NEET PG Pathology Sample Paper-9

Duration: 20 Minutes **Maximum Marks: 100**

Instructions

- This paper contains **25** Multiple Choice Questions.
- Each correct answer carries **+4** mark. Incorrect answer: **−1** marks. Only **one** correct option.
- Unattempted questions carry **0** marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

Q1. A 67-year-old female with osteoporosis undergoes a vertebral compression fracture. Histological examination of the fractured bone reveals abnormal mineralization with excessive unmineralized osteoid accumulation within the bone matrix. Which of the following represents the primary biochemical disturbance in this condition?

(A) Defective type I collagen crosslinking preventing proper matrix formation

(B) Failure of osteoid mineralization despite normal biochemical availability of mineral ions

(C) Excessive osteoclastic resorption leading to net loss of mineralized bone mass

(D) Impaired osteoblastic synthesis of alkaline phosphatase enzyme

Q2. A 29-year-old male with a history of inflammatory bowel disease develops sudden-onset abdominal pain. Exploratory laparotomy reveals a perforated small bowel with extensive necrotic ulceration and hemorrhage. Histological examination shows transmural inflammation with destruction of the muscularis propria. This pattern of tissue damage is primarily driven by which pathological process?

(A) Ischemic coagulative necrosis from vascular insufficiency

(B) Enzymatic liquefactive necrosis from inflammatory cell infiltration

(C) Caseous necrosis from granulomatous inflammation

(D) Fat necrosis from pancreatic enzyme spillage

Q3. A 52-year-old male with previously undiagnosed hypertension presents with acute myocardial infarction. Autopsy reveals a myocardial infarct spanning the anterior wall. Histological examination demonstrates contraction band necrosis with hypereosinophilic myofibers displaying a characteristic "wavy fiber" appearance. At what point post-infarction would this histological feature most prominently manifest?

(A) Within the first 2-4 hours (hyperacute phase)

(B) 24-48 hours (early acute phase)

(C) 3-7 days (organizing phase)

(D) 2-8 weeks (chronic phase)

Q4. A 45-year-old female with a family history of premature coronary artery disease undergoes genetic testing. Sequencing reveals a heterozygous missense mutation in the APOB gene affecting apolipoprotein B-100 function. This genetic alteration impairs the recognition of which lipoprotein clearance receptor?

(A) LDL receptor-mediated endocytosis

(B) Scavenger receptor-mediated uptake

(C) HDL receptor-facilitated reverse cholesterol transport

(D) VLDL receptor-dependent triglyceride metabolism

Q5. A 38-year-old construction worker presents with progressive dyspnea and chronic productive cough. Chest X-ray reveals bilateral upper lobe-predominant reticular opacities with "eggshell" calcification of hilar lymph nodes. Histology shows non-caseating granulomas. This clinical and histopathological presentation is most consistent with which occupational lung disease?

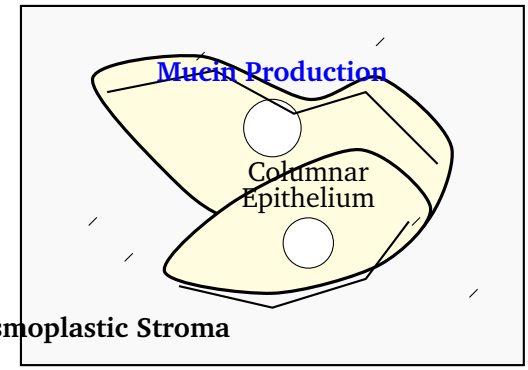
(A) Asbestosis with pleural fibrosis and mesothelioma

(B) Silicosis with silica particle deposition and fibrosis

(C) Berylliosis with chronic beryllium disease manifestations

(D) Talcosis with talc granulomatosis

Q6. A 56-year-old male smoker develops a cough and hemoptysis. CT chest reveals a 4 cm lung mass suspicious for malignancy. A core needle biopsy is obtained, and the tissue is examined microscopically. The diagram below illustrates the architectural pattern observed:



Based on the histological pattern illustrated above, which histological subtype of lung cancer is most likely?

(A) Small cell carcinoma with neuroendocrine differentiation

(B) Squamous cell carcinoma with keratin pearl formation

(C) Adenocarcinoma with mucin production and columnar epithelium

(D) Large cell carcinoma with undifferentiated pleomorphic cells

Q7. A 35-year-old female with systemic sclerosis (scleroderma) presents with progressive renal dysfunction. Laboratory studies reveal a sudden spike in blood pressure and elevated serum creatinine. Renal biopsy shows acute fibrinoid necrosis of arterioles and acute tubular necrosis. This rapidly progressive renal disease is best classified as which type of vasculitis?

(A) Large vessel vasculitis affecting aorta and major branches

(B) Medium vessel vasculitis with nodal involvement

(C) Small vessel vasculitis with fibrinoid necrosis of arterioles

(D) Immune complex vasculitis with granuloma formation

Q8. A 41-year-old male with a 20-year history of chronic alcoholism presents with progressive abdominal distension and ascites. Liver biopsy demonstrates extensive fibrosis bridging portal areas with regenerative nodules creating a nodular hepatic architecture. At which stage of liver fibrosis does this histological appearance best correspond?

(A) Stage 1: Portal fibrosis without bridging

(B) Stage 2: Portal fibrosis with rare bridging septa

(C) Stage 3: Bridging fibrosis (portal-to-portal or portal-to-central)

(D) Stage 4: Cirrhosis with complete architectural distortion

Q9. A 19-year-old male presents with severe hemolytic anemia following ingestion of certain medications. Laboratory studies reveal a hemoglobin of 7.8 g/dL, elevated reticulocytosis of 12%, elevated indirect bilirubin, elevated LDH, and a negative direct Coombs test. Which enzyme deficiency best explains this drug-induced hemolysis?

(A) Pyruvate kinase deficiency impairing glycolytic ATP generation

(B) Glucose-6-phosphate dehydrogenase (G6PD) deficiency compromising antioxidant defense

(C) Adenylate kinase deficiency reducing nucleotide phosphorylation

(D) Phosphofructokinase deficiency limiting glycolytic flux

Q10. A 67-year-old male with a history of smoking and COPD develops a palpable mass in the upper outer quadrant of his breast. Excisional biopsy reveals infiltrating lobular carcinoma (ILC). Which cellular feature distinguishes ILC from infiltrating ductal carcinoma (IDC)?

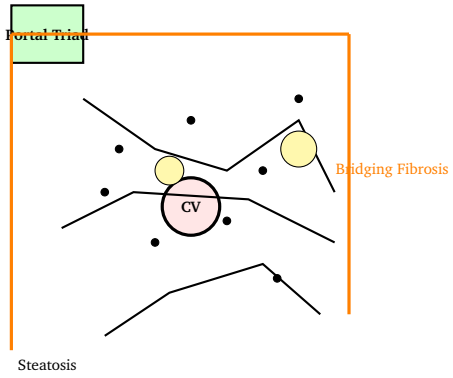
(A) ILC maintains in situ growth pattern with ductal origin

(B) ILC displays single-file infiltrative growth pattern with loss of E-cadherin

(C) ILC shows nested arrangement with prominent fibrosis and stromal desmoplasia

(D) ILC exhibits perineural invasion and lymphatic channel invasion exclusively

Q11. A 44-year-old female with a prior history of blood transfusion presents with fatigue, arthralgia, and elevated liver enzymes. Serological testing reveals positive anti-hepatitis C antibodies and detectable HCV RNA. A liver biopsy demonstrates portal inflammation with lymphoid aggregates and steatosis. Which histological pattern in chronic hepatitis C most commonly corresponds to this presentation? The diagram below illustrates the hepatic lobular architecture:



Which of the following best describes the histological stage of this chronic hepatitis?

(A) Stage 1: Mild portal inflammation without fibrosis

(B) Stage 2: Portal inflammation with portal-to-portal bridging fibrosis

(C) Stage 3: Bridging fibrosis with early architectural distortion

(D) Stage 4: Cirrhosis with complete architectural destruction

Q12. A 56-year-old male with a history of ulcerative colitis presents with progressive dyspnea and productive cough. High-resolution CT of the chest reveals peripheral, basilar-predominant reticular opacities. Bronchoalveolar lavage demonstrates elevated lymphocyte percentage with CD4/CD8 ratio less than 1. This pulmonary complication of IBD is best classified as which type of interstitial lung disease?

(A) Pulmonary hemorrhage from vasculitis

(B) Bronchiolitis obliterans organizing pneumonia (BOOP)

(C) Usual interstitial pneumonia (UIP) pattern

(D) Lymphocytic interstitial pneumonia (LIP)

Q13. A 31-year-old female presents with amenorrhea, galactorrhea, and visual field defects. Pituitary MRI reveals a 2.5 cm tumor with suprasellar extension. Immunohistochemistry of the surgical specimen confirms prolactin positivity. Which of the following represents the most likely pathological classification of this tumor?

(A) Chromophobe adenoma with oncocytic differentiation

(B) Prolactin-secreting adenoma (lactotroph adenoma)

(C) Non-functioning adenoma with incidental hormone positivity

(D) Plurihormonal adenoma with simultaneous hormone co-secretion

Q14. A 73-year-old male presents with progressive cognitive decline, tremor, and rigidity. Pathological examination reveals neuronal loss in substantia nigra with depigmentation. Intracytoplasmic inclusions composed of alpha-synuclein protein are identified using immunohistochemistry. These pathological inclusions are called:

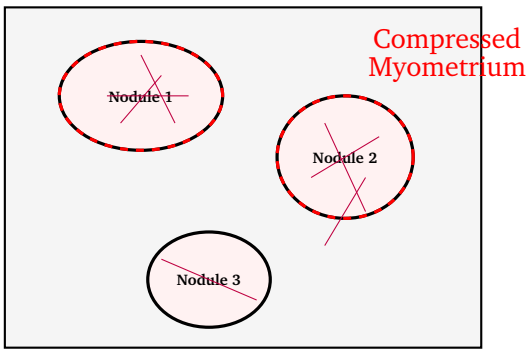
(A) Lewy bodies in Parkinson disease

(B) Pick bodies in Pick disease

(C) Hirano bodies in Alzheimer disease

(D) Marinesco bodies in substantia nigra

Q15. A 42-year-old female undergoes total abdominal hysterectomy for symptomatic uterine fibroids. Gross examination of the uterus reveals multiple well-circumscribed nodules with a whorled, tan-white appearance on cut surface. Histologically, the specimen demonstrates interlacing bundles of smooth muscle cells with variable cellularity and mitotic activity. The schematic representation of the pathological features is shown below:



Which of the following best characterizes the pathological features of uterine leiomyoma as illustrated above?

(A) Benign smooth muscle tumors with interlacing bundles and fibrous pseudocapsule

(B) Malignant leiomyosarcoma with increased mitotic rate and necrosis

(C) Adenomyosis with ectopic endometrial glands embedded in myometrium

(D) Diffuse myometrial hypertrophy from hormonal stimulation

Q16. A 48-year-old female with a history of carcinoma of the breast (estrogen receptor positive) develops bone pain and pathological fractures. Imaging reveals lytic bone lesions in the pelvis and femur. Bone biopsy demonstrates metastatic adenocarcinoma. Which of the following mechanisms best explains the osteolytic pattern of bone destruction observed in this metastasis?

(A) Direct tumor production of RANKL (receptor activator of NF-kB ligand) stimulating osteoclasts

(B) Osteoblastic formation of new bone matrix by tumor-derived osteoid production

(C) Vascular endothelial growth factor (VEGF)-mediated angiogenesis creating osteogenic reaction

(D) Parathyroid hormone-related peptide (PTHrP)-induced osteoclastogenesis via RANKL

Q17. A 51-year-old male with a history of chronic hepatitis B infection develops acute jaundice, fatigue, and right upper quadrant pain. Laboratory evalu-

ation reveals markedly elevated transaminases and bilirubin. Ultrasound demonstrates cirrhotic liver with ascites and a heterogeneous 6 cm mass in the right lobe. Alpha-fetoprotein is significantly elevated. Which of the following represents the most likely diagnosis?

(A) Benign hemangioma of the liver

(B) Cholangiocarcinoma with intrahepatic spread

(C) Hepatocellular carcinoma (HCC) arising in cirrhotic liver

(D) Metastatic colon carcinoma to the liver

Q18. A 26-year-old male presents with severe testicular pain and swelling. On examination, he has a unilateral testicular mass with a "bell clapper" anatomical finding. Ultrasound shows testicular torsion with absent perfusion. Histological examination of the surgically removed testicle reveals extensive coagulative necrosis and hemorrhagic infarction. Which pathological mechanism explains the testicular necrosis in this condition?

(A) Inflammatory orchitis with lymphocytic infiltration

(B) Vascular occlusion and subsequent ischemic necrosis

(C) Direct bacterial infection with suppurative necrosis

(D) Autoimmune-mediated apoptosis of germ cells

Q19. A 34-year-old female presents with amenorrhea and infertility. Pelvic ultrasound reveals bilateral ovarian cysts with a characteristic "string of pearls" appearance. Hormonal evaluation shows elevated LH, normal FSH, and elevated testosterone. Which of the following ovarian pathologies best corresponds to this clinical and radiological presentation?

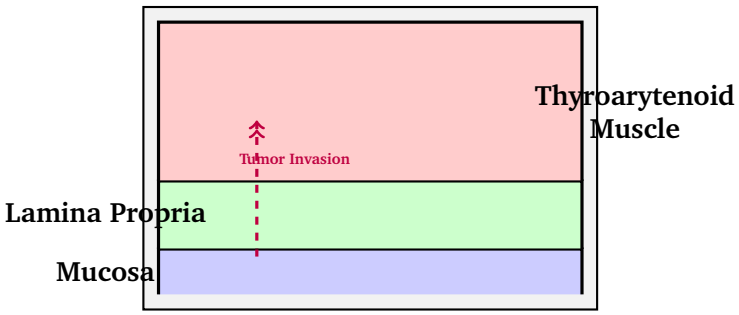
(A) Polycystic ovarian syndrome (PCOS) with anovulation

(B) Ovarian granulosa cell tumor with hormone secretion

(C) Ovarian dysgenesis with fibrous streak formation

(D) Simple ovarian cyst with follicular retention

Q20. A 58-year-old male presents with hoarseness and stridor. Laryngoscopy reveals a mass on the vocal cord. Biopsy demonstrates squamous cell carcinoma with histological evidence of invasion through the lamina propria into the underlying thyroarytenoid muscle. The TikZ diagram below represents the laryngeal wall architecture:



Based on the depth of invasion illustrated above, what is the T-staging classification for this laryngeal carcinoma?

(A) T1: Limited to vocal cord with normal mobility

(B) T2: Invasion of adjacent laryngeal structures with preserved airway

(C) T3: Fixation of vocal cord with compromised airway

(D) T4: Invasion into thyroid cartilage or extralaryngeal structures

Q21. A 47-year-old female with a history of inflammatory bowel disease undergoes colonoscopy for surveillance. Multiple polyps are identified and biopsied. Histology reveals tubular adenocarcinoma with mucinous differentiation. Flow cytometry analysis of the tumor cells demonstrates aneuploidy. Which of the following genetic pathways is most commonly altered in sporadic colorectal cancer development?

(A) p53 tumor suppressor and APC (adenomatous polyposis coli) gene mutations in adenoma-carcinoma sequence

(B) BRCA1/BRCA2 mutations in hereditary nonpolyposis colorectal cancer

(C) KRAS proto-oncogene activation exclusive to mucinous tumors

(D) Mismatch repair gene deficiency universal in all colorectal cancers

Q22. A 52-year-old male with a history of occupational asbestos exposure presents with progressive dyspnea. Chest X-ray reveals pleural thickening and pleural

plaques. Pleural fluid analysis shows exudative characteristics. Histological examination of pleural biopsy demonstrates malignant mesothelioma with epithelioid and sarcomatoid components. This biphasic tumor is best classified as which histological variant?

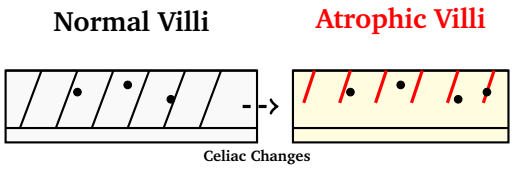
(A) Epithelioid mesothelioma with sarcomatoid metaplasia

(B) Sarcomatoid mesothelioma with adenocarcinoma differentiation

(C) Biphasic mesothelioma with both epithelioid and sarcomatoid components

(D) Desmoplastic mesothelioma with prominent fibroblastic stroma

Q23. A 37-year-old male presents with weight loss and chronic diarrhea. Duodenal biopsy demonstrates villous atrophy, crypt hyperplasia, and increased intraepithelial lymphocytes. Serological testing reveals positive tissue transglutaminase (tTG) IgA antibodies. The histological pattern in the diagram below illustrates the architectural changes:



Which of the following represents the Marsh classification stage for this duodenal biopsy finding?

(A) Type 0: Normal duodenal architecture

(B) Type 2: Increased intraepithelial lymphocytes with crypt hyperplasia (normal villi)

(C) Type 3b: Partial villous atrophy (PVA) with increased IELs and crypt hyperplasia

(D) Type 3c: Total villous atrophy with crypt hyperplasia and increased IELs

Q24. A 55-year-old female with a history of breast cancer undergoes sentinel lymph node biopsy. Histological examination reveals metastatic carcinoma cells within the lymph node parenchyma. Immunohistochemistry confirms

epithelial markers in the malignant cells. Which of the following represents the pathological mechanism by which metastatic cells preferentially lodge and proliferate within regional lymph nodes?

(A) Passive embolic lodgment in lymph node sinuses

(B) Chemokine-mediated homing via CCL21 and CCR7 expression

(C) Direct lymphatic invasion with growth in the afferent lymphatic channels

(D) Spontaneous apoptosis and phagocytosis within germinal centers

Q25. A 71-year-old male presents with acute coronary syndrome. Cardiac catheterization reveals a 90% stenosis of the left anterior descending artery. The atherosclerotic plaque is characterized by a lipid-rich necrotic core with a thin fibrous cap overlying it. This unstable plaque morphology is most susceptible to which catastrophic event?

(A) Plaque regression and spontaneous recanalization

(B) Plaque rupture with superimposed thrombosis causing acute MI

(C) Plaque calcification with stable atherosclerotic matrix

(D) Plaque encapsulation with formation of fibrotic scar tissue

Detailed Solutions

Q1.

Solution

Concept:

Osteomalacia is a metabolic bone disease characterized by defective mineralization of osteoid matrix. Understanding the distinction between normal osteoid formation and the failure of mineralization is critical for diagnosing and treating this condition.

Solution:

(a) Osteomalacia results from insufficient vitamin D availability or metabolism, leading to impaired calcium and phosphate homeostasis.

(b) Unlike osteoporosis, which involves decreased bone mass with normal mineralization, osteomalacia demonstrates abnormal mineralization ratios.

(c) Osteoblasts continue to synthesize osteoid (unmineralized bone matrix) normally, but the mineralization process is impaired.

(d) This impairment occurs because mineralization requires adequate serum calcium and phosphate concentrations, both dependent on vitamin D-mediated intestinal absorption and renal handling.

(e) Vitamin D deficiency leads to hypocalcemia and hyperphosphatasia, creating a mineralization milieu that prevents normal calcium deposition into osteoid.

(f) The primary biochemical disturbance is failure of osteoid mineralization despite intact osteoid synthesis and normal alkaline phosphatase enzyme activity.

(g) Histologically, excessive unmineralized osteoid seams accumulate within the bone matrix, a hallmark finding on bone biopsy.

Final Answer: The primary biochemical disturbance is failure of osteoid mineralization despite normal biochemical availability of mineral ions.

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept:

Inflammatory bowel disease causes chronic intestinal inflammation capable of triggering multiple patterns of necrosis depending on the severity and location of inflammation. Recognizing these necrotic patterns guides severity assessment and treatment decisions.

Solution:

(a) Severe inflammatory bowel disease, particularly Crohn's disease, can perforate the bowel through transmural inflammation extending through all wall layers.

(b) The pattern of tissue destruction in perforated IBD is characterized by liquefaction, where inflammatory cells and enzymes digest tissue architecture.

(c) This liquefactive necrosis results from intense infiltration by neutrophils, macrophages, and other inflammatory cells releasing proteolytic enzymes.

(d) The enzymes digest both structural proteins and the collagen framework of the intestinal wall, creating zones of complete tissue dissolution.

(e) Unlike coagulative necrosis, where cell outlines are preserved, liquefactive necrosis obliterates the normal tissue architecture.

(f) This destructive process explains the acute presentation with perforation, peritonitis, and the need for emergent surgical intervention.

(g) The extensive hemorrhage reflects both the inflammatory vasculitis and loss of vascular integrity from the necrotic process.

Final Answer: This pattern of tissue damage is primarily driven by enzymatic liquefactive necrosis from inflammatory cell infiltration.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept:

Myocardial infarction undergoes characteristic morphological changes through distinct temporal phases. Contraction band necrosis represents a specific early manifestation of irreversible ischemic injury with distinctive ultrastructural features.

Solution:

(a) Contraction band necrosis is an early histological feature of myocardial ischemic injury, appearing within 2-4 hours of the ischemic insult.

(b) These bands appear as hypereosinophilic, prominent transverse striations within individual myofibers.

(c) Mechanistically, contraction band necrosis results from dysregulated calcium influx during the immediate post-ischemic period.

(d) As oxygen becomes available during reperfusion, calcium floods into damaged myocytes, hyperactivating the contractile apparatus.

(e) This uncontrolled contraction creates areas of extreme fiber shortening and myofibrillar disruption, producing the characteristic "wavy fiber" appearance.

(f) Electron microscopy reveals disrupted Z-bands, fragmented myofibrils, and mitochondrial distortion.

(g) This early finding represents the transition between reversible and irreversible injury.

(h) Later phases (3-7 days) show macrophage infiltration and granulation tissue formation, while chronic phases (2-8 weeks) demonstrate mature fibrosis and scar formation.

Final Answer: This histological feature would most prominently manifest 24-48 hours post-infarction in the early acute phase.

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept:

Familial defective apolipoprotein B-100 (FDB) is a genetic disorder affecting the structural component of atherogenic lipoproteins. Understanding the molecular basis of this defect clarifies the mechanism of accelerated atherosclerosis in affected individuals.

Solution:

(a) Familial defective apolipoprotein B-100 results from specific missense mutations in the APOB gene affecting the ligand-binding domain of apoB-100.

(b) Apolipoprotein B-100 is the sole structural protein of LDL particles and serves as the ligand for LDL receptor recognition and binding.

(c) The APOB mutations create conformational changes that impair the ability of LDL particles to bind to LDL receptors on hepatocyte surfaces.

(d) This impaired binding prevents normal receptor-mediated endocytosis of LDL particles, causing accumulation of LDL in the circulation.

(e) Despite elevated LDL cholesterol, other lipoprotein metabolism remains relatively intact.

(f) The clinical consequence is severe hypercholesterolemia and premature atherosclerosis, similar to heterozygous familial hypercholesterolemia.

(g) This condition is distinguished from FH by the presence of structurally abnormal LDL rather than deficient LDL receptors.

Final Answer: This mutation impairs the recognition of LDL receptor-mediated endocytosis.

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept:

Occupational lung diseases produce characteristic patterns of fibrosis linked to specific mineral exposures. The radiological pattern of "eggshell" lymph node calcification provides a pathognomonic clue to specific occupational exposures.

Solution:

(a) The clinical presentation of an occupational lung disease worker with eggshell lymph node calcification is highly specific for silicosis.

(b) Silicosis results from chronic inhalation of crystalline silica dust in mining, sandblasting, and foundry work.

(c) Inhaled silica particles are engulfed by alveolar macrophages, which then undergo apoptosis due to silica crystal-induced membrane damage.

(d) The resulting granulomatous inflammation creates nodular opacities, particularly in upper lung lobes.

(e) The distinctive "eggshell" calcification occurs when hilar and mediastinal lymph nodes develop central calcification with peripheral calcified rims.

(f) Simple silicosis progresses to complicated silicosis with progressive massive fibrosis when exposure continues.

(g) Asbestosis produces pleural plaques and pleural thickening, not eggshell calcification.

(h) Berylliosis causes acute beryllium disease with acute respiratory symptoms or chronic beryllium disease with granulomatous lung disease.

(i) Talcosis is rare and does not produce eggshell calcification.

Final Answer: This is most consistent with silicosis with silica particle deposition and fibrosis.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept:

Lung adenocarcinoma is the most common histological subtype of lung cancer in non-smokers. Recognizing the characteristic glandular architecture with mucin production guides accurate histological classification and prognostication.

Solution:

(a) The schematic illustrates a malignant lung neoplasm with distinctive glandular structures producing mucin (as shown by the lumens).

(b) The tumor demonstrates columnar epithelial cells lining these glandular lumens, characteristic of adenocarcinoma differentiation.

(c) The desmoplastic stromal response surrounding the glandular elements is typical of infiltrating adenocarcinoma.

(d) Adenocarcinoma represents the most common lung cancer subtype, accounting for over 40% of lung malignancies.

(e) Unlike squamous cell carcinoma, which displays intercellular bridges and keratin pearl formation, adenocarcinoma shows glandular differentiation.

(f) Small cell carcinoma presents as sheets of small round cells with neuroendocrine differentiation and high mitotic rate.

(g) Large cell carcinoma displays large pleomorphic cells without specific differentiation patterns.

(h) The mucin production depicted is a hallmark of adenocarcinoma differentiation, distinguishing it from other lung cancer subtypes.

Final Answer: The histological subtype is adenocarcinoma with mucin production and columnar epithelium.

Answer: (C)

Go Back to Question 6

Q7.

Solution

Concept:

Systemic sclerosis-associated renal crisis represents a medical emergency characterized by acute renal failure and malignant hypertension. Understanding the specific pattern of vascular injury guides appropriate classification and urgent intervention.

Solution:

(a) Systemic sclerosis (scleroderma) renal crisis is a feared complication occurring in 5-10% of patients with diffuse cutaneous disease.

(b) The pathological hallmark is acute fibrinoid necrosis of small arterioles and arteries, a hallmark of small vessel vasculitis.

(c) Fibrinoid necrosis results from immune complex deposition and complement activation within vessel walls, causing protein denaturation and vessel wall destruction.

(d) The acute renal manifestation includes acute tubular necrosis from severe vasoconstriction and ischemia.

(e) Large vessel vasculitis affects aorta and major branches, not arterioles.

(f) Medium vessel vasculitis affects medium-sized arteries, presenting with different clinical patterns.

(g) Small vessel vasculitis directly affects arterioles and small arteries, causing fibrinoid necrosis and rapid renal deterioration.

(h) Immune complex vasculitis with granulomas describes a different pattern of vascular injury.

Final Answer: This is best classified as small vessel vasculitis with fibrinoid necrosis of arterioles.

Answer: (C)

Go Back to Question 7

Q8.

Solution

Concept:

Hepatic fibrosis is staged histologically based on the extent and pattern of collagen deposition and architectural distortion. Understanding the progressive stages guides assessment of liver disease severity and prognosis.

Solution:

(a) The histological description demonstrates portal-to-portal bridging fibrosis with regenerative nodules creating a nodular architecture.

(b) Bridging fibrosis indicates septa extending from portal area to portal area (portal-to-portal) or from portal to central vein (portal-to-central).

(c) This pattern of bridging indicates advanced fibrosis with significant architectural distortion.

(d) Stage 1 fibrosis shows only portal fibrosis without bridging.

(e) Stage 2 demonstrates portal fibrosis with rare or occasional bridging septa.

(f) Stage 3 fibrosis is defined by more extensive bridging fibrosis with portal-to-portal or portal-to-central connections.

(g) Stage 4 represents complete cirrhosis with loss of normal hepatic architecture and formation of regenerative nodules surrounded by complete fibrous septa.

(h) The clinical presentation of ascites indicates portal hypertension, consistent with advanced fibrosis approaching cirrhosis.

Final Answer: This corresponds to Stage 3: Bridging fibrosis with portal-to-portal or portal-to-central connections.

Answer: (C)

Go Back to Question 8

Q9.

Solution

Concept:

Drug-induced hemolysis in glucose-6-phosphate dehydrogenase deficiency represents an oxidative stress-induced process. Understanding the molecular mechanism guides diagnosis and prevention of future episodes.

Solution:

(a) The clinical presentation describes acute hemolytic anemia triggered by medication exposure, with negative Coombs test ruling out immune-mediated hemolysis.

(b) The elevated reticulocytosis indicates compensatory bone marrow erythropoiesis, appropriate for hemolytic anemia.

(c) Elevated LDH and indirect bilirubin confirm hemolysis.

(d) G6PD deficiency impairs the pentose phosphate pathway's production of NADPH, which normally reduces oxidative damage via glutathione antioxidant systems.

(e) When G6PD-deficient patients ingest oxidant medications, hemoglobin undergoes oxidative damage without adequate antioxidant protection.

(f) Hemoglobin precipitation into insoluble Heinz bodies damages the red blood cell membrane, causing hemolysis.

(g) Pyruvate kinase deficiency affects glycolytic ATP generation, causing chronic hemolysis independent of oxidant triggers.

(h) Adenylate kinase deficiency affects nucleotide metabolism, not hemolysis.

(i) Phosphofructokinase deficiency causes muscle symptoms and glycolytic impairment, not selective hemolysis.

Final Answer: The enzyme deficiency is glucose-6-phosphate dehydrogenase (G6PD) deficiency compromising antioxidant defense.

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept:

Infiltrating lobular carcinoma of the breast represents a distinct histological subtype with unique biological characteristics. Understanding the cellular and molecular features distinguishes ILC from more common infiltrating ductal carcinoma.

Solution:

(a) Infiltrating lobular carcinoma accounts for 10-15% of invasive breast cancers.

(b) The defining characteristic of ILC is a single-file infiltrative growth pattern, where individual tumor cells separate and infiltrate through the stroma.

(c) This distinctive pattern results from loss of E-cadherin, an adhesion molecule essential for maintaining cell-cell contact in epithelial tissues.

(d) E-cadherin loss causes the cells to lose cohesion and assume individual migration, creating the pathognomonic single-file pattern.

(e) IDC, by contrast, demonstrates nested and clustered arrangement of cells with retained E-cadherin expression.

(f) The single-file pattern makes ILC more difficult to detect clinically and radiographically compared to IDC.

(g) ILC tends to be multifocal and multicentric more frequently than IDC.

(h) Perineural invasion is not a distinguishing feature specific to ILC.

Final Answer: ILC displays single-file infiltrative growth pattern with loss of E-cadherin.

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept:

Chronic hepatitis C demonstrates characteristic histological features that stage the extent of liver damage. Understanding the relationship between histological stage and clinical progression guides treatment decisions and prognostication.

Solution:

(a) The patient presents with serologically confirmed chronic hepatitis C with portal inflammation.

(b) The diagram illustrates portal inflammation with lymphoid aggregates, steatosis, and bridging fibrosis spanning from portal triad to another portal triad.

(c) Chronic hepatitis C typically progresses through defined stages of fibrosis.

(d) Stage 1 involves only portal inflammation without architectural distortion or fibrosis.

(e) Stage 2 demonstrates portal inflammation with portal-to-portal bridging fibrosis, as illustrated.

(f) Stage 3 shows more extensive bridging fibrosis with early architectural distortion.

(g) Stage 4 represents cirrhosis with complete architectural loss and nodular regeneration.

(h) The presence of bridging fibrosis (visible in the diagram) is the key feature distinguishing stage 2 from stage 1.

(i) Steatosis is common in chronic hepatitis C and may reflect metabolic abnormalities.

(j) Lymphoid aggregates are characteristic of chronic hepatitis C.

Final Answer: This corresponds to Stage 2: Portal inflammation with portal-to-portal bridging fibrosis.

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept:

Pulmonary complications of inflammatory bowel disease represent extra-intestinal manifestations occurring in 5-10% of IBD patients. Recognizing the specific interstitial lung disease pattern guides diagnosis and treatment decisions.

Solution:

(a) The clinical presentation of ulcerative colitis with pulmonary involvement showing lymphocytic interstitial inflammation is characteristic of IBD-associated lung disease.

(b) The BAL findings of elevated lymphocyte percentage with inverted CD4/CD8 ratio ($<$ 1) indicate T-cell predominance with increased CD8 cells.

(c) The pattern of peripheral, basilar-predominant reticular opacities on HRCT with lymphocytic interstitium defines lymphocytic interstitial pneumonia (LIP).

(d) LIP is the most common pulmonary manifestation of IBD.

(e) Other IBD-associated lung diseases include bronchiolitis obliterans organizing pneumonia (BOOP), diffuse alveolar hemorrhage, and pulmonary fibrosis.

(f) BOOP typically presents with focal consolidations and organizing pneumonia pattern.

(g) UIP pattern is more commonly associated with autoimmune connective tissue diseases and idiopathic pulmonary fibrosis.

(h) Pulmonary hemorrhage presents with different clinical and radiological patterns including hemoptysis.

Final Answer: This is best classified as lymphocytic interstitial pneumonia (LIP).

Answer: (D)

Go Back to Question 12

Q13.

Solution

Concept:

Pituitary adenomas are benign neoplasms arising from anterior pituitary cells. Classification based on hormone production guides clinical management and predicts specific endocrine manifestations.

Solution:

(a) The patient presents with the classic triad of galactorrhea, amenorrhea, and visual field defects from suprasellar mass effect.

(b) Immunohistochemical confirmation of prolactin positivity in the tumor cells establishes the diagnosis of prolactin-secreting adenoma.

(c) Prolactin-secreting adenomas (lactotroph adenomas) represent the most common functional pituitary adenomas.

(d) These tumors preferentially occur in women of reproductive age and manifest with menstrual irregularities and galactorrhea.

(e) Chromophobe adenomas lack specific hormone differentiation and are often non-functioning, though chromophobe adenomas can occasionally retain hormone granules.

(f) Non-functioning adenomas are hormone-negative on immunostaining.

(g) Plurihormonal adenomas co-secrete multiple hormones simultaneously, which is rare.

(h) The diagnosis is specifically prolactin-secreting adenoma based on the clinical presentation and immunohistochemical confirmation.

Final Answer: The most likely pathological classification is prolactin-secreting adenoma (lactotroph adenoma).

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept:

Parkinson disease is characterized by progressive dopaminergic neuron loss in the substantia nigra with intracytoplasmic protein aggregates. Understanding the pathological hallmark inclusions guides diagnosis and understanding of disease pathogenesis.

Solution:

(a) The clinical presentation of progressive bradykinesia, rigidity, and tremor with substantia nigra degeneration defines Parkinson disease.

(b) The pathological hallmark is the presence of Lewy bodies, which are intracytoplasmic inclusions composed primarily of alpha-synuclein protein.

(c) Lewy bodies are eosinophilic, spherical inclusions with a central core surrounded by radiating filaments.

(d) Alpha-synuclein is normally a soluble protein involved in synaptic function, but in Parkinson disease, it misfolds and aggregates into insoluble fibrils.

(e) Lewy bodies accumulate in remaining dopaminergic neurons and in other CNS regions in more advanced disease.

(e) Pick bodies represent pathological inclusions in Pick disease (frontotemporal dementia) composed of tau protein.

(f) Hirano bodies are intraneuronal crystalline inclusions seen in Alzheimer disease and other conditions.

(g) Marinesco bodies are intranuclear inclusions in substantia nigra cells but are not specific to Parkinson disease.

Final Answer: These pathological inclusions are called Lewy bodies in Parkinson disease.

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept:

Uterine leiomyomas are the most common benign pelvic tumors in women. Understanding the pathological features and growth characteristics guides clinical management and surgical planning.

Solution:

(a) Uterine leiomyomas arise from smooth muscle cells of the myometrium.

(b) The diagram illustrates multiple well-defined nodules with compressed surrounding myometrium, characteristic of benign leiomyomas.

(c) Histologically, leiomyomas display interlacing bundles of smooth muscle cells with variable cellularity.

(d) The interlacing architecture demonstrates the benign, organized growth pattern.

(e) Unlike leiomyosarcoma, benign leiomyomas have low mitotic activity (less than 5 mitoses per 10 HPF).

(f) Leiomyomas are typically surrounded by a pseudocapsule of compressed myometrium, as illustrated in the diagram.

(g) The benign nature allows for conservative management or hysterectomy depending on symptoms.

(h) Adenomyosis involves ectopic endometrial glands within the myometrium, creating a different clinical and histological picture.

(i) Diffuse myometrial hypertrophy represents uniform thickening without nodular masses.

Final Answer: These are benign smooth muscle tumors with interlacing bundles and fibrous pseudocapsule.

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept:

Osteolytic bone metastases result from a complex interplay between tumor cells and bone-resorbing osteoclasts. Understanding these mechanisms guides therapeutic targeting and treatment selection.

Solution:

(a) The patient presents with metastatic breast cancer producing osteolytic bone lesions.

(b) Osteolytic metastases develop through mechanisms distinct from osteoblastic lesions.

(c) The primary mechanism involves tumor cells producing parathyroid hormone-related peptide (PTHrP) and other cytokines.

(d) PTHrP indirectly stimulates osteoclasts through activation of osteoblasts and stromal cells.

(e) Osteoblasts and stromal cells respond to PTHrP by increasing RANKL (receptor activator of NF-kB ligand) expression.

(f) RANKL acts on osteoclast precursor cells via the RANK receptor, driving differentiation and activation of mature osteoclasts.

(g) Activated osteoclasts resorb bone, creating the lytic lesions visible on imaging.

(h) Direct tumor production of RANKL is another mechanism of osteolytic disease.

(i) VEGF promotes osteogenesis, not osteolysis.

(j) Osteoblastic lesions result from direct tumor production of bone-forming factors.

Final Answer: The mechanism is parathyroid hormone-related peptide (PTHrP)-induced osteoclastogenesis via RANKL.

Answer: (D)

Go Back to Question 16

Q17.

Solution

Concept:

Hepatocellular carcinoma is one of the most common and deadly malignancies worldwide. Understanding the risk factors, clinical presentation, and diagnostic markers guides diagnosis and prognostication.

Solution:

(a) The patient presents with clinical features highly suggestive of hepatocellular carcinoma: cirrhotic liver, heterogeneous mass, and elevated AFP.

(b) Chronic hepatitis B infection is a major risk factor for HCC development, with increased risk in cirrhotic patients.

(c) The combination of hepatic cirrhosis, a large solitary mass, and markedly elevated AFP is pathognomonic for HCC.

(d) Benign hemangiomas are typically homogeneous, well-defined lesions that do not produce AFP elevation.

(e) Cholangiocarcinoma arises from biliary epithelium and may present with different clinical features and serological markers.

(f) Metastatic colon carcinoma typically presents with multiple lesions and different radiological patterns.

(g) The elevated AFP specifically supports HCC diagnosis, as AFP is produced by hepatocellular carcinoma cells.

(h) HCC frequently arises in the setting of cirrhosis from chronic hepatitis B or C infection.

Final Answer: The most likely diagnosis is hepatocellular carcinoma (HCC) arising in cirrhotic liver.

Answer: (C)

Go Back to Question 17

Q18.

Solution

Concept:

Testicular torsion represents a urological emergency requiring immediate surgical intervention. Understanding the pathophysiology guides rapid diagnosis and treatment decisions.

Solution:

(a) Testicular torsion occurs when the spermatic cord twists on itself, cutting off blood supply to the testis.

(b) The "bell clapper" anatomy predisposes to torsion by allowing increased testicular mobility within the tunica vaginalis.

(c) The vascular occlusion immediately compromises testicular blood supply, initiating ischemic damage.

(d) The ischemic environment rapidly progresses to coagulative necrosis and hemorrhagic infarction.

(e) Within 6 hours of torsion, irreversible damage typically occurs.

(f) The histological examination reveals coagulative necrosis with extensive hemorrhage throughout the testicular parenchyma.

(g) This represents pure vascular ischemia rather than inflammatory, infectious, or autoimmune mechanisms.

(h) The surgical findings on Doppler ultrasound confirm absent perfusion, confirming the ischemic etiology.

Final Answer: The testicular necrosis results from vascular occlusion and subsequent ischemic necrosis.

Answer: (B)

Go Back to Question 18

Q19.

Solution

Concept:

Polycystic ovarian syndrome represents a complex endocrinopathy affecting ovarian follicle development. Understanding the pathophysiology guides diagnosis and management decisions.

Solution:

(a) PCOS affects approximately 10% of reproductive-age women and is a leading cause of anovulatory infertility.

(b) The "string of pearls" radiological appearance represents multiple follicular cysts in various stages of development arranged around the ovarian periphery.

(c) The elevated LH and normal or low FSH create an abnormal LH/FSH ratio that impairs follicular maturation.

(d) This hormonal imbalance prevents normal dominant follicle selection and triggers anovulation.

(e) The elevated testosterone reflects increased androgen production from theca cells stimulated by excess LH.

(f) The hyperandrogenism manifests clinically as hirsutism, acne, and male-pattern alopecia.

(g) Ovarian granulosa cell tumors specifically produce inhibin and AMH, different from the hormonal profile of PCOS.

(h) Ovarian dysgenesis presents with streak ovaries and primary amenorrhea, not functional cysts.

(i) Simple follicular cysts are typically solitary, not the multiple follicular arrangement seen in PCOS.

Final Answer: This is polycystic ovarian syndrome (PCOS) with anovulation.

Answer: (A)

Go Back to Question 19

Q20.

Solution

Concept:

Laryngeal squamous cell carcinoma staging depends on the extent of invasion through the laryngeal wall layers. Understanding the T-staging system guides treatment selection and prognostication.

Solution:

(a) The diagram illustrates the laryngeal wall architecture with distinct tissue layers.

(b) The tumor demonstrates invasion through the lamina propria into the thyroarytenoid muscle layer beneath.

(c) The depth of invasion is critical for T-staging in laryngeal cancer.

(d) T1 tumors are limited to one laryngeal site (vocal cord, arytenoid, etc.) without fixation.

(e) T2 tumors extend beyond one anatomical site or to supraglottic space without vocal cord fixation.

(f) T3 tumors show vocal cord fixation OR tumor extension to paraglottic space or minor thyroid cartilage erosion.

(g) T4a tumors invade major thyroid cartilage or epiglottic cartilage.

(h) T4b tumors invade extralaryngeal tissues (thyroid, trachea, esophagus, etc.).

(i) The invasion illustrated extends into the thyroarytenoid muscle, indicating fixed vocal cord involvement.

(j) This depth of invasion with vocal cord fixation defines T3 staging.

Final Answer: This corresponds to T3: Fixation of vocal cord with compromised airway.

Answer: (C)

Go Back to Question 20

Q21.

Solution

Concept:

Colorectal cancer develops through a well-characterized adenoma-carcinoma sequence involving accumulation of genetic alterations. Understanding these pathways guides surveillance and early intervention strategies.

Solution:

(a) The adenoma-carcinoma sequence describes the stepwise genetic transformation from normal mucosa to adenoma to carcinoma.

(b) The initial critical alteration involves loss of the APC (adenomatous polyposis coli) gene, which normally functions as a tumor suppressor through Wnt pathway regulation.

(c) Loss of APC function leads to aberrant Wnt signaling and polyp formation.

(d) Subsequent mutations in KRAS proto-oncogene promote proliferation and adenoma growth.

(e) Later mutations in p53 tumor suppressor allow escape from apoptotic checkpoints and malignant transformation.

(f) Additional alterations in mismatch repair genes (MLH1, MSH2, MSH6) can accelerate progression, particularly in hereditary nonpolyposis colorectal cancer (HNPCC).

(g) BRCA1/BRCA2 mutations are associated with hereditary breast and ovarian cancer, not colorectal cancer.

(h) KRAS mutations are not exclusive to mucinous tumors; they occur across histological subtypes.

(i) Mismatch repair deficiency is universal in HNPCC but not in sporadic colorectal cancers.

Final Answer: The pathway is p53 tumor suppressor and APC gene mutations in adenoma-carcinoma sequence.

Answer: (A)

Go Back to Question 21

Q22.

Solution

Concept:

Mesothelioma represents a highly aggressive malignancy arising from pleural or peritoneal mesothelium in response to asbestos exposure. Recognizing histological variants guides prognosis and treatment planning.

Solution:

(a) The patient's history of asbestos exposure with pleural thickening and malignant tumor confirms mesothelioma diagnosis.

(b) Mesothelioma exhibits three major histological variants: epithelioid, sarcomatoid, and biphasic.

(c) Epithelioid mesothelioma displays cuboidal to columnar cells with glandular differentiation, resembling adenocarcinoma.

(d) Sarcomatoid mesothelioma shows spindle cell proliferation mimicking fibrosarcoma or other soft tissue sarcomas.

(e) Biphasic mesothelioma contains both epithelioid and sarcomatoid components in varying proportions.

(f) The presence of both epithelioid and sarcomatoid components in this case defines biphasic mesothelioma.

(g) Biphasic mesothelioma carries a prognosis intermediate between epithelioid (better) and sarcomatoid (worse) variants.

(h) Desmoplastic mesothelioma is characterized by prominent fibrous stroma obscuring the neoplastic cells.

(i) Immunohistochemistry typically shows positivity for calretinin, WT1, and CK5/6 in mesotheliomas.

Final Answer: This is biphasic mesothelioma with both epithelioid and sarcomatoid components.

Answer: (C)

Go Back to Question 22

Q23.

Solution

Concept:

Celiac disease is an autoimmune condition affecting the small intestine in genetically predisposed individuals. The Marsh classification system provides a standardized framework for histological grading based on architectural changes.

Solution:

(a) The patient presents with classic celiac disease symptoms: diarrhea, weight loss, and positive serological tests (anti-tTG IgA antibodies).

(b) The diagram demonstrates the characteristic histological changes from normal villi to atrophied villi with increased intraepithelial lymphocytes.

(c) The Marsh classification system stages celiac disease severity based on intestinal biopsy findings.

(d) Type 0 represents normal duodenal architecture without inflammation.

(e) Type 1 (increased IELs) shows increased intraepithelial lymphocytes without villous atrophy.

(f) Type 2 (hyperplastic) demonstrates increased IELs with crypt hyperplasia but preservation of normal villous architecture.

(g) Type 3a (partial villous atrophy/PVA) shows partial villous height reduction with crypt hyperplasia and increased IELs.

(h) Type 3b (PVA) shows more extensive villous atrophy with pronounced crypt hyperplasia.

(i) Type 3c (total villous atrophy/TVA) demonstrates complete loss of villous architecture with crypt hyperplasia and increased IELs.

(j) The diagram illustrates total villous atrophy with crypt hyperplasia and increased intraepithelial lymphocytes, consistent with Type 3c.

Final Answer: This represents Marsh Type 3c: Total villous atrophy with crypt hyperplasia and increased IELs.

Answer: (D)

Go Back to Question 23

Q24.

Solution

Concept:

Lymph node metastasis involves a complex process of malignant cell homing and colonization. Understanding the molecular mechanisms guides understanding of tumor progression and therapeutic targeting.

Solution:

(a) The sentinel lymph node biopsy demonstrates metastatic carcinoma cells, indicating tumor dissemination.

(b) While passive embolic lodgment in lymph node sinuses can occur, this does not explain selective lymph node involvement.

(c) The preferred mechanism involves active chemokine-mediated homing, where tumor cells express specific chemokine receptors.

(d) CCL21 (C-C motif chemokine ligand 21) is highly expressed by lymph node stromal cells, including lymph node fibroblasts and endothelial cells.

(e) CCR7 (C-C motif chemokine receptor 7) is frequently upregulated on malignant cells during epithelial-to-mesenchymal transition.

(f) CCL21-CCR7 interactions facilitate specific homing of tumor cells to regional lymph nodes.

(g) This chemokine-mediated mechanism explains why metastases preferentially lodge in draining regional nodes rather than random lymph nodes.

(h) Direct lymphatic invasion may contribute but does not explain the selective regional node involvement.

(i) Apoptosis within germinal centers would prevent metastatic outgrowth.

Final Answer: The mechanism is chemokine-mediated homing via CCL21 and CCR7 expression.

Answer: (B)

Go Back to Question 24

Q25.

Solution

Concept:

Atherosclerotic plaque rupture represents the pivotal event precipitating acute coronary syndromes. Understanding plaque morphology and structural vulnerability guides risk stratification and aggressive preventive therapy.

Solution:

(a) The patient presents with acute coronary syndrome caused by a high-grade coronary stenosis.

(b) The plaque morphology described—lipid-rich necrotic core with thin fibrous cap—represents an unstable or vulnerable plaque.

(c) Unstable plaques are characterized by: 1. Large lipid-rich necrotic core accumulating cholesterol and foam cell debris 2. Thin, dysfunctional fibrous cap separating the thrombogenic core from the lumen 3. Inflammatory infiltration with macrophages and T cells

(d) The thin fibrous cap cannot withstand hemodynamic forces and mechanical stress.

(e) When the cap ruptures, the highly thrombogenic lipid core is exposed to circulating blood.

(f) Tissue factor and phosphatidylserine on the lipid core activate the coagulation cascade.

(g) Superimposed thrombosis rapidly occludes the vessel, causing acute myocardial infarction.

(h) This mechanism explains why some plaques cause acute MI despite only moderate severity stenosis.

(i) Plaque regression would reduce thrombotic risk, not destabilize the plaque.

(j) Calcification and fibrotic encapsulation stabilize plaques, not destabilize them.

Final Answer: This morphology is most susceptible to plaque rupture with superimposed thrombosis causing acute MI.

Answer: (B)

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Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	B	3	B	4	A	5	B
6	C	7	C	8	C	9	B	10	B
11	B	12	D	13	B	14	A	15	A
16	D	17	C	18	B	19	A	20	C
21	A	22	C	23	D	24	B	25	B