LAB 3: THE MUSCLE AND CARDIOVASCULAR SYSTEM

The focus of this week’s lab will be pathology of the cardiovascular system.

The cases we will cover are:

A. **Atherosclerosis** Refer to virtual slide p_8, should be available in the virtual slides by Monday

B. **Hypertension (hyaline and hyperplastic arteriolar sclerosis)**

C. **Myocardial Infarction (different timepoints)** Refer to virtual slide p_259 heart, recent MI with mucal thromb at [https://med-vmicro.med.illinois.edu/v/360/](https://med-vmicro.med.illinois.edu/v/360/) and virtual slide p_36 heart, infarct at [https://med-vmicro.med.illinois.edu/v/364/](https://med-vmicro.med.illinois.edu/v/364/)

D. **Infective Endocarditis (Acute)**

E. **Lymphocytic Myocarditis** Refer to virtual slide p_166 heart, myocarditis, rheumatic at [https://med-vmicro.med.illinois.edu/v/357/](https://med-vmicro.med.illinois.edu/v/357/)
A. Atherosclerosis

CC/HPI: A 59-year-old man complains of pain in calf muscles during exercise (claudication) that is relieved by rest. His symptoms have been occurring for a year. He is a smoker and smokes two packs of cigarettes per day.

PE: Physical exam reveals hypertension (BP 150/100); diminished peripheral pulses bilaterally; carotid and femoral arterial bruits.

Labs: Elevated LDL (>60) and decreased HDL (<100); elevated total serum cholesterol (>200).

Pathology: Histology of a coronary artery reveals:

- What are the layers of the blood vessel wall?
- Which layer is thickened in this sample due to the presence of an atherosclerotic plaque?
- Describe the histological components of an atherosclerotic plaque.
- What cell types are present in the shoulder of the atherosclerotic plaque?
- Where do these cells come from?

B. Hypertension (Hyaline and Hyperplastic Arteriolosclerosis)

CC/HPI: A 42-year-old man presents with chest pain and a headache. He has a history of labile essential hypertension for a few years.

PE: No fever, severe hypertension (BP 230/150). Bilateral papilledema; no focal neurological defects.

Labs/Imaging: Urinalysis reveals hematuria. Increased serum BUN and creatinine. Left ventricular hypertrophy.
What type of blood vessel is most significant for regulation of blood pressure?

How would luminal narrowing alter vascular resistance in this vessel?

If everything else remained constant, how would this change mean arterial pressure (MAP)? How could this change in total peripheral resistance (TPR) and MAP result in the left ventricular hypertrophy seen in this patient?

Pathology: Representative sections of his arterioles are shown below. The left picture indicates an earlier stage in this disease process and the right is more advanced.

What extracellular matrix feature is thickened (usually by duplication) in the sample on the left?

What cell type has proliferated in the section on the right?

C. MYOCARDIAL INFARCTION

CC/HPI: A 65-year-old white man is brought to the emergency room with nausea, dyspnea (shortness of breath), and a crushing substernal chest pain that radiates to his left arm and jaw. The pain has lasted for 30 minutes and is not relieved by rest. One sublingual nitroglycerin tablet did not relieve his pain. He has a history that includes a sedentary lifestyle, moderate hypercholesteremia, and obesity. He is also diabetic and a smoker.

PE: Physical exam reveals hypotension; diaphoresis (sweating).

Labs/Imaging: EKG indicates ST elevation with peaking of T waves; subsequent development of inverted T waves and prominent Q waves. Later, ST and T waves normalize. Elevated CK-MB; elevated troponin T and I. Complete blood count shows leukocytosis.

What are CK-MB and troponin T? Why are they high in this patient?

Note: You are not expected to cover the EKG changes in your presentation of this case. Normal EKG will be discussed in Case E. We have provided the EKG information so that interested students can examine the complete presentation of a myocardial infarction, but you do not need to go over the EKG information in any detail.
Pathology: Myocardium biopsies taken at different times after a myocardial infarction are shown below:

A: 1 day post infarct. B: 2-3 days. C: 7 days. D: 10 days. E: 2-3 weeks.

What looks different in the sample taken one day after myocardial infarction?

What looks different in the well-healed myocardial infarction?

What cell types have infiltrated at 2-3 days after infarction? What about at 7-10 days after infarction?

What does the sequence of cell infiltration tell you about the progression of healing after an MI?

D. INFECTIVE ENDOCARDITIS (ACUTE)

CC/HPI: A 34-year-old man with a history of IV drug use presents to the emergency department complaining of fever, chills, and weakness.

PE: Patient has fever (102.3 F). Physical exam reveals holosystolic murmur heard best at the 4th left sternal border that intensifies with inspiration. S3 heart sound observed.

Labs/Imaging: Leukocytosis with increased bands. Blood cultures are taken and later indicate s. aureus infection. Echocardiogram indicates abnormal contour on tricuspid valve.

Where does s. aureus usually reside in a healthy person?

A holosystolic murmur heard best over the 4th left sternal border that intensifies with inspiration suggests that which valve is affected?

Based on your knowledge of blood flow and this patient’s history explain why this valve is infected with this organism in this patient.
Pathology: A histological section of the patient’s valve shows the following:

What looks abnormal about this heart valve?

Structurally heart valves are composed of...

E. LYMPHOCYTIC MYOCARDITIS

CC/HPI: A 25-year-old man complains of increasing shortness of breath and ankle edema that have worsened over the past two weeks. He also reports fatigue, heart palpitations, and a low fever. The development of his symptoms was preceded by an upper respiratory tract infection. He denies any rashes or pain in his joints.

PE: Physical exam reveals JVP elevated; pitting edema in lower extremities; fine inspiratory crepitations heard at both lung bases; mild hepatosplenomegaly.

Labs: ASO titers not elevated. Lymphocytosis in blood. EKG indicates first-degree AV block. Erythrocyte sedimentation rate (ESR) elevated; increased titers of antibodies to coxsackievirus in serum. CRP elevated.

What does the increased antibody titer to coxsackie virus indicate?

Draw a normal EKG. Label the stages and the corresponding electrical activity of each stage.

What tissue generates the electrical signals in the heart, and how does the contraction spread?

Pathology: A cardiac biopsy reveals the following:
What cells are present in this biopsy that are not normally in cardiac tissue?

What do these cells do?