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MCQs for General Duty Medical Officers (GDMO)

With Explanatory Answers

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JAYPEE

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General Duty Medical Officers

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1. **Which one of the following infections is mainly responsible for recurrent exacerbation of COAD in elderly?**
   a. Rota virus
   b. *Mycobacterium tuberculosis*
   c. *Staphylococcus aureus*
   d. *Hemophilus influenza*

Ans. d. Hemophilus influenza

Explanation

Chronic obstructive airways disease or chronic obstructive pulmonary disease (COAD/COPD).

- Disease state characterized by airflow limitation that is not fully reversible.
- Third most common cause of death and fifth most common cause of disability.
- It consist of either chronic bronchitis, chronic asthma or emphysema.
- Cigarette smoking is most common etiological factor (> 90%), infection with H. influenza, pseudomonas, Streptococcus pneumoniae, Moraxella catarrhalis increase acute exacerbation of disease. α_1- antitrypsin(protease inhibitor which is produced in liver secreted into blood vessels and diffuse into lung) deficiency accounts for about 2% emphysema (mostly pan-acinar).
- Most consistent pathological finding is increased numbers of mucus secreting goblet cells in the bronchial mucosa, which blocks small airway, other mechanism of airway obstruction is due to inflammation and scarring and loss of elasticity of alveoli.

Ref: *Kumar and Clerk, 7/e p835-8; Harrison, 18/e p2132*

2. **The following diseases have autosomal dominant inheritance, except:**
   a. Polycystic kidney disease
   b. Peutz Jegher syndrome
   c. Von Willebrand disease
   d. Phenylketonuria

Ans. d. Phenylketonuria

Explanation

Autosomal dominant traits Prevalence 2–10 per 1,000	ABO blood group system, Achondroplasia, Acute intermittent porphyria, Brachydactyly, Ehlers–Danlos syndrome, Familial breast cancer, Familial hypercholesterolemia, Familial hypertrophic cardiomyopathy, Familial malignant melanoma, Familial poliposis coli, Hereditary hemorrhagic telangiectasia, Hereditary nonpolyposis colon cancer, Hereditary spherocytosis, Hereditary eliptocytosis, Huntington's chorea, Hyperkalemic period paralysis, Hyperlipoproteinemia I, II, III, IV, Hypokalemic periodic paralysis, Malignant hyperthermia, Marfan's syndrome, Maturity onset diabetes of the young (MODY), Multiple endocrine neoplasia type 1, Multiple endocrine neoplasia type 2, Neurofibromatosis, Osteogenesis imperfecta, Peutz–Jeghers syndrome, Polycystic kidney (adult), Retinoblastoma,Rotor syndrome, Crigler–Najar type II, Tuberous sclerosis types 1 and 2, Von Hippel-Lindau syndrome, Von Willebrand disease
Autosomal recessive traits Prevalence 2 per 1,000	Agammaglobulinemia, Swiss type, Albinism, Alkaptonuria, Congenital adrenal hyperplasia, Crigler-Najjar type I, Cystic fibrosis, Dubin–Johnson syndrome, Familial Mediterranean fever, Fibrocystic disease of pancreas, Friedreich's ataxia, Galactosemia, Gaucher's disease, Glycogen storage disease type I-VIII, Hemochromatosis, Hemoglobinopathies, Hirschsprung's disease, Homocystinuria, Maple syrup urine disease, Mucopolysaccharidosis types I–VII, Niemann–Pick disease, Obesity, Phenylketonuria, Sickle-cell anemia, Tay–Sachs disease, Thalassemia, Wilson's disease, α1-Antitrypsin deficiency, Fragile X syndrome
X-linked recessive traits Prevalence 1–2 per 1,000	Hemophilia type A and type B, Duchenne type of muscular dystrophy, Color blindness, G6-phosphate-dehydrogenase deficiency, Hydrocephalus, Retinitis pigmentosa, Agammaglobulinemia, (Bruton type), Fragile X syndrome, Adrenoleukodystrophy, Becker's muscular dystrophy, Fabry disease, Hunter syndrome, Nephrogenic DI, Wiskott–Aldrich syndrome
X-linked dominant traits	Vitamin D–resistant rickets, Familial hypophosphatemia, Blood group Xg

Ref: *Park, 21/e p763; Nelson, 19/e p383–9; Devidson, 21/e p52–5; Harrison, 16/e p373*

3. A young female presents with right heart failure. On examination she has an elevated JVP with giant wave, systolic murmur at left sternal border, soft delayed P2 with an ejection sound. The ECG shows right ventricular hypertrophy. She is likely to have:

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a. Pulmonary stenosis
b. Tricuspid regurgitation
c. Atrial septal defect
d. Ventricular septal defect

Ans. a. Pulmonary stenosis

Explanation

Pulmonary stenosis	***Tricuspid regurgitation***	***Atrial septal defect***	***Ventricular septal defect***
Pulmonary ejection systolic murmur and a thrill in left second interspace, concentric right ventricular hypertrophy, S1 normal, wide and variable split of second heart sound, in valvular pulmonary stenosis wide ejection click audible soon after S1. **C/F**-Mild to moderate PS are asymptomatic, In severe stenosis dyspnea on effort. palpitation easy fatiguability and rarely chest pain. Cardiac size normal, left parasternal heave. Infundibular stenosis distinguish from valvular PS by absence of click, absence of post stenotic dilatation, relatively lower point of maximum intensity of systolic murmur in 2–3th left interspace.	Feature of TR is seen in 20–50% RHD in India. It may be organic (associated with RHD) or functional (due to pulmonary hypertension). All patient of TR having feature of pulmonary hypertension. TR associated with MS may be functional or organic but TR associated with MR is mostly organic. Both systolic and diastolic murmur. **C/F-** right hypochondial pain due to congested liver, fatiguability. Pancystolic murmur over left sternal border. **ECG-** right ventricular hypertrophy. **T/T-** decongestive therapy, TR may resolve following mitral vulvotomy.	Enlarged right ventricle results in parasternal impulse. Ejection systolic murmur. S1 Loud, S2 wide and fixed split P2 ECG of ostium secundum – right axis deviation with right ventricular hypertrophy. Left axis deviation beyond -300 suggest ostium primum. **CXR-** shows mild to moderate cardiomegaly, right atrial and right ventricular enlargement, prominent main pulmonary segment, plethoric lung field. Echo- enlarge right ventricle with paradoxical ventricular motion. Larger the shunt, more cardiomegaly, larger pulmonary and tricuspid murmur **C/F-** Heart failure rare in infancy. Pulmonary hypertension, may develop arrhythmia and heart failure in late adulthood.	Most common congenital cardiac lesion. 90% in communication of membranous part of ventricle. Shunting of blood from left to right ventricle. Murmur start early masking S1, pansystolic murmur at left sternal border. Ejection systolic murmur at pulmonary valve, delayed diastolic at mitral valve. S2 widely split and variable in large shunt. S3 may audible at apex. **C/F-** symptomatic around 6–10 weeks with CCF. Palpitation, dyspnea on exertion and frequent chest infection are main symptoms in older children. If VSD is small, left to right shunt murmur continue to be pansystolic, absence of delayed diastolic. If VSD is very small

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Pulmonary stenosis	*Tricuspid regurgitation*	*Atrial septal defect*	*Ventricular septal defect*
ECG- right axis deviation, right ventricular hypertrophy, P pulmonale – severe stenosis, Pulmonary oligemia occurs if right to left shunt at atrial level. Balloon pulmonary valvuloplasty is TOC In isolated valvular PS... surgical treatment for failure balloon valvoplasty or infundibular PS		**T/T-** closure of fossa ovalis percutaneously, closure recommended before school entry to prevent late complication	murmur is ejection systolic, if VSD is large left to right shunt murmur is shorter and softer and appear as ejection systolic murmur. Spontaneous closure (highest for muscular type) within 3 month in small VSD. It can complicate to infective endocarditis **CXR**- pulmonary plethora. **T/T-** control CCF, chest infection Surgery indication – CCF in infancy, LR shunt is large (pulmonary flow more than twice as systemic flow), If PS present than pulmonary atrial hypertension or aortic regurgitation. Surgery – closure of VSD with patch through right atrium. Complication of surgery:- complete heart block, bifascicular block, residual VSD

Ref: *Ghai, 8/e p413-443*

4. Nocturnal hypoxemia in COPD is due to all the following except:
   a. An increase in upper airway resistance because of muscle tone
   b. Stimulation of peripheral chemoreceptors
   c. Shallow breathing in REM sleep, reduced ventilation
   d. Inhibition of intercostals and accessory muscles in REM sleep

Ans. b. Stimulation of peripheral chemoreceptors

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Explanation

In nocturnal hypoxemia the mechanism of alveolar hypoventilation:

- Inhibition of intercostal and accessary muscle in REM sleep.
- Shallow breathing in REM sleep, which reduces ventilation, particularly in severe COPD.
- An increase in upper airway resistance because of a reduction in muscle tone.

This nocturnal hypoxemia associated with further rise in pulmonary arterial pressure because of vasoconstriction, and majority of death in patient of COPD occurs during night probably due to cardiac arrhythmia. The patient may develop secondary polycythemia.

Treatment

Avoid sleeping pill. Noninvasive bilevel positive pressure ventilation.

Ref: *Kumar and Clerk, 7/e p840*

5. A 50-year-old male presented with acute onset dyspnea, orthopnea and pinkish frothy sputum. The clinical diagnosis is:

a. Acute bronchial asthma b. Acute pulmonary edema
c. Acute pulmonary embolism d. Pneumonia

Ans. b. Acute pulmonary edema

Explanation

Acute pulmonary embolism	***Acute pulmonary edema***	***Pneumonia***	***Acute bronchial asthma***
It is third leading cause of death in hospitalized patient. PE develop in 50–60% of proximal DVT, 60–70% of symptomatic PE will have lower segment DVT. The risk factor of PE are risk factor of thrombus formation like venous stasis, injury to vessel wall and hypercoagulability (Virchow triad). **C/F**- one or more of three finding, dyspnea, and chest pain with breathing, or	Pulmonary edema may be cardiogenic (due to AMI, severe ischemia causing heart failure, acute volume load due to valvular regurgitation and MS) or non-cardiogenic (due to IV opioid, ↑ intacerebral pressure, high altitude, sepsis, shock, DIC, severe medications, inhaled toxin), **C/F-** severe dyspnea, orthopnea, pink frothy sputum, diaphoresis and cyanosis. Sings-rales, as generalized wheezing. CXR- pulmonary vascular redistribu-	The incidence higher in the very young and the elderly, accounts for almost one-fifth of childhood deaths. Usually spread by droplet infection. *Strep. Pneumonia*- most common cause **C/F**: acute onset , fever, rigors, shivering and vomiting often predominate, loss of appetite, headache, cough is characteristically short, painful and dry, but later with mucopurulent sputum.	Characterised by chronic airway inflammation and increased airway hyperresponsiveness leading to symptoms of wheeze, cough, chest tightness and dyspnea **C/F**- Typical symptoms include recurrent episodes of wheezing, chest tightness, breathlessness and cough. Common precipitants include exercise, particularly in cold weather, exposure to airborne allergens or pollutants, and viral upper respiratory

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Acute pulmonary embolism	*Acute pulmonary edema*	*Pneumonia*	*Acute bronchial asthma*
tachypnea. Hemoptysis, tachycardia, hypotension, ↑ JVP, right ventricular gallop rhythm, split P_2 Severe cyanosis ↓ Urinary output CXR- in medium PE-Pleuropulmonary opacities, pleural effusion, linear shadows, raised hemidiaphragm, in massive PE it is normal **ECG-** S_1 Q_3 T_3 anterior T-wave inversion Right bundle branch block (RBBB) ABG- May be normal or in severe case reduced *Pa*CO_2, low *Pa*O_2, Metabolic acidosis	tion, blurriness of vascular outline, increase interstitial marking, the butterfly pattern of distribution of alveolar oedema. In cardiogenic pulmonary edema pulmonary capillary wedge pressure elevated(>25mm Hg), in noncardiogenic it is normal or low.	Rust-coloured sputum in patients with *Streptococcus pneumonia*,; hemoptysis complications of *Mycoplasma pneumoniae* include hemolytic anemia, Stevens-Johnson syndrome, erythema nodosum, myocarditis, pericarditis, meningoencephalitis, Guillain-Barré syndrome. In *Staphylococcus aureus* multilobar shadowing, cavitation, pneumatoceles and abscesses seen. Dissemination to other organs may cause osteomyelitis, endocarditis or brain abscesses *Klebsiella:*- More common in men and alcoholics. Upper lobe involvement typical. Low platelet count and leucopenia **CXR**- homogeneous opacity localized to the affected lobe appears within 12–18 hours from the onset of the illness	tract infections Beta-adrenoceptor antagonists, Aspirin and other nonsteroidal antiinflammatory drugs are associated with asthma in about 10% of patients • Diagnosis: • Compatible clinical history *plus either/or*: – FEV_1 ≥ 15% (and 200 ml) increase following administration of a bronchodilator/trial of corticosteroids – > 20% diurnal variation on ≥ 3 days in a week for 2 weeks on PEF diary – FEV_1 ≥ 15% decrease after 6 mins of exercise • IgE is typically elevated in atopic asthma, ↑ sputum or peripheral blood eosinophil count

Ref: *CMDT 51/e p397, Devidson 21/e p716-18, 680*

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6. Pseudo membranous colitis is caused by which of the following organisms?

a. *Shigella* b. *E. coli*
c. *Clostridium perfringens* d. *Clostridium difficile*

Ans. d. Clostridium difficile

Explanation

Pseudomembranous Colitis

- Acute, potentially severe disease of the colon characterized by exudative plaques that cover the intestinal mucosa.
- The disease is caused by an enterotoxin produced by C. difficile, an anaerobic bacteria.
- Symptoms begin 3 days to 4 weeks after initiating antibiotic therapy. Virtually all antibiotics have been associated with this disease, but clindamycin, fluoroquinolones, and the cephalosporins are the most common offenders.
- Signs and symptoms include watery diarrhea, crampy abdominal pain, lower, abdominal tenderness, and fever, leukocytosis.
- Dehydration and electrolyte disturbances may develop in severely ill patients. Toxic megacolon and colonic perforation are rare, but serious, complications.
- Approximately 20% of patients relapse after primary treatment.
- Demonstration of C. difficile toxin in the stool or sigmoidoscopic visualization of the characteristic yellow-white plaques in an erythematous and edematous mucosa suggests the diagnosis. Biopsy of the plaques shows a mucinous, fibrinous, polymorphonuclear exudate. Most patients have disease throughout the colon; however, the disease may be confined to the right colon, and in such cases sigmoidoscopic findings are negative.

Treatment

- The first step in treatment is to discontinue unnecessary antibiotics. metronidazole is first-line therapy, vancomycin should be used in severe cases. Relapse is treated with a second course of therapy. Multiple relapses can be treated with tapering or pulsed doses of vancomycin; or probiotics including Lactobacillus or Saccharomyces boulardii, or cholestyramine.

Ref: *NMS medicine, 6/e p212*

7. The ventricular tachycardia can respond to all of the following *except*:

a. D C Cardioversion b. IV Amiodarone
c. IV Lidocaine d. IV Digitalis

Ans. d. IV Digitalis

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Explanation

Ventricular Tachycardia (VT)

- Feature in favor of ventricular tachycardia in the differential diagnosis of board-complex tachycardia
  - A history of myocardial infarction
  - AV dissociation (pathognomonic)
  - Capture/fusion beats (pathognomonic)
  - Extreme left axis deviation
  - Very broad QRS complexes (> 140 ms)
  - No response to carotid sinus massage or IV adenosine
- The common causes of ventricular tachycardia include acute myocardial infarction, cardiomyopathy and chronic ischemic heart disease.
- Patients may complain of palpitation or the symptoms of low cardiac output, such as dizziness, dyspnea or syncope.
- The ECG shows tachycardia with broad, abnormal QRS complexes with a rate above 120/min.
- Prompt action to restore sinus rhythm is required which should followed by prophylactic therapy. DC cardioversion is the treatment of choice if systolic BP is less than 90 mm Hg If the arrhythmia is well tolerated, IV amiodarone maybe given as a bolus followed by an IV infusion.
- Intravenous lidocaine can be used but it can depress left ventricular function, causing hypotension or acute heart failure. Beta-blockers may be effective at suppressing VT.
- Aggravating factor like Hypokalemia, hypomagnesemia, acidosis and hypoxemia should be corrected.

Ref: *Devidson, 20/e p566-67*

8. Which one of the following cardiac enzymes does *not* remain elevated for more than 48 hours after myocardial infarction?

a. Creatinine kinase MB
b. Troponin T
c. Lactic dehydrogenase
d. Aspartate amino transferase

Ans. a. Creatinine kinase MB

Explanation

- Troponin T and Troponin I, become positive as early as 4–6 hours of onset of MI and should be abnormal by 8–12 hours. Level of troponin elevated upto 5–7 days (7–10 days in Harrison) so not useful for evaluating suspected reinfarction.

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- Creatinine Kinase MB become positive as early as 4–6 hours of onset of MI and should be abnormal by 8–12 hours and normalize within 24 hours hence more useful for evaluating reinfarction.
- LDH level begins to rise by 12 hours, reaches maximum at 48 hours and return to normal by 11th days.
- Aspartate amino transferase rises from normal level 5–17 to 200IU/L is useful diagnostically in the first few days then the level drops to normal after 3 days.

Ref: *Walter & Israel general pathology, 7/e p715; CMDT 51/e p357*

9. All the following are helpful in the management of acute pulmonary edema *except* :

a. Administration of high flow high concentration oxygen
b. Use of Morphine
c. Administration of loop diuretics
d. Make the patient lie in supine position

Ans. d. Make the patient lie in supine position

Treatment of Pulmonary Edema

- Patient should be sitting position with leg dangling over side of bed.
- Oxygen is delivered by mask or to obtain arterial $Po_2 > 60$ m Hg. If needed endotracheal intubation and mechanical ventilation may be needed.
- Morphine is highly effective. It increase venous capitance, lowering LA pressure, and relieves anxiety, which can reduce the efficiency of ventilation. It should be avoid in patient with opioid induced pulmonary edema.
- IV diuretics (furosemide and bumetanide) indicated even patient is not exhibited prior fluid retention.
- Nitrate therapy accelerates clinical improvement by reducing both BP and LV filling pressure. It ameliorate dyspnea rapidly. IV nesiritide when given as a bolus followed by infusion, improves dyspnea more rapidly than IV nitroglycerine. Norepinephrine, dopamine or dobutamin can be given to increase BP which maybe due to nitroglycerin induced hypotension.
- β-adrenergic agonist or IV amynophyllin may useful to reduce bronchospasm.

Ref: *CMDT, 51/e p398*

10. All of the following aggravate heart failure *except*:

a. Intercurrent Infection
b. Arrhythmia
c. Fluid restriction
d. Anemia

Ans. c. Fluid restriction

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Explanation

Factors That May Precipitate Acute Decompensation in Patients with Chronic Heart Failure:

- Dietary indiscretion
- Myocardial ischemia/infarction
- Arrhythmias (tachycardia or bradycardia)
- Discontinuation of Heart Failure therapy
- Infection
- Anemia
- Drug (Calcium antagonists (verapamil, diltiazem) Beta blockers, NSAID, Antiarrhythmic agents [all class I agents, sotalol (class III)], Anti-TNF antibodies
- Alcohol consumption
- Pregnancy
- Worsening hypertension
- Acute valvular insufficiency.

Ref: *Harrison, 18/e p1907*

11. The following are true for hypertrophied cardio myopathy *except*:

a. Systolic murmurs
b. Triple apical impulse
c. Diastolic murmur
d. Jerky carotid pulse

Ans. c. Diastolic murmur

Explanation

Some important point regarding Hypertrophic cardiomyopathy:

- Hypertrophic cardiomyopathy is characterized by marked left ventricular hypertrophy in the absence of other causes, such as hypertension or valve disease.
- This was previously termed *hypertrophic obstructive cardiomyopathy* (HOCM), as distinguished from *nonobstructive hypertrophic cardiomyopathy*. Other terms that have been used include *asymmetric septal hypertrophy* (ASH) and *idiopathic hypertrophic subaortic stenosis* (IHSS).
- The prevalence is 1:500 adults. Autosomal dominant pattern, and spontaneous mutations also arise.
- Characterized hemodynamically by diastolic dysfunction, originally attributed to the hypertrophy, fibrosis, and intraventricular gradient when present.
- Usually presents between the ages of 20 and 40 years.
- Dyspnea on exertion MC, orthopnea, and paroxysmal nocturnal dyspnea
- Chest pain with either an atypical or typical exertional pattern occurs.

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- Palpitations may result from atrial fibrillation or ventricular arrhythmias. May be episodes of presyncope or syncope.
- Sudden death from ventricular tachycardia or fibrillation. It is the most common lesion found at autopsy of young athletes dying suddenly.
- Loud fourth heart sound and prominent 'a' wave in JVP - due to decreased ventricular compliance.
- The electrocardiogram usually shows left ventricular hypertrophy, often with prominent septal Q waves. Systolic anterior motion (SAM) of the mitral valve is a classic finding on the echocardiogram.
- Jerky pulse.
- Triple apical impulse at the apex (due to prominent atrial filling wave and early and late systolic impulses).
- Mid-systolic murmur at the base. Murmur may be enhanced by Valsalva maneuver, or standing may be decreased by squatting or handgrip.
- Pansystolic murmur (due to mitral regurgitation) at the apex.

Ref: *Harrison, 18/e p1968; CMDT, 51/e p404*

12. 'Shrinking lungs' in systematic lupus erythematosis is attributed to:

a. Fibrosing alveolitis
b. Vasculitic infarcts
c. Pulmonary emboli
d. Diaphragmatic myopathy

Ans. d. Diaphragmatic myopathy

Explanation

Clinical Feature of SLE or Presentation of SLE

System involved	*Presentation*
Systemic	Fatigue, malaise, fever, anorexia, weight loss
Musculoskeletal	Arthralgia and symmetrical arthritis often are features of acute SLE, but the rare joint deformities (Jaccoud's arthropathy) that occur, are a function of tendon or ligament laxity rather than erosive joint disease. Myalgia, Nonerosive polyarthritis, Hand deformities, Myopathy/myositis, Ischemic necrosis of bone are other features
Cutaneous	Photosensitivity, Malar rash(**butterfly rash**), Oral ulcers, alopecia, Discoid rash(coin-shaped lesions with hyperemic margins, Central atrophy, and depigmentation), Vasculitis rash, other {e.g., urticaria, subacute cutaneous lupus(associated with the Ro antibody)}
Hematologic	Anemia (chronic disease), Leukopenia (<4000/L), Lymphopenia (<1500/L), Thrombocytopenia (100,000/L), Lymphadenopathy, Splenomegaly, Hemolytic anemia
Neurologic	Reactive depressions, Psychoses (antibody to ribosomal P protein), Cognitive disorder, Mood disorder, Headache, Seizures, Mono-, polyneuropathy, Stroke, TIA, Acute confusional state or movement disorder, Aseptic meningitis, Myelopathy

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System involved	Presentation
Cardiopulmonary	Pleurisy, pericarditis, effusions, myocarditis (conduction abnormalities, arrhythmias, and CHF), endocarditis, Lupus pneumonitis, Coronary artery disease, Interstitial fibrosis, Pulmonary hypertension, ARDS, hemorrhage, Diaphragmatic fibrosis or diaphragm dysfunction may manifest as 'shrinking lung syndrome'.It has restrictive picture on pulmonary function testing
Renal	Proteinuria 500 mg/24 h, cellular casts, Nephrotic syndrome, End-stage renal disease
Gastrointestinal	Nonspecific (nausea, mild pain, diarrhea), Abnormal liver enzymes, Vasculitis
Thrombosis	Venous, Arterial
Ocular	Sicca syndrome, Conjunctivitis, Episcleritis, Vasculitis

Ref: *NMS Medicine, 6/e p483-6; Kumar and Clerk, 7/e p543*

13. Which of the following is a longer acting insulin?

a. Aspart
b. Glulisine
c. Lispro
d. Glargine

Ans. d. Glargine

Explanation

Type of Insulin

Type	Onset (hour)	Peak (hour)	Duration (hour)
Rapid acting			
Insulin lispro	0.2–0.3	1–1.5	3–5
Insulin aspart	0.2–0.3	1–1.5	3–5
Insulin glulisine	0.2–0.4	1–2	3–5
Short acting			
Regular (soluble)insulin	0.5–1	2–3	6–8
Intermediate acting			
Insulin –zinc suspension or Lente	1–2	8–10	20–24
Natural protamine hagedom or isophane insulin	1–2	8–10	20–24
Long insulin			
Insulin glargine	2–4		24
Insulin detemir	1–4		20–24

Ref: *KDT, 7/e p263*

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14. Which one of the following is *not* a risk factor for alcoholic liver disease?

a. Gender
b. Hepatitis C
c. Quality of alcohol
d. Malnutrition

Ans. c. Quality of alcohol

Explanation

Risk Factors for Alcoholic Liver Disease

Risk Factor	*Comment*
Quantity	In men, 40–80 g/d of ethanol produces fatty liver; 160 g/d for 10–20 years causes hepatitis or cirrhosis. Only 15% of alcoholic develop alcoholic liver disease
Gender	Women exhibit increased susceptibility to alcoholic liver disease at amounts >20 g/d
Hepatitis C	HCV infection concurrent with alcoholic liver disease is associated with younger age for severity, decreased survival
Genetics	Genepolymorphisms may include alcohol dehydrogenase, cytochrome P4502E1, and those associated with alcoholism
Malnutrition	Alcohol injury does not require malnutrition, but obesity and fatty liver from the effect of carbohydrate on the transcriptional control of lipid synthesis and transport may be factors.

Ref: *Harrison, 18/e p2589*

15. All of the following are true about renal cell carcinoma *except*:

a. Secondaries more common than Primary
b. More common in males
c. Mainly arise from renal medulla
d. Mostly occur in sixth and seventh decade

Ans. a. Secondaries more common than Primary

Explanation

Renal Cell Carcinoma

- Account for 90–95% of malignant neoplasms arising from the kidney.
- The male to female ratio is 2:1.
- Peak incidence between the ages of 50 and 70 years, may be at any age.
- Associated with cigarette smoking. Risk increased in patients who have acquired cystic disease of the kidney associated with end-stage renal disease, and for those with tuberous sclerosis.

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- First guide for MCQs having questions of 2015
- Written by experienced author who wrote many MCQs guide including *MCQs in Pediatric* by Jaypee Brothers Medical Publishers
- In explanations, important line from which questions has been asked or probably will be asked, has been incorporated.



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