Ischaemia & infarction

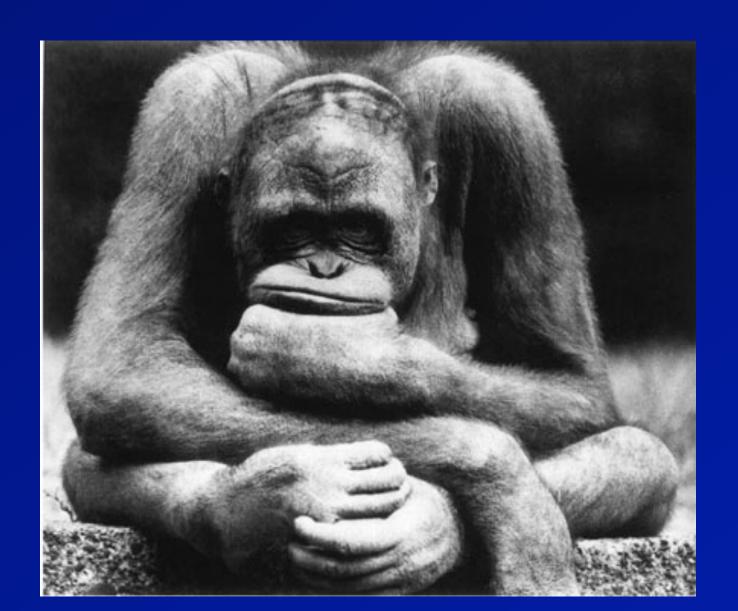
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Dept of Pathology

- A 60 year old man presented with vague fatigue
- Chest discomfort and
- Malaise
- Relieved with rest

- A substernal tightening chest pain
- Radiating to the left arm and shoulder
- Not relieved with rest
- Associated with nausea and vomiting

What is Ischaemia & Infarction??



Ischaemia

Deficient blood supply

Complete / partial

Arterial obstruction is more important than venous obstruction.

Ischaemia

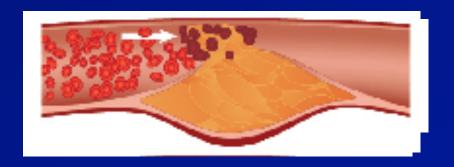
Causes - Thrombosis

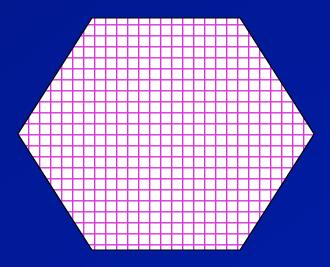
 Embolism
 Atheroma
 Proliferation of the intima

Arterial spasms

Ischaemia

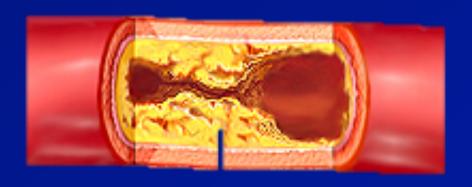
Progressive narrowing ---- ischaemic fibrosis





Infarction

Cessation of blood flow necrosis (infarction)





Susceptibility of tissues – blood supply

- Anatomy and physiology of the blood vessels
- Organs with a dual blood supply
- Liver portal circulation
- Lung pulmonary circulation

Collateral circulation

- Neck arteries supplying circle of Willis
- Femoral artery with collaterals in the legs
- Mesenteric vessels form overlapping arcades

If the narrowing is gradual collaterals may develop in some organs e.g.heart

End arteries

- Kidney
- Spleen
- Arteries beyond the circle of Willis

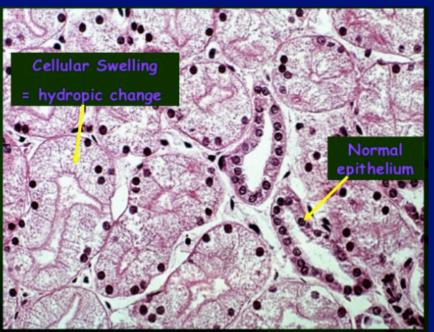
When arteries are narrowed a sudden drop in blood pressure causes reduction in perfusion which may lead to infarction.

Tissue metabolism & Ischaemia

- Parenchymal cells are more susceptible than supporting tissue.
- Tissue depending on exogenous fuel
 - with a high metabolic rate
 - depending on aerobic metabolism
- are susceptible to ischaemia

Effects of ischaemia on cell metabolism

Reduced O₂
Anaerobic respiration
Reduced ATP generation
ATPase cation pump affected



Cell swelling
Enzyme activity increases
Free radical injury
Necrosis
Membrane injury

Reperfusion injury

- Restoration of blood supply may cause a paradoxical increase in cell injury due to free radicals.
- However restoration of blood supply reduce the mortality rate

Infarction

- Tissue necrosis due to reduction / loss of blood supply.
- Usually due to obstruction of arteries by thrombosis or embolism.
- Occasionally due to obstruction of a draining vein. (venous infarct)

Factors that influence the development of an infarct

- 1. Nature of the vascular supply
- 2. Rate of the development of the occlusion
- 3. Vulnerability of a given tissue to hypoxia
- 4. O₂ content of the blood

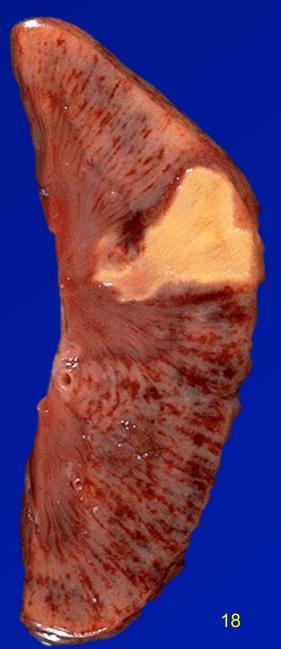
General features

- Pale / red
- sterile / septic
- Undergo coagulative / colliquative necrosis.
- Size depends on -
- Amount of tissue affected
- Severity and
- Duration
- Susceptibility of the cells to hypoxic injury

Macroscopy

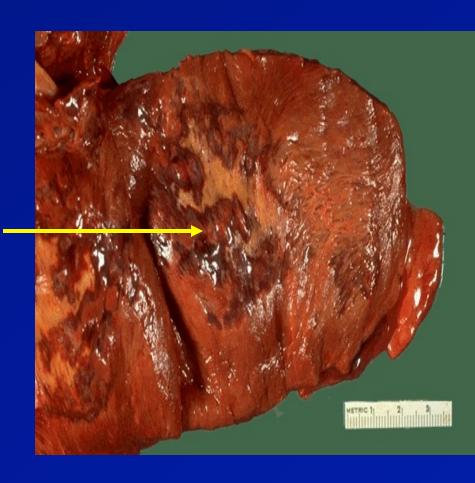
 No histological changes during the first few hours

Wedge shaped in many organs
 Base in the periphery



Macroscopy

- Overlying fibrinous exudate.
- Initially poorly defined
- Slightly haemorragic
- Narrow rim of hyperaemia at the margin
- Pale / red
- Later fibrous scar



Microscopy

- initially cells are swollen
- margin acute inflammatory reaction vascular congesion oedema
- Polymorphs and macrophages enter the dead tissue
- granulation tissue grows in
- later fibrous scar forms

Pale infarct

- In solid organs (Heart, spleen, kidney)
- Tissue limits the amount of haemorrhge that can seep in.



Myocardial infarction

- Usually from occlusive thrombosis of an existing atheromatous narrowing
- Coagulative necrosis replaced by scar tissue
- Less commonly subendocardial infarction

Red infarct

- a)venous infarct
- b) loose tissue (lung)
- c)tissue with dual circulation (lung, SI)
- d)congested tissue
- e)when blood flow is re established following arterial occlusion

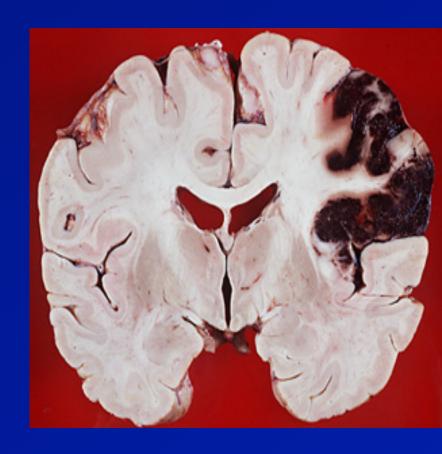
Lung infarction

- red infarction
- conical shape



Cerebral infarction

- Pale / haemorrhagic
- colliquative necrosis
- debris removed by macrophages
- residual cavity remains



Splenic / kidney infarct

- pale infarct
- conical shape



Intestinal infarction

- occlusion of a major artery
- occlusion of mesenteric vein



Venous infarction

Ovary and testes

- Acute renal vein thrombosis kidney infarct
- Marantic thrombosis of the superior longitudinal sinus in severely debilitated children
- Venous infarction of the gut

Septic infarction

Infarct caused by septic emboli



