Pathology 68 Questions

1. Regarding acute inflammation page 52-55):

    A. Circulating inflammatory cells include nerutrophils, monocytes, fibroblasts and lymphocytes
    B. Acute inflammation involves proliferation of blood vessels and connective tissue
    C. Vasodilation initially occurs by opening of venules
    D. Increased vascular permeability is the hallmark of acute inflammation
    E. Relaxation of endothelial cells leads to increased vascular leakage

2. Regarding acute inflammation (page 55-57):

    A. Contraction of arteriolar and capillary endothelial cells results in increased vascular leakage
    B. Direct endothelial injury causes leakage of venules, capillaries and arterioles and is known as the immediate sustained response
    C. Leakage ceases when capillaries start to regenerate
    D. Rolling and adhesion of leakocytes along the endothelial wall is known as diapedesis
    E. Only neutrophils traverse the basement membrane into the extravascular space

3. Regarding binding of leukocytes and transmigration (page 58-59)

    A. On exposure to histamine, P-selectin redistributes to the endothelial surface to bind leukocytes
    B. Cytokines induce E-selectin on endothelial membrane almost immediately
    C. Leukocyte diapedesis occurs predominantly in capillaries
    D. Neutrophils are always the first inflammatory cells to arrive at a site of inflammation
    E. After 24hours, monocytes are the predominant cell type at all sites of infection

4. Regarding the action of leukocytes in the extravascular space (page 60-63):

    A. C4 is the component of complement that acts as a chemoattractant
    B. Leukocytes move via a pseudopod containing contractile actin filaments
    C. Bacteria and extraneous matter are labeled for phagocytosis by opsonins such as C3b and IgG (F fragment)
    D. Lysosomal granules remain intracellular during phagocytosis
    E. pH in the leukocyte is lowered following phagocytosis

5. Regarding chemical mediators of inflammation (:

    A. Histamine and serotonin are plasma derived mediators of inflammation
    B. Histamine causes constriction of all blood vessels
    C. C3 and C4 are the most important inflammatory mediators of the complement components
    D. Bradykinin decreases vascular permeability
    E. Inflammatory properties of thrombin include increased leukocyte adhesion and fibroblast proliferation

6. Regarding Eiconasoids (page 68-69):

    A. Arachidonic acid is formed from cell membrane phospholipids by cyclo-oxygenase
    B. Vascular endothelium produces thomboxane A2
    C. Thromboxane A2 causes vasoconstriction and promotes platelet aggregation
    D. Prostaglandins and prostacycline cause vasoconstriction
    E. Leukotrienes cause bronchodilation

7. Regarding chemical mediators of inflammation (page 67-74):

    A. IL-1 and TNF are the main cytokine mediators of inflammation
    B. NO increases platelet aggregation and adhesion
    C. Acid proteases from neutrophils and monocytes degrade extracellular components
    D. Substance-P is a neuropeptide causing vasoconstriction
    E. Leukotrienes are major mediators o pain in inflammation

8. Regarding Chronic Inflammation (page 75-):

    A. It is always preceeded by acute inflammation
    B. It is primarily mediated by monocytes
    C. Macrophages only accumulate by recruitment of monocytes from the circulation
    D. Tissue destruction is a hallmark feature
    E. Macrophages are activated by monokines realeased from lymphocytes.

9. Regarding Chronic inflammation (page 79-82):

    A. Repair is due to regeneration of parenchymal cells
    B. Newly developing capillary vessels extend via exing basement membrane protrusions
    C. The main cell in granulomatous inflammation is a giant cell
    D. Accumulation of modified 'epitheleoid' macrophages characterizes granulomatous inflammation
    E. Caeseous necrosis is a feature of all granulomas

10. Regarding the morphology and systemic effects of inflammation (page):

    A. Serous inflammation contains fluid derived from blood
    B. Fibrinous exudate is characteristic of inflammation in body cavities such as pleara and peritoneum
    C. Purulent inflammaton fluid is secreted by bacterial organisms
    D. Fever is due to skin vasodilation
    E. Acute leukocytosis is due to increased production by bone marrow

11. In wound healing by primary intention (page 85, 88):

    A. Collagen fibres are present by 24hours
    B. Leukocytic infiltrate and oedema disappear by day 5
    C. Epithelial cells migrate and grow alone the cut dermis between 24-48hours
    D. Tensile strength is about 50% normal when sutures are removed at one week
    E. Neovascularisation is maximal at day 3

12. Regarding wound healing (page 86-90):

    A. Myofibroblasts assist the phenomenon of wound contraction
    B. Metalloproteinases produce collagen
    C. Vitamin C inhibits collagen synthesis
    D. Keloid scarring is due to formation of excessive granulation tissue
    E. Keloid scarring is more common in pale people

13. Regarding the pathology of oedema (page 93-97):

    A. A transudate has a specific gravity greater than 1.020
    B. CCF directly causes oedema by increased hydrostatic pressure and indirectly through a decrease in rennin
    C. Normal end-arteriolar hydrostatic pressure is about 15mmHg
    D. Cirrhosis may lead to secondary aldosteronism
    E. Cerebral oedema does not involve neurons or glial cells

14. As part of normal haemostasis (page 100-101):

    A. Activated platelets are prevented from adhering to uninjured cells by NO and PGI2
    B. Thromboxane A2 and PGI2 have similar effects
    C. Thrombomodulin activates thrombin
    D. Protein C is a cofactor for Protein S
    E. Protein C inactivates factor Xa

15. Regarding haemostasis (page 101-105):

    A. VWF is essential for platelet-platelet adhesion
    B. GpIIb-IIIa forms a complex with vWF
    C. Haemostatic plugs do not contain red or white blood cells
    D. Activation of factor VIII marks the conversion of the extrinsic and intrinsic clotting pathways
    E. Cytokines IL-1 and TNF stimulate synthesis of t-PA inhibitor

16. Regarding arterial and venous thrombosis (page):

    A. An arterial thrombus 'tail' develops in the direction of blood flow
    B. The 'tail' of a venous thrombus is the most likely section to embolise
    C. Embolism of an arterial thrombosis usually occurs in fragments
    D. A mural thrombus occludes an entire vessel
    E. Thrombosis of the saphenous system is at a high risk of embolisation

17. Regarding pulmonary embolism (p111):

    A. 70% arise from the large deep veins of the lower limb
    B. 60-80% are 'silent'
    C. obstruction of large pulmonary vessels causes pulmonary infarction
    D. pulmonary hypertension and chronic right heart failure are frequent sequelae
    E. sudden death, cardiovascular collapse and acute right heart failure may result when >40% of total pulmonary vasculature is obstructed

18. Regarding different types of embolism (p112-114)

    A. Brain is the most common site of systemic arterial embolism
    B. Rapidity of progression correlates well with the volume of amniotic fluid infused in amniotic fluid embolism
    C. Pulmonary insufficiency, anaemia, thrombocytosis and neurologic symptoms constitute the 'fat embolism syndrome'
    D. Air/gas embolism to the lungs does not cause dyspnoea
    E. Decompression sickness is a form of gas embolism

19. Regarding infarction (page 114-116):

    A. All infarction results in coagulative necrosis
    B. 'Red' infarcts are encountered in arterial occlusion of normal solid tissue
    C. the lung, small intestine and brain may all show haemorrhagic (red) infarcts)
    D. Infarction of the hand is relatively common as it is an end appendage.
    E. Fibroblasts in myocardium are highly sensitive to hypoxia

20. Regarding septic shock (page 117-):

    A. Is mostly caused by gram positive bacteria
    B. IL-! and TNFα are key cytokines
    C. The distinction between progressive and irreversible shock is when urine production ceases
    D. Endotoxin bacterial wall lipopolysaccharides from a complex with platelets
    E. It causes increased corticolipids in the adrenals at autopsy.

21. Regarding T lymphocytes (page 172):

    A. They make up 10-20% of circulating lymphocytes
    B. T-cells with CD4 proteins bind to MHC-I molecules on antigen presenting cells
    C. CD4 protein is expressed on more T cells than CD8
    D. CD4 T-cells secrete IL-4 and IL-5
    E. CD8 T cells are known as 'helper' or 'inducer' cells

22. Regarding B lymphocytes (page 172-173):

    A. B-cells require antigen to be presented by another cell
    B. On antigenic stimulation B-cells form plasma cells which mediate humoral immunity
    C. IgM is bound to the surface of some B cells
    D. B cells are found in the paracortical region of lymph nodes
    E. Igα and Igβ bind to antigen on the B cell surface.

23. Regarding components of the immune system (173-175):

    A. Macrophages are essential in presenting antigen to B cells
    B. Dendritic cells and Langerhan's cells are specialized phagocytic cells
    C. Following sensitization, Natural Killer Cells are able to destroy other cells
    D. Cytokines are produced only by mast cells
    E. Cytokines bind to specific high affinity receptors

24. Regarding Type I hypersensitivity reactions (page 179-181):

    A. Require the assistance of the TH1 subset of CD4+ T cells
    B. Involve recruitment of mast cells from the blood stream
    C. Involve release of histamine from mast cells as a secondary mediator
    D. Leukotrienes are extremely important secondary mediators
    E. Eosinophils and mast cells release primary mediators

25. Regarding Type I hypersensitivity (page 179-181):

    A. The 'late' phase is resolved in 24 hours
    B. The 'late' phase is characterized by infiltration of eosinophils, other inflammatory cells and tissue destruction
    C. Systemic anaphylaxis always causes pulmonary oedema
    D. Leukotrienes do not contribute to the initial phase reaction
    E. TH1 cells assist eosinophil growth and activation

26. Type II hypersensitivity (page 182-184):

    A. Is mediated by mast cells
    B. Always involves comletment mediated opsonisation of cells
    C. Destruction of cells may be due to membrane attack complexes
    D. Goodpastures syndrome is an example of antibody-dependent cell mediated cytotoxicity (ADCC)
    E. Myasthaenia gravis is an example of antibody mediated cellular destruction

27. Type III hypersensitivity (page 184-185):

    A. Is cell mediated
    B. Includes SLE
    C. Is usuallymore severe for larger immune complexes
    D. Is more severe when the mononuclear phagocytic system is intact
    E. Does not involve changes in vascular permeability

28. Regarding type III and type IV hypersensitivity reactions (page 184-):

    A. Single large antigen exposure usually results in chronic type III hypersensitivity disease
    B. The 'Arthus reaction' is an example of type III hypersensitivity
    C. The Mantoux test is an example of the 'Arthus reaction'
    D. TH2 T cells are central to the development of type IV hypersensitivity
    E. Contact dermatitis is IgE mediated.

29. Regarding vascular wall response to injury (page 471):

    A. The endothelial lining must be denuded for thromboses to occur
    B. Vasc smooth muscle cells are each surrounded by a basal lamina
    C. Vascular smooth muscle cells are confined to the media at all times
    D. Vasc smooth muscle cells become contractile on migration to the intima
    E. Migration/proliferation of smooth muscle cells is inhibited by PDGF

30. Regarding vascular disease (page 472-473):

    A. Vascular diseases affect arteries and veins evenly
    B. Arteriosclerosis is a subtype of atherosclerosis
    C. Any artery may be affected by atherosclerosis
    D. ICA is usually involved in more atherosclerotic lesions than the popliteal artery
    E. Atheromas are usually occlusive in large arteries

31. Regarding atherosclerosis (page 474-479):

    A. Hyperlipidaemia, HT, smoking and increasing age are the most significant risk factors for atherosclerosis
    B. Increased cholesterol is a higher risk factor than hypertension for atherogenesis in people over 45yrs
    C. Decreased risk of dying from IHD occurs within a few years of smoking cessation
    D. Diabetes double the frequency of lower limb gangrene compared to non-diabetics
    E. The key morphologic feature is atrophy of the media

32. Regarding atherosclerotic changes (page 476-478):

    A. Fatty streaks are only present in children of developed countries
    B. Atheromatous plaques do not always impinge on the lumen of the artery
    C. Macrophages are the only cells that form foam cells
    D. Aortic lesions are more prominent at the ostia of its major branches
    E. Foam cells are typically located in the superficial fibrous cap of atheromatous plaques

33. Regarding complications of atheromatous lesions page 478):

    A. Atheromatous plaques rarely calcify
    B. The media is involved in disease process most frequently in smaller vessels
    C. Aneurismal dilatation is unrelated to formation of atheromatous plaques
    D. Superficial thrombosis usually occurs over fissured or ulcerated lesions
    E. Haemorrhage into a plaques is most uncommon in the coronary arteries

34. Regarding pathogenesis of atherosclerosis (page 479-482):

    A. Vascular cell adhesion molecule (VCAM-1) and intercellular adhesionmolecule (ICAM-1) are expressed on the smooth muscle cells of atheromatous plaques
    B. The scavenger receptor on macrophages allows the uptake of oxidized LDL
    C. Foam cells are not present in ‘fatty streaks’
    D. PDGF is produced only by platelets
    E. Lipoprotein abnormalities are seen in less than 50% of myocardial infarction survivors

35. Regarding Cardiac Structure (page 517-518):

    A. The normal male heart weighs 400g
    B. Normal left ventricular free wall is 1.3-1.5cm thick
    C. The lunula of heart valves is the insertion point to the annulus
    D. Lesions in the lunula of valves significantly impair valve competence
    E. Chordae tendinae connect each MV leaflet to its own separtate papillary muscle

36. Regarding cardiac structure (page 518):

    A. Cardiac valves leaflets have their own vascular supply
    B. Myocytes only make up 25% of cardiac tissue volume
    C. Collagen is abundant in normal myocardium
    D. Purkinje cells do not contain contractile filaments
    E. 4/5 of the population have a right dominant coronary circulation

37. Regarding Cardiac structure and changes within it (page 519):

    A. occlusion of the RCA does not cause LV damage
    B. Collateral blood flow is important in normal blood supply
    C. The subendocardium is well perfused by collateral flow
    D. Brown atrophy is partly due to lipofuscin deposition in the aging heat
    E. Elderly myocardium contains the same number of myocytes as younger

38. The aging heat (page 519):

    A. Shows deacreased subepicardial fat
    B. Should not normally develop basophilic degeneration
    C. Shows increased LV chamber size
    D. Frequently develops clinical complications due to calcification of the mitral annulus
    E. Shows Lambl's excrescences on valves of nearly over 60yo's

39. In congestive cardiac failure (page 520):

    A. Hypertrophied cardiac cells have more sarcomeres
    B. Pressure loaded ventricles develop concentric hypertrophy
    C. Volume overloading shows an increase in the the ratio of the wall thickness to the cavity radius
    D. Increased myocyte size is accompanied by increased capillary density
    E. Gene expression is not affected

40. Regarding Congestive Cardiac Failure (page 521-522):

    A. LVH is not a risk factor for IHD
    B. Physiologic hypertrophy has marked deleterious effects
    C. Increased muscle mass and wall tension are major determinants o increased O2 demand
    D. A damaged, decompensated heart is morphologically different to a damaged but compensated one
    E. Hypoxic and congestive changes in peripheral tissues are unique to CHF

41. Left heart failure (page 522):

    A. Is most commonly caused by valve disease
    B. Commonly causes pleural effusions
    C. May result in haemosiderin containing alveolar macrophages
    D. Is uncommonly accompanied by cough
    E. Leads to renal congestion

42. Right heart failure (page 523):

    A. Frequently occurs as an isolated entity
    B. Is clinically simulated by constrictive pericarditis
    C. Results in congested peripheral hepatic lobules with pale centers
    D. Does not cause splenomegaly
    E. Causes a renal condition known as anasarca

43. Regarding IHD (page 524-525):

    A. IDH accounts for 50% of cardiac mortality
    B. Only one coronary artery is affected in most IHD
    C. 90% of patients with IHD have advanced stenosing coronary atherosclerosis
    D. Significant stenosing plaques in the RCA are usually in the middle 1/3
    E. Completely stenosing plaques are the cause of acute coronary syndromes in most patients

44. Regarding IHD (page 526):

    A. Incidence of AMI peaks between 9 and 11am
    B. Plaques that rupture tend to have a concentric configuration
    C. Plaque fissures frequently occur at the central portion of the fibrous cap
    D. Thrombus formation over a fissured plaque always results in symptoms
    E. Thrombosis is not involved in UAP

45. Regarding Angina (page 528):

    A. Stable angina is commonly associated with >90% coronary artery stenosis
    B. ECG ST depression occurs in stable and Prinzmetal's angina
    C. Ischaemia in Prinzmetal's angina only occurs in the subendocardial region
    D. Prinzmetal's angina rarely relates to BP, HR or activity level
    E. Thromboemboli are rare in UAP

46. Subendocardial Infarction (page 529):

    A. Is more common than transmural
    B. Necrotic area often spreads laterally beyond the perfusion territory of a single coronary artery
    C. Is usually associated with a single vessel lesion
    D. Is commonly associated with plaque rupture
    E. Is reliably predicted by absence of Q waves on ECG

47. Regarding Myocardial infarction (page 529-530):

    A. 5% infarcts occur in people under 40yrs
    B. 90% subendocardial infarts are due to occlusive intracoronary thrombus overlying a fissured plaque
    C. occlusive thromus occurring in a severely stenosed vessel has a worse prognosis
    D. ADP is a potent vasodilator
    E. Tissue thromboplastin activates the intrinsic coagulation pathway

48. Regarding Myocardial infarction (page 530):

    A. Angiography shows a thrombosed coronary artery in 90% MI when performed at 12hours
    B. The "area at risk" is most pronounced in the subendocardium
    C. Emboli from vegetative endocarditis does not cause coronary occlusion
    D. Anaerobic glycolysis occurs within minutes of ischaemia
    E. Loss of contractility is a late manifestation of ischaemia

49. In Myocardial infaction (page 530-531):

    A. Irreversible injury occurs only after 40mins ischaemia
    B. Majority of patients developming VF do so due to acute myocardial infarction
    C. Extent of necrosi in humans is usuallycomplete at 6hours
    D. Septal involvement is rare
    E. Isolated RV infarction occurs in only 1-3%of cases

50. Regarding MI (page 532-533):

    A. LCX lesion make up 30-40% critical stenoses
    B. Gross examination shows changes at 6-12 hours
    C. Dehydrogenases increase in ishaemic region at 2-3hours
    D. Acute inflammation is seen at 2-3 days post infarct
    E. A fibrous scar does not form until 12weeks

51. Regarding MI and reperfusion (page 535-537):

    A. Thrombolysis reestablishes patency of occluded CA in 90% of cases
    B. Reperfusion may prevent all necrosis if done within 15-20mins
    C. Contraction bands are seen in all cardiac cells following ishaemia
    D. Reperfusion injury is accelerated disintegration of already dead myocytes
    E. Myocardial 'stunning' only exists for a few hours

52. Regarding AMI (page 537):

    A. MI is assympotomatic in 10-15%
    B. LDH rises after 8hours
    C. Cardiac tissue is the only tissue containing CKMB isoenzyme
    D. Sudden cardiac death occurs within 1-2 hours of AMI in 5% patients
    E. LV failure occurs in 15% AMI

53. Regarding complications of MI (page 539):

    A. Cardiogenic shock has an almost 70% mortality
    B. Rupture of the septum is more common than rupture of the free wall
    C. Cardiac rupture is most commonly seen within 2 days of infarct
    D. Fibrinous or fibrinohaemorrhagic pericarditis occurs usually at 1-2weeks post infacrt
    E. Infarct is expansion is new necrosis adjacent to an existing infact

54. Regarding complications and prognosis following AMI (page 539):

    A. Ventricular aneurysm most commonly results from a posterior infarct
    B. Anterior infarcts are the most likely to develop conduction abnormalities
    C. Subendocardial infarcts do not result in the formation of mural thrombus
    D. Including patients dying before reaching hospital, 1 year mortality for MI is 35%
    E. Early mortality rate during hospitalization is 4%

55. Regarding Sudden Cardiac Death (page 541):

    A. It ismost commonly caused by congenital structural abnormalities
    B. Stenosis greater than 75% is found in one or more coronary arteries in 80-90% victims
    C. Refers to death occurring within 24hours of symptoms onset
    D. Increased parasympathetic activity may contribute to formation of lethal arrhythmias
    E. In those saved by rapid intervention, new myocardial infarction is found in 50%

56. Regarding lung microstructure (page 673-674):

    A. The vocal cords are lined by pseudostratified epithelial cells
    B. Bronchial mucosa contains neuroendocrine cells
    C. Type I alveolar cells secrete surfactant
    D. Type II alveolar cells line >95% of the alveolar surface
    E. Alveolar walls are a soid continuous layer

57. Regarding atelectasis (page 675-676):

    A. Atelectasis is most frequently irreversible
    B. Absorption atelectasis results in deviation of the trachea away from the affected side
    C. Obstructive atelectasis is caused principally by excessive secretions or exudates within smaller bronchi
    D. Compressive atelectasis most commonly results from tumour masses
    E. Contraction atelectasis develops due to loss of pulmonary surfactant

58. Pulmonary oedema/congestion (page 676):

    A. Is most commonly due to microvascular injury
    B. Is characterized by heavy, wet lungs regardless of clinical setting
    C. May lead to lipofuscin laden macrophages known as ‘heart failure cells’
    D. May lead to intracellular changes known as brown induration
    E. Does not result from primary injury to alveolar cells

59. ARDS (page 676-677):

    A. Is a subtype of haemodynamic pulmonary oedema
    B. Does not show development of hyaline membranes
    C. Causes decreased type II alveolar cells
    D. Final common pathway is diffuse damage to alveolar capillary walls
    E. Initial injury is most frequently to alveolar epithelium

60. Regarding ARDS (676-678):

    A. Neutrophils do not play a large role in ARDS
    B. Does not develop in systemic neutropaenia
    C. Endotoxin is likely to be important in may cases
    D. Early CXR shows diffuse basal infiltrates
    E. Changes in the the lungs are diffuse and uniform

61. Regarding Pulmonary Embolus (page 679-680):

    A. PE can be attributed to DVT in 60%
    B. May result in haemorrhage rather than infarction
    C. Majority cause infarction
    D. Most infarcts involve upper lobes
    E. LDH levels are not elevated

62. Regarding Emphysema (page 683-684):

    A. Fibrosis is a feature
    B. Both emphysematous and normal airspaces coexist in the same acinus in centrilobular emphysema
    C. Walls of airspaces rarely contain pigment
    D. Smoking typically causes pan-acinar emphysema
    E. Centrilobular emphysema typically involves lower lung zones more

63. Regarding emphysema (page 684-686):

    A. Core pulmonale is common in emphysema predominant COPD
    B. 'irregular'emphysema is the subtype most likely underlying spontaneous pneumothorax
    C. alpha1-AT is normally present in serum, tissue fluids and macrophages
    D. more neutrophils are sequestered in upper than lower zones of the lung
    E. O2 free radicals stimulate alpha1-AT activity

64. Regarding emphysema (page 686-687):

    A. Smoking stimulates increased neutrophil presence in alveoli
    B. Cetriacinar and panacinar emphysema have similar macroscopic appearance
    C. Emphesema does not manifest linically until 1/5 functioning pulmonary parenchyma is incapacitated
    D. Diffusing capacity does not change
    E. Hypercapnea frequently occurs in emphesema predominant COPD

65. Chronic bronchitis (page 684, 688):

    A. Results in cor pulmonale less than emphesema
    B. Is defined by persistent cough with sputum production for at least 2 months in 3 consecutive years
    C. Always shows airflow limitation
    D. The hallmark is infection
    E. Causes atypical metaplasia and dysplasia of respiratory epithelium

66. Regarding Chronic Bronchitis (page 688-689):

    A. Increased mucus secretion is primarily due to increased goblet cells
    B. Infection has a primary role in initiation
    C. Cigarette smoke interferes with both ciliary action and ability of bronchial and alveolar leukocytes to clear bacteria
    D. The Reid Index is decreased
    E. Bronchioloes show marked dilatation

67. Regarding Asthma (page 690-691):

    A. All asthma is IgE mediated
    B. IgE mediated asthma elicits an acute immediate response and a late phase response
    C. Histamine does not cause cholinergic reflex actions
    D. Leukotriene B4 is a secondary mediator
    E. PAF is a primary mediator

68. Regarding Asthma (page 692):

    A. Non-atopic asthma is most frequently triggered by bacterial infection
    B. Aspirin sensitive asthmatics also experience urticaria
    C. Mucus plugs are not found macroscopically
    D. Basement membrane is unchanged
    E. Bronchial muscle does not show changes

1. D
2. B
3. A
4. C
5. E
6. C
7. A
8. D
9. D
10. B
11. C
12. A
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