Heart and Coronary Arteries

Brett Hambly
The Pathogenesis of Atherosclerosis

A: The normal artery has three layers, the intima, media and adventitia. Endothelial injury is the initiating factor in atherosclerosis.

B: The fatty streak is formed by accumulation and oxidation of lipoproteins (mainly LDL) and the initiation of monocyte adhesion and migration.

C: Monocytes adhere to the wall and migrate between endothelial cells to the intima, where they become mature macrophages. T cells also invade the intima and VSMCs migrate from the media to the intima. Lipids continue to accumulate and undergo oxidation.

D: After migration to the intimal layer, VSMCs proliferate and become synthetic. More lipid accumulates and some is engulfed by VSMCs and macrophages, which become foam cells.

E: Advanced lesions are characterised by continued localisation of macrophages, VSMCs, T cells and lipids and the formation of the necrotic core and fibrous cap.
Atheromatous artery

- Media greatly thinned
- Necrotic core pale, under the fibrous cap

- Irregular thickness of wall of artery due thickened intima
- Lumen is narrow and eccentric.

Necrotic core of atheromatous artery

- Numerous pale clefts
- Contained crystals of cholesterol (extracellular lipid)

Thickened intima of atheromatous artery

- Foam cells (arrows)
- Lipid engorged macrophages
- Contain some of the excess intimal lipid.
Coronary atherosclerosis

- Thickened intima.
- Necrotic core covered by a fibrous cap (double arrow).
- Lumen is occluded by thrombosis (arrow).
- Thrombus = RBCs enclosed within a meshwork of fibrin.
**Myocardium: recent infarct**

Pale infarct with hyperemic (red, inflamed) border

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**Normal myocardium**

- Branching fibres
- Central nuclei
Myocardial infarct ~ day 2

- Necrotic myocytes with few recognisable nuclei
- Infiltrated by acute inflammatory cells, the nuclei of which have disintegrated.
- Vascular congestion (arrows).

- Higher magnification
- Inflammatory cells are recognisable as neutrophils, some with lobed nuclei (arrows).

Evolution of myocardial infarction

<table>
<thead>
<tr>
<th>Macro</th>
<th>Micro</th>
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<tbody>
<tr>
<td>0-4 hours</td>
<td>None</td>
</tr>
<tr>
<td>4-24 hours</td>
<td>Dark mottling</td>
</tr>
<tr>
<td>1-7 days</td>
<td>Tan infarct centre</td>
</tr>
<tr>
<td>7-14 days</td>
<td>Hyperemic border, tan centre</td>
</tr>
<tr>
<td>2-8 weeks</td>
<td>Grey/white collagenous scar forms</td>
</tr>
<tr>
<td>2 months</td>
<td>Scarring complete</td>
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</tbody>
</table>
Myocardial repair

LV aneurysm from old MI

Areas of myocardial fibrosis