Author(s): Gerald Abrams, M.D., Stephen Ramsburgh, M.D., 2009

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Pathology

- Etiology of disease
- Pathogenesis of disease
- Structural alterations resulting from disease (gross and microscopic)
- Natural history of the disease process
- Functional consequences of disease
Pathology

- Systemic Pathology
- Specific Diseases in Specific Organs

- If you understand basic disease process such as cell death, inflammation, and neoplasia, the questions of etiology, pathogenesis, natural history and functional consequences of specific diseases will fall into place in a rather common sense fashion.
Aortic atherosclerosis
Coronary atherosclerosis
Hyperplastic arteriolosclerosis
Hyaline arteriolosclerosis
Polyarteritis nodosa
Aortic dissection
Aortic Atherosclerosis
Coronary Atherosclerosis
Coronary Atherosclerosis
Hyperplastic Arteriolosclerosis
Hyaline Arteriosclerosis
Polyarteritis Nodosa
Aortic Dissection

Dissecting Aneurysm of the Aorta

Lumen

Cystic medial necrosis

basophilic amorphous (mucoid) material in the media

Intimal Tear

Intima

Inner Media

Outer Media

Adventitia

Hemorrhage into the media with laminar dissect of the aorta

Cross section of a dissecting aneurysm of the aorta

Department of Pathology, University of Michigan
Heart and Aortic Arch
Left Subclavian Artery
Aortic Dissection
Aortic Dissection
Normal Aortic Elastin  

Fragmented Aortic Elastin Marfan’s

G.D. Abrams, University of Michigan Medical School (Both Images)
Abdominal Aortic Aneurysm
Syphilitic Aortitis
Aneurysm
Syphilic Aortitis
<table>
<thead>
<tr>
<th>Page</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>54</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>35</td>
<td>Thrombus in IVC</td>
</tr>
<tr>
<td>36</td>
<td>Pulmonary infarction</td>
</tr>
<tr>
<td>5</td>
<td>Renal infarction</td>
</tr>
</tbody>
</table>
Recent Myocardial Infarction
After ~ 40 minutes of ischemia myocardial cells die and membranes leak enzymes and proteins.

<table>
<thead>
<tr>
<th>Test</th>
<th>Onset</th>
<th>Peak*</th>
<th>Return</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac creatine kinase (CK-MB)</td>
<td>2 - 4 hours</td>
<td>24 hours</td>
<td>72 hours</td>
</tr>
<tr>
<td>Cardiac troponins (-I and -T)</td>
<td>2 - 4 hours</td>
<td>48 hours</td>
<td>7 - 10 days</td>
</tr>
</tbody>
</table>

*Peak accelerated if myocardium reperfused (enzyme “washout” from necrotic myocardium)
Histological Evolution of Myocardial Infarct

Fig. 3.49 Diagram of histology at various stages of infarct repair.
Antemortem Thrombus/Embolus
The Stages of Venous Thrombosis

1. Partial occlusion
   - Initial thrombus in vein packed
   - Fibrin
   - Platelet

2. Total occlusion
   - Formation of thrombus progressively
   - Cutting of sheath
   - Blood vessel (retrograde指引)
   - Fibrin
   - Mediate
   - Connect sheath/tissue
Thrombus in IVC
Pulmonary Infarct
Pulmonary Infarct
Renal Infarct
Renal Infarct
M-2 Cardiovascular Laboratory - 3

30  
   Acute pulmonary edema

32  
   Central hemorrhagic necrosis

31  
   Chronic pulmonary congestion

88  
   Colon ischemia

37  
   Myocardial hypertrophy

9   
   Gangrene
Acute Pulmonary Edema
Acute Pulmonary Edema
Central Hemorrhagic Necrosis
Central Hemorrhagic Necrosis
Congested Lung
Chronic Pulmonary Congestion
Ischemic Colitis
Ischemic Colitis
Colon Ischemia
Normal Heart

Hypertrophied Heart

Department of Pathology, University of Michigan (both images)
Myocardial Hypertrophy
Gangrene

G.D. Abrams, University of Michigan Medical School
50  Rheumatic heart disease
    Pericarditis
    Myocarditis
    Endocarditis
51  Infective endocarditis
58  Myocarditis
21  Pericarditis
   Atrial Myxoma
Rheumatic Heart Disease
Rheumatic Pericarditis

G.D. Abrams, University of Michigan Medical School
Rheumatic Myocarditis
Rheumatic Endocarditis

Vegetations

Department of Pathology, University of Michigan
Rheumatic Endocarditis
Bacterial Endocarditis - Aortic Valve
Bacterial Endocarditis - Aortic Valve
Pericarditis (epicarditis)
Constrictive Pericarditis
Pericarditis (epicarditis)
Case Presentation

Atrial Myxoma
Case presentation - Atrial Myxoma
Following slides are intended for review
1. Discuss the pathologic findings and offer a diagnosis

2. Why did circulatory collapse occur?

3. What blood test may have aided in the diagnosis of this disorder?

4. What risk factors are associated with this disorder?
Pulmonary embolus secondary to DVT
Cardio Lab 5
Case Presentation 2
(Refer to lab syllabus for the clinical information)

1. Describe the pathologic findings and form a diagnosis.

2. What other pathologic abnormalities might have been observed, with regard to the arterial system?

3. What pathologic finding was most likely found in his cerebrum?

4. What abnormal physical findings might have been present?
Intra-retinal hemorrhage in ischemic area, "cotton wool" hemorrhage in ischemic area ("cotton wool")
Malignant hypertension
Cardio Lab 5
Case Presentation 3
(Refer to lab syllabus for the clinical information)

1. Describe the pathologic findings and form a pathologic diagnosis.

2. Discuss or explain the clinical course and physical findings, in light of the pathologic findings
hypopyon
gram-positive cocci
Bacterial endocarditis
1. Describe the pathologic findings.

2. How do the pathologic findings explain the clinical course, and the clinical and laboratory findings?

3. What additional pathologic feature(s) might have been found in the heart if the patient had died on the 12th hospital day?
Myocardial infarction
Cardio Lab 5  
Case Presentation 5  

(Refer to the lab syllabus for the clinical information)  

1) Based on the pathologic features manifested in the sample, please offer a differential diagnosis.  
2) Based on the pathologic and clinical features, please offer a diagnosis.  
3) What is the relationship between this patient’s disorder and hepatitis B infection?  
4) What other clinical signs and symptoms have been associated with the patient’s disorder?
Polyarteritis Nodosa