Cardiac Pathology 2:
Heart Failure, Ischemic Heart Disease and other assorted stuff

Kristine Krafts, M.D.
Cardiac Pathology Outline

- Blood Vessels
- Heart I
- Heart II
Cardiac Pathology Outline

• Blood Vessels
• Heart I
  • Heart Failure
  • Congenital Heart Disease
  • Ischemic Heart Disease
  • Hypertensive Heart Disease
Cardiac Pathology Outline

- Blood Vessels
- Heart I
  - Heart Failure
Heart Failure

- End point of many heart diseases
- Common!
  - 5 million affected each year
  - 300,000 fatalities
- Most due to systolic dysfunction
- Some due to diastolic dysfunction, valve failure, or abnormal load
- Heart can’t pump blood fast enough to meet needs of body
Heart Failure

• System responds to failure by
  • Releasing hormones (e.g., norepinephrine)
  • Frank-Starling mechanism
  • Hypertrophy

• Initially, this works

• Eventually, it doesn’t
  • Myocytes degenerate
  • Heart needs more oxygen
  • Myocardium becomes vulnerable to ischemia
Clinical consequences of left and right heart failure

- cyanosis
- pulmonary edema
- hepatomegaly
- splenomegaly
- ascites
- peripheral edema
Left Heart Failure

• Left ventricle fails; blood backs up in lungs

• Commonest causes
  • Ischemic heart disease (IHD)
  • Systemic hypertension
  • Mitral or aortic valve disease
  • Primary heart diseases

• Heart changes
  • LV hypertrophy, dilation
  • LA may be enlarged too (risk of atrial fibrillation)
Left Heart Failure

- Dyspnea
- Orthopnea, paroxysmal nocturnal dyspnea too
- Enlarged heart, increased heart rate, fine rales at lung bases
- Later: mitral regurgitation, systolic murmur
- If atrium is big, irregularly irregular heartbeat
Right Heart Failure

- Right ventricle fails; blood backs up in body

- Commonest causes
  - Left heart failure
  - Lung disease ("cor pulmonale")
  - Some congenital heart diseases

- Heart changes
  - right ventricular hypertrophy, dilation
  - right atrial enlargement
Right Heart Failure

- Peripheral edema
- Big, congested liver (nutmeg liver)
- Big spleen
- Most chronic cases of heart failure are bilateral
Hepatic blood flow
“Nutmeg” liver  

Nutmeg
Cardiac Pathology Outline

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  - Congenital Heart Disease
Congenital Heart Disease

- Abnormalities of heart/great vessels present from birth
- Faulty embryogenesis, weeks 3-8
- Broad spectrum of severity
- Cause unknown in 90% of cases
Congenital Heart Disease

- Left-to-right shunts
  - atrial septal defects
  - ventricular septal defects
  - Patent ductus arteriosus

- Right-to-left shunts
  - tetralogy of fallot
  - transposition of the great arteries

- Obstructions
  - aortic coarctation
Atrial Septal Defects

• Initially, left-to-right shunt (asymptomatic)

• Eventually, pulmonary vessels may become constricted (pulmonary hypertension), leading to right-to-left shunt (Eisenmenger syndrome)

• Surgical repair prevents irreversible pulmonary changes and heart failure
Ventricular Septal Defects

- Most common congenital cardiac anomaly
- Most close spontaneously in childhood
- Small VSD: asymptomatic
- Large VSD: big left-to-right shunt, may become right-to-left
VSD
Patent Ductus Arteriosus

- Ductus: allows flow from PA to aorta
- Closes spontaneously by day 1-2 of life
- Small PDA: asymptomatic
- Large PDA: shunt becomes right-to-left
PDA
Most common cause of cyanotic congenital heart disease

Four features:
- VSD
- obstruction to RV outflow tract
- overriding aorta
- RV hypertrophy

Cyanosis, erythrocytosis, clubbing of fingertips, paradoxical emboli
Tetralogy of Fallot
Clubbing of fingertips
Normal (L) and clubbed (R) fingertips
Transposition of Great Arteries

- Aorta arises from R ventricle; pulmonary artery arises from L ventricle
- Outcome: separation of systemic and pulmonary circulations
- Incompatible with life unless there is a big shunt (VSD)
Aortic Coarctation

- Coarctation = narrowing
- “Infantile” (preductal) and “adult” (postductal) forms
- Cyanosis and/or low blood pressure in lower extremities
- Severity depends on degree of coarctation
Coarctation of the aorta
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Ischemic Heart Disease

• Myocardial perfusion can’t meet demand
• Usually caused by decreased coronary artery blood flow (“coronary artery disease”)
• Four syndromes:
  • angina pectoris
  • acute MI
  • chronic IHD
  • sudden cardiac death
Angina Pectoris

• Intermittent chest pain caused by transient, reversible ischemia

• Typical (stable) angina
  • pain on exertion
  • fixed narrowing of coronary artery

• Prinzmetal (variant) angina
  • pain at rest
  • coronary artery spasm of unknown etiology

• Unstable (pre-infarction) angina
  • increasing pain with less exertion
  • plaque disruption and thrombosis
Myocardial Infarction

- Necrosis of heart muscle caused by ischemia
- 1.5 million people get MIs each year
- Most due to acute coronary artery thrombosis
  - sudden plaque disruption
  - platelets adhere
  - coagulation cascade activated
  - thrombus occludes lumen within minutes
  - irreversible injury/cell death in 20-40 minutes
- Prompt reperfusion can salvage myocardium
Morphologic Changes in Myocardial Infarction

<table>
<thead>
<tr>
<th>Time</th>
<th>Gross changes</th>
<th>Microscopic changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4h</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>4-12h</td>
<td>Mottling</td>
<td>Coagulation necrosis</td>
</tr>
<tr>
<td>12-24h</td>
<td>Mottling</td>
<td>More coagulation necrosis; neutrophils come in</td>
</tr>
<tr>
<td>1-7 d</td>
<td>Yellow infarct center</td>
<td>Neutrophils die, macrophages come to eat dead cells</td>
</tr>
<tr>
<td>1-2 w</td>
<td>Yellow center, red borders</td>
<td>Granulation tissue</td>
</tr>
<tr>
<td>2-8 w</td>
<td>Scar</td>
<td>Collagen</td>
</tr>
</tbody>
</table>
Acute Myocardial Infarction
MI: day 1, day 3, day 7
Myocardial Infarction

- **Clinical features**
  - Severe, crushing chest pain ± radiation
  - Not relieved by nitroglycerin, rest
  - Sweating, nausea, dyspnea
  - Sometimes no symptoms

- **Laboratory evaluation**
  - Troponins increase within 2-4 hours, remain elevated for a week.
  - CK-MB increases within 2-4 hours, returns to normal within 72 hours.
Myocardial Infarction

• Complications
  • contractile dysfunction
  • arrhythmias
  • rupture
  • chronic progressive heart failure

• Prognosis
  • depends on remaining function and perfusion
  • overall 1 year mortality: 30%
  • 3-4% mortality per year thereafter
Rupture of papillary muscle after MI
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Hypertensive Heart Disease

- Can affect either L or R ventricle
- Cor pulmonale is RV enlargement due to pulmonary hypertension caused by primary lung disorders
- Result: myocyte hypertrophy
- Reasons for heart failure in hypertension are poorly understood
Left ventricular hypertrophy (L) and cor pulmonale (R)