

PLAB 2 (OSCE): Miscellaneous/ Important Topics

Acute confusional state.

Any unexplained behavioural change in a hospital patient is likely to be part of an acute confusional state.

Clinical features: impaired conscious level with onset over hours or days. The patient is likely to be disoriented in time and with greater impairment in place. The patient may be unusually quiet and drowsy, and sometimes he/she may be agitated. On other occasions the patient may be deluded (accusing staff of plotting against him/her) or to be hallucinating (seeing things or hearing voices which are not there). In the later two cases the question of primary mental illness is raised e.g. paranoid state and schizophrenia. However if there is no past psychiatric history, and in the setting of a general hospital, i.e. the patient is physically ill or has had recent surgery, mental illness is rare and acute confusional state is common. A confusional state is more likely if the symptoms are worse in the late afternoon and at night.

DD: if agitated consider an anxiety state. If the onset is uncertain consider dementia.

Causes: infection: chest, UTI, surgical wound, IVI sites

Drugs: especially analgesics and sedatives.

*Alcohol withdrawal,

*Metabolic disorder,

*Hypoxia

Vascular: stroke, MI

Head injury, especially subdural haematoma.

Management: find the cause, review the case notes, examine the patient with above causes in mind, do necessary investigations, start relevant treatment.

If hypoxic give oxygen. If agitated and disruptive sedation is necessary, sometimes before examination and investigations are possible.

Use major tranquillizers: haloperidol and chlorpromazine.

In alcohol withdrawal use chlormethiazole.

Nurse in a moderately lit quiet room with the same staff in attendance to minimize confusion. Repeated reassurance and orientation.

Refer: **Mini mental state.**

Immediate management of Myocardial Infarction.

Reassure the patient.
35% oxygen (unless COAD)
site an IV cannula
take bloods for cardiac enzymes, U and E, FBC and ESR(elevated).

Arrange for continuous ECG monitoring and for CXR.

Diamorphine 5-10mg IV + anti-emetic cyclazine 50mg IV for analgesic, anxiolytic, anti-arrhythmic and venodilator effects.

Glyceryl trinitrate 0.5mg SL for coronary artery vasodilatation.

If presentation is less than or ~12hrs after the onset of pain, give streptokinase 1.5 million units in 100ml 0.9% saline IVI.

Aspirin 160mg/day PO.

Heparin 5000U/ 8h SC until mobilized as prophylaxis against DVT.

If pain continues give isosorbide dinitrate infusion.

Discontinue pre infarction drugs. Prohibit smoking

Continuous ECG monitoring. 24h bed rest.

4 hourly TPR and BP.

Examine Heart, Lungs and legs for complications.

Streptokinase side effects: hypotension and anaphylaxis.

Streptokinase Contraindications: stroke or active bleeding in the past 2 months, BP more than 200mmHg, surgery or trauma in the past 10 days, bleeding disorders, pregnancy, menstrual period, proliferative Diabetic retinopathy, previous streptokinase treatment in the last 5 days to 1 year.

Acute myeloid leukaemia.

This is neoplastic proliferation of blast cells derived from myeloid elements within the bone marrow. It is among the most rapidly progressive malignancies.

Clinical features: marrow failure: anemia, infection, bleeding, DIC.

Leukaemic infiltration: bone pain, tender sternum, CNS signs like cord compression and cranial nerve lesions, gums, testis and orbit infiltration, hepatosplenomegaly, lymphadenopathy, skin and perianal involvement.

Constitutional features: malaise, weakness, fever, polyarthritis.

Diagnosis: WCC is variable. Diagnosis depends on bone marrow biopsy.

Complications: leucostasis may cause pulmonary and cerebral infarcts and death. To reduce the blast count quickly a single dose of daunorubicin or doxorubicin may be given with allopurinol to counter the high uric acid level. Emergency cranial radiotherapy may be given. Massive mediastinal lymphadenopathy may give rise to dyspnoea.

Treatment: supportive care: blood and platelet transfusion, barrier nursing, IV antibiotics.

Chemotherapy: is very intensive, resulting in long periods of neutropenia and thrombocytopenia. The main drugs used are daunorubicin and thioguanine.

Bone marrow transplant (BMT): allogenic transplant from histocompatible siblings or from unrelated donors syngeneic transplants from a twin may be indicated in the first or subsequent remissions. The idea is to destroy the leukaemic cells and the entire immune system by cyclophosphamide and total body irradiation, and repopulate the marrow by transplantation from a matched donor infused IVI. Cyclosporin ` methotrexate may be used to prevent graft versus host disease.

Prognosis: chemotherapy: 20%. BMT: perhaps 50%.

Refer: **Leukaemia**.

Leukaemia counselling.

Adult basic life support.

SAFE approach: Shout for help, Approach with care, Free from danger and Evaluate.

check responsiveness (shake)

responsive: check his condition, get help if needed and reassure him regularly.

unresponsive: shout for help

open airway: head tilt, chin lift. (avoid head tilt if trauma to the neck is suspected).

Check breathing: look listen and feel up to 10 seconds.

breathing present : recovery position.

no breathing: call 999 and give 2 effective breaths, check airway and ensure head tilt and chin lift.

Assess circulation, movement/pulse up to 10 sec. (carotids)

circulation present: breath for one minute and reassess

no circulation: give chest compressions: 100 per minute (position yourself vertically above the victim's chest and, with your arms straight press down on the sternum to depress it between 4-5 cm.). Single rescuer:- 2:15, two rescuers:- 1:5.

Continue resuscitation until:- (i) qualified help arrives, (ii) the victim shows signs of life, (iii) you become exhausted.

Anorexia and Bulimia nervosa.

The person chooses not to eat.

Intense fear of becoming obese even under weight. Weight perception: feeling fat when thin. Excessive exercise and induced vomiting. Abuse of laxatives, diuretics, and appetite suppressants. Dieting and excessive importance to weight reduction.

Stages: binge eating, remorse, vomiting and concealment.

Amenorrhoea, when not on pill, more than 3 cycles absent.

Feeling self worth as being embodied in shape and weight. Depression and suicide attempts coexist.

View: struggle for identity and control.

Physical complications: sensitivity to cold, constipation, amenorrhoea, faints, weakness, fatigue, BP↓, glucose↓, K+↓.

Parental preoccupation with food may precipitate and sometimes leave the person without a sense of identity.

Management: if extremely weak - prompt admission to the hospital under the mental health act.

IV feeding and work on building a trusting relationship. Gaining weight: targets and aims.

Treat the medical complications of starvation. Help building relationships. On admission privileges are removed and restored as weight increases.

Cardiac complications are the commonest cause of death.

Asthma - Management.

British Thoracic Society guidelines: Check inhaler technique: address any fears of the patient. Prescribe peak flow meter.

Step 1: try occasional β agonist inhaler. If needed more daily add step 2.

Step 2: add inhaled beclomethasone or budesonide.

Step 3: increase inhaled steroid by a large volume spacer, \pm theophylline

Step 4: add ipratropium \pm salmeterol.

Step 5: check compliance; add regular prednisolone tabs.

Reference: [History taking Asthma.](#)

Pitfalls: failing to prescribe steroids soon enough.

Failing to notice severe morning dips in peak flow.
Always ask about nocturnal waking: it is a sign of dangerous asthma.

Not admitting those with moderate severe attacks to hospital.

Signs of severe asthma: wheeze prevents finishing sentences in 1 breath; > 25 breaths/min; pulse ≥ 110 ; peak flow less than 50% of predicted; pulsus paradoxus > 10 mmHg.

Life threatening signs: peak flow $< 33\%$ of predicted; silent chest; coma; BP \downarrow ; pulse \downarrow ; PaO₂ < 8 kPa; PaCO₂ raised; pH \downarrow .

DD: Respiratory obstruction from inhaled foreign body; swollen epiglottitis; mediastinal and neck lumps;

These usually cause inspiratory stridor, rather than expiratory wheeze.

Chronic lymphocytic leukaemia (CLL).

This is a monoclonal proliferation of well-differentiated lymphocytes. They are almost always B cells. The patient is usually over 40. Men are affected twice as often as women are.

Staging (0-6) correlates with survival.

Symptoms: bleeding, weight loss, infection and anemia.

Signs: enlarged, rubbery, non-tender nodes. Late hepatosplenomegaly.

Film: lymphocytosis, normochromic normocytic anemia and thrombocytopenia.

Complications: autoimmune haemolysis, bacterial infections and bone marrow failure.

Treatment: chemotherapy: chlorambucil (decrease lymphocyte count).

Radiotherapy: use for relief of lymphadenopathy or splenomegaly.

Supportive care: transfusions, prophylactic antibiotics and occasionally IV immunoglobulins.

Reference: **Counselling: Leukaemia, AML, CLL, CML and Leukaemia.**

Chronic myeloid leukaemia (CML).

CML is characterized by uncontrolled proliferation of myeloid cells. It is a myeloproliferative disorder (feature: massive splenomegaly).

Mostly common in middle aged males.

Philadelphia chromosome: this is chromosome 22, which has lost about half of its long arm. In nearly all the cases this is translocated to the long arm of chromosome 9. This Philadelphia chromosome is present in granulocyte; RBC and platelet precursors in over 95% of those with CML. Those without Ph¹ have a particularly bad prognosis.

Symptoms: weight loss, tiredness, gout, fever, sweats, haemorrhage, abdominal pain. Signs: splenomegaly, hepatomegaly, anaemia and bruising.

Tests: WBC , Hb↓ or↔ , platelets variable, leucocyte alk phos↓ , plasma uric acid and alk phos , B12.

Natural history: variable, typically 2 phases. Chronic, lasting month or years of few, if any, symptoms followed by blast transformation with features of acute leukaemia and usually rapid death.

Treatment: Busulphan, α- interferon. BMT starting with chemotherapy and whole body radiotherapy. 50-60% of transplanted patients will be cured.

Diarrhoea and rectal bleeding.

Diarrhoea is the passage of >300ml of liquid faeces/24hrs.

In determining the cause there are three major questions to ask:

1. Is the diarrhoea acute or chronic? Infections are often acute (has the patient been abroad?). Chronic diarrhoea alternating with constipation suggests irritable bowel syndrome. Medication abuse eg. Antacids.
2. Is the large or small bowel to blame? In the former stools are watery with mucus or blood and there is lower abdominal pain with tenesmus and urgency; in the later any pain is often periumbilical or in the RIF and the stools are bulky and stink.
3. Is there a non-GI cause? Eg thyrotoxicosis, anxiety, or autonomic neuropathy from DM (nocturnal diarrhoea) drugs like antacids, cimetidine, digoxin, antibiotics, thiazide diuretics and alcohol.

Osmotic causes of diarrhoea: laxatives: lactulose, magnesium sulphate.

Secretory causes: infections: bacteria: *Campylobacter*, *V.cholerae*, *Staphylococcus*, *E coli*, *Salmonella*, *Shigella*, *Clostridium difficile*; giardiasis; rotavirus; amoebiasis.

Inflammatory bowel disease: UC, Crohn's disease.

Laxative abuse; bile salts, malabsorption.

Increased motility: irritable bowel syndrome; thyrotoxicosis.

Refer: **Diarrhoea.**

Causes of bloody diarrhoea: dysentery: *Campylobacter*, *Salmonella*, *Shigella* and *E coli* infections; amoebiasis; UC; Crohn's disease; colorectal cancer; pseudomembranous and ischaemic colitis.

Causes of rectal bleeding (\pm diarrhoea): diverticulitis; colonic cancer; polyps; haemorrhoids; radiation proctitis; trauma; *fissure-in-ano*; angiodysplasia, a common cause of bleeding in the elderly due to arteriovenous malformation.

Investigations: PR to exclude overflow diarrhoea. Large bowel diarrhoea: fresh stool for pathogens, ova and cysts. Sigmoidoscopy, barium enema \pm colonoscopy if prolonged.

If a small bowel cause is suspected: rule out malabsorption; do faecal fats analysis and measure serum folate and iron. Consider a small bowel barium meal and biopsy.

Management: treat the cause, Fluids PO. Check U and E, if IV fluid is needed, give 0.9% saline with \geq 20mmol K/l. If it is necessary to reduce symptoms try codeine phosphate.

Refer: **Rectal bleed.**

Drugs in CVS.

β-blockers: Blockade of β₁ adrenoreceptors is negatively inotropic and chronotropic, and delays AV conduction. Blockade of β₂ receptors causes coronary and peripheral vasoconstriction and bronchoconstriction. β₁ receptors are atenolol, metoprolol etc.

β-blockers are used to diminish myocardial oxygen demand in coronary artery disease, to control hypertension and certain arrhythmias, and beneficial effect prophylactically following an MI

CI: asthma, obstructive airway disease, claudication, and heart failure/heart block

SE: nightmares, depression, decreased glucose tolerance, lethargy, impotence, and headache etc.

Diuretics: used in hypertension and heart failure. SE: frusemide: hypokalaemia, ototoxicity. Thiazides: hypokalaemia, hypomagnesaemia, glucose and ↓ platelets

Vasodilators: ↓ BP in hypertension, decrease the work of the heart in heart failure and coronary artery disease.

Venodilators: nitrates, arterial dilators: hydralazine

ACE inhibitors also act by vasodilation.

SE: hypotension and dizziness.

Aspirin: prevents blood clotting

GI upset and black stool.

Epilepsy.

Causes: Often none is found. Trauma, tumour, alcohol withdrawal.

Physical: space occupying lesions, stroke, raised BP, Tuberous sclerosis, SLE, PAN, sarcoid and vascular malformations.

Metabolic causes: hypoglycaemia, hyperglycaemia, hypoxia, uraemia, hypo and hypernatraemia, hypocalcaemia, liver disease, drugs (phenothiazines, tricyclics, cocaine or withdrawal of benzodiazepines).

Infections: encephalitis, syphilis, cysticercosis and HIV.

Refer: **Blackouts.**

Commonly used drugs: carbamazepine: toxic effects: rash, nausea, dizziness, diplopia, fluid retention, hyponatraemia and blood dyscrasias.

Phenytoin: toxic effects: rash, sedation memory and behaviour disturbance, gum hypertrophy, acne, facial coarsening, blood dyscrasias, folate deficiency and cerebellar syndrome.

Sodium valproate: monitor liver function and give after food. Toxic effects: sedation, tremor, weight gain, hair thinning, ankle swelling, hyperammonaemia (causing encephalopathy) and liver failure.

Other drugs: ethosuximide, vigabatrin or lamotrigine as second line treatment for partial seizures, phenobarbitone and benzodiazepine.

Only consider maintaining on two drugs if all appropriate drugs tried singly at maximum dose.

Refer: **Epilepsy counselling.**

Gout.

History:

Age of the patient?

H/o pain, stiffness (morning or evening). Swelling and redness.
Any other joints are involved?

Weight? Diet? (red meat, fish, offal precipitates.)
H/o skin rash, genital and oral ulcers. Fever?

Excessive exercise and starvation.

H/o trauma, surgery and infection.

H/o alcohol consumption. Past history of similar complaint?

H/o DM, HTN, IBD, polycythaemia, leukemia, renal failure.

Hypothyroidism and hyperparathyroidism.

Use of drugs like diuretics, aspirin, cytotoxics.

F/h Gout.

The metatarsophalangeal joint of the big toe is usually effected. Attacks are due to hyperuricaemia and the deposition of sodium monourate crystals in the joints.

Diagnosis: microscopy of synovial fluid: negatively birefringent crystals, x-rays: soft tissue swelling and punched out lesions in the bone. The joint spaces are preserved until late.

Treatment: acute gout: NSAID such as Naproxen. If contraindicated eg. Peptic ulcer, give Colchicine.

Prevention: avoid purine rich foods, obesity and alcohol excess. No aspirin. Consider reducing serum urate with long term Allopurinol.

DD for joint swelling: trauma, septic arthritis, seronegative arthritides and Gout.

Hodgkin's Lymphoma.

Lymphomas are malignant proliferation of the lymphoid system. They are divided into Hodgkin's and non-Hodgkin's types by histology. In Hodgkin's lymphoma characteristic cells with mirror image nuclei are found (Reed-Sternberg cells).

Classification: Nodular sclerosing, Mixed cellularity, lymphocyte predominance and lymphocyte depletion (bad prognosis).

Clinical features: Enlarged painless lymph nodes, usually in the neck or axilla. Rarely there may be alcohol-induced pain or features due to the mass effect of the nodes. Constitutional symptoms such as fever, weight loss, night sweats, pruritus and loss of energy.

Signs: lymphadenopathy (note position, consistency, mobility and tenderness). Look for anaemia, weight loss and hepatosplenomegaly.

Tests: lymph node biopsy for diagnosis. FBC, Film, ESR, LFT, uric acid, Ca²⁺, CXR, bone marrow biopsy, abdominal CT or lymphangiography.

Staging: 1- 4. Each stage is subdivided into A (no systemic symptoms) or B- with weight loss, fever and night sweats.

Treatment: Radiotherapy for stages: Ia and IIa.

Chemotherapy for IIa-IVb, usually a MOPP type regime.

Complications of treatment: Radiation lung fibrosis and hypothyroidism. Chemotherapy- nausea, alopecia, infertility in men, infection and secondary malignancies especially AML.

Prognosis: 5y-survival rate 80%, depending on stage and grade.

Emergency presentations: Infection, marrow failure and SVC obstruction (presenting with raised JVP, sensation of fullness in the head, dyspnoea, blackouts and facial oedema).

HRT.

When is HRT particularly desirable: - Premenopausal bilateral oophorectomy, hysterectomy, increased risk of osteoporosis eg: inactive or smokers, Ischaemic heart disease or risk factors like DM and hypercholesterolaemia and Rheumatoid arthritis.

Rule out thyroid and psychiatric problems before starting HRT as they present similarly.

counselling helps psychosocial problems, contraception should be continued for a year after the last period (PoP, IUCD and barrier methods are suitable).

Hot flushes and vaginal dryness respond to HRT.

HRT postpones menopausal bone loss and protects against

cardiovascular disease and ovarian carcinoma.

Women with uterus should receive oestrogen & progesterone

preparations. If bleeding - endometrial biopsy after 8 months.

SE: - weight gain, cholestasis & vomiting.

CI to HRT: - breast carcinomas, undiagnosed PV bleeding, breast feeding, h/o DVT or PE.

Types of HRT: Oral, creams, pessaries, rings, transdermal patches and oestradiol implants.

Refer: HRT Counselling.

Leukaemia.

Neutropenic regime: (for all patients with a WCC $\leq 1.0 \times 10^9/l$) close liaison with a microbiologist is essential. Barrier nursing. Look for infection (mouth, perineum, axilla, and IVI site). Do swabs. Avoid IM injections (the danger is an infected haematoma). Do swabs. Avoid IM injections (the danger is an infected haematoma). Wash perineum after defecation. Swab moist skin with chlorhexidine. Avoid unnecessary PR examinations. Give hydrogen peroxide mouthwashes and candida prophylaxis. TPR every 4h. High calorie diet.

If Temperature >38 degC. for $>4-6$ h or the patient is toxic start blind broad spectrum antibiotics. If there are chest signs consider treatment for *Pneumocystis*.

Prevent hyperkalaemia and hyperuricaemia following massive destruction of cells by giving a high fluid intake and allopurinol before cytotoxic therapy.

If the WCC is raised WBC thrombi may form in the brain, lung and heart (leucostasis). Transfusing here before the WCC is reduced increases the blood viscosity, so increasing risk of leucostasis.

DIC: this pathological activation of coagulation mechanisms may occur in any malignancy. The release of procoagulant agents leads to clotting factors and platelet consumption (consumption coagulopathy). Fibrin strands fill small vessels, slicing passing RBC.

Signs: extensive bruising, old venepuncture sites start bleeding, renal failure, gangrene, bleeding anywhere.

ALL:

This is a neoplastic proliferation of lymphoblasts.

Causes: unknown- but preconception paternal exposure to sawdust, benzene and radiation has been implicated.

Clinical Features: these are due to marrow failure: anaemia, infection and bleeding. Also: bone pain, arthritis, splenomegaly, lymphadenopathy, thymic enlargement, CNS involvement - cranial nerve palsy.

Common infections: CMV, candida, pneumocystosis, zoster, measles, bacterial septicaemia.

Treatment: supportive care: blood and platelet transfusion, IV antibiotics.

Preventing infections: Neutropenic regime, barrier nursing, prophylactic antibiotics.

Chemotherapy: remission induction: vincristine, prednisolone

CNS prophylaxis and maintenance chemotherapy;

Marrow transplant: consider in poor prognosis groups.

Quality of life: wigs for alopecia

Prognosis: cure rates high in children, low in adults.

Refer: Counselling Leukaemia, **Acute myeloid leukaemia, CLL.**

Meningitis is an infection of the pia matter and arachnoid. If you think the patient is having meningitis nothing should delay prompt blind treatment.

Clinical features: meningeal signs: headache, stiff neck, kernig's sign may be positive: pain and resistance on p forward; opisthotonus.

ICP signs: headache, irritable, drowsy, vomiting, fits, pulse↓ , consciousness↓ , coma, irregular respiration a

Septic signs: fever, arthritis, odd behaviour, rashes - any, petechiae suggest meningococcus, shock DIC, pul

Tests: LP is the key test, and should be done at once unless signs of raised ICP, or focal CNS signs in which hypotension and rapid progression) when benzylpenicillin must be given urgently. An early LP in Pyogenic M stain, culture, virology and glucose.

Blood culture, FBC and U & E, plasma glucose, SXR if h/o head injury, culture urine and nose swabs for virolo

Treatment: Prompt antibiotics save life. Give penicillin before admission. If on LP pus emerges from the needle, start a broad-spectrum agent, cefotaxime at once while awaiting its analysis. Liaise early with a senior and a microbiologist.

Set up an IVI. Treat shock with plasma IVI until BP >80mmHg systolic and urine flows as guided by CVP on IT

Diamorphine may be needed for severe pain, for nausea try domperidone.

Once the organism is known use most effective antibiotic after discussing with a microbiologist.

Continue parenteral antibiotics for 10 days. Follow with rifampicin to eliminate nasal carriage.

Look for complications: cerebral oedema, cranial nerve lesions, deafness, cerebral venous sinus thrombosis

Meningococcal prophylaxis: to all household and nursery contacts, those who have kissed the patient's mouth group A and C strains and may be used for school contacts (if >2yrs) or for travellers to endemic areas.

Meningitis.

Meningitis is an infection of the pia matter and arachnoid. If you think the patient is having meningitis nothing should delay prompt blind treatment.

Clinical features: meningeal signs: headache, stiff neck, kernig's sign may be positive: pain and resistance on passive knee extension when hips fully flexed. Brudzinski's sign, hips flex on bending head forward; opisthotonus.

ICP signs: headache, irritable, drowsy, vomiting, fits, pulse↓, consciousness↓, coma, irregular respiration and papilloedema.

Septic signs: fever, arthritis, odd behaviour, rashes - any, petechiae suggest meningococcus, shock DIC, pulse raised, tachypnoea, WCC- increased.

Tests: LP is the key test, and should be done at once unless signs of raised ICP, or focal CNS signs in which case do CT scan, or suspected meningococcaemia (epidemic area, petechial rash, hypotension and rapid progression) when benzylpenicillin must be given urgently. An early LP in Pyogenic Meningitis may be normal. Repeat LP if signs persist. Send 3 bottles of CSF for urgent gram stain, culture, virology and glucose.

Blood culture, FBC and U & E, plasma glucose, SXR if h/o head injury, culture urine and nose swabs for virology.

Treatment: Prompt antibiotics save life. Give penicillin before admission. If on LP pus emerges from the needle, start a broad-spectrum agent, cefotaxime at once while awaiting its analysis. Liaise early with a senior and a microbiologist.

Set up an IVI. Treat shock with plasma IVI until BP >80mmHg systolic and urine flows as guided by CVP on ITU. If ICP raised ask a neurosurgeon's advice, is dexamethasone indicated?

Diamorphine may be needed for severe pain, for nausea try domperidone.

Once the organism is known use most effective antibiotic after discussing with a microbiologist.

Continue parenteral antibiotics for 10 days. Follow with rifampicin to eliminate nasal carriage.

Look for complications: cerebral oedema, cranial nerve lesions, deafness, cerebral venous sinus thrombosis and mental retardation.

Meningococcal prophylaxis: to all household and nursery contacts, those who have kissed the patient's mouth. Give rifampicin or ciprofloxacin 500mg PO one dose for adults. Vaccination protects from group A and C strains and may be used for school contacts (if >2yrs) or for travellers to endemic areas.

Multiple Myeloma.

Myeloma is a plasma cell neoplasm, which produces diffuse bone marrow infiltration and focal osteolytic deposits. A Monoclonal band is seen on serum or urine electrophoresis.

Peak age: 70yrs. Sex ratio is equal.

Classification is based on principal neoplastic cell product. 60% of IgG and IgA myelomas also produce free Ig light chains, which are filtered by the kidney and may be detectable as Bence Jones protein. These may cause renal damage or rarely amyloidosis.

Symptoms: bone pain, mainly back, ribs, long bones and shoulder - not extremities. Increased Ca, pathological fractures, hyperuricaemia, anaemia, infection, neuropathy, blurred vision and bleeding.

Examination: bone tenderness, urine, fundoscopy, for hyperviscosity-induced haemorrhages and exudates, macroglossia (amyloid).

Investigations: FBC, ESR (increased), bone marrow, serum/urine electrophoresis, Ca increased, alk phos (normal), urea, creatinine and uric acid, increased, CXR, skeletal X-ray shows punched out lesions (pepper pot skull) and background osteoporosis.

Diagnosis: abundant plasma cell in marrow, M band or urinary light chains, osteolytic bone lesions.

Treatment: supportive: bone pain, anaemia and renal failure are the main problems so give analgesics and transfusions as needed. Solitary lesions may be given radiotherapy.

Chemotherapy: Melphalan is standard.

Prognosis: survival is worse if urea >10mmol/l or Hb <7.5gm/dl.

Dangers: Hypercalcaemia: IV saline, steroids eg hydrocortisone and consider diphosphonates.

Hyperviscosity: causing mental impairment, disturbed vision and bleeding. May need plasmapheresis.

Acute renal failure.

Multiple Sclerosis.

This is a chronic or relapsing disorder caused by episodic demyelination and formation of many small plaques throughout the CNS. Peripheral nerves are not affected. The pathogenesis involves focal disruption of the blood-brain barrier with associated immune response and myelin damage. Relapses may be triggered by otherwise innocuous infections.

Clinical: usually presents in young adult either with single focal lesion, especially unilateral optic neuritis which causes rapid deterioration in central vision, isolated numbness or with weakness of the legs. Other features are vertigo, nystagmus, double vision, pain, incontinence, cerebellar signs and Lhermitte's sign - paraesthesiae in arms or legs on flexing neck. Also less commonly: facial palsy, epilepsy, aphasia, euphoria and dementia. The usual course involves initial remissions and relapses with later progressive accumulation of disability. Steady progression from onset sometimes occurs.

Examination: look carefully for signs of CNS lesion. The abdominal reflexes may be lost.

Diagnosis is based on h/o recurrent episodes of neurological disturbance implicating more than one part of the CNS, where no other explanation found. Isolated features are never diagnostic, but may become so if a careful history reveals previous neurological features such as blindness for a day or two, or investigations reveal involvement of distant parts of the CNS.

Tests: CSF: pleocytosis, protein increased, electrophoresis reveals oligoclonal bands. Visual, auditory and somatosensory evoked potentials prolonged.

Treatment: there is no cure. Dietary supplements with polyunsaturated fats may help. Treat optic neuritis with prednisolone. β - interferon appears to reduce the relapse rate. Treat spasticity. Offer incontinence aids. Help patient to live with disability. Advice is available from Disabled Living Foundation and MS Society.

Relapse last a few months and remissions may last many years, especially if the first sign is optic neuritis, when the remission may outlive the patient.

Oral contraceptive pills.

OCP is a combination of ethinyl oestradiol and progesterone. They inhibit ovulation and make the cervical mucus thick, thereby inhibiting the sperm penetration.

Advantages: effective contraception; less painful periods; reduced premenstrual syndrome; decreased risk of ovarian tumours and endometrial carcinoma; ?PID and endometriosis.

Disadvantages: increased risk of arterial and venous disease i.e. DVT and MI. increased risk of breast cancer. It doesn't protect against STD.

How and when to start: day 1 of the menstrual cycle, on the day of ToP, 3 weeks postpartum, 2 weeks after full mobilization after major surgery.

Stopping the pill: stop the pill in case of sudden severe chest pain, sudden breathlessness, severe calf pain, severe stomach pain, unusual severe prolonged headache.

Absolute contraindications: cardiovascular disease, liver disease, focal migraine, undiagnosed uterine bleeding, gross obesity, immobility, abnormal lipid profile, and recent trophoblastic disease.

Relative contraindications: Prescribe with caution in those with a family history of MI, HTN, DVT and breast cancer; epilepsy, sickle cell disease, DM, severe migraine and oligomenorrhoea, heavy smokers, abnormal cervical smear.

Counselling: Pre-eclampsia.

(BP: systolic >130 and diastolic >90 over booking BP or BP >160/100 or BP 140/90 with proteinuria).

Good morning Mrs. Smith I am Dr. XYZ senior house officer in the department obstetrics and gynecology. You are now 30 weeks pregnant and we have been monitoring your BP closely over the last 2 weeks. I am sorry to say that your BP is still high. This condition is called Pre-eclampsia or pregnancy induced hypertension. This condition needs to be managed very carefully to prevent any complications to you or your baby. I am afraid that we will have to admit you to the hospital to control your BP. You need to take bed rest. We'll also start you on medication to lower your BP. It is important to test your urine and blood regularly. We'll also check the well being of the baby by CTG and scan. We hope that your BP will be controlled by these measures.

If how ever there is a problem we might need to deliver the baby early.

Pre-eclampsia: PIH with proteinuria \pm oedema. The arterial wall doesn't distend enough to allow sufficient blood flow to the placenta in late pregnancy, and increasing BP is a mechanism, which partially compensates for this.

Symptoms: headache, fever, chest pain, epigastric pain, vomiting and visual disturbance. There may be tachycardia, shaking, hyperreflexia, irritability and papilloedema.

Effects: plasma volume is reduced, increased peripheral resistance. If BP is >180/140 microaneurysms develop in the arteries. DIC may develop. Uric acid level is increased.

Management: admit, measure BP 2-4 hourly. Weigh daily, test urine for protein, monitor fluid balance, check uric acid, renal, liver and platelet function. Do regular CTG and USG to check growth and biophysical profiles.

Methyldopa to reduce BP. If signs worsen delivery is the only cure (give H2-blockers if they go into labour).

Symptomatic pre-eclampsia and eclampsia: diazepam + hydralazine. Labetalol provides an alternative. Visual symptoms, twitching or ankle clonus indicate that the baby must be delivered urgently (by caesarean section).

Ergometrine should not be used for the third stage. Syntocinon may be used. Ergometrine further increases BP so would

risk stroke.

Peptic ulcer

Ulcer sites: oesophagus, stomach, duodenum, and meckel's diverticulum.

Possible exacerbating factors: stress, smoking, alcohol, NSAIDS, steroids and *Helicobacter pylori*.

Clinical features: DU: epigastric pain, radiation to the back, eating relieves the pain (so weight gain), worse at night, waterbrash. With gastric ulcers the pain may be worse by day. Chief dangers are GI bleeding and perforation.

Investigations: endoscopy, biopsy, barium studies and consider rebiopsying all gastric ulcers after 4 weeks of treatment to exclude malignancy.

Treatment: remove exacerbating factors, use antacids, and give histamine antagonist for 8 weeks eg cimetidine or ranitidine. 90% of all ulcers heal, but relapse on stopping the drug is very common. 60-95% of those relapsing on stopping H2 antagonists has infection with *Helicobacter pylori*. This is best treated with triple regime, bismuth chelate + amoxicillin + metronidazole. Consider surgery.

Other drugs: omeprazole may heal resistant ulcers and work by blocking the proton pump of gastric parietal cells. Sucralfate may protect mucosa from acid pepsin attack.

SE: cimetidine: gynaecomastia ± impotence.

Bismuth: stool blackening; CI: renal disease and pregnancy

Sucralfate: pruritus, vertigo, back pain, dry mouth and insomnia.

Schizophrenia.

Thought insertion: 'some one is putting thoughts into my head'

Thought broadcasting: 'people hear my thoughts as they occur'

Thought withdrawal: 'thoughts are being taken out of my head'

Passivity feelings: 'someone is directing me'

Hearing voices.

Primary delusions.

Somatic hallucinations: bodily sensations imposed by some external agency.

Most frequent symptoms:

lack of insight,

auditory hallucinations,

ideas of reference,

suspiciousness,

flatness of affect,

voices speaking to the patient

delusions of persecution,

thought delusions.

Most frequent behaviour:

social withdrawal

underactivity

lack of conversation

little leisure interests

slowness, over eating

neglect of appearance

odd postures or movements.

Management: Phenothiazines: Chlorpromazine
Thioridazine.

Community psychiatric nurse, regular appointments and frequent follow up.

Severe Pulmonary Oedema.

Sit the patient up.

O₂ by face mask 100% if there is no existing lung disease.

Insert IV cannula and take bloods for blood gases, U & E and cardiac enzymes.

Continuous ECG monitoring

Arrange for a CXR. Take a brief history if possible. Note drugs;

precipitating factors: MI and infection; pre existing lung disease, hypertension and previous heart disease. Examine for signs of pulmonary oedema: pink frothy sputum, pulsus alternans, fine crackles on auscultation of the chest and gallop rhythm.

Drugs: Frusemide 40-80mg iv

Diamorphine 5mg iv

GTN spray 2 puffs SL

In AF give: Digoxin 0.5mg iv

Baseline tests and markers of progress: BP, heart rate, cyanosis, Respiratory rate, JVP, peripheral oedema and enlarged liver.

Continuing management: Frequently check BP, pulse rate, heart sounds, restrict fluids, measure urine output, further IV frusemide and check U and E and ECG.

If improving oral frusemide, sequential CXR, daily weighing, 6 hourly BP and pulse.

If BP↓ the danger is cardiogenic shock. Consider venesection to remove 500ml blood quickly

In patients with severe pulmonary oedema following MI may develop VSD or Mitral regurgitation.

DD: Bronchospasm and Pneumonia. All 3 conditions may coexist in elderly. Treat with Salbutamol nebulizer, Frusemide IV, Diamorphine and ampicillin.

Stroke.

Stroke results from ischaemic infarction of the brain or from intra cerebral haemorrhage.

Risk factors: HT, smoking, DM, hyperlipidaemia, contraceptive steroids, obesity, heart and peripheral vascular disease, AF, previous TIA, increased PCV, excessive alcohol intake.

€€€€€€€ Clinical features: usually sudden onset of signs and symptoms. Sometimes step wise progression over hours, even days. Focal damage relates to distribution of affected artery, but collateral supplies cloud the issue.

Tests: look for hypo and hyperglycaemia

€€€€€€€ AF (look for large left atrium on CXR, consider echo, and anticoagulation, but first exclude cerebral haemorrhage with CT scan)

€€€€€€€ Hypertension: HT retinopathy, large heart,

€€€€€€€ Giant cell arteritis: increased ESR, history of temporal headaches or tenderness. Give steroids promptly.

€€€€€€€ Look for active disease of syphilis. Rule out polycythaemia, thrombocytopenia, embolic stroke and carotid artery stenosis.

€€€€€€€ Management of stroke: explain to the patient and relatives what has happened. Consider the kindest level of intervention. No drug is of proven value. Admit to stroke unit for skilled nursing and physiotherapy.

Maintain hydration, but take care not to over hydrate. Risk of cerebral oedema. Nil by mouth if there is no gag reflex.

Turn regularly and keep dry to stop bedsores.

Monitor BP but treat if only high. If conscious level impaired consider treating cerebral oedema with glycerol. If cerebral haemorrhage suspected consider immediate referral for evacuation.

Consider naftidrofuryl and heparin.

€€€€€€€ Secondary prevention: Prevent risk factors. Consider aspirin and warfarin if embolic stroke or in AF patients.

€€€€€€€ Complications: pneumonia, depression, contractures, constipation and bedsores.

€€€€€€€ Primary prevention: Good medical care to prevent strokes. Control risk factors like HT, DM, Smoking and hyperlipidaemia. Lifelong anticoagulation in patients with rheumatic or prosthetic heart valves and in chronic AF.

Thyroid: history, Investigations & DD.

History: simple goiter is common in girls approaching puberty. Deficient iodide, goitrogens and dysmorphogenesis are also the causes of simple goitre. Multinodular and solitary nodular goitres are found in 20's and 30's. Papillary carcinoma is seen in young girls and follicular Ca in middle aged women. Anaplastic Ca is mainly a disease of old age. Primary toxic goitre (Graves' disease) and Hashimoto's disease are seen in middle age women.

Investigations: Thyroid function tests: T4, T³, TSH (serum thyroid stimulating hormone), radioiodine uptake tests, T³ suppression test.

Thyroid scan: will show which part of the gland is functioning or which part is not (hot or cold).

Miscellaneous tests: BMR, serum cholesterol, serum creatinine, tendon reflexes, ECG, CXR.

Bone scan: exclude early bone metastasis.

DD of thyroid swellings:

Non-toxic (simple) goitre: 1. Diffuse parenchymatus, 2. Colloid, 3. multinodular, 4. solitary nodular.

Toxic goitre: 1. diffuse (graves' disease), 2. multinodular, 3. Toxic (solitary) nodule.

Neoplastic: 1. Benign, 2. Malignant.

Thyroiditis: 1. Acute bacterial, 2. Granulomatous, 3. Autoimmune, 4. Riedel's, 5. Chronic bacterial from TB or syphilis.

Other rare types: Amyloid goitre.

Tricyclic antidepressants.

(Amitriptyline hydrochloride and dothiepin)

Indication: Tricyclic antidepressants are useful in moderate to severe endogenous depression

Dosage: needs to be adjusted according to the individual response. Tricyclics take two weeks to act.

Caution: - The tricyclics should be used with caution in the following conditions: cardiac diseases, liver failure, epilepsy, pregnancy, breast feeding, thyroid disease, mania, glaucoma, urinary retention.

Driving: drowsiness may effect performance of skilled tasks; effects of alcohol enhanced.

CI: tricyclic antidepressants are contraindicated in recent MI, heart block and arrhythmias and severe liver disease.

SE: palpitations (arrhythmias), dizziness (heart block), dry mouth, blurred vision, constipation, urinary retention, rash tremor, nausea, sedation, sweating.

The patient should be encouraged to persist with the treatment as some tolerance to these side effects develops.

Withdrawal of tricyclics should be done very slowly.

Upper GI bleeding.

Haematemesis: is the vomiting of blood. Melaena is altered blood passed PR.

Common causes: peptic ulcer, gastritis, Mallory-Weiss tear, NSAIDS, steroids, thrombolytics and anticoagulants.

Others: oesophageal varices, nose bleeds, oesophageal or gastric malignancy, oesophagitis, angiodysplasia, bleeding disorders.

Treatment: bed rest, nil by mouth, 6 units of crossed matched blood, I.V. 0.9% saline and blood transfusion as needed. Monitor BP, pulse, CVP, ECG and fluid balance. Do U&E, FBC, LFT, platelets, INR.

Liaise with surgeons early.

Bleeding varices. In the presence of portal hypertension, blood from the gut can reach the right heart bypassing liver via collateral veins around the oesophago-gastric junction. With pressure >12mmHg these veins dilate forming varices, which may bleed catastrophically.

Causes of portal hypertension: Presinusoidal (obstruction before the hepatic sinus): portal vein thrombosis, pancreatic tumour or pseudocyst, myelofibrosis, Hodgkin's, schistosomiasis, sarcoid. Post sinusoidal: cirrhosis, Budd-Chiari syndrome.