TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Infarction LOA: 1	1. What is an infarct? 2. What mechanisms lead to infarction? 3. What factors determine the development of an infarct? Prompt- What influences whether an infarct will develop?	1. Area of **ischaemic necrosis** caused by **arterial** or venous occlusion **2 Arterial thrombosis, embolism**, vasospasm, haemorrhage into plaque, extrinsic vascular compression (by tumour or oedema), torsion of vessel, traumatic rupture, entrapment in hernial sac, venous thrombosis 3. Factors that determine development of an infarct • *Nature of vascular supply eg dual vs end arterial* • Rate of occlusion development – time for collaterals to develop • Vulnerability to hypoxia of the tissue type • Oxygen content of blood	Bold Bold + 2 2 of 4
Question 2 Type 2 Hypersensitivity Reaction LOA: 1	1. What is Type 2 hypersensitivity? 2. Describe the mechanisms involved giving examples for each mechanism.	**1 Hypersensitivity caused by antibodies that react with antigens present on cell surfaces or in the extracellular matrix** Antigens can be intrinsic to the membrane or matrix or extrinsic eg. Drug metabolite **2 a) Opsonisation & phagocytosis: IgG antibodies opsonise cells plus complement activation** generates C3b & C4b recognized by phagocyte Fc & protein receptors resulting in **phagocytosis & destruction of opsonised cells** ADCC- cells coated with Abs killed by monos, neutros, eosinos and NK cells **Examples:** transfusion reaction, erythroblastosis fetalis, autoimmune haemolytic anaemia, agranulocytosis,, thrombocytopaenia, drug reactions when a drug acts as a hapten **b) Complement and Fc receptor mediated inflammation: antibodies bind to fixed tissue** such as basement membranes, extracellular matrix ... **activates complement** ... generate by-products particularly chemotactic agent C5a ... direct PMN migration and C3a and C5a = **increase vascular permeability. PMNs activated** by C3a and Fc receptors... release of pro-inflammatory substances like prostaglandins, production of lysosomal enzymes, reactive O2 species **Examples:** glomerulonephritis, vascular rejection in organ grafts, vasculitis caused by ANCA, Goodpastures c) **Antibody mediated cellular dysfunction:** antibodies directed against cell surface **receptors impair or dysregulate function without causing cell injury or inflammation** **Examples:** myasthenia gravis, Graves's disease, insulin resistant diabetes, pemphigus vulgaris, pernicious anaemia	Bold (concept)_ Bold 2/3 With 1 example in each

Question 3 Community Acquired Pneumonia LOA:1	1.What organisms cause community acquired pneumonia? PROMPTS: What organisms cause atypical pneumonia? What viruses may cause atypical pneumonia?	**1 Bacterial** • **Strep pneumoniae** • Haemophilus influenza • Moraxella catarrhalis • Staph aureus • Legionella pneumophilia • Others eg klebsiella pneumonia, pseudomonas **Atypical pneumonia** • Mycoplasma pneumonia • Chlamydiae spp • Coxielle burnetti (Q fever) • RSV, parainfluenza, influenza A+B, adeno virus. SARS virus	Need • Bacteria bold +2 • Atypical 1
	2. What conditions predispose to the development of pneumonia?	2 Extremes of age, malnutrition, alcoholism Chronic conditions – CCF, COPD, DM Neurological/swallowing disorders-aspiration pneum Congenital or acquired immune deficiencies Decreased or absent splenic function- splenectomy, sickle cell disease Recent viral infection (esp staph). IVDU & staph	4 broad categories 2/3 bold
	3. What are the potential complications of pneumonia Prompt-Pathological sequelae	3 **Abscess formation** (type 3 pneumococcus, Kleb) **Empyema** **Bacteraemic dissemination** – endocarditis, pericarditis, meningitis, abscesses of kidney, spleen, brain, septic arthritis	
Question 4 Infective enterocolitis LOA: 2	1. What are the organisms that cause infectious enterocolitis?	**1. Bacterial**- E.coli, Salmonella, Shigella, Campylobacter, C.difficile, Cholera, Yersinia, Mycobacteria **Viral**- Norovirus, Rotavirus, Adenovirus Parasitic- Giardia, Amoeba, Cryptosporidium, other (nematodes, cestodes, trematodes)	Bold with 1 bact & 1 viral 3 examples total
	2. What is pseudomembranous colitis?	2. Colitis caused by **overgrowth of C. difficile** (also Salmonella, C.perfringens typeA, S.aureus) Associated with antibiotic use Forms a **pseudomembrane** made up of adherent layer of inflammatory cells and debris	Bold
	3. What are the risk factors for development of pseudomembranous colitis?	3. **Risk factors- advanced age, hospitalisation, antibiotic treatment**	2/3 Bold
	What are the clinical features of pseudomembranous colitis?	30% hospitalised patients colonised, but most aymptomatic **Fever**, leucocytosis, **abdominal pain**, cramps, hypoalbuminaemia, watery **diarrhoea**, dehydration, rarely gross bloody diarrhoea Diagnosis-usually detection of toxin Treat with metronidazole, vancomycin	Bold

Question 5 Gout LOA: 2	1. What are the causes of gout?	**Hyperuricaemia:** 1. Primary Gout (90%; often idiopathic): Overproduction (diet, unknown enzyme defects); Reduced filtration/excretion with normal production. 2. Secondary Gout (10%; known cause, secondary effect is gout): Leukaemias/tumor lysis/psoriasis, inborn errors of metabolism (overproduction with increased excretion); Chronic renal disease (reduced excretion).	Hyperuricaemia + 1 Primary and 1 Secondary cause Or 1 overproduction and 1 decreased excretion
	2. Describe the pathogenesis of acute gouty arthritis. Prompt- What are the steps involved?	1. Hyperuricaemia 2. **Precipitation of urate crystals into joints** (in synovium / cartilage) 3. **Release of crystals into synovial fluid** (?trauma) 4. **Inflammatory response initiated** – crystals phagocytosed by macrophages and neutrophils; release of inflammatory mediators by macrophages (interleukins, cytokines (IL-1B)); resulting in further neutrophil chemotaxis; neutrophils also release inflammatory mediators (free radicals, leukotrienes (LT B4), lysosomal enzymes) – acute arthritis.	Bold to pass
	3. (only if needed) What factors contribute to the conversion of asymptomatic hyperuricaemia into gout	Age & duration of hyperuricaemia; genetic predisposition/etoh/obesity/drugs e.g. thiazides/lead toxicity	

ACEM PRIMARY 2013/1 Pathology VIVA **Afternoon Session 2** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: **Oedema formation** LOA: 1	1. What are the mechanisms of oedema formation?	**1.↑ hydrostatic pressure –** impaired venous return, eg CHF,Constrictive pericarditis,ascites,venous obstruction(internal/external +immobility), arteriolar dilatation eg heat **Decr plasm oncotic pressure (hypoproteinaemia)** – nephrotic syndrome, ,malnutrition, protein losing enteropathy. **Lymphatic obstruction** - inflammatory, neoplastic, post-surgery/radiation **Sodium and water retention** –XS salt with renal insufficiency, incr renin-angiotensin-aldosterone secretion **Inflammation** –acute/chronic, anglogenesis	3 out of 5 bold, example from each
	2. What is the pathogenesis of cardiogenic oedema?	**2.** Decreased cardiac output, decr renal perfusion, secondary aldosteronism, Incr blood volume, incr venous pressure	At least 3 steps.
Question 2 Hep B LOA: 2	1. How can Hepatitis B infection be transmitted?	1. Vertical – perinatal during childbirth Horizontal – skin or mucosal breaches - intercourse - shared needles / syringes in IVDU - blood transfusion	**3/5**
	2. What are the potential outcomes following ACUTE Hepatitis B infection?	**2. Recovery** >90% Fulminant hepatitis necrosis <0.5% **Chronic Hepatitis** <5% - cirrhosis 12-20% +/- hepatocellular Ca - healthy **carrier state** - non progressive chronic hepatitis <2%	**Bold to pass**
	3. What are the serum markers of ACUTE infection with Hepatitis B? Prompt: What antigens and antibodies are present during acute hepatitis B?	**3. HBeAg, HBsAg** HBV-DNA, **Anti-HBc IgM** Anti-HBe, (not Anti-HBs)	**2/3 Bold**

Question 3 IHD LOA: 1	1. In myocardial infarction, what sequence of events leads to acute coronary artery occlusion? Prompt- pathological events 2. Describe the time course of myocardial injury after acute coronary artery occlusion. Prompt- What happens to the myocardial tissue over time?	**1. Sudden change in atheromatous plaque** haemorrhage, erosion, ulceration, rupture, fissure **Platelet** adherence, activation & aggregation leading to microthromi Vasospasm from plt released mediators Activation of coagulation pathway causing **thrombus** Vessel occlusion **2. Reversible** • cessation of aerobic metabolism seconds • decreased ATP production • lactic acid production (noxious metabolites) • loss of contractility, acute heart failure 1 min • ultrastructural changes – myofibrillar relaxation, glycogen depletion, cell & mitochondrial swelling few minutes • ATP depletion up to 40 min **Irreversible** • myocyte injury – defects in sarcolemmal membrane and cell leakage 20 - 40min • initially subendocardial then transmural myocyte death • microvascular injury 1 hour • coagulation necrosis > 2 hours (more protracted if collaterals)	**Bold to pass** **Bold to pass with minutes to hours concept**
Question 4 Asthma LOA: 1	1. What are the pathological features of asthma? 2. Asthma may be categorized as atopic or non-atopic. What are the characteristics of each of these types? Prompt - What is the underlying mechanism of atopic asthma? What are some of the triggers? 3. In atopic asthma, what happens in the early-phase reaction?	**1. Increase airway responsiveness** to variety of stimuli; episodic **bronchoconstriction**; bronchial wall **inflammation**; incr mucus 2. Atopic- **IgE mediated type1 hypersensitivity** (allergen sensitisation); **environmental allergen triggers** e.g. dust, pollen, dander e.g. house dust mite, foods. Family Hx common; skin test positive to allergen; RAST shows allergen sensitivity Non-Atopic- **hyperirritability of bronchial tree**-no allergen sensitisation, skin tests usual negative; family Hx uncommon; triggers-resp infection secondary viruses common; inhaled air pollutants may contribute (SO2, ozone, NO2) **3. Allergen exposure**=> IgE. • Reexposure=> Mast cell degranulation with release of cytokines/mediators • =>bronchoconstriction (via subepithelial vagal/parasympathetic receptors), • mucus production, • vasodilation with increased vasc permeability	Bold Bold. One trigger for each Bold plus concept

Question 5 Obstructive uropathy LOA: 2	1. What are the causes of urinary tract obstruction?	1. Congenital- urethral valves & strictures; bladder neck obstruction; ureteropelvic narrowing; reflux **Calculi; Prostatic hypertrophy** **Tumors**- prostate; bladder; cervix/uterus; other Inflammation- prostatitis; urethritis; ureteritis; retroperitoneal fibrosis Sloughed papillae, clots; Pregnancy; Uterine prolapse; cystocele Functional- neurogenic (spinal cord/diabetic); dysfunctional; ureter or bladder	Bold plus one other.
	2. What are the clinical features of acute obstruction?	**2. Pain** due to distension or Sx of underlying process e.g. renal colic, LUTS in prostatic disease asymptomatic (in Unilateral complete or partial) Polyuria and nocturia. Calculi, HT, distal tubular acidosis- (In Bilateral partial) oligo/anuria, hyperkalaemia, incr urea & creat- (in Complete bilateral)	Bold
	3. What are the possible clinical sequelae of urinary tract obstruction?	3. Infection Stone formation Atrophy/hydronephrosis/obstructive uropathy (if chronic)- => renal failure Complications of renal failure.	3/5

ACEM PRIMARY 2013/1 Pathology VIVA **Morning Session 3** ***Candidate Number:*** **AGREED MARK:**

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 Vascular changes of acute inflammation	1. In acute inflammation what changes occur in blood vessels? Prompt: What happens next? 2. What are the mechanisms for the increased vascular permeability seen in acute inflammation?	1. Changes in blood flow: (transient constriction), **vasodilation** (NO mediated) lead to **increased flow** • **Increased permeability,** loss of protein-rich fluid • Fluid loss & dilation lead to **stasis/congestion** • Leukocytes accum at vasc endothelium, endothelium expresses adhesion molecs, leuks adhere & migrate out 2. Chem mediated **endothelial cell contraction** (caused by eg histamine, LKT, sub P) • Endothelial injury direct/microbes/leuks eg burns • Increased transcytosis of fluids/proteins via channels of connected vesicles/vacuoles (vesiculovacuolar organelles) stim by factors eg VEGF	3/4 Bold 2/3 must include bold
Question 2 LOA: 2 The normal immune response	1. What are the major classes of lymphocytes? 2. What is the role of each class of lymphocytes in the normal immune system? Prompt- What is the role of B-cells? What is the role of T-Cells?	1. B lymphocytes CD4+ helper T- Lymphocytes CD8+ Cytotoxic T Lymphocytes Natural Killer (NK) Cells 2. Adaptive immunity – circulate widely & rec-circulate esp Ts - respond to foreign substances/Ag. Can become effector or memory cells B cells: recognise Ag via memb IgM/IgD –plasma cell -secretes Ig/Ab = humoral immunity. (B cells also have compl R, FcR, CD40) T cells: Ag specific T cell R - binds to Ag on cells (on MHC molecules on APCs) – activates cell depending on type = cell-mediated immunity CD4/T helper recog class II MHC bound Ag: cytokine release – leads to macrophage activation, inflam, B cell stimulation CD8/ T cytotoxic recog class I MHC bound Ag: infected cell destruction NK Cells- kill inf&tumor cells. No prior exp needed. Healthy cell Class I MHC=>inhibits NK. Can secrete cytokines=>inflame	B&T B-Humoral plus concept T-Cell mediated plus concept
Question 3 Pulmonary Embolism LOA: 1	1. From where do pulmonary thromboemboli originate? 2. What are some risk factors for thrombus formation? 3. What are the clinical effects of pulmonary thromboemboli?	1/95% **arise in the deep veins of the leg** – pass up to R side of heart and into pulm vasculature. Size determines where they lodge. 2. **Primary** – (genetic factors) – factor 5 Leiden, protein C+S deficiency, antiphospholipid syn **Secondary-** (acquired) – stasis/Immobilisation, long haul flights, active malignancy, trauma/burns/surgery, pregnancy, OCP. Indwelling catheters 3. most clinically silent 60-80%, Cough, SOB, fever, CP, haemoptysis, tachy-cardia/pnoea through to sudden death,cor pulmonale,CVS collapse Pulm haemorrhage / infarction, over time multiple emboli may cause pulm hypertension & cor pulmonale	Bold to pass (exact % not required but rough idea) At least one example from Primary, and 2 from secondary 5 features

Question 4 Portal Hypertension LOA: 2	1/What are the causes of portal hypertension? May need to prompt for examples/classification. 2/What are the clinical consequences of portal hypertension? 3/What mechanisms are involved in the formation of Ascites?	1/ Incr resistance to portal blood flow Prehepatic – portal vein thrombosis or narrowing Hepatic – (most important)- **cirrhosis**, massive fatty change, schistosomiasis, granulomatous disease eg sarcoid/Tb Post hepatic - severe RHF, constrictive pericarditis hepatic vein occlusion 2/ **Ascites** – with potential for infection **Porto-systemic shunts** : varices, haemorrhoids, spider naevi Congestive **splenomegaly** – thrombocytopaenia/pancytopaenia **Hepatic encephalopathy** 3/ Sinusoidal hypertension – Starling forces : Incr pressure and decr albumin Incr formation of hepatic lymph – exceeds capacity of thoracic duct- percolates into peritoneum Splanchnic vasodilation with dec BP=> Renal retention of sodium and water due to secondary hyperaldosteronism	Bold. One from each other group 2/4 bold 2/3 concepts
Question 5 Traumatic CNS Injury LOA: 1	1/ What types of intracranial bleeding can be seen in a patient with a head injury? 2/What sequence of events occur in an extradural haemorrhage 3/Define concussion and what are its clinical features?	1/ **Extradural** **Subdural** **Subarachnoid (including intraventricular)** **Intra-parenchymal** 2/Dural **artery** (eg. middle meningeal) tear**, usually associated with a skull fracture** Strips off the dura from the skull May be a lucid period before ALOC 3/**Altered consciousness secondary to a head injury** **Transient** neurological dysfunction Transient resp arrest Transient loss of reflexes (pathogenesis is unclear, may be dysregulation of RAS) Features inc headache, amnesia, N&V, Concentration and Memory issues, perseveration, irritability, behaviour/personality changes, dexterity loss, neuropsychiatric syndromes	3 of 4 Must get bold Must get bold 3 features