Jiwaji University, Gwalior
SOS MBA (CSMM)

TOPIC –INFARCTION (405 B)

LECTURE BY–
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INFARCT

An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue.

Causes

Thrombosis or embolism

Atherosclerosis

Occlusion

Venous outflow obstruction (single outflow organs)

Others : Hypotensive,local vasospasm, compression of, vessel by hematoma or tumor, torsion

INFARCTION

- Tissue necrosis due to ischaemia
- vascular insufficiency of any cause
- usually arterial occlusion due to thrombosis/embolism
- Mainly due to oxygen deficiency, but toxin accumulation & reperfusion injury may contribute
- Number of determining factors
- Size of vessel and size of vascular territory
- Partial / total vascular occlusion
- Duration of ischaemia

INFARCT DEVELOPMENT

- Dependent on a number of factors
- Nature of vascular supply
- Dual supply e.g. lungs, liver
- End arteries e.g. kidneys, spleen
- Rate of vascular occlusion
- Time for development of collateral circulation
- Vulnerability to hypoxia
- Neurons – 2-3mins, Myocardium – 20-30mins, Fibroblasts – hours.
- Oxygen content of blood
- Anaemia, cyanosis, congestive heart failure
- Can result in infarction due to otherwise inconsequential blockage
- Size of vessel and size of vascular territory
- Partial / total vascular occlusion
- Duration of ischaemia

MORPHOLOGY OF INFARCTS

on basis of Colour-

a.Pale /anemic /white- absence of RBCs

b.Red (hemorrhagic) Infarct –presence of RBCs

On basis of presence of infection
a.Septic –presence of infection b.bland-absence of infection

TYPES OF INFARCT

Red (haemorrhagic) infarcts

Venous occlusion/congestion e.g. torsion

Loose tissues where haemorrhage can occur and blood can collect in infarcted zone e.g. lung

Tissues with dual blood supply e.g. lung small intestine (permitting blood flow from unobstructed

vessel into infarcted zone – note flow is insufficient to rescue ischaemia)

Tissues that were previously congested due to sluggish venous Outflow

When flow is re-established e.g. fragmentation of an

occlusive embolus, angioplasty

PALE / WHITE INFARCT

- Ischemia following obstruction of nutrient artery or hypoperfusion of tissue

- Solid organs with end-arterial circulation such as kidney, heart, spleen

- Wedge shaped.occluded vessel at the apex,base at the serosal surface

- Better defined with time, paler, hyperemic margins

MICROSCOPY

- Ischemic coagulative necrosis
- Demonstrable only >12-18 hrs.
- Inflammation in response to necrosis
- Phagocytosis of cellular debris by neutrophils & macrophages 1-2 days
- Healing response
- Scar tissue (brain- liquefactive necrosis)

Red (hemorrhagic)infarcts

Sites :venous occlusion of organ with single venous outflow e.g. testicular torsion

Loose tissues- e.g. lung

Tissues with dual circulations: lung and gut

Previously congested tissue

With reperfusion of previously infarcted tissue

PULMONARY INFARCTS

- Ischemic necrosis of lung parenchyma following pulmonary embolism & lack of blood from bronchial arteries.
- When blood from bronchial arteries reperfuses the ischemic area, blood leaks into the alveolar spaces
- Appears triangular, red & airless.
- Becomes more firm &brown with time

SEPTIC INFARCT

- Following fragmentation of a bacterial vegetation from a heart valve or following microbes seeding a necrotic area.
- Converted into an abscess
- Greater inflammatory response
- scarring

EVENT SEQUENCE

1. Coagulative necrosis
2. Infiltration by neutrophils
3. Infiltration by macrophages
4. Phagocytosis of debris
5. Granulation tissue formation
6. Scar formation

Time	Microscopic Features	Gross Features
0 – 4 hr	None	None
4 – 12 hr	Early coagulation necrosis (nucleus: pyknosis, cytoplasm: eosinophilia)	None
12- 24 hr	Further necrosis, haemorrhage, early neutrophil infiltrate	Dark mottling
1 – 3 days	Marked neutrophil infiltrate and necrosis	Mottled with yellow-tan necrotic centre
3 – 7 days	Early phagocytosis of dead cells by macrophages (at border)	Hyperaemic border, central yellow-tan softening
7 – 10 days	Well-developed phagocytosis, early granulation tissue formation	Maximal yellow-tan softening, depressed red-tan margins.
10 – 14 days	Well-developed granulation tissue, early collagen deposition	Red-gray depressed infarct borders
2 – 8 wk	Increased collagen deposition, decreased cellularity	Grey-white scar progresses from border toward centre
> 2 months	Acellular collagenous scar	Dense gray scar

Thank You