A 25yr old woman known to have type 1 DM and adherent to her medications is admitted with fever, polyuria, polydipsia semi concious and with Kussmaul's breathing.

a) List the investigations you would do and the reason for each.

RBS - to check for blood glucose levels; assess hyperglycemia.

HbA1C - a marker for response to therapy / treatment.

Urinalysis - glycosuria, ketones +++, albuminuria / proteinuria.

U/E/Cs - Electrolyte derangements: hypokalemia, hypophosphatemia, hypomagnesemia, hypocalcemia??

Urinary function

BGAs - Anion gap, low blood pH.

Lactic acidosis: Compensated vs decompensated metabolic acidosis.

FHG - Evidence of infection; neutrophilia

Anemia

Albumin levels :- Albumin : creatinine ratio for evidence of proteinuria.

Fundoscopy - Evidence of retinopathy; Glaucoma

ECG - Cardiovascular sequale of DM, HTN (Risk factors for DM)

Blood toxicology - commonly abused drugs; cocaine, alcohol.

b) How would you manage her?

Check for airway patency and adequacy of breathing.

Ensure adequate circulation and expose the patient adequately.

Admit to ICU and for monitoring.

Assume fluid deficit of 6-8 litres. Give IV fluids N/S

1st litre - 1st half hour

1 litre - next 1 hr.

1 litre - next 2 hrs.

Maintenance: Then 0.45% saline at 150-300mls / kg / hr

when RBS < 14 mmol/L alternate 0.45% saline with 5% dextrose at 100-200 mls / hr.

Question 2.

A 32 year old man known to have rheumatic heart disease presents with shortness of breath on exertion, leg swelling and cough. HF AF
He is tachypnoeic, has a raised JVP and an irregularly irregular pulse.

a) What investigations would you do on this patient.

- Full blood count
- urinalysis
- Arterial blood gases
- U/E/Cr
- BNP
- liver function tests
- SPO_2
- ECG ⇒ To assess cardiac rhythm, and rule out causes of heart failure such as myocardial infarction, LV/RV hypertrophy

- Chest x ray ⇒ To assess cardiac size and shape, and pulmonary vasculature. evidence of HF includes cardiomegaly, peribronchial cuffing, pleural effusion, prominent kerley 'B' lines, dilated upper lobe vessels, fluid in the alveoli
- Echocardiography ⇒ Assessment of cause (Valvular heart disease), left ventricular size and function

b) List the possible modalities of his management and their usefulness in his management.

~~Sit patient upright in bed (45°)~~
~~Give oxygen (100%) if oxygen saturation is low.~~

- General measures

Counselling the patient and family. it improves adherence to treatment plan
Dietary measures ⇒ reduce salt in take, fluid intake, stop smoking and no alcohol.
Physical activity and weight reduction.

- Pharmacological management

- Diuretics for relief of fluid retention
- ACE-I eg captopril, enalapril - improves symptoms and reduces hospitalization

beta blockers e.g carvedilol, metoprolol improves symptoms, left ventricular function and lowers hospitalisation and decrease mortality

ARBs → if ACE-I are not tolerated, same efficacy as ACE-I

Aldosterone antagonists e.g spironolactone. It reduces mortality and when added to conventional therapy in those still symptomatic

Cardiac glycosides e.g Digoxin, digitoxin particularly useful in patients with atrial fibrillation.

Vasodilators e.g hydrallazine Improves survival rate when added to standard therapy especially black patients

Anticoagulants patients with A. fib are at risk of VTE Given as prophylaxis

Surgical intervention

Correction of underlying pathology; for this case valve replacement / valvuloplasty

Cardiac transplantation

I. MED QN

#3. A 40year Sld man presents with hematemesis.

(a) List the differential diagnosis:

- Peptic ulcer / Duodenal ulcer
- Esophageal varices
- Mallory-Weiss tear
- Gastritis / Gastric erosions
- Drugs eg. NSAIDs, Aspirin, Anticoagulants, Thrombolytics
- Esophagitis
- Duodenitis
- Malignancy
- Bleeding disorder
- Angiodysplasia

(b) He is found to have splenomegaly, which is thought to be related to his presentation.
What other investigations should be done to ascertain the diagnosis & the primary problem?

- Thorough history. Ask about:
  - ✓ Previous GI bleeds
  - ✓ Dyspepsia or known ulcers
  - ✓ Known liver disease
  - ✓ Alcohol use
  - ✓ Chronic NSAID use or anticoagulants
  - ✓ Dysphagia, weight loss, fever (Constitutional Sx)
  - ✓ Color of stool — Black, tarry stool Etc
  - ✓ Residence / work near water bodies (Schistosomiasis)
- Examination:
  - ✓ Any stigmata of chronic liver disease eg. Jaundice, ascites, palmar erythema, leuconychia caput medusae etc.
  - ✓ Assess signs of shock
  - ✓ Per rectal exam for melena stool or bright red

blood per rectum (hematochezia)
✓ Hepatomegaly

- Initial laboratory tests:
  - ✓ FBC - ↓Hb, ↓Plt, ↑WBC
  - ✓ Triple serology (HIV/HBV/HCV)
  - ✓ UECr - ↑UE
  - ✓ Stool for M/C/S & Ova + Cyst
  - ✓ LFTs = ↑
  - ✓ INR = ↑
  - ✓ GxM - In case blood transfusion is necessary
- Imaging - Abdominal ultrasound
- Diagnostic test:
  - ✓ Upper GI endoscopy (Diagnostic & therapeutic)

(c) If stool examination establishes the primary problem, what is the likely problem and how would you manage it?

Stool examination could reveal blood in stool (melena), which is a sign of upper GI bleed. It could also reveal frank blood in stool that could be due to bleeding anorectal varices. Ova and cyst stool examination may also reveal Schistosoma mansoni eggs, which may be the primary problem.

Schistosoma mansoni cause periportal fibrosis that leads to portal hypertension, which may present as ena + hematemesis due to bleeding varices.
ol

Treatment of schistosoma mansoni is with Praziquantel 20mg/kg PO TID every 4-6hrs x 1 day.

Treatment of bleeding varices is ABC and fluid/blood resuscitation till patient is stable. Urgent endoscopy is done to diagnose and treat bleeding esophageal varices via banding ligation.

INTERNAL MEDICINE:

(5) Write short notes on typhoid fever under the following sub-headings:

(a) Pathology
(b) Clinical Presentation.
(c) Diagnostic workup.
(d) Complications.

(a) Pathology:

- Salmonella possesses cellular mechanisms enabling bacterial proteins to be transferred to enterocytes and M cells with subsequent growth within endosomes.
- This is followed by: inflammatory response with neutrophil recruitment & mucosal damage.
- Bacteria are phagocytosed by histiocytes in underlying lymphoid tissue.
- The bacteria proliferate and widely disseminate through blood vessels and lymphatic channels.

(b) Clinical Presentation:

- Causes symptoms shortly after bacteria are ingested.
- Patient presents with:
  - → Severe abdominal pain
  - → Bloody diarrhoea.
  - → Anorexia.
  - → Nausea & Vomiting.
  - → Headache.
  - → High-grade fever

(c) Diagnostic workup:

- FBC with WBC differential (elevated).
- ESR (elevated).
- Blood (dual culture.
- Salmonella antibodies/antigen with ELISA (must be supported by cultures/DNA evidence.
- PCR for.

(d) Complications:

- Extraintestinal complications:
  - → CNS disease: meningitis → Osteomyelitis.
  - → Endocarditis. → Disease relapse.
  - → Myocarditis.
  - → Pneumonia.
  - → Cholecystitis.
- Intestinal perforation with peritonitis.

IRON DEFICIENCY ANEMIA

i) Discuss the causes of IDA

a) Insufficient intake - dietary, Upper GI obstruction, loss of appetite, low social economic status, Eating disorder

b) Lack of absorbtion - malabsorbtion syndrome, chronic diarrhea (DM +IBS associated), therapy with antacids, bowel resection

c) Blood loss - hookworms, menorrhagia, malignancies, GI bleeds

ii) Tests done to determine IDA

Peripheral blood film → anisocytosis, poikilocytosis

MCV, MCH, MCHC → All reduced

Serum iron levels → Reduced

Serum ferritin → reduced

TIBC → Increased

Transferrin → Increased

Iron staining in marrow → Absent

Reticulocytes → Normal or reduced.

IMED REVISION

Briefly Discuss the differential diagnoses, investigations of a 45 y.o HIV +ve woman presenting with severe headache.
How would you manage her if your investigations show a positive CRAG in CSF ??

=> Discussion: Cryptococcosis is caused by a yeast-like fungus, C. neoformans. It has a worldwide distribution and appears to be spread by birds, esp pigeons in their droppings. Spores gain entry via the respiratory tract where they elicit a granulomatous reaction. However, pulmonary symptoms are uncommon. Rarely, lung cavitation, hilar lymphadenopathy, pleural effusions and occasionally pulmonary fibrosis.

Meningitis is more common and occurs in those with HIV infection or lymphoma. It develops subacutely. Skin and bone involvement is rare.

CSF characteristics
- clear/turbid
- ↑ protein
- N/↓ glucose
- mononuclear pleocytosis

=> Diagnosis: Examination of CSF (perform a CT scan before LP to r/o a SOL).
- India Ink staining shows the organism directly
- CSF cryptococcal antigen is positive at various titres (NB!: It is unusual for the CRAG to become -ve after tx although levels should fall substantially)
- +ve latex cryptococcal agglutinin test performed on CSF is diagnostic. This is how Ag is detected
- Cryptococci can also be cultured from CSF and/or blood

=> Treatment: Induction phase: ✓IV amphotericin B (1 mg/kg/d) 2 weeks
✓Oral Fluconazole at 1200mg
(NB!: If amp B unavailable or renal function is impaired or if side effects are troublesome, give 1600mg Fluconazole)

- Coinfection: Fluconazole 800mg OD PO for 8 weeks
- Maintenance: Fluconazole 200mg OD PO
  For 1 year at least and until CD4 > 100 cells/mm³ for two measurements 6 months apart and viral load is undetectable.

* Therapeutic LPs (Supportive Tx)
- done daily until pressure is normal for 3 consecutive days or until severe headache subsides if measuring pressure is impossible.
- Remove 10-20ml of CSF each time

✓ ↓ mortality
✓ ↓ risk of blindness
✓ Alleviates pain
✓ Improves consciousness level

* Defer HAART in the HAART naive until after completing 5 weeks of tx and symptoms have resolved

⇒ Managing and Monitoring for Amphotericin B Therapy
- Give 1L of normal saline with 20mmol of KCL over 2-4 hours before each infusion
- Give Amp B with 1L of 5% dextrose.
- Give 1-2 tablets of 8mEq KCL orally BD. An additional 8mEq KCl tablet BD may be added in week 2
- Mg trisilicate supplements 250mg BD OR 4mEq of $MgCl_2$ PO BD
- Manage hypoK+ and ↑ creatinine levels -
  - obtain a routine baseline and then weekly potassium and creatinine
    ✓ if K^+ < 3.3mmol/L → 1L Normal saline with 40mmol KCl or 1-2 tablets 8mEq KCl TID. Add Mg. Monitor K daily
    ✓ If creatinine ↑ > 2 fold from baseline, omit Amp B, ↑ hydration to 1L TID. If there is improvement, restart Amp B at 0.7 mg/kg/d on alternate days. If no improvement, discontinue Amp B and give 1600 mg Fluconazole. Monitor

creatinine daily.

⇒ Ddx:
- Lymphoma of CNS
- TB meningitis
- Neurosyphilis
- Acanthamoeba infection
- Progressive disseminated histoplasmosis with CNS involvement

Nicole

IMED

Question 9

Clinical presentation of Rheumatoid arthritis

- pain / swelling and tenderness mostly of small joints of hands, feet and wrist. Spares DIP. Symmetrical.
- Morning stiffness of greater than 1hr duration or after periods of inactivity.
- May have a palindromic onset
- May have pitting oedema
- Constitutional symptoms → weakness, easy fatiguability, anorexia, weight loss
- Warmth of joint. Erythema suggests sepsis and is rare.
- Deformities with longstanding uncontrolled disease:
  - Swan neck deformity (hyperextension of PIP with flexion of DIP)
  - Boutonnière deformity (Flexion of PIP with extension of DIP)
  - Radial deviation at the wrist with ulnar deviation of the digits with z deformity of the thumb.
  - Similar changes in the feet with flat feet due to rupture of tibialis posterior tendon.
- Baker's cyst
- Hand extensor tendons may rupture
- Atlanto-axial joint subluxation

- Extra articular manifestations
  - Rheumatoid nodules (elbows etc)
  - Rheumatoid vasculitis
  - Pleuropulmonary manifestations → pleural disease, interstitial fibrosis, bronchiolitis obliterans, organizing pneumonia, pleuropulmonary nodules
  - Pericarditis, LHS, pericardial effusion, myocarditis, endocarditis

Felty's syndrome → RA, splenomegaly, neutropenia

- Neurologic manifestations → neuropathies of median, ulnar, radial (interosseous branch) or anterior tibial nerves, cervical cord compression
- Ocular → Episcleritis, scleritis, scleromalacia, keratoconjunctivitis sicca
- Hematological & lymphatic → Anemia, eosinophilia, thrombocytosis, Felty's syndrome, splenomegaly
- Others → Amyloidosis, osteoporosis

B. Multidisciplinary management

- General principles
  - Rest
  - splinting the joint
  - Exercise to maintain muscle strength & joint mobility → physiotherapy & occupational therapy
  - Orthotic & assistive devices
  - ...

- Medical principles
  - NSAIDs
  - Glucocorticoids

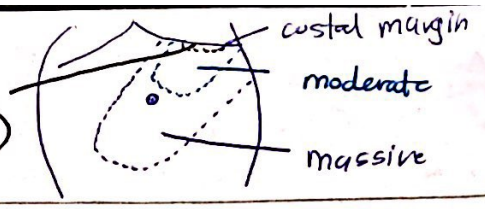
1st line DMARDs.

2nd line Biological agents eg. TNF-α inhibitors, B-cell depletion compounds (Rituximab), IL1 & IL6 inhibitors, T cell inhibitors

- Surgery → for pain relief, improve function & prevent deformity.

Fatma.

6. Lymphoma
7. Thalassemia major



Imed

Somy Naisola I won't be late again.

4a) Causes of Massive splenomegaly → Beyond umbilicus, crosses midline 7&8m into pelvis / LHQ

1. Chronic myeloid leukemia (CML) → commonest cause
2. Myelofibrosis
3. TSS (tropical splenomegaly syndrome) due to malaria. (abnormal immune response to malaria)
4. Visceral leishmaniasis
5. Storage diseases eg. Gaucher's disease

b) Investigations and Reasons.

1. A complete blood count
   - Leukocytosis will be present in CML and normocytic normochromic anemia (CML)
   - Erythrocytosis in Polycythemia vera
2. A peripheral blood film
   - malaria parasites can be visualized.
   - increased mature lymphocytes " and
   - Amastigotes visualized in WBC's
3. A bone marrow biopsy
   visualize amastigotes on Giemsa stained slides
   Hyperallular in CML
   May have increased blasts in blastic crisis transformation
4. Splenic aspirate
   100% sensitive for leishmaniasis ∴ risk of splenic rupture.
5. Reticulocyte count.
   Reduad in Myelofibrosis.
6. Direct agglutination test
   Positive in leishmaniasis

A 30yr old is hospitalized with a history of ingestion of methanol. Enumarate the potentially lethal >1g/kg.

a) Clinical features

First 24hrs : CNS depression -> dyspnoea, vertigo, dizziness, headache, euphoria. Followed by visual disturbances: blurry vision, reduced acuity, photophobia, 'snowstorm' (black & white dots in the visual field). central scotoma, absent pupillary response (afferent pupillary defect).

b) Complications

Seizures Coma

Permanent optic nerve atrophy/ blindness.

Residual extrapyramidal movement disorders.

c) Therapeutic approach to this px

The goal of acute management is to increase excretion and decrease metabolism to toxic byproducts, and to maintain serum glucose and address metabolic derangement to avoid 2o injury.

ABCs

Vitals: BP, RR, temp, SpO_2, RBS

If hypoglycaemic, give 50mls 50% dextrose IV. Also give 100mg thiamine IV followed

by 100mg PO BD for 6wks.

IV normal saline / Ringers lactate 20ml/kg bolus to achieve a mean arterial pressure MAP > 65 mmHg.

Maintenance 4:2:1 - 4ml/kg for the 1st 10kg. 2ml/kg for the next 10kg. 1ml/kg for the remaining kgs.

If stable 5% dextrose saline infusion at 3L/24hrs.

Do not perform gastric lavage or give activated charcoal. Instead, perform gastric aspiration if presented within 1hour of ingestion.

Treat seizures with IV benzodiazepines.

Ethanol as antidote. IV or oral

Haemodialysis indications:
serum pH <7.25 osmolar gap >10
acute renal failure visual symptoms
deterioration despite maximal supportive care
End points are correction of acidosis and osmolar gap <10.

Investigate: LFTs, FBC, ABG, UEC, lipase

SIE of ethanol Rx: hypoglycaemia, CNS depression, thrombophlebitis, intoxication, hypotension.

A patient presents with chronic cough, fever and wt loss of 2m duration. Examination reveals tracheal deviation to the left and a stony dull percussion note with no breath sounds on the right base

a) What is the diagnosis?

b) List the investigation you would carry out to confirm the diagnosis.

Diagnosis: pulmonary tuberculosis with a pleural effusion on the right side.

Investigations:

Gene xpert testing of sputum:

Sputum culture

Chest x ray to detect pleural effusion:

- blunting of the costophrenic angles.
  - blunt costophrenic sulcus, visible lateral view; occurs with 75ml of fluid
  - once fluid is abt 300ml, it blunts the lateral costophrenic angle, visible on frontal CXR
- meniscus sign: pleural fluid rises higher along the the lateral margin of the thorax than it does medially.
- opacified hemithorax if > 2litres of fluid
- heart & trachea displaced away from the side of opacification

IMED ESSAY (Number 8)

a) Clinical diagnosis Portal hypertension (SAAG > 11g/L)

b) Possible causes for this condition (prehepatic, posthepatic, hepatic)

i) Liver cirrhosis

ii) Portal vein thrombosis

iii) Budd chiari disease

iv) Hepatic vein obstruction

c) Complications that the patient is at risk of

i) Variceal bleeding

ii) Spontenous bacterial peritonitis

iii) Hepatic encepalopathy

iv) Coagulopathy

vi) Hepatorenal syndrome

vii) Hepatopulmonary syndrome

d) Tests carried out on the acitic fluid and interpretation of results

i) Cell count and differential - leucocytosis

ii) Total protein concentration - increased

iii) Culture - positive culture

iv) Glucose concentration - reduced

vi) Gram stain + microscopy

vii) TB smear

viii) Cytology

e) 3 principles of management of the ascites in this patient

i) Diuresis

ii) Paracentesis

iii) Portosystemic shunting

A 40 year old woman presents with a 5 week history of palpitations, heat intolerance and an anterior neck swelling. She has lost 3 kg despite an increased appetite.

O/E the mass moves on swallowing.

a) What is the most likely diagnosis?

Hyperthyroidism

b) list signs you would expect to find on clinical examination:

✓ Cardiorespiratory signs
- tachycardia
- ankle oedema

✓ Neuromuscular signs
- tremor
- hyper-reflexia
- ill-sustained clonus
- muscle weakness (proximal myopathy, bulbar myopathy)

✓ Dermatological
- increased sweating
- palmar erythema
- spider naevi
- alopecia
- hyperpigmentation, vitiligo (Graves disease)
- digital clubbing (Graves disease)
- pretibial myxoedema (Graves disease)
- onycholysis

✓ Ocular
- lid retraction, lid lag (Graves dx)
- Gritiness (Graves disease)
- Excessive lacrimation)
- Exophthalmos, Corneal ulceration, Opthalmoplegia, Diplopia (Graves)
- Chemosis

✓ Other
- Gynecomastia
- Lymphadenopathy (Graves disease)

-X. Bonus !!

Symptoms:

✓ GIT

- weight loss
- hyperdefecation (Grave's)
- Diarrhoea, steatorrhoea
- Anorexia
- Vomiting

✓ Cardiorespiratory

- palpitations
- dyspnoea on exertion
- exacerbation of asthma

=> Investigations will show: sinus tachycardia, a-fib, increased pulse pressure, angina, cardiomyopathy and cardiac failure

✓ Neuromuscular

- Nervousness, irritability, emotional lability, psychosis

✓ Reproductive

- Amenorrhoea / Oligomenorrhoea
- Infertility, spontaneous abortion
- Loss of libido, impotence

✓ Ocular

- papilloedema
- loss of visual acuity

Other:

- heat intolerance
- fatigue, apathy
- thirst

=> Investigations will show osteoporosis

c) Priority Investigation

- TFTs: • Elevation of serum T3 and T4
  • Decreased TSH

- Autoantibody studies:
  • anti TPO antibody test titres are ↑ in autoimmune thyroiditis, Graves thyrotoxicosis and low/absent in toxic multinodular goiter and toxic adenoma.
  • thyroid stimulating immunoglobulin (TSI) diagnoses Grave's disease
  • Antithyroglobulin (anti-TG) antibodies present in Graves disease.

- Scintigraphy confirms the diagnosis if physical exam and lab tests are insufficient.
  • Iodine-123 or Technetium-99m are used for thyroid scanning
  • Normally the isotope distributes homogeneously throughout both glands but in hyperthyroidism, the pattern of uptake (e.g. diffuse vs nodular) varies with the underlying disorder.

Other tests:

- LFTs: • Slightly raised concentration of bilirubin, ALT and GGT
  • Elevated ALP (derived from bone and liver)

- U/E/C: • Mild hypercalcaemia

- FBC: • mild normocytic anaemia
  • mild neutropenia (Graves dx)

d) Definitive Treatment

- antithyroid drugs: • Carbimazole and its active metabolite methimazole
  • propylthiouracil

These drugs reduce the synthesis of new thyroid hormones by inhibiting the iodination of tyrosine.

- Subtotal thyroidectomy:
  - patient must be euthyroid before the operation.
  - antithyroid drug is stopped 2 weeks before and replaced with potassium iodate 170mg daily PO. This maintains euthyroidism in the short term by inhibiting thyroid hormone release and reduces the size and vascularity of the gland.

- Radioactive iodine
  - acts by destroying functioning thyroid cells or by inhibiting their ability to replicate.

- ≠ adrenoceptor antagonists
  - e.g propranolol or nadolol will alleviate but not abolish the symptoms
  - only useful short-term

For management of ophthalmopathy:
- Lid retraction will resolve when the patient becomes euthyroid
- [illegible]
- Grittiness treated with methylcellulose eyedrops
- Side shields attached to spectacle frames to decrease excessive lacrimation triggered by wind or sun
- Lid lengthening procedure or lateral tarsorrhaphy for corneal ulceration
- Extraocular muscle surgery for diplopia
- 60 mg prednisolone for papilloedema, loss of visual acuity or visual field defects

For management of dermopathy
- Pretibial myxoedema if persistent - local injection of triamcinolone or application of betamethasone ointment under occlusive dressing

Complication

- CVS:
  - afib
  - hypertension
  - sudden cardiac arrest
  - cardiac dilation and congestive heart failure
- MSK:
  - osteoporosis

IMED

EOY SAQ 2018 (special class)

Question 2

A 22 year old woman presents with a 2 month history of shortness of breath, irregular heart beat and haemoptysis. She has previously been followed up in the cardiac clinic for 1 year during which she has been relatively stable.

Physical exam reveals dyspnoea at rest, temperature of 36.7°C, pulse rate of 120 beats/min with irregularly irregular rhythm.

The JVP is 14 cm H_2O

Precordial exam reveals a tapping apex beat in the 5th intercostal space, midclavicular line. The heart sounds are ~~reg~~ irregular and there is a mid-diastolic murmur at the apex. There are fine basal crepitations.

A) What is the clinical diagnosis

Congestive cardiac failure secondary to mitral stenosis

B) List 6 factors that could have contributed to the worsening of her clinical state.

Infection

Severe anaemia

Thyrotoxicosis

Pregnancy

~~Overrest~~ Overexertion as in exercise

Atrial fibrillation

Infective endocarditis

C) List 3 investigations that will elicit her underlying structural cardiac lesion and the expected finding for each.

Investigation	Expected finding
1) Echocardiogram.	• Morphology of the valve: fusion of the commissural fibres, thickening of the valvular leaflets, calcifications on the valve. Reduced normal mobility of the valves.

35.8 - 37 - 0.5 axilla 35.3 - 36.5
mouth

	Doppler echocardiogram	• Reduced size of the mitral valve orifice $<1cm^2$ = critical stenosis • Secondary changes in the heart chambers such as left atrial enlargement, right ventricular hypertrophy • Increased pulmonary pressures (cor pulmonale) Pressure gradient across mitral valve
2.	Chest x ray	• Features of left atrial enlargement such as double shadow, filling of pulmonary bay, widened carina • Features of pulmonary congestion: prominent pul. vessels. • Normal cardiothoracic ratio • Mitral valve calcification • Interstitial oedema (Kerley A + B lines)
3.	Electrocardiogram (ECG)	• Signs of left atrial enlargement: - P wave duration in lead II > 0.12 seconds ~~Right~~ - P mitrale • Right ventricular hypertrophy - Right axis deviation in frontal plane - Tall R waves in V1-V2 - Wide QRS / T angle. • Atrial fibrillation.

Q. ~~Name~~ ~~pros~~ List 4 long term complications of this cardiac lesion

Heart failure

Infective endocarditis

Thrombo-embolism

Atrial fibrillation

E) Outline 5 principles of management of this patient

Anticoagulation to ~~prevent~~ reduce risk of thromboembolism.

Antibiotic cover for prophylaxis against infective endocarditis and RF

Anti arrhythmic agents to correct arrhythmias

Anti-heart failure medication to increase cardiac output

Definitive management is valvuloplasty to repair damaged valve.

J. MED QN

#3 A 63 year old man presents with a 3 month history of cough, frank hemoptysis & weight loss. He has a 30 pack-year history of cigarette smoking. Sputum microscopy and culture is negative for TB.

(a) What is the most likely diagnosis?

Bronchogenic Carcinoma

(b) Outline other clinical features that would be indicative of the diagnosis under the areas:

General Exam

i) Pallor
ii) Cyanosis
iii) Finger clubbing
iv) Lymphadenopathy
v) Tachypnea
vi) Wasting

Local Effects

i) Stridor
ii) Wheezing
iii) Decreased chest expansion on affected side
iv) Dullness on percussion due to the mass
Stony dull on percussion if there's pleural effusion, and also decreased tactile fremitus plus decreased & absent vesicular breath sounds
v) Tracheal deviation if a pleural effusion pushes the trachea away & the mass can also push both the trachea and mediastinum to the contralateral side

(c) Outline 4 priority investigations you would carry out and the expected findings.

Diagnostic (i) Chest xray – A mass in the lung fields can be seen. Also a pleural effusion can be

detected from blunted costophrenic angles.
- Tracheal deviation with or without a mediastinal shift can also be detected.

(ii) Cytology of any fluid drained eg. effusion or sputum
May show malignant cells.

(iii) Bronchoscopy for biopsy - Most common lung cancer is squamous cell carcinoma (35%)

(iv) CT scan of the chest - To delineate the mass further

Supportive i) FBC - Anemia
ii) UECs
iii) LFTs - ALP is elevated in bone metastasis.
Abnormal LFTs also indicate liver metastasis.
iv) Calcium - Elevated in bone metastasis
v) LDH - Elevated levels indicate increased tumor mass and high cell turnover

(d) Outline 2 approaches to the definitive management of this condition

Chemotherapy
Surgery
Radiation

Nicole

1 MED

A 50 yr old male from Makueni presents with a 2 day history of confusion & 3 weeks history of jaundice. He went into a semi comatose state within 2 hours of admission to the hospital. He visited both traditional & medical doctors for a troublesome headache 2 months ago.
Physical examination revealed jaundice, semi comatose GCS 13/15, BP 80/60 mm Hg. temp 35.0° C. Prothrombin time 80 sec & petechiae.

Working diagnosis → Hepatic encephalopathy secondary to Acute (8-28 days time: Jaundice to HE) liver failure

A) Aetiological factors
Drugs → Paracetamol, Halothane, Isoniazid,
Toxins → Amanita phalloides mushroom, carbon tetrachloride
Viral infections → Hepatitis B, C, CMV
Vascular → Budd chiari syndrome, veno occlusive disease
Metabolic → Wilson's disease, hemachromatosis
Autoimmune hepatitis

B) Management
- Airway → Assess for patency and suction if needed. Intubation or airway adjuncts.
- Breathing → look, feel, listen. O_2
- Circulation → patient is hypotensive hence 2 wide bore IV lines and Normal saline 2 litres

Supportive

Drugs → Assess for overdose (eg: paracetamol overdose give antidote → acetylcysteine)
Assess for hypoglycemia (RBS) and administer 50ml of 50% dextrose if hypoglycemic.

- Elevate the bed at 30°. Avoid sedatives
- Administer mannitol IV
- Hyperventilate. Nasogastric tube to prevent aspiration
- Administer lactulose 30-45ml TID
- Limit dietary protein intake to 30-40g/day
- Administer antibiotics → Rifaximin / metronidazole / neomycin to reduce urease producing bacteria in the gut.
- Transfusion → Fresh frozen plasma, vitamin K 10mg IV, platelets, whole blood.
- Phenytoin → seizure prophylaxis
- Omeprazole → for stress ulcers.
- Manage hypoglycemia if present → 50ml of 50% dextrose IV.

Supportive

A 27yr old with 3 wk hx of progressive SOB, dry cough, Low grade fever. Recieved a course of oral amoxicillin without much improvement. Completed Rx for sputum positive PTB 2 months ago, and responded well to Rx. Physical exam reveals a cachexic patient in severe resp distress with floricl oral thrush. PR 111 bpm Temp 37.9°C RR 32 bpm BP 110/75 mmHg O_2 Sats <90% on RA. Wght 46kg. HIV test +ve after counselling. What is the most likely diagnosis?

Pneumocystis Pneumonia in a newly diagnosed seropositive 27yr old man.

- What is the WHO clinical stage of this patient.

WHO stage 4. of HIV/AIDs.

Extrapulmonary cryptococcosis including menengitis
Disseminated extrapulmonary TB
Progressive multifocal leukoencephalopathy
Candida of trachea, bronchi, lungs, Eosophagus.
Any disseminated mycosis
Recurrent non typhoidal salmonella
HSV, CMV (Chronic infection)
Kaposi Sarcoma.
CNS toxoplasmosis.
HIV wasting syndrome.
Pneumocystis Pneumonia.
Ca Cervix Invasive. Lymphoma (Non Hodgkin B cell)

Investigation	Expected Finding
CXR	Patchy ground glass appearance/lesions. Parenchymal/subpleural cysts (Pneumatoceles)
Bronchoalveolar Lavage M/C/S.	Culture PCP. Staining Toluidine blue O stain shows PCP cysts. Other stains: Silver stain, Periodic acid schiff stain all showing cysts.
Viral Load.	Many copies of HIV virus.
CD4 count	<200/ml.
Full Hemogram	Anemia of chronic dx. Leukopenia.

List 2 parameters that objectively indicate severity of respiratory dx and the need for adjunctive therapy in this patient.

- SPO_2 on pulse oximetry.
- Respiratory function tests - Spirometry.
- Blood gas analysis.

Outline 5 principles of manggement.

~~A B C s~~ Supportive Rx - ABC, Oxygen supplementation

Oxygen supplementation., Nutritional support.

Rx of Disease {
- Corticosteroid supplementation on presentation.
- Antibiotics - Trimethropim-sulfamethoxazole.
  Clindamycin.
  for about 21 days.

Prevention of Dx Recurrence →
- Chemoprophylaxis using Cotrimoxazole (life long).
- HAART should be started - How soon?
- Treatment of oral thrush - fluconazole 150mg.

Qn/ A 57 year old man presents with a 7 month history of progressive left upper quadrant swelling, early satiaty and fatigue. Physical exam reveals an enlarged spleen extending 20cm below the left costal margin.

(a) List 5 differential diagnoses for this presentation

His complete blood count reveals WBC = $348 \times 10^9/L$, platelets = $602 \times 10^9/L$, Hb = 9 g/dL

(b) What is the most likely diagnosis

(c) List 4 priority investigations & the expected findings

(d) List 3 phases in the natural course of this condition

(e) Name 3 drugs used in the management of this condition

Ans/

(a) Ddx (Massive Splenomegaly)

1. Leishmaniasis
2. Hyperacute malarial splenomegaly (HMS)
3. Chronic Myeloid Leukemia (CML)
4. Lymphomas, CLL
5. Myelofibrosis
6. Schistosomiasis (usually moderate splenomegaly)
7. Hereditary spherocytosis
8. Gaucher's disease
9. Essential thrombocythemia
10. Polycythemia vera

(b) Massive splenomegaly with leukocytosis, thrombocytosis & anemia = Leukemia, most probably CML.

(c)

Investigations	Expected findings
① FBC + differential counts	Leukocytosis with predominant granulocyte elevation
② PBF	Myeloid cells @ different stages of development (promyelocytic to mature)
③ BMA	Hypercellular marrow with expansion of the myeloid cell line & megakaryocytes
④ Cytogenetics	BCR-ABL positive

(d) Phases of CML

1. Chronic – Blasts <10% (on BMA & PBF); patient is stable
2. Accelerated – (10–19)% in PBF &/or BMA; may have new cytogenetic changes in addition to BCR-ABL
3. Blast – >20% blasts on PBF & BMA

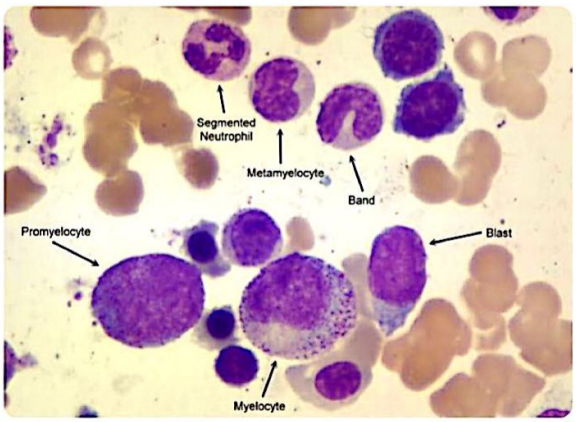
(a) CML Tx

① Tyrosine kinase inhibitors (mainstay): Imatinib, nilotinib, dasatanib

② Hydroxycarbamide / hydroxyurea

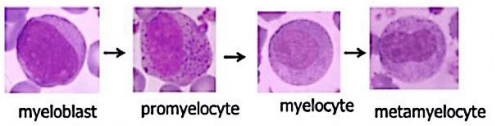
③ α-interferon

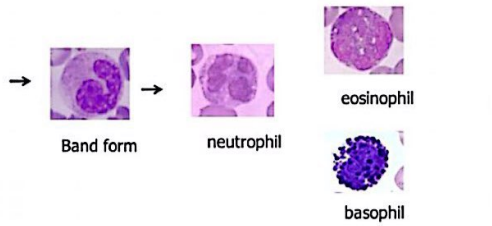
Mgmt classified into: Targeted therapy (tyrosine kinase inhibitors), chemotherapy, & immunotherapy.



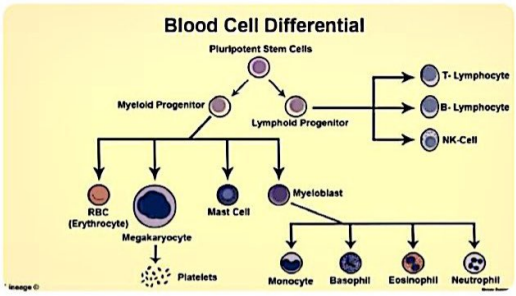
Myeloid cells @ different stages of development (pro-myelocytic to mature) = CML

Additional notes
* CLL on PBF has smear cells.
* AML & ALL have blast cells (AML blast cells contain more cytoplasm, contains granules & the pathognomic Auer rods.





The different stages



CML vs Leukemoid Reaction

Characteristic feature	CML	Leukemoid Reaction (leukocytoisis)
Age	>40 yrs	Any age
Leukocytosis	>100,000	30,000 – 50,000
Absolute Basophilia	Present	May not
Differentials	Mainly myelocytes & segmented	Mainly bands
Splenomegaly	Prominent	May not
Philadelphia Chromosome	Present (BCR/ABL ⊕)	Absent ⊖
LAP / NAP	Very low / Absent	High
Transformation to Acute leukemia	Yes	No
	Onset Morphology	Chronic Hypogranular
		Acute Toxic

→ Due to physiological response to stress or infection.

1. MED:

(i) A 23 yr old woman presents with general malaise, leg swelling, progressive reduction in urine output, poor appetite and N/V. She has pallor ++, bilateral pitting oedema ++ and bp is 182 mmHg. She is nulliparous. She has been managed for hypertension for the last 3 yrs.

Investigations reveal:

- Urinalysis - protein +++, rbcs ++, granular casts, no growth obtained on culture.
- FBC - Hb 9.2 g/dl, MCV = 86 fL
- WBC = 5.3×10^9/L
- Plts = 176×10^9/L
- UECs = serum creatinine 231 µmol/L
- Urea = 13 mmol/L
- K+ = 5.3 mmol/L
- Na+ = 131 mmol/L
- Renal USS → echogenic kidneys, right → 8.1 x 3.6 cms; left → 7.9 x 3.6 cms

(a) What is her clinical diagnosis?

Chronic Kidney Disease (CKD)

(b) List 4 possible secondary causes of this condition and give screening tests for these secondary causes.

Cause	Screening Test
1) Chronic Glomerulonephritis	* Renal biopsy
2) HTN	Bp measurements
3) SLE nephritis	ANA + ab's
4) Diabetes mellitus	RBS

(c) What is the commonest hematological complication associated with this condition?

Anaemia.

(d) List 4 causes of hematological complication?

1) Reduced production of erythropoietin (primarily produced by kidneys) hormone which usually stimulates red blood cell production.
2) Poor nutrition due to ~~reduced appetite~~ could ~~lead to~~ reduced iron intake.
3) Bone marrow toxins retained in renal failure.
4) Hematinic deficiency e.g. iron, B12, folate
5) Increased red cell destruction.

e) Outline 5 principles / modalities used to slow down the progression of this condition:

1) Good bp control: < 140/90 mmHg; if DM, < 130/80 mmHg
2) DM control: HbA1C < 7%
3) ACE's / ARB which are renoprotective.
4) Immunosuppressive therapy for autoimmune disorders
5) lifestyle: exercise, healthy weight, smoking cessation

61 year old man. Sudden onset Right sided weakness and loss of sensation. Difficulty speaking. Long standing hypertension.

BP on exam 184/100 mmHg. Right facial droop and right hemiplegia.

Brain CT shows hemorrhage.

a) Diagnosis? Hypertensive emergency with a hemorrhagic stroke.

b) Vascular territory? Left Middle cerebral artery (MCA)

c) Risk factors for this condition?

1. Hypertension.
2. Smoking
3. Diabetes Mellitus
4. Vasculitis
5. Coagulopathies
6. Family history (Aneurysms are familial)
7. Connective tissue disorders
8. Use of drugs like cocaine.
9. HIV

d) List 4 priority investigations to establish predisposing factors and findings you expect.

1. HbA1c may be high if patient was diabetic
2. CT angiogram or MR Angiogram can visualize any vascular abnormalities if they are present eg. aneurysms or Arterio-venous malformations.
3. ~~Rapid determine test for HIV - may be positive~~ 3. INR/coagulation profile - May be elevated if there's an underlying coagulation defect.
4. Complete Blood count - Platelet levels could be low.
5. Lipid profile - High triglycerides or high & LDL can be present
6. HIV test
7. ANA - to screen for an underlying ~~HIS~~ connective tissue disorder, (I know it's reaching but they always do it in the ward)
8.

Management Principles

1. ABC's
2. Gain IV access
3. Monitor and correct sugars
4. Monitor BP and only give antiHTNs if SBP >200 to a SBP <180
   DBP >110mmHg (a) if there is raised ICP.
   (b) If no raised ICP, SBP < 160mmHg (Note reduction)
   pg 24 CVS guideline
5. Control seizures if present.
6. Reduce ICP → Elevate head of the bed, give osmotic diuretics.
7. Pain relief
8. Consult neurosurgery
9. DVT/PE prophylaxis.
10. Ensure nutrition and hydration
11. Ensure early mobilization to prevent/minimize complications eg Pneumonia, DVT/PE, Pressure sores

3(a) What are the differential diagnoses in a 50yr old man from mmeg irigation scheme who presents with haematemesis.

Considering the geographical information provided, the most likely diagnosis would be: Hematemesis due to esophageal varices 2° to portal hypertension 2° to schistosomiasis.

Differentials: ① Other causes of portal hypertension: cirrhosis, Budd-chiari syndrome

② Local factors ✓Gastritis (alcoholic, NSAIDs, H.pylori), Esophagitis (e.g. medication-induced esophagitis)
✓Peptic ulcer disease
✓Mallory-Weiss Tear
✓Esophageal/gastric ca
✓Trauma/Iatrogenic rupture
✓Boerhaave syndrome (Mackler triad of chest pain, vomiting + subcutaneous emphysema)
✓Dieulafoy's lesion

③ Systemic factors (bleeding diasthesis): Congenital (coagulopathies) vs Acquired (hematological malignancies, anticoagulation therapy, liver failure, DIC, viral hemorrhagic fevers)

4. A 55 year old patient presents with a one month history of painless hematuria. Physical examination reveals a left lumbar mass. Discuss the investigations & management of this patient.

(Epidemiology: 90% of RCC, mean age = 55yrs, ♂:♀ ≈ 2:1, 15% of hemodialysis pts develop RCA).

Investigations
- BP: ↑ renin secretion)
- FHG: Polycythaemia (Erythropoietin secretion)
- ESR, U&E, ALP (bony met?)
- Ca^{2+} (↑ due to lytic bone mets, production of PTHrP = paraneoplastic syndrome, or ↑ production of PGs that promote bone resorption = responsive to indomethacin).
- LFTs (to evaluate for metastatic dx; hepatic dysfunction however can occur in RCC in the absence of liver mets = Stauffer syndrome).
- Urine · RBCs (>3/hpf)
  · Cytology (malignant cells)
- Imaging
  · U/S to differentiate benign/complex cyst from solid tumor
  * CT/MRI
  · IVU (filling defect +/- calcification)
  · CXR ("cannon ball" metastases)

Mgmt
- Supportive vs definitive
- Definitive
  · Radical nephrectomy
  · Targeted therapy: sunitinib, bevacizumab, sorafenib
  · Immunomodulatory therapy: IFN, IL-2, BCG

5. Write short notes on the differential diagnoses & investigations in a patient who presents with a right sided pleural effusion.

Transudative	Exudative
- Congestive heart failure	- Hemothorax (RBCs)
- Liver cirrhosis	- Infections (Parapneumonic effusions)
- Nephrotic syndrome/ESRD/ Peritoneal dialysis	✓Bacterial pneumonia (Gram stain, culture)
- Severe hypoalbuminemia	✓Tuberculosis (ZN stain, culture, PCR for TB DNA/GeneXpert, adenosine deaminase, interferon gamma levels).
- Acute atelectasis	✓Viral infection
- Myxedema	- Empyema
* Meig's syndrome	- Malignant pleural effusions (Lung, breast, ⅕ lymphoma = 75%) 1° or 2°s.
	- Trauma
	- Pulmonary embolism
	- Pancreatitis/Rupture esophagus (↑ amylase)
	- Rheumatoid pleurisy / lupus pleurisy

Investigations
* Demonstrate pleural effusion on CXR
* Investigations depending on the most likely cause as suggested by hx & physical exam
* Diagnostic thoracocentesis (usually not needed for transudative causes)

① Determine if it's a transudate or exudate using light's criteria:
- ✓ Pleural protein to serum levels >0.5
- ✓ Pleural LDH to serum levels >0.6
- ✓ Pleural LDH > ⅔ of the upper limit of the lab's normal serum levels.

② Gross appearance: Straw-like/pale yellow (transudate), Red/hemorrhagic (trauma, malignancies - 1° or 2°s, pulmonary embolism, asbestosis), Black (aspergillosis, metastatic melanoma, bronchogenic adenoca), Yellow-green (rheumatoid pleurisy), Pus (empyema), Ammonia-smell (urinothorax)

③ Cell count (nucleated cell count)
- ✓ Counts >50,000/microL = usually only in complicated parapneumonic effusions e.g. empyema.
- ✓ Exudative effusions (from bacterial pneumonia, lupus pleurisy, acute pancreatitis usually have a count >10,000/microL except chronic exudates (TB, malignancies) tend to have a count <5,000/microL.
- ✓ Lymphocytosis (85-95%) = TB, malignancy, sarcoidosis, R.A, lymphoma, yellow nail syndrome, chylothorax

④ Cytology = malignant cells

⑤ Biochemistry
- ✓ Protein - Transudates = absolute protein level <3 g/dL (30g/L)
  * TB effusions = protein level >4 g/dL (almost always)
  - Waldenstroms macroglobulinemia or M.M. = (7-8)g/dL
- ✓ LDH - Very high (>1000IU/L) = Empyema, rheumatoid pleurisy, pleural paragoniniasis
  - Elevated pleural fluid LDH with low pleural fluid protein = Pneumocystis jirovecii pneumonia, urinothorax, some malignancies
- ✓ Triglycerides = chylothorax
- ✓ Glucose - Low glucose (<0.06g/dL or 3.33 mmol/L or pleural fluid to serum glucose ratio of less than 0.5) = TB, malignant effusion, rheumatoid pleurisy, lupus pleurisy, complicated parapneumonic effusion (empyema), esophageal rupture.
- ✓ pH - Low pH (↑ production due to pleural fluid cells or bacteria e.g. empyema or ↓ H^+ efflux due to fibrosis, pleuritis, tumor e.g. R.A, malignancy, TB).
- ✓ Amylase = Pancreatitis, esophageal rupture, malignancy
- ✓ Creatinine = Urinothorax

⑥ Gram staining, microscopy, culture