Cardiac Pathology 1: Blood Vessels

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Cardiac Pathology Outline

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

- Blood Vessels
  - Atherosclerosis
  - Hypertension
  - Aneurysms
  - Vasculitis
  - Tumors
Cardiac Pathology Outline

- Blood Vessels
  - Atherosclerosis
Normal blood vessel
Atherosclerosis

- Characterized by atheromas (plaques)
- Half of deaths in US!
- Most common cause of myocardial infarction
- Most common cause of cerebral infarction
Major Risk Factors for Atherosclerosis

Non-modifiable
- Increasing age
- Male gender*
- Family history
- Genetic abnormalities

Modifiable
- Hyperlipidemia
- Hypertension
- Cigarette smoking
- Diabetes

* Sort of. MI is uncommon in premenopausal women, but after menopause, incidence in women exceeds that in men.
Other Risk Factors for Atherosclerosis

- Inflammation
- Metabolic syndrome
- Obesity
- Lack of exercise
- “Stress”
How Plaques Form

It all starts with chronic endothelial injury (from smoking, hyperlipidemia, etc.). The endothelium looks normal at first.
Eventually, the injured endothelium shows signs of damage. It becomes more permeable, and monocytes and platelets begin to stick to it.
Monocytes slip into the subendothelium and become macrophages. Smooth muscle cells proliferate in the subendothelium. Lipid accumulates.
How Plaques Form

Macrophages and smooth muscle cells ingest lipids. T cells show up. A fatty streak is now visible.
How Plaques Form

Smooth muscle cells proliferate in the intima and start laying down collagen. Now there’s a plaque with a soft core and a brittle surface.
Fatty streak
Mild (L) and severe (R) atherosclerosis
Atheromatous plaques
Natural history of atherosclerosis
Prevention of Atherosclerosis

- Primary prevention
  - Lessen risk factors
  - Statins

- Secondary prevention
  - Aspirin, statins, beta blockers
  - Surgery
Cardiac Pathology Outline

- Blood Vessels
  - Atherosclerosis
  - Hypertension
Hypertension

• Common problem (25% of population)
• Asymptomatic until late
• Contributes to coronary artery disease, stroke, cardiac hypertrophy, heart failure
• Mechanisms largely unknown - called “essential hypertension”
• >140/90
Types of Hypertension

- Benign hypertension
  - Essential (idiopathic) hypertension
  - Secondary hypertension
- Malignant hypertension
Essential Hypertension

- Idiopathic! But probably related to...
- Reduced renal sodium excretion
- Vascular changes
- Genetic factors
- Environmental factors
Essential Hypertension

- Accelerates atherogenesis
- Potentiates aortic dissection and stroke
- Arterioles eventually become damaged
  - Hyaline arteriolosclerosis
  - Hyperplastic arteriolosclerosis
Hyaline (L) and hyperplastic (R) arteriolosclerosis
Cardiac Pathology Outline

- Blood Vessels
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  - Hypertension
  - Aneurysms
Aneurysms

- Aneurysm: localized abnormal vessel dilation
- “True” aneurysm: involves all three layers
- “False” aneurysm: hole covered with hematoma
True aneurysm

Normal vessel

False aneurysm

- Extravascular connective tissue
- Extravasation of blood
- Hematoma
Causes of Aneurysms

- Atherosclerosis
- Cystic medial degeneration of wall
- Trauma
- Congenital defects (berry aneurysm)
- Infection (mycotic aneurysms)
Abdominal Aortic Aneurysm

- Males >50
- Atherosclerosis, genetic defects (Marfan)
- Below renal arteries, above bifurcation
- May present as pulsating abdominal mass
- May rupture, obstruct branches or embolize
Abdominal aortic aneurysm
Abdominal aortic aneurysm
Abdominal aortic aneurysm repair

Graft being sewn into place
Aortic Dissection

• Blood tracks up through media, creating a channel
• Hypertensive men, 40-60 (most cases)
• Sudden onset excruciating pain
• Can rupture → massive hemorrhage or cardiac tamponade
• Rapid diagnosis, surgery = 65-75% of patients survive
Type A
Worse prognosis

Type B
Better prognosis
Cardiac Pathology Outline

- Blood Vessels
  - Atherosclerosis
  - Hypertension
  - Aneurysms
  - Vasculitis
Vasculitis

- Inflammation of vessel walls
- Many possible symptoms
- Constitutional signs/symptoms common
- Immune-mediated or infectious
## Summary of Vasculitides

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Disease</th>
<th>ROS *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large</td>
<td>Giant-cell arteritis</td>
<td>&gt;50. Arteries of head.</td>
</tr>
<tr>
<td></td>
<td>Takayasu arteritis</td>
<td>F &lt;40. “Pulseless disease”</td>
</tr>
<tr>
<td>Medium</td>
<td>Polyarteritis nodosa</td>
<td>Young adults. Widespread.</td>
</tr>
<tr>
<td>Small</td>
<td>Wegener granulomatosis</td>
<td>Lung, kidney. c-ANCA.</td>
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<tr>
<td></td>
<td>Churg-Strauss syndrome</td>
<td>Lung. Eosinophils. Asthma. p-ANCA.</td>
</tr>
<tr>
<td></td>
<td>Microscopic polyangiitis</td>
<td>Lung, kidney. p-ANCA.</td>
</tr>
</tbody>
</table>

* Ridiculously oversimplified summary
Giant-Cell (Temporal) Arteritis

- Most common type of vasculitis
- Patients >50
- Chronic, granulomatous inflammation of large to small arteries, especially in head
- Symptoms vague (fever) or localized (headache, vision loss)
- Treatment: corticosteroids
Giant cell (temporal) arteritis
Takayasu Arteritis

- Women <40
- Granulomatous vasculitis of aortic arch
- Severe narrowing of major branches
- Weakening of pulses in upper extremities ("pulseless disease")
- Ocular disturbances
Takayasu arteritis
• Young adults
• Necrotizing vasculitis in many different organs
• Different stages coexist even in same artery
• Puzzling, varied symptoms
• Fatal if untreated, but steroids and cyclophosphamide are curative
PAN: fibrinoid necrosis and thrombotic occlusion
Kawasaki Disease

- Children <4
- Acute, febrile, usually self-limiting
- Danger: involvement of coronary arteries
- “Mucocutaneous lymph node syndrome”
- Delayed-type hypersensitivity reaction?
- Treatment: intravenous Ig
Kawasaki Disease: hand edema, mouth lesions
Kawasaki Disease: “strawberry” tongue
Wegener Granulomatosis

- Most common age = 40s
- Triad: respiratory tract granulomas, vasculitis, renal disease
- c-ANCA positive
- T-cell mediated hypersensitivity response?
- Untreated: fatal in 1 year
- Churg-Strauss: similar, but associated with allergies and asthma, and no renal disease
Wegener granulomatosis: cavitating lung lesions
Wegener granulomatosis: palatal ulceration
Wegener granulomatosis: palatal destruction
Mnemonic:
Wegener Granulomatosis is the “C” disease

- C through the body: in through nose, out through kidneys
- c-ANCA
- Cell-mediated response
- Saddle nose “C” shaped
- Treatment: cyclophosphamide, corticosteroids
- Churg-strauss similar
Microscopic (Leukocytoclastic) Polyangiitis

- Widespread, necrotizing vasculitis of smaller vessels
- Lung and kidney especially
- Antibody response to drugs or bugs
- Neutrophils in vessels
- Type III hypersensitivity reaction?
- Removing offending agent usually works
Leukocytoclastic polyangiitis: vessel with fragmented PMNS
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Hemangioma

- Very common benign tumor of blood vessels
- Capillary hemangioma
  - Skin, oral mucosa, sometimes organs
  - “Strawberry” type present at birth, regresses
- Cavernous hemangioma
  - Organs, sometimes skin
  - Cosmetic problem (unless in brain)
- Pyogenic granuloma
  - Rapidly growing red nodule on skin, in mouth
  - Microscopically resembles granulation tissue
Capillary hemangioma
Pyogenic granuloma
• Benign but very painful
• Arise from glomus body cells
• Distal digits, especially under fingernails
• Excision is curative
Glomus tumor
Kaposi Sarcoma

- Low-grade malignancy of endothelial cells
- Four forms: Chronic (older Ashkenazi Jewish males), African, transplant-associated, AIDS-associated
- Clinical course varies (chronic = best)
- Excision can be curative
Kaposi sarcoma
Kaposi sarcoma
Kaposi sarcoma
Angiosarcoma

- Malignancy of endothelial cells
- Prefers skin, soft tissue, breast, liver
- Arsenic and PVC increase risk
- Well-differentiated to anaplastic
- Metastasize rapidly. 5ys 30%.
Angiosarcoma
Angiosarcoma cells positive for CD31 (an endothelial marker)