CLD

PARTICULARS OF PATIENT:

Name: Md. Kasem
Age: 35 years
Sex: Male
Marital status: married
Occupation: Bus driver
Religion: Islam
Address: Dhobaura , Mymensingh
Date of admission: 1.12.09 at 7pm
Date of examination: 3.12.09 at 10 am

Presenting complaints

Abdominal distension for 6 month
Yellow coloration of skin and sclera for 2 month

History of present illness

According to the statement of patient he was relatively well 6 months ago then he developed generalized swelling of whole body which first noticed at abdomen gradually spread whole over the body**.** For the last the 2 months the patient developed yellow coloration of skin, eye and urine .This was not associated with vomiting, nausea, joint pain and itching .Color of stool was not pale. The patient has no history fever and abdominal pain, vomiting out of blood and black tarry tool, alter level of consciousness and alteration of sleep pattern. The patient also had no history of transfusion of blood and blood product and no history of abdominal surgery previously .For the last few month he gradually losing his body and pubic hair and decreased frequency of saving .the patient had history of shaving in salon and was unaware about using of disposable blade every time . But patient complained of loss libido. The patient's urine out put is normal and bowel moves once daily. The patient has no alternation bowel habit and passage of mucus with or without blood. The patient had no history of breathlessness on exertion, rest or in lying position. Now patient is anorexic and with above complaint he was admitted into medicine uint-1 MMCH. He also gave history aspiration of fluid from his abdomen twice after admission in medicine unit-1 and color of the fluid was clear ..

History of past illness

No previous history of jaundice. He had no history hypertension and Diabetes. He had no history of chronic lung disease or heart disease

Drug history

The patient has no significant drug history

PERSONAL HISTORY

The patient is Non smoker, Non alcoholic. And have no history IV drug user. The patient had history multiple extra marital sexual exposure

FAMILY HISTORY
None his family member is suffering from this type of disease.
They are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION
He comes from low socioeconomic condition and lived in crowding house
Housing: Tin shade house.
2 rooms, which accommodate 8 0f
His family member.
Sanitation: 1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history
The patient was not immunized against hepatitis B

GENERAL EXAMINATION

- **Appearance –hepatic face (sunken eye ball , malar prominence, muddy color)**
- Body built – average
- Nutritional status –average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – Absent
- **Jaundice : mild**
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- **Leukonychia : present**
- Dupuytren's contracture : Absent
- **palmar erythema: Absent**
- **hepatic flap / flapping tremor : absent**
- **Spider nevi : present**
- **Gynaecomastia : present**
- **Oedema : + + + +**
- Dehydration – Nill
- **Skin –general skin condition and is normal . no evidence of scratch marks and generalized pigmentation . there is bandage on right iliac fossa .**
- **Body hair distribution –loss of axillary & pubic hair**
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 130/80 mm of Hg
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :
- **Bed side urine examination show s no proteinuria**
  Sugar= Nill

Following general examination are to be mentioned
Appearance
Jaundice
Leukonychia
Palmar erythema flapping tremor
Spider nevi , Gynaecomastia
Oedema
Skin
Bed side urine examination to exclude DD (NS)
Body hair distribution

Systemic examination

Examination of Alimentary system

■**Mouth & pharynx**

■Tongue – normal

■**Abdomen Proper**

■**Inspection**

■Abdomen – distended and flanks are full

■Umbilicus – centrally placed and everted and slit is transverse

■Visible vein – there is several engorged vein at upper abdomen and direction of flow upward

■Movement with respiration – present

■Scar mark – no scar mark and no striae

■Visible peristalsis – absent

■Hernial orifice – intact

■Hair distribution – loss of pubic and axillary's hair

■External genitalia – scrotal swelling due to edema

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,

■ Liver and spleen cannot be delineated as due to huge ascites

■Fluid thrill present (but may absent if ascites is mild)

■Testicular atrophy (when said it – if it small , soft . pain less)

(If spleen is palpable which is 5 cm from left costal margin at ant? Axillary line toward its long axis having smooth surface , firm in consistency , non tender ,notch can not be identified / there is a notch at its upper border and there is no splenic bruit—but in this patient spleen is not palpable)

■**Percussion**

■Shifting dullness is present

(Paddle sign in case of mild ascites)

■**Auscultation**

■Bowel sound –present

CARDIVASCULAR SYSTEM

•Pulse : 88 b/min

Regular, normal in volume & character, all the peripheral pulses are normal.

•BP : 130/80 mm of Hg

•**JVP : Not raised**

•**Precordium** :

Inspection: Normal

Palpation: Apex beat in lt 5th intercostals space 9 cm from midline

No para-sternal heave and no palpable

AuscultationS1&S2 audible in all auscultatory area

No added sound and no murmur

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Movement is symmetrical

•No evidence of respiratory distress

•**Palpation** :

Trachea: Trachea central

Apex beat: in left 5th intercostals space 9 cm from midline normal in character

Vocal fremitus : normal

Percussion: resonance

Auscultation:

Breath sound is vesicular in all parts of the chest.

No added sound

Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.

Fundoscopic exam: Normal

Cranial nerves: intact

Motor system examination

Motor functions are normal in all four limbs

Sensory examination

All modalities of sensation are intact in both upper and lower limbs

Cerebellar signs: Absent

Signs of meningeal irritation: Absent

Salient feature:

Md. Kasem 35 yrs old Normotensive ,Nondiabetic, Nonalcoholic ,Nonsmoker Muslim bus driver hailing from Dhobaura , Mymensingh got admitted into MU-1 MMCH with gradual distension of abdomen followed by generalized pitting edema for 6 months and jaundice for the last 2 months . Jaundice is mild, non progressive in nature without any prodomal sign and symptoms like nausea, vomiting or malaise. The patient had no history of fever, abdominal pain, pruritus, pale stool, haematemesis, malaena, transfusion of blood and blood product and no any major surgery .The patient had history of multiple extramarital sexual relations but he denied any IV drug abuse. For the last few months he gradually losing his body hair and libido .The patient urine out put is normal and bowel moves once daily with semi-solid consistency. The patients had no history unconsciousness or alter mental sate previously .He used to shave in salon with unawareness of one time blade or razor. The patient had no history alteration of bowel habit, mucus stool with or without blood, Dyspnea in exertion and rest or orthopnea, chronic cough and old TB. After admission in medicine unit -1 his paracentesis had done twice color of which was serous. The patient is fully conscious and oriented General examination reveals the patient have hepatic faces with mild jaundice, leukonychia, spider nevi, gynaecomastia, moderate pitting edema and bandage over right iliac fossa. The patient has no anaemia, palmer erythema ,flapping tremor with normal blood pressure, JVP not raised. Alimentary system examination reveals huge ascites as shifting dullness and fluid thrill present .with engorged vein over epigastric region with upward flow. Liver and spleen can not be delineated due to huge ascites and both testis are atrophied .Bed side urine examination reveals no sugar and protein

Provisional diagnosis

Decompensate chronic liver disease

Differential diagnosis

Nephrotic syndrome
Congestive cardiac failure

Investigation

What investigation u wants to done in case of CLD?

Liver function test

SGPT------N / ↑
S.Bilirubin --- N / ↑
Prothrombin time— N / ↑
s.ablumin----↓
AG ratio---- alter

Viral marker

HBs Ag
Anti-HBc Ig G
Anti-HCV

RBS----- normal
Urine RME --- no proteinuria or no RBC

Imaging

USG of HBS and pancreases—coarse echo structure, Splenomegaly, ascites
Ascitic fluid study – Transudative and SAAG > 1 .1
CXR-PA
ECG
Urine copper (Wilson's disease),
Serum ferritin in case of haemochromatosis
Endoscopy of upper GIT --- to see varcies

Following will be the provisional diagnosis :

If the patient present with only ascites

Then provisional Dx :
Decompensate chronic liver disease

If pt has ascites with engorged vein or splenomegaly

Then provisional Dx :
Decompensate chronic liver disease with portal HTN

If patient present history of either fever or abdominal pain or both

Then provisional diagnosis will be
Decompensate chronic liver disease with SBP

If the patient ascites with haematomesis and melaena

Then provisional Dx :
Decompensate chronic liver disease with portal HTN with rupture esophageal varices

Sometime u may get onlyCLD hav only ascites no edema :

Then provisional diagnosis will be

- Decompensate chronic liver disease

DD will b

- Abdominal Tb
- Intra abdominal malignancy metastasis to peritoneum

Treatment decompensate CLD

Diet
Salt restriction
No fluid restriction until < 120 m mol / l
Diuretic
Combination spirolactone and frusemide
Paracentesis
Aspiration of 2-5 L/ d ascitic fluid is safe.
If more than 5 L is done in one day then need 6-8 gm albumin for each litre .
Syp. Lactulose is given for bowel movement .
Treatment of complication such as
SBP
Hepato-renal syndrome
Specific R_x -- liver transplantation

Sometime u may get pt of CLD with palpable liver:

- Then provisional diagnosis is :
  - Hepatoma (or HCC) on the top of CLD
- Differential diagnosis
  - Secondaries in the liver and metastasis in the peritoneum

What is ur provisional diagnosis

My provisional diagnosis is Decompensated chronic liver disease

What are the points in favour of ur provisional diagnosis

Following points are favour my diagnosis

- ✓ The patient has history of generalized edema that appear at abdomen first and loss libido & body hair
- ✓ HO of multiple sexual exposure
- ✓ On general examination patient has stigmata CLD such as
  - Hepatic faces
  - Jaundice
  - Leukonychia.
  - Spider nevi
  - loss of axillary and pubic hair,
  - Gynaecomastia.
  - Edema
- ✓ Examination of abdomen reveals that
  - Ascites
  - Engorged vein in upper abdomen with up ward flow of direction
  - Testicular atrophy

Box -a

What are sign of hepatic insufficiency?

Hepatic faces (sunken eye, Malar prominent)
Jaundice
Flapping tremor
Gynaecomastia
In case of female breast atrophy
Spider nevi
Loss of body and pubic hair
In hand
leukonychia.
Dupuytren's contracture
Palmar erythema
Testicular atrophy

When will u called the testes are atrophied?

If the testes are soft, small and loss of pain sensation.

Box- b

What are the signs of portal hypertension?

To remember it keep mind SEA

S—Slepnomegaly

E—Engorged vein

Abdomen –

Above umbilicus direction of flow – upward

Below umbilicus direction of flow – down ward

Caput medusa –arround the umbilicus, direction of flow – away from the umbilicus

Esophageal varices –

Clinically –haematomesis and malaena

Via upper GIT endoscopy

A—Ascites

Other • Fetor hepaticus ,Hepatic encephalopathy

What is the cause of CLD in this patient?

Due to chronic viral hepatitis

What are the culprit viruses in this pt.?

Hepatitis B and C virus

What type of virus is hepatitis B is?

It is DNA virus.

Between HIV @ HBV which one is more infectious?

HVB is more infectious than HIV

Why Decompensate CLD

Because patient has

- **J**--Jaundice
- **E**--Encephalopathy and
- **A--**Ascites

To remember it **JEA**

- Next Question will be what are the sign of hepatic insufficiency u got in this patient ?

  Write the positive sign u got in ur patient from box a

- Next Question will be what are the sign of portal HTN u got in this patient ?

  Write the positive sign u got in ur patient from box b

What is ur differential diagnosis what are point in favour of ur diagnosis ?

The point in favor of CCF is
only dependent edema
Point against
Other sign and symptoms CCF is absent such
No history exertional dyspnea . orthropnea , chronic cough with productive sputum
On Exam –
most important
JVP not raised
No tender hepatomegaly
Others sign
lung crep + , spasm ,ronchi , vesicular breath sound with prolong expiration – are absent
murmur , left parasternal heave absent

why this is not a case of NS ?

point in
is favor is only generalized edema

point against are
no urinary complaints
in bed side heat coagulation test – protein is absent
the patient has stigmata of CLD

What is cirrhosis ?Cirrhosis is defined pathologically as a diffuse liver abnormality characterized by fibrosis and abnormal regenerating nodules
•Micronodular cirrhosis ---nodules about 1 mm in diameter (seen in alcoholic cirrhosis.)
•Macronodular cirrhosis – nodules is about > 1 mm in diameter
Mixed --- Both micro and macro nodular
Which cell is activated during cirrhosis ?
activation of **the hepatic stellate** cells

CAUSES OF CIRRHOSIS
TO remember ---abcdefghi
A--- Alcohol
B---Biliary
Primary biliary cirrhosis
C--- Chronic viral hepatitis (B or C)
D---Drug
E--- Endocrine - Wilson's disease
F--- Non-alcoholic fatty liver disease
G—genetic- α1-antitrypsin deficiency
H--- Haemochromatosis
I--- Autoimmune liver disease/hepatitis
Primary sclerosing cholangitis

Complication of CLD
1. Hepatic encephalopathy.
2. Ascites
3. Spontaneous bacterial peritonitis.
4. Hepatorenal syndrome.
5. Hepatopulmonary syndrome
6. Hepatocellular carcinoma
7. Portal hypertension.
   - Variceal haemorrhages.
   - Portal gastropathy
8. Coagulopathy and feature of hypersplenism

If u got a spider nevi following question may be asked ?

- Spider telangiectasia is a central arteriole from which small vessels radiate.
- Site : usually found only above the nipples along the area of superior venacava distribution
- Normally found: 1or 2 in 2 % people
- Cause due to: hyper dynamic circulation . in case of CLD due to access oestrogen as metabolism of oestrogen decreased by diseased liver.

  Other cause pregnancy , viral hepatitis ,OCP ,

  thrytoxicosis ,
- How will u see it

  With the help of pin head or glass slide
- How will differentiate between purpura and spider nevi
  - Purpura does not blanch on pressure (as it extravascular)
  - Spider nevi : Blanch on pressure and when release the pressure it will reappear

What is gynaecomastia

Enlargement of male breast tissue due to proliferation of glandular component.

To remember --- $BLAST_3$

B—Bronchogenic carcinoma
L—chronic liver disease
A—Adrenal carcinoma
S—spirolactone
T_1--Testicular tumour (leydig cell),
T_2-- Testicular failure (trauma, orchitis, radiation)
T_3---Thrytoxicosis
Mechanism: Either due to increase activity of oestrogen or decrease activity of testosterone

Name some drug responsible gynaecomastia

Spirolactone , cemitidine , digoxin

Cause of gynaecomastia is in CLD

Due CLD it self @ drugs spirolactone

Painful gynaecomastia found in

Spirolactone

<u>FACTOR PREDISPOSING HEPATIC ENCEPHALOPATHY</u>

To remember it BCDEF minus TOP

B—Bleeding from GIT haematemesis @ malaena
C--- Constipation
D---drug sedative, hypnotic, NSAID,
E---electrolyte imbalance, hypokalaemia
F—Fever indicate infection

Minus top

T--- Trauma
O—operation

What follow u will give in a patient with CLD in ur ward ?

- Level of consciousness –orientation & alternation sleep rhythm and
- Jaundice
- Dehydration
- Flapping tremor
- Pulse , BP, Cyanosis
- Abdomen
  - Abdominal pain
  - Percussion distension
  - Bowel sound
  - Fever / Temp.
  - Constipation / bowel pass
  - Bladder (urine out put)
  - Rebound tenderness
- Abdominal girth
- Planter extensor
- Daily weight

What the clinical signs are of decompensate CLD?

To remember JEA

J—jaundice

E—Encephalopathy

A---Ascites

From **The Child-Pugh** score we can identify whether this CLD is compensated or decompensate

To remember it PABA-encephalopathy

	Point 1	Point 2	Point 3
P--Prothrombin time	< 4	4-------6	> 6
A--Albumin	> 3	2.5----3.5	> 2.5
B--Bilirubin	< 2	2-------3	> 3
A--Ascites	None	Mild to moderate	Marked
E--Encephalopathy	None	1 / 2	3 / 4

Child 1 = < 7 = well compensated

Child 2 = 7--9 = slightly decompensate

Child 3 = > 9 = decompensate

Bad prognostic features are

- Increasing plasma bilirubin,
- Falling plasma albumin or
- An albumin concentration < 30 g/l,
- Marked hyponatraemia (< 120 mmol/l, not due to diuretic therapy) and
- A prolonged prothrombin time.

Is hepatomegaly present in CLD

No , Usually is liver is small due to fibrosis

What are the condition where cirrhosis is associated with hepatomegaly ?

- Alcoholic cirrhosis
- Haemochromatosis
- Primary biliary cirrhosis

Feature of alcoholic cirrhosis

- Florid spider telangiectasia,
- Gynaecomastia
- Parotid enlargement
- Hepatomegaly

What is 1st neurological sign in encephalopathy ?

- Constructional apraxia (ask to draw star)

How the portal vein is formed

It is formed by combination superior mesenteric vein and splenic vein

What is the normal portal pressure?

- **Normally 2-5 mmHg**
- Clinical features Developed when portal venous pressures **above 12 mmHg**

What are blood supply of liver

Liver has dual blood supply

2/3 is supplied by portal vein rich in nutrients

1/3 is supplied by hepatic artery rich in O_2

Oxygen supply of liver

Half of the oxygen supply is met by the portal vein and rest half by the hepatic artery

COMPLICATIONS OF PORTAL HYPERTENSION

- Variceal bleeding (Oesophageal)
- Congestive gastropathy
- Hypersplenism
- Ascites
- Renal failure
- Hepatic encephalopathy

Classification of acute hepatic failure

Hyperacute	< 7 days
Acute	8-28 days
Subacute	29 days-12 weeks

Site of proto-systemic anastomasis

Site	Portal vein	Systemic circulation
Oesophageal (varices)	Left gastric vein	Hemi-azygos vein
Para umbilical (caput medusa)	paraumbilical veins	superficial epigastric vein
Rectal (hemorrhoid)	superior rectal vein	middle and inferior rectal veins
Bear area of liver		
Retroperitoneal		

Management of esophageal Varices
Restoration of circulation with blood or plasma
For bleeding control
Local
- Banding or sclerotherapy,
- Balloon tamponade and
- Oesophageal transaction

Pharmacological
Terlipression and octreotide
Prevention
- Transjugular intrahepatic portosystemic stent shunting (TIPSS)
- Pharmacological
  Beta-adrenoceptor antagonists (β-blockers)
  propranolol
- Banding

How propanolol act
It reduce portal HTN by splanching vasodilation .

Dose
Dose is 80 – 160 mg / day ,tritrate the dose up to reduction of 25 % of Basal pulse rate

How will diagnosed oesophageal varices ?

By upper GIT endocopy

PROGNOSIS
- The overall prognosis in cirrhosis is poor.
- only 25% of patients survive 5 years from diagnosis
- Where liver function is good, 50% survive for 5 years and 25% for up to 10 years.

What do you know about the serum-ascites albumin gradient **SAAG?**
- It is calculated by subtracting the ascitic fluid albumin concentration from the serum albumin concentration from samples obtained at the same time.
- SAAG >1.1 g/dl indicate portal hypertension
- SAAG< 1.1 g/dl do not portal hypertension
- The accuracy of such determinations is 97%

What investigation u want to done in case of CLD
Liver function test
SGPT------N / ↑
S.Bilirubin --- N / ↑
Prothrombin time— N / ↑
s.ablumin----↓
AG ratio---- alter
Viral marker
HBs Ag
Anti-HBc Ig G
Anti-HCV

daily accepted wt loss only in ascites
ascites –**0.5** kg
with peripheral oedema 1 kg

Dose	minimum	max
frusemide	40 mg	160 mg
spirilactone	50-100	400

what is refractory ascites
failure to decrease wt loss 0.5 kg/d after 1 wk of max dose of combin diuretic (f-160 , s—400)

paracentesis
aspiration of ascitic fluid is called paracentesis
- can draw 2-4 l fluid /day with out albumin

Don't draw fluid if patient in encephalopathy

Imaging
USG of HBS and pancreases—coarse echo structure, splenomegaly , ascites
Ascitic fluid study – Transudative and SAAG > 1 .1

urine copper (Wilson's disease),
Serum ferritin in case of haemochromatosis
Endoscopy of upper GIT --- to see varcies

Ascetic fluid study
Color
- straw – TB
- Turbid / pus –peritonitis
- Hemorrhagic—malignancy
- Serous -- transudative **/ CLD / NS**
- milky-white chylous--Lymphatic obstruction:

Biochemical
See protein and glucose
- If protein more than > 3 gm Exudative or (2.5)

Cytology
See inflammatory cell
Neutrophil and lymphocyte
Malignant cell

Micro biological
GM stain and AFB stain

Common causes
- Malignant disease
  - Hepatic
  - Peritoneal
- Cardiac failure
- Hepatic cirrhosis

Exudative cause
Infection
Tuberculosis
Spontaneous bacterial Peritonitis
Malignancy
Budd-Chiari syndrome
hepatic venous obstruction
Pancreatitis
Lymphatic obstruction
Spontaneous bacterial peritonitis
Hypothyroidism

Transudative
Nephrotic syndrome
CLD
CCF
Meigs' syndrome
Hypoproteinaemia
Malnutrition @
Protein-losing enteropathy

A patient with splenomegaly with ascits
- CLD
- Lymphoma leukaemia
- Dessiminated TB

A patient with hepatomegaly with ascitis
- CCF
- Hepatoma with secondary in the peritoneum
- Lymphoma
- Dessiminated TB
- Chirrohsis of liver with portal HTN

CLINICAL GRADING OF HEPATIC ENCEPHALOPATHY
Grade 1 **Poor concentration, slurred speech**, slow mentation, disordered sleep rhythm
Grade 2 **Drowsy but easily rousable**, occasional aggressive behaviour, lethargic
Grade 3 Marked confusion, **drowsy, sleepy but responds to pain and voice**, gross disorientation
Grade 4 **Unresponsive to voice**, may or may not respond to painful stimuli, unconscious

Acute viral hepatitis	Chronic viral hepatitis / CLD
Clinical	
Prodrome present (nausea / vomiting /anorexia)	No prodome
Short HO < 1 month Jaundice present	Usually absent if present duration is > 3 month
No stigmata of CLD	Stigmata CLD present Ascites , splenomegaly , spider and gynaecomastia , testicular atrophy
Bio-chemical	
Prothrombin time increased	Prothrombin time increased in Acute on chronic
Albumin and A:G ratio normal	hypoAlbuminia and A:G ratio alter
Viral marker HBs Ag + < 6 months	Viral marker HBs Ag + > 6 months
Anti-HBC I_g G negative	Anti-HBC I_g G positive
Imaging (USG)	
Shows liver Hypo echoic Inflammation of gall bladder (Chloe cystitis) Normal	Coarse echo structure Ascites @ or Spleno-megaly

Treatment decompensate CLD

Diet

Salt restriction

No fluid restriction until < 120 m mol / l

Diuretic

Combination spirolactone and frusemide

Paracentesis

Aspiration of 2-5 L/ d ascitic fluid is safe

If more than 5 L is done in one day then need 6-8 gm albumin for each litre

Syp. Lactulose is given for bowel movement

Treatment of complication such as

SBP

Hepato-renal syndrome

Specific R_x -- liver transplantation

A patient with CLD suddenly comes to u recently appearing lump in the right upper abdomen .what is ur diagnosis ?

- Hepatocellular carcinoma

Anti – viral therapy

See history file of viral hepatitis

If patient of CLD developed back pain or bone pain what is ur Dx ?

- Hepatic osteodystrophy

If patient with developed cyanosis then what is ur Dx ?

- Hepato-pulmonary syndrome

If a patient comes to u with ascites and jaundice for 6 wk and flapping tremor what is ur Dx ?

Sub acute hepatic failure

Spontaneous bacterial peritonitis (SBP)
Infection of ascitic fluid in absence of primary source.
Usually by single organism.
Clinical feature is to remember -- BAFAR
B—Absence of bowel sound,
A- ascites,
F—fever,
A –abdominal pain,
R—rebound tenderness
Aspiration of fluid :
Shows cloudy –exudative (protein ↑and glucose↓)
neutrophil count >250 mm ^{3}
culture – single organism mostly *Escherichia coli*

Treatment
- Broad spectrum antibiotic
- Injectable Cefotaxime or ceftriaxon

For prevention
- Prophylactic antibiotic
- Norfloxacin (400 mg daily) or Ciprofloxacin (250 mg daily)

In secondary bacterial infection
Neutrophil > 10000
Culture multiple shows organism

Q if a patient with CLD but u hav not find any cause specially then what may be the other causes ?

In a patient with CLD if no underlying causes are found then we have to search for the following

- Wilson disease
- Haemochromatosis
- Auto-immuno hepatitis

Q if patient with young with will u look for? Or what will u look for in a patient with CLD due to Wilson ?

If u suspects any patient of CLD due to Wilson disease then looks for the following:
Take family history
Young age
Neurological manifestation such
- Dementia ,
- Tremor

What will u see in the eye?
- I will see the K-F ring in eye

What investigation you want to do?
- Serum ceruloplasmin—decreased
- Urinary copper ----increased
- Biopsy ---

If CLD patient comes to u with anemia or pancytopenia what may be the causes?
- Hypersplenism

Why not autoimmune hepatitis is the cause of CLD in this patient?
- No autoimmune hepatitis is not the cause of CLD in this patient
- Because autoimmune hepatitis occur in
  - Mainly female
  - Younger age
  - Associated with other autoimmune diseases such Thyroid , DM,

We take the history for the following reason

- To establish diagnosis (CLD)
- To exclude differential diagnosis (CCF , NS ,abdominal TB)
- To find out it etiology (viral , alcohol , willsion , autoimmune)
- To see complication (rupture esophageal varices)

If ur pt has only ascites with generalized edema then

Provisional diagnosis is the decompensated CLD and

DD – NS & CCF

If ur pt has only ascites then

Provisional diagnosis is the decompensate CLD and

DD –abdominal TB or intra-abdominal malignancies

Following history should be taken

swelling of body,	**where it appear first , face or leg or abdomen**
recent or past history of jaundice	
if jaundice present	**then take ho of vomiting, nausea, joint pain (for viral hepatitis)**
	then take HO Itching .Color of stool pale or not to see obstructive jaundice
to see the etiology	**H/O transfusion of blood and blood product (HBV)** **abdominal surgery previously (biliary cirrhosis) ,** **shaving in salon & using of disposable blade or not**
HO of hepatic insufficiency	**Loss of body and pubic hair and decreased frequency of saving & loss libido**
to exclude DD	**The patient has no alternation bowel habit and passage of mucus with or without blood (TB & malignancy)** **history of breathlessness on exertion, rest or in lying position or cough (CCF) and no urinary complained (NS)**
To see complication of CLD	**fever and abdominal pain(SBP),** **vomiting out of blood and black tarry tool(rupture esophageal varices) ,** **alter level of consciousness and alteration of sleep pattern(encephalopathy)** **urine output (hepato renal)and** **Bowel moves per day (as constipation is risk for hepatic encephalopathy)**
HO of aspiration of fluid and how many time and its color	
past HO jaundice	
PERSONAL HISTORY	**Alcoholic. IV drug user, multiple extra marital sexual exposure to see etiology**
FAMILY HO	**Patient's partner is suffering from jaundice or not**
immunization HO	**immunized against HBV or not**

Bronchial Asthma

Long case of COPD and bronchial asthma:

- ✓ **If patient is young and non-smoker (may be smoker)**
  - ➢ Provisional diagnosis: Acute severe asthma
  - ➢ Differential diagnosis: Acute exaggeration of COPD

- ✓ **If patient is old and smoker**
  - ➢ Provisional diagnosis: Acute exaggeration of COPD
  - ➢ Differential diagnosis: Acute severe asthma

Age	young —asthma , old > 40-COPD
Onset	in both onset is gradual
	family history -bronchial asthma
	atopy — bronchial asthma
Bronchial asthma patient has some triggering factor that provoke the symptoms	such as • Tree and grass pollen, Cold air exposure • Cat and dog dander, • Food allergen , drugs NA1D • Viral RTI
	Orthropnea , paroxysmal nocturnal dyspnea or exertional dyspnea
Cough	Productive (COPD/ br asthma)or non productive (br asthma)
	Amount 1/4 cup per day
	• Nature mucoid , whitish — asthma • Purulent, yellow color -COPD • Foul-smelling or not • Blood stained • Relation with posture — brochiectasis • Diurnal variation —asthma -more in the morning • Episodic – asthma • Persistent and most of day –COPD
Smoking	Positive in COPD , how many stick per day ,for how long
Chest pain	
Fever	
Drug history	on going -medication or previous HO inhaler

PARTICULARS OF PATIENT:
Name: Samir Kumar Das
Age: 50 years
Sex: Male
Marital status: Married
Occupation: Farmer
Religion: Hindu
Address: Fulbaria, Mymensingh.
Date of admission: 8.12.13 at 7pm
Date of examination: 10.12.13 at 7.15am

PRESENTING COMPLAINT:
Respiratory distress for 7 days
Cough with sputum for 7 days

HISTORY OF PRESENT ILLNESS:
According to the statement of the patient he was reasonably well 7 days back then he gradually developed intermittent breathlessness. Initially it was mild to moderate in nature which subsided after taking inhaler (only mention if patient use it) but for last one day it increased in severity and is not responding to inhaler. The patient's breathlessness is so severe that he could not speak full sentence. The breathlessness has no association with exertion, lying posture or no has history of sudden severe breathlessness that woke him from the sleep. The patient has similar type's episodic attack for last 10 years most of them were not severe as like this which subsided after taking inhaler or some oral medication name of which he cannot mention. On query, patient gives history of some trigger factors for breathlessness such as tree and grass pollen, cat and dog dander, cold air exposure, perfumes, and bleaches and food allergens. The patient also noticed that his symptoms are more marked on winter and early summer seasons and also worsens at night. The patient also complained of cough for same duration. The cough was productive in nature (dry if patient mention), contain viscous mucoid sputum, less than 1/6 cup amount in 24, whitish in colour, not blood stained, no relation with posture change but worsen at morning. This cough also aggravated in exposure to above mentioned trigger factors. The patient also complained of episodic running nose, sneezing, localized transient skin swelling (urticarial, pain and fever) exposure to above mentioned allergens. But the patient has no history of chest pain and fever. Patient has no history of leg swelling (cor pulmonale). But for the last few months the frequency of this type of attack was increasing but most of the attacks was relieved at home after medication. This attack is so severe that he has to admit in this hospital under MU-I for emergency management.

HISTORY OF PAST ILLNESS:
No history of DM/HTN/TB
Patient has previous history of attack of breathlessness for several occasions for which he has to admit in this hospital.

PERSONAL HISTORY:
Occasional non-smoker
Non-alcoholic
FAMILY HISTORY:
One of sisters is suffering from bronchial asthma.

SOCIO-ECONOMIC CONDITION:
He comes from low socio-economic condition and lived in crowding house.
Housing: Tin shade house.
3 rooms which accommodate 8 of his family member.
Sanitation: 1 sanitary latrine.
Water supply: Arsenic free tube-well water.

IMMUNIZATION HISTORY:
The patient is immunized according to EPI schedule.

DRUG HISTORY:
Patient is taking two types inhaler and some oral medications name which he cannot mention.

GENERAL EXAMINATION:

a) **Appearance**: Ill looking, dyspnic
b) **Body built**: Average
c) **Nutritional status**: Below average/ average
d) **Decubitus:** On choice but feels better in sitting
e) **Co-operation**: Well co-operative
f) **Anaemia:** Absent
g) **Jaundice:** Absent
h) **Cyanosis**: Absent/ may present
i) **Clubbing:** Absent
j) **Koilonychia**: Absent
k) **Leuconychia**: Absent
l) **Oedema:** Absent
m) **Dehydration**: Moderate
n) **Skin**: Pale , thin and wrinkled
o) **Body hair distribution**: Normal
p) **Bony tenderness**: Absent
q) **Lymph node**: No lymphadenopathy
r) **Thyroid gland**: Not palpable
s) **Neck vein**: Not engorged
t) **Pulse**: 110/minute, low volume, regular
u) **BP:** 120/85 mm of Hg
v) **Respiratory rate**: 30/minute
w) **Temperature**: 98 degree F
x) **Weight**: 45kg
y) **Height**: 1.6 m
z) **BMI**: 17.57 kg/m^2
aa) **Flapping tremor**: Absent

REPIRATORY SYSTEM:

- **INSPECTION:**

Size and shape of the chest: Normal
Evidence of respiratory distress: -Intercostal fullness or recession/ in drawing.
-Suprasternal, supraclavicular excavation

No asymmetry, scar mark, visible impulse and engorged vein, gynaecomastia and spider nevi and pigmentation.

- **Palpation:**

Trachea: Central
Apex beat: Left 5th intercostal space 9 cm from midline, normal in character.
Vocal fremitus: Normal.

- **PERCUSSION:** Resonance
  Upper border of liver dullness is 5th intercostals space.

- **AUSCULTATION:**
  Breath sound is vesicular with prolong expiration.
  Added sound: Expiratory Ronchi and Wheeze present all over the lung field .
  Vocal resonance: Normal.

CARDIOVASCULAR SYSTEM:

Pulse: 72 beats/min.
Regular, normal in volume and character, all the peripheral pulses are normal.
BP: 120/75 mm of Hg
JVP: Not raised

PECORDIUM:
Inspection: Normal
Palpation: Apex beat in it 5th intercostal space 9 cm from midline.
Auscultation: S1 and S2 audible in all auscultatory area. No added sounds.
ALIMENTARY SYSTEM:
Inspection:
-Mouth and oral cavity: Normal
-Abdomen proper: Normal
Palpation:
-Liver, spleen, kidney: Not palpable
-No intra abdominal lymphadenopathy or palpable lump.
Percussion: Tympanic
Auscultation: Bowel sound present
Testes: Normal
D/R/E: Normal

SALIENT FEATURE:
Samir Kumar Das, 35years old normotensive, non-diabetic, non-alcoholic, non-smoker, Hindu farmer hailing from Fulbaria, Mymensingh got admitted into MU-1 MMCH with gradual development of dyspnea and cough for 7 days. Initially it was mild to moderate in nature then turned severe form which not responds to bronchodilator. He has no history of orthopnea, paroxysmal nocturnal dyspnea or exertional dyspnea. Patient is suffering from this type of episodic intermittent dyspnea for last 10 years. This dyspnea have some precipitating factors like tree and grass pollen, cat and dog dander, cold air exposure, perfumes and bleaches and food allergens. These worsen at night and winter season. Patient also complained productive coughs with mucoid sputum which worsen at early morning. The patient also has history of atopy and one of his family member is

suffering from bronchial asthma. The patient is non- smoker and has no history of fever or chest pain. With this type of attack he had to admit several times in hospital. General examination reveals the patient is ill looking, dyspnic, Pulse-110/min, low volume, regular, Respiratory rate- 30/min, cyanosis, cyanosis, oedema, flapping tremor are absent and eye is not congested, Neck vein- not engorged. Respiratory system examination reveals evidence of respiratory distress like intercostal recession/ in drawing and supraclavicular, suprasternal excavation. Size and shape of chest wall is normal, trachea is central and apex beat is normal in position. Percussion reveal upper border of liver dullness is in 5^{th} intercostal space with normal percussion note all over the chest. Auscultation reveals vesicular breath sound with prolong expiration with expiratory ronchi and wheeze all over the chest. No crepitation is present.

PROVISIONAL DIAGNOSIS:
Acute severe asthma

DIFFERENTIAL DIAGNOSIS:
Acute exaggeration of COPD

INVESTIGATION:

Pulmonary function test:

-The diagnosis is certain if:

- 20% diurnal PEF variation on >3 days per week, in a week of peak flow diary measures.
- FEV_1>15% decrease after 6 minutes exercise.
- FEVA>15%(and 200ml) increase after 2 week trial of oral steroid (30mg prednisolone od)

-Bronchodilator reversibly testing FEV_1>15%(or 200ml)increase after short-acting beta agonist therapy.

Blood:

- Total eosinophil count: Increased
- Serum total IgE is typically elevated in atopic asthma.
- Skin prick tests are simple and provide a rapid assessment of atopy.
- Metacholine/ histaminechallenge measures bronchial hyperresponsiveness.
- Sputum analysis: Sputum eosinophilia
- CXA- Normal
- ECG-Normal

Treatment:

1. High flow O_2 inhalation
2. Nebulization stat and sos or 4/6 hourly
   (Sulbutamol .sol 1ml+1mlipratropium .sol +2ml normal sal.)
3. Inj. Hydrocortosone
   2 amp. IV stat and 1 amp. IV 6 hourly
4. Salbutamol inhaler
   2 puff qds
5. Beclomethason inhaler
   2 puff tds
6. Tab. Montelucast 10mg
   0+0+1

✓ **What is your provisional diagnosis?**

My provisional diagnosis is acute severe asthma.

✓ **What are the points in favor of your diagnosis?**

1. **In history:**
   - Gradual onset of dyspnea with cough
   - Young age
   - Non-smoker
   - Family positive
   - History of atopy
   - Dyspnea provoke in response to some triggering factors like
     - Tree and grass pollen
     - Cold air exposure, perfumes, and bleaches and food allergens
     - These worsens at night and winter season
   - Cough is mucoid and having diurnal variation
   - General examination:
     - Dyspnic
     - Tachycardia (Pulse-110/min)
     - Tachypnea (Respiratory rate-30/min)

2. **Respiratory System:**
   - Intercostal recession/ in drawing and Suprasternal, Supraclavicular excavation
   - Vesicular breath sound with prolong expiration
   - With expiratory ronchi and wheeze all over the chest

✓ **What is differential diagnosis?**

Acute exaggeration of COPD

The point in favor in diagnosis:

- Gradual onset of dyspnea and cough with productive sputum
- Vesicular breath sound with prolong expiration
- With expiratory ronchi and wheeze all over the chest

Point disfavor of diagnosis:

In history:

- Young age
- Non-smoker
- Family positive
- History of atopy
- Dyspnea provoke in response to allergen

On examination:

- Eye is not congested and hand is not warm and absence of bounding pulse
- Absence of barrel shaped chest
- Upper border of liver dullness is not lowered down
- Percussion note is not hyper-resonance

✓ **Write difference between Asthma and COPD.**

Write difference between Asthma and COPD

Asthma	COPD
age —young	old usually, after 40
family history , atopy HO of triggering factor such as tree and grass pollen, cat and dog dander, Cold air exposure, drugs NAID , viral RT1 may provoke the symptoms	usually not , but. respiratory tract infection aggravate fee symptoms
cough usually non productive or mucoid sputum, .	Cough are productive
have diurnal variation more marked in the morning	not so
general examination tachycardia, and tachypnea in sever case	usually feature of Type II Resp. failure Eye- congested • Tongue -cyanosed • Palm— warm • Pulse -bounding pulse • Flapping tremor may present
Respiratory exam	
not so	lip pursing, prominence of accessory muscle of neck , engorge neck vein ,
apex beat palpable	in emphysema not palpable
upper border of liver dullness is normal	upper border of liver dullness is lower in emphysema
crepitating absent	may present
feature of pulmonale HTN and corpulmonale	
not so	palpable-JP2
	left para sternal heave
	epigastric pulsation
	loudP2
	tender hepatomegaly raised JVP depended edema
investigation The diagnosis is certain if: • 20% diurnal PEF variation on >*3* days per week, in a week of peak flow diary measures. • FEVl > 15% decrease after 6 minutes exercise • FEVl > 1 5% (and 200 ml) increase after 2 week trial of oral steroid (30 mg prednisolone od) Bronchodilator reversibility testing FEVl > 15% • CXR and ECG normal	Reduced FEV, to <80% predicted) FEV,/FVC<0.7 N CXR -- Hyper inflated lung fields with Low Flat diaphragm with tubular heart EGC — P— pulmonale , RVH and poor progression of R wave

- ✓ **Define asthma.**
  - Asthma is characterized by chronic airway inflammation and increased airway hyper-responsiveness leading to symptoms of wheeze, cough, chest tightness and dyspnea. It is characterized functionally by the presence of airflow obstruction which is variable over short periods of time or is reversible with treatment.

- ✓ **What are the clinical presentations of asthma?**

  Recurrent episodes of
  - Breathlessness
  - Cough
  - Wheeze
  - Chest tightness

- ✓ **Name some provoking or triggering factors for asthma.**
  - Asthma is associated with specific triggers e.g. tree and grass pollen, cat and dog dander, and non-specific triggers e.g. cold air exposure, perfumes, and bleaches, food allergens and also viral upper respiratory tract infection.

- ✓ **What are the cardinal pathophysiological features of asthma?**

  - Airflow limitation
  - Airway inflammation
  - Airway hyper-reactivity

- ✓ **Which immunoglobulin is responsible for asthma and what type of hypersensitivity reaction is it?**

  - IgE
  - Type I hypersensitivity reaction

- ✓ **What is the immunological mechanism of asthma?**

A subgroup of asthmatics are atopic, and therefore inhalation of an allergen producing specific IgE from B lymphocytes. This leads to the formation of IgE dependent "antigen complexes that bind to mast cells, basophils and macrophages and release of mediators such as histamine and eosinophil chemotactic factor. These factors cause bronchoconstriction and airway edema.

- ✓ **What investigations you want to do?**

Pulmonary function test:

-The diagnosis is certain if:
  - 20% diurnal PEF variation on >3 days per week, in a week of peak flow diary measures.
  - FEV_1>15% decrease after 6 minutes exercise.
  - FEVA>15%(and 200ml) increase after 2 week trial of oral steroid (30mg prednisolone od)

-Bronchodilator reversibly testing FEV_1>15% (or 200ml)increase after short-acting beta agonist therapy.

Blood:

- Total eosinophil count: Increased
- Serum total IgE is typically elevated in atopic asthma.
- Skin prick tests are simple and provide a rapid assessment of atopy.
- Metacholine/ histaminechallenge measures bronchial hyperresponsiveness.
- Sputum analysis: Sputum eosinophilia
- CXA- Normal
- ECG-Normal

✓ **What is the treatment of acute exaggeration?**

Treatment:

7. High flow O_2 inhalation
8. Nebulization stat and sos or4/6 hourly
   (Sulbutamol sol 1ml+1mlipratropium .sol +2ml normal sal)
9. Inj. Hydrocortosone
   2 amp IV stat and 1 amp IV 6 hourly
10. Salbutamol inhaler
11. Beclomethason inhaler
    2 puff tds
12. Tab. Montelucast 10mg
    0+0+1

If not controlled then what will you do?

- IV aminophylline drip

If not controlled then what will you do?

- IV magnesium sulphate

If is still not controlled refer patient to the ICU

✓ **What is the management of chronic asthma?**

Step wise management

Step 1: Occasional use of inhaled short-acting beta adrenoceptor agonist bronchodilators
Step 2: Introduction of regular preventer therapy (inhaled corticosteroid-ICS)
(Start Beclomethasone (BDP) at 400 micro gram in a twice daily dose)
Step 3: Add-on therapy

- Low to moderate dose of inhaled corticosteroid plus
  Long acting beta-2 agonist(LABAs),such as salmeterol
  Or
  Leukotriene receptor antagonist (Montelucast)

Step 4:"poor control on moderate dose inhaled steroid and add on therapy addition of fourth drug.
-Leukotrine receptor antagonist.
-Theophylline.

Step 5: Continuous or frequent use of oral steroid.

✓ **What are the indications of regular preventing therapy?**

-Has experienced an exacerbation of asthma in the last 2 years.
-Uses inhaled beta-2 agonists three times a week or more.
-Reports symptoms three times a weeks or more.
-Is awakened by asthma one night per week.

✓ **What do you mean by rescues therapy?**

To prevent frequent exacerbation and control symptoms short courses of 'rescue' oral corticosteroids are therefore often required and this is called rescue therapy.

✓ **Indications for rescue courses include:**

- Symptoms and PEF progressively worsening day by day.
- Fall of PEF below 60% of the patient's personal best recording.
- Onset or worsening of sleep disturbance by asthma.
- Persistence of morning symptoms until midday.
- Progressively diminishing response to an inhaled bronchodilator.
- Symptoms severe enough to require treatment with nebulized or injected bronchodilators.

✓ **What are the newer drug used in treatment of asthma? Or biological agent used in asthma?**

- It is omalizamub,
- It is monoclonal antibody against IgE.

✓ **What do you mean by brittle asthma?**

- Most of acute attacks are characterized by gradual deterioration over several hours to days.
- But some appear with little or no warning which is called brittle asthma.

✓ **When will do step down?**

- Once asthma control the dose of inhaled corticosteroid is titrate at lowest dose at which symptom is controlled.
- Then decrease inhaled corticosteroids around 25-50% in every three months.

✓ **Mention some non-pharmacological management of bronchial asthma.**

Non-pharmacological management

- Allergen avoidance may reduce severity of disease in sensitized individuals.
- House dust mite control measures.
- Pet removal may be useful.
- Smoking cessation may reduce asthma severity.
- Dietary manipulation avoids allergen food.
- Weight reduction in obese asthmatic leads to improve control.

Immunotherapy

- Desensitization using allergen-specific immunotherapy may be beneficial in small subgroup of patients.

✓ **In following topic you have memorized only features of acute severe asthma. (??)**

- Acute severe asthma (to remember PHIR)
  - P- PEFR 33-50%
  - R- RR > 25

- H- HR> 110
- I- Inability to complete sentence in one breath.

➢ Life threatening asthma
Any one of
- ❖ PEFR<33%
- ❖ SaO_2 <92% (NB needs ABG)
- ❖ PaO2 <8 kPa
- ❖ Normal CO_2
- ❖ Silent chest
- ❖ Cyanosis
- ❖ Poor respiratory effort
- ❖ Bradycardia/ arrhythmia/ Hypotension
- ❖ Exhaustion
- ❖ Confusion
- ❖ Coma
- ❖ Raised $PaCO_2$ and/or
- ❖ Needing mechanical ventilation with raised inflation pressure

✓ **What will you see before discharge?**

➢ Prior to discharge patient should be stable on discharge medication.
➢ Nebulised therapy should have been discontinued for at least 24 hours and
➢ The PEF should have reached 75% of predicted or personal best.

✓ **Indications for assisted ventilation in acute severe asthma.**

➢ To remember it ACR
- A- Arterial blood gas
  - Pa O_2 <8 kPa (60 mm Hg) and falling
  - Pa CO_2> 6 kPa (45 mm Hg) and rising
  - pH low and falling (H^+ high and rising)
- C-Coma
- R- Respiratory arrest
- Exhaustion, confusion, drowsiness

Step wise management of bronchial asthma ? SABA— short-acting β2-r agonist LABA— Long-acting β2-agonists ICS-- inhaled corticosteroids
Step 1: Occasional use of inhaled SABA
Step 2: step 1 plus regular use of ICS such as beclometasone
Step 3: Add-on therapy ❖ inhaled SABA as required plus **select any one :** ❖ high dose ICS 800 µg/day ❖ low dose ICS 4oo µg/day and LABAs (salmeterol and formoterol) ❖ low dose ICS & Oral leukotriene receptor antagonists (montelukast 10 mg daily) ❖ low dose ICS & Sustained released Oral theophyllines
Step 4: addition of a fourth drug ❖ inhaled SABA as required plus add one or more drug ❖ medium and high dose ICS 800 –2000 µg/day plus LABAs

- ❖ Oral leukotriene receptor antagonists (e.g. montelukast 10 mg daily)
- ❖ Sustained released Oral theophyllines

Step 5:

- ❖ inhaled SABA as required plus

add one or both

- ❖ prednisolone therapy (as a single daily dose in the morning)
- ❖ omalizumab, a monoclonal antibody directed against IgE

name some special variant asthma ?

cough variant asthma
Occupation asthma
NSAID induced asthma
exercise induced asthma

What is cough variant asthma?

when cough is the only symptoms without wheeze or breathlessness is called cough variant asthma

diagnostic criteria:

1. dry cough more than 6—8wk
2. absence of wheeze and dyspnea
3. presence of bronchial hyper-responsiveness

Occupation asthma?

when symptoms of asthma appear when patient remain in working place or exposure to occupational hazard and patient remained symptoms free during holi days

brittle asthma ?

this is an unusual variant of asthma characterized by sudden severe (life threatening asthma) attack occur within hours in apparently control patient without any warning symptoms

Complication of asthma?

acute severe asthma
respiratory failure
pneumothorax
coma
atelectasis

What does u mean by asthma control? LADEN ---Rescue

	Controlled	Partly controlled (any in any week) present	Uncontrolled
L- Lung function (PEF or FEV1)	normal	< 80%	3 features of partly controlled asthma present in any wk and 1 exaggeration
A- Limitations of activities	None	any	
D- Daytime symptom	None (<2 /wk)	(>2/wk)	
E- Exacerbation	None	1/ yr	
N- Nocturnal symptoms	None	any	
Rescue -- Need for rescue/'reliever' treatment	None (<2/wk)	(>2/wk)	

Demonstrate the procedure of inhaler?

what is indication of rescues therapy ?

M-- morning symptoms persist up to mid day
D-- diminishing response to inhaled bronchodilator
F-- fall of PEF < 60% of person best recording
C—sleep disturbance (C—means sleep)
P—progressive worsening of PEF and symptoms day by day
s—severe symptoms need nebulization / IV bronchodilator
dose and duration

- oral prednisolone 30-60 mg daily
- given for 3 weeks.

A patient is taking medicine but his asthma is not controlled? What may be the causes?

Difference between cardiac asthma and bronchial asthma clinically?

	bronchial asthma	**cardiac asthma**
age	young age	older
history	family HO ++ HO atopy ++	HO I
timing of dyspnea	occur at late part of night	early part of night
orthopnea	Absent	present (PND—in case OF LVF)
cough and sputum	scanty mucoid sputum and tenacious	profuse and frothy expectoration
wheeze and	more marked (++++)	less marked ()
pulse	pulsus paradoxus	pulsus alternans
BP	normal	hypertension
JVP	normal	may be raised
apex beat	normal	heaving – in hyptertension
auscultation	Rhonchi more marked (+++++) creps is absent	less marked (+++) creps present gallop rhythm

1. poor inhalation technique
2. inadequate drug and dose
3. triggering factor not controlled
4. non-compliance
5. wrong diagnosis
6. underlying copd / heart failure

when steroid / oral steroid is given ?

oral steroid is given
acute exaggeration
frequent exaggeration
stage III/IV disease
dose –0mg for 10 day

how will look for prognosis ?

composite score (BODE index) comprising the

1. body mass index (B),
2. the degree of airflow obstruction (O),
3. a measurement of dyspnoea (D) and
4. exercise capacity (E),

COPD

If patient is young and non-smoker (may be smoker)
-Provisional diagnosis: Acute severe asthma
-Differential diagnosis: Acute exaggeration of COPD

If patient is old and smoker
-Provisional diagnosis: Acute exaggeration of COPD
-Differential diagnosis: Acute severe asthma
……table…….

PARTICULARS OF PATIENT:
Name: Mustofa Kamal
Age: 50 years
Sex: Male
Marital status: Married
Occupation: Teacher
Religion: Muslim
Address: Fulbaria, Mymensingh.
Date of admission: 8.12.13 at 7pm
Date of examination: 10.12.13 at 7.15am

PRESENTING COMPLAINT:
Respiratory distress for 7 days
Cough with sputum for 7 days

HISTORY OF PRESENT ILLNESS:
According to the statement of the patient he was reasonably well 7 days back then he gradually developed intermittent breathlessness. Initially it was mild to moderate in nature which subsided after taking inhaler (only mention if patient use it) but for last one day it increased in severity and is not responding to inhaler. The patient's breathlessness is so severe that he could not speak full sentence. The breathlessness has no association with exertion, lying posture or no has history of sudden severe breathlessness that woke him from the sleep. But during this attack patient feels breathlessness walking few steps to toilet. The patient has similar type's episodic attack for last 10 years most of them were not severe as like this which subsided after taking inhaler or some oral mediction name of which he cannot mention. On query, patient gives history of some trigger factor for breathlessness such as tree and grass pollen, cat and dog dander, cold air exposure, perfumes, and bleaches and food allergens. The patient also noticed that his symptoms are more marked after any injection such as fever. The patient also complained of cough for same duration. The cough was productive in nature (dry if patient mention), contained yellow coloured, foul smelling sputum, less than 1/4 cup amount in 24, not blood stained, no relation with posture change, has no diurnal variation. On query the patient gives history that his cough persists most of the days of the month around the year with acute exaggeration which usually occurs after any infection. But the patient has no history of chest pain but had fever 15 days ago. Patient has no history of leg swelling (cor pulmonale). But for the last few months the frequency of this type of attack was increasing but most of the attacks were relieved at home after medication. This attack was so severe that he had to be admitted in this hospital under MU-I for emergency management.

HISTORY OF PAST ILLNESS:
No history of DM/HTN/TB
Patient has previous history of attack of breathlessness for several occasions for which he has to admit in this hospital.

PERSONAL HISTORY:
The patient is smoker. He smokes 20 sticks/day for last 20 years. For the last 3 years he is abstained from smoking.
Non-alcoholic

FAMILY HISTORY:
His wife and children are healthy and none of his family member is suffering from this type of diease and enjoying sound health.

SOCIO-ECONOMIC CONDITION:
He comes from low socio-economic condition and lived in crowding house.
Housing: Tin shade house.
3 rooms which accommodate 8 of his family member.
Sanitation: 1 sanitary latrine.
Water supply: Arsenic free tube-well water.

IMMUNIZATION HISTORY:
The patient is immunized according to EPI schedule.

DRUG HISTORY:
Patient is taking two types inhaler and some oral medications name which he cannot mention.

GENERAL EXAMINATION:

A. **Appearance**: Ill looking, dyspnic
B. **Body built**: Average
C. **Nutritional status**: Below average/ average
D. **Decubitus:** On choice but feels better in sitting
E. **Co-operation**: Well co-operative
F. **Anaemia:** Absent
G. **Eye:** Congested
H. **Jaundice:** Absent
I. **Cyanosis**: Absent/ may present
J. **Clubbing:** Absent
K. **Koilonychia**: Absent
L. **Leuconychia**: Absent
M. **Oedema:** Absent
N. **Dehydration**: Moderate
O. **Skin**: Pale , thin and wrinkled
P. **Hand**: Palm is warm
Q. **Body hair distribution**: Normal
R. **Bony tenderness**: Absent
S. **Lymph node**: No lymphadenopathy
T. **Thyroid gland**: Not palpable
U. **Neck vein**: Not engorged
V. **Pulse**: 110/minute, low volume, regular
W. **BP:** 120/85 mm of Hg
X. **Respiratory rate**: 30/minute

Y. **Temperature**: 98 degree F
Z. **Weight**: 45kg
AA. **Height**: 1.6 m
AB. **BMI**: 17.57 kg/m^2
AC. **Flapping tremor**: Absent

If patient has Corpulmonale or P-HTN

- Palpable P2
- Left parasternal heave
- Loud P2
- Epigastric pulsation
- Tender hepatomegaly

REPIRATORY SYSTEM:

- **INSPECTION:**

Size and shape of the chest: Barrel shaped chest
Evidence of respiratory distress: -Intercostal fullness or recession/ in drawing.
-Suprasternal, supraclavicular excavation
- Prominence of accessory respiratory muscle
-Lip pursing
No asymmetry, scar mark, visible impulse and engorged vein, gynaecomastia and spider nevi and pigmentation.

- **Palpation:**

Trachea: Central
Apex beat: Not palpable
Vocal fremitus: Normal.

- **PERCUSSION:** hyperesonance all over the lung

Upper border of liver dullness is 6th intercostals space.

- **AUSCULTATION:**

Breath sound is vesicular with prolong expiration.
Added sound: Expiratory Ronchi and Wheeze present all over the lung field .
Coarse inspiratory and expiratory crepitation all over the lung field.
Vocal resonance: Normal.

CARDIOVASCULAR SYSTEM:

Pulse: 72 beats/min.
Regular, normal in volume and character, all the peripheral pulses are normal.
BP: 120/75 mm of Hg
JVP: Not raised

PECORDIUM:
Inspection: Normal
Palpation: Apex beat not palpable.
Auscultation: S1 and S2 audible in all auscultatory area. No added sounds.

ALIMENTARY SYSTEM:
Inspection:
-Mouth and oral cavity: Normal
-Abdomen proper: Normal

Palpation:
-Liver, spleen, kidney: Not palpable
-No intraabdominal lymphadenopathy or palpable lump.

Percussion: Tympanic

Auscultation: Bowel sound present
Testes: Normal
D/R/E: Normal

SALIENT FEATURE:
Mustofa Kamal, 50 years old normotensive, non-diabetic, non-alcoholic, smoker for 20 years, Muslim college teacher, hailing from Fulbaria, Mymensingh got admitted into MU-1 MMCH with gradual development of dyspnea and cough for 7 days. Initially it was mild to moderate in nature then turned severe form which not responds to bronchodialator. He has no history of orthopnea, paroxysmal nocturnal dyspnea or exertional dyspnea. Patient is suffering from this type of episodic intermittent dyspnea for last 10 years. This dyspnea have no precipitating factors like tree and grass pollen, cat and dog dander, cold air exposure, perfumes and bleaches and food allergens. These worsen at night and winter season. Patient also complained productive coughs with foul smelling, yellow discolored sputum and has no relation with posture change and no diurnal variation. This cough persists most of the days of the month around the year with acute exaggeration which usually occurs after any infection. But the patient has no history of chest pain but had fever 15 days ago. The patient has no history of atopy and none of his family member is suffering from bronchial asthma. With this type of attack he had to admit several times in hospital. General examination reveals the patient is ill looking, dyspnic, eye congested, warm periphery, Pulse-110/min, bounding pulse, regular, Respiratory rate- 30/min, cyanosis, oedema, flapping tremor are absent, neck vein not engorged. Respiratory system examination that patient has barrel shaped chest, evidence of respiratory distress like intercostal recession/ in drawing and supraclavicular, suprasternal excavation, trachea is central and apex beat is not palpable. Percussion reveal upper border of liver dullness is in lowered down with hyper-resonant all over the chest. Auscultation reveals vesicular breath sound with prolong expiration with expiratory ronchi and wheeze all over the chest. The patient also has crepitation both in expiration and inspiration.

PROVISIONAL DIAGNOSIS:
Acute exaggeration of COPD

DIFFERENTIAL DIAGNOSIS:
Acute severe asthma

INVESTIGATION:

Pulmonary function test:
Obstructive spirometry and flow volume loop

- Reduced FEV1 to <80% (FEV1 is the measurement of choice to assess progression of COPD)
- FEV1/FVC <0.7
- Minimal bronchodilator reversibility (<15%, usually <10%)

CXR-PA

- Hyper inflated lung fields with attenuation of peripheral vasculature
- Low diaphragm
- Flattened diaphragm
- More horizontal ribs and tubular heart
- May see bullae

ECG

- P-pulmonale, RVH and poor progression R wave

Treatment:

- ✓ Low flow O_2 inhalation
- ✓ Nebulization stat and sos or 4/6 hourly

(Sulbutamol sol 1ml+1ml ipratropium .sol +2ml normal sal)

- ✓ Inj. Hydrocortisone
  2 amp IV stat and 1 amp IV 6 hourly
- ✓ Salbutamol inhaler
  2 puffs qds
- ✓ Ipratropium inhaler
  2 puffs tds
- ✓ Beclomethason inhaler
  2 puffs tds
- ✓ Tab. Theophylline

- ✓ **What is your provisional diagnosis?**
  My provisional diagnosis is acute exaggeration of COPD

- ✓ **What are the points in favor of your diagnosis?**

In history

- Gradual onset of dyspnea with cough
- Old age
- No family history, no atopy, no allergen provoking factor
- Productive cough persist more of the day of the month around the year with acute exaggeration which usually occur after any infection

In general examination:

- Dyspnic, lip pursing
- Eye congested
- Cyanosis (absent was this patient but may have in your patient)
- Tachycardia (pulse-110/min)
- Tachypnea (Respiratory rate- 30/min)
- Warm periphery and bounding pulse

Respiratory system exam reveals

- ➢ Barrel shaped chest
- ➢ Intercostal recession/ in drawing and Suprasternal, Supraclavicular excavation
- ➢ Apex beat is not palpable
- ➢ Upper border of liver dullness is lowered down
- ➢ Percussion note is hyper-resonance
- ➢ Vesicular breath sound with prolong expiration
- ➢ Crepitation both in expiration and inspiration

If the patient has Corpulmonale what will you got?

In general examination

-Eye: congested.

-Tongue: cyanosed.

-Edema:+

Sign of right heart failure

-Raised JVP

-Tender hepatomegaly

Sign of pulmonary hypertension

-Palpable P2 and loud P2

-Left parasternal heave

-Epigastric pulsation

- ✓ **Why not bronchiectasis?**
  - ➢ In bronchiectasis, productive cough with profuse amount of foul smell that increases with changing posture.
  - ➢ Clubbing with bilateral coarse crep present

- ✓ **What is your differential diagnosis?**
  - ➢ Acute severe asthma

Points in favor:

Gradual onset of dyspnea and cough with productive sputum.

- Vesicular breath sound with prolong expiration
- With expiratory ronchi and wheeze all over the chest

Points disfavor:

- Old age
- Smoker
- No family history, no atopy
- No allergy provoking factor
- Cough has no diurnal variation
- Barrel shaped chest
- Upper border of liver dullness is lowered down
- Percussion note is hyper-resonance
- Crepitation both in expiration and inspiration

……
……

- ✓ **Define COPD**
  - ➢ COPD is chronic obstructive pulmonary disease characterized by
    - o Fixed airflow obstruction
    - o Minimal or no reversibility with bronchodilators
    - o Minimal variability day-to-day symptoms
    - o Slowly progressive and irreversible deterioration in lung function, leading to progressively worsening symptoms.

- ✓ **What diseases are with in COPD?**
  - ➢ Chronic bronchitis
  - ➢ Emphysema
  - ➢ Chronic bronchiolitis/ chronic asthma

- ✓ **Define chronic bronchitis.**
  - ➢ Chronic bronchitis is the condition where the patient suffers from cough with most of the day at least 3 consecutive months for at least 2 consecutive years.

- ✓ **Define emphysema**.
  - ➢ Emphysema is abnormal permanent destructive enlargement of air space distal to terminal bronchiole due to loss of elastic recoil.

Types emphysema:

- o Panacinar – all the alveoli and alveolar ducts in acinus are involved
- o Centriacinar – involve proximal part of acinii.
- o Periacinar or paraseptal emphysema along the septa, blood vessels and pleura
- o Scar and irregular emphyesema

- ✓ **What will you find in patient with COPD?**

  - ➢ The patient is dyspnic
  - ➢ May have lip pursing- if emphysema
  - ➢ Eye- Congested
  - ➢ Tongue- cyanosis
  - ➢ Hand- palm is warm and bounding pulse- due to increased CO_2
  - ➢ Edema
  - ➢ Nicotine stain in nail

In respiratory system examination:

- Prominence accessory muscle of neck, engorged neck vein.
- Barrel shaped chest.
- Evidence of respiratory distress like intercostal recession /in drawing and suprasternal , supraclavicular excavation.
- Apex beat is not palpable in emphysema.
- In percussion upper border of liver dullness is lower down.
- **Auscultation** : vesicular breath sound with prolong expiration
  Add sound:
  -Expiratory ronchi and wheeze all over the chest .
  -Crepitation both in expiration and inspiration.

- ✓ **What will you get if a patient have pulmonary hypertension and cor pulmonale?**

In general examination

-Eye: congested.
-Tongue: cyanosed.
-Edema:+

Sign of right heart failure

-Raised JVP

-Tender hepatomegaly

Sign of pulmonary hypertension

-Palpable P2 and loud P2

-Left parasternal heave

-Epigastric pulsation

✓ **What investigation will you want to do in patient with COPD ?**

To Dx

- **Spirometry**
  - Reduced FEV1 to <80% (FEV1 is the measurement of choice to assess progression of COPD)
  - FEV1/FVC <0.7
  - Minimal bronchodilator reversibility (<15%, usually <10%)

- **To see etiology**
  - Young patient: serum alpha -anti –trypsin.
    - High resolution CT scan to see emphysema

- **To see complication**
  - -Arterial blood gas analysis
  - -Low PCo2 and low PO2

CXR-PA

- Hyper inflated lung fields with attenuation of peripheral vasculature
- Low diaphragm
- Flattened diaphragm
- More horizontal ribs and tubular heart
- May see bullae
- Loss of vascular marker and prominent pulmonary vessel at both hilum

ECG

- P-pulmonale, RVH and poor progression R wave

ECHOCARDIGRAPHY

✓ **What are the treatment of acute attack?**

1. Low flow O_2 inhalation
2. Nebulization stat and sos or 4/6 hourly
   (Sulbutamol sol 1ml+1ml ipratropium .sol +2ml normal sal)
3. Inj. Hydrocortisone
   2 amp IV stat and 1 amp IV 6 hourly
4. Antibiotics
5. Salbutamol inhaler
   2 puffs qds
6. Ipratropium inhaler
   2 puffs tds
7. Beclomethason inhaler
   2 puffs tds
8. Tab. Theophylline

✓ **What is the role of high-resolution CT in the diagnosis of emphysema?**

✓ **What is the definitive test to D_X emphysema?**

✓ **Why low flow of oxygen is given in COPD?**
- **To preserve the hypoxic drive for respiration**

We know that respiratory center is stimulated by CO_2 and hypoxia (O_2) and H_2. CO_2 plays main role in stimulating the respiratory center. But in COPD, there is hypoxia and hypercapnea. So, due to elevated CO_2 for long time, respiratory center becomes insensitive to CO_2. So, hypoxia is the only drive that maintain the respiration by stimulating the respiratory center. So, if hypoxia is totally corrected then respiratory center loses the drive and there will be respiratory arrest.

- It is the most sensitive technique for the diagnosis of emphysema
- It is useful in evaluating symptomatic patient with almost normal pulmonary function
- It helps emphysema, to differentiate the interstitial lung disease and pulmonary vascular disease.

✓ **Mention the treatment of stable COPD**

- **NONPHARMACOLOGICAL**
  - **Smoking cessation**
  - **Pulmonary rehabilitation** :
    - Multidisciplinary programmes that incorporate physical training,disease education and nutritional counseling to reduce symptoms ,improve health status and increase confidence
- **Diet:**
  - Weight loss is recommended if the patient is obese
- **PHARMACOLOGICAL**
  - **Bronchodilator:**
    - First short acting beta-adrenoceptor agonist(Sulbutamol inhaler)
    - If not controlled then add a short acting anti-cholinergic(Ipratropium inhaler)
    - If still symptomatic,regular long acting bronchodilator with anti-cholinergic
  - **Corticosteroid**
    - If still patient symptomatic add inhaled corticosteroids with bronchodilator
  - **Theophylline**
    - Theophyllines
  - **Oxygen therapy**
    - Long term domiciliary oxygen therapy(LTOT)
    - Low flow oxygen 2-4L/min for a minimum of 15 houres/day

- **Vaccination**
  - Influenza vaccine annually and pneumococcal vaccine

- **Antibiotics**

- **Surgery**
  - Bullectomy if large bulla

- **Lung transplantation in young patient**

Long term domiciliary oxygen therapy:
If prevent
- Progression of pulmonary hypertension
- Decrease secondary polycythemia
- Improve neuropsychological health

✓ **Mention the complications of COPD.**

- Pulmonary hypertension
- Corpulmonale
- Type ii respiratory failure
- Pneumothorax
- Polycythaemia
- Secondary infection
- Weight loss

✓ **How will you differentiate between pink puffer and blue bloater?**

Traits	Pink puffer	Blue bloater
Cause	Emphysema	Chronic bronchitis
Clinical feature	Dyspnea is more than cough and lip pursing	Cough with sputum is more than dyspnea
Cyanosis and edema	absent	Present
Arterial gas analysis	PO_2 and PCO_2 are normal	Low PO_2 increased PCO_2
Corpulmonale	absent	Present
Lean and thin	yes	no

✓ **Mention the MRC grading for dyspnea.**

Grade	Degree of breathlessness related to activities
0	No breathlessness except with strenuous exercise
1	Breathlessness with hurrying on the level or walking up a slight hill
2	walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stop for breath after walking about 100m or after a few minutes in level ground
4	Too breathlessness to leave the house, or breathless when dressing or undressing

- ✓ **How will you classify COPD according to severity?**

Mild	FEV1/FVC<0.7 FEV1 >80%
Moderate	FEV1/FVC <0.7 50% <FEV1<80% predicted
Severe	FEV1/FVC<0.7% 30% <FEV1 <50%
Very severe	FEV1/FVC<0.7 FEV1 <30%

- ✓ **A patient of known case of COPD comes to you sudden severe chest pain and breathlessness. What is your DX?**
  - ➢ (If you can't answer then sir may tell rest HO "on examination patient have hyper resonance and absent breath sound on right side)
  - ➢ Right sided pneumpthorax

- ✓ **Why?**
  - ➢ Due to rupture of bullae.

Which varieties it and why?
my diagnosis is COPD and it is emphysema • Breathlessness is more prominent then cough • lip pursing present (may absent) • Barrel shape chest • Apex beat is impalpable • upper border of liver dullness is lower down • auscultation ---vesicular breath sound with prolong expiration and rhonchi less marked **my diagnosis is COPD and it is chronic bronchitis** • cough is more prominent than breathlessness • on exam --- Rrhonchi present
Step wise management of bronchial asthma ? SABA— short-acting β2-r agonist LABA— Long-acting β2-agonists ICS-- inhaled corticosteroids
Step 1: Occasional use of inhaled SABA
Step 2: step 1 plus regular use of ICS such as beclometasone
Step 3: Add-on therapy ❖ inhaled SABA as required plus **select any one :** ❖ high dose ICS 800 µg/day ❖ low dose ICS 4oo µg/day and LABAs (salmeterol and formoterol) ❖ low dose ICS & Oral leukotriene receptor antagonists (montelukast 10 mg daily) ❖ low dose ICS & Sustained released Oral theophyllines
Step 4: addition of a fourth drug ❖ inhaled SABA as required plus add one or more drug

❖ medium and high dose ICS 800 –2000 µg/day plus LABAs ❖ Oral leukotriene receptor antagonists (e.g. montelukast 10 mg daily) ❖ Sustained released Oral theophyllines
Step 5: ❖ inhaled SABA as required plus **add one or both** ❖ prednisolone therapy (as a single daily dose in the morning) ❖ omalizumab, a monoclonal antibody directed against IgE
step wise management of COPD

name some special variant asthma ?

cough variant asthma
Occupation asthma
NSAID induced asthma
exercise induced asthma

What is cough variant asthma?

when cough is the only symptoms without wheeze or breathlessness is called cough variant asthma

diagnostic criteria:

1. dry cough more than 6—8wk
2. absence of wheeze and dyspnea
3. presence of bronchial hyper-responsiveness

Occupation asthma?

when symptoms of asthma appear when patient remain in working place or exposure to occupational hazard and patient remained symptoms free during holi days

brittle asthma ?

this is an unusual variant of asthma characterized by sudden severe (life threatening asthma) attack occur within hours in apparently control patient without any warning symptoms

Complication of asthma?

acute severe asthma
respiratory failure
pneumothorax
coma
atelectasis

What does u mean by asthma control? LADEN ---Rescue

	Controlled	**Partly controlled (any in any week) present**	**Uncontrolled**
L- Lung function (PEF or FEV1)	normal	< 80%	3 features of partly controlled asthma present in any wk and 1 exaggeration
A- Limitations of activities	None	any	
D- Daytime symptom	None (<2 /wk)	(>2/wk)	
E- Exacerbation	None	1/ yr	
N- Nocturnal symptoms	None	any	
Rescue -- Need for rescue/'reliever' treatment	None (<2/wk)	(>2/wk)	

Demonstrate the procedure of inhaler?

what is indication of rescues therapy ?
M-- morning symptoms persist up to mid day D-- diminishing response to inhaled bronchodilator F-- fall of PEF < 60% of person best recording C—sleep disturbance (C—means sleep) P—progressive worsening of PEF and symptoms day by day s—severe symptoms need nebulization / IV bronchodilator dose and duration ➢ oral prednisolone 30-60 mg daily ➢ given for 3 weeks.

Difference between cardiac asthma and bronchial asthma clinically?		
	bronchial asthma	**cardiac asthma**
age	young age	older
history	family HO ++ HO atopy ++	HO I
timing of dyspnea	occur at late part of night	early part of night
orthopnea	Absent	present (PND—in case OF LVF)
cough and sputum	scanty mucoid sputum and tenacious	profuse and frothy expectoration
wheeze and	more marked (++++)	less marked ()
pulse	pulsus paradoxus	pulsus alternans
BP	normal	hypertension
JVP	normal	may be raised
apex beat	normal	heaving – in hyptertension
auscultation	Rhonchi more marked (+++++) creps is absent	less marked (+++) creps present gallop rhythm
A patient is taking medicine but his asthma is not controlled? What may be the causes?		
7. poor inhalation technique 8. inadequate drug and dose 9. triggering factor not controlled 10. non-compliance 11. wrong diagnosis 12. underlying copd / heart failure		
when steroid / oral steroid is given ?		
oral steroid is given acute exaggeration frequent exaggeration stage III/IV disease dose –0mg for 10 day		
how will look for prognosis ?		
composite score (BODE index) comprising the 5. body mass index (B), 6. the degree of airflow obstruction (O), 7. a measurement of dyspnoea (D) and 8. exercise capacity (E),		

COPD	
MILD –I FEV1/ FVC < 0.7% FEV1 >80%	**Reduction of risk factors** ❖ vaccination influenza, pneumococcal ❖ smoking -- **short acting bronchodilator** when needed ❖ β_2-agonists (salbutamol and terbutaline,) ❖ anticholinergic, (ipratropium bromide)
MORDERATE –II FEV1/ FVC < 0.7% FEV1 : 80% ----50%	**stage I plus** ❖ add one or more long acting bronchodilator ○ LABA(salmeterol)/ ○ Theophylline ❖ add Pulmonary rehabilitation
SEVERE –III FEV1/ FVC < 0.7% FEV1 : 50%---30%	**stage I & II plus** add Inhaled corticosteroids (ICS)
VERY SEVERE—IV FEV1/ FVC < 0.7% FEV1 <30%	**above all plus** **add** **Long-term domiciliary oxygen therapy (LTOT)** **surgery** – ❖ lung volume reduction surgery (LVRS) and ❖ bullectomy **Lung transplantation**
Prescription of long-term oxygen therapy (LTOT) in COPD	❖ PaO2 < 7.3 kPa (55 mmHg) irrespective of PaCO2 and FEV1 < 1.5 L ❖ PaO2 7.3-8 kPa (55-60 mmHg) plus pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia ❖ patient stopped smoking. Use at least 15 hours/day at 2-4 L/min target to achieve a PaO2 > 8 kPa (60 mmHg) without rise PaCO2.

Which varieties it and why?
my diagnosis is COPD and it is emphysema • Breathlessness is more prominent then cough • lip pursing present (may absent) • Barrel shape chest • Apex beat is impalpable • upper border of liver dullness is lower down • auscultation ---vesicular breath sound with prolong expiration and rhonchi less marked **my diagnosis is COPD and it is chronic bronchitis** • cough is more prominent than breathlessness • on exam --- Rrhonchi present

Diabetes Mellitus

PARTICULARS OF PATIENT:

Name: Muhammad mohsin
Age: 55 years
Sex: Male
Marital status: married
Occupation: service holder
Religion: Islam
Address: jamalpur
Date of admission: 1.2.13 at 7pm
Date of examination: 4.2.13at 10 am

PRESENTING COMPLAINTS

Burning and tingling sensation of both limbs for 1 yr
Generalized swelling for 6 months

HISTORY OF PRESENT ILLNESS:

According to statement of the patient he was reasonable well 5 yr ago. Then he was diagnosed a case of diabetes mellitus on the basis of classical symptoms like increase thirst , polyuria , polydipsia , chronic fatigue and weight gain and laboratory finding (blood sugar) by a registrar physician or (in hospital if patient says) (or he was diagnosed a case of diabetes mellitus insidiously during blood checkup for another physical illness / chronic fatigue). After diagnosed he was advised for dietary control , exercise , oral hypoglycaemic drug and with those management his Diabetes was under controlled for several years .later he become reluctant regarding dietary control ,exercise and irregular on medication .1 yr ago patient developed burning and tingling sensation of both limbs. at first it began at feet then involved hands . Initially it was intermittent and now become persistent distributed in gloves and stocking patter (some patient may complain numbness instead of burning sensation). But he has no history limb weakness.
6 months ago he notice gradual swelling of whole body .initially it appear at face more marked around peri-orbital region after awaking and subsequently it became generalized . At the beginning of the swelling urine output was normal but for the last 15 days he noticed reduction in the urinary volume and frequency. Color of urine is normal .The patient denied of chest pain, cough , fever , shortness of breath in exertion , rest , lying position or sudden sever breathlessness that awake him from sleep (PND) **((((** if patient complain chest pain/ cough then describe it like this – patient also noticed central chest pain which chocking or band like compression in nature appear and aggravated by exercise and walking relived by resting , mild to moderate severity without any radiation or nausea vomiting or sweating …if patient complained cough describe it like this---patient also having episodic/ intermittent dry cough without diurnal variation or seasonal variation or triggering factors **)))))**. Patient having abdominal discomfort like abdominal fullness , early satiety (gastroparesis) ,anorexia ,nausea (CKD), loss libido and sexual dysfunction (erectile) , light-headness /dizziness specially during stand from sitting position (tell only if u got Postural hypotension in BP measuring).Patient bowel habit is normal (ask patient for nocturnal diarrhea and constipation) and had history of several episode of burning sensation of micturation which

improved after antibiotics treatment .the patient have no history dimness of vision, dysphagia , loss of consciousness(hypoglycaemia . DKA, CVD), leg pain during walking (intermittent claudication) , palpitation either in rest or exertion, Postprandial sweating , foot ulcer and no history of hospitalization for any serious illness. Patient had history several episode of hypoglycaemic attack which was reversed by patient himself by taking oral glucose (or have to be hospitalized) .patient was switch on insulin therapy twice or thrice daily (according to pt HO) 3 months ago as the oral drug was not sufficient enough to control his diabetes .

HISTORY OF PAST ILLNESS

The patient is hypertensive for 3 yr which control on drug .No previous history of jaundice and TB and contract with TB patient. The patient have on significant medical and surgical disorder.

DRUG HISTORY

The patient is taking insulin, metformin , antihypertensive (osartan potassium) , diuretics(if patient cant name the drug—then write that –pt is taking insulin or oral medication for DM and HTN but he cant mention the name). He had no HO adverse drug reaction. NO history taking NSAID(NS) and corticosteroid (as it causes DM)

PERSONAL HISTORY

Non smoker, Non alcoholic. No history of extra marital sexual exposure

FAMILY HISTORY

He has two daughters. All of his family members are healthy and enjoining sound health.

His father was diabetic and hypertensive died at the age of 65 yr due to IHD.

SOCIOECONOMIC CONDITION

He comes from middle class family
Housing: building .
3 rooms, which accommodate 4 0f
His family member.
Sanitation: sanitary latrine.
Water supply: Arsenic free tube well water

IMMUNIZATION HISTORY

The patient was not immunized according to EPI schedule

GENERAL EXAMINATION

- Appearance – puffy face (only if patient hav edema)
- Body built – average / obese in type II DM/
- Nutritional status –average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – absent (mild –if dm nephropathy)
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent

- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : + + , pitting
- Dehydration – Nill
- Skin –dry , thick , rough
- Body hair distribution – normal distribution of axillary & pubic hair
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – Blood pressure: lying, right arm 165/96 mmHg; sitting, right arm 140/90 mmHg
- Respiratory rate – 18/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :
- Bed side urine examination show s massive proteinuria (+ +)
  Sugar= Nill / ++ (according to finding)

Examination of foot

SYSTEMIC EXAMINATION

If depend on patient presentation
If no symptoms ---cardiovascular examination is first then neuro
If oedema –alimentary system , cardiovascular system then neuro
If only neuropathy ---nervous system first and cardio

Examination of Alimentary system
■**Mouth & pharynx**
■Tongue – Dry & coated

■**Abdomen Proper**
■**Inspection**
■Abdomen – distended and flanks are full
■Umbilicus – centrally placed and everted and slit is transvers
■Movement with respiration – present
■Scar mark – no scar mark but some striae
■Visible peristalsis – absent
■Visible vein – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia – normal

■**Palpation**
■Superficial & deep palpation: No muscle guard, no tenderness, no organomegaly
■Fluid thrill present (if with huge ascitis)or absent (if ascites is mild)
■**Percussion**
■**Shifting dullness is present**
■**Paddle sign in case of mild ascites**

■**Auscultation**
■Bowel sound –present t

■**NERVOUS SYSTEM**

Motor system examination

	Right LL	left LL	Right UL	left UL
Wasting and fasciculation	Absent	Absent	Absent	Absent
Bulk of the muscle	Normal	Normal	Normal	Normal
Tone of muscle	Increased	Increased	Increased	Increased
Power of the muscle	5/ 5	5/ 5	5/ 5	5/ 5
Reflex				
Knee jerk	+	+		
Ankle jerk	absent	absent		

SENSORY EXAMINATION
Inspection :
Upper limb
Wasting (theaner , hypotheaner muscle, dorsal guttering)
clawing hand , wrist drop
lower limb:
ulcer , hair loss , foot drop , pes cavus , loss of arching of foot

	Rt LL	Lt LL	Rt UL	Lt UL
pain and fine touch	Intact	Intact	Intact	Intact
crude touch	intact	intact	intact	intact
joint-sense position	diminish / loss up to ankle	diminish / loss up to ankle	present	present
vibration	do	do	do	do
Romberg sign	absent			

Not in this case
If pain and touch loss write in this way

pain and fine touch	loss up to ankle	loss up to ankle	up to wrist	up to wrist
pain and fine touch	loss along dermatome S1,L5,L4	loss along dermatome S1,L5,L4	loss along ulnar or median nerve distribution or dermatome C7,C6	loss along ulnar or median nerve distribution or dermatome C7,C6

If also sensation intact write in this way
All modalities of sensation are intact

Higher psychic function including speech: normal.
Fundoscopic exam: Normal / Not done
Cranial nerves: intact

CARDIVASCULAR SYSTEM
•Pulse : 72 b/min
Regular, normal in volume & character, all the peripheral pulses are normal.
•BP : 120/75 mm of Hg
•**JVP : Not raised**
•**Precordium** :
Inspection: Normal
Palpation: Apex beat in lt 5th intercostals space 9 cm from midline
No para-sternal heave and no palpable
AuscultationS1&S2 audible in all auscultatory area
No added sound and no murmur

SALIENT FEATURE:

MD Mohsin 55 years old man diabetic for 5 yr , hypertensive for 3 yr , non smoker , non alcoholic service holder hailing from jamalpur got himself admitted into MU-I of MMCH with complains of Burning and tingling sensation of both limbs for 1 yr and Generalized swelling for 6 months. 5 yr ago he was diagnosed a case type II DM by a local doctor on the basis of classical symptoms and lab investigation and was managed well by lifestyle modification and drug until he became reluctant and irregular on medication . 1 yr ago he developed burning and tingling sensation in gloves and stocking pattern . 6 months ago he also developed progressive generalized swelling of whole body start initially around face. recently his urinary output reduced with decrease frequency but color is normal (frothy). The patient have no motor complain. The patient also noticed abdominal complain like epigastric fullness , early satiety , anorexia , nausea , loss of libido , sexual dysfunction , dizziness . His bowel habit is normal. He denied any chest pain , dyspnea in exertion or rest , orthropnea , PND, visual disturbance , fever , cough , abdominal pain, intermittent claudication . He also denied symptoms of autonomic dysfunction like palpitation, dysphagia , urinary incontinence , nocturnal diarrhea , gustatory sweaty . he had recurrent UTI and hypoglycaemic attacked which was managed well . but no history unconsciousness , foot ulcer and other serious illness for which he needed hospitalization .recently he switch on insulin . His hypertension is managed well with medication .His father was also diabetic and died of MI / stroke .
General examination reveal patient is obese (sir may aked BMI) having puffy face ,mildly anaemic, bilateral pitting edema , pulse normal in rate and rhythm , Having postural hypotension , canula in situ (if present), temperature normal . The patient have no jaundice , dehydration , lymphadenopathy , neck vein not engorged , No thyromegaly . systemic examination reveal patient having ascites evidence by shifting dullness and fluid thrill but no organomegaly. Neurological examination reveal patient loss of vibration and joint sense position up to ankle in lower limb and upto wrist in upper limb . romberg sign absent . pain and fine touch are intact . motor function are normal except loss or diminish of ankle jerks . there is no trophic ulcer . all the peripheral pulse are palpable .rest other system reveal no abnormality

Provision diagnosis:

Type II diabetes mellitus with complication nephropathy and neuropathy (if chest pain --IHD) and HTN

Differential diagnosis:

Type II diabetes mellitus with heart failure with neuropathy and HTN

Investigation:

- Urine RME—proteinuria , glucose ,
- 24 hr total urinary protein
- FBS and 2hr ABF
- Renal function test –urea and creatinine , electrolyte
- Fasting lipid profile –dyslipidaemia
- ECG---feature of IHD
- CXR—PA
- HbA1c

TREATMENT:
Treatment of DM is combination of **3D**

1. **Discipline/ lifestyle modification**
2. **Diet**
3. **Drug**

In discipline
Educate the pt about the disease –and life style modification
Exercise
Diet

- Depend on patient body weight or BMI
- Occupation / life style Sedentary worker or heavy worker
- If patient is obese than ---low calorie diet
- If patient is non obese or underweight ----weight maintaining diet

Composition of diet
Carbohydrate 45-60% (avoid sugar , sweets)
Fat (total) < 35%, Polyunsaturated
Protein 10-15% (do not exceed 1 g/kg body weight)
vegetables and fruits
we give 5 meal –3 large meal morning, noon and night and two snacks at 8 am—11am –2pm—5 pm –8pm

In this patient:
Drugs

- Insulin ---as patient have –complication

For nephropathy and hypertension

- ACE inhibitor or ARB

For neuropathy
Pain and paraesthesiae

- Anticonvulsants (gabapentin, pregabalin, carbamazepine, phenytoin)
- Tricyclic antidepressants (amitriptyline, imipramine)

Postural hypotension :
non pharmacological :

- **correct hypovolemia**
- **stop the drug**
- **Support stockings—compression bandage**

pharmacological :

- **Non-steroidal anti-inflammatory drugs (NSAIDs)**
- **Fludrocortisone**
- **α-adrenoceptor agonist (midodrine)**

Drugs

- In type one -----insulin

- In type II OHA (**oral hypoglycemic agent**:)

If patient is obese

- First add --- Metformin (Monotherapy)
- If not control Add ----Sulphonylurea ((combination therapy))
- If not control add insulin or give only insulin

If patient is non-obese

- First add --- Sulphonylurea(Monotherapy)
- If not control Add ---- Metformin ((combination therapy))
- If not control add insulin or give only insulin

Ques and ans

why it is type II DM
Because Age –(because type I occur in young pt < 40 yr) Obese –(type one pt is cachexic) insidious onset ,(type—I is abrupt onset) Absence of other auto immune disease

why nephropathy
patient have –oedema anemia oligouria anorexia and nausea --CKD bed side urine positive

what is the causes of edema in DM patient ?
➢ Diabetic nephropathy ➢ NS ➢ CCF ➢ Autonomic neuropathy –vaso-motor tune
why not heart failure
point favour depended edema **point disfavor** Other sign and symptoms CCF is absent such No history exertional dyspnea . orthropnea , chronic cough with productive sputum On Exam – most important JVP not raised No tender hepatomegaly lung crep are absent

Write down the difference between types I and Type II diabetes mellitus?

	Type 1	Type 2
age of onset	young less than 4o yrs	more than 50 yrs
duration of onset	abrupt onset	insidious onset
Body weight	low / underweight	Obese
Diabetic complications at diagnosis	absent	usually present in 25 %
ketonuria	present	absent
Autoantibodies & autoimmune disease	usually present	absent
family history	absent	present

What other investigation will u do in this pt with diabetes milieus
• **fasting and two hr after break fast** • Urine RME---proteinuria ----nephropathy • s.creatinine and urea , serum electrolyte • fasting lipid profile • ECG • CXR • HbA1c • USG
What examination will you do if u want to know the glycaemic status of previous months?
• Glycated haemoglobin (HbA1c) • Normal value 7 • 1% HbA1c = 2 mmol / L

how will treat the patient

treat of DM and also treatment of complication

Treatment of DM is combination of **3D**

1. **Discipline/ lifestyle modification**
2. **Diet**
3. **Drug**

In discipline

Educate the pt about the disease –and life style modification

- it is the disorder of glucose metabolism
- it can't be cured and only pt have maintain normal blood sugar by dietary and drug control
- he has to maintain a discipline life with daily regular exercise with calculated food and timely food intake
- avoid sugar and sugar containing food
- counseling the pt about or educate about complication of the disease
- teach the pt about foot care
- Teach he sign symptom of hypoglycemia and what will do when these appear

Diet

- Depend on patient body weight or BMI
- Occupation / life style Sedentary worker or heavy worker
- If patient is obese than ---low calorie diet
- If patient is non obese or underweight ----weight maintaining diet

composition of diet

Carbohydrate 45-60% (avoid sugar , sweets)
Fat (total) < 35%, Polyunsaturated
Protein 10-15% (do not exceed 1 g/kg body weight)
vegetables and fruits
we give 5 meal –3 large meal morning, noon and night and two snacks
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Drugs

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- First add --- Sulphonylurea(Monotherapy)
- If not control Add ---- Metformin ((combination therapy))
- If not control add insulin or give only insulin

treatment of complication

What is the target BP in DM ? glucose level ?

130/ 80 mm of hg if proteinuria is more than > 1 gm per 24 125/75
target blood sugar :

- fasting level= 6 mg dl
- HbA1C =7
- 2hr ABF = 8

Which ant-HTN u will use in a pt with DM ?

ACEinhibitor or angiotensin receptor blocker

indication of insulin in DM patient ?

Indication of insulin in DM patient:

Insulin therapy is indicated in those who meet the following criteria:

1. Type 1 DM patients
2. Type 2 DM patients
    1. Who remain persistently symptomatic hyperglycaemic on maximum dose of oral agents and diet (primary/secondary failure).
    2. Acute stress, such as –
        *severe Infection
        * Myocardial infarction
        * Stroke
        DKA and HONK
    3. Diabetes with complication
        * Eye disease: Prolifertive retinopathy
        * Kidney disease: nephropathy Serum Creatinine >2.5 mg/dl.
        * Acute metabolic neuropathy
    4. Prior to surgery;
    5. pregnancy

Here sir may ask u what exercise and how long u do it?

Methods of exercise:

Stretching Exercise –

- Free hand exercise – **Duration** 10 minutes

Aerobic Exercise –

- i.e., brisk walking, swimming, cycling, jogging. Treadmill, static cycling

Duration at least 30 min at **least 3 times a week.**

Tell the Contraindication of exercise?

1. Coronary heart disease,
2. Proliferative retinopathy,
3. Severe neuropathy,
4. Osteoarthritis,
5. Neprhopathy,
6. Ketonuria

Name of oral hypoglycemic agent

1. **Insulin Secretagogues:**
    - **A. SULPHONYLUREA**
        - Glibenclamide ,

- Glimeperide ,
- Gliclazide

B GLINIDES

- Rapaglinide
- Nateglinide.

2. INSULIN SENSITIZERS

A. BIGUANIDES

- Metformin 500 mg /850 mg

B. THIAZOLIDINEDIONES

- Rosiglitazone 4 mg / 8 mg
- Pioglitazone 15 mg / 30 mg

3. OTHERS :

The α-glucosidase inhibitors

- Acarbose 50 mg

Name drug there mechanism of action ?	
SULPHONYLUREA	stimulate the release of insulin from the pancreatic β cell
INSULIN SENSITIZERS	Increase insulin sensitivity and peripheral glucose uptake in muscle and impairs glucose absorption by the gut and inhibits hepatic gluconeogenesis
The α-glucosidase inhibitors	delay carbohydrate absorption in the gut by selectively inhibiting disaccharidases

Now sir will ask about metformin

What type of drug it is	Sensitizer
Why it is use in obese	It reduce weight also
What invest. U do before prescribe it and why	S.creatinine and SGPT Because if S. Creatinine > 1.5 mg/dl in male and 1.4 mg/dl in female metformin cant given
Mention one complication of metformin	Lactic acidosis
Name another disease where it use ?	Polycystic ovarian syndrome

Now sir some scenario and ask u to give treatment

A 40 years obese leady newly diagnosed DM .BMI 30 or wt -60 kg FBS—13 mmol/l and 2HABF 18 mmol/ l	• Diet ---Diabetic diet (low calorie diet) • Exercise : 30 min each day • Drug ----OHA----Metformin 500 mg
A 40 years non-obese leady newly diagnosed DM .BMI 22 or wt -50 kg FBS—13 mmol/l and 2HABF 18 mmol/ l	• Diet ---Diabetic diet (weight maintaining diet) • Exercise : 30 min each day • Drug ----OHA----Sulphonylurea

A 20 years obese leady newly diagnosed DM .BMI 22 or wt -30 kg FBS—13 mmol/l and 2HABF 18 mmol/ l	• Diet ---Diabetic diet (weight maintaining diet) • Exercise : 30 min each day • Drug ----insulin
Diabetic patient with MI , stroke , severe infection –TB, CLD , jaundice , pregnancy	• insulin

Classify insulin:	
Type	**Generic name**
Ultra short acting	insulin analogues- lispro, aspart,
Short acting / rapid	Soluble regular
Intermediate acting	Basal Lente Isophane (NPH-Neutral protamin hagedorn)
Long acting	Insulin analogues- Glargine, Detemir

Dose of insulin is:

(**0.2 – 0.4 unit/kg body wt/day**), usually we count 0.3unit / kg body wt /day

Tell the side affect of insulin?
➢ Hypoglycaemia ➢ Weight gain ➢ Peripheral oedema (insulin treatment causes salt and water retention in the short term) ➢ Local allergy (rare) ➢ Lipodystrophy at injection site
How insulin is given and what are the sites?
Insulin is given subcutaneously **Site of insulin:** ➢ Outer aspect of thighs, upper arms and ➢ Abdomen below the umbilicus ➢ Buttocks

new drug use in DM
Incretin-based therapies ➢ GLP-1 (glucagon like peptide) receptor antagonists – exenatide ➢ DPP-4 inhibitors (dipeptidyl peptidase 4)--sitagliptin, vildagliptin and saxagliptin

How will u Diagnosis of diabetes
Patient complains of symptoms suggesting diabetes Test urine for glucose and ketones Measure random or fasting venous blood glucose. Diagnosis confirmed by2: ➢ Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) ➢ Random plasma glucose ≥ 11.1 mmol/L (200 mg/dL)
Indications for oral glucose tolerance tes?
➢ Fasting plasma glucose 6.1-7.0 mmol/L (110-126 mg/dL) ➢ Random plasma glucose 7.8-11.0 mmol/L (140-198 mg/dL)

How to perform an oral glucose tolerance test (OGTT)?		
Preparation before the test ➢ Unrestricted carbohydrate diet for 3 days ➢ Fasted overnight for at least 8 hrs ➢ Rest for 30 mins ➢ Remain seated for the duration of the test, with no smoking		
Plasma glucose is measured before and 2 hrs after a 75 g oral glucose drink		
Interpretation	**Fasting**	**2 hrs after glucose**
Fasting hyperglycaemia	6.1-6.9 mmol/L	< 7.8 mmol/L (< 140 mg/dL)
Diabetes	≥ 7.0 mmol/L (≥ 126 mg/dL)	≥ 11.1 mmol/L (≥ 200 mg/dL)
Impaired glucose tolerance	< 7.0 mmol/L (< 126 mg/dL)	7.8-11.0 mmol/L (140-199 mg/dL)

Mention the complication of DM ?
acute complication • Hypolgycaemia • DKA • Hyper osmolar nonketotic coma, (HONK

chronic complication of DM are two types

***Microvascular* (to rmember RNN)**

- **Retinopathy, (R)**
- **Nephropathy (N)**
- ***Neuropathic* (N)**
  - Peripheral neuropathy or somatic
  - Autonomic neuropathy
- **Foot disease**
  - Ulceration
  - Arthropathy

Macrovascular (to rmember CCP)

- **Coronary circulation (C)**
  - Myocardial ischaemia/infarction
- **Cerebral circulation (C)**
  - Transient ischaemic attack
  - Stroke
- **Peripheral vascular disease (P)**
  - Claudication
  - Ischaemia

A diabetic patient comes to you with complaint unconsciousness or semi consciousness? What will b ur diagnosis?

Hypolgycaemia
DKA
Hyper osmolar nonketotic coma, (HONK)

autonomic neuropathy ?		
Somatic neuropathy		
polyneuropathy	Sensory	loss joint sense position and vibration loss of Touch and pain sensation Buring and Parasthesia , Numbness
	Motor	Diabetic amyotrophy—pain and wasting jerk absent (knee)
mono neuropathy		mononeuritis multiplex
Autonomic neuropathy		
Cardiac		Resting tachycardia Fixed heart rate Postural hypotension
Gastro-intestinal		Gastro-paresis Dyphagia Diarrhea (nocturnal) Constipation

Gentio-urinary	Urinary incontinence Erectile disfucntion Retrograde ejaculation
SUDOMOTOR	Gustatory sweating Anhidrosis;
Vasomotor	Cold Feet Dependent oedema
Cranial nerve	Pupillary reflex Opthalmoplagia

How will u differentiated from hypoglycemic coma from hyperglycemic coma?	
hypoglycemic coma	**hyperglycemic coma**
Sweating with cold periphery	Not so
Pulse –bounding pulse	Low volume pulse or tachycardia
BP –normal	Bp- hypotension
No sign of dehydration	Sign of dehydration

If u fail to differentiate the between hypoglycemic coma from hyperglycemic coma then what will do ? Why ?
I will treat the patient as hypoglycaemia coma Because hypoglycaemic coma is more dangerous than hyperglycemic coma . it causes irreversible brain damage

What are the feature of hypoglycemia ?	
Autonomic symptoms Sweating Pounding heart Hunger Tachycardia Tremor	**CNS: / Neuroglycopenic** Confusion Convulsion Drowsy Inability to concentrate Slurring speech
How will manage hypoglycaemic coma ?	
will manage the pt in following ways : **If look at the patient is unconsciousness or not** Or he is able to take food orally or not If the patient is able to take food orally then give **Oral carbohydrate as form of** Orange juice / glucose Sugar (4 –6 tsf sugar in a glass of water) Whatever carbohydrate u gets near ur hand **IV fluid:** Inj. 25 % glucose 100 ml (in text book ---50 ml of 50 % glucose if any one ask to know the text book) ---------- IV @ 20 D / min (if u do not get 25 % then 1st give 5%DA and ask the pt bring immediately)	

Followed by

Inj. 10 % DA 1000 ml

IV @ 20 D/ min

- Stop insulin or other oral Hypoglycemic drug immediately
- Do FBS and 2 hr ABF next day @
- 20to 30 % reduction of dose of insulin and oral hypoglycemia drug .

What will u do if a patient with hypoglycemia come to u with unconsciousness but 25% or 5% DA is not available?

Give him NG tube and give glucose / orange juice/ sugar via it

What may the causes of hypoglycaemia in this pt ? or what history u will ask the pt ?

Pt usually present With following HO

- HO insulin or oral hypoglycaemic drug intake followed by
  - Missed, delayed or inadequate meal
  - Unexpected or unusual exercise
  - Vomiting

define **HYPOGLYCAEMIA and SPONTANEOUS HYPOGLYCAEMIA ?**

HYPOGLYCAEMIA When hypoglycaemia (blood glucose < 3.5 mmol/l (63 mg/dl)) occurs in a person with diabetes it is a result of treatment and not a manifestation of the disease itself.

SPONTANEOUS HYPOGLYCAEMIA When hypoglycaemia develops in non-diabetic people,(if < 3 mmol/L) it is called 'spontaneous' hypoglycemia

How will manage a patient with DM nephropathy ?

vigorous efforts should be made to reduce the risk of progression and of cardiovascular disease by:

- Improved control of blood glucose
- Aggressive reduction of blood pressure
- Aggressive cardiovascular risk factor reduction by
  - statins and aspirin

How will u control BP?

- In type 1 diabetes ACE inhibitors
- with type 2 diabetes angiotensin II receptor blockers

where both contraindicated:

- Non-dihydropyridine calcium antagonists (diltiazem, verapamil)

When and how % of type I DM develop nephropathy?

30% of patients with type 1 diabetes

20 years after diagnosis

What significance of microalbuminuria (MA) ?

- Identifies incipient nephropathy in type 1 and type 2 diabetes;
- independent predictor of macrovascular disease in type 2 diabetes

What are the risk factor for microalbuminuria ?
Risk factors include • increased blood pressure, • poor glycaemic control, • smoking

When to screen ?
Patients with type 1 diabetes annually from 5 yrs after diagnosis Patients with type 2 diabetes annually from time of diagnosis
what does MA indicate in type –II ?

what is microalbuminuria ?
Microalbuminuria describes the urinary excretion of small amounts of albumin
what is significance of microalbuminuria ?
indicator of the development of diabetic nephropathy in case of typeII it is marker of an increased risk of macrovascular disease
what is value of
➢ confirm MA overnight or 24 hrs to quantify the albumin excretion 30-300 mg/24 hrs)
when to screen for DM ?
type 1 diabetes annually from 5 yrs after diagnosis type 2 diabetes annually from time of diagnosis
when can Standard dipstick testing for albumin detects urinary albumin?
at concentrations > 300 mg/L,

What do you mean by metabolic syndrome?
Following are collective called metabolic syndrome: • Type-2 diabetes mellitus • HTN • Central obesity • Hypertriglyceridaemia

How will u differentiated between HTN & Diabetic retinopathy?	
HTN retinopathy	**Diabetic retinopathy**
• Artery –venous nipping • Slivery wiring • Flamed shape is hemorrhage • Cotton wool exxudate	• Dot and blot hemorrhage • Micro-aneurysm • Hard exudates

Treatment of retinopathy

prevention and management

• tight glycemic control with insulin
• laser photocoagulation (eliminates neovascularization)
• vitrectomy
• frequent follow-up visits with an ophthalmologist yearly
  - immediatereferral after diagnosis of type 2 DM
  - in type 1, only after 5 years of DM

Management options for peripheral neuropathy

Pain and paraesthesiae

strict glycaemic control-- Intensive insulin therapy

- Anticonvulsants (gabapentin, pregabalin, carbamazepine, phenytoin)
- Tricyclic antidepressants (amitriptyline, imipramine)

what is postural hypotension ?

Orthostatic hypotension/ postural hypotension
it is defined as a fall in systolic blood pressure of at least 20mm Hg and diastolic blood pressure of at least 10 mm Hg when a person assumes a standing position from sitting position

causes of postural hypotension ?

- hypovolemia
- Drug
  - Diuretics, vasodilators, antidepressants
- Addison's disease
- DM
- Parkinson's disease

How will measure?

- first measure the BP in sitting or lying position and then deflate the cuff
- now ask the patient to stand up
- measure the BP again after two minute and before three minutes

what is the treatment of postural hypotension

non pharmacological :

- correct hypovolemia
- stop the drug
- Support stockings—compression bandage

pharmacological :

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Fludrocortisone
- α-adrenoceptor agonist (midodrine)

What is the cause of impotency or erectile dysfunction in DM pt ?

affects 30% of diabetic males

Multifactorial

- neuropathy and vascular causes are common,
- psychological factors, including depression, anxiety and reduced libido
- Alcohol and antihypertensive --thiazide diuretics and β-blockers
- Endocrine-- testosterone deficiency or hyperprolactinaemia

Rx

- Psychological counselling; psychosexual therapy
- Phosphodiesterase type 5 inhibitors (**sildenafil**,)
- **Prostaglandin E1** (alprostadil)-

When and what do u mean by gestation dm?

Gestational diabetes is defined as diabetes with first onset during pregnancy

- Gestation diabetes fasting plasma glucose > 5.5 mmol/l or
- 2 hours after a glucose > 9.0 mmol/l

Treatment

- Pregnancy should be planned
- Folic acid supplementation begins before conception
- Maintain strict glycaemic control
  - first with diet
  - if nt control add short acting soluble insulin

Can you give oral hypoglycacemic drug in pregnancy ?

no

they are teratrogenic

Name two drug given in pregnancy ?

metformin

glibenclamide

write down the management of DM foot ?

Prevention

Prevention is the most effective way of dealing with the problem of tissue necrosis in the diabetic foot. Advice to all diabetic patients includes: **IM WW ABCD-1231**

- I— **Ins**pect feet every day
- M— **Mois**turise skin if dry
- W— **Wa**sh feet every day
- W— **Wea**r suitable good-fitting shoes
- A— A**void** over-the-counter corn/callus remedies
- B— Avoid walking **barefoot**
- B— Do not burst b**listers**
- C— **Cut or file** toenails regularly
- C— **Change socks** or stockings every day
- C— **Check footwear** for foreign bodies
- D— Cover minor cuts with sterile **dressing**s

Advice to high-risk patients is as above plus:

- Do not attempt corn removal
- Avoid high and low temperatures

treatment –to remember : REGIA

R— Remove callus
E— Control oedema
G— Ensure good glycaemic control
I— Treat infection
A— Avoid weight-bearing
A— Undertake angiogram to assess feasibility of vascular reconstruction where indicated

what is the treatment of gastroparesis ?

Gastroparesis DEP

- D-- Dopamine antagonists (metoclopramide, domperidone)

if want to more than only say the following

- E-- Erythromycin
- P-- Gastric pacemaker; percutaneous enteral (jejunal) feeding

What is DM?

Diabetes mellitus is a clinical syndrome characterised by hyperglycaemia caused by absolute or relative deficiency of insulin

Etiological classification of diabetes mellitus
Type 1 diabetes

- Immune-mediated
- Idiopathic

Type 2 diabetes
gestational Dm
Other specific types

WHAT ARE THE OTHER AND SPECIFIC CAUSES OF dm?

Pancreatic disease	➢ Pancreatitis, ➢ Pancreatectomy, ➢ Neoplastic disease, ➢ Cystic fibrosis, ➢ Haemochromatosis, ➢ Fibrocalculous pancreatopathy
ENDOCRINE CAUSES	➢ growth hormone-acromegaly; ➢ glucocorticoids-Cushing's syndrome; ➢ glucagon-glucagonoma; ➢ catecholamines-phaeochromocytoma; ➢ thyroid hormones-thyrotoxicosis
Drug-induced	➢ corticosteroids, ➢ thiazide diuretics, ➢ phenytoin

genetic syndromes	➢ Down's syndrome; ➢ Klinefelter's syndrome; ➢ Turner's syndrome
DIDMOAD (Wolfram's syndrome)-	Associated with (e.g.; diabetes insipidus, diabetes mellitus, optic atrophy, nerve deafness; Friedreich's ataxia; myotonic dystrophy)

AGN

PARTICULARS OF PATIENT:

Name: Rabiual islam
Age: 20 years
Sex: Male
Marital status: unmarried
Occupation: student
Religion: Islam
Address: Mouchak, mymensingh
Date of admission: 1.12.09 at 7pm
Date of examination: 3.12.09 at 10 am

Presenting complaints
Scanty micturation for 5 days
Generalized body swelling for 5 days

History of present illness
According to the statement of the patient, he was reasonable well 5 days ago .Then he suddenly developed scanty micturation for 5 days. The urine is high color and he have to evacuate bladder 2-3times pre 24 hours and amount is one and half glass per day .He also complained of generalized body swelling for the same duration which he first noticed at face around the eye lid and later on spread all over the body .The patient also gave history itching followed by skin infection 3 wks before (sore throat) of presenting complaints. The patient has no history breathlessness and palpitation either in rest or exertion. The patient has no recent or past history of yellow coloration of sclera or any other parts of the body. The patient also complaint of mild head ache and blurring of vision for the last 5days but no vomiting. Patient has no HO convulsion, unconsciousness and sudden severe breathless ness that awake him from sleep.

History of past illness
The patient has no HTN and diabetic. Patient had no previous history of generlalized body swelling
Drug history
The patient has no history taking pain killer and anti hypertensive drug. After admission in hospital he getting some inject able and oral drug but name of which he could not mention.

PERSONAL HISTORY

Non smoker, Non alcoholic.

FAMILY HISTORY

- None his family member is suffering from this type of disease.

They are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition and lived in Crowding house
Housing: Tin shade house.

2 rooms, which accommodate 8 0f
his family member.

Sanitation:1 sanitary latrine.

Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized according to EPI schedule

GENERAL EXAMINATION

- Appearance – puffy face
- Body built – average
- Nutritional status –average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia –mild
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : + +
- Dehydration – Nill
- Skin –scratch mark present over hand and abdomen and old scar of healed skin infection
- Body hair distribution – normal distribution of axillary & pubic hair
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 150/95 mm of Hg
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :
- Bed side urine examination show s massive proteinuria (++)

  Sugar= Nill

Systemic examination

Examination of Alimentary system

■**Mouth & pharynx**

■Tongue – Dry & coated

■**Abdomen Proper**

■**Inspection**

■Abdomen – mildly distended and flanks are full

■Umbilicus – centrally placed and everted and slit is transvers

■Movement with respiration – present

■Scar mark – no scar mark but some striae

■Visible peristalsis – absent
■Visible vein – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia – normal

■**Palpation**
■Superficial & deep palpation: No muscle guard, no tenderness, no organomegaly
■Fluid thrill absent (if with huge ascitis may present)
■**Percussion**
■**Shifting dullness is present**
■**Paddle sign in case of mild ascites**
■**Auscultation**
■Bowel sound –present t
■**Examination of respiratory system**

CARDIVASCULAR SYSTEM
•Pulse : 72 b/min
Regular, normal in volume & character, all the peripheral pulses are normal.
•BP : 150/95 mm of Hg
•**JVP : Not raised**
•**Precordium** :
Inspection: Normal
Palpation: Apex beat in lt 5th intercostals space 9 cm from midline
No para-sternal heave and no palpable
AuscultationS1&S2 audible in all auscultatory area
No added sound and no murmur

RESPIRATORY SYSTEM
•**Inspection** : Size and shape of the chest : Normal
•Movement is symmetrical
•No evidence of respiratory distress
•**Palpation** :
Trachea: Trachea central
Apex beat: in left 5th intercostals space 9 cm from midline normal in character
Vocal fremitus : normal
Percussion: resonance

Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM
•**Higher psychic function including speech**: normal.
Fundoscopic exam: Normal
Cranial nerves: intact
•**Motor system examination**
Motor functions are normal in all four limbs

Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

Salient feature

Rabiual Islam 20 years nonsmoker hypertensive , nondiabetic, muslim student hailing from Mouchak, mymensingh got admitted into MU-1 MMCH with suddenly developed scanty micturation and generalized body swelling for 5 days. The urine is high color and amount is one and half glass per day. Edema was first noticed at face around the eye lid and later on spread all over the body .The patient also gave history itching followed by skin infection 3 wks before (sore throat) of presenting complaints. The patient has no history breathlessness, orthropnea and palpitation, convulsion, unconsciousness and recent and previous history of jaundice and no history of similar type of attack . General examination reveals patient is ill looking with puffy face, mildly anaemic and mild pitting edematous, non ecteric and with blood pressure 150/ 95 mm of hg , JVP not raised The patient has no feature of mal -absorption and hepatic insufficiency but there is scratch mark present over hand and abdomen and old scar of healed skin infection and IV canula in situ. Bed side Heat coagulation test shows mils protein uria and absence of reducing substance. Examination of alimentary system reveal mildly distended abdomen due to ascites with out engorged vein and organomegaly . Rest of the system reveals no abnormality

Provisional diagnosis
Acute glomerulonephritis
Differential diagnosis
Nephrotic syndrome

What ur diagnosis why?
Acute glomerulonephritis

- Sudden onset
- HO of post. streptococcus infection skin infection /Sore throat
- Oligouria
- HTN
- Generalized edema
- Heat coagulation test –massive protein uria

What is ur differential diagnosis _ Nephrotic syndrome
Point in favor of ur differential diagnosis

- Pitting edema

Point disfavor

- Acute on set
- Bed side heat coagulation test protein + + +
- Normal blood pressure
- Oligouria
- evidence of streptococcal infection

Why not case of CCF
On history
The point in favor of CCF is only dependent edema

Other sign and symptoms CCF is absent such
No history exertional dyspnea . orthropnea , chronic cough with productive sputum
On Exam –
most important
JVP not raised
No tender hepatomegaly
Others sign
lung crep + , spasm ,ronchi , vesicular breath sound with prolong expiration – are absent
murmur , left parasternal heave absent

what types heart failure occur In AGN --- left heart failure ?

Cardinal feature of right heart failure /CCF

- Dependent edema
- Tender hepatomegaly
- Raised JVP

Cardinal feature of left heart failure

Pulsus alternus / Tachycardia
Gallop rhythm
Bilateral Basal crep ++

Investigation :
Urine RME
Protein + +
Pus cell 2-5 cell / mm
RBC @RBC cast = +
24 hr total urinary protien < 3 gm / 24 hrs
S.creatinine
urea
ASO titre
USG of kUB
Complement $C_{3\downarrow}$ and C_4
CXR and ECG

What is cast
Csat is coagulated protein ( ) secreted from the tubules .

When it contain blood Then called RBC cast
When it contain WBC Then called WBC cast
What is the pathognomic AGN ?
RBC cast is the pathognomic of nephritic syndrome

How will u differ glomerular RBC from other RBC
RBC from glomerular orging is dysmorphic (shap change as the RBC come glomerulus's during passing through tubule change its morphology)

RBC in urine due to other cause are isomorphic

Complication
1. Hypertensive encephalopathy,
2. acute left ventricular failure
3. Renal failure / Azotemia
4. Hyperkalemia

Treatment

Treatment is mainly supportive and symptomatic

Diet –

- Protein restriction
- Fruits restriction (to avoid hyperkalaemia)
- Fluid 500ml + previous day out

Antibiotic

- **Tab. Pen-V 250 mg**
  1 + 1 + 1 + 1

Diuretic

Tab. Lasix or inj .lasix depend on out put
1 + 1 +0 / 1amp IV BD

Anti HTN

And treatment of complication

Cause of haematuria
Pain ful haematuria
Renal stone
UTI
Painless haematuria
GN / AGN
TB
Tumour
Polycystic kidney
Schistosomiasis
Bleeding disorder

Which organism is responsible from AGN

group A β-hemolytic streptococci

- S. pyogenes

Why AGN occur 1-3 weeks after skin infection

It is the time need to develop antibody after Bacterial infection

N**ame the organism responsible for scabies**

Cause by female gravid Sarcoptes scabiei

Tell the mechanism AGN from scabies

Following scabies –scratching due to itching --- Breach the continuity of skin and –secondary infection with streptococcus infection --- immunity develop against strep.--- antibody +antigen + complement

C_3

Why kidney affect in after strep. Infection

1. AGN develop only when skin or sore throat infection by "nephritogenic strain of " streptococci
2. kidney is involved due antigenic mimicry between the bacteria and GBM of kidney

what type reaction it is

Type -3 hypersensitivity reaction

CASE – 1

1. This patient suddenly Develop head ache followed by convulsion and unconsciousness
2. 4 boys with scabies were playing in field –but suddenly Develop head ache followed by convulsion and unconsciousness

DX **is hypertensive encephalopathy**

CASE -2

1. If This patient suddenly Develop severe respiratory distress or orthopnea

D_X is **Acute left ventricular failure**

How follow up the patient

Every day maintain BP chart
Maintain heat coagulation
Daily weight chart
Urine RME

How long the patient should be kept in hospital

Until Bp control
Urine free of RBC

Q .A patient of AGN S. creatinine is progressively increasing day by day what is ur diagnosis?

Ans. RPGN

Causes :

- Post.streptococcal glomerulonephritis
- Good posture
- Ig A nephropathy
- Mesangiocapillary glomerulonephritis

Q. What will u got in histology?

- Cresent formation

Q . What will be the treatment?

- IV methyl prednisolone

History

- Patient with edema + without HTN + heat coagulation test (+++)
  - Provisional Dx NS
  - DD is AGN

If edema with HTN

Then

- Provisional Dx AGN
- DD is NS

Onset	sudden –AGN , insidious or gradual ---NS
Swelling	where first appear
Urine out put	scanty , amount (glass) , color (dark or frothy), how many time he have to evacuate his bladder
For AGN	history skin infection , sore throat and ear infection or any other infections prior to onset of this swelling
To exclude CCF	history breathlessness and palpitation either in rest or exertion history chronic cough
Recent or past history of jaundice	to exclude CLD also for secondary causes of viral hepatitis
to exclude CLD	history of blood transfusion previously
	of vomiting out of blood and black tarry stool,
	Loss of body and pubic hair and decreased frequency of saving & loss libido
HO fever	to exclude malaria
To exclude DM	polyuria and polyphagia
To exclude 2ndary causes SLE	joint swelling and pain ,rash

mal-absorptions	History alteration of bowel habit and passage mucous stool.
past history	Diabetic , malaria , previous edema (relapse)
drug history to exclude secondary causes	History taking pain killer, anti hypertensive drug (captopril). After admission in hospital he getting some inject able and oral drug but name of which he could not mention

If patient is AGN

HTN	Take history of headache and blurring vision
hypertensive encephalopathy	head ache , convulsion and unconsciousness
for LVF	orthropnea , sudden severe dyspnea

Prognosis of AGN?
prognosis is good 85% achieved recovery 5% develop RPGN
How long will u prescribe bed rest?
until disappearance of following clinically a) HTN b) edema c) oligouria **investigation:** Urine free of RBC and RBC cast until urea and creatinine level back to normal
oliguria , anuria , haematuria and dark color urine causes ?
Why HTN occur in AGN?
due to salt and water retention due to secondary hyperaldosteronism
why hyper lipidaemia occur in NS?
in NS there is hypoalbuminaemia -> decrease oncotic pressure → stimulate liver for lipoprotein synthesis → causes lipidaemia
When NS— become hypertension ?
HTN – is not feature of NS – but it may present only in following cases when it associated with a) nephro-nephritic syndrome b) diabetic nephropathy c) turn into CKD d) SLE e) focal glomerulosclerosis
steroid resistance and steroid depended , steroid toxic ?
steroid resistance : poor response with full dose of corticosteroid for 8 weeks **steroid dependent** : when symptoms appear after withdrawal of steroid **steroid toxic** : when side effect of steroid appear with treatment of steroid
The treatment of relapse ?
first relapse treat with oral prednisolone second relapse / frequent relapse –give cyclophosphamide
How will differentiate AGN , NS, CRF, UTI/PYELONEPHRITIS in urine RME ?
AGN—RBC cast , RBC UTI – WBC cast, pus cell NS—fatty cast CRF—granular cast

difference between nephrotic syndrome and AGN		
	AGN	nephrotic syndrome
onset	sudden	insidious
HO	sore throat skin infection	not so in adult HO 2ndary cause e.g. DM
urine color	red / dark /cocacola color	frothy , smoky
HTN	present	absent
edema	mild to moderate	massive
oligouria	present	may present at late stage
urine RME	RBC and RBC cast protein uria + to ++	no RBC or RBC cast protein uria ++++

causes of AGN and nephrotic syndrome ?

AGN	**NS**
1st say 1. post streptococcal glomerulonephritis if sir want to more than say 2. connective tissue disease a. SLE 3. vasculitis 4. Henoch scholein purpura **other infection** other bacterial infection 1. infective endocarditis 2. meningo coccal infection 3. pneumococcal infection 4. plasmodium malarae 5. viral hepatitis	primary causes FM3 M—minimal change M—membranous GN M—mesangio-capillary GN F—Focal and segmental glomerulosclerosing I—IgA nephropathy secondary causes **CID** **C—collagen disease –**SLE , RA **C—Carcinoma –**bronchial , non-Hodgkin lymphoma **I—infection** 1. HBV,HCV, HIV 2. plasmodium malarae 3. secondary syphilis 4. leprosy (type II lepra reaction) 5. bacterial endocarditis **D—DM** **D—drug –**pencillamine , captopril (ACE), gold, NSAID

Name some infective cause of nephrotic syndrome ?
see above
HIV, obese, heroine addicted → with NS occur ?
Focal and segmental glomerulosclerosing

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Name some infective cause of nephrotic syndrome ?
see above
HIV, obese, heroine addicted → with NS occur ?
Focal and segmental glomerulosclerosing

Nephrotic Syndrome

PARTICULARS OF PATIENT:

Name: Rabiual islam
Age: 15 years
Sex: Male
Marital status : unmarried
Occupation: student
Religion: Islam
Address: Mouchak, mymensingh
Date of admission:1.12.09 at 7pm
Date of examination:10.12.09 at 10 am

Presenting complaints
Generalized swelling of body for 25days
Reduced volume of urine for 5 day

History of present illness
According to the statement of the patient, he was reasonable well 25 days ago .Then he gradually develop generalized body swelling which he first noticed at face around the eye lid and later on spread all over the body hampering his daily activity .At the beginning of the swelling urine out put was normal but for the last 5 days he noticed reduction in the urinary volume and frequency. Color of urine is normal and he have to evacuate bladder 3-4 times pre 24 hours and amount 750 ml per day .The patient dose not gave any history skin infection ,sore throat and ear infection or any other infections prior to onset of this swelling .The patient has no history breathlessness and palpitation either in rest or exertion. The patient has no recent or past history of yellow coloration of sclera or any other parts of the body. The patient has no history of blood transfusion previously. The patient has no history joint swelling and pain .The patient has no history of vomiting out of blood and black tarry stool, alteration of bowel habit and passage mucous stool.

History of past illness

The patient suffered from same type of problem at the age of six years and treated in local hospital and was cured completely. The patient is Normotensive, non diabetic. No previous history of jaundice and malaria. Rather this there is no other significant medical and surgical disorder.

Drug history
The patient has no history taking pain killer , and anti hypertensive drug . after admission in hospital he getting some inject able and oral drug but name of which he could not mention . for previous attack take some oral medication and he forgot those name

PERSONAL HISTORY
Non smoker, Non alcoholic.

FAMILY HISTORY

- None his family member is suffering from this type of disease .

They are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition and lived in Crowding house

Housing: Tin shade house.
2 rooms, which accommodate 8 0f his family member.

Sanitation:1 sanitary latrine.

Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized according to EPI schedule

GENERAL EXAMINATION

- Appearance – puffy face
- Body built – average
- Nutritional status –average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – mild
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : + + + +
- Dehydration – Nill
- Skin –there are some striation over the lower abdomen for ascites . other wise general skin condition is normal . no evidence of scratch marks
- Body hair distribution – normal distribution of axillary & pubic hair
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 130/80 mm of Hg
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :
- Bed side urine examination show s massive proteinuria (+ + + +)
  Sugar= Nill

Systemic examination

Examination of Alimentary system

■Mouth & pharynx

■Tongue – Dry & coated

■**Abdomen Proper**
■**Inspection**
■Abdomen – distended and flanks are full
■Umbilicus – centrally placed and everted and slit is transvers
■Movement with respiration – present
■Scar mark – no scar mark but some striae
■Visible peristalsis – absent
■Visible vein – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia – normal

■**Palpation**
■Superficial & deep palpation: No muscle guard, no tenderness, no organomegaly
■Fluid thrill present (if with huge ascitis)or absent (if ascites is mild)
■**Percussion**
■**Shifting dullness is present**
■**Paddle sign in case of mild ascites**
■**Auscultation**
■Bowel sound –present t

CARDIVASCULAR SYSTEM
•Pulse : 72 b/min
Regular, normal in volume & character, all the peripheral pulses are normal.
•BP : 120/75 mm of Hg
•**JVP : Not raised**
•**Precordium** :
Inspection: Normal
Palpation: Apex beat in lt 5th intercostals space 9 cm from midline
No para-sternal heave and no palpable
AuscultationS1&S2 audible in all auscultatory area
No added sound and no murmur

RESPIRATORY SYSTEM
•**Inspection** : Size and shape of the chest : Normal
•Movement is symmetrical
•No evidence of respiratory distress
•**Palpation** :
Trachea: Trachea central
Apex beat: in left 5th intercostals space 9 cm from midline normal in character
Vocal fremitus : normal
Percussion: resonance
Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM

- **Higher psychic function including speech**: normal.

Fundoscopic exam: Normal
Cranial nerves: intact

- **Motor system examination**

Motor functions are normal in all four limbs
Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

Salient feature

Rabiual islam 15 yrs smoker normotensive, nondiabetic, muslim student hailing from Mouchak, mymensingh got admitted into MU-1 MMCH with gradually development of generalized body swelling which he first noticed at face around the eye lid and later on spread all over the body. With Recently reduction in the urinary volume and frequency. and The patient dose not give any history haematuria, skin infection ,sore throat any other infections of body prior to onset of this swelling .The patient has no history exertional dyspnea, orthopnea and palpitation, jaundice and malaria . The patient has no history of blood transfusion arthritis, haematomesis and malenae. The patient has no feature of mal absorption and hepatic insufficiency and NSAID intake .Patient had experience similar incident at age 6 yrs and was cured with medical treatment. general examination reveals patient is ill looking with puffy face, mildly anaemic and severely edematous, non ecteric and with normal blood pressure, JVP not raised and there some striation on abdomen and IV canula in situ. Bed side Heat coagulation test shows massive protein uria and absence of reducing substance. Alimentary system examination reveal
Abdomen is distend with out engorged vein, flanks are full , umbilicus is central everted with transverse slit . Shifting dullness and fluid thrill present and no organomegaly with normal testis and body hair distribution. Other system reveals no abnormality

Provisional diagnosis
Nephritic syndrome

Differential diagnosis
AGN

Investigation :

Urine RME
Protein + + + +
Pus cell 2-5 cell / mm
RBC @RBC cast = -
24 hr total urinary protien > 4 gm / 24 hrs
s.albumin ↓
S.cholesterol ↑
S.creatinine
RBS
HBs Ag
USG of kUB
CXR and ECG

What is the provision diagnosis and what are s point in favour of your diagnosis

The provisional diagnosis is ---- Nephrotic syndrome

Insidious on set
Have the previous HO similar episode
Massive pitting edema
Bed side heat coagulation test protein + + +
Normal blood pressure
No oligouria
No evidence of streptococcal infection

What is ur differential diagnosis
AGN
Point favor of ur D/D
Generalized edema
Point against of ur D/D
No HO of post. streptococcus infection skin infection /Sore throat
No Sudden on set
No Oligouria
No HTN
Heat coagulation test –massive protein uria

Why not case of CCF
On history
The point in favor of CCF is only dependent edema
Other sign and symptoms CCF is absent such
No history exertional dyspnea . orthropnea , chronic cough with productive sputum
On Exam –
most important
JVP not raised
No tender hepatomegaly
Others sign
lung crep + , spasm ,ronchi , vesicular breath sound with prolong expiration – are absent
murmur , left parasternal heave absent

Why this is not case of CLD
- There in no history of jaundice
- On examination there are no stigmata of CLD such
  - Gynaecomastia, spider, palmer erythma
  - leuconychia ,testicular atrophy ,jaundice
  - loss of pelvic and axillaries hair
  - Splenomegaly, engorged veins

What do mean by Nephritic and Nephrotic Syndrome
Nephritic syndrome1
Haematuria (brown urine)
Oedema and generalised fluid retention
Hypertension
Oliguria

Nephrotic syndrome2

Massive proteinuria-usually > 3.5 g/24 hrs (urine may be frothy)
Hypoalbuminaemia (< 30 g/l)
Oedema and generalised fluid retention
Hyper lipideamia

Difference between

AGN	**NS**
1. Onset acute	1. Onset insidious
2. Older children	2. Younger children
3. Preceding throat or skin infection	3. no such infection
4. Oliguria at onset	4. Oliguria later
5. Edema mild to moderate	5. Edema marked, ascites, genital edema, pleural effusion
6. Hypertension and hematuria	**6.** Hypertension **30 %**
7. Urine ---protein ++ , RBC and RBC cast +	**7. Urine ---protein ++ , RBC and RBC cast -**
8. UTP < 3 gm	8. **UTP > 3 gm**

Proteinuria

24 -hr urine protein	**Significance**
< 0.03 g	Normal
0.03-0.3 g	**Microalbuminuria**
0.3-0.5 g	Dipsticks positive
0.5-2.5 g	Source equivocal
> 2.5 g	Glomerular disease likely
> 4.0 g	Nephrotic range-always glomerular

Micro albuminuria
When 24 hrs urinary protein between 0.03 to 0.3 gm

Cause of nephrotic syndrome	**Cause of nephritic syndrome**
Primary cause	Post streptococcus glomerulonephritis
Minimal change disease	Ig A nephropathy
Membranous glomerulo nephritis	RPGN
Focal segmental glomerulo sclerosis	God posture syndrome
secondary cause of nephrotic syndrome	
DM	
Amyloidosis	
SLE	
Infection	
HBV HCV	
Malaria –palsmodium Malarae	
HIV	
DRUG – NSAID ,	
AEI – captopril	
Gold and penicilinamide	
Malignancy	
Hodge king lymphoma	

CAUSES OF GLOMERULONEPHRITIS ASSOCIATED WITH LOW SERUM COMPLEMENT

Post-infection glomerulonephritis
Subacute bacterial infection-especially endocarditis
SLE
Cryoglobulinaemia
Mesangiocapillary glomerulonephritis-usually type II

What are the primary cause of nephrotic syndrome and 2ndary cause of nephrotic syndrome

How ill investigation in patient with nephrotic syndrome ?

Treatment of patient with nephrotic syndrome

Treatment is supportive treatment, specific treatment and treatment of complication

Bed rest

Diet

Normal and salt restriction
Fluid restriction only in case massive edema

Why bed rest?
- To decrease catabolism and
- Increase renal perfusion

Diuretic

Frusemide oral or injectable form

Specific treatment

Steroid Tab. Prednisolone 1 mg / kg body weight

If u r in pediatric board then follow their protocol for steroid

Treat ment of complication

If HTN then give anti hypertensine --- ACE I

If infection give antibiotic

Treatment of thromboemolism if present

Complication of nephrotic syndrome

- Infection --- Due to loss of immune globulin in blood =more prone to pneumococcal infection
- Venous Thrombo-embolism / hyper coagulability (due to relative loss of anti-coagulant such anti-thrombin II , protein C , protein S and increase secretion of procoagulate by liver .)
- Hypercholesterolemia --- Due to increase secretion of lipoprotein
- Edema

What is underling cause of NS in this patient

If the patient is child ----then answer is minimal change disease
If the patient is adult ---- the answer will be the membranous glomerulo nephritis

Prognosis in case minimal change disease

Respond to treatment is good , not turn in to chronic GN

Prognosis in membranous disease

1/3 achieve remission and 1/3 remain in nephrotic state or CGN and 1/3 turn in to CRF

What will give if u adult patient not respond to steroid

Cyclophosphamide (2mg / kg body wt)

What type drugs the cyclophosphamide is ?
It is the cytotoxic drug . use in cancer chemotherapy

How it act here ?
It act as immunosuppressant . as the disease is immune mediate

When Cyclophosphamide use in Minimal change disease
If more than 2 relapse

How can u confirm the diagnosis
By doing renal biopsy
Minimal change is responsible for 90 % NS in adult

Why HTN in AGN then NS ?
For following two reason

1. In AGN there is Low GFR that causes stimulation of renin-aldosterone axis and retention of salt and water
2. this water remain in intravascular then extra vascular

Q a patient with nephritic syndrome come to RTA and haematuria what Is the Dx ?
Ans. Ig nephropathy

Q a patient with nephritic syndrome come to u fever and loin pain what Is the Dx ?
Ans. Renal vein thrombosis
Q. Before giving cyclophosphamide in patient with relapse case of NS what will u do ?
Ans. Will do renal biopsy
THIS ONLY FOR THOSE WHO WANT TO GET MORE MARKS

RENAL BIOPSY
Indications

- Acute renal failure that is not adequately explained
- Chronic renal failure with normal-sized kidneys
- Nephrotic syndrome or glomerular proteinuria in adults
- Nephrotic syndrome in children that has atypical features or is not responding to treatment
- Isolated haematuria or proteinuria with renal characteristics or associated abnormalities

Contraindications

- Disordered coagulation or thrombocytopenia. Aspirin and other agents causing platelet dysfunction should be omitted for elective biopsies
- Uncontrolled hypertension
- Kidneys < 60% predicted size
- Solitary kidney (except transplants) (relative contraindication)

Complications
Pain, usually mild

- Bleeding into urine, usually minor but may produce clot colic and obstruction
- Bleeding around the kidney, occasionally massive and requiring angiography with intervention, or surgery
- Arteriovenous fistula, rarely significant clinically

In children < 15 yrs, nephrotic syndrome almost always caused by primary renal disease (~ 98 %)

In adults nephrotic syndrome may often be associated with secondary renal disease

History

- Patient with edema + without HTN + heat coagulation test (+++)
  - Provisional Dx NS
  - DD is AGN

If edema with HTN
Then
  - Provisional Dx AGN
  - DD is NS

Onset	sudden –AGN , insidious or gradual ---NS
Swelling	where first appear
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If patient is AGN

HTN	Take history of headache and blurring vision
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for LVF	orthropnea , sudden severe dyspnea

Kala-azar

PARTICULARS OF PATIENT:

Name: Md. Kamrul Hassan
Age: 40yrs
Sex: Male
Marital status: Married
Occupation: Farmer
Religion: Islam
Address: Fulbaria, Mymensingh
Date of admission: 8.12.09 at 7pm
Date of examination: 10.12.09 at 7.15am

PRESENTING COMPLAINTS

- Fever for 2 months
- Mass (or lump) in left upper abdomen for 1 ½ months

HISTORY OF PRESENT ILLNESS

According to statement of the patient, he was reasonably well 2 months back then he developed high grade fever which did not follow any specific pattern. The fever used to rise some times at the evening and some times at the morning ,lasted for about six to eight hrs, which was not associated with chills and rigors but disappeared without sweating after taking paracetamol (or spontaneously with out medication).The highest recorded temperature was 103 F ^{o}. Some times The patient remained afebrile for a week in between episodes of fever. Fever was not associated cough, sputum, chest pain or breathlessness. The patient had no night sweating or itching. The patient also noticed a pain less mass in the left upper abdomen for last 1 ½ months. The lump was gradually increasing in size toward the umbilicus day by day. The patient has no history coughing out of blood, bleeding per nose, vomiting out of blood, black tarry stool. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. There was no history of loose motion, abdominal pain, and alteration of bowel habit, joint pain and rash, area of hypo-pigmented or hyper-pigmented, headache or vomiting. He gave no history of increased frequency, urgency, hesitancy and red urine. Despite of good appetite he lost about 10 kg weight in last two months. With above complained he visited several times to local doctors every time he was treated with different types of antibiotic name of which he could not mentioned. Now he got admitted in to MU-1, MMCH for further evaluation and better management .

HISTORY OF PAST ILLNESS

No history of DM/HTN/TB

No history of such type of illness before

PERSONAL HISTORY

smoker (10 Stick/day) for last 20 years. Non alcoholic. Betel nut chewer. No history of extra-marital sexual exposure . no history IV drug abuse (to exclude infective endocarditis)

FAMILY HISTORY

- His wife and children are healthy.
- His other family members are also healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition. and lived in Crowding house
Housing: Kacha house
2 rooms, which accommodate 8 0f
His family member.
Sanitation:1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized

Drug History

Patient received some oral and parental antibiotics and ant malarial drug (write only if patient give u this history)

GENERAL EXAMINATION

- Appearance –ill looking
- Body built – average
- Nutritional status – below average /average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – mildly anaemic
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : Absent
- Dehydration – Absent
- Skin –normal
- Body hair distribution –normal
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse –88/min, low volume, regular
- B.P – 140/85 mm of Hg
- Respiratory rate – 25/min
- Temperature –100^0F
- Weight : 45 kg
- Height : 1.6 meter
- BMI : 17.57 kg/m2

SYSTEMIC EXAMINATION

Examination of Alimentary system

■**Mouth & pharynx**

Normal

■**Abdomen Proper**

■**Inspection**

■Abdomen – upper abdomen is distended and flanks are not full

■Umbilicus – centrally placed and inverted

■Visible vein – absent

■Movement with respiration – present

■Scar mark – no scar mark and no striae

■Visible peristalsis – absent

■Hernial orifice – intact

■Hair distribution – normal

■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,

■Liver is palpable which 5 cm from right costal margin in mid clavicular line having smooth surface , sharp margin (rounded), non tender ,firm in consistency , upper border of liver dullness is in right 5 the intercostals space with absent hepatic bruit and liver span is 15 cm .

■ spleen is enlarged which is 6 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Finger insinuation is not possible

■Kidney and urinary bladder is not palpable

■No intra abdominal lymph adenopathy

■Fluid thrill absent

■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present

■Renal bruit absent

CARDIVASCULAR SYSTEM

•Pulse : 88 b/min

Regular, normal in volume & character, all the peripheral pulses are normal.

•BP : 140/85 mm of Hg

•**JVP : Not raised**

•**Precordium** :

Inspection: Normal

Palpation: Apex beat in lt 5th intercostals space 9 cm from midline

Auscultation of S1&S2 audible in all auscultatory area

No added sound and no murmur

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Movement is symmetrical

•No evidence of respiratory distress

•**Palpation** :

Trachea: Trachea central

Apex beat: in left 5th intercostals space 9 cm from midline normal in character

Vocal fremitus: normal

Percussion: resonance

Auscultation:

Breath sound is vesicular in all parts of the chest.

No added sound

Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.

Fundoscopic exam: Normal

Cranial nerves: intact

Motor system examination

Motor functions are normal in all four limbs

Sensory examination

All modalities of sensation are intact in both upper and lower limbs

Cerebellar signs: Absent

Signs of meningeal irritation: Absent

SALIENT FEATURE

Md. Kamrul Hassan aged 40 yrs old married muslim smoker normotensive nondiabetic farmer hailing from Fulbaria Mymensingh got admitted into MU-1 MMCH with high grade intermittent fever for 2 months and mass in left upper abdomen for 1 ½ months . The fever was irregular in natural, some times it appeared in morning and sometimes in the evening but every time it lasted for about 6 to 8 hours. The fever was not associated with chill and rigor and subsided with sweating after taking paracetamol and again reappeared. Some times the patient enjoy period of a pyrexia in between attack. Highest recorded temperature is 103 ^{0} F. The patient also noticed a painless mass in the left upper quadrant abdomen for last 1 ½ months. The lump was gradually increasing in size toward the umbilicus day by day. Fever was not associated cough, sputum, haemoptysis, chest pain or breathlessness. The patient had no night sweating or itching. On query, he had no history of contact with known TB patient or traveling into hilly or border area and abroad, epistaxis, haematemesis and malena ,abdominal pain ,alteration of bowel habit, joint pain, rash, headache or vomiting and urinary problem. The patient had no significant past history but he took several antibiotics both parental and oral and one course of anti-malarial drugs prescribed by local physician. Despite of his good appetite he lost about 10 kg weight in last 2 months .the patient has no history of blood transfusion and extramarital sexual exposure and IV drug use. General examination revealed the patient is ill looking, mildly anaemic temp. 100^{0}F , pulse is regular 88 beat / minute with normal blood pressure (140/85 mm of Hg) respiratory rate is 18 / min and pattern is normal . The patient is non-ecteric, non-edematous, boney tenderness absent; The patient has no lymph-adenopathy, clubbing, splinter hemorrhage, hypo or hyper pigmentation. Examination of elementary system reveals non tender hepatosplenomegaly. (Liver is enlarged which 5 cm from right costal margin in mid clavicular line having smooth surface , sharp margin (rounded), firm in consistency , upper border of liver dullness is in right 5 the intercostals space with absent hepatic bruit and liver span is 15 cm . Spleen is enlarged which is 6 cm from left costal margin in ant

Axillary line toward its long axis having smooth surface , firm to hard in consistency,notch in its upper border and there is no splenic bruit .—if examiner ask u to say only salient feature then u can tell this other wise tell him only non tender hepatoplenomegaly). He has no ascites or intra-abdominal lymphadenopathy . Other systemic reveals no abnormality.

Provisional diagnosis
Kala-azar

Differential diagnosis
Chronic malaria
Disseminated tuberculosis

Investigation
- First do

CBC ---progressive leucopenia with relative lymphocytosis
ESR – is highly raised
Hb --- ↓
PBF—pancytopenia
Immunological test
ICT for Kala-azar
RK-39
DAT—Direct agglutination test
Definitive test
Splenic puncture
Bone marrow aspiration

TREATMENT

Specific treatment
- **Sodium antimony gluconate (SAG) / Na stiboglocunate :**
20 mg/kg / day IV daily for 30 days

Supportive treatment

- Adequate nutrition and hydration
- Anti-pyretic and tepid sponging
- Treatment of complication

Question on the basis of history

What is u r provisional diagnosis? What are the points in favour of ur diagnosis
My provisional diagnosis is kala-azar
Point in favor
Patient hailing from Endemic Zone (fulbaria)
Prolong high fever with irregular pattern
Aneamia, weight loss
Hepato-splenomegaly

<u>What r ur differential diagnosis and what are the point in favor and against ur diagnosis ?</u>

- Chronic malaria
- Disseminated tuberculosis

Chronic malaria

Point in favor diagnosis

- Prolong fever
- Hepato-splenomegaly

Point against

- Not coming from hilly or border area (although malaria occur any area)
- Not associated with chill and rigors
- In malaria splenomegaly is mild to morderate not huge

Disseminated tuberculosis

Point in favor

- Prolong fever
- Weight loss
- Hepatosplenomegaly

Point against

- Hailing from endemic Zone of kala-azar
- No feature of other system involvement
- Such as – no respiratory problem such cough , haemoptysis , pleural effusion
- Abdominal— absence of Ascites , alteration of bowel habit , doughy feeling of abdomen or feature of recurrent intestinal obstruction

Why this is not case of leukemia

Although patient has high fever and hepato-splenomegaly it is not a case leukemia because of

- The patient is not toxic.
- Absence of boney tenderness.

***If it is chronic leukaemia what would ur be diagnosis*?**

Chronic myeloid leukemia

Why u call it CGL / CML?

Due to huge splenomegaly

Which age is Common for CGL*?*

It occur in older age.

Why it is not a case of lymphoma?

Point favor

Fever

Hepatosplenomegaly

Point against Ares

No lymphadenopathy (including intra abdominal) and

Absence drenching night sweating and itching

Why this not a case of enteric fever?

This not a case of enteric fever because

Enteric fever does not persist 2 months

Usually fever is continued in nature and may have relative bradycardia

Mild splenomegaly

Is this may be infective endocarditis?

Fever and splenomegaly goes favor of sub-acute infective endocarditis but in endocarditis there is clubbing, splinter hemorrhage , changing murmur and HO IV drug use or tooth extraction

What are the investigation u want to establish ur diagnosis?

CBC ---progressive leucopenia with relative lymphocytosis
ESR – is highly raised
Hb --- ↓
PBF—pancytopenia
Immunological test
ICT for Kala-azar
RK-39
DAT—Direct agglutination test
Definitive test
Splenic puncture
Bone marrow aspiration

What are the finding of CBC*?*

Leucopenia –monocytosis
Progressive leucopenia with lymphocytosis
Hb—decrease and ESR is raised

***what do u mean by Progressive leucopenia* ?**

if do u serial CBC total count will decrease day by day
suppose 7 days ago TC was 7000 then 5 day later TC become 4000

what will be the ESR ?

ESR will be highly raised more than 100 or near to 100

What is finding of PBF ?

Pancytopenia --- anemia , leucopenia , thrombocytopenia

What investigation will u do ?

Immunological examination
Like ICT for Kala-azar , RK-39(recombinate kynoplast) , DAT (direct agglutination test)

What they detect (antigen or antibody) @ when they appear ?

All these detect antibody and appear after 2/3 wks after infection . These tests remain positive for several months after cure.

How will confirm this D_x or what will be the next examination?

Bone marrow examination ---sensitive 85 %
Splenic puncture ----95-98 % sensitive
Others –liver and lymph node

Which one is more sensitive?

Splenic puncture

What will u see in spleen and bone marrow material*?*

LD body (Leishman-Donovan bodies).

Where u see it?
It is seen in macrophage

What is the form of LD in bone marrow and spleen
It is the form amastigot

Where promastigote form is found
It is found in mosquito and culture media

What is the differentiate between amastigote and promastigote?
Flagella absent in Amastigot form

Which is media needed for culture and duration of culture
NNN media (Nicolle-Nove McNeal) it take 1-4 wks

Can LD body found in blood ?
Rarely it found in peripheral blood in Buffy coat

What is the cardinal the feature of kala-azar ?
- H/O fever more than 2 weeks
- Residing/Traveling in endemic area
- Splenomegaly
- Weight loss
- Anemia

Describe the fever of kala-azar
Fever- usually insidious and may be associated with chills and rigors. Fever intensity decreases over time and patient may become afebrile for weeks to month followed by relapse of fever.

Cause of anemia in kala-azar
Anaemia may result from
- Bone marrow infiltration,
- Hypersplenism,
- Autoimmune hemolysis &
- Bleeding.

Cause of bleeding in kala-azar
This occurs as a result of thrombocytopenia
Or also due drug sodium stibo gluconate

What are the complication of kala-azar
1. **Secondary infections:**
   - **Pneumonia**
   - **Tuberculosis**
   - **Amoebic or bacillary dysentery**
   - **Gastroenteritis**
   - Herpes zoster
   - Chickenpox
   - Skin infections, boil, cellulitis, scabies
   - Cancrum oris

2.**Bleeding manifestation-**
from nose, retina, GIT etc.

3. **Post Kala-azar Dermal Leishmaniasis(PKDL)**
4. Post kala-azar laryngitis and colitis
5. Post kala-azar splenomegaly
6. **Glomerulonephritis**
7. **Nephrotic syndrome**
8. Cirrhosis of liver

What is the treatment of KALA-AZAR ?

1ST line

Sodium antimony gluconate (SAG)

Dose of SAG is given in a dose of 20 mg/kg IM or slow IV or dilute with NS for a period of 30 days

Next

- **Amphotericin B**
- **Liposomal amphotericin** B

Liposomal amphotericin B in the treatment of kala-azar --The recommended schedule is 3mg/kg for 5 days , 7th day , 14th day --- it is better it have no nephrotoxicity and save in pregnancy

amphotericin B—1 mg / kg body weight slow iIV infusion over 4-6 hours for 20 days

Paromomycin

Paromomycin has been registered in India The recommended dose is 15 mg/kg IM for 21 days

Pentamidine isetionate

This was used to treat Sb-refractory patients with VL

What is practice in our ward ?

Sodium antimony gluconate (SAG***)***

How it given in ur ward?

In our ward it has been given slow iv dilute with normal saline

How will u understand Response to treatment?

A good response results in abatement of fever, / decrease fever
A feeling of well-being,
Gradual decrease in splenic size,
Weight gain and recovery of blood counts.

When will u told it relapse?.

Relapse is indicated by

- Enlargement of the spleen,
- Return of fever,
- Weight loss and
- Decline in blood counts

What are the comoplicaton in stibatin

1st –cordiatoxicity

Minor- myalgia, arthralgia
Major-arrhythmias, Heart failure, oedema, jaundice, decreased urine

What will u do before and after giving sodium antimony?

Before starting I will do ECG and CXR, S.creatinine, SGPT

Follow up

Every day see pulse and rhythm

ECG should be done every 5-7 days interval

What are side effects of Amphotericin B?

thrombophlebitis, diarrhoea and vomiting, are extremely common. Serious adverse events, such as renal or hepatic toxicity, hypokalaemia, thrombocytopenia, myocarditis . main side effect is nephrotoxic .

what are the advantage of Liposomal amphotericin B over Amphotericin B ?

Liposomal amphotericin B is safe and less toxic than Amphotericin B and it safe in pregnancy but costly

What will do if patient is pregnant?

We have to treat as kala-azar is fatal disease.

Drug choice is Liposomal amphotericin B . if patient not able take this drug due to high cost then u give Sodium antimony gluconate (SAG)

What drug use in our ward ?

Sodium antimony gluconate (SAG) /inj. Stibatin

inj. Stibatin I vail = 30 ml , 1 ml = 100 mg Sodium antimony gluconate

in our ward we practice 8 ml + 42 ml NS or 8 ml + 80 ml NS slow IV

whar will u do if patient develop bleeding after Sodium antimony gluconate (SAG) /inj. Stibatin

drug should be stop and do PBF with platelet count , PT and liver function test .

Bleeding due to abnormal preparation , expire date

What do u mean by Relapse , Reinfection , Resistant ?

Relapse –after cure again occurrence of kala-azar with in 6 months

Reinfection –after cure again occurrence of KAlA-azar after 6 month

Resistance—no respon to drug

Treatment of Relapse, Reinfection, Resistance?

Relapse – Sodium antimony gluconate (SAG) /inj. Stibatin for 40- 60 days or Amphotericin B

Reinfection – Amphotericin B

Resistance---- Amphotericin B or pentamidine isothionate

What complication patient with kala –azar may come after 2-3 year

Post Kala-azar Dermal Leishmaniasis(PKDL)

Why PKDL developed ?

PKDL usually develops 6 months-5 years following an attack of untreated or incompletely treated kala–azar

Can PKDL occur with out previous HO of kala-azar ?
However 15% of PKDL cases occur without thepreceding history of kala-azar.

What are present of PKDL ?
They have only skin lesion which are the area of hypo-pigmentation or hyper pigmentation. It may be macular, papular, nodular or mixed.

Is these lesions are infective or not?
Yes, these contain LD body

What is investigation to D_x
Skin slit smear --- it see LD body

What is the differential diagnosis of PKDL?
In contrast to leprosy, sensation over the lesions is preserved& the lesions do not ulcerate.

What is the treatment of PKDL?
Sodium Antimony Gluconate (SAG)
Dose: 20 mg/kg body wt daily for 20 days per cycle
Route: IM/IV
Duration: Six cycles with 10 days interval between cycles

Organism and vector of kala-azar?
It is a protozoa name Leishmania donovani
Vector is the phlebotomine sandfly.
Man is the only reservoir

How it transmit to human ?
Sandflies pick up amastigotes when feeding on infected patients or animal reservoirs.in gut of sandfly it multiply .Flagellar promastigotes are introduced in to human by the feeding female sandfly. Sandfly saliva helps Leishmania evade immunity.

What are the form of leishmania ?
Amastigotes----found in human
Promastigotes (Flagellar)--- sandfly and culture

Causes of weight loss in with increases appetite and decrease appetite ?	
weight loss with increase appetite	weight loss with decrease appetite
DM	malignancy
thyrotoxicosis	TB,HIV
malabsorption syndrome	depression
phaechromocytoma	Anorexia nervosa
Kala-azar	Addison disease
worm infestation in children	CRF

What is the pattern of fever in kala-azar ?
any type of fever can occur in Kala-azar intermittent fever double quotidian fever is usually not so high –101 to 102 irregular pattern patient may hav apyrexia
what is the treatment of kala-azar according to national guide line

Causes of anaemia in Kala-azar ?
due to bone marrow infiltration hypersplenism Auto-immune haemolysis bleeding
jaundice in kala-azar ?
due to hepatic infiltration
edema/ ascites in kala-azar ?
due to hypoalbuminaenia
What is the incubation period in kala-azar ?
The incubation period of Kala-Azar 1-2 months

causes of huge splenomegaly ?
when spleen size is more than > 8 cm or spleen cross the umbilicus ? Kala-azar chronic Malaria Thalassaemia major Chronic myeloid leukaemia Ploycythemia rubra vera Cirrhosis with portal hypertension Myelofibrosis Storage disorder gauchers diseases

how many upazilla of Bangladesh is kala-azar affected ?
45 out of 64 zilla and 130 upazilla
what do mean by KAFT ? (kala-azar treatment failure)
a case that earlier diagnosed as KALa-azar and took complete treatment within one year reappearance of symptoms and sign of Kala-azar and any positive lab evidence of parasite from bone marrow / splenic aspiration RX usually alternative first line drug : ioposomal amphotericin B or second line drug : sodium stibogluconate
what type of drug amphotericin B is ? Mechanism of action
it is antifungal it bind with ergosterole of cellwall and make a pore and intracellular calcium and other ions come outs and cell death occur

Write down treatment of kala-Azar according to national guide line?
the drug uses as first line drug in Bangladesh is –meltifosine **1st line drug :** • Meltifosine alternative first line drug : • Paromomycin • Liposomal amphotericin B **second line drug** • Sodium stibogluconate • Amphotericin-B deoxycolate
when PkDL occur ?
it occur 6 month to 5 yr after in untreated and incompletely treated pt
what do u mean by cancrum oris ?
it is a form of sloughing ulcerative gingivitis which spread to buccal mucosa cheek , mandible Maxilla , resulting in widespread destruction of bone and soft tissue . it is due to invasion of tissue by bacteroids , fusebacterium and other normal commonsals of the mouths .

Leukaemia

Acute leukaemia or aplastic anaemia

PARTICULARS OF PATIENT:

Name: Fazlul Haque
Age: 30 years
Sex: Male
Marital status: unmarried
Occupation: school teacher
Religion: Islam
Address: Gouripur Mymensingh
Date of admission:
Date of examination:

PRESENTING COMPLAINTS

- Fever for 1 months
- Bleeding from gum for 7 days

HISTORY OF PRESENT ILLNESS

According to statement of the patient, he was reasonably well 1 months back then he gradually developed high grade fever which did not follow any specific pattern. The fever used to rise some times at the evening and some times at the morning ,lasted for about 10 to 12 hrs and it was not associated with chills and rigors but disappeared with sweating only after taking paracetamol (or spontaneously with out medication if patient told u).The highest recorded temperature was 104 F ^{o} . The Fever was not associated with cough, sputum, chest pain or breathlessness. The patient had not any history of drenching night sweating or itching (**exclude lymphoma**). 10 ago days patient notice bleeding from gum during brushing (or spontaneously)and two days later patient notice pain less , non itchy rash of variable size and shape which first appeared at face and arm (u will write the site what) then it involve the whole body more on the trunk .Initially these were red in color then as the days progress it turn into blackish color . The patient had no history of loose motion or bloody diarrhea , abdominal pain, and joint pain(**Henoch scheonlein** purpura) .The patient has no recent or past history of yellow coloration of sclera or any other parts of the body(**CLD/acute viral hepatitis**). The patient has no history of blood transfusion previously. He has no history coughing out of blood, bleeding per nose, vomiting out of blood, black tarry stool or alteration of bowel habit, headache, retro orbital pain (**dengue**) or vomiting. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. He also gave no history of increased frequency, urgency, hesitancy and red urine. The patient have no history of radiation and exposure insecticide or chemical (take this history if the case is **aplastic anemia**). The patient does not give history of significant weight loss. Patient also complained of loss of appetite and become anorexic in last one month. Gradually the patient became fatigue day by day. Initially he became fatigue in mild exertion. Now it begins to hamper his daily activity such going to bathroom and simple walking. With above complained he visited several times to local doctors every time he was treated with different types of antibiotic name of which he could not mentioned. Now he got admitted in to MU-1, MMCH for further evaluation and better management **(**if the patient is female then take HO hair loss and photo sensitivity and oral ulcer to **exclude SLE)**

HISTORY OF PAST ILLNESS

No history of DM/HTN/TB

No history of such type of illness before

(In case of aplastic anemia pt may admitted before for blood transfusion)

PERSONAL HISTORY

Smoker (10 Stick/day) for last 20 years. Non alcoholic. No history of extra-marital sexual exposure. No history IV drug abuse (to exclude infective endocarditis)

FAMILY HISTORY

- His wife and children are healthy.
- His other family members are also healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from middle class socioeconomic condition.

Housing: pacca house

Sanitation:1 sanitary latrine.

Water supply: Arsenic free tube well water

Immunization history

The patient was not immunized

Drug History

Patient received some oral and parental antibiotics (write only if patient give u this history).

Received

3 unit of blood last three days in hospital .

patient have no history Cytotoxic drugs Antirheumatic agents , Antithyroid and Immunosuppressives drugs (take this if ur provision diagnosis is aplastic anaemia)

GENERAL EXAMINATION

- **Appearance –ill looking and Toxic**
- Body built –Average
- Nutritional status – average
- Decubitus: on choice
- Co-operation : Well co-operative
- **Anaemia –severly anaemic (u may find –morderate anemia due to –blood transfusion)**
- **Jaundice : non icteric**
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : absent
- Dehydration – Absent
- Body hair distribution –normal
- **Bony tenderness – present**
- **Lymph node – No lymphadenopathy**
- hyroid gland – not palpable
- Neck vein – not engorged
- Pulse –110 /min, low volume, regular

If the pt is child the cause will b **ALL u have to describe the lymph node as follow :**
examination of lymph node of this patient reveals that patient have generalized lymph adenopathy involving cervical, right axillary s group and left inguinal group . There multiple, discrete, rubbery ,non tender lymph node of variable size and shape largest of them in cervical region is 2x 1 cm and in right axillary's region is 1.5x1 cm and left inguinal region is 2x 1.5 cm .these lymph node are not fixed with underlying structure or over lying skin and having no discharging sinus

- B.P – 110/80 mm of Hg
- Respiratory rate – 25/min
- Temperature –102^0F
- Weight : kg
- Height : meter
- **There is IV canula in situ**
- **Examination of Skin shows multiple painless ,not palpable purpuric spot and Echymosis of variable of size and shape not fade on pressure . these are present in the oral cavity , face and trunk and upper extremity**

SYSTEMIC EXAMINATION

Examination of Alimentary system

■**Mouth & pharynx**

Normal

■**Abdomen Proper**

■**Inspection**

■Abdomen – Abdomen is normal in size and shape l

■Umbilicus – centrally placed and innverted

■Visible vein – absent

■Movement with respiration – present

■Scar mark – no scar mark and no striae

■Visible peristalsis – absent

■Hernial orifice – intact

■Hair distribution – normal

■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,

■Liver is palpable which 2 cm from right costal margin in mid clavicular line and 3.cm from xephoid process having smooth surface , sharp margin , non tender ,soft in consistency , upper border of liver dullness is in right 5^{th} intercostals space with absent hepatic bruit and liver span is 14cm .

■ spleen is enlarged which is 3 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Finger insinuation is not possible

■Kidney and urinary bladder is not palpable

■There are no intra abdominal lymph adenopathy

■Fluid thrill absent

■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present

■Renal bruit absent

CARDIVASCULAR SYSTEM

- Pulse : 88 b/min
  Regular, normal in volume & character, all the peripheral pulses are normal.
- BP : 140/85 mm of Hg
- **JVP : Not raised**
- **Precordium** :
  Inspection: Normal
  Palpation: Apex beat in lt 5th intercostals space 9 cm from midline
  Auscultation of S1&S2 audible in all auscultatory area
  No added sound and no murmur

RESPIRATORY SYSTEM

- **Inspection** : Size and shape of the chest : Normal
- Movement is symmetrical
- No evidence of respiratory distress
- **Palpation** :
  Trachea: Trachea central
  Apex beat: in left 5th intercostals space 9 cm from midline normal in character
  Vocal fremitus: normal

Percussion: resonance

Auscultation:

Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.
Fundoscopic exam: Normal
Cranial nerves: intact
Motor system examination
Motor functions are normal in all four limbs
Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

Salient feature:

Mr.FAZlul haque , 30 yrs old normotensive, non diabetic, smoker and non alcoholic Muslim shopkeeper hailing from ……..mymesingh with the complaint of fever for 1 months, gum bleeding and rash for 7 days . The fever used to rise some times at the evening and some times at the morning ,lasted for about 10 to 12 hrs and it was not associated with chills and rigors but disappeared with sweating only after taking paracetamol (or spontaneously with out medication if patient told u).The highest recorded temperature was 104 F º . The Fever was not associated with cough, sputum, chest pain or breathlessness. The patient had no drenching night sweating or itching (**exclude lymphoma**). 10 ago days patient notice bleeding from gum during

brushing (or spontaneously) and two days later patient noticed multiple pain less , non itchy purpuric spot of variable size and shape which first appeared at face and arm (u will write the site what) then it involve the whole body more on the trunk .Initially these were red in color then progressively it turn into blackish color . The patient has no history of arthritis, bloody diarrhea, and abdominal pain and jaundice **.**The patient has no history of blood transfusion previously. He has no history Haemoptysis ,Haematemasis ,Maleana, Epistaxis ,alteration of bowel habit, headache, retro orbital pain (**dengue**) or vomiting. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. He had not any history of increased frequency, urgency, hesitancy and red urine. The patient have no history of radiation and exposure insecticide or chemical (take this history if the case is **aplastic anemia**). The patient does not give history of significant weight loss. But the Patient complained of loss of appetite and become anorexic in last one month. Gradually the patient became fatigue day by day. Initially he became fatigue on mild exertion. Now it begins to hamper his daily activity such going to bathroom and simple walking. General examination reveal that the patient is toxic , ill looking ,moderately anaemic , non icteric and presence of boney tenderness The patients temp is 101^0F, pulse is regular 110 beat / minute with normal blood pressure (110/80 mm of Hg) respiratory rate is 18 / min and pattern is normal . Examination of Skin shows multiple painless ,not palpable purpuric spot and Echymosis of variable of size and shape which not fade on pressure . The patient has no clubbing, splinter hemorrhage, hypo or hyper pigmentation . Examination of elementary system reveals that abdomen is normal in size and shape, flanks are normal , umbilicus is in centre inverted, no muscle guard or rigidity or tenderness , shifting dullness and fluid thrill are absent . Organ palpation reveal hepato-splenomegaly . Liver is enlarge which 3 cm from right costal margin in mid clavicular line and 4.cm from xephoid process having smooth surface , sharp margin , non tender ,firm in consistency , upper border of liver dullness is in right 5^{th} intercostals space with absent hepatic bruit and liver span is 14 cm . Spleen is enlarged which is 4 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Testis is normal in size and consistency. Examination of rest of the system reveals no abnormality

PROVISIONAL DIAGNOSIS:

In this patient

Acute leukemia

Differential diagnosis

If the patient has no boney tenderness and no organomegaly
Then ur provisional diagnosis is --- **Aplastic anemia**

Aplastic anemia
Lymphoma (if lymphnode present)
Kala-azar (if hepatosplenomegaly present)

Investigation

Complete blood count

Hb%
TC----increased
ESR –increased

PBF------Anemia ---

WBC ---increased blast cell
Platelete – thrombocytopenia

Bone marrow examination

To see hyper celularity with alter myloid erythoid ratio and increased blast cell (>20%)

cytogenetics and immunological phenotyping

USG and CXR

Treatment:
Treatment of acute leukemia is Supportive and specific
Supportive treatment:
- Maintain nutrition and hydration
- Correction of anaemia -----With fresh blood
- Correction of thrombocytopenia----With platelet transfusion
- Control of infection--- Empirical therapy with broad spectrum antibiotics
- Hyperuracemia----allopurinol

Specific therapy: **Chemotherapy sees --- later**
Definite treatment: **Bone marrow tranplantaion**

What is ur provisional diagnosis? What are points in favor of ur diagnosis?

My provisional diagnosis is acute leukemia
Point in favor your diagnosis
- Fever
- Anaemia
- Gum bleeding
- Patient is toxic
- Boney tenderness present
- Hepato-splenomegaly present

What type of leukaemia is it? Why not ALL?

It is acute myeloblastic leukaemia
Because
ACUTE – Due to short history
Toxicity
Presence of boney tenderness

AML---due to age
Age ---AML is more common in adult
ALL
ALL is more common in child and
ALL may have lymphadenopathy

What are ur differential diagnosis?
My differential diagnosis are
- Lymphoma
- Aplastic anemia
- Kala-azar

Lymphoma

Point in favor	**Point against**
Fever	No lymphadenopathy
Hepatospleno megaly	Presence of Boney tenderness
Anemia	Absent of Drenching night sweating and itching

Aplastic anemia

Point in favor	Point against
Fever Anemia Bleeding menifestation	Hepatospleno megaly Boney tender ness

Kala-azar

Point in favor	Point against
Fever Anemia Bleeding menifestation Hepatosplenomegaly	Presence of Boney tender ness Not coming from endemic ZONE Toxicity of patient

Why it is not enteric fever?
It is unlikely to be enteric fever as

Point in favor	Point against
High grade fever Hepato-splenomegaly	More than one month fever usually not enteric Presence of boney tenderness As the pt is toxic Bleeding manifestation absent in enteric fever

What do u mean by sub-leukamic leukemia and Leukemic leukemia ?

Subleukaemic leukNr. Or subnormal WBCs count with predominant blasts.
A leukaemic leuk----- normal WBCs with no blast in B.M

How will u differentiate between aplastic anaemia / leukemia / ITP?

	Acute leukemia	Aplastic anemia	ITP
Clinical feature	Fever –high grade Toxic bleeding Present	Only fever and Bleeding manifestation Nontoxic	Only bleeding manifestation Non fever or non toxic
Boney tenderness	Present	Absent	Absent
Hepato-splenomegaly	Present	Absent	Absent
PBF	Leukocytosis with Blast cell	Pancytopenia	Only thrombocytopenia
Bone marrow	Hyper cellularity with increased blast cell	Hypocellur marrow or dry tap	Increased megakaryocyte

Classify the leukemia?
Classification (According to the cell origin and the rapidity of the course).

- **Acute leukemia**
  - Acute lymphoblastic leukemia
  - Acute myeloblastic leukemia
- **Chronic leukemia**
  - Chronic lymphoblastic leukemia
  - Chronic myeloblastic leukemia

Name a single investigation in to Dx leukaemia ?
PBF

Acute : Rapid, clinical course resulting in death within months without effective ttt, this is due to early B.M failure

Chronic: A more prolonged natural history, this is due to late B.M failure

What are the clinical presentation of acute leukemia?

Infection : fever / sore throat
Anemia
Bleeding manifestation
Lymphadenopathy
Organomegaly ---hepatosplenomegaly
Arthritis

How will u differ from AML from ALL

	ALL	**AML**
Age	**Child (**1-5 age)	**Adult**
Lymphadenopathy	**Present**	**Usually absent**
PBF---type of blast cell	**Lymphoblast**	**Myeloblast**
Bone marrow	**Lymphoblast**	**Myeloblast**
Auer rods in the cytoplasm of blast cells	**Absent**	**Present**

Who can u recognize AML in PBF or differentiate from AML from ALL in PBF?

By presence of Auer rods in the cytoplasm of blast cells. which present in AML

What history u have to take in patient with leukaemia ?

- HO of radiation---- radiotherapy , repeated X—ray
- HO cytotoxic drug
- Exposure **t**o benzene
- Genetic –family history
- Immunosupression
- Some viral infection

What do u mean by neutropenic fever?
Fever (> 38°C) lasting over 1 hour in a neutropenic patient (absolute neutrophil count < 1.0×10^9/l) indicates possible septicaemia.
Rx : Aminoglycoside (e.g. gentamicin) and a broad-spectrum penicillin (e.g. piperacillin/tazobactam).but we use ceftriaxone 2 gm IV BD
How long Continue treatment
At least 3 days after fever subside

Write the treatment o acute leukemia?

Treat of acute leukaemia is
Supportive and specific

Supportive treatment:

- Maintain nutrition and hydration
- Correction of anaemia
  With fresh blood
  Target haemoglobin is10gm/dl
- Correction of thrombocytopenia
  With platelet transfusion
- Control of infection
  Empirical therapy with broad spectrum antibiotics

Anti-fungal
Anti-viral if herpes simplex
Prophylaction drugs against --*Pneumocystis carinii (with co-trimoxazole)*

- Hyperuracemia
  allopurinol

Specific treatment is chemotherapy:

ALL

- **Phase of in induction**
  VAP @ DM
  - **V--**Vincristine (i.v.)
  - A--L-asparaginase (i.m.)
  - P-Prednisolone (oral)
  - D-Daunorubicin (i.v.)
  - M-Methotrexate (intrathecal)
- **Phase of Consolidation**
  CDE-M
  - C--Cytarabine (i.v.)
  - D--Daunorubicin (i.v.)
  - E--Etoposide (i.v.)
  - M--Methotrexate (i.v.)
- **Phase of Maintenance**
  PVM-2
  - P--Prednisolone (oral)
  - V--Vincristine (i.v.)
  - M--Mercaptopurine (oral)
  - M--Methotrexate (oral

What r the Signs of remission?
Signs of remission are
- Improvement of C/P
- B.M blasts below 5%
- No blast in peripheral blood

AML

- **Induction phase** :
  CDE
  - C--Cytarabine (i.v.)
  - D--Daunorubicin (i.v.)
  - E---Etoposide (i.v. and oral)

- **Consoilidation Phase**
  CAM
  - C--Cytarabine (i.v.)
  - A---Amsacrine (i.v.)
  - M--- Mitoxantrone (i.v.)

What is the definitive treatment of acute leukemia ?
Allogenic bone marrow transplantation from HLA match person

Where lymphnode and hepatosplenomegaly found ?

Liver, Spleen ++, L.N. common with lymphoblastic leuk,

Which one have better prognosis ?

Acute lymphoblastic leuk have better prognosis than acute myeloblastic leukemia

Time of survivable with out treatment in acute leukaemia ?

Without treatment the median survival is about 5 weeks

What are the poor prognostic factors of acute lymphoblastic leuk ?

Poor of acute lymphoblastic leuk

* Age < 2 yrs > 10
* TLC > 1,00,000
* Plat < 25,000
* L3 – CNS infiltration

What will u do if patient developed meningeal leukaemia ?

In case of Meningeal leukaemia treatment is

- Cranial irradiation.
- Intrathecal methotrexate.

What will u differentiate ALL and AML by staining or cytochemically ?
Or how will u differentiate by staining ALL and AML ?

By staining blood or marrow material with myeloperoxidase or Sudan black stain we can differentiate AML from ALL . Both positive in case of AML .

Name some indication of bone marrow transplantation ?

General indications for allogeneic bone marrow transplantation

- Leukaemias
- Aplastic anaemia
- Thalassaemia,
- Inborn errors of metabolism with missing enzymes or cell lines

Complication of bone marrow transplantation ?

Complication of bone marrow transplantation are :

- Mucositis
- Infection
- Bleeding
- Pneumonitis
- Chronic graft-versus-host disease
- Acute graft-versus-host disease
- Secondary malignant disease

What r the newer treatment In leukemia ?

Recent trends in Rx of leuk

- Monocional Abs to the leukemia blasts.
- BCG
- Interleuk in 2
- Activated natural killer cells
- B.M. transplantation

If the case is Aplastic anemia : u have to learn the following :

Aplastic anemia

Pt present with severe anemia and infection in late case bleeding manifestation

PBF –Normocytic with pancytopenia (anemia, neutropenia, thrombocytopenia) 7

No Organomegaly or bony tenderness

Bone marrow dry trap or hypo plastic marrow

What is the causes in aplastic anemia ?

Primary causes or idiopathic

What HO u have to take to find out secondary cause?

Recent drug

- Viral hepatitis –HBV, HCV
- Pregnancy
- Radiation
- Insecticide ---DDT, OPC , Carbamate
- Fanconi anemia

To increase Cell count what we give in the ward ?
We give granulocyte stimulating factors
Inj . filastin
I amp S/C

Rx

Supportive Rx :

For infection-----

- **Antibiotic** ---usually broad spectrum, if fever then gives Inj. Ceftron 1 gm BD

For anemia----

- **Blood transfusion** to keep it 10 gm/dl

Specific treatment

If the patient age < 30 years Allogenic bone marrow transplantation

If not then

Immunesupressor therapy –cyclosporine + antithymocyte globulin

Prognosis is poor 50 % will die.

OLD age

Only supportive therapy and follow up (monthly CBC and PBF)

Immunesupressor therapy –cyclosporine + antithymocyte globulin

If multiple releaps think for MDS and even AML

In MBBS final examination u will get a patient with fever & bleeding spot.

If boney tenderness presents:

- Then provisional diagnosis –acute leukaemia
- DD is------Aplastic anemia

If boney tenderness is absent:

- Then provisional diagnosis –Aplastic anemia
- DD is -----acute leukaemia

Fever

Duration of	
High grade or low grade	
Chills and rigor travel to hilly area	
Subsides with sweating with sweating	
other history of fever	Is it associated with cough, sputum, chest pain or breathlessness.
Exclude lymphoma.	History of drenching night sweating or itching

History of bleeding manifestation

site of bleeding	from gum during brushing (or spontaneously)a
	• Coughing out of blood, • Bleeding per nose, • Vomiting out of blood, • Black tarry stool or alteration of bowel habit, • Associated with headache, retro orbital pain (**dengue**)
history about purpura	• Patient notice pain less , non itchy rash of variable size and shape • Which first appeared at face and arm (u will write the site what) then it involve the whole body more on the trunk • Initially these were red in color then as the days progress it turn into blackish color
Henoch scheonlein purpura	• history of loose motion or bloody diarrhea , abdominal pain, and joint pain
Recent or past history of jaundice	• CLD • acute viral hepatitis –secondary causes of aplastic anemia
HO of blood transfusion	• previous • also after in admission in hospital
2ndary causes of aplastic anaemia	• history of radiation and exposure insecticide or chemical
others	• weight loss ,loss of appetite, fatigability, anorexic
joint pain and rash , hair loss , photosensitivity in female pt	• SLE

What do you mean by **aleukaemic leukaemia ? sub lukaemic leukaemia? leukemoid reaction** ?

sub lukaemic leukaemia:

A form of leukaemia in which abnormal cells (blast cell)are present in the peripheral blood, but the total leukocyte count is not elevated.

aleukaemic leukaemia:

a form of Leukaemia in which abnormal (blast) cells are absent in the peripheral blood. But bone marrow show blast cell.

in both case diagnosis is done by bone marrow aspiration

leukemoid reaction

The term leukemoid reaction describes an elevated white blood cell count, or leukocytosis, that is a physiological response to stress or infection

What is blood picture in different type leukemia ?

	ALL	AML	CML	CLL
age	1-5	> 50 yr	30---80 average 55	65 to 70
sex ratio m:f	3:1	3:1	1. 3: 1	2:1
TC	1---5oo X 10^9 or 1ooo to 500000	1---5oo X 10^9 or 1ooo to 500000	10—600 X 10^9 or 1000 to 600000	
DC	lymphoblast	myeloblast	protmyelocytes metamyelocyte meyloblast	lymphocyte > 5 X 10^9 or

What is leuko-erythroblastic blast picture?

Immature RBC and WBC in blood cell but bone marrow normal

causes

1. infection
2. post haemolysis / haemorrhage
3. marrow infiltration ---by carcinoma
4. myelofibrosis

causes of anaemia , jaundice , in lymphoma ?

anaemia	**jaundice**
ineffective erythropoiesis hypersplenism haemolysis	hepatic involvement obstruction of billiary duct at porta hepatis haemolysis

What is the fever pattern in lymphoma?

different types of fever :
low grade
high garde
pel-ebstin fever
irregular fever

composition of waldyer ring ?

adenoid
faucal tonsil
lingual tonsil

What is vircows node? What is troisers sign ?
Can lymphoma occur without lymphadenopathy ?
yes abdominal lymphoma
Causes of night sweat?
TB lymphoma CLL myelofibrosis AIDS
Is bone marrow needed for diagnosis of leukaemia?
not necessary to diagnosis leukaemia need for treatment and prognosis

What is the difference between hereditary and congenital disease?		
	hereditary/ familiar	congenital
Genes	mutant genes present	not produce by mutant genes
Mendelian law	follow the mendelian law transmit of one generation to another generation	not follow the mendelian law and not transmite from one generation to another
Onset	may not present since birth	always present since birth
Example	DM , thalassamia ,	congenital heart disease , cleft lip
Causes of cardiomegaly or heart failure in thalassamia ?		
Causes abdominal pain?		
Biliary colic due to pigment stone in gall bladder Dragging pain due to splenomegaly (if sir ask think pt have no stone)		
difference between hemosederosis and haemochromatosis		
Feature	haemosiderosis	heamochromatosis
inheritance	always acquired	genetic or acquired
tissue affected	Reticulo-endothelial tissue	paranchymal cell (liver , pancrease)
damage	less tissue damage	more tissue damage
after giving desfrrioxamine	urinary excretion of iron doesn't excrete more than 4 mg/ 24	exceed more than 4 mg / 24 hr

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New therapy in Thalassamia?
Inj.Erythropoietin—it stimulate bone marrow **Hydroxyurea---** it prevent in effective erythopoiesis
Target Hb level in thalassamia ? serum feretin level ?

Indication of splenectomy in thalassamia ?

- Requirement of excessive transfusion to maintain Hb level > 10 gm / dl (more than I unit / month)
- Feature of hypersplenism
- Massive splenomegaly causing mechanical problem (dragging pain / early satiety)

Name some causes of refractory anaemia ?

- Aplastic anaemia
- Sideroblastic anaemia (Rx—pyridoxine)
- Myelodysplastic syndrome
- chronic renal failure

what do u mean by super transfusion and hyper transfusion ?

- Super transfusion : Hb level is kept > 10 gm / dl
- Hyper transfusion : Hb level is kept > 12 gm / dl

What are the advantages of hyper transfusion?

- GIT iron absorption decreased
- less chance of facial disfigurement
- less chance of hypersplenism
- less need for early splenectomy
- growth and development are increased

A patient admitted in your hospital for repeated blood transfusion what may the possibility?

- Thalassamia
- Aplastic anaemia
- ITP(immune thrombocytopenic purpura)
- Haemophilia
- CRF
- Recurrent haematemesis and melaena

Two other test can be done or

- Alkali denaturation test : ---test positive (HbF is resistance to alkali denaturation)
- Osmatic fragility test : it is decrease – that mean there is increased resistance to red cell to osmotic lysis (osmotic fragility test is increased in hereditary spherocytosis)

Complication of repeated blood transfusion?

risk of iron over load / haemochromatosis
transfusion hazard – hepatitis B C , AIDs
chance of isoimmunization
volume overload ---heart failure

Name some organism transmit by blood donation.

malaria , hepatitis B , C , HIV , syphilis , toxoplasmosis ,

Causes of target cell in blood.

iron deficiency anaemia , thalassamia , after splenectomy , cholestatic jaundice

Liver Abscess

PARTICULARS OF PATIENT:

Name: Md. Asraf
Age: 40yrs
Sex: Male
Marital status: Married
Occupation: Farmer
Religion: Islam
Address: Tishal , Mymensingh
Date of admission: 8.12.09 at 7pm
Date of examination: 10.12.09 at 7.15am

PRESENTING COMPLAINTS

- Fever for 25 days
- Pain in the right upper abdomen for 25 days
- (Yellow coloration eye for last 15 days (may be absent))

HISTORY OF PRESENT ILLNESS

According to statement of the patient, he was reasonably well 25 days back then he developed high grade fever which persisted most of the time of the day and was associated with chills and rigors and disappeared with sweating after taking Paracetamol(or spontaneously with out medication). The highest recorded temperature was 104 F $^{o.}$ The patient also complained of mild to moderate pain in right upper abdomen and also in right lower chest which was sharp in nature, had no radiation and increased by deep breathing and coughing and relieved after taking medication. Fever was not associated cough, sputum, breathlessness, night sweating and itching. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. The patient gave history loose motion one month ago. There was no history of bloody diarrhea and alteration of bowel habit, joint pain and rash, headache or vomiting. He gave no history of increased frequency, urgency, hesitancy and red urine. But the patient is anorexic and no history of significant weight loss . The patient had no history of previous severe abdominal pain (appendicitis / cholelithiasis), abdominal surgery, abdominal trauma and jaundice (gall stone). The patient had no history breathlessness in exertion or rest or in lying posture and chronic cough and swelling of the leg (congestive cardiac failure) .With above complained he visited several times to local doctors every time he was treated with different types of antibiotic name of which he could not mentioned. Now he got admitted in to MU-1, MMCH for further evaluation and better management . He also gave history aspiration of pus from his right upper abdomen after admission in this hospital and color of the pus was chocolate in color (anchovy sauce).

If patient have jaundice the following history should be taken

The patient also notice yellow coloration of skin and eye for the last 15 days which is progressively increasing .but patient had no history headache, malaise, nausea and vomiting , itching . The color of stool was not pale. The patient had not the history of loss of body hair , vomiting out of blood and black tarry tool, Alter level of consciousness and alteration of sleep pattern. No blood transfusion and always use disposable blade for shaving

History of past illness

No previous history of jaundice, gall stone or liver disease. He had no history hypertension and Diabetes.

Drug history

The patient has no significant drug history

PERSONAL HISTORY

The patient is smoker he smokes 5-8 sticks per day, Non alcoholic and has no history IV drug user. The patient had no history multiple extra marital sexual exposures.

FAMILY HISTORY

None his family member is suffering from jaundice.
They are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition and lived in crowding house
Housing: Tin shade house.
2 rooms, which accommodate 8 0f
His family member.
Sanitation: 1 sanitary latrine.
Water supply: Arsenic free tube well water but sometimes he used to drink Tap water when he remained out side the house.

Immunization history

The patient was not immunized against hepatitis B

GENERAL EXAMINATION

- Appearance –ill looking
- Body built – Average
- Nutritional status –Average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – Absent
- Jaundice : absent (mild)
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leukonychia : Absent
- Dupuytren's contracture : Absent
- palmar erythema: Absent
- hepatic flap / flapping tremor : absent
- Spider nevi : Absent
- Gynaecomastia : Absent
- Oedema : Absent
- Dehydration – Nill
- Skin –general skin condition and is normal
- Body hair distribution –normal
- Bony tenderness – absent

- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 130/80 mm of Hg
- Respiratory rate – 25/min
- Temperature – 101^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :

Canula in situ:

Systemic examination

Examination of Alimentary system

■**Mouth & pharynx**

Normal

■**Abdomen Proper**

■**Inspection**

■Abdomen –swelling in right hypochrondium and fullness of right inter costal space

■ Movement with respiration – restricted movement of right upper abdomen present

■flank not full

■Umbilicus – centrally placed and inverted

■Visible vein – absent

■Scar mark – no scar mark and no striae

■Visible peristalsis – absent

■Hernial orifice – intact

■Hair distribution – normal

■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, tenderness present in right hypochrondium ,

■Liver is palpable which 3 cm from right costal margin in mid clavicular line which tender having smooth surface , sharp margin (rounded), soft in consistency , upper border of liver dullness is in right 5^{th} intercostals space with absent hepatic bruit and liver span is 15 cm .

■ spleen ,Kidney and urinary bladder is not palpable

■No intra abdominal lymph adenopathy

■Fluid thrill absent

■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present

■Renal bruit absent

CARDIVASCULAR SYSTEM

•Pulse : 88 b/min

Regular, normal in volume & character, all the peripheral pulses are normal.

•BP : 140/85 mm of Hg

•**JVP : Not raised**

•**Precordium** :

Inspection: Normal

Palpation: Apex beat in lt 5th intercostals space 9 cm from midline

Auscultation of S1&S2 audible in all auscultatory area

No added sound and no murmur

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Movement is symmetrical

•No evidence of respiratory distress

•**Palpation** :

Trachea: Trachea central

Apex beat: in left 5th intercostals space 9 cm from midline normal in character

Vocal fremitus: normal

Percussion: resonance

Auscultation:

Breath sound is vesicular in all parts of the chest.

No added sound

Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.

Fundoscopic exam: Normal

Cranial nerves: intact

Motor system examination

Motor functions are normal in all four limbs

Sensory examination

All modalities of sensation are intact in both upper and lower limbs

Cerebellar signs: Absent

Signs of meningeal irritation: Absent

SALIENT FEATURE:

Md. Asraf 40 yrs old married muslim smoker normotensive nondiabetic farmer hailing from Tishal Mymensingh and got admitted into MU-1 MMCH with high grade intermittent fever and mild to moderate pain in the right hypochrondium for 25 days .the fever persisted most of the time of the day and was associated with chills and rigors and subside with sweating after taking Paracetamol(or spontaneously with out medication). The highest recorded temperature was 104 F o. The pain was sharp in nature, had no radiation and increased by deep breathing and coughing and relieved after taking medication. Fever was not associated cough, sputum, breathlessness, night sweating and itching. .He had no history of contact with known TB patient or traveling into hilly or border area or abroad. The patient had history of loose motion one month ago but no history bloody diarrhea and alteration of bowel habit, joint pain and rash, headache or vomiting. He gave no

history of increased frequency, urgency, hesitancy and red urine. But the patient is anorexic but not toxic and no history of significant weight loss. The patient had no previous history of appendicitis / cholelithiasis , abdominal surgery, abdominal trauma and jaundice (gall stone). The patient had no history exertional dyspnea, orthopnea, COPD. with these complained he visited several times to local doctors every time he was treated with different types of antibiotic .He also gave history aspiration of pus from his liver after admission in this hospital and color of the pus was chocolate in color .General examination reveals the patient is well oriented, non toxic ill looking, co-operative, body built is average, temperature is raised and mildly anaemic(but u may got anemia absent) . The patient is non icteric(may be icteric) , pulse is regular 88 beat / minute with normal blood pressure (140/85 mm of Hg), respiratory rate is 18 / min and pattern is normal . The patient is non-edematous, boney tenderness absent; the patient has no lymph-adenopathy, clubbing, splinter hemorrhage, hypo or hyper pigmentation. Systemic examination revealed only tender hepatomegaly (Liver is enlarge which 3 cm from right costal margin in mid clavicular line ,tender having smooth surface , sharp margin , soft in consistency , upper border of liver dullness is in right 5^{th} intercostals space with absent hepatic bruit and liver span is 15 cm. Spleen ,Kidney and urinary bladder is not palpable). No intra abdominal lymph adenopathy, Shifting dullness and Fluid thrill absent .Testes are normal is size and consistency, no engorged vein. Respiratory system examination reveals inter costal fullness and tenderness at right lower chest. Other systems reveal no abnormality .

PROVISIONAL DIAGNOSIS

Amoebic Liver abscess (or if have enough point in favor of ur diagnosis u may write as amoebic liver abscess)

DIFFERENTIAL DIAGNOSIS

Viral hepatitis

- Acute cholecystitis

Investigation :

CBC : neutrophilic leucocytosis
TC – 1800/mm3
DC –
neutrophil-84%
Lymphocyte-10%
monocyte-02%
Eosinophil-04%
ESR-55 mm in 1st hr Hb-09g/dl**.**

USG of hepato billiary system : shows liver abscess

CXR –raised of hemi diaphragm and complication such as pleural effusion
LFT---S.Bilirubin –according to presence jaundice
Alk phosphates –may increased
Serum albumin ---is often low
USGuided aspiration of pus and send for culture and sensitivity

For amoebic liver abscess:

Immunofluorescent antibody test (95% ---positive)
Indirected haemoaglutination test (positive in –95 %)
ELISA

Treatment

Incase of amoebic **liver abscess:**

- Metronidazole (800 mg 8-hourly for 5 days) or
- Tinidazole (2 g daily for 3 days)

In case of pyogenic abscess:

- Amoxicillin
- Metronidazole and
- Gentamycin

Cause of fever with chill @ rigor
Common

- Malaria
- UTI (pyelonephritis)
- Cholangitis

Other cause
Any abscess
Pneumonia

What is ur provisional diagnosis?

My provision diagnosis is ameobic liver abscess

What are the points in favor of ur diagnosis?

HO of lose of motion 1 month ago.
Fever for 25 days
Upper abdominal pain for 25 days
(Jaundice if present)
On examination
Tender hepatomegaly
Intercostals fullness and tenderness on percussion over the right lower chest
HO aspiration of pus from liver which color is anchovy sauce.

Why have u called it amoebic rather than pyogenic liver abscess?

Patient is not toxic
Fever has not any chill and rigor.
Ho of diarrhea one month ago
Pus is anchovy sauce in aspiration
No history of billiary obstruction or portal pyaemia

What is the different between pyogenic and amoebic liver abscess ?

In pyogenic liver abscess	**Amoebic liver abscess**
Fever is high and with chill and rigor	Fever is mild to moderate usually not with chill @rigor
Patient is toxic	Not toxic
No history of diarrhea ,Ho Cholangitis ,septicaemia may present	History of diarrhea/ amoebiasis may present
USG of shows multiple abscess	Single Fever is mild to moderate usually not with chill @rigor
Aspiration of pus reveals –frank pus	Anchovy sauce / chocolate brown color
Organism –E.coli	E.histolytica
Prognosis Is fatal	Prognosis is less fatal
Neutrophilic leucocytosis	Absent

What are the differential diagnosis?

Acute viral hepatitis

if sir want to know what will be other differential diagnosis . Then say the following
Acute Cholecystitis
Right lower lobe pneumonia

What are the point in favor and disfavor of ur diagnosis?

Point in favor	**Point in against**
Jaundice (if it present in ur case) Fever Abdominal pain Tender hepatomegaly	No prodorme such as nausea, vomiting , malaise High fever (in hepatitis fever is mild) Absence of jaundice / mild jaundice

Why this is a not case of acute cholecystitis

Point in favor	**Point in against**
Fever Abdominal pain	Murphy sign absent Tender hepatomegaly

What are the causes of tender hepatomegly
Acute viral hepatitis
Hepatocellular carcinoma
Liver abscess
Congestive cardiac failure

Why this is a not case of Hepatocellular carcinoma

Point in favor	**Point in against**
Only tender hepatomegaly HCC some time associated with PUO	Consistency is soft (in HCC irregular firm to to hard) No stigmata of CLD No hepatic bruit Fever

Why this is a not case of CCF

Point in favor	**Point in against**
Only tender hepatomegaly Consistency is soft	Fever No history of cough and dyspnea JVP not raised and no depended edema

Why have u not think it as enteric fever or kala-azar?
Enteric fever
Prolong Fever with abdominal pain is one of the cause is enteric fever but in enteric fever splenomegaly is more common then hepatomegaly alone. In enteric fever liver is non tender and here other features of enteric fever are absent (relative Bradycardia, coated tongue, rash, constipation)
Kala-azar;
Though patient comes from endemic Zone but like enteric fever splenomegaly is more common and appear first and before hepatomegaly. And lever is non tender in Kala-azar .

If u ask Only one cause pyogenic abscess ? why ?
- Biliary obstruction
- As bile is good media for bacterial growth

What are the causes of pyogenic liver abscess?

TO remember it –**PTB তে DIC হয়**
P—portal pyaemia
(PIA—perforation , intra. abdominal abscess , appendicitis)
T—trauma
B—ascending cholangitis due to Biliarry obstruction by (SSC—stone , stricture , carcinoma)

D—direct extension from peripheral abscess (sub diaphramgtic)
I—idiopathic
C—septicaemia

What are the organisms responsible for pyogenic liver abscess @ amoebic liver abscess?

pyogenic liver abscess
- *E. coli*
- streptococci. *milleri*,
- anaerobes, including streptococcus faecalis and *Bacteroides*,

Amoebic liver abscess
- E.histolyctia

Common site of liver abscess ?
- Post . surface of right lobe . as it is bare area of liver

What are the indication of aspiration liver abscess::

1. more than 5 cm
2. empending to rupture
3. if in the left lobe (chanc of pericardial effusion)
4. failure to respond medical therapy

What investigation u want to do ?

CBC : neutrophilic leucocytosis in case of pyogenic liver abscess
TC – 1800/mm3
DC –
neutrophil-84%
Lymphocyte-10%
monocyte-02%
Eosinophil-04%
ESR-55 mm in 1st hr Hb-09g/dl**.**

USG of hepato billiary system : shows liver abscess

CXR –raised of hemi diaphragm and complication such as pleural effusion
LFT---S.Bilirubin –according to presence jaundice
Alk phosphates –may increased
SGPT ---normal
Serum albumin ---is often low
US Guided aspiration of pus and send for culture and sensitivity
CT-scan of abdomen

For amoebic liver abscess:

Immunofluorescent antibody test (95% ---positive)
Indirected haemoaglutination test (positive in –95 %)
ELISA

Which liver abscess is more common? Which is more dangerous?

Amoebic liver abscess and pyogenic is more dangerous.

What history u should take in case of amoebic liver abscess?

HO diarrhea (10% case associated with amoebic abscess)

What are the complication liver abscess?

Rupture in peritoneal cavity
Rupture in the lung and cause pleural effusion and empyema thorasis
Rupture in to pericardium and cause pericardial effusion
Septicemia
Metastatic abscess
Broncho pleural fistula.

Jaundice is more common in where ?

In pyogenic liver abscess

What is the color of pus in liver abscess ?

In pyogenic ----yellow color and foul smelling
In amoebic liver abscess ---anchovy sauce

What do u mean by lax sign?

If u purcus over the Right lower chest and u will get tenderness in case of liver abscess . it is called lax sign .

In which position they lie?

Left lateral position patient lie and because in that position intercostals space widen and pt feel comfort.

Common site of abscess?

Apex and anterior aspect of Right lobe

Mechanism of amoebic liver abscess?

- It is occur due to ischaemic necrosis .

We eat it as cyst --After ingestion –enter into colon and form flask shape ulcer in caecum and enter in to portal circulation and goes to liver sinusoids and block portal circulation and ischaemic necrosis of hepatocyte and formation of abscess .

Why color in amoebic liver abscess is anchovy sauce?

- As pus mixed with blood

Treatment of liver abscess?

In amoebic liver abscess oral form

Tab . metronidazole 800 mg 8 hrly for 5 days or
Tab . Tinidazole (2 g daily for 3 days)

In case of pyogenic liver abscess ?
AGM

A—inj.amoxycillin
M—inj metronidazole
G—inj. Gentamycin
This are injectable form for 2 wk and then oral if necessary

What treatment u may give to eradicate cyst ?
To luminal cyst eradicate
Tab. Diloxanide 800 mg 1+1+1 for 10 days
To tissue cyst
Tab. Chloroquine 300 mg 1 +0 1 for 2 days then
150 mg 1+0+1 for 14 days

What are the bad prognostic criteria?

BAD prognostic criteria to remember it JAAL
J-- Jaundice bilirubin > 3.5
A--Hypoalbuminia < 2 g/ dl
A --Ascites
L--Liver abscess >10 cm

A patient with liver abscess comes to u cough out of pus what is cause?

- The liver abscess may be ruptured into the lung and made broncho pleural fistula.

Is it good or bad for the patient?

- Good for the patient as auto drainage done

A patient with liver abscess come to u on examination u got absent breath sound and stoney dull on percussion then what is ur diagnosis ?

- My diagnosis is liver abscess burst in pleural cavity

Provisional diagnosis is

- Liver abscess

Differential diagnosis is

- Acute viral hepatitis
- Acute cholecystitis

You have to take history to differentiate it from pyogenic liver abscess to amoebic liver abscess

Following history you have to take in pt with suspected liver abscess

Fever

Duration of	
High grade or low grade	high—pyogenic liver abscess , low grad—amoebic
Chills and rigor travel to hilly area	chill & rigor—pyogenic liver abscess malaria
Rise with shivering or subsides with sweating	DO
right lower Chest pain or upper abdominal pain	• Sharp in nature, radiation present or not . • Pain Increased by deep breathing and coughing . • Pain Relieved after taking medication.
other history regarding fever	• contact with known TB patient • history of bloody diarrhea and alteration of bowel habit, joint pain and rash, headache or vomiting • urinary problem,
History loose motion	• Amoebic liver abscess
Previous severe abdominal pain (appendicitis / cholelithiasis),	
Abdominal surgery, abdominal trauma and jaundice (gall stone)	
congestive cardiac failure / CCF as patient have tender hepatomegaly	• history breathlessness in exertion or rest or in lying posture and chronic cough and swelling of the leg
	• History of anorexic and weight loss .
• History aspiration of pus from his right upper abdomen and color of pus	

Lymphoma

PARTICULARS OF PATIENT:

Name: salam
Age: 27 years
Sex: Male
Marital status: married
Occupation: shope keeper
Religion: Islam
Address: Gouripur Mymensingh
Date of admission:
Date of examination:

PRESENTING COMPLAINTS

- Fever for 3 months
- Mass (or lump) in left upper abdomen for 2 months

HISTORY OF PRESENT ILLNESS

According to statement of the patient, he was reasonably well 3 months back then he developed high grade fever which did not follow any specific pattern. The fever used to rise some times at the evening and some times at the morning ,lasted for about six to eight hrs, most of the time which was associated with chills and rigors but disappeared with sweating after taking paracetamol (or spontaneously with out medication).The highest recorded temperature was 103 F º. Fever was not associated cough, sputum, chest pain or breathlessness. The patient had drenching night sweating but no itching. The patient has no recent or past history of yellow coloration of sclera or any other parts of the body. The patient has no history of blood transfusion previously. The patient also noticed a pain less mass in the left upper abdomen for last 1 month. The lump was gradually increasing in size toward the umbilicus day by day. The patient has no history coughing out of blood, bleeding per nose, vomiting out of blood, black tarry stool. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. There was no history of loose motion, abdominal pain, and alteration of bowel habit, joint pain and rash, bleeding from nose and area of hypo-pigmented or hyper-pigmented skin , headache or vomiting. He gave no history of increased frequency, urgency, hesitancy and red urine. He has no history of swelling of neck He lost about 10 kg weight in last two months. Patient also complained of lost of appetite and become anorexic in last few month. With above complained he visited several times to local doctors every time he was treated with different types of antibiotic name of which he could not mentioned. Now he got admitted in to MU-1, MMCH for further evaluation and better management.

HISTORY OF PAST ILLNESS

No history of DM/HTN/TB

No history of such type of illness before

PERSONAL HISTORY

FAMILY HISTORY

His other family members are also healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition. and lived in Crowding house
Housing: Kacha house
…..rooms, which accommodate 8 0f
His family member.

Sanitation:1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized

Drug History

Patient received some oral and parental antibiotics and ant malarial drug (write only if patient give u this history)

GENERAL EXAMINATION

- Appearance –ill looking and cachetic
- Body built – bellow average
- Nutritional status – severely malnourish
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia –moderately anaemic
- Jaundice : non icteric
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : abscent
- Dehydration – Absent
- Skin –pale
- Body hair distribution –normal
- Bony tenderness – absent
- Lymph node – examination of this patient reveals that patient have generalized lymph adenopathy involving cervical, right axillary group and left inguinal group . There multiple, discrete, rubbery ,nontender lymph node of variable size and shape largest of them in cervical region is 2x 1 cm and in right axillary's region is 1.5x1 cm and left inguinal region is 2x 1.5 cm .these lymph node are not fixed with underlying structure or over lying skin and having no discharging sinus
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse –110 /min, low volume, regular
- B.P – 110/80 mm of Hg
- Respiratory rate – 25/min
- Temperature –100^0F
- Weight : kg
- Height : meter
- BMI : kg/m2

SYSTEMIC EXAMINATION

Examination of Alimentary system

■Mouth & pharynx

Normal

■Abdomen Proper

■Inspection

■Abdomen – abdomen is distended which more marked on upper part and flanks are full
■Umbilicus – centrally placed and inverted
■Visible vein – Absent
■Movement with respiration – present

■Scar mark – no scar mark and no striae
■Visible peristalsis – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,
■Liver is palpable which 8 cm from right costal margin in mid clavicular line and 11.cm from xephoid process having smooth surface , sharp margin , non tender ,firm in consistency , upper border of liver dullness is in right 5th intercostals space with absent hepatic bruit and liver span is 17 cm .
■ spleen is enlarged which is 9 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Finger insinuation is not possible
■Kidney and urinary bladder is not palpable
■There few intra abdominal lymph adenopathy which are rubber discrete and non tender and mobile
■Fluid thrill absent
■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present
■Renal bruit absent

CARDIVASCULAR SYSTEM

•Pulse :110 b/min
Regular, normal in volume & character, all the peripheral pulses are normal.
•BP : 110/80 mm of Hg
•**JVP : Not raised**
•**Precordium** :
Inspection: normal
Palpation: Apex beat in left 5th intercostals space 9 away from midline and normal in character
Auscultation of S1&S2 audible in all auscultatory area

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal
•Movement is symmetrical
•No evidence of respiratory distress
•**Palpation** :
Trachea: Trachea central
Apex beat: in left 5th intercostals space 9 cm from midline normal in character
Vocal fremitus: normal
Percussion: resonance
Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.
Fundoscopic exam: Normal
Cranial nerves: intact

Motor system examination
Motor functions are normal in all four limbs expect generalized wasting of muscle
Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

Salient feature:

- Mr. Salam 17yrs old normotensive, nondiabetic , nonsmoker and non alcoholic Muslim shopkeeper hailing from gouripur , mymesingh with the complaint of fever for 3 months , generalized body swelling and gradually developing lump in left upper abdomen for 1 months . The fever was high grade in nature used to rise some times at the evening and some times at the morning ,lasted for about six to eight hrs, most of the time which was associated with chills and rigors but disappeared with sweating after taking paracetamol and some time spontaneously with out medication .The highest recorded temperature was 103 F ^{o}. Fever was not associated cough, sputum, chest pain or breathlessness. The patient had drenching night sweating but no itching.The patient has no recent or past history of jaundice. The patient has no history of blood transfusion previously. The patient also noticed a pain less mass in the left upper abdomen for last 1 month. The lump was gradually increasing in size toward the umbilicus day by day. The patient has no history of haemoptysis , epistaxis ,haematemesis and malaena. On query, he had no history of contact with known TB patient or traveling into hilly or border area or abroad. There was no history of loose motion, abdominal pain, and alteration of bowel habit, joint pain , rash, and area of hypo-pigmented or hyper-pigmented, headache or vomiting. He gave no history of increased frequency, urgency, hesitancy and red urine. He has no history of swelling of neck and bladder and bowel abnormality. He lost about 10 kg weight in last two months. Patient also complained of lost of appetite and become anorexic in last few month . With above complained he visited several times to local doctors every time he was treated with different types of antibiotic name of which he could not mentioned. Now he got admitted in to MU-1 , MMCH for further evaluation and better management .General examination reveal that the patient is cachetic, ill looking ,no toxic , malnourish moderately anaemic having generalized lymphadenopathy involving cervical , right axillary and left inguinal lymph node . There multiple, discrete, rubbery ,nontender lymph node of variable size and shape largest of them in cervical region is 2x 1 cm and in right axillary's region is 1.5x1 cm and left inguinal region is 2x 1.5 cm .these lymph node are not fixed with underlying structure or over lying skin and having no discharging sinus . The patients temp. 101^{0}F, pulse is regular 110 beat / minute with normal blood pressure (110/80 mm of Hg) respiratory rate is 18 / min and pattern is normal . The patient is non-ecteric, and boney tenderness is absent; The patient has no clubbing, oedema ,splinter hemorrhage, hypo or hyper pigmentation , stigmata of CLD and feature of superior Vena caval obstruction .heat coagulation test and benedict test are negative .Examination of elementary system reveals that abdomen is moderately distended ,flanks normal , umbilicus is in centre inverted, Temperature is normal , no muscle guard or rigidity or tenderness .shifting dullness and fluid thrill is absent and there are multiple intra abdominal lymph nodes of variable size and shape which are discrete, rubbery ,non tender not fixed with under lying structure .Organ palpation reveal non tender hepato-splenomegaly . Liver is enlarge which 8 cm from right costal margin in mid clavicular line and 11.cm from xephoid process having smooth surface , sharp margin , non tender ,firm in consistency , upper border of liver dullness is in right 5th intercostals space with absent hepatic bruit and liver span is 17 cm . Spleen is enlarged which is 9 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Testis is normal in size and consistency . Examination of others systems reveal no abnormality .

Provisional diagnosis

Lymphoma stage IV B

Differential diagnosis

Disseminated Tuberculosis , leukemia

Investigation

***Full blood count* and PBF**

may be normal.
A normochromic, normocytic anaemia
lymphopenia,
An eosinophilia or a neutrophilia may be present.

ESR may be raised.

Chest X-ray may show Bilateral hilar lymphadenopathy @ mediastinal widening

USG of whole abdomen to see intra-abdominal lymphadenopathy

FNAC or biopsy of lymph node

LDH measurements, as raised levels are an adverse prognostic factor

CT scan of **chest and abdomen** for staging.

***Renal function** tests* before start chemo.

***Liver function test** to see before chemotherapy* or see hepatic infiltration.

Treatment

Supportive treatment

Correction of anemia

Specific treatment

According to the staging and classification of lymphoma
Radiotherapy
Chemo therapy
Or both

What are the points in fever of ur provision diagnosis diagnosis?

- Fever
- Night sweating and weight loss
- Aneamia
- Generalized lymphadenopathy which r discrete, rubbery, non tender and mobile
- Hepatosplenomegaly

What is ur differential diagnosis?

My differential diagnosis is **leukemia**

Point in favor	**Point against**
Fever and anaemia Lymphadenopathy Hepato-splenomegaly	Absence of boney tenderness The patient is not toxic

Next: Disseminated Tuberculosis

Point in favor	**Point against**
Fever and anaemia Lymphadenopathy Weight loss Hepato-splenomegaly	Lymph node are usually matted in TB Absence cough with sputum and no alteration of bowel habit

What type of leukemia is common in this age?

Acute lymphoblastic leukemia (if the patient is child)
Acute myeloblastic leukemia (if the patient is adult)

pt will be toxic and boney tenderness will present

If the patient is old

It is chrinic myeloblastic leukaemia (in between 30 and 80 years, peak incidence at 55 years)
it Is chronic lymphoblastic leukaemia (between 65 and 70 years)

What type of lymphoma it is u think? Why?

This is may be Non Hodgkin lymphoma. Because

Peripheral lymph node are involved (incase of Hodgkin axial lymph node)
Extra nodal involvement (hepato-spleno megaly , bone marrow)
Absent of systemic feature and pruritus and pel-ebstin fever goes fever NON HOGKIN lymphoma

In this age which lymphoma is common?

If patient is Young age

Hodgkin lymphoma is common
First peak at 20-35 years and second at50-70 year's .Median age 31 years;

if the patient is old aged person

Then in that age **non Hodgkin** lymphoma is common
Median age 65-70 years

How will u differentiated between and Hodgkin and non Hodgkin lymphoma

	Hodgkin	**Non Hodgkin lymphoma**
1. Lymph node	Localized to single Axial group (cervical , mediastinal , para aortic)	Peripheral
2. Mesenteric and waldeyer ring	Not involved	Commonly involved
3. Spread of LN	Contagious	Non contagious
4. Systemic feature	Common	Less common
5. Pruritus	Common	Less common
6. Pel-ebstein fever	May occur	Does not occur
7. Extranodal involvement	Less common	common
8. Histology	Reed –Sternberg cells (hall mark) present	Absent
9. Prognosis	High cure rate	Low cure rate

What is the staging of lymphoma and why?

Stage IV B

Stage IV is due to involvement of liver and bone marrow
Stage B is due to presence of B symptoms

What are the B symptoms ?

B Symptoms

- ❑ **unexplained fever > 38ºC**
- ❑ **unexplained weight loss (> 10% of body weight in 6 months)**
- ❑ **night sweats**

What do u mean by pel-Ebstein fever?

Recurrent bouts pyrexia followed by apyrexial period

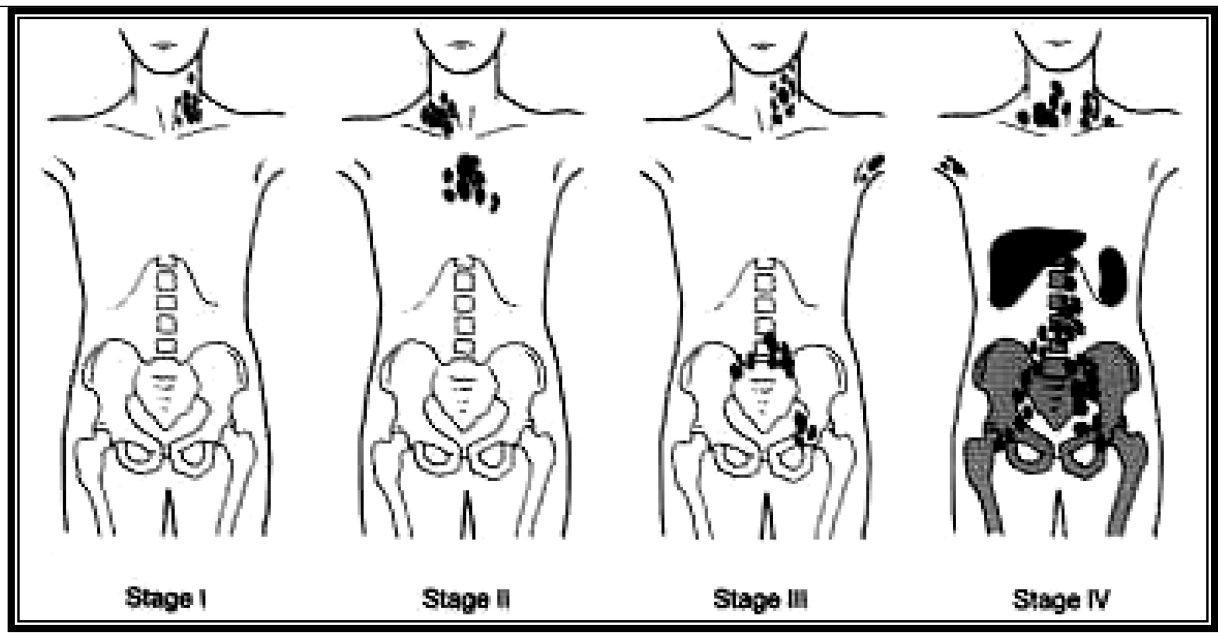
Which virus has link with the lymphoma ?
Epstein-Barr virus has been linked to the pathogenesis of HD**.**

Who will u confirm the Hodgkin disease ?
By seeing Reed-sternberg gaint cell

CLINICAL STAGES OF HODGKIN LYMPHOMA (ANN ARBOR CLASSIFICATION)

Stage	**Definition**
I	Involvement of a single lymph node region (I) or extralymphatic site (IA_E)
II	Involvement of two or more lymph node regions (II) or an extralymphatic site and lymph node regions on the same side of (above or below) the diaphragm (II_E)
III	Involvement of lymph node regions on both sides of the diaphragm with (III_E) or without (III) localised extralymphatic involvement or involvement of the spleen (III_S) or both (III_{SE})
IV	Diffuse involvement of one or more extralymphatic tissues, e.g. liver or bone marrow
A	No systemic symptoms
B	Weight loss, drenching sweats

The lymphatic structures are defined as the lymph nodes, spleen, thymus, Waldeyer's ring, appendix and Peyer's patches.



What investigation u want to do t o Dx lymphoma ?

- ***Full blood count*** **and PBF**
  may be normal.
  A normochromic, normocytic anaemia
  lymphopenia,
  An eosinophilia or a neutrophilia may be present.

ESR may be raised.
Chest X-ray may show Bilateral hilar lymphadenopathy @ mediastinal widening
USG of whole abdomen to see intra-abdominal lymphadenopathy
FNAC or biopsy of lymph node
LDH measurements, as raised levels are an adverse prognostic factor
CT scan of **chest and abdomen** for staging.
Renal function *tests* before start chemo.
Liver function test *to see before chemotherapy* or see hepatic infiltration.

Treat of Hodgkin lymphoma ?

Treat of Hodgkin lymphoma depend on the staging of

Indications for radiotherapy

- Stage I disease
- Stage IIA disease with three or fewer areas involved
- After chemotherapy to sites where there was originally bulkdisease
- To lesions causing serious pressure problems

Indications for chemotherapy

- All patients with B symptoms
- Stage II disease with more than three areas involved
- Stages III and IV disease

Chemotherapy schedule for Hodgkin lymphoma ?

ChlVPP---Therapy

Chl--Chlorambucil
V--Vinblastine
P--Procarbazine
P--- Prednisolone 40 mg/m2 days 1-14

Name the other schedule of chemotherapy

MOPP	COPP	ABCD
M-mustine hydrochloride	C—Cyclophosphamide	A-Adriamycin
O-Vincristine(Onchovin)	O-Vincristine(Onchovin)	B-Bleomycin
P-Procarbazine	P-Procarbazine	V- Vincristine
P-Prednisolone	P-Prednisolone	D- Dacarbazine

Write down the treatment of Non Hodgkin lymphoma?

Treatment depend on it is low grade or high grade

Incase of low grade

Asymptomatic patients may not require therapy.

Indications for treatment include

To remember it **BCS in law**

- **B--B**one marrow failure or
- **C--C**ompression syndromes.
- S--Marked **S**ystemic symptoms
- **L--Ly**mphadenopathy causing discomfort or disfigurement

The options are:

- ***Radiotherapy***. This can be used for localised stage I disease.
- ***Chemotherapy***.
- Oral therapy with chlorambucil,
- ***Monoclonal antibody therapy***.
- **Rituximab**

In case of high grade lymphoma

Patients with high-grade NHL need treatment at initial presentation:

Chemotherapy.

CHOP regimen remains the mainstay of therapy

- C--Cyclophosphamide,
- H--Doxorubicin,
- O--Vincristine
- P--Prednisolone

Radiotherapy.
Is need in case of

- A few stage I patients without bulky disease
- To destroy residual localised site of bulk disease after chemotherapy,
- For spinal cord and other compression syndromes.

Monoclonal antibody therapy.

- When combined with CHOP chemotherapy, rituximab (R) increases the complete response rates and improves overall survival.
- The combination of R-CHOP is currently recommended for those with stage II or greater diffuse large-cell lymphoma as first-line therapy.

What is newer drug in NHL? How it act?

Rituximab-- it is Humanised monoclonal antibodies'
It is the antibody against CD20 cell

What does u mean by salvage therapy? Or what will u do in case relapse in lymphoma?

In case of Hodgkin lymphoma
If relapse occur after 6 months start again with same regime of chemo therapy
If relapse occur with in 6 month of pl started with new or another regime of chemotherapy

What is the prognosis or lymphoma?

Hodgkin lymphoma	Non Hodgkin lymphoma
Over 90% of patients with stage IA disease are cured by radiotherapy alone. Approximately 70% of patients treated with chemotherapy are cured. The 15% of patients who fail to respond to initial chemotherapy have a poor prognosis	**Low-grade NHL** runs an indolent remitting and relapsing course, with an overall median survival of 10 years. **In high-grade** NHL, about 80% of patients overall respond initially to therapy but only 35% will have disease-free survival at 5 years

Type	**Histology**	**Incidence**
Nodular lymphocyte- predominant HL		5%
Classical HL	Nodular sclerosing	70% young female
	Mixed cellularity	20% elderly male
	Lymphocyte-rich	5% male
	Lymphocyte-depleted	Rare

Who pathological classification and incidence of hodgkin lymphoma

Non hodgkin lymphoma

Low grade	**High grade**
o has low proliferation rates, o Slow an indolent course, o Asymptomatic **for many year** o It is not curable by conventional therapy but survive long time o No treatment is need if the disease is advance or symptomatic o Median survival rate 10 years / good prognosis	o High proliferation rate o early symptoms o curable and respond to chemo therapy is good o fatal if not treated o it is large cell type

What r the poor prognostic criteria ?
Increasing age
Lymphopenia
High LDH
If abdominal lymph node size > 10 cm
Advance age

Stroke

PARTICULARS OF PATIENT:

Name: Nasir uddin
Age: 55 years
Sex: Male
Marital status : married
Occupation: farmer
Religion: Islam
Address: Ramonichor, Ishwarganj, Mymensingh.
Date of admission: 1.12.09 at 7pm
Date of examination: 2.12.09 at 10 am

Presenting complaints

- In ability to move right side of body for 5 days.
- Unable to talk. For same duration

History of present illness

According to the statement of the patient s wife, he was reasonable well 2 days ago .At mid noon of that day he was working in his house suddenly he complaint of headache and vomiting followed by inability to move right side of the body. The headache was spontaneous onset, continuous and associated with vomiting for several time and the vomiting was non projectile and contained semi digest food particle. His wife also notice deviation of her husband mouth toward the left side of face and food accumulate in right check and dribbling of saliva from same side .The patient was fully conscious but Drowsy and Had no history of fever and convulsion, discharge from ear (this exclude CNS infection, abscess) .With this complained he was taken local hospital from there he was referred to MMCH and was admitted to MU-1. On query patient attendant state that patient has difficulty in Swallowing specially liquid food, No visual disturbance, no difficulty in micturation and defecation .But they noticed that patient unable to speak but respond to command such as protrude tongue (motor aphasia). The patient had no history of chronic daily morning headache and vomiting associated with weakness of any part of the body (ICSOL) .The patient also had no recent and previous history of head injury. On query patient attendant state that the patient was hypertensive for 5 years with irregular medication and for last few months he was abstinence from anti-hypertensive drugs.

History of past illness

The patient had no previous history of similar type attack (MS , recurrent stroke), TB and malignancy ..The patient has HO of hypertension with irregular medication but non diabetic. Not known a case of heart disease .

Drug history

History of irregularly taking anti-HTN drug s Betanol fro last 2 year, from admission he is taking NG feeding, IV fluid and other oral and injectable drugs but name of which she cannot mentioned.

PERSONAL HISTORY

- The patient is smoker an taking 10 stick / per day for last 45 years, non alcoholic and no history of IV drug user and addiction**.**

Family history

Both of father and mother was hypertensive and used died from acute attack of stroke and rest of the family members healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from middle class family
Sanitation: 1 sanitary latrine.
Water supply: Arsenic free tube well water

GENERAL EXAMINATION

- Appearance –ill lokimg ,there is IV canula , NG tube and catheter in situ
- Nutritional status –average
- Decubitus: supine
- Co-operation : Well co-operative
- Anaemia – mild
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : absent
- Dehydration – Nil
- Skin – normal.
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 190 / 105 Hg of mm
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :

Systemic examination

NERVOUS SYSTEM

- **Higher psychic function including speech** :
  Cannot be evaluated as patient is unable to Talk
  Patient has motor aphasia (as patient cannot talk but obey command)
  (u will write normal if patient able to talk)
- **Cranial nerves** : intact expect Right 7^{th} nerve that shows upper motor lesion because the angle of mouth deviated toward the left and loss of nasolabial fold of right side and wrinkling present on right side and right can properly closed
- Fundoscopic exam : hypertensive retinopathy grade ii (u will write –fundoscopy not done)

Motor system examination

Lower limb

	Right	Left
Wasting and fasciculation	Absent	Absent
Bulk of the muscle	Normal	Normal
Tone of muscle	Increased	Normal
Power of the muscle	2/ 5	5/5
Reflex		
Knee jerk	Exaggerated	Normal
Ankle jerk	Exaggerated	Normal
Clonus	Absent	Absent
Planter	Extensor	Flexor

Upper limb

	Right	Left
Wasting and fasciculation	Absent	Absent
Bulk of the muscle	Normal	Normal
Tone of muscle	Increased	Normal
Power of the muscle	2/ 5	5/5
Reflex		
Bicep jerk	Exaggerated	Normal
Triceps jerk	Exaggerated	Normal
Supinator jerk	Normal	Normal
Hoffman	Present	Absent

Sensory examination

Cannot be evaluate as patient cannot speak

(If patient speak write that ---- all modalities of sensation is intact)

Cerebellar

Sign cannot be evaluated

Signs of meningeal irritation: Absent (neck rigidity and kernig's sign)

CARDIVASCULAR SYSTEM

•Pulse : 72 b/min

Regular, normal in volume & character, all the peripheral pulses are normal.

Carotid bruit : absent

•BP : 190 / 105 mm of Hg

•**JVP : Not raised**

•**Precordium** :

•Inspection: Normal

•Palpation:

Apex beat in lt 5th intercostals space 9 cm from midline

No para-sternal heave and no palpable P_2 and no thrill

•Auscultation

S1&S2 audible in all auscultatory area

No added sound and no murmur

RESPIRATORY SYSTEM

- **Inspection** : Size and shape of the chest : Normal
- Movement is symmetrical
- No evidence of respiratory distress
- **Palpation** :

Trachea: Trachea central
Apex beat: in left 5th intercostals space 9 cm from midline normal in character
Vocal fremitus : normal
Percussion: resonance
Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

Examination of Alimentary system

■**Mouth & pharynx**
Tongue – Dry & coated

■**Abdomen Proper**

■**Inspection**
normal

■**Palpation**
No organomegaly is present

■**Percussion**
Shifting dullness is absent t

■**Auscultation**
Bowel sound –present

Salient feature

Mr. Nasir uddin 55 years married Muslim farmer smoker, non alcoholic, non diabetic, known hypertensive for 5 years with history of irregular anti-hypertensive drug hailing from Ishwarganj , Mymensingh and was admitted in mu-1 of MMCH with the complaints of sudden headache and vomiting followed by development of right sided hemi paresis and Dysphasia during his normal activity . He was fully conscious before and after attack had no history of fever and convulsion and purulent discharge from ear .He had also no history of recent and previous head injury or same type of attack previously. On query patient attendant state that patient has dysphagia for liquid food, no nasal regurgitation, no diplopia or visual disturbance, no bladder and bowel involvement. The patient had no history of chronic daily morning headache and vomiting associated with no focal neurological sign (ICSOL). General examination reveals the patient is hypertensive B.P –190 / 105 Hg of mm , pulse is regular and patient is mildly dehydrated, non ecteric, there is xanthalasma over right eye lid and there is IV canula , NG tube and catheter in situ. Nervous system examination reveals patient has motor dysphasia and so other higher psychic function test could not be done (if ur patient is able to talk u write here the following -----patient is conscious and orientation to time and place and person preserved , speech normal , both recent and remote memory is intact .)Cranial nerve examination reveals upper motor type of Right seven nerve palsy .Pupil is equal and reaction to light and accommodation present .motor examination reveals spastic hemiparasis of right limb with exaggerated jerks and planter extensor. Cerebellar sign and gait and sensory function could be evaluate as the patient has hemparesis and dysphasia (if patient is able to talk then u write here that – all modalities of sensation are intact). The patient has no carotid bruit and murmur on auscultation. Rest of the system reveals normal.

PROVISIONAL DIAGNOSIS

Acute stroke with right sided hemiparesis with motor dysphasia with right sided UMNL type facial nerve palsy with Grade II HTN with dyslipidemia (If no HTN or no xanthalasma u will not write the HTN and dyslipidaemia)

Differential diagnosis:

ICSOL

Investigation

To establish the diagnosis, exclude DD and find out etiology and see complication and comorbid factors

Imageing

- CT Scan of Brain

Others

- ECG
- RBS
- S.creatinine
- CXR-ray
- s.electrolyte
- Echo
- Fasting lipid profile

Treatment of stroke

Q**. in case ICSOL what will u find in general exam. ?**

Pulse ---brady cardia

BP---- HTN

Why this is a stroke

Because it sudden onset ,preceded by head ache and vomiting and focal neurological sign

Risk factor for stroke such hypertension with irregular medication

Dyslipidaemia

Family history

On neurological examination reveal UMNL

Why have u told that the pt have upper motor type VII nerve palsy?

Ans . Because only lower part of rt VII nerve involved & paper part is spared (such pt can close eye and wrinkled the fore head). If it is the lower motor type then all fuction of VII on rt side will loss (such as pt cannot close eye or wrinkle)

What is ur differential diagnosis ?

ICSOL and why

Point favor

Focal neuro logical sign

UMNL

Point disfavor

Sudden onset ---goes favor of stroke

In ICSOL it ill be gradual

Daily chronic head ache and vomiting

No feature of raised ICP

In Fundus papillaedema absent

CT—Scan of brain :

- Haemorrhage –hyper dens ----white
- Infarction ----hypodens ---black

Why this is not a case of abscess or /cns infection

Patient has no fever and convulsion or neck rigidity

Name some example of upper motor lesion

- Stroke
- ICSOL
- Cerebral abscess
- Multiple sclerosis
- Subdural haematoma

Why this not a case of multiple sclerosis

Because the patient has no history of remission and relapse of similar attack

How will u differentiate between and infarctive and hemorrhagic stroke

Haemorrhagic stroke	**Infarctive stroke**
Occur during activity , excitement Patient has HTN with irregular anti-HTN therapy Head ache and vomiting and unconscious BP – highly raised	Occur in normal activity and even in sleep Have feature of risk factor HTN , hyper lipidaemia Carotid bruit and murmur and AF

What type stroke happened here

Tell hemorrhagic -- head ache + vomiting + unconscious ness
Sub arachnoid haemorrhage ---above plus neck rigidity
In Other cases infarctive stroke

What is the site of lesion in this patient?

Left cerebral cortex

If ur patient is hemiparesis / hemiplagia

Site of the lesion is ant 2/3 of post.limb of left internal capsule

What is the supply or which vessel involved?

Lenticulo –strial branch of middle cerebral artery. these are the end artery here lacunar infarction happened

What cause of lesion ?

May be thrombosis or emobolism

What is the source of embolism

Cardio-artery and artery to artery

Why hemiparesis occur

Because all the compact fiber pyramidal cell pass through narrow area

Why dysphasia happens

Because also involvement of speech area

Why u told it motor dysphasia

Patient can not talk. But can respond to command such as if u ask the patient protrude tongue he Can protrude tongue

When u told it sensory dysphasia

Patient can not talk. Can not respond to command such as if u ask the patient protrude tongue he will not do it

Define and classify stroke

ACUTE STROKE

Acute stroke is characterised by the rapid appearance (usually over minutes) of a focal deficit of brain function, most commonly a hemiplegia with or without signs of focal higher cerebral dysfunction (such as aphasia), hemisensory loss, and visual field defect or brain-stem deficit

Classification of stroke

- ***Transient ischaemic attack (TIA)***. When symptoms resolve within 24 hours of onset
  Progressing stroke (or stroke in evolution). The stroke in which the focal neurological deficit

worsens after the patient first presents. Such worsening may be due to increasing volume of infarction, haemorrhage or related oedema.

- ***Completed stroke.*** The stroke in which the focal deficit persists and is not progressing

What are the features of brainstem involvement?

- Ataxia,
- diplopia,
- vertigo and/or bilateral weakness
- and 3D disarthia , dysphagia , dysphonia
- cross hemiplegia

What do u means by cross hemiplegia ?

it means LMN type of cranial nerve lesion in one side and hemiparesis is in the opposite sight

where it found ?

it found in the brain stem lesion

Risk factors in stroke

un modifiable to remember **GRAPH**

- **G--**Gender (male > female, except in the very young and very old)
- **R--**Race (Afro-Caribbean > Asian > European)
- **A--**Age
- **P--**Previous vascular event, e.g. myocardial infarction, stroke or peripheral embolism
- **H---**Heredity

Modifiable to remember ABCD-SHOP

- A--Excess alcohol consumption
- B--High blood pressure
- C--Heart disease (atrial fibrillation, heart failure, endocarditis)
- D--Diabetes mellitus
- S--Smoking
- H--Hyperlipidaemia
- O--Oral contraceptives
- Polycythaemia

cause of haemorrhic stroke in
Risk factors to remember **ABCD**
A---**Arteriovenous malformation**
Amyloid angiopathy
B --**Hypertension**
C--- Coagulopathy
Anticoagulant therapy
Blood dyscrasia
Thrombolytic therapy
3.DRUGS
Alcohol
Amphetamines
Cocaine

Cause of stroke in young patient
To remember CAT HAS vasculitis
C-- Cardiac embolism (MS)
A-- Premature atherosclerosis
T-- Thrombophilia
Protein C
Protein S
Antithrombin III
H—Homocystinuria
A-- Antiphospolipid antibody syndrome
S-- Systemic lupus erythematosus

vasculitis

Of the all stroke

complication of bed ridden patient

Infarctive is 85 %

Haemorrhagic stroke 15 %

Infarction

- mostly to thromboembolic disease to 2dary atherosclerosis in the major extracranial arteries (carotid artery and aortic arch).
- 20% due embolism from the heart
- 20 % due to intrinsic disease of small perforating vessel of lenticulostriate artery of (lacunar infarction)

A- Aspiration pneumonia
B- Bedsore
C- Constipation
D- DVT
E- Epilepsy
F- Frozen shoulder / pain full shoulder
Urinary tract infection
Depression and anxiety
Oteosporosis

What do u mean by UMN

Pyramidal cell and their Axon up to ant. Horn cell and up to motor nuclei of brain stem

What do u mean by LMN

Ant. Horn cell and their homologous neuron in the brain-stem and their axon up to effector organ is called LMN

	Upper motor neuron lesion	**Lower motor neuron**
Fasciculation and wasting	Absent	Present
Tone	Hypertonic	Hypotonic
Reflex	Deep reflex exaggerate	Both superficial and deep reflex are lost
Planter	Extensor	Flexor
Clonus	Present	Absent
Paralysis	Spastic paralysis	Flaccid paralysis

Cause of UMNL

Stroke
ICSOL
Cerebral abscess
Multiple sclerosis
Spinal cord compression
3 T Trauma
TB
Tumour –2ndary and

multiple myloma

Cause of LMNL

GBS
Poly myelitis
Motor neuron disease
Diabetic amytrophty

Muscle power grading
0 – no movement
1—only flicker of movement
2—side movement possible but not against
Against the gravity
3—movement against the gravity is possible
4—move against resistant is possible
5—normal movement

How will u evaluate muscle power?

- First ask the patient to lift his leg with out bending his knee .—if can –power is > 3
- Then check movement against resistant
- If muscle power is < 3
- Then look for side to side move if possible then muscle power is >2
- When it is <2 then ask to move it finger if patient can do it then power is 1
- HEMIPLAGIA – complete paralysis muscle power is 0
- Hemiparesis ---- muscle power is 1/2

Read the root value of jerk
Reflex arc
Tendon and muscle of knee and ankle jerks

What is u see in patient with stroke other than neurological examination
See eye

- Xanthelasma
- Fundoscopy
  - Diabetic changes
  - Hypertensive changes
- Rashes (arteritis, splinter haemorrhages, livedo reticularis)
- Pulse AF/ bradycardia (AF pulsus deficit present, that is hear rate > pulse)
- Bp : hypertension
- Heart rhythm (atrial fibrillation)
- Murmurs (sources of embolism) and Apex beat shift
- Peripheral pulses (generalised arteriopathy) and bruits (carotid)

Young patient with stroke cause
See following are present or not
Valvular heart disease
HTN
Vasculitis
AVM
Rupture Barry aneurysm
Hyperlipidaemia

Old **patient with stroke cause**

- **HTN**
- **DM**
- **IHD**
- **Atherosclerosis**

A young patient comes to u with left sided hemi paresis and on auscultation the patient irregular pulse and diastolic murmur . what is the D_x

Patient is a case of mitral stenosis with AF and infarctive stroke due to thrombo embolism that arise from the left atrium and goes to cerebral vessel

- ***Jerk absent but planter extensor what are the cause***
  - SCD
  - MND
  - Tabes dorsalis
  - Fredrich ataxia
  - DM with stroke

Cause of peripheral neuropathy
To remember GBS @ PLID-3
GBS
P-paraneoplastic
L-lead
I –Infective –leprosy , HIV
D—DM
D—deficiency B_1 B_6 B_{12}
D---DRUGS ---INH

Q . A patient comes to u with absent right knee jerk but exaggerated of right ankle jerk ?
It happen if lesion is in L3 and L4 level of spinal cord .
As it LMN lesion is at level L3 and L4 causes lose of knee jerk and upper motor lesion for bellow it so ankle jerk is exaggerated

- **If patient comes to u with stroke u have to write provisional diagnosis as**
  - **Acute stroke with right sided (**affected side **) hemi-paresis with right sided upper motor type (**remember that hemi paresis and VII will same side if hemiparesis rt side then it will associated with rt sided upper motor type of facial nerve palsy **) with grade –I** hypertension +/ - dysphasia

    if patient not able to talk then add
    - **sensory dysphasia ----**can not talk but also can not obey command or communicate. such as ask to show tongue it do not follow the command
    - **motor dysphasia --** can not talk but obey command or communicate such as if asked to show tongue he can follow the command

What will be the differential diagnosis?

- **ICSOL**

Onset of symptom	sudden –stroke , gradual ICSOL
consciousness	
for hemorrhagic stroke	headache , vomiting
if vomiting	frequency , amount , projectile or non projectile , food particles
focal neurological sign	Unable to move one side of body Deviation of mouth (vii nerve palsy) Unable to talk and if unable then see can he follows command such (ask to show tongue)
	Difficulty in Swallowing specially liquid food
	No visual disturbance
	bowel & bladder involvement
for ICSOL	chronic daily headache and vomiting
History of fever and convulsion	meningo-encephalitis
History discharge from ear	To exclude CNS infection, abscess)
recent or previous history of head injury	

hypertensive	if present then is he taking medication , if yes then regularly or irregularly and name of the HTN drug if patient can tell
past or	previous attack to exclude recurrent stroke
in general examination	look carefully the patient have NG or not , cather or IV fluid in situ

Thalassaemia

PARTICULARS OF PATIENT:

Name: Mahbub.
Age: 17 years
Sex: Male
Marital status: Unmarried
Occupation: Student
Religion: Islam
Address: Muktagacha, Mymensingh .
Date of admission: 1.12.09 at 7pm
Date of examination: 3.12.09 at 10 am

Presenting complaints

Gradual development of lump in left upper abdomen for 7 years
Generalized weakness and pallor for last 6 months

History of present illness

According to the statement of the patient he was reasonable well 7 years back then he noticed a lump in left upper abdomen .The lump was gradually increasing in size toward the umbilicus day by day. At the same time patient also developed weakness, easily fatigability and palpitation. With that complained he was admitted in hospital and was treated with 3 unites of fresh blood .After blood transfusion he was improved and was discharged from the hospital. He had to admit several times in the hospital with same complaints and every times he was treated with blood transfusion. As far as patient remember he has received approximately 30 unites of blood till today. He received last blood transfusion 1 year ago. For last 6 month patient gradually developed pallor associated with lethargy and weakness. On query patient give repeated history of yellow coloration of skin and urine which subside spontaneously .For last 6 months patient again develop yellow coloration of urine and skin without nausea, vomiting, itching, and color and consistency of stool is normal. The patient has good appetite with normal body hair and distribution (to exclude hypogonadism/ CLD) , No history of alteration of bowel habit, vomiting out of blood and black tarry stool(to exclude PUD),bleeding per rectum and chronic blood loss . fever with and with out chill and rigor ,weight loss , cough with sputum (to exclude the infective cause), joint pain and swelling(pseudogout) , increased frequency of micturation and increased thrust (DM), no history recurrent upper abdominal pain (To exclude gall stone) .The patient had no history of breathlessness on exertion, rest or in lying position(or some patient may present with breathslessness due to anaemic hear failure).But the patient complaint that his skin become darker day by day . with above complaint he was admitted into medicine uint-1 MMCH for blood transfusion

History of past illness

The patient

PERSONAL HISTORY

The patient is Non smoker, Non alcoholic. And have no history IV drug user. The patient has no history of homo or heterosexuality

Drug history

The patient had received 1 unite of fresh blood one day ago . he is regularly taking tab. folison daily .

FAMILY HISTORY

He has two bothers and three sisters. One of his bother is suffering from similar types of disease and is on

regular blood transfusion .his father and mother were cousin .His father was died 5 years ago but cause of death he cannot mention .the rest of the family members are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition and lived in crowding house
Housing: Tin shade house.
2 rooms, which accommodate 7 0f
His family member.
Sanitation: 1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized against hepatitis B

GENERAL EXAMINATION

- **Appearance –Haemolytic face (frontal bossing , depressed nasal bridge , mal occlusion of upper incision teeth**
- Body built – average
- Nutritional status –average
- Decubitus: on choice
- Co-operation : Well co-operative
- **Anaemia – severe anaemia (may be moderate)**
- **Jaundice : mild**
- Cyanosis : Absent
- Clubbing : Absent
- **Koilonychia : Absent**
- **Leukonychia : Absent**
- Dupuytren's contracture : Absent
- **palmar erythema: Absent**
- **hepatic flap / flapping tremor : absent**
- **Spider nevi : Absent**
- **Gynaecomastia : Absent**
- **Oedema : Absent**
- Dehydration – Nill
- **Skin –general skin condition and is normal. No evidence of scratch marks and generalized pigmentation. There is iV canula on right hand.**
- **Body hair distribution –normal**
- **Bony tenderness – absent**
- **Lymph node – no lymphadenopathy**
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 130/80 mm of Hg
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight :40 kg
- Height :
- BMI :
- **Bed side urine examination show ,Sugar= Nill**

SYSTEMIC EXAMINATION

Examination of Alimentary system

■**Mouth & pharynx**

■Tongue – white and loss of papilla

■Malocclusion of upper incision teeth

■**Abdomen Proper**

■**Inspection**

■Abdomen – upper abdomen is distended and flanks are not full
■Umbilicus – centrally placed and inverted
■Visible vein – absent
■Movement with respiration – present
■Scar mark – no scar mark and no striae
■Visible peristalsis – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,
■Liver is palpable which 5 cm from right costal margin in mid calvicular line having smooth surface , sharp margin (rounded), non tender ,firm in consistency , upper border of liver dullness is in right 5 the intercostals space , with absent hepatic bruit and liver span is 15 cm .
■ spleen is enlarged which is 9 cm from left costal margin in ant Axillary line toward its long axis having smooth surface , firm to hard in consistency , non tender ,notch in its upper border and there is no splenic bruit. Finger insinuation is not possible
■Kidney and urinary bladder is not palpable
■No intra abdominal lymph adenopathy
■Fluid thrill absent
■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present

CARDIVASCULAR SYSTEM

•Pulse : 88 b/min

Regular, normal in volume & character, all the peripheral pulses are normal.

•BP : 130/80 mm of Hg

•**JVP : Not raised**

•**Precordium** :

Inspection: Normal

Palpation: Apex beat in lt 5th intercostals space 9 cm from midline (may be shift due to heart failure)

No para-sternal heave and no palpable P_2

AuscultationS1&S2 audible in all auscultatory area

No added sound and no murmur

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Movement is symmetrical

•No evidence of respiratory distress

•**Palpation** :

Trachea: Trachea central

Apex beat: in left 5th intercostals space 9 cm from midline normal in character

Vocal fremitus: normal

Percussion: resonance
Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.
Fundoscopic exam: Normal
Cranial nerves: intact
Motor system examination
Motor functions are normal in all four limbs
Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

SALIENT FEATURE

Mr. mahbub 17 years old unmarried Normotensive ,Nondiabetic, Nonalcoholic ,Nonsmoker Muslim student hailing from Mukta ghacha , Mymensingh wad admitted in medicine unit-1 of MMCH with the complained of gradually developing lump from left upper quadrant of abdomen toward the umbilicus for 7 years with repeated history of generalized weakness and easily fatigability, pallor , palpitation and jaundice which has been persisting for last 6 month. Each times he had to admit in the hospital and was treated with blood transfusion. The patient got relieved temporally from these symptoms and few months later patient again developed these symptoms. Gradually the interval between these remissions and relapses was decreasing. He had to admit several time in the hospital fro blood transfusion The Jaundice was not associated with nausea, vomiting, itching, and color and consistency of stool was normal. The patient has no history of hematemesis and malena, chronic blood loss like hemorrhoid, alteration of bowel habit, fever with chill and rigor , joint pain and swelling , poly uria and poly depsia . He also has no history of recurrent upper abdominal pain, weight loss and cough with sputum, contact with TB patients, exertional dyspnea or orthropnea. The patient developed generalized pigmentation during this period. The appetite was normal. Previously patient received near about 30 unite human fresh blood, last transfusion was done one year ago. After admission in this hospital he transfused 1 unit of fresh blood. There is history of *Consanguineal* marriage between his parent and one of his elder bother is suffering from this type of this disease and on regular blood transfusion . General examination reveals the patient has haemolytic faces, severe anemia with mild jaundice with huge hepato-splenomegly with out stigmata of CLD.The patient has no lymphadenopathy, edema, boney tenderness, raised JVP and other system reveals no abnormality. Systemic examination reveals only hepato-splenomegaly

PROVISIONAL DIAGNOSIS

Congenital haemolytic anaemia with secondary haemochromatosis

Differential diagnosis

Chronic liver disease with portal hypertension

Investigation

CBC ----- Hb % ↓
Increased reticulocyte count
PBF --- Microcytic-Hypochromic , Anisopoikilocytosis, Target cells, fragmented cells , large number Of Nucleated red cells

Definite test ---
haemoglobin electrophoresis
. Minimal to no HbA, elevated HbF and HbA2
USG OF whole abdomen
To see hepato-splenomegaly
Gall stone
X-Ray Skull shows
Widening of the diploeic space with a "hair on end" appearance.
LFT
Elevated total and unconjugated bilirubin,
Elevated LDH
Serum ferritin

TREATMENT

DEIT
Avoid iron contain food an oral iron tablet
Liver, beef,

Regular blood transfusion
To keep the Hb level above 10 gm / dl
Iron chelation
Parental

Desferrioxamine : 30 to 50 mg/kg/day given by subcutaneous infusion over 8 to 12 hours, 5 to 7 nights per week

Oral drug
Deferiprone (cap .Kelfer) 50-75 mg/kg/day in 3 divided dose

Vitamin and folic acid supplementation
Vitamin –C increase iron excretion
Folic Acid daily 5 mg daily

Definite treatment
Haematopoietic stem cell transplantation

Surgical treatment
Splenectomoy

What is ur provisional diagnosis?

My provisional diagnosis is congenital hemolytic anemia with secondary heamochromatosis
Because of family history of *Consanguineal* marriage
One of his brothers is suffering from this disease
Typical history and
On general examination
- Haemolytic face
- Severe anaemia
- Mild jaundice

Alimentary system examination
- Hepato-splenomegaly

Why u told secondary haemochromatosis ?

Because of increased pigmentation the skin become black

What are the cause of haemochromatosis ?

Due to deposition of iron due to blood transfusion and excessive break down of RBC

If the patient comes above feature and edema ?

Diagnosis will be **congenital hemolytic anemia with secondary heamochromatosis with anemic hear failure**

What is u r differential Diagnosis ?

Chronic liver disease with portal hypertension

Point in favor of u r diagnosis

Jaundice

Splenomegaly

Point in against of u r diagnosis

No stigmata of CLD such as

- Leukonychia, spider nevi, gynaecomastia,
- Palmer erythama , ascites , engorged vein and
- Testicular atrophy , loss of body hair distribution
- Haematomesis and malena

Why this is not a case of leukemia?

Point against Ares

Because of long duration acute leukemia is unlikely.

Also due to

Patient is not toxic with no high fever

Boney tenderness absent

If it is chronic leukaemia what would ur be diagnosis?

Chronic myeloid leukemia

Why u call it CGL / CML?

Due to huge splenomegaly

Is this age Common for CGL?

No, it occur in older age.

Why it is not a case of lymphoma?

Point favor

Hepatosplenomegaly

Point against Ares

Long history

No lymphadenopathy and absence of fever and night sweating

(if the pt have fever then following Que .may be asked)

Why this is not case of Kala-azar / chronic malaria ?

Not from endemic zone (in this pt MUKTA GHACHA is endemic zone for kala-azar)

In case malaria – no chill and rigor fever

In case of kala-azar --- pattern of fever not correlate with kala-azar ,

In Both case long history of 7 years with splenomegaly with fever life is unlikely

***What investigation u will do*?**
First what will u do?
CBC ----- Hb % ↓
Increased reticulocyte count (normal < 2 %)
PBF --- Microcytic-Hypochromic , Anisopoikilocytosis,
Target cells, fragmented cells , large number Of Nucleated red cells
What is the Definite test for diagnosis thalassaemia ?
haemoglobin electrophoresis
. Minimal to no HbA, elevated HbF and HbA2
What are the other investigations?

USG OF whole abdomen
To see hepato-splenomegaly
Gall stone
LFT
Elevated total and unconjugated bilirubin,
Elevated LDH
Serum **ferritin**

What is common haemolytic disease in bangladesh ?
- Haemoglobin E diseases

What is cause of haemolytic aneamia in this patient?
- Haemoglobin E beta thalassaemia
- Because here patient have severe aneamia & huge hepato-splenomegaly
- In sub-continent it is common
- Oneparent is suffering from Hb E disease and another parent is suffering from thalassaemia minor

What is the X-ray finding in thalassamia ?
X-Ray Skull shows
Widening of the diploeic space with a "hair on end" appearance.

What are causes of microcytic and hypochromic anaemia ?
- Iron deficiency
- Thalassaemia
- Sederoblastic anaemia

What are the complications of thalassaemia ?
- Bronz diabetes –deposition of iron
- Growth retard / dwarfism
- Gallstone ---pigmented stone –
- CLD
- Hypogonadism –due to deposition of iron in to hypothalamus
- (Hypopituitarism)The anterior pituitary is involved
- Heart failure – Due to anaemia –
  - Due to haemochromatosis –
- Joint pain – pseudogout –due deposition of iron in to synovial fluid
- Neurological examination – encephalopathty
- Hyper pigmentation

What did u mean by bronz diabetes?
Deposition of iron in pancreatic islet cells cause insulin dependent (type 1) diabetes and deposition of iron in the skin increase pigmentation cause dark or bronz coloration skin .when this two think present together then it is called Bronz diabetes .

What will u give in this DM ?
- Insulin as it is type I

A patient with thalassaemia comes to u with Right hypochrondium pain what is ur diagnosis ?
Cholelithiasis ----pigmented stone in the gall bladder due to –bilirubrin

What is the effect heart in patient with thalassaemia ?

Due to anaemia ---anaemic heart failure ---DCM
Due to haemochromatosis ----
Arrhythmia

A patient with thalassaemia comes to u with the complained left knee joint pain ?

Cause is pseudogout

Why hypogandism ?

Due to accumulation of iron in hypothalamus . Patient will loss body hair and libido

Tell the treatment of thalassamia ?

TREATMENT
DEIT
Avoid iron contain food an oral iron tablet
Liver , beef ,

Regular blood transfusion
To keep the Hb level above 10 gm / dl
Iron chelation
Parental

Desferrioxamine : 30 to 50 mg/kg/day given by subcutaneous infusion over 8 to 12 hours, 5 to 7 nights per week

Oral drug

Deferiprone (cap .Kelfer) 50-75 mg/kg/day in 3 divided dose

Vitamin and folic acid supplementation
Vitamin –C increase iron excretion
Folic Acid daily 5 mg daily

What is the new iron chelating agent in Bangladesh ?
- Ans : Asunra

Definite treatment
Haematopoietic stem cell transplantation

Surgical treatment
Splenectomy

What are the Indication of splenectomy ?

- Huge splenomegaly due to pressure effect
- If patient need repeated blood transfusion in a short interval (200 to 250ml / kg packed cell per year to maintain an Hb level at 10 g/dl).
- Feature of hypersplenism

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- Feature of hypersplenism

What are the iron chelating agent use in treatment thalassaemia ?
See before in treatment part

How does Desferrioxamine given?
In given subcutaneously via infusion pump . but in our ward it given iv infusion in drip .
2 amp inj. Desferal in 500 ml DA iv @ 10 drop/ min .

100 mg desferal will bind 8 gm iron
One unit blood will accumulate 250mg of iron
When transfusion exceeds 100 ml / kg of body weight needs chelation
Or serum feritinin level > 1000 µgm / L

Name the oral iron chelating agent?
Deferiprone (cap .Kelfer) 50-75 mg/kg/day in 3 divided dose

Counseling patient with of thalassamia ?

- It is genetic disorder (Autosomal recessive) run from generation to generation
- One forth of his children will be affected and one forth will be carrier
- So before married u has to test ur wife Hb electrophoresis to detect she is trait of carrier
- Patient only developed thalassamia major if both his partent affect or trait
- Pain pathology is defective production of Hb
- So early destruction of RBC so need regular blood transfusion
- Main hazard is due secondary haemochromatosis due to iron overload

Can perinatal diagnosis of thalassaemia possible?

Yes perinatal diagnosis is possible by collecting DNA material from chroionic villus sampling during 8 wk of gestation or by Amniocentesis from 14- 20 wks of pregnancy . if positive termination of pregnancy

What is the definite Treatment of thalassamia?
Allogenic Bone marrow transplantation
Which one is the normal Hb in adult ?

- Hb A is normal haemglobin in adult

Hb A	adult haemoglobin
Hb A2	Normally 2% , increased in β-thalassaemia minor
Hb F	Normal haemoglobin in fetus , in adult less 1% , increased in β-thalassaemia Major
Hb S	sickle cell anemia
Hb C	

What type anemia in thalassaemia quantitative or qualitative ?

- It is qualitative anemia as defect in the globin chain of haemoglobin .

Which chromosome is responsible for globins chain formation ?

- Chromosome 16

What is the cause of haemolysis in thalassaemia ?

- in beta-thalassaemia excess alpha chains are present. The excess chains precipitate, causing red cell membrane damage and reduced red cell survival.

What type of disease it is?

- It is autosomal recessive diseases

What do u mean by thalassaemia major and minor?

Thalassaemia major	Thalassaemia minor (trait)
• it is homozygous disease • both parents have thalassaemia minor • profound hypochromic anemia • Hb electrophoresis usually shows a raised Hb F • Require regular transfusion	• it is heterozygous disease • one parents have thalassaemia minor • asymptomatic • Anaemia is mild or absent. • Hb electrophoresis usually shows a raised Hb A2 • Usually does not require transfusion

What do mean by thalassaemia intermediate?

- It is in between thalassaemia major and minor .
- Thalassaemia intermedia includes patients who are symptomatic with moderate anaemia (Hb7–10 g/dL) and who do not require regular transfusions.

What is the cause of hepatosplenomegaly & haemolytic facies in thalassaemia ?

- Extramedullary haemopoiesis that soonleads to hepatosplenomegaly and bone expansion, giving rise to the classical thalassaemic facies

What will u do before splenectomy ?

Before splenectomy against organism you hav to vaccinated the patient ?

- Meningococcal group C
- *Haemophilus influenzae* type B,
- Pneumococcal

When will you give the patient vaccine?

At least 2-3 weeks before elective splenectomy

When boster dose given ?

- **Pneumococcal re-immunisation should be given at least 5-yearly**
- **Influenza vaccine annually**
- **Life-long prophylactic penicillin V 250 mg 12-hourly**

What extra measure you should take in patient with splenectomy ?

- In septicaemia, splenectomised patients should be resuscitated and given intravenous antibiotics to cover pneumococcus, *Haemophilus* and meningococcus
- The risk of malaria is increased
- Animal bites should be promptly treated with local disinfection and antibiotics, to prevent serious soft tissue infection and septicaemia

What will he carry with him ?

A card or bracelet should be carried by splenectomised patients to alert health professionals to the risk of overwhelming sepsis,

New therapy in Thalassamia?

Inj.Erythropoietin—it stimulate bone marrow
Hydroxyurea--- it prevent in effective erythopoiesis

Target Hb level in thalassamia ? serum feretin level ?

Indication of splenectomy in thalassamia ?

- Requirement of excessive transfusion to maintain Hb level > 10 gm / dl (more than I unit / month)
- Feature of hypersplenism
- Massive splenomegaly causing mechanical problem (dragging pain / early satiety)

Name some causes of refractory anaemia ?

- Aplastic anaemia
- Sideroblastic anaemia (Rx—pyridoxine)
- Myelodysplastic syndrome
- chronic renal failure

prognosis of

what do u mean by super transfusion and hyper transfusion ?

- Super transfusion : Hb level is kept > 10 gm / dl
- Hyper transfusion : Hb level is kept > 12 gm / dl

What are the advantages of hyper transfusion?

- GIT iron absorption decreased
- less chance of facial disfigurement
- less chance of hypersplenism
- less need for early splenectomy
- growth and development are increased

A patient admitted in your hospital for repeated blood transfusion what may the possibility?

- Thalassamia
- Aplastic anaemia
- ITP(immune thrombocytopenic purpura)
- Haemophilia
- CRF
- Recurrent haematemesis and melaena

Two other test can be done or

- Alkali denaturation test : ---test positive (HbF is resistance to alkali denaturation)
- Osmatic fragility test : it is decrease – that mean there is increased resistance to red cell to osmotic lysis (osmotic fragility test is increased in hereditary spherocytosis)

Complication of repeated blood transfusion?
risk of iron over load / haemochromatosis
transfusion hazard – hepatitis B C , AIDs
chance of isoimmunization
volume overload ---heart failure

Name some organism transmit by blood donation?

malaria , hepatitis B , C , HIV , syphilis , toxoplasmosis ,

Causes of target cell in blood ?

iron deficiency anaemia , thalassamia , after splenectomy , cholestatic jaundice

Thalassaemia

Usually thalassaemia patient usually admitted into hospital only for blood transfusion . u have write history from the very beginning when it was first detected . in chief complaint u also mention recent reason of admission in the hospital .

The provision diagnosis is

- Congenital haemolytic anaemia with secondary haemochromatosis

Differential diagnosis

- Chronic liver disease with portal hypertension

Following history u have to take

Anemia history	weakness , fatigability ,
	history of blood loss haematomasis & melaena (PUD / CLD) Bleeding per rectum and chronic blood loss
jaundice history	then take ho of vomiting, nausea, joint pain (for viral hepatitis)
	then take HO Itching .Color of stool pale or not to see obstructive jaundice
History of CLD	Loss of body and pubic hair and decreased frequency of saving & loss libido , haematomasis & melaena
History lump / spleen	appearance of a lump in left upper abdomen for how many days
history of blood transfusion	how many times in yr , date of last blood transfusion
consanguial marriage	marriage of parent with in relative
HO brother and sister	is any of them is suffer from this disease or any of them had died in this disease
to exclude infective causes	fever with or without chill and rigor ,weight loss , cough with sputum
PERSONAL HISTORY	Alcoholic. IV drug user, multiple extra marital sexual exposure to see etiology
immunization HO	immunized against HBV or not

History Complication of

DM	• Increased frequency of micturation , increased thrust and polyphagia
haemocromatosis	• Progressive darkening of skin
pseudogout	• Joint pain and swelling
gall stone	• History recurrent upper abdominal pain
anaemic hear failure	• History of breathlessness on exertion, rest or in lying position
hypogonadism/ CLD	• Normal body hair and distribution

Viral Hepatitis

PARTICULARS OF PATIENT:

Name: Mostofa Kamal
Age: 35 yrs
Sex: male
Marital status: married
Occupation: fisher man
Religion: Islam
Address: Valuka, Mymensingh.
Date of admission: 1.12.09 at 7pm
Date of examination: 3.12.09 at 10 am

Presenting complaints
Nausea and anorexia for 10 days.
Yellow coloration of skin, eye and urine for 7 days.

History of present illness
According to the statement of patient he was relatively well 10 days ago then he developed nausea and anorexia, loss of appetite for all types' food including distaste for cigarettes. 3 days later his family members noticed him that his eyes became yellow. He initially thought nothing of it but became concerned when this yellowish discoloration gradually spread to his face then whole over the body. His urine became yellow to dark in color. The patient no history of fever with chills and rigor but he complained of malaise, mild nonspecific headache and vomiting for several times which was non projectile in nature, sometimes green in color, contain mostly water and few food particles not contained blood and sometimes sour or bitter in taste .The patient also complained generalized itching and fatigue . The color of stool was not pale .The patient has no history of weight loss, abdominal pain, loss of body hair , vomiting out of blood and black tarry tool, Alter level of consciousness and alteration of sleep pattern,. Joint pain and swelling. The patient also had no history of transfusion of blood and blood product and no history of abdominal surgery previously. The patient had history of shaving in salon and was unaware about using of disposable blade every time. (Or he used to shave in home and did not share razor). The patient's libido and urine out put is normal and bowel moves once daily.

History of past illness
No previous history of jaundice, gall stone or liver disease. He had no history hypertension and Diabetes.

Drug history
The patient has no significant drug history

PERSONAL HISTORY

The patient is smoker he smokes 5-8 sticks per day, Non alcoholic and has no history IV drug user. The patient had no history multiple extra marital sexual exposures .

FAMILY HISTORY
None his family member is suffering from jaundice.
They are healthy and enjoining sound health.

SOCIOECONOMIC CONDITION
He comes from low socioeconomic condition and lived in crowding house
Housing: Tin shade house.
2 rooms, which accommodate 8 0f his family member.

Sanitation: 1 sanitary latrine.
Water supply: Arsenic free tube well water but sometimes he used to drink Tap water when he remained out side the house.

Immunization history

The patient was not immunized against hepatitis B

GENERAL EXAMINATION

- Appearance –ill looking
- Body built – Average
- Nutritional status –Average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – Absent
- **Jaundice : moderate**
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- **Leukonychia : Absent**
- **Dupuytren's contracture : Absent**
- **palmar erythema: Absent**
- **hepatic flap / flapping tremor : absent**
- **Spider nevi : Absent**
- **Gynaecomastia : Absent**
- **Oedema : Absent**
- Dehydration – Nill
- **Skin –general skin condition and is normal and evidence of scratch marks**
- **Body hair distribution –normal**
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 88/min, low volume, regular
- B.P – 130/80 mm of Hg
- Respiratory rate – 25/min
- Temperature – 98^0F
- Weight : 53 kg
- Height : 152 cm
- BMI :

Systemic examination

Examination of Alimentary system

■**Mouth & pharynx**

Normal

■**Abdomen Proper**

■**Inspection**

■Abdomen –normal in size and shape and flank not full
■Umbilicus – centrally placed and inverted
■Visible vein – absent
■Movement with respiration – present
■Scar mark – no scar mark and no striae

■Visible peristalsis – absent
■Hernial orifice – intact
■Hair distribution – normal
■External genitalia –normal

■**Palpation**

■Superficial & deep palpation: normal temperature , No muscle guard, no tenderness,
■Liver is palpable which 3 cm from right costal margin in mid clavicular line which tender having smooth surface , sharp margin (rounded), firm (or soft) in consistency , upper border of liver dullness is in right 5th intercostals space with absent hepatic bruit and liver span is 15 cm .
■ spleen ,Kidney and urinary bladder is not palpable
■No intra abdominal lymph adenopathy
■Fluid thrill absent
■Testes are normal is size and consistency

■**Percussion**

Shifting dullness absent

■**Auscultation**

■Bowel sound –present
■Renal bruit absent

CARDIVASCULAR SYSTEM

- •Pulse : 88 b/min
  Regular, normal in volume & character, all the peripheral pulses are normal.
- •BP : 140/85 mm of Hg
- •**JVP : Not raised**
- •**Precordium** :

Inspection: Normal
Palpation: Apex beat in lt 5th intercostals space 9 cm from midline
Auscultation of S1&S2 audible in all auscultatory area
No added sound and no murmur

RESPIRATORY SYSTEM

- •**Inspection** : Size and shape of the chest : Normal
- •Movement is symmetrical
- •No evidence of respiratory distress
- •**Palpation** :

Trachea: Trachea central
Apex beat: in left 5th intercostals space 9 cm from midline normal in character
Vocal fremitus: normal
Percussion: resonance
Auscultation:
Breath sound is vesicular in all parts of the chest.
No added sound
Vocal resonance: normal

NERVOUS SYSTEM

Higher psychic function including speech: normal.
Fundoscopic exam: Normal
Cranial nerves: intact
Motor system examination
Motor functions are normal in all four limbs
Sensory examination
All modalities of sensation are intact in both upper and lower limbs

Cerebellar signs: Absent
Signs of meningeal irritation: Absent

Salient feature:

■Md. Mostofa kamal 35 yrs old Normotensive ,Nondiabetic, Nonalcoholic ,Smoker Muslim fisher man hailing from : Valuka, Mymensingh got admitted into MU-1 MMCH with complained of Nausea and anorexia for 10 days followed by Yellow coloration of skin, eye and urine for 7 days . The patient also complained of malaise, mild nonspecific headache and vomiting for several times in last few days but he had no history of fever with chills and rigor. The patient also complained generalized itching and fatigue. The color of stool was not pale .The patient has no history of weight loss, abdominal pain, loss of body hair , haematemesis, malaena, Alter level of consciousness and alteration of sleep pattern, joint pain and swelling. The patient also had no history of transfusion of blood, blood product and abdominal surgery previously. The patient used to shave in salon and was unaware about using of disposable blade every time. The patient's libido and urine out put is normal and bowel moves once daily. The patient had no previous history of jaundice, gall stone or liver disease. The patient has no history of hepato toxic drug intake and denied IV drug abuse or extramarital sexual relations. The patient is fully conscious and oriented. General examination reveals the patient have moderate jaundiced with scratching mark in all over the body. No evidence other stigmata of CLD like, leukonychia, spider nevi, gynaecomastia, edema palmer erythema, flapping tremor. The patient has no anaemia, Lymphadenopathy with normal pulse, BP, temperature and respiratory pattern and JVP not raised. systemic examination reveals Liver is enlarge which 3 cm from right costal margin in mid clavicular line ,tender having smooth surface , sharp margin (rounded), firm (or soft) in consistency , upper border of liver dullness is in right 5^{th} intercostals space with absent hepatic bruit and liver span is 15 cm. spleen ,Kidney and urinary bladder is not palpable. No intra abdominal lymph adenopathy, Shifting dullness and Fluid thrill absent .Testes are normal is size and consistency no engorged vein.

Provision diagnosis:

Acute viral hepatitis

Differential diagnosis

Liver abscess
Leptospirosis

Investigation

Liver function test

SGPT (ALT) : > 6 times
Alkaline phosphatase : normal or increased
S. Blirubin : increased
Prothrobin time : increased

Viral marker

HBs Ag
Anti HAV Ig M
Anti HEV Ig M
Anti HCV
USG of hepato billiary system

Treatment :

Treatment of viral hepatitis is mainly bed rest
Then supportive and symptomatic
Bed rest

Diet normal
If nausea and vomiting
Anti-emetic
Lactulose
Inj . konakion (10mg) vit-k 2
1 amp iv for 5 days if PT difference with control is more than 4 sec

What is ur provisional diagnosis? What r the point in favor of ur diagnosis
Acute viral hepatitis

Point in favor of diagnosis
Typical prodrome – nausea and vomiting, malaise, head ache
Jaundice, itching
Tender hepatomegaly

What is differential diagnosis?
Liver abscess
Leptospirosis

Point favor of u r DD
Only hepato megaly
(Fever—if present –but in this case absent**)**

The Point against of u r DD
Absent of the high fever (pyogenic abscess)/ fever
Absent of abdominal pain
Absent of toxicity in the patient
Presence of jaundice and prodrome of viral hepatitis

Why DD is leptospirosis
Point in favor
Fever and jaundice
Point disfavor
In leptospirosis
Fever is more marked than > prodome
Subconjunctival hemorrhage
Kidney involvement
increase S.creatinin ,
urine—RBC and cast , protein

Why this is not a case of CLD
Short History
In history and examination
No stigmata of CLD such as
- Leukonychia, spider nevi, gynaecomastia,
- Palmer erythama , ascites , engorged vein and
- Testicular atrophy , loss of body hair distribution
- Haematomesis and malena

Why this is not a case hemolytic jaundice?
In haemolytic anemia there is severe anemia and mild jaundice and splenomegaly ,haemolytic faces and history of repeated blood transfusion

Why this is not a case of obstructed jaundice?

- In obstructed jaundice, jaundice is more marked with pale color stool , intense itching Xanthelasma and xanthomas ,Steatorrhoea –which is absent in this case except itching
- On examination only tender hepatomegaly , no lump or palpable gall bladder

Define jaundice and classify and when jaundice is clinically detectable ?

Jaundice refers to the yellow coloration of the skin, sclerae and mucous membranes resulting from an increased bilirubin concentration in the body fluids.

Three type of jaundice A) prehepatic / hemolytic B) hepatocellular C) obstructive jaundice

It is usually detectable clinically when the plasma bilirubin exceeds 50 μmol/l (3 mg/dl)

What is the cause of jaundice in this patient?
Acute viral hepatitis

What are the viruses responsible for viral hepatitis?
CAUSES OF VIRAL HEPATITIS
Common
Hepatitis A
Hepatitis B ±
Hepatitis D
Hepatitis C
Hepatitis E
Less common
Cytomegalovirus
Epstein-Barr virus

What is the DNA virus?
Hepatitis B is the DNA virus rest are RNA virus

What are the features of viral hepatitis?
Non-specific prodromal features like headache, myalgia, arthralgia, nausea and anorexia usually precedes the development of jaundice by a few days to 2 weeks. Vomiting and diarrhoea may follow and abdominal discomfort is common. Dark urine and the liver is often tender but only minimally enlarged.

What are the routes of HAV / HEV transmission?
Oro faecal route

How hepatitis B and C transmit?
It transmit parentally via blood, sexually and vertical (from mother to child)

Which viral hepatitis have chronic phase?
Hepatitis B and C

How does hepatitis D spread along ?
The hepatitis D virus (HDV) is an RNA-defective virus which has no independent existence; it requires HBV for replication . it infected people only in presence of HBV infection

What are the complication of viral hepatitis?
In Most of viral hepatitis the fate is complete resolution .

In some case following complication may occur:
Acute liver failure.
Cholestatic hepatitis.
Aplastic anaemia.
Chronic liver disease and cirrhosis (hepatitis B and C).
Relapsing hepatitis .

Whar are the fate of acute viral hepatitis ?

Most of them spontaneous resolution
Plus above complication

What are the investigation u want to do in viral hepatitis?

Liver function test
SGPT (ALT)
Alkaline phosphatase
S. Blirubin
Prothrobin time
Viral marker
HBs Ag
Anti HAV Ig M
Anti HEV Ig M
Anti HCV
USG of hepato billiary system

What is the USG findings u may got in viral hepatitis?
Following are the USG finding in viral hepatitis
Shows liver Hypo echoic or
Inflammation of gall bladder (Chloe cystitis)or
Normal

what is the finding of SGPT and alkaline phosphatase in jaundice
In hepatocelluar jaundice
ALT > 6 times and Alkaline phospahtase < 2.5

In obstructive jaundice
Alkaline phospahtase > 2.5 and ALT < 6 times

How will u differentiate between acute and chronic viral hepatitis

Acute viral hepatitis	**Chronic viral hepatitis**
Clinical	
Prodrome present (nausea / vomiting /anorexia)	No prodome
Short HO < 1 month Jaundice present	Usually absent if present duration is > 3 month
No stigmata of CLD	Stigmata CLD present Ascites , splenomegaly , spider and gynaecomastia , testicular atrophy
Bio-chemical	
Prothrombin time increased	Prothrombin time increased in Acute on chronic
Albumin and A:G ratio normal	hypoAlbuminia and A:G ratio alter
Viral marker HBs Ag + < 6 months	Viral marker HBs Ag + > 6 months
Anti-HBC I_g G negative	Anti-HBC I_g G positive
Imaging (USG)	
Shows liver Hypo echoic Inflammation of gall bladder (Chloe cystitis) Normal	Coarse echo structure Ascites @ or Spleno-megaly

What are the causes of tender hepatomegaly?
Acute viral hepatitis
Liver abscess
Primary hepatocelular carcinoma
Congestive cardiac failure

Why liver become tender?
Liver is pain insensitive except its capsule. As in viral hepatitis liver is enlarge in very short time that it stretch the capsule and it become pain full.

What is the treatment of acute viral hepatitis/?
Treatment of viral hepatitis is mainly bed rest
Then supportive and symptomatic
Bed rest
Diet normal

Inj. 5%DA1000 ml / INJ. DNS 1000 ml if pt is nausea
I V @ v 20 drop / min//vomiting

Cap. omeprazole 20 mg
1 + 0 +1

Tab. omidone 10 mg
1+1+1

Syp. D-LUC
3 tsf tds
inj. konakion 10 mg
1 amp iv stat and daily for 5 days

How long Hepatitis A spread via stool?
The virus excrete in faeces for about 2-3 weeks before the onset of symptoms and then for a further 2 weeks.

Which one has more chance of turn in to chronic viral hepatitis between B @C? or which form of acute viral hepatic needs treatment ?
Acute B viral hepatitis no needs of treatment --- 95 % resolve spontaneously
Acute C viral hepatitis needs of treatment ----- 95 % turn into chronic

If patient comes to u with HBS Ag positive what will u do
wait for 6 months and tell him comes after 6 month
then do HBs Ag again if positive and it is either

- Carrier or --------no R_x is needed
- Chronic active viral hepatitis ---- R_x is needed

Criteria for carrier

1. HBs Ag positive > 6months
2. ALT normal
3. HBV-DNA level undetectable
4. Biopsy: minimal hepatitis (Knodal score)
5. anti- HBc (+)

if all these are positive than it is carrier

Do the following

- No treatment is needed
- Patient will do normal activity
- Stop smoking and alcohol consumption
  it aggravate CLD
- Do not donate blood
- Sexual partner should be vaccinated
- Breast feeding allowed

Chronic active hepatitis

1. HBs Ag positive > 6months
2. ALT > 2 times (persistent / intermittent)
3. HB_e Ag +
4. HBV-DNA level > 10^5 copies
5. Biopsy: moderate / bridging hepatitis
   (Knodal score)

in our subcontinent there is mutated virus so HB_e Ag may be negative . So negative HBe Ag Does not exclude the chronic active hepatitis so treatment give if other criteria fulfilled

Practically we see

HBs Ag positive > 6months
ALT > 2 times (persistent / intermittent) (60)
Give anti viral therapy

Single test for Chr. Viral hepatitis

- Anti-HBC I_g G positive

Dug use in HBV

- Interferons/ Pegylated interferons
- Adefovir
- Lamivudine

Interferon should not given compensate, if u give it in compensated CLD it may be turn into De-compensate.

Interferons/ Pegylated interferons

DOSE
Interferon standard
Sub cutaneous thrice weekly
Pegylated interferons
180 µmg Sub cutaneous wkly

Duration

If HBe Ag positive ---- 6 month
If HBe Ag negative ------ 1 years

Contractindication
Decompensated CLD
Thyroid anti body
Neuro-psychiatric manifestation
Pregnancy

Sign of decompensation

- Ascites
- Jaundice
- Encephalopathy

Side effect

- Flu like syndrome
- Alopecia
- Bone-marrow suppression
- Reversible Azospermia
- Neutropenia

Lamivudin
Oral and cheap and easily avail able

Dose
Tab . lamivir 100 mg
1 + 0 + 0 ----------before meal
In case of child 3 mg / kg body wt
Duration
At least one year

Can given in pregnancy
Can given in decompensated CLD

Chance of resistance so some body prefer to give With Adefovir with lamivudin

Tab. Adefovir 10 mg

1+ 0+ 0 --- given at morning ----1 year
Complication
Nephrotoxic
- Renal function monitor
- Advantage does not grow resistance
- Can use in Decompensate CLD

Goal of therapy
Short term goal
- ALT normal
- ↓ DNA level
- Sustained Seroconversion

Long term goal
- Prevent cirrhosis
- Prevent HCC

Seroconversion
If patient is HBe Ag positive developed Anti-HBe Ag then it is called Seroconversion
Sustained seroconversion
If anti- HBe Ag persist more than 6 month then it is called sustained seroconversion

If patient HBe Ag negative
In such patient seroconversion will be achieved when HBs Ag will be negative in the blood

A **patient comes to u with HBs Ag and want to go foreign ?**

Or patient with HBs Ag positive and want to make it negative ?

- No , He can not go to foreign as there is no chance of spontaneous Removal of **HBs Ag**(chance of spontaneous recovery 0.5% year)
- Interferon can clear the virus 10 % per year , no role of oral anti viral therapy
- Come to 6 month later and Repeat HBs Ag
- if positive then look for whether it carrier and chronic Active hepatitis

- Mother HBs Ag + chance to spread to baby is 90 %
- HBs Ag does not cross the placenta
- It spread peri natal via blood during delivery vis umbilical vein

A pregnant woman with HBs Ag +
- R_x usually not given in pregnancy
- During Anti natal check up see
  - Patient DNA level
- If DNA is increasing Rx may given
- Choice of drug is--- **Lamivudin**

If lady on interferone but recently become pregnant what will u do ?
- Stop interferon
- Switch on to **lamivudin**

What will u do A HBs Ag + mother give birth a child?
- With in 24 hr s of Delivery give Ig G
- Both active and passive immunization should be done after delivery

Wife of HBs Ag + husband

- Do HBs Ag of wife
- Immunized her if negative
- Until developing immunization (3—6 mon) pl. use barrier contraceptic method
- If she become positive do not immunized her .

Indication of Immunoglobin

- Infant born Of HBs Ag (+)mother
- After needle prick injury
- After sexual contact with HBs Ag (+) women

If doctors get needle is stick injury what Will u do?

- First see he is vaccinated or not
- If not give immunoglobulin (Ig G)
- Followed by active immunization
- If doctors is immunized then see triter Do accordingly level of triter

Which one is more infectious?

HBV > HIV

HBV > HCV

- **HBeAg----** indicates continued active replication of the virus in the liver .
- **Anti-HBe** ----implies that replication is occurring at a much lower level .

Patien is chronic viral hepatitis

HBs Ag + and Anti-HBC I_g G positive

A patient is vaccinated

HBs Ag -ve and Anti – HBS + ve

Level of vaccination

If triter is

< 10 ---non responder ---Double dose full immunization 10 --- 100 mild responder --Double dose single immuniz.

> 100 ------ full immunized --- no vaccine needed the patient is fully protective

Vaccine schedule inj. engerix-B

1 amp. IM on 0--- 1 ------6 month

Hepatitis B Virus –not cytopathic it act immunologically

HAV --- is not also cytopathic

HEV ---t is cytopathic (damage the liver cell)

HEV—it dangerous in old age

In late pregnancy –mortality high

Chance of fulminative hepatic failure is more jaundice more deeper and prolong than the acute HAV

Feature of acute hepatitis

Non-specific prodromal

- Headache,
- Myalgia, arthralgia,
- Nausea and anorexia usually begin few days to 2 weeks before development of jaundice
- Vomiting and diarrhoea may follow and abdominal discomfort is common.
- Dark urine and pale stools

PHYSICAL SIGNS.

- The liver is often tender but only minimally enlarged.
- Occasionally, mild splenomegaly and cervical lymphadenopathy are seen. (with Epstein-Barr virus infection.)
- Symptoms do not last more than 3-6 weeks.

Test for

- Hepatitis A --- Anti-HAV Ig M
- Hepatitis C --- Anti HCV and HCV RNA Hepatitis E – Anti HEV Ig M
- Only B @ C have chronic form only 5 -10 %
- Only 15 – 20 % of them develop complication , HCC , cirrhosis , hepatic decompensarion
- They spread in parental and sexual routes.
- Other have only faecoral routes
- HAV—can spread sexually in perverted (oro-anal sex)

Describe the metabolism of bilirubin

- Bilirubin in the blood is unconjugated and not water-soluble and bound to albumin and does not pass into the urine.
- RBC destruction →Unconjugated bilirubin →in liver conjugated by glucuronyl transferase→ into bilirubin mono- and diglucuronide→ These bilirubin conjugates are water-soluble →secret into the bile→ goes to colon → Conjugated bilirubin is metabolised by colonic bacteria to form stercobilinogen→ which oxidised to stercobilin→both stercobilinogen and stercobilin are then excreted in the stool →LA small amount of stercobilinogen (4 mg/day) is absorbed from the bowel→ passes through the liver →is excreted in the urine, where it is known as urobilinogen or, → oxidize in to urobilin.

CAUSES OF CHOLESTATIC JAUNDICE	
Intrahepatic	**Extrahepatic**
Primary biliary cirrhosis	Choledocholithiasis
Primary sclerosing cholangitis	Carcinoma
Alcohol	Ampullary
Drugs	Pancreatic
Viral hepatitis	Bile duct (cholangiocarcinoma)
Autoimmune hepatitis	Secondary
Hodgkin lymphoma	Parasitic infection
Pregnancy	Traumatic biliary strictures

What is the mechanism of jaundice viral hepatitis A, B, E ?

- HAV , HBV cause viral hepatitis by immunological mechanism
- HEV ---Here jaundice occur due to direct cell destruction –that why it is cytopathic

Why jaundice is more severe / deep acute viral hepatitis than CLD ?

In CLD jaundice less because here hepatocyte are fibrosed

A patient comes to you with deep jaundice for 1 ½ month what may be the cause ?

It may be due to hepatitis E virus

Counseling the patient with viral hepatitis due to HBV ?

- Informed the pt that it is an contagious diseases
- It spreads in parental route
- So not donate blood to any one
- Use barrier method of contraception and immunized his partner against
- There 10 % chance turn the viral hepatitis in chronic form
- so avoid alcohol and smoking
- Do regular activity and periodic liver function test to see whether it is turn in to chronic form or not

What follow up u will give in patient with viral hepatitis?

- Level of consciousness
- Flapping tremor
- Planter extensor
- Bowel passed and urine out put
- Any bleeding manifestation

What will u do A HBs Ag + mother give birth a child?

- Within 24 hr s of Delivery give Ig G
- Both active and passive immunization should be done after delivery

Pleural Effusion

PARTICULARS OF PATIENT:

Name: Md. Kamrul Hassan
Age: 50yrs
Sex: Male
Marital status: Married
Occupation: Farmer
Religion: Islam
Address: Fulbaria, Mymensingh
Date of admission:8.12.09 at 7pm
Date of examination:10.12.09 at 7.15am

PRESENTING COMPLAINTS

- Fever for 2 months
- Cough with sputum for 2 months
- Breathlessness for lasts15 days

HISTORY OF PRESENT ILLNESS

According to statement of the patient, he was reasonably well 2 months back then he developed low grade fever which used to rise at the evening, lasted for about six to eight hrs, which was not associated with chills and rigors but disappeared with sweating after taking Paracetamol(or spontaneously with out medication). Fever was not associated chest pain. He also complained of cough with sputum for 2 months. Sputum was mucoid, whitish, non foul smelling and not blood stained, was about ½ cup/day .Cough was not aggravated on exposure to dust, cold air or allergens and has no diurnal variation. For the last 15 days patient also complained of gradual development of mild breathlessness and heaviness of right chest which not related with exertion or lying position . On query, he had no history of contact with known TB patient or traveling into hilly area. There was no history of loose motion, joint pain, rash, headache or vomiting. He gave no history of increased frequency, urgency, hesitancy or red urine, vomiting out of blood, black tarry stool or alteration of bowel habit. The patient has no history voice change , swelling of neck and face , shoulder pain The patient lost about 1/5 his total body wt in last 6 month With the above complaints he got admitted into MU-1,MMCH for further evaluations. He also gave history aspiration of fluid from his right chest twice after admission in this hospital and color of the fluid was straw (hemorrhagic in malignancy).

HISTORY OF PAST ILLNESS

No history of DM/HTN/TB

No history of such type of illness before

PERSONAL HISTORY

Occasional smoker(10 – 12 Stick/day) for last 20 years

Non alcoholic. Betel nut chewer

FAMILY HISTORY

- His wife and children are healthy and not suffering from TB or such type of illness.
- His other family members are also healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition.and lived in Crowding house
Housing: Tin shade house.
32rooms, which accommodate 8 0f

his family member.
Sanitation:1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized against TB (Immunized against tuberculosis_)

Drug History

GENERAL EXAMINATION

- **Appearance –ill looking**
- Body built – average
- Nutritional status – below average
- Decubitus: on choice
- Co-operation : Well co-operative
- **Anaemia – absent**
- Jaundice : Absent
- Cyanosis : Absent
- **Clubbing : Absent**
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : Absent
- Dehydration – moderate dehydration
- Skin – pale , thin & wrinkled.
- Body hair distribution – total loss of axillary & pubic hair
- Bony tenderness – absent
- **Lymph node – no lymphadenopathy**
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 68/min, low volume, regular
- B.P – 80/40 mm of Hg
- **Respiratory rate – 25/min**
- **Temperature – 98^{0}F**
- Weight : 45 kg
- Height : 1.6 meter
- BMI : 17.57 kg/m2

Important general examination :
Appearance
Anaemia –malignancy
Clubbing : Absent
Lymph node – cervical lymphadenopathy
Respiratory rate
Temperature
if the patient have MT mark on hand then measure it mention in general examination

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Chest movement restricted in Rt lower part of chest

•**Palpation** :

•Trachea: Trachea shifted to left

Apex beat: in leftt 5th intercostal space 9 cm from midline&normal in character

Chest expansion: Diminished in rt lower part of chest & normal in other part

Vocal fremitus : Diminished from

Right 5th intercostal space to downwards along midclavicular line

Right 6th intercostal space to downwards along midaxillary line

Right 7th intercostal space to downwards along infrascapular line &

Normal in other part of the chest.

Percussion: Stony dull at above mentioned area & normal in other parts of the chest

Auscultation:

Breath sound absent at above mentioned area &vesicular in other parts of the chest.

No added sound

Vocal resonance: Diminished in above mentioned area & normal in other parts of the chest

ALIMENTARY SYSTEM

- **Inspection** :
- Mouth and oral cavity : Normal
- **Abdomen proper**

  Normal
- **Palpation** :

  Liver,Spleen , Kidney : Not palpable

  No intraabdominal lymphadenopathy or palpable lump
- **Percussion** :

  Tympanic
- **Auscultation** :

  Bowel sound present
- Testis : Normal
- D/R/E : Normal

CARDIVASCULAR SYSTEM

- Pulse : 72 b/min

  Regular, normal in volume & character, all the peripheral pulses are normal.
- BP : 120/75 mm of Hg
- JVP : Not raised
- **Precordium** :

  Inspection : Normal

  Palpation: Apex beat in lt 5th intercostal space 9 cm from midline

  AuscultationS1&S2 audible in all auscultatory area No added sound

NERVOUS SYSTEM

- **Higher psychic function including speech**: normal.

Fundoscopic exam: Normal

Cranial nerves: intact

- **Motor system examination**

  Both upper and lower limbs
- No muscle wasting or fasciculation
- Bulk of the muscle : normal

Tone of the muscle: normal

Power of the muscle: G-5

Sensory examination

All modalities of sensation are intact in both upper and lower limbs

Cerebellar signs: Absent

Signs of meningeal irritation: Absent

SALIENT FEATURE

Md. Kamrul Hassan aged 50 yrs old married muslim smoker normotensive nondiabetic farmer hailing from Fulbaria Mymensingh got admitted into MU-1 MMCH with low grade intermittent fever for 2 months which was not associated with chill and rigor and subside with sweating . He also complained of productive cough for 2 months with significant weight loss. He has no history of chest pain and haemoptysis .For the last 15 days he developed mild dyspnea and heaviness of right chest without any HO of orthropnea, exertion dyspnea and paroxysmal nocturnal dyspnea .On query, he had no history of contact with known TB patient or traveling into hilly area, alteration of bowel habit, joint pain, rash, headache or vomiting and urinary problem . The patient has no history voice change, swelling of neck and face , shoulder pain and any Para neoplastic syndrome.he also give history of aspiration of straw color pleural fluid twice after admission General examination reveals no anaemia jaundice, clubbing, edema , lymph-adenopathy and no

feature of superior vena cava obstruction . Examination of respiratory system reveals restricted Chest movement in Rt lower part Trachea: Central in position. Apex beat: normal ,Vocal fremitus : Diminished from Anteriorly Right 5th intercostal space to downwards along midclavicular line And laterally Right 6th intercostal space to downwards along midaxillary line Right 7th intercostal space to downwards along infrascapular line &Normal in other part of the chest. Percussion: Stony dull. Breath sound absent, and vocal resonance Diminished at above mentioned area.

PROVISIONAL DIAGNOSIS

Right sided Tubercular pleurla effusion

DIFFERENTIAL DIAGNOSIS

Right sided Pleurla effusion due to Bonchrogenic carcinoma (in old patient)

Investigation

FBC:

TC – 9800/mm3

DC –

neutrophil-64%
Lymphocyte-30%
monocyte-02%
Eosinophil-04%

ESR-75 mm in 1st hr Hb-09g/dl**.**

CXR P/A view : Right sided pleual efffusion

Sputum for AFB -----Negative

Sputum for malignant cell --- not found

Mantoux test : Induration 15 mm after 72 hrs

RBS 7 mmol / L

Pleural fluid study :

Appearance :

Straw

Biochemical :

Sugar : 30 mg/dl

Protein :6.6 gm/dl

Cytological examination

Lymphocyte predominant

Microbiological examination

Gm stain = -

AFB = -

Malignant cell

Not found

Pleural biopsy

What is ur provisional diagnosis

Tubercular pleurla effusion

What are the point in favour in ur diagnosis ?"

- Long HO (2 to months)
- Fever is low grade with evening rise temperature
- Weight loss
- TB is common in our country
- Fluid is Straw in color according to patients statement

Why not this is a case parapneumonic effusion ?
Because the patient had not the following feature

- High grade fever (days to week)
- Short HO
- Chest pain marked
- Patient is toxic

What is the deferential diagnosis of this patient and mention point favour and disfavour of D_x ?
Pleural effusion due bronchogenic carcinoma

Point in favor of diagnosis
Old age
Ho of cigarette smoking

Point in against of ur differential diagnosis

- No Clubbing , presence of fever (in malignancy fever is usually absent)
- Hoarseness of voice
- Cervical lymph adenopathy
- Feature of superior vena-cava obstruction
- No Horner syndrome
  Meiosis, Ipsilateral partial ptosis,Enophthalmos, anhydrosis
- No feature of Pancoast chest ((pain in the shoulder and inner arm)
- Fluid is straw color (if malignancy)

Why u called it pleural effusion ?

- Trachea shift to left side (only in massive effusion
- Percussion stony dull
- Vocal resonance and Fremitus --- decreased
- Breath sound ---- decrease

Q. when will U tell that media-stenium is shift?
Only when both trachea and apex beat is shifted

Q.**why trachea is not shifted to opposite site**?
Two reason:

- Pleural effusion was not passive
- Or due to aspiration of pleural effusion

Q. **Is it possible** right sided pleural effusion with trachea is shifted to right side?
Ans: yes possible if pleural effusion is associated with underlying collapse .

Why this not a case of consolidation ?
In Consolidation

- Trachea central and
- breath sound bronchial,
- Vocal resonance and Fremitus --- increased
- Percussion –woody dull
- cerps +

Why this not a case of fibrosis
Fibrosis

- Trachea same side and
- breath sound bronchial
- Vocal resonance and Fremitus --- increased
- wasting of over lying chest ,
- Rib crowding present (space between corresponding rib is decrease)

Why this not a case of collapse
If bronchus is patent

- Trachea same side
- Bronchial breath sound

If bronchus is not patent

- Trachea same side
- Breath sound diminish

Q. pleural effusion what type of TB is it pulmonary or extra pulmonary TB ?
Ans: It is extra pulmonary TB

How will u confirm the pleural effusion at bed side ?

By aspiration of fluid

How color of fluid help in Diagnosis ?

Color

- straw – TB
- Turbid / pus –pneumonia /empyma
- Hemorrhagic—ca bronchus
- Serous -- transudative

Q. mention the site of extra pulmonary TB ?
Ans:

- Intestine
- Bone
- Braine and meninges
- Skin
- Lymph node
- Pleura

Maximum aspiration per day is ?

1.5 L

- removing more than 1.5 litres in one episode is inadvisable as there is a small risk of re-expansion pulmonary oedema..

Describe the procedure of aspiration of pleural fluid ?

Amount of fluid

Pleural effusion is clinaclly detect if Fluid is

- 500 ml

Radiological detected in PA view if Fluid is

- 200 ml

Radiological detecte in Lateral view if Fluid is

- 100 ml

USG can detect as small amount Fluid

Investigation for Diagnosis pleural effusion ?

CBC -- Neutrophilic Leucocytosis in pneumonia

Normal CBC with persistent high ESR- in TB / CA

RBS

MT

Sputum for AFB and CXR PA

Pleural fluid aspiration and study for biochemical , cytology , malignant , microbiology

If suspect malignancy Do

- sputum for Malignant cell
- FNAC for lymphnode
- USG or CT guided FNAC
- Central lesion – bronchoscopy and biopsy

What do means by AFB?

AFB means acid fast bacilli .

Why M.Tuberculosis is called AFB ?

Because M. tuberculosis have mycolic acid in their cell wall . So they retain the primary stain (carbol fuchsin) after washing by acid –alkali . So it is called AFB

What are the finding of pleural fluid study

Pleural fluid study

Color

- straw – TB
- Turbid / pus –pneumonia /empyma
- Hemorrhagic—ca bronchus
- Serous -- transudative

Biochemical

See protein and glucose

- If protein more than > 3 gm Exudative

Cytology

See inflammatory cell

Neutrophil and lymphocyte

Malignant cell

- Only given
  - when suspected malignancy
  - Or hemorrhagic effusion .

What other investigation you want to do ?

I want to do pleural biopsy

How much sensitive is pleural biopsy?

In case of TB pleural biopsy is positive 80% case

In case of Malignancy biopsy is positive in 40 % case

Which type of needle use to do pleural biopsy?

Abraham needle or copes needle

Recently what is seen in pleural fluid to Dx TB?

Pleural fluid for ADA --ADA-Adenin De Aminase

- Otherwise not routinely given

Micro biological

GM stain and AFB stain

Practically valueless

TB

Exudative with lymphocyte predominant

Parapneumonic

Exudative with Neutrophil predominant

Malignant

Exudative with malignant cell present with hemorrhagic fluid

What stain is done to see malignant cell ?

How will u differentiate between hemorrhagic effusion from Traumatic haemorrhage **?**

In Hemorrhagic effusion

Does not clot and uniformed distribution

In Traumatic haemorrhage

Clot on the tube or on standing

LIGHT'S CRITERIA

Pleural fluid is an **exudate** if one or more of the following criteria are met:

- Pleural fluid protein:serum protein ratio > 0.5
- Pleural fluid LDH: serum LDH ratio > 0.6
- Pleural fluid LDH > two-thirds of the upper limit of normal serum LDH

Causes for an exudative pleural effusion:

Tuberculosis

Bronchogenic carcinoma ().

· Pneumonia.

· Pulmonary infarction.

· Lymphoma (in young individuals).

· Mesothelioma**.**

Connective tissue disease RA,SLE

Causes of a transudate:

· Nephrotic syndrome.

· Cardiac failure.

· Liver cell failure.

· Hypothyroidism**.**

What are the Radiological finding of TB in CXR

- Patchy opacity
- Pleural effusion
- Hilar lymphadenopathy- unilateral, paratracheal or mediastinal)
- Collapse
- Consolidation
- Cavitation / lung abscess

If you give the Rx of TB but no cured what r the cause ?

- Patient compliance
- Multi-drug resistance TB
- Wrong diagnosis (bronchogenic ca)

If TB patient does not take drug what will be the complication ?

He develop complication like fibrosis

M. TB. ?

When TB is resistant to both INH and Rifampicine

X-DRTB ?

Extreme drug resistant TB means resistance to INH and Rifampicine , fluroquinolone and at least one injectable drug

False negatives

- Severe TB (25% of cases negative)
- Newborn and elderly
- HIV (if CD4 count < 200 cells/ml)
- Recent infection (e.g. measles) or Immunisation
- Malnutrition
- Immunosuppressive drugs

U have to know the following about MT

MT test positive

when induration more than > 10 mm

It is not diagnostic test but supportive investigation

The test does not differentiate between

- TB infection
- TB disease
- BCG vaccination

Treatment of tubercular pleural effusion

CAT 1

Intensive phase

4FDC drugs such as ------- 2 month

RIEZ --

- Rifampicin
- Isoniazid
- Ethambutol
- Pyrazinamide

Continuation phase

2FDC drugs ----------4 month

RI

- Rifampicin
- Isoniazid

Weight	4 FDC (Rimstar 4 FDC)
30-37	2
38-54	3
55-70	4
>70	5

Weight	2 FDC
30-37	1 Rematazid 300
38-54	1 Rematazid 450
55-70	2 Rematazid 300

Rifampicin ------ 10 mg/kg, max 600 mg
INH-------- 5 mg/kg, max 300 mg
Ethambutol---- 15–20 mg/kg
Pyrazinamide----- 20–25 mg/kg, max 2 g
Streptomycin -----15 mg/kg daily,

Indication of CAT-1

- New smear-positive patient
- New smear negative PTB extensive parenchyma involve
- Extra pulmonary TB
- Meningeal ,
- Miliary
- Pericardial , Pleural effusion
- Spinal , Intestinal TB, dessiminiated TB

Indication of CAT 2

Should be given

- Relapse
- Treatment after interruption / default
- Treatment failure

In single word, if a patient get previously anti TB

Next time u have to give CAT--2

Indication of steroid in TB

- TB with Serosal involvement
  Pleural effusion
  Pericardial effusion
  Ascitis
- Tuberculous meningitis
- Genitourinary TB
- Endocrine TB (Addison)

Why steroid use in pleural effusion ?

- To prevent adhesion
- To early healing and absorption

Cat 2 to remember it 235
(2-streptomycin, 3- Remstar4 FDC, 5-Remactazid**)**
Intensive phase
First 2 months --- Inj.Streptomycin IM daily
First 3 months ----- Remistar FDC
Continuation phase
Next 5 months ---- Remactazid + Ethambutol

What adv. U will give a patient with on Anti-TB drugs

- Do not miss any does and take drugs regularly
- Ur urine ,saliva will turn into orange colour so do not affarid.
- Stop the drugs if patient develop Jaundice
- and seek for medical advice if patient develop visual disturbance ,

name some important complication of anti –TB chemotherapy

Isoniazid	Rifampicin	Pyrazinamide	Streptomycin	Ethambutol
Peripheral neuropathy[1]	**Hepatitis**	**Gout**	**8th nerve damage**	**Retrobulbar neuritis**

A patient developed jaundice after taking Anti TB drug?
what will u do ?
Do the following

- Stop the drugs immediately
- Do liver function test (SGPT and s.bilirubin)
- When test become normal or near to normal
- Strar anti TB drug in challenging dose
- Start with low dose single less hepato toxic drug
- Goes its optimum dose gradually and
- Start one by one drug and
- Finally give combination drug
- Due to unknown mechanism jaundice does not develop
- First give pyrazinamide ---Ethambutol ----INH---last Rifampicin
- Choice of drug is deffer from DR to DR

Tab. Pyrazinamide 500 mg
¼ + 0 +0----3days
1/3 + 0 +0
½ + 0 +0
1 + 0 +0
2 + 0 +0
Then
Ethambutol 400 mg
½ + 0 +0—1 days
1 + 0 +0---1days
2 + 0 +0 ---1 days
Then start such manner
INH @ rifampicin
Then goes to combination drug again

Different between active and passive TB

	ActiveTB	**Inactive TB**
Sputum	Positive	Negative
Symptoms	Equivocal	Equivocal
Creps	Marked	Less Marked
Radiology	Soft shadows Cavitation Serial extension pleural effusion	Calcification Tracheal shift Hilar elevation Diaphragm tenting Change of fissure

Pregnancy
All drugs are safe in pregnancy
Except in streptomycin
Which is Ototoxic to fetus

Breast feeding
No contractindication . baby should be breast feed

A patient came to u with the compliant of dimness of vision after taking anti TB

----------Ethambutol

A patient came to u with the compliant of burning sensation after taking anti TB
----------Isoniazid
A patient came to u with complaint of joint pain after taking ant-TB
----------Pyrazinamide

QName some condition where TB patient comes with emergency?

- Tubercular meningitis
- Cord compression
- Pericardial temponad

When will tell open TB?

Ans: Sputum for AFB is positive

If the cause is bronchogic following question may be asked :

Classify bronchogenic carcinoma :
Small cell carcinoma: 20 %
Non small cell carcinoma :80%
- Squamous cell carcinoma 35%
- Adenocarcinoma 30%
- Large cell carcinoma 15%

What will u look in general examination in patient with Br.Ca?

- Clubbing
- Cervical lymphadenopathy
- Feature of superior venacava obstruction
- Feature of Honers syndrome (ptosis , enophthalmus , meosis , anhydrosis)

If patient have clubbing in brochogenic carcinoma what will do and why?
I will press over just below to wrist joint to see the tenderness of lower radius and ulna to see the hypertrophy of osteo-arthropathy.
It occur due to sub-periosteal new born formation

What r the paraneoplastic syndrome will u search for in pt with bronchogenic carcinoma ?

Para-Neoplastic Syndrome	
SIADH **ACTH** **Carcinoid**	**Small cell carcinoma**
Hypercalcaemia	**Squamous cell carcinoma**
Gynaecomastia	**large cell**

Neurological • **polyneuropathy** • **MND** **MSK** • **polymyocitis** • **myasthenia gravis** • **Clubbing with hypertrophy osteoarthropathy** **Nephrotic syndrome**	**non small cell**

What investigation will u do in patient with suspected bronchogenic carcinoma ?

- **CBC----ESR…increased**
- **Sputum for AFB …(to exclude TB)**
- **Sputum for malignant cell …….**
- **CXR—PA**
- **FNAC or biopsy cervical lymphnode**
- **If central lesions ---bronchoscopy and biopsy**
- **----bronchial brushing**
- **If peripheral lesion ----CT—guided or USG FNAC for suspected lesion**

Treatment:

- **Surgery**
- **Radiotherapy—squamous cell carcinoma radio--sensitive**
- **Chemotherapy---small cell carcinoma chemo-sensitive**

Which carcinoma is more dangerous ?

- **Small cell carcinoma**

What drug use in chemotherapy?

CVD or CE

C—cyclophosphamide **V—vencristin** **D—Doxorubocin**	**C—Cisplatin** **E---Etoposide**

If the patient is young or middle age

- Then provisional diagnosis is right sided tubercular pleural effusion
- Then dd will be para-pneumonic pleural effusion

If patient is old and smoker then

- Diagnosis will be such right sided tubercular pleural effusion if no feature of malignancy found (such as lymph node, clubbing)
- Then dd will be pleural effusion due to bronchogenic carcinoma

Following history may be taken in patient of pleural effusion:

Fever

Duration of	long TB , short pneumonia
High grade or low grade	high—pneumonia , low grad—TB
Chills and rigor travel to hilly area	malaria
Rise with shivering or subsides with sweating	DO
Chest pain	pneumonia

Cough

Duration , productive or not
sputum mucoid , color , foul smelling and non foul smelling , blood stained or not , amount such as ½ cup per day
Cough --aggravated or not exposure to dust, cold air or allergens and Diurnal variation

Other History

Contact with known TB patient	
joint pain, rash,–viral ,	connective tissue disease
History of loose motion, alteration of bowel habit	abdominal TB
urinary problem	to exclude UTI
headache or vomiting , malaise	to exclude viral causes
history of wt lost in last 6 month	TB
history of aspiration	how many times and color
socio-economic condition , house overcrowding , less ventilated	
immunization against TB	

History to exclude bronchogenic carcinoma:

voice change , ,	recurrent laryngeal nerve palsy
swelling of neck and face	superior vena cava obstruction
shoulder pain	pancoast tumor
smoking for how long and how many stick per day	

Classify pneumonia?
Community acquired pneumonia Hospital acquired pneumonia Suppurative pneumonia Pneumonia in the immunocompromised
Define hospital acquired pneumonia? Name the organism responsible for HAP? Treatment of HAP ?

New episode of pneumonia occurring at least 2 days after admission to hospital
if **occur within 4-5 days**—organism of CAP
if after 5 day

- Gram-negative bacteria (e.g. Escherichia, pseudomonas and klebsiella species),
- Staph. Aureus (including meticillin-resistant staph. Aureus (MRSA)) and
- Anaerobes.

Difference between viral and bacterial pneumonia?

	viral pneumonia	**Bacterial pneumonia**
Onset	less abrupt ,H/O RTI	acute or abrup onset
Cough	Dry	Productive
Pain	uncommon	common
CXR	normal	feature of consolidation
CBC	Leucopenia	leucocytosis

What do mean by atypical pneumonia? Rx

pneumonia causes by atypical organism like (to remember CML)
C--Chlamydia pneumonia, M--Mycoplasma pneumonia, L--Legionella pneumophila

Causes of delayed resolution of pneumonia? A patient getting treatment of pneumonia but not improving ?

1. inappropriate chemotherapy
    a. incomplete course / sub optimal dose/ wrong anti-biotics
    b. non-compliance / drug resistance
2. developed complication –pl.effusion or empyema
3. wrong diagnosis—TB, bronchial carcinoma
4. if the patient is immunesupressed
5. if pneumonia occur by atypical organism

Difference between pleura thickening and pleural effusion?

pleural effusion	thicken pleura
short HO **Inspection –** intercostals space normal **palpation** trachea and apex beat shifted **percussion** stony dull **Auscultation** absent	**long HO** **Inspection –** flattening of chest , and depressed intercostals space , rib crowding -/+ **palpation** trachea and apex beat not shifted **percussion** dull **Auscultation** diminish

Extra questions for TB

Name new diagnostic test in TB?		
ADA—in fluid –such as pleura l , ascites , CSF.(> 40 u/dl) IGRA—interferone gama release assay ALS		
Difference between primary tB and secondary tB ANGEL , HIS		
	primary TB	**secondary TB**
age	child	adult
Nature	first infection	reactivation or re-infection
ghon focus	present	absent
erythema nodosum	present	absent
Lymphnode	involved (hilar)	not involved
Healing	spontaneous healing in 95% case by calcification	healing by fibrosis
immunity	not developed / uncommon	common or developed
site	lower part of upper lobe or upper part of lower lobe (subpleural region)	apical / apex (post segment of upper lobe) apical segment of lower lobe
miliary TB	occur	doesn't occur

Treat of MDR TB? what are causes of MDR TB
initial phase or intensive phase : **COPE__K---6 to 8 months** **C**—Cycloserine **O**—Ofloxacine **P**—Pyrazinamide **E**— Ethionamide **K**—(Inject able drug)—Kanamycin continuation phase **: COPE**---16 to18 months **C**—Cycloserine **O**—Ofloxacine **P**—Pyrazinamide **E**—Ethionamide l
Indication of paracentesis in pleural effusion ?
1. diagnostic purpose (only 50 ml) 2. therapeutic : a. respiratory distress b. massive collection c. rapid collection d. if suspected secondary infection
What do u mean by refractory pleural effusion?

Treatment of refractory pleural effusion? Malignant pleural effusion treatment?

Treatment of refractory pleural effusion **is Pleurodesis**
this can be achieved by
Chemical pleurodosis→by give Inj. Tetracycline, Kaolin or Talc via IT tube
Surgical pleurodosis→ pleural abrasion or parietal pleurectomy
In case of malignancy pleurodosis is done by injecting --bleomycin

Pleural effusion with lymphadeno pathy ?

CLAST
C—carcinoma (bronchial carcinoma)
L—lymphoma
A—acute leukaemia
S—SLE
T—TB

Complication of pleural effusion?

1. thicken pleura
2. empyema thoracis
3. hydro-pneumothorax
4. if long standing –collapsed lung may turn into fibrosis

haemorrhagic pleural effusion ?

haemorrhagic pleural effusion
to remember **CML—TIPS**

C—carcinoma (bronchial carcinoma) M—mesothelioma (pleural mesothelioma) L—lymphoma (lymphoma is common in young malignant pleural effusion)	T--trauma I—Infarction (pulmonary infarction) P-pancreatitis S---SLE

recurrent pleural effusion ?

to remember CML—TS (like that of hemorrhagic just "IP" delete from TIPS and T= transudative)

C—carcinoma (bronchial carcinoma) M—mesothelioma (pleural mesothelioma) L—lymphoma	T—transudative causes 1. Heart--CCF 2. Liver--cirrhosis of liver 3. Kidney --nephrotic syndrome 4. GIT--malabsorption / malnutrition / protein losing enteropathy S---SLE / RA

Causes of left sided pleural effusion?

Dr. READ
Dr—Dressler syndrome
R—rheumatoid arthritis
E—Esophageal rupture
A—acute pancreatitis
D—Dissecting aneurysm

Bilateral pleural effusion?

TO remember
LIST
L—Lymphoma

I—infarction (pulmonary infarction)
S—SLE
T— all Transudative causes (first mention this cause to examiner) (to remember 4 system heart , liver, kidney , GIT)

1. Heart--CCF
2. Liver--cirrhosis of liver
3. Kidney --nephrotic syndrome
4. GIT--malabsorption / malnutrition / protein losing enteropathy

chylothorax?
injury / obstruction of thoracic duct by any cause. Such as **LITON** L—Lymphoma I—infection –TB , filariasis T—traumatic injury (during surgery / trauma) O—obstruction of thoracic duct N—neoplasm (bronchial Ca / metastasis)
Case definition? New case , relapse , defaulter
What is DOTS?
Risk factor bronchial carcinoma?
Complication of plural fluid aspiration?
hydropneumothorax secondary infection → empyema subcutaneous emphysema and pleural shock
causes of empyema ?
BREAST B—bronchiectasis R—Rupture of liver/ subphrenic abscess E—effusion –complication of parapneumonic effusion (/ pneumonia) A—Abscess –lung abscess S—secondary infection –mainly due aspiration T—-TB

exudative	tarnsudative
common causes **MP3** 1. Malignant disease a. bronchial carcinoma old b. lymphoma --young 2. Pulmonary Tuberculosis 3. Pneumonia ('parapneumonic effusion') 4. Pulmonary infarction* **uncommon --MCPS** M—mesothelioma (pleural mesothelioma) C—connective tissue disease a) SLE b) RA P—pancreatitis S—subdiaphramitic a) subphrenic abscess b) liver abscess **other** **dressler syndrome** **(**Post-myocardial infarctionsyndrome)	**common causes :** to remember 4 system heart , liver, kidney , GIT 1. Heart--CCF 2. Liver--cirrhosis of liver 3. Kidney --nephrotic syndrome / CKD 4. GIT--malabsorption / malnutrition / protein losing enteropathy **uncommon causes :** **CMH** **C**—constrictive pericarditis **M**—Meigs syndrome --ovarian tumor + rt sided effusion **H**—Hypothyroidism / Myxoedema
female, nonsmoker , old scar -→ which bronchial carcinoma occure?	
adenocarcinoma	

Pneumonia

PARTICULARS OF PATIENT:

Name: Md. Mofazzol Hossain
Age: 35yrs
Sex: Male
Marital status: Married
Occupation: Farmer
Religion: Islam
Address: Telegram,Fulbaria, Mymensingh
Date of admission: 18.12.09 at 7pm
Date of examination: 18.12.09 at 7.15pm

PRESENTING COMPLAINTS

- Fever for 7days
- Cough with sputum 7days
- Chest pain for 7days

HISTORY OF PRESENT ILLNESS

- According to statement of the patient, he was reasonably well 7 days back then he developed high grade fever which persisted most of the time of days and was not associated with chills and rigors but disappeared with sweating only after taking Paracetamol.the heighst recorded temperature was 104 .the Fever was associated with Rt lower chest pain for last 7 days which was sharp, increased by deep breathing and coughing without radiation and breathlessness. He also complained of cough with scanty sputum for 7. Sputum was mucoid, whitish, non foul smelling and not blood stained, was about ¼ cup/day .Cough was not aggravated on exposure to dust, cold air or allergens and has no diurnal variation. On query, he had no history of contact with known TB patient or traveling into hilly area. There was no history of loose motion, joint pain, rash, headache or vomiting. He gave no history of freqency,urgency,hesitancy or red urine, vomiting alteration of bowel habit.The patient has no history voice change , swelling of neck and face , shoulder pain The patient had no history of weight loss . With the above complaints he got admitted into MU-1,MMCH for further evaluations. He also gave history aspiration of fluid from his right chest twice after admission in this hospital and color of the fluid was hazy (hemorrhagic in malignancy)

HISTORY OF PAST ILLNESS

No history of DM/HTN/TB

No history of such type of illlness before

PERSONAL HISTORY

Occasional smoker (3 –4 Sticks/day) for last 20 years

Non alcoholic. Betel nut chewer

FAMILY HISTORY

- His wife and children are healthy and not suffering from TB or such type of illness.
- His other family members are also healthy and enjoining sound health

SOCIOECONOMIC CONDITION

He comes from low socioeconomic condition. and lived in Crowding house
Housing: Tin shade house.

3rooms, which accommodate 8 0f his family member.
Sanitation:1 sanitary latrine.
Water supply : Arsenic free tube well water

Immunization history

The patient was not immunized against TB (Immunized against tuberculosis_)

Drug History

GENERAL EXAMINATION

- Appearance –ill looking
- Body built – average
- Nutritional status – below average
- Decubitus: on choice
- Co-operation : Well co-operative
- Anaemia – absent
- Jaundice : Absent
- Cyanosis : Absent
- Clubbing : Absent
- Koilonychia : Absent
- Leuconychia : Absent
- Oedema : Absent
- Dehydration – moderate dehydration
- Skin – pale , thin & wrinkled.
- Body hair distribution – Total loss of axillary & pubic hair
- Bony tenderness – absent
- Lymph node – no lymphadenopathy
- Thyroid gland – not palpable
- Neck vein – not engorged
- Pulse – 68/min, low volume, regular
- B.P – 80/40 mm of Hg
- Respiratory rate – 28/min
- Temperature – 102^0 F
- Weight : 45 kg
- Height : meter
- BMI : kg/m2

RESPIRATORY SYSTEM

•**Inspection** : Size and shape of the chest : Normal

•Chest movement restricted in Rt lower part of chest

•**Palpation** :

•Trachea: Central in position.

Apex beat: in leftt 5th intercostal space 9 cm from midline&normal in character

Chest expansion: Diminished in rt lower part of chest & normal in other part

Vocal fremitus : Diminished from

Right 5th intercostal space to downwards along midclavicular line

Right 6th intercostal space to downwards along midaxillary line

Right 7th intercostal space to downwards along infrascapular line &

Normal in other part of the chest.

Percussion: Stony dull at above mentioned area & normal in other parts of the chest

Auscultation:

Breath sound absent at above mentioned area &vesicular in other parts of the chest.

No added sound

Vocal resonance : Diminished in above mentioned area & normal in other parts of the chest

ALIMENTARY SYSTEM

- **Inspection** :
- Mouth and oral cavity : Normal
- **Abdomen proper**

  Normal
- **Palpation** :

  Liver,Spleen , Kidney : Not palpable
  No intraabdominal lymphadenopathy or palpable lump
- **Percussion** :

  Tympanic
- **Auscultation** :

  Bowel sound present
- Testis : Normal
- D/R/E : Normal

CARDIVASCULAR SYSTEM

- Pulse : 72 b/min

  Regular, normal in volume & character, all the peripheral pulses are normal.
- BP : 120/75 mm of Hg
- JVP : Not raised
- **Precordium** :

  Inspection : Normal
  Palpation: Apex beat in lt 5th intercostal space 9 cm from midline
  AuscultationS1&S2 audible in all auscultatory area No added sound

NERVOUS SYSTEM

- **Higher psychic function including speech**: normal.

Fundoscopic exam: Normal
Cranial nerves: intact

- **Motor system examination**

  Both upper and lower limbs
- No muscle wasting or fasciculation
- Bulk of the muscle : normal

Tone of the muscle: normal
Power of the muscle: G-5
Sensory examination
All modalities of sensation are intact in both upper and lower limbs
Cerebellar signs: Absent
Signs of meningeal irritation: Absent

SALIENT FEATURE

Md. Mofazzol Hossain aged 35 yrs married muslim smoker normotensive nondiabetic farmer hailing from Fulbaria Mymensingh got admitted into MU-1 MMCH with developed high grade fever which persisted most of the time of days and was not associated with chills and rigors but disappeared with sweating only after taking Paracetamol.the heighst recorded temperature was 104 .the Fever was associated with Rt lower chest pain for last 7 days which was sharp, increased by deep breathing and coughing without radiation and breathlessness. He also complained of cough with scanty sputum for 7. Sputum was mucoid, whitish, non foul smelling and not blood stained, was about ¼ cup/day .Cough was not aggravated on exposure to dust,

cold air or allergens and has no diurnal variation. He had no history of contact with known TB patient or traveling into hilly area, alteration of bowel habit, joint pain, rash, headache or vomiting and urinary problem . The patient has no significant weight loss .The patient has no history voice change, swelling of neck and face , shoulder pain and any Para neoplastic syndrome.he also give history of aspiration of turbid or hazy color pleural fluid twice after admission General examination reveals no anaemia jaundice, clubbing, edema , lymph-adenopathy and no feature of superior vena cava obstruction temp. is 102 ^{0}. Examination of respiratory system reveals restricted Chest movement in Rt lower part Trachea: Central in position. Apex beat: normal ,Vocal fremitus : Diminished from Anteriorly Right 5th intercostal space to downwards along midclavicular line And laterally Right 6th intercostal space to downwards along midaxillary line Right 7th intercostal space to downwards along infrascapular line &Normal in other part of the chest. Percussion: Stony dull. Breath sound absent, and Vocal resonance Diminished at above mentioned area.

PROVISIONAL DIAGNOSIS

Right sided Para pneumonic pleurla effusion

DIFFERENTIAL DIAGNOSIS

Right sided Pleurla effusion due to pulmonary TB

INVESTIGATION

FBC:

TC – 15,800/mm3

DC –

neutrophil-84%
Lymphocyte-10%
monocyte-02%
Eosinophil-04%

ESR-75 mm in 1st hr Hb-09g/dl**.**

CXR P/A view : Right sided pleual efffusion

Sputum for AFB -----Negative

Sputum for malignant cell --- not found

Mantoux test : Induration 05 mm after 72 hrs

RBS 7 mmol / L

Pleural fluid study:

Appearance:

Turbid or hazy

Biochemical:

Sugar: 30 mg/dl
Protein :6.6 gm/dl

Cytological examination

Neutrophil predominant

Microbiological examination

Gm stain = -
AFB = -

Malignant cell

Not found

What is ur provisional diagnosis?

Para pneumonic effusion

Because the patient had the following feature

- High grade fever (days to week)
- Short HO

- Chest pain marked
- Patient is toxic
- No history of weight loss
- Color of fluid is not straw

PLEASE SEE HISTORY FILES OF PLUERAL EFFUSION DUE TO TB OTHER QUESTION

A patient with pneumonia fever not subsides after taking antibiotic? What is the underlying cause?

- D_x may be wrong (it may be TB , CA)
- Inadequate dose or wrong drug , not taking drug
- Complication has been developed (emphysema)

Complication of pneumonia
common

- Para-pneumonic effusion-
- Empyema-
- Pneumothorax- *Staph. Aureus*
- lung abscess

Uncommon

- ARDS, renal failure
- Hepatitis,
- Pericarditis,myocarditis,
- Meningoencephalitis

Common organism in CAP – **MSC in low**

- **S**--Streptococcus pneumoniae
- **C---**Chlamydia pneumoniae
- **M**---Mycoplasma pneumoniae
- **Law**--Legionella pneumophila

Less common to remember **HSC**

- **H--**Haemophilus influenzae
- **S—**Staphylococcus aureus
- **C --**Chlamydia psittaci

What due u mean by CURB -65

C---Confused patient
U--- urea > 7mmol/l
R----respiratory rate >30/MIN
B--- BP systolic <90, diastolic <.60
65—Age more than 65

1 point for each feature present

If score
0/1 – home treatment
2—short hospital stay R_x
3-- R_x hospital as severe pneumonia
4/5—ICU support needed .

ANTIBIOTIC TREATMENT FOR
COMMUNITY-ACQUIRED PNEUMONIA (CAP)

Uncomplicated CAP

Amoxicillin 500 mg 8-hourly orally
If patient is **allergic** to penicillin
Clarithromycin 500 mg 12-hourly orally or
Erythromycin 500 mg 6-hourly orally
If **Staphylococcus is cultured** or suspected
Flucloxacillin 1-2 g 6-hourly i.v. plus
Clarithromycin 500 mg 12-hourly i.v.
If Mycoplasma or Legionella is suspected
Clarithromycin 500 mg 12-hourly orally or i.v. or
Erythromycin 500 mg 6-hourly orally or i.v. plus
Rifampicin 600 mg 12-hourly i.v. in severe cases

severe CAP

Clarithromycin 500 mg 12-hourly i.v. o
Erythromycin 500 mg 6-hourly i.v. plus
Co-amoxiclav 1.2 g 8-hourly i.v. or
Ceftriaxone 1-2 g daily i.v. or
Cefuroxime 1.5 g 8-hourly i.v. or

Pleura effusion due to pneumonia:

Classify pneumonia?

Community acquired pneumonia
Hospital acquired pneumonia
Suppurative pneumonia
Pneumonia in the immunocompromised

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    a. incomplete course / sub optimal dose/ wrong anti-biotics
    b. non-compliance / drug resistance
7. developed complication –pl.effusion or empyema
8. wrong diagnosis—TB, bronchial carcinoma
9. if the patient is immunesupressed
10. if pneumonia occur by atypical organism

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pleural effusion	thicken pleura
short HO **Inspection –** intercostals space normal **palpation** trachea and apex beat shifted **percussion** stony dull **Auscultation** absent	**long HO** **Inspection –** flattening of chest , and depressed intercostals space , rib crowding -/+ **palpation** trachea and apex beat not shifted **percussion** dull **Auscultation** diminish