Ischemia & Infarction

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ISCHAEMIA

- Def -deficient supply of blood flow
- maybe partial due to abnormal low flow of blood or complete when blood flow ceases
- arterial obstruction a more an important cause of ischaemia thanVenous blockage
- arterial obst. maybe due to: thrombosis,embolism leading to complete ischaemia or atheroma, proliferation of intima in small arteries and arterial spasm leading

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- Partial ischaemia.
- Progressive obstruction of artery leading to partial ischaemia results in ischaemic atrophy of parenchyma with overgrowth of fibrous tissue

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- cessation of blood flow leads to Necrosis of affected part or the whole organ = infarction
- E.g. chronic myocardial ischaemia results in fibrosis and angina pectoris

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- complete ischaemia leads to MI, which can lead to sudden death
- susceptibility of tissue to ischaemia depends on:
- I. Anatomical/physiological properties of the vessels
- II. Metabolism of tissue

- Blood supply and collateral circulation eg lungs and liver make them resistant to ischaemia
- brain arterial anastomosis at the circle of Willis allow leeway if carotids are blocked

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- The kidneys and the spleen have end arteries that do not anastomose any sudden occlusion leads to infarction
- in vessels narrowed by atheroma, sudden drop in blood pressure can

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- Lead to infarction eg watershed infarction in brain and global infarct in the heart
- tissue metab and ischaemia depends on
- a. type of cell

- Parenchymal cells of tissue with high metab rate are susceptible to acute and chronic ischaemia
- support tissue such as fibrous connective ts, fatty ts, bone and cartilage are less susceptible

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- b.use exogenous fuel as opposed to endogenous fuel
- c. ability to have anaerobic metabolism
- brain and heart have high metabolic rates and rely on

- Exogenous fuel with limited endogenous stores of fuel as such they have poor tolerance to hypoxia and ischaemia
- in normal metabolism of fats oxygen is required

- In ischaemia triglycerides are not oxidized and glycogen is only broken down to pyruvate and eventually to lactate' this results in release of enzymes that can be assayed to confirm tissue necrosis E.g. heart, liver and skeletal muscle

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- restoration of blood flow results in re-perfusion injury due to increased formation of free radicals
- treatment with streptokinase and calcium channels blockers reduce formation of free radicals in re-perfusion

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INFARCTION

- Def- tissue necrosis due to reduction of blood supply
- usually due to obst. Of arteries by thrombosis or embolism
- occasionally venous obstruction may result in venous infarcts

Obst: obstruction

General features of infarcts

- Size depends on amount of tissue rendered ischaemic, severity and duration of ischaemia
- appearance maybe red or pale, may undergo coagulative or colliquative necrosis

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- hypoxic cells unable to maintain ionic gradient and hence absorb water become swollen
- recent infarcts are raised above the surface-in the brain causes increased ICP

Living tissue around infarcted area becomes inflamed

- Diffusion of material from cell cause acute inflammatory reaction at the margins with vascular congestion, edema, migration of macrophages and polymorphs
- Inflammation is followed by healing that starts with granulation ts growing from the periphery that is finally replaced by fibrous tissue to give a sunken scar

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Infarctions- Types

Red (Hemorrhagic)	White (Anemic)
Venous occlusion	Arterial occlusion
In loose tissues (lung)	Solid organs
Organs with dual blood supply (Lungs, Liver)	End arterial supply (Kidney, Spleen, Retina, Heart)
Previously congested organs (nutmeg liver)	
Re- perfused tissues	

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MYOCARDIAL INFARCTION

- Arises due to occlusive thrombosis supervening on atheroma of major coronary artery
- it undergoes coagulative necrosis giving a pale infarct
- with time a fibrous scar ts forms
- Inner region of the heart may undergo necrosis leading to subendocardial infarction

Low BP- leads to global subendocardial infarction

- Global MI is circumferential and occurs in sudden drop of BP in a patient with severe atheroma of the coronary arteries
- **CEREBRAL INFARCTS** may be pale or haemorrhagic, due to high brain water content has colliquative necrosis

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- Removal of infarct is by marcrophages (gitter cells) and results in residual cavity referred to as apopletic cyst. Eventually contains clear fluid and is walled off by gliosis

Gitter cell: an enlarged phagocytic **cell** of microglial origin

Lung infarcts

- Typically dark red
- Conical shape (wedge)
- firm and haemorhagic because bronchial artery bleed into the dead tissue

Splenic infarct

- Conical and subcapsular
- initial red due to congestion but change to pale yellow before it organizes
- seen in splenomegaly and in sickle cell may result in autosplenectomy

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- Similar infarcts are seen in kidneys and result in release of renin resulting in hypertension
- **venous infarct**
- acute renal vein thrombosis results in renal infarction

Renal tumor can grow along the vein causing blockage-infarct

- Marantic thrombosis in severely debilitated children lead to haemorrhagic infarction of cerebral cortex
- **septic infarction**
- in infarcts caused by septic emboli Bacteria invade necrotic tissue leading to suppuration and abscess formation