### Final Exam. Third year. Paper I

**Date:** 22/5/2013.

**Important instructions For All Students:** Please Read Carefully. All Questions to be answered.

<table>
<thead>
<tr>
<th>ALLOCATED TIME</th>
<th>(2 HOURS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total allocated marks</td>
<td>(75 marks)</td>
</tr>
<tr>
<td>Number of pages.</td>
<td>(11 pages)</td>
</tr>
<tr>
<td>Number of questions</td>
<td>(7 questions)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Question number</th>
<th>Mark</th>
<th>First signature</th>
<th>Second signature</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

الدرجة بالحروف

**Student name:**

**Student number:**

**Oral Exam** will be in the pathology department, on 28 May at 9.00 AM.

**Secret number**
Question 1: Define the following (One mark each)

1- **Necrosis.**
Morphological changes that follow death of group of cells within the living body which occur either directly or follow reversible injury

2- **Membranous inflammation.**
Severe type of acute non suppurative inflammation characterized by formation of a pseudomembrane over the inflamed area

3- **Granuloma.**
Chronic specific inflammation consisting of a microscopic aggregation of chronic inflammatory cells mainly macrophages, lymphocytes, langhan's giant cells

4- **Dystrophic Calcification.**
Calcification of injured and necrotic tissues. It is the commonest type of pathological calcification.

5- **Atherosclerosis.**
It is a chronic, progressive, multifocal disease of the vessel wall intima whose characteristic lesion is the atheroma or plaque.

6- **Pneumonia.**
Inflammation associated with consolidation of the lung parenchyma.

7- **Goiter.**
Enlargement of thyroid gland, which is noninflammatory, non-neoplastic, due to hypertrophy and hyperplasia of follicular epithelium

8- **Aneurysm.**
Localized abnormal dilatation of a blood vessel wall or cardiac wall

9- **Cirrhosis.**
Chronic diffuse irreversible process affecting the liver characterized by: loss of normal liver architecture, hepatocellular necrosis, regenerating nodules separated by fibrotic bands.

10- **Ulcerative colitis.**
Chronic non-specific inflammation of large intestine, characterized by ulceration
**Question 2: Explain the pathogenesis of: (2.5 marks each)**

1- **Secondary amyloidosis.**

All these diseases cause chronic persistent tissue breakdown. In these diseases there is a persistent proliferation and activation of macrophages and polymorphs, secreting proteolytic enzymes and various cytokines → tissue breakdown and stimulation the liver to secret SAA protein in large amounts → deposit extracellularly in many organs (secondary amyloidosis).

2- **Tuberculosis (Tubercle formation only).**

- Polysaccharide fraction of the tubercle bacilli attracts the neutrophil leucocytes within few hours. They phagocytose the bacteria but are unable to destroy them as the bacteria are protected by the lipid capsule and the neutrophils do not contain the enzyme lipase.
- The lipid fraction of the capsule attracts the macrophages (tissue histiocytes & blood monocytes) after the first day. They collect and phagocytose the free bacilli and those inside the neutrophils and are now called epithelioid cells. The bacilli are partially digested with the release of tuberculoprotein. The tuberculoprotein stimulates a cell mediated immune response (delayed hypersensitivity) within a period of about 10 days. Sensitized T-lymphocytes appear and surround the epithelioid cells.
- The sensitized lymphocytes release various cytokines

3- **Rheumatic fever.**

An autoimmune disease mediated by either one of the following mechanisms:

1- **Antigenic similarity (cross reaction mechanism):** Antibody against streptococcal M protein cross react with heart muscles.

2- **Altered antigenicity mechanism:** Streptococcal toxins change antigencity of heart collagen to become autoAg → autoAbs → Ag-Ab reaction → complement formation → secretion of chemical mediators and degeneration of collagen and inflammation around it.

3- **Immune complex theory:** Streptococci act as antigen → antibodies formation → Ag-Ab complex in circulation precipitate which share in blood vessels wall and collagen of the heart → complement fixation and inflammation.

4- **Bronchiectasis.**

Both infection & obstruction are required:

1- Bronchial infection: lead to:
   - Destruction of bronchial epithelium → interferes with cough mechanism → retention of secretions
   - Destruction of muscle and elastic tissue → fibrosis → weakness of the wall
   - Pneumonitis from spread of infection → pneumatitis → fibrosis → pull the weak wall of bronchi.

2- Bronchial obstruction: after obstruction, air is resorbed → atelectasis → loss of elastic force → relax of airways → superadded infection → inflammation → weakening and further dilatation

5- **Colorectal carcinoma.**

A) **Environmental causes:**
(1) Dietary practices:
- Low content of unabsorbable vegetable fiber, high concentrated toxic products for long time
- Corresponding high content of refined carbohydrates → toxic oxidative products in intestine
- High fat content → synthesis of cholesterol & bile acids (carcinogens)
- Decreased intake of protective micronutrients such as vitamins A, C
(2) Adenoma, villous & FAP
(3) Ulcerative colitis.

B) Adenoma Carcinoma sequence:
**Question 3: a- Compare between (5 marks):**

<table>
<thead>
<tr>
<th></th>
<th>Ulcerative colitis</th>
<th>Crohn's Disease.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Site</strong></td>
<td>colon</td>
<td>Ileum ± colon</td>
</tr>
<tr>
<td><strong>Distribution</strong></td>
<td>Diffuse</td>
<td>Skip lesions</td>
</tr>
<tr>
<td><strong>Gross picture</strong></td>
<td>Hyperemia, edema, and granularity with friability, pinpoint hemorrhage</td>
<td>– Skip lesion: segmental areas of involvement that are sharply demarcated from contiguous normal gut</td>
</tr>
<tr>
<td></td>
<td>Mucopurulant discharge → necrosis → ulceration (large, irregular, superficial undermined edges) → pseudopolyps (edematous mucosa in between ulcer)</td>
<td>– Affected bowel wall thick &amp; inflexible “lead-pipe or rubber hose”</td>
</tr>
<tr>
<td></td>
<td>Mesenteric L.N: may be enlarged</td>
<td>– Lumen narrow</td>
</tr>
<tr>
<td><strong>Microscopic picture</strong></td>
<td>Ulcerated mucosa</td>
<td>Mucosa ulcerate (long &amp; serpentine), linear deep penetrating (fissuring), separated by edema → mucosa with cobblestone appearance</td>
</tr>
<tr>
<td></td>
<td>– Non-specific inflammation in mucosa &amp; submucosa:</td>
<td>– Mesenteric L.N.: enlarged, firm</td>
</tr>
<tr>
<td></td>
<td>*Congestion - Edema</td>
<td>– Mesentery: thick, with adhesion</td>
</tr>
<tr>
<td></td>
<td>*Neutrophils and plasma cells infiltrate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crypt abscess: collected neutrophils and pus cells in the depth of mucosal crypts</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pseudopolyps consist of hyperplastic mucosa and granulation tissue</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Healing of ulcers occurs, but crypts do not reform completely</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mucosa at the margins of the ulcer shows mucus depletion, metaplasia.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Transmural inflammation (chronic inflammatory cells; lymphocytes, plasma cells), extending to serosa, mesentery.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non caseating granuloma → Submucosa and Subserosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dilatation of lymphatic channels</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lymphoid aggregates in all levels of the bowel wall.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mucosa → ulcers and fissures, hyperplasia, metaplasia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Healing by fibrosis</td>
<td></td>
</tr>
</tbody>
</table>
**Question 3: b-Compare between : (5 marks):**

<table>
<thead>
<tr>
<th></th>
<th>Benign Hypertension</th>
<th>Malignant hypertension.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diastolic Blood pressure</strong></td>
<td>Not exceeding 120 mmHg</td>
<td>Exceed 120 mmHg</td>
</tr>
<tr>
<td><strong>Arterioles</strong></td>
<td>Hyalinosis &amp; elastosis</td>
<td>Onion skin thickening &amp; fibrinoid necrosis</td>
</tr>
<tr>
<td><strong>Heart</strong></td>
<td>Concentric hypertrophy of left ventricles → bulging of interventricular system to the right &amp; papillary muscle thickening → left ventricular dilatation &amp; failure</td>
<td>Depends on the duration. If rapidly progressive → death without significance cardiac changes</td>
</tr>
<tr>
<td><strong>Kidney</strong></td>
<td>Benign nephrosclerosis:</td>
<td>Malignant nephrosclerosis:</td>
</tr>
<tr>
<td></td>
<td>• Decrease in size &amp; contracted</td>
<td>• Normal size</td>
</tr>
<tr>
<td></td>
<td>• Granular surface.</td>
<td>• Smooth surface</td>
</tr>
<tr>
<td></td>
<td>• Increased perinephric fat.</td>
<td>• Normal perinephric fat</td>
</tr>
<tr>
<td></td>
<td>• Capsule is thick &amp; adherent</td>
<td>• Capsule stripped easily</td>
</tr>
<tr>
<td></td>
<td>• Progressive periglomerular &amp; glomerular fibrosis &amp; hyalinosis.</td>
<td>• Focal glomerulonecrosis &amp; hemorrhage (hge)</td>
</tr>
<tr>
<td></td>
<td>• Atrophied tubules &amp; others of the functioning glomeruli are dilated.</td>
<td>• Thick, prominent arterioles.</td>
</tr>
<tr>
<td></td>
<td>• Interstitial fibrosis &amp; blood vessels show end arteritis obliterans.</td>
<td>• Malignant arteriosclerosis (Onion skin thickening of the all with fibrinoid necrosis).</td>
</tr>
<tr>
<td></td>
<td>• Afferent &amp; efferent arterioles show benign arteriosclerosis (hyalinosis and elastosis).</td>
<td>• Cortex: normal easily differentiated from medulla.</td>
</tr>
<tr>
<td></td>
<td>• Cortex is thin, fibrosis not easily differentiated from medulla.</td>
<td>• Focal glomerulonecrosis &amp; hge</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Thick, prominent arterioles.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Malignant arteriosclerosis</td>
</tr>
</tbody>
</table>
**Question 4:** Describe in short (2.5 marks each)

1- Stages of lobar pneumonia.

<table>
<thead>
<tr>
<th>Period</th>
<th>Congestion</th>
<th>Red hepatization</th>
<th>Gray hepatization</th>
<th>Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2 days</td>
<td>Enlarged, congestion, dark red and well demarcated, wet sponge, exudes frothy fluid on squeeze</td>
<td>Enlarged dark red, congested, dry, solid, granular (hepatized), fibrinous pleurisy, enlarged hilar L.N.s.</td>
<td>Enlarged and gray, reduced congestion, consolidated, firm, airless (hepatized), dry, granular, fibrinous pleurisy, enlarged hilar L.N.s.</td>
<td>The fibrin is gradually liquefied by proteolytic enzymes. Phagocytosis and lymphatic drainage of liquefied exudates restore normal alveolar aeration. The lung will then return to normal except that healing of pleurisy may lead to some adhesions</td>
</tr>
</tbody>
</table>

**MP**

<table>
<thead>
<tr>
<th>Congestion</th>
<th>Red hepatization</th>
<th>Gray hepatization</th>
<th>Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congested septal capillaries, thick edematous alveolar wall, Alveoli contains fluid and cellular exudates</td>
<td>Alveolar capillaries thick, edematous and congested, thickened alveolar walls, distended alveolar spaces with infl. exudate and bacteria.</td>
<td>Alveolar capillary congestion is reduced, thin alveolar wall, alveolar spaces show dead bacteria, shrunked fibrin network, histocytes, hemolysed RBCs</td>
<td></td>
</tr>
</tbody>
</table>

2- Types and microscopic picture of thyroid carcinoma.

1- Papillary carcinoma:

**Grossly:** Solitary or multifocal. Well circumscribed and encapsulated; or infiltrating and ill-defined. Areas of fibrosis, calcification and papillary foci

**Microscopic picture:** Branching papillae formed of fibrovascular core show Psammoma bodies and covered by single or multiple layers of malignant cuboidal epithelium show empty, ground-glass nuclei.

2- Follicular carcinoma:

**Gross picture:** Solitary well-circumscribed encapsulated mass. Cut section is grayish to tan to pink in color wit central fibrosis and focal calcification.

**Microscopic picture:** formed of small follicles, trabeculae and sheets composed of uniform malignant cells with capsular or vascular invasion.

3- Medullary carcinoma:

**Grossly:** Large lesion with necrosis and hage, or multifocal and bilateral.

**Microscopic:** nests or trabeculae of spindle cells with amyloid deposits.

4- Anaplastic carcinoma:

Undifferentiated, highly anaplastic cells (spindle, giant, small cell carcinoma

3- Morphology of hepatocellular carcinoma.

**Grossly:** solitary, multinodular, or massive. Yellow –white, variable in consistency, bile stained, and show areas of hage and necrosis.

**Microscopically:** The following histological variants are recognized:

a) **Trabecular pattern:** cords of tumor cells separated by sinusoidal blood spaces.
b) Psuedoglandular and Acinar pattern: cells arranged around lumen contain bile.
c) Compact pattern: Sinusoid- like blood spaces are slit like (solid appearance).
d) Scirrhouls: Characterized by marked fibrosis.
e) Fibrolamellar: cords of large well differentiated oncocyctic cells separated by parallel collagenous bundles.

4- Pathologic features of secondary syphilis.
1- skin lesions:
   a. Generalized skin rash, macules, papules and pustules.
   b. Alopecia.
   c. Condyloma latum: Flat, red brown elevations
   d. Leukoderma.
2- Mucous patches:
3- Generalized lymph node enlargement

5- Microscopic Picture Gastric carcinoma.
1] Adenocarcinoma: polypoid and ulcerative usually moderately differentiated
2] Mucinous carcinoma
3] Signet ring carcinoma
4] Undifferentiated carcinoma.

Question 5: Enumerate: (2 marks each)
1- Four Complications of lung cancer.
2- Bronchial obstruction leads to pneumonia, lung abscess, bronchiectasis, collapse and wheezes.
3- Cancer cachexia.
4- Direct spread to: Esophagus, Pleura & pericardium, Nerves (Recurrent laryngeal nerve, Phrenic nerve, Vagus nerve)
5-Distant spread: Lymphatic spread, Hematogenous spread (Liver, Bones, Brain and Kidneys)

2- Four sites of metastatic calcification.
1- Renal tubules (nephrocalcinosis).
2- Gastric mucosa.
3- Walls of lung alveoli.
4- Media of the blood vessels.

3- Four Types of Giant cells.
1- Langhan's giant cells
2- Foreign body giant cells
3- Tumor giant cells
4- Multinucleated osteoclast-like giant cells
5- Neoplastic Reed-Sternberg giant cells
6- Aschoff giant cells

4- Effects of portal hypertension.
I- Splanchnic congestion:
Ascities, Congestive splenomegaly, Formation of portosystemic veins anastomosis
(Hemorrhage: rupture varices, Hepatic encephalopathy)

5- Four causes of cirrhosis.
I- Congenital: primary hemochromatosis, Wilson dis., α-1 antitrypsin def.,
glycogen storage disease, thalassemia
II- Acquired: postviral (HBV, HCV, HDV+HBV), alcoholic or nutritional liver
diseases, biliary cirrhosis (primary, secondary), drug-induced, cardiac cirrhosis.
III- Cryptogenic (unknown)

Question 6: 10 marks:
Case I: A 18-year-old male has thalasemia intermedia. Due to recurrent
attacks of hemolysis, he has required multiple transfusion of compact red
blood cells, for all of his life. Lastly, his skin became darker and he
complained of heaviness in his right hypochondrium due to
hepatomegaly. What is the most probable cause of hepatomegaly? (2
marks)
   a- Primary hemochromatosis.
   b- Secondary hemosederosis.
   c- Fatty change liver.
   d- Chronic viral hepatitis.

Case:2: A 59-years old male has had increasing difficulty in
swallowing during the past 6 months. There were no significant finding
on physical examination. Upper GIT endoscopy shows erythematous
mucosa in the most lower part of the esophagus. Biopsy reveals mucosal
lining formed of columnar epithelium with goblet cells.
   a- What is the most probable diagnosis? (2 marks).
      - Barrett’s eosophagus
   b- What is the most dangerous complication likely to occur? (2
      marks).
      - Eosophageal adenocarcinoma

Case 3: A 58-year-old man suffers from a worsening cough for several
months, followed by dyspnea. He feels easy fatigability with the least
effort, and experienced unexplained weight loss. A chest x-ray shows
hilar lymph node enlargement and a right lung mass. He claims that for being a heavy smoker (more than 40 cigarettes / day for several years).

a- What is the most likely diagnosis? (2 marks)
   - Bronchogenic carcinoma

b- Enumerate 3 other etiologic factors which might be involved in the pathogenesis of this condition? (2 marks)
   - Environmental Factors
   - Scar cancer
   - Genetic mechanisms

**Question 7:** (one mark each):

**A- Choose the most appropriate answer:**

1- In cases of fibrocaseous tuberculosis the followings are correct except:
   c
   a- The lesions started at apex.
   b- Caseation is common.
   c- Hilar lymph node enlargement is common.
   d- Basal lesions may occur due to transbronchial spread.

2- Inflammatory exudate is characterized by the following except:
   c
   a- increased protein content.
   b- Increased fibrinogen.
   c- Low specific gravity.
   d- Numerous leucocytes.

3- All the followings are Complications of suppurative inflammation except:
   c
   a- Acute lymphangitis.
   b- Thrombophlebitis.
   c- Caseative lymphadenitis.
   d- All of the above

4- The commonest complication of periportal hepatic fibrosis is:-
   b
   a- Liver cell failure
b- Portal hypertension.
c- Portal and splenic vein thrombosis.
d- Hepatocellular carcinoma.

5- Which of the following conditions is LEAST likely to play a role in the causation of aneurysms?  
a- Atherosclerosis.
b- Polyarteritis nodosa.
c- Congenital weakness of the arterial wall.
d- Rheumatic fever.

6- Infective endocarditis:
   a
   a. In intravenous drug abuses.
b. Untreated acute type is fatal within 3 years
c. Subacute bacterial endocarditis affect healthy valve
d. Carcinoid endocarditis is an example

7- All of the following are likely complications of duodenal ulcers EXCEPT:  
d
   a. Hemorrhage
   b. Perforation
   c. Scarring with obstruction
   d. Complete healing
   e. Malignant change

8- Which of the following is NOT a feature of Familial adenomatous polyposis:
   b
   a. Is an autosomal dominant disorder
   b. Never turn malignant
   c. May reach 2500 colonic adenomas
d. All of the above
e. None of the above.

9- Atopic bronchial asthma:
   b
   a. Mostly appears in adulthood
   b. Associated with positive family history
   c. Is an example for hypersensitivity type II
d. None of the above.
10- Which of the following is predisposed by gall stones:

d
a. Hepatocellular carcinoma
b. Cholangiocarcinoma
c. Hepatoblastoma
d. Gall bladder carcinoma.

Best wishes