HEMAT/ONC (Document prepared & compiled by Ward 8D members — MBChB class of 2019). Yusra

3 a) List the causes of Iron deficiency Anemia (IDA)?
b) Briefly discuss Investigations done in patient suspected with to suffer from IDA?

3a) Etiologies of Iron Deficiency Anemia:

1) Chronic blood loss
- Uterine
- Gastrointestinal e.g esophageal varices, hiatal hernia, peptic ulcer dx, Aspirin ingestion; Ca of stomach, Cecum, Colon or rectum; hookworm infestation, colitis; piles; Diverticulosis etc.
- Rarely, hematuria, hemoglobinuria, pulmonary hemosiderosis, self inflicted bloodloss
- Disorders of hemostasis, Intravascular hemolysis

2: Increased demand
- Prematurity of newborns
- Rapid growth (as in adolescent) growth spurt
- Pregnancy

3: Malabsorption of Iron
- Gastrectomy, Celiac dx
-

4: Poor diet
- Lack of nutritional food rich in iron

3b) Investigations → (1) Lab values.
- Full blood count: Hb - Low; MCV - Low; MCH - Low. Reticulocyte count - Low.
- Peripheral blood film: Hypochromic microcytic RBCs with Anisopoikilocytosis - ie Target cells and pencil shaped poikilocytes.

- Serum Iron and Total Iron binding capacity.
  Serum Iron - Low
  TIBC - Increased.
  Serum ferritin - decreased.

- Bone marrow Iron - Decreased Iron stores ie (macrophages.)

(ii) Investigating the Cause:
- Stool for occult blood.
- CXR to exclude pulmonary haemosiderosis
- Urinalysis for haematuria/ haemosiderinuria.
- Pelvic-gynecologic studies ie U/S
- Upper & Lower GI radiologic & endoscopic studies ie OGD & colonoscopy
- Bronchoscopy.
- Coeliac serology.

4. A 46yr old man presents with severe wasting. He has oral thrush with a purplish macular lesion on the palate. He also has widespread dark nodular lesions involving the feet & trunk.
   a) What is the differential diagnosis:
   - Kaposi sarcoma in immunosuppressed individual
   - Oral candidiasis " "
   - Melanocytosis
   - Addison's disease.
   - Histoplasmosis
   - Cryptococcosis

4b) List the available modes of therapy for his condition?

- Primary Treatment: If RVD positive; start them on ARVs. Lesions regress with HAART use.
- Local therapy for skin lesions:
  - * Topical retinoids e.g Alitretinoin
  - Local radiation / radiotherapy
  - Intralesional vinblastine / Vincristine / Bleomycin
  - Cryotherapy
  - Photodynamic therapy or surgical excision
- Systemic treatment
  - Immunotherapy: Interferon alpha - Immunomodulatory antiviral & angiogenic; superior efficacy if combined with HAART

    Others: IL-12

    Thalidomide, VEGF, Sirolimus, Monoclonal antibodies
  - Chemotherapy: Indicated for rapid progressive oral / visceral dx

    Liposomal doxorubicin / Daunorubicin - (Superior to conventional chemotherapy & less toxicity)

Naisola

Write short notes on anaemia in chronic renal failure:

a) Causes

- Deficiency of erythropoietin
- Toxic effects of uraemia on marrow precursor cells
- Reduced red blood cell survival
- Increased blood loss due to capillary fragility and poor platelet function.
- Reduced intake, absorption and utilization of dietary iron

b) Investigations

- Full Blood Count
  - assess red blood cell status, HB, HCT - assess platelet levels
  - assess MCV, MCH, MCHC
- Haematinic level
  - Serum iron level, ferritin
  - Serum B12 levels
  - Serum and red cell folate levels
- Urea and Creatinine
  - to assess levels of urea (because of uraemia)
  - to assess progression of disease
- Urinalysis
  - to rule out haematuria
- LFTs (particularly albumin)
  - to assess nutritional status

- EPO test
  - to assess for levels of EPO ; it is increased in anaemia

c) Methods of Treatment

- Depends on the cause of the anaemia:
  - ferrous sulphate 200 - 300mg TDS . Give IV if on dialysis.
  - EPO given to patients with a Hb < 10g/dL given either SC or IV. Note that its use can cause cardiovascular events such as hypertension and thrombosis. Target Hb is between 10 and 20g/dL
  - RBC transfusions
  - Vitamin B12 and Folic Acid supplements
  - Lastly a balanced diet should be maintained regardless of cause.

A 65 y.o man presents with bone pain, tenderness over the lumbar spine. Investigations show Hb = 6.5g/dL, normocytic normochromic picture and ESR = 70mm/hr

a) Give 2 differential diagnoses

- multiple myeloma
- bone metastasis secondary to prostate cancer

b) Investigations to ascertain the diagnosis

- Skeletal survey using x-rays to look for presence of lytic lesions and/or bone fractures of MM vs osteoblastic lesions of prostate Ca.
- Isotope bone scan which is the most sensitive way of detecting bone metastasis
- TRUS for prostate size and guided prostate biopsy. PSA studies (assessment of the velocity of PSA level increase / % of free PSA).
- BMA & trephine to assess if there are any plasma cells in the bone marrow.
- blood and urine protein electrophoresis which will detect the presence and type of M protein.

NKfrote

Qn/ A 57 year old man presents with a 7 month history of progressive left upper quadrant swelling, early satiety 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

(e) CML Tx

1. Tyrosine kinase inhibitors (maintain): Imatinib, nilotinib, dasatanib
2. Hydroxycarbamide / hydroxyurea
3. α-interferon

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

GIT

Joshua

Question 1

A 35 year old African man presents with
① Tense Ascites
② Leg Oedema
③ & Liver span of 3 cm

He has no pruritus and no respiratory signs. Serum Albumin was found to be 20g/L and serum albumin gradient was > 11 g/L.

a) What is the clinical diagnosis (2mks)
Liver cirrhosis resulting in portal hypertension

b) List 4 possible causes of this condition (4mks)
① Alcoholism
② Chronic viral hepatitis
③ Autoimmune hepatitis
④ Non-alcoholic steatohepatitis (NASH)

c) List 3 complications that this patient is at risk of (3 mks)
① Portal hypertension
② Hepato-renal syndrome
③ Hepatic encephalopathy

d) List 4 tests you would carry out on the ascitic fluid and how you would interpret the results
① cultures and sensitivity - if the culture is positive, it is indicative of ascitic fluid infection causing bacterial peritonitis

② Blood Cell count - a high count is indicative of a traumatic tap, hepatocellular carcinoma, or ruptured omental vein

③ Protein Concentration - Helps in calculating SAAG which can rule out portal hypertension as a cause

④ Amylase levels - To rule out or in a pancreatic cause

⑤ Glucose levels

e) Outline 3 principles of management of the ascites in this patient (3mks)

① Sodium intake restriction

② Diuretics - spironolactone and furosemide

③

Question 2

A 60 year old Kenyan female patient presents with features consistent with peptic ulcer.

(a) List 3 likely causes of PUD in her (3mks)

① H. pylori infections
② Prolonged NSAID use

(b) Outline the diagnostic evaluation of this patient

① Confirm the presence of ulcers (2mks)
  ① Barium Swallow
  ② Endoscopy (OGD)

(ii) Determine the Causative factor (5mks)
  ① H. pylori breath test (urea breath test)
  ② H. pylori stool test (stool antigen)
  ③ Biopsy for histology } Invasive biopsy / Endoscopy required
  ④ Rapid urease
  ⑤ Culture
  ⑥ Serology

Question 3

(a) Classify the causes of portal hypertension giving one example for each disease (3 m

Pre-Hepatic	Hepatic	Post Hepatic
(1) Portal vein thrombosis	(a) Pre-Sinusoidal (1) Schistosomiasis	(1) Budd-chiari syndrome
(2) Splenic vein thrombosis	(2) Congenital Hepatic Fibrosis	(2) Superior vena caval obs
(3) Massive splenomegaly (Banti's Syndrome)	(b) Sinusoidal -Cirrhosis -Alcoholic hepatitis	(3) Cardiac causes -Restrictive cardiomyopathy -Constrictive pericarditis -Severe CHF
	(c) Post Sinusoidal -Hepatic sinusoidal obstruction (Veno-occlusive syndrome)	

(b) List the modalities of management of acute variceal bleeding in a year old boy from Machakos (5 mks)

(A) -Acute treatment of bleeding
Vasoconstrictors s.a vasopressin 0.4 units/min
Terlipressin at 2mg 4-6hrly then half dose 5-7 da
Somatostatin 250 µg IV bolus

(B) Prevention of recurrence
-Use a β adrenoceptor blocker s.a propranolol

(C) Infection prophylaxis
- 7 days of IV ceftriaxone 1g/1 day

(D) Vessel embolization / banding of bleeding vessels.

(E) Treat for possible schistosomal infection with praziquantel

GIT

A 35 yr old African man presents with tense ascites, leg oedema and a liver span of 3cm. He has no Pruritus and no respiratory signs. Serum albumin was found 2 be 20g/L and SAAG was >11g/L

(a) What is the clinical diagnosis (2mks)
Hepatic failure secondary to Cirrhosis.

(b) List 4 possible causes of this condition.

- Infections - Hep B, C D, CMV, EBV, Schistosomia
- Drugs ; Toxins - Alcohol, amiodarone, OCP (Budd chiari)
- Inherited and metabolic disorders - Gauchers, CF, Galactosemia
- Biliary disorders - PBC, Biliary atresia, PSC
- Cardiovascular causes - chronic heart failure, Budd Chiari syndrome, Portal vein thrombosis
- OTHERS: NASH, Sarcoidosis, Scleroderma, Autoimmune hepatitis, cryptogenic.

(c) List 3 complications that this Patient is at risk of:

- portal HTN *
- Variceal bleeding.
- Spontaneous bacterial peritonitis (SBP)
- Hepatic Encephalopathy
- Hepatorenal Syndrome
- Hepatopulmonary syndrome.
- HCC

(d) List 4 tests you would carry out on the ascitic fluid and how you would interpret the results.

TEST - 4MKS	RESULT - 4MKS
1. cell count	normal WBCs <500cells/2L ↑ WBC - infections PMNs >250cells/ml - SBP
2.	
2. Total protein	Ascitic fluid protein ≥ 25g/L - Exudative
3. Culture/Gram stain/AFB stain	- Identify infecting organisms.
4. Ascitic fluid amylase	>1000 mg/dl in Pancreatic ascitis
5. Malignant cytology	- in suspected malignant causes.
6. Adenosine Deamse	>40 U/L in TB asmil
7. Gene Xpert	- suspect TB.
8. Biochemicals	- Glucose levels ↓ in Bacterial infections
9. SAAG ⊛	>11g/L - Transudative <11g/L - Exudative.
10. Appearance	- Straw-coloured in cirrhosis.

(e) Outline 3 Principles of management of the ascites in this Patient.

(i) Sodium and water restriction - salt 2mg/day, water - 1L/day

(ii) Promoting urine output with diuretics
- Spironolactone - DOC
- diuresis improved with resting in bed.

(iii) Removing ascites directly by PARACENTESIS

(iv) SHUNTS: TIPSS, portocaval shunts, Peritoneovenous Shunts.

RENAL

Index Number _____

5. A 23 year old woman presents with general malaise, leg swelling, progressive reduction in urine output, poor appetite and nausea. She has pallor ++, bilateral pitting oedema++ and blood pressure is 182/115 mmHg. She is nulliparous. She has been managed for hypertension for the last 3 years.

Investigations reveal:
Urinalysis – protein +++, RBCs ++, granular casts, no growth obtained on culture.
FBC – Haemoglobin 9.2 g/dl, MCV 86 fl,
WBC – 5.3 x 10^9/L, platelets – 176 x 10^9/L
UECr – Serum creatinine 201 µ mol/L,
Urea 13 mmol/L, K+ - 5.3 mmol/L,
Na+ - 131 mmol/L
Renal ultrasound shows – echogenic kidneys, right – 8.1 x 3.6 cm, left – 7.9 x 3.6 cm

(a) What is the clinical diagnosis? (2 Marks)

_____

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

Secondary Cause (4 Marks)	**Screening Test** (4 Marks)
1. _____	1. _____
2. _____	2. _____
3. _____	3. _____
4. _____	4. _____

(c) What is the commonest haematological complication associated with this condition? (1 Mark)

_____

(d) List 4 causes of the haematological complication. (4 Marks)

1. _____
2. _____
3. _____

_____

Outline 5 principles/modalities used to slow down the progression of this conditi

(5 M

Qn:2 (a) Define acute renal failure/AKI (4mks)
(b) Outline the causes of pre-renal azotemia (6mks)

David

RENAL QUESTION 5.

① (a) Nephrotic syndrome.

(b) - Kidney biopsy - Glomerulonephritis.
- Connective tissue disease eg. SLE - ANA, ANCA., Anti GBM ab.
- HIV - PCR,
- Haemolytic uremic syndrome (HUS) - shiga toxin E. coli - Blood culture. / Stool culture. / Stool microscopy

Random blood sugar → Diabetes mellitus.

(c) Haemorrhage / Thromboembolism.

(d) Reasons
- Loss of inhibitors of coagulation. Antithrombin III, Protein C and S.
- Increase of liver synthesis of procoagulant factors.
- Impaired platelet function.
- Uraemia. - impairs platelet function.

(e) Modalities to slow the disease progression.
- Use of diuretics to reduce the oedema.
- fluid and salt restriction
- Use of Angiotensin receptor blockers and Angiotensin II convertin enzyme inhibitors to reduce the proteinuria. and control HTN.
- Albumin replacement.
- Anticoagulation prophylaxis.
- Treat the underlying cause.
- Use of lipid lowering drugs eg. Statins
- Treat anemia.

2(a) Acute kidney injury - Sudden reversible reduction in renal function that develops days / weeks accompanied with reduction in urine volume.

(b) Causes of prerenal azotemia.

Prerenal azotemia refers to the rise in serum creatinine or Blood urea nitrogen (BUN) concentration due to inadequate renal plasma flow and intraglomerular hydrostatic pressure. to support renal filteration.

- Hypovolemia - e.g. bleeding.
- Decrease Cardiac output - heart failure
- Decrease in effective circulating volume eg. Congestive heart failure, liver failure
- Impaired renal auto regulation, drugs nephrotoxic. - NSAIDs, ACE-I, ARB Cyclosporine, Chemotherapy drug.
- 3rd spacing - sepsis, ascites.
- Renal artery stenosis
-

Hypovolemia losses
- GIT - Diarrhoea
- Skin - Burns, dehydration
- Renal - over diuresis
- Sequestration of blood (sepsis)

An 1y & 61yr old ♂ is brought to the casualty with acute onset right sided body weakness. The patient was eating lunch when he suddenly lost strength in the right side of his body. He was unable to move his right arm & leg and also noted a loss of sensation in the right arm and leg. He had difficulty speaking. His medical history is remarkable for long-standing hypertension. Physical exam reveals blood pressure of 184/100mmHg. Neurological exam reveals a facial droop & dense right hemiplegia. CT scan of the brain shows no evidence of hemorrhage.

(a) What is the diagnosis? (2mks)
(b) What vascular territory is involved (1mk)
(c) List 4 risk factors for this condition (4mks)
(d) List 4 priority investigations to establish the predisposing factors & expected findings (8mks)
(e) Outline 5 principles of mgmt of this patient (5mks)

(a) Ischaemic stroke

(b) Left Middle cerebral artery territory

(c) Risk factors
- (i) Hypertension
- (ii) Diabetes mellitus
- (iii) Hyperlipidaemia
- (iv) Heart disease (A. fib, valvular, ischemic)
- (v) Peripheral vascular disease / carotid bruit
- (vi) Smoking
- (vii) Past TIA/stroke
- (viii) ↑PCV/clotting ↑/the pill.

(d)

Investigations	Expected findings
(i) Lipid profile	→ Hyperlipidemia (LDL/TGs/chol ↑, HDL ↓)
(ii) Blood sugars	→ Random blood sugar may be ↑ (>11.1mmol/L) Raised HbA1C (>6%)
(iii) EKG (Holter monitor)	→ May show atrial fibrillation
(iv) Duplex ultrasound of carotids	→ carotid artery stenosis
(v) Echocardiography	→ There may be left atrial enlargement, mitral stenosis, mural thrombosis, RHD ASD (paradoxical emboli).

(Davidson's pg. 1158 & Oxford handbook of Clinical Med pg. 474)

(e) (i) Patient resuscitation (ABC: keep NPO if swallowing unsafe/aspiration risk; give O_2 if saturations <95%; correct any circulation abnormalities e.g. anti-arrhythmics; Do NOT lower BP abruptly in the 1st week → may ↓ cerebral perfusion as autoregulation is impaired → only lower BP if encephalopathy or aortic dissection).

(ii) Identify the cause (non-contrast CT/MRI)

(iii) Minimize the brain volume that is irreversibly damaged
- ✓ Reperfusion - Thrombolysis / thrombectomy (within 4.5hr from onset of symptoms if there are no contraindications)
- ✓ Glycemic control - keep RBS between 4-10mmol/L
- ✓ Pyrexia - use anti-pyretics
- ✓ Antiplatelet therapy (300mg Aspirin) - after excluding hemorrhage

(iv) Prevent & treat complications
- ✓ Edema - Mannitol or artificial ventilation, surgical decompression
- ✓ Seizures - maintain cerebral oxygenation / avoid metabolic disturbance / anticonvulsants.

CNS

Nemex

- ✓ chest infection - nurse semi-erect, avoid aspiration (NPO, NG)
- ✓ UTIs - avoid catheterization if possible (use penile sheath)
- ✓ constipation - appropriate aperients and diet
- ✓ Musculoskeletal
  - Painful shoulder - avoid traction injury, shoulder/arm supports, physiotherapy
  - Pressure sores - frequent turning, monitor pressure areas, avoid urine damage to skin, nursing care, pressure-relieving mattress.
  - DVT/PE - maintain hydration, early mobilization, heparin (for high-risk patients only).
- ✓ Depression & anxiety - maintain positive attitude & provide information / anti-depressants.

(v) Secondary prevention (after a stroke has already occured i.e. preventing further strokes)
- ✓ Control risk factors as in 1° prevention:
  - Look for & treat HTN/DM/↑lipids (statins) & cardiac disease
  - Help quit smoking
  - Exercise helps ↑HDL & ↑glucose tolerance
  - Folate supplements may also help (↓ homocysteine)
- ✓ Antiplatelet therapy: Aspirin 300mg/24hr for 2wks, then 75mg/day (unless imaging showing 1° hemorrhage)
- ✓ Anticoagulation therapy: Used instead of antiplatelet agents only in embolic stroke or chronic AF & only from 2 weeks after the stroke: Warfarin (Do not do both antiplt & anti-coag therapy @ the same time; only ↑ to ↑ hemorrhage risk without added benefit)

(vi) Neurorehabilitation (speech and physiotherapy)

CVS Assignment

Fredrick

1. 22 year old with 2 month history of shortness of breath, irregular heart beat and hemoptysis previously on follow up at the cardiac clinic for 1 year where she was relatively stable, now presents with dyspnoea at rest.
temp of 36.7°C
pulse rate of 120bpm - irregularly irregular
JVP 14 cm of water
precordial exam reveals:- tapping apex 5ICS, mcl
- mid-diastolic murmur - apex
- irregular heart sounds
- fine bibasal crepitations

Clinical diagnosis-
mitral stenosis in decompensated congestive cardiac failure in possible chronic rheumatic heart disease

6 factors that have contributed to worsening clinical state
✓ infection - infective endocarditis
✓ poor adherence to medication
✓ arrythmias
✓ pulmonary embolism
✓ acute valvular disease
✓ renal failure
✓ acute coronary event
✓ pericardial tamponade

3 investigations and expected findings
Doppler echocardiography → increased pulmonary pressure
→ mitral stenosis - thick cusps, - reduced valve area.
ECG (electrocardiogram) → Atrial fibrillation
P - mitrale
→ right ventricular hypertrophy
→ tall R waves in V1-V3.
chest x-ray → kerley B lines at the base.
→ enlarged left atrium.

4 long term complications:
→ development of mitral regurgitation with resultant left ventricular enlargement
This results in reduced cardiac output thus increased risk of:- thrombi formation and dislodgement
→ stroke.
→ pulmonary embolism.

→ Renal failure
→ predisposition to infective endocarditis
→ fatal arrythmias.
→ biventricular heart failure.

Principles of management
- Aim is to limit progressive cardiac damage and to relieve symptoms.

(i) Anticoagulation → reduce risk of systemic embolism
(ii) Diuretic control → reduce pulmonary congestion
(iii) Antibiotic prophylaxis → prevent infective endocarditis
(Davidsons says its no longer recommended routinely)
(iv) Rate-limiting calcium antagonists in atrial fibrillation
(v) ventricular rate control with beta blockers and digoxin.

- If significant symptoms, with no mitral regurgitation and left atrium is free of thrombus → mitral valvuloplasty.

2. 32 year old with heart failure secondary to dilated cardiomyopathy who has been stable at NYHA-2 presenting with rapid worsening of symptoms.

(i) Potential causes of acute decompensation
- infection → infective endocarditis.
- poor adherence to medication.
- new onset arrythmia
- pulmonary embolism
- acute valvular disease
- Renal failure.

(b) Relevant investigations-
(i) ECHO → chamber integrity
(ii) Doppler ECHO → pulmonary artery pressure
(iii) ECG → chamber hypertrophy
(iv) cardiac catheterization → pulmonary artery pressure.
(v) CXR → pulmonary congestion.
→ heart size.

3 Management of acute pulmonary edema.
Principles.
- Sit patient up
- Give oxygen for hypoxemia
- loop diuretic for fluid overload
- Pain and anxiety → morphine ~~+ met~~
- prevent aspiration with promotility drug
- measure systolic blood pressure
if >110 → consider vasodilator → nitroglycerin titrate according to blood pressure
- treat underlying cause
- if dyspnea does not improve → consider CPAP

Resp

Kevin

Resp:

Question 1:

A 63 year old man presents with a 3 month history of Cough, frank haemoptysis and weight loss. He has a 30 pack-year history of Cigarette Smoking. Sputum Microscopy and Culture is negative for tuberculosis (2 marks)

a) What is the most likely Diagnosis?

⇒ Squamous Cell Carcinoma of the lung.

b) Outline other Clinical features that would be Indicative of the diagnosis Under the;

General Exam; (4 marks)

⇒ 1. Finger Clubbing.
2. Cachexia
3. Anaemia
4. Supraclavicular or Axillary lymphadenopathy.

Local effects; (4 marks)

⇒ 1. Lung Consolidation.
2. Lung Collapse.
3. Pleural effusion
4. Chest pain
5. Dyspnoea
6. Recurrent or Slowly resolving pneumonia.
7. Lethargy
8. Anorexia.

c) Outline 4 priority Investigations You would Carry out And the expected Findings;

Investigation (4 marks)	Expected finding (4 marks)
1. Chest X-ray	1. - Mass or Spot in the lung. - Hilar enlargement - Lung collapse. - Pleural effusion.
2. Sputum Cytology	2. Atypical or Malignant cells in the phlegm.
3. Fine Needle Aspiration or Biopsy	3. Cancerous cells, Differentiation into Squamous cells and their stage of differentiation.
4. CT Scan	4. Staging of the tumor Location of the tumor Size and shape.

d) Outline 2 approaches to the definitive Management of this Condition; (2 marks)

=> 1. Lobectomy for patients with borderline fitness and smaller tumors (T1a-b, N0, M0)
2. Radical Radiotherapy for stage I, II, III
3. Chemotherapy + Radiotherapy
(pemetrexed + Cisplatin)
(Cetuximab)

Mutami

A patient presents with chronic cough, fever & weight loss of 2 months 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?
- Right sided pleural effusion 2° to either;
  i) Primary lung malignancy
  ii) mets to the lung
  iii) pulmonary tuberculosis

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

Baseline blood tests: FBC, CRP, ESR, U&E, & LFTs.

Diagnostic tests (for TB): Tuberculin skin test
: Ziehl-Neelsen / Auramin fluorescence stains (for sputum, pleural fluid, bronchopy washings ... etc)
: Nucleic acid amplification
: Culture in Lowenstein-Jensen
: Response to anti-TB's
(Davidson's pg 714)

THURSDAY

diagnostic tests for lung tumours:

i) imaging
CXR: Might see:
- Peripheral nodules ~ lung collapse
- hilar enlargement ~ pleural effusion
- Consolidation ~ bony secondaries
(Oxford handbook pg 174)

Cytology: pleural fluid & sputum.

Biopsy & histopathology:
* biopsies are taken by use of a flexible bronchoscope.
* bronchoscopy also allows an assessment of operability.
(Davidson's page 723)

staging: ✓ CT scan
✓ EBUS (endobronchial ultrasound)
✓ Radionuclide bone scan
✓ Pet scan
(Davidson's pg 723)

A 55 yr old Man presents with a 4 day hx of cough, right sided chest pain, shortness of breath & fever. His sputum is rusty brown & sometimes blood stained.

a) What's the most likely diagnosis?

Pneumococcal community acquired/lobar pneumonia. ~ (Hutch pg 179, Davidson's 704)

b) list 5 diagnostic investigations.

1. FBC: very high white cell count with a neutrophilia (suggests bacterial etology)
2. U/E/Cr: Urea > 7mmol/L is a marker of severity.
3. Inflammatory markers: ESR (↑), CRP (↑)
4. Blood culture: shows bacteremia which is a marker of severity.
5. Sputum microscopy, culture & sensitivity.
6. Urine: pneumococcal antigen.
7. Chest xray: Lobar pneumonia ~ homogenous consolidation of one lobe.
8. Pleural fluid M,C & S if present. (Davidson's pg 705)

c) Outline the treatment of this patient.

~ The most important aspects of management are:

i) oxygenation
ii) fluid balance
iii) antibiotic therapy

a) Oxygenation

- Given at high concentrations (> 35%) & humidified except for patients with hypercapnia associated with COPD.
- Maintain PaO_2 at or > 60mmHg or SPO_2 at or > 92%.
- If still hypoxic ⇒ Mechanical ventilation.

b) fluid balance

(Davidson's page 705)

c) Antibiotics

- as per pneumonia antibiotic protocol.

N/B: Patient falls in category 2.

d) Analgesia with paracetamol or NSAIDs to relieve the chest pain.

Infections Diseases

Wakini

A 27 y/o man presents with a 3 week hx of progressive SOB, dry cough and low grade fever. He has received a course of oral Amoxicilin without improvement. He completed treatment for sputum positive PTB 2 months ago; and has responded well to PTB treatment. PE findings- cachexic patient with severe respiratory disease (distress?) and with florid oral thrush. Pulse rate-111bpm, temp 37.9, resp rate- 32 breaths/min, BP 110/75mmhg and oxygen saturation is less than 90% on room air. He weighs 46kg. HIV test done after counselling came back positive

1. What is the most likely diagnosis?
    - Pneumonia infection secondary to Pneumocystis jirovecii
2. What is the WHO clinical stage of this patient?
    - WHO stage 4
3. Priority investigations

	Investigation	*Expected findings*
a	Sputum for microscopy	Trophic/cyst forms visualized
b	Chest radiograph	Diffuse bilateral pulmonary infiltrates extending from perihilar region
c	Ct scan	Bilateral ground glass opacities with a background of interlobular septal thickening
d	Baseline CD4 count	Less than 200cells/mm3
e	LDH levels	Elevated greater than 220 U/L (levels reflect the degree of lung injury. Decline with treatment)

4. List 2 parameters that objectively indicate severity of respiratory disease and the need for adjunctive therapy in this patient.
    - Respiratory rate- 32 breaths per min
    - Oxygen saturation less than 90% on room air
5. Outline five principles of management of this patient
    - Supportive treatment - Oxygen supplementation & nutritional support.
    - Treatment of disease- Septrin – 15-20mg/kg TMP plus 75-100mg/kg SXM IV/PO given in divided doses
    - Prevention of recurrence of disease and other opportunistic infections-Chemoprophylaxis with septrin (160mg TMP to 800mg SMX) daily
    - Treatment of oral thrush- fluconazole 150mg
    - Start ART and counselling.

SIPUL BRIAN.

Sipul

ENDOCRINOLOGY ESSAY.

28/07/2019.

10. A 40year old woman presents with a 5 wk hx of palpitations, heat intolerance, & an anterior neck swelling. She has lost 3 kg wt despite an ↑ed appetite. On examination, the neck swelling moves up with swallowing.

a) What is the most likely diagnosis? (2mks).

Hyperthyroidism.

b) 5 signs you would expect a find during physical examination (5mks)

Common Signs:

1. Weight loss.
2. Tremor.
3. Palmar erythema.
4. Sinus tachycardia.
5. Lid retraction, Lid lag.

Less Common Signs:

1. Atrial fibrillation.
2. Increased pulse pressure / Systolic HTN.
3. Goitre with bruit.
4. Hyperreflexia.
5. III-sustained clonus
6. Proximal myopathy
7. Bulbar myopathy.
8. Cardiac failure. (esp in elderly)

Rare: 1. Gynaecomastia.
2. Spider naevi.
3. Onycholysis
4. Pigmentation.

(c) Outline 4 priority Ix you would carry out to establish diagnosis and expected findings: (4mks Ix) (4mks findings)

1. 1st line Ix:
   Serum T_3, T_4 and TSH → ↑ T_3 ↑ T_4 TSH ↓ / undetectable

2. Thyroid autoantibodies → Elevated.

3. Anti thyroid peroxidase antibodies → Non specific elevation in Graves Dx. (Low or absent in Toxic MultiNG / Toxic adenoma)

4. TSH receptor antibodies – Graves Dx. → Positive in Graves Dx.

5. Radioisotope scanning: / 99m technetium scintigraphy (Thyroid scintigraphy)
   → Diffuse iodine uptake seen in Graves Dx.
   → ↑ radioactive iodine uptake / uptake of isotope.

6. U/S scan Thyroid → Detect nodules if > 3mm.

7. Other non specific labtests:

7. FHG → mild normocytic anaemia.
   → mild neutropenia. (Graves Dx).

8. ESR → ↑.

9. Ca^{2+} → mild hypercalcemia.

10. LFTs → ↑

11. Serum enzymes → ↑ alanine aminotransferase, GGT. and alkaline phosphatase from liver and bone.

12. Bilirubin → ↑.

13. Glycosuria → Ass. DM, 'lag storage' glycosuria).

14. ECG may demonstrate sinus tachycardia / Atrial fibrillation

10 (d) Outline 3 definitive Rx options for this Px. (3 marks).

1. Antithyroid drugs.
2. Radioactive iodine.
3. Surgery.

1. Antithyroid drugs:

a) Non-selective β adrenoceptor antagonist (β blockers).

such as: Propranolol. (160 mg daily: 40 mg/6hrs)

or Nadolol (40-80 mg) daily. for rapid ctrl of symptoms

- will alleviate but not abolish symptoms in most Pxs within 24-48 hrs.

- β-blockers should not be used for long term Rx of thyrotoxicosis but are extremely useful in short term, while Pxs are awaiting hospital consultation / following ^{131}I / Tx.

Verapamil → may be used as an alternative to β-blockers eg: in Pxs with asthma, but usually is only effective in improving tachycardia & has little effect on the other systemic manifestations of thyrotoxicosis.

- ANTITHYROID:

A. Titration Eg:

Carbimazole 20-40 mg/24hrs PO for 4wks. (Titrate according to TFTs every 1-2 months.

B. Block-replace

Give carbimazole + Thyroxine simultaneously. (Less risk of iatrogenic hypothyroidism.

In Graves, maintain on either regimen for 12-18 months then withdraw.

2. Radioiodine (^{131}I): Most become hypothyroid post Rx.

(Surgery)

3. Thyroidectomy: Risk of damage 2 recurrent laryngeal n. & hypoparathyroid

Pxs may become hypothyroid.

e) List 2 expected complications of this condition.

MAIN
1. Atrial fibrillation in thyrotoxicosis
2. Thyrotoxicosis crisis (Thyroid storm)

Others
3. Heart failure (Thyrotoxic cardiomyopathy, ↑ in elderly).
4. Angina.
5. Opthalmopathy.
6. Gynecomastia.
7. Osteoporosis.

Rheumatology

later

Qn a) List 5 risk factors for osteoarthritis (5 mks)

① Genetics - genetic factors play a major role particularly for hand and generalised OA, but also for the hip and knee - Twin and family studies.

② Gender / hormonal status - OA is more prevalent and more commonly symptomatic in women except at the hip where men are equally affected.

③ Trauma.

- fractures - particularly osteochondral fractures.
- Joint instability eg. cruciate ligament injury, joint hypermobility syndrome.
- Mechanical causes including limb leg. discrepancy, instability, repetitive (occupational) injuries

④ Endocrine

Diabetes mellitus

Hypo / hyperthyroidism

Acromegaly

⑤ Secondary to inflammatory disorders:

- Septic arthritis
- Rheumatoid arthritis
- Ankylosing spondylitis

⑥ Developmental:

Congenital hip dislocation

Slipped upper femoral epiphysis (SUFE)

Genu valgum or varum.

Qn b) Outline the management of the arthritis. (5 mks)

Early treatment principles.

① Full explanation of the condition - should include relevant risk factors; the fact that established structural changes are permanent but pain and fxn can improve as well as the prognosis

- Exercise - This should cover both strengthening and aerobic exercise, preferably with reinforcement of a physiotherapist
- Reduction of adverse mechanical factors - includes
  - weight loss if obese
  - Shock absorbing footwear
  - Modify daily activities
  - Walking aids
- Relieve pain - analgesics and anti-inflammatory therapy
  - Give an initial trial of paracetamol and consider the addition of a topical NSAID / oral NSAIDs and capsaicin
  - opioids may occasionally be required.

b) Intermediate tx

Maintain movement and muscle strength - physiotherapy.

Injection of depot intra-articular long acting steroids 6/12

c) Late tx

Minimally invasive procedures - arthrotomy, arthroscopy

Intertrochanteric realignment osteotomy - may prolong life of malaligned joints and relieve pain by reducing intraarticular pressure

Arthrodesis - surgical immobilization of a joint so that bones grow solidly together.

Total joint replacement - required for the minority of people with large joint OA.

Qn 2. Enumerate the therapeutic strategies for Rheumatoid arthritis.

① Agents that are effective in controlling the signs and symptoms of RA, but have no effect on disease progression:

- NSAIDs - reduce inflammation and pain
- COX-2 inhibitors are similar to NSAIDs but with improved GI safety and tolerability
- Corticosteroids have anti-inflammatory and immunoregulatory activity but ~~not~~ minimal disease modifying activity

- Corticosteroids can be used to bridge the gap between initiation of DMARD therapy and onset of action.
- Intra-articular steroid injections can be used for individual joint flares.

However, early use of DMARDs is recommended before radiologically evident damage develops.

Large body of evidence shows joint damage is an early phenomenon and joint erosions occur in upto 93% of patients with less than 2 years of disease activity

① Mild RA - usually hydroxychloroquine or sulfasalazine.

② Severe RA - usually methotrexate or combination of traditional drugs including methotrexate

→ If there is no satisfactory response - modify DMARD therapy as follows:

① substitute with or add mxt

② substitute with alternative DMARD.

③ Combination treatment with traditional DMARDs

④ Biological agent ± methotrexate.

Combination of DMARDs - better than mono therapy in inducing remission. The following combinations are feasible:

MXT + SSZ

MXT + Hydroxychloroquine

MXT + cyclosporine

MXT + leflunomide.

Triple therapy - SSZ + MXT + HCQ.

→ Non drug interventions - patient education, physical therapy, occupational therapy

NB: When in doubt; consult.