Cardiovascular system. Atherosclerosis. Respiratory diseases.

Dentistry

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Endocarditis

1. Non-rheumatic endocarditis
   
   non-infectious
   
   • atypical verrucous (Libman-Sacks) – „collagen disorders“; SLE
   
   • non-bacterial thrombotic (cachexia)

   infectious
   
   • bacterial (BE)
     
     acute – ulcerous – highly virulent bacteria (S.aureus, S.pyogenes)
     
     subacute – polypous – less virulent bacteria (S.viridans, epidermidis)

2. Rheumatic endocarditis (rheumatic fever) - verrucous
Endocarditis – pathogenesis

- **endocardial injury** (cathetrisation, iv drug abuse, more prone - valvular disease and deformities, inborn heart defects, prosthetic valves)
- **exposure of thrombogenic BM**
- **adhesion of platelets**
- **stabilisation by fibrin network**
- **bacterial colonisation during bacteriaemia** (teeth hygiene, diagnostic and treatment procedures – dentistry, gynecology, urology…)

- in ABE – some bacteria can stick to previously non – injured endothelium (they produce polysacharides that injure the endothelium)

**Complications:**

- **CARDIAC** (valves – insufficiency, rupture, abscess, spread of infection)
- **SYSTEMIC** (embolism + septic embolism)
Endocarditis

- Ao, Mi, (Tri – drugs)

- **typical lesions**: **VEGETATIONS** – verrucous masses of platelets, fibrin, inflammatory cells and mo

- clinics: fever (often spiking), septic emboli (causing circulatory problems such as stroke or gangrene of fingers), Janeway lesions (painless hemorrhagic cutaneous lesions on the palms and soles), intracranial hemorrhage, conjunctival hemorrhage, splinter hemorrhages on nails, glomerulonephritis, Osler’s nodules (painful subcutaneous lesions on distal parts of fingers)

- **Dg**: Blood culture, lab (CRP, Leu, FW), ECHO, ECG

- **Th**: Atb
Cardiac valve

Ulcerous acute

Polypous subacute
Endocarditis - histologic appearance

Vegetations – 3 zones:

1. **the outer layer** — eosinophilic material (fibrin and platelets)

2. **underneath** — basophilic zone (colonies of bacteria) – not always present

3. **the deepest zone**
   - in ABE — non-specific inflammatory reaction (mainly Neu, tissue necrosis, abscesses)
   - in SABE — healing by granulation tissue (mainly mononuclear i. cells, proliferation of fibroblasts)
Valve perforation in ulcerative endocarditis
Ulcerative endocarditis, HE

- Platelets and fibrin
- Clusters of bacteria
- Acute inflammatory infiltrate
Ulcerative endocarditis, HE

Platelets and fibrin

Clusters of bacteria

Acute inflammatory infiltrate
Polypous endocarditis
Polypous endocarditis on a mechanical valve
Polypous endocarditis, HE

Fibrin

Bacteria

Leukocyte infiltration
Polypous endocarditis, HE

Fibrin

Leukocyte infiltration
Polypous endocarditis, Gram
Myocardial abscess, HE

- Neutrophils
- Cluster of bacteria
Myocardial abscess, HE

- Fibrous tissue
- Clusters of bacteria
- Neutrophils
Myocardial abscess, Gram

Clusters of bacteria
Cardiomyopathy (255)

• primary (intrinsic) or secondary (extrinsic) chronic heart muscle disease with
deterioration of myocardial function
• generalised inflammation, ischemia, toxic substances (alcohol, cytostatics, As, Co, Pb),
endocrine and metabolic diseases (amyloidosis), ...

<table>
<thead>
<tr>
<th>Dilated CMP</th>
<th>Hypertrophic CMP</th>
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<td>• most common form</td>
<td>• heart muscle (especially of the septum) is thickened</td>
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<td>• with or without obstruction of LV outflow</td>
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<td>• diastol. dysfunction</td>
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Dilated type | Hypertrophic type
CARDIOMYOPATHY
Hypertrophic cardiomyopathy
1. **Dilated CMP** – non-specific, variable changes, hypertrophy of some CMC and atrophy of others, +- interstitial fibrosis

2. **Hypertrophic CMP** – cell disorganisation, cells are irregularly arranged, they cross each other, separated by interstitial fibrous tissue
Hypertrophic cardiomyopathy, HE

- Disorganised architecture of cardiomyocytes
- Markedly abundant fibrous tissue
Hypertrophic cardiomyopathy, HE

Interstitial fibrosis with fibroblasts
Dilated cardiomyopathy
Atherosclerosis (66)

- intimal disease of elastic and muscular arteries.
- chronic inflammatory response in the walls of arteries, in large part to the endothelial damage and to the deposition of lipoproteins.

Risk factors (constitutional and modifiable)

- old age
- male gender: IM is 5 times less frequent in premenopausal women than men; in postmenopausal women incidence equals to men
- genetic factors: positive family history
- hypercholesterolemia: (↑LDL)
- hypertension
- smoking
- obesity
- inactivity
- stress
- alcohol
- diabetes mellitus
Atherosclerosis (66)

Pathogenesis:
• chronic endothelial damage (↑permeability for lipids, monocytes)
• deposition of lipids in artery wall (LDL, VLDL, oxidized forms)
• adhesion of monocytes, leukocytes → migration and change of Ma → foam cells → lysis and release of enzymes
• adhesion of thrombocytes
• release of cytokines from Tr, Leu → migration of smooth muscle cells (SMC) from media to intima → proliferation of SMC and collagen deposition

Clinical manifestation:
• asymptomatic → complications in organs and tissues
• ischaemic changes (IM), aneurysm, thrombosis, embolism
• CP depends on the type of affected tissue
Atherosclerosis – morphology

1) fatty streaks, dots, plaques (lipid-laden foam cell accumulation in intima)

2) atheromas – atheromatous plaques
   central core – cholesterol, foam cells
   fibrous cap

3) fibrous lesions (scaring of atheromatous plaque)

4) complicated lesions (thrombus formation, embolism, ulceration/rupture, calcification, hemorrhage, aneurysm...)
Atherosclerosis of thoracic aorta

- Fatty streaks on intimal surface
- Calcification
Atherosclerosis of abdominal aorta

Calcification

Thrombotic mass on the luminal surface

Atherosclerosis of abdominal aorta
Calcification

Lumen partially obstructed by the plaque

Atherosclerosis, HE
Emphysema of the lungs (141)

• permanent dilatation of air spaces distal to the terminal bronchioli caused by loss of elasticity and destruction of the walls
• included in COPD

Etiology: imbalance between proteases and anti-proteases (↓ α1-antitrypsin – smoking)

Forms: centrolobular, panlobular, parasepta, irregular

Macro: lungs are inflated, larger, softer
Micro: alveolar wall destruction
Complications: pneumothorax (rupture of bulla), chronic bronchitis, cor pulmonale
Emphysematous bulla

Lung emphysema
Lung emphysema

Ruptured emphysemous bulla
Markedly dilated alveolar spaces

Lung emphysema, HE
Markedly dilated alveolar spaces

Torn alveolar septa

Lung emphysema, HE
Pneumonia (86)

Forms: Superficial → 1. Lobar, 2. Bronchopneumonia; 3. Interstitial pneumonia

A. Bacterial pneumonia, B. Primary atypical pneumonia (viral, chalmydia, mycoplasma), C. Special types (aspirative, hypostatic, ...)

- etiology - Nosocomial (staphylococcus, G- Klebsiella, Pseudomonas, Proteus, ...), Community aquired (pneumococcus, streptococci, hemophilus, mycoplasma, chlamydia, legionella, viral, ...)

Pathogenesis: inhalation pathway, aspirative, hematogenous dissemination

Complications: abscess formation, sepsis, pleuritis
Lobar pneumonia (83)

- affecting the entire lobe / several lobes (also bilaterally)
- *Pneumococcus*
- 4 stages: congestion (intra-alveolar fluid), red hepatisation (ery, fibrin, neu), grey hepatisation (fibrin, neu), resolution
- Complications: organisation – lung carnification, empyema, abscess formation, hematogenous dissemination

Bronchopneumonia (86)

- caused by infection of terminal bronchioles which extend into adjacent alveoli resulting into **patchy consolidation of the lungs**
- *S.aureus, S.pyogenes, H.influenzae*
- often bilateral, multilobar
- Forms: aspirative, hypostatic, post-operative, dystelectatic, bronchostenotic
- Micro: neu, exudate extends from bronchi into adjacent alveoli

Interstitial pneumonia, primary atypical pneumonia

- **viruses, mycoplasma, chlamydia**
Lobar pneumonia

Consolidation of an entire lobe
Lobar pneumonia, HE - congestion
Dilatation and congestion of capillaries

Intraalveolar fluid with more neu

Fibrin

Lobar pneumonia, HE – red hepatisation
Lobar pneumonia, HE – grey hepatisation

- Dilatation and congestion of capillaries
- Abundant neutrophils in alveoli
- Fibrin
Dilatation and congestion of capillaries

Fibrin

Macrophages with a few neu

Lobar pneumonia, HE - resolution
Bronchopneumonia

Patchy infiltration of lung parenchyma

Abundant liquid

Pus
Bronchopneumonia

Foci of pus
Bronchopneumonia, HE

- Bronchiolus with numerous neu
- Neu in alveoli
- Dilatation and congestion of capillaries
- Edema
- Unaffected alveolus
1. **Epithelial tumors**
   1. Benign
      1. Papiloma
      2. Adenoma
   2. Malignant

2. **Soft tissue tumors** (chondroma, myoepithelioma, ....)

3. **Lymphohistiocytic tumors** (lymphoma, ....)

4. **Tumors of ectopic origin** (malignant melanoma, teratoma, ...)

5. **Secondary tumors**
Carcinoma of the Lung

Classification: Malignant epithelial tumors

a) Small cell carcinoma (poor prognosis, neuroendocrine activity, small round basophilic cells, different therapeutic approach)

b) Squamous cell carcinoma

c) Adenocarcinoma (lepidic adenocarcinoma)

d) Large cell carcinoma

e) Adenosquamous carcinoma

f) ...

Other tumors (mesenchymal tumors, lymphomas, metastatic...)

Non-small cell carcinoma
Carcinoma of the Lung

Etiology: **smoking**, atmospheric pollution, radiation, asbestos, nickel, beryllium, arsenic, iron, vitamin A deficiency, chronic scarring

Localisation: **central** (often hilus), **peripheral**, „Pancoast tumor“ in apex of lungs

Clinical picture:

- **primary tumor** (coughing, hemoptysis, dyspnoe, chest pains, recurrent pneumonia, Horner triad, vena cava sy, dysphagia, …)
- **spread of the primary tumor**
- **systemic**
- **paraneoplastic sy** (migrating thrombophlebitis, Lambert-Eaton sy., polymyositis, Cushing sy – ACTH, hypercalcaemia – parathormone…)
Lung carcinoma

Poorly defined area of infiltrative growth (peripheral type)

Anthracosis
Lung carcinoma

Poorly defined area of infiltrative growth (hilar type)
Poorly defined area of infiltrative growth

Lung carcinoma
Small cell lung carcinoma, HE

Tumor composed of small blue round cells infiltratively growing into lung tissue.
Small cell lung carcinoma, HE

Tumor composed of pleomorphic small blue round cells

Atypical mitosis
Small cell lung carcinoma – cardiac infiltration
Small cell lung carcinoma – liver metastasis
Tumor composed of small blue round cells infiltratively growing into liver

Hepatocytes with steatosis

Small cell lung carcinoma – liver metastasis, HE
Squamous cell lung carcinoma
Squamous cell lung carcinoma, HE

Squamous tumor cells infiltratively growing into lung tissue

Anthracosis
Squamous cell lung carcinoma, HE

Keratin pearl formation amid squamous cells with cellular and nuclear atypia
Lepidic lung carcinoma
Lepidic lung carcinoma, HE

Alveolar structure is preserved, tumor cells grow inside the alveolar space, copying its shape
Lepidic lung carcinoma, HE

Mitosis
Questions

1. Can infectious endocarditis cause infarction of remote tissues?
2. Which conditions can lead to concentric hypertrophy of left ventricle?
3. What are the main symptoms of interstitial pneumonia? How is it diagnosed?
4. What is the clinical significance of histologic evaluation of lung tumors?