Hemodynamic Disorders

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LEC 4

Infarction

- **Area of ischemic necrosis caused by occlusion of either arterial supply or venous drainage in particular tissue.**
- **90 % results from thrombotic or embolic events & almost result from arterial occlusion.**
- **Other mechanisms:**

1- Local vasospasm
2- Enlargement of an atheroma
3- Secondary to hemorrhage within atherosclerotic plaque
4- Extrinsic compression of vessels e.g by tumor.

Types of infarction:

1-red (hemorrhagic)
2- White (anemic)

Red Infarct: occur with:

1- Venous occlusion like ovarian torsion.
2- Loose tissues e.g. lung that allow blood to collect in infarcted zone.
3- Tissues with dual circulation e.g. lung & small intestines.
4- Tissues that previously congested because of sluggish venous outflow.
5- When flow re-established to a site of previous arterial occlusion & necrosis (fragmentation of occlusive embolus).

White Infarct: occur with:

1- Arterial occlusion.
2- Solid organs (heart, spleen, kidneys) where solidity of tissues limits the amount of hemorrhage into ischemic necrosis.
The few of extravasated RBCs lysed & hemoglobin released which remain in form of hemosiderin, while in spongy organs, the hemorrhage is extensive.

Histologically:

- **The infracted area shows ischemic coagulative necrosis.**
- **Inflammatory response begin within few hours along margin & becomes well defined in 1-2 days caused by necrotic tissues then gradual degradation of dead tissues with phagocytosis by inflammatory cells.**
- **Reparative response begin in margin & most infarction replaced by scar tissues.**

Shock:

It is a state in which the supply of blood to the tissues is inadequate to meet the metabolic demands (either real loss or relative decrease in blood volume).

Classification:

1- **Hypovolemic shock**: In which there is a real decrease in blood volume.

Causes:

* Hemorrhage.
* Fluid loss as in severe vomiting, diarrhea & burns.

Mechanism of development: is inadequate blood or plasma volume.

2- **Cardiogenic shock:** There is relative decrease in blood volume (pooling of blood).

Causes:

- Myocardial infarction.
- Rupture of the heart.
- Pulmonary embolism.
- Arrhythmias.
- Cardiac temponade.

Mechanism of development:

Failure of myocardial pump due to intrinsic myocardial damage or extrinsic pressure or obstruction to outflow.

3- **Septic shock:** Causes:

Overwhelming bacterial infection (gram negative or positive septicemia or endotoxic shock) .

Mechanism of development:

* Peripheral vasodilatation &pooling of blood.
* Cell membrane injury.
* Endothelial cell injury with DIC (disseminated intravascular coagulopathy)

4- **Neurogenic shock:** Causes:

Anesthesia & spinal cord injury.

Mechanism: peripheral vasodilatation.

Stages of shock:

1- **Non progressive phase:** which is the compensatory phase .In this stage a compensatory mechanisms operate to maintain cardiac output &blood pressure near normal levels .The compensatory mechanisms include:

a- Arteriolar constriction leading to increase blood pressure.

b- Increase heart rate &cardiac output.

c- Retention of fluid through increase secretion of ADH & activation of rennin angiotensin aldosterone axis to retain fluid.

2- **Progressive phase**: When an additional factor is added like extensive burn complicated by bacterial infection .In this stage, despite the compensatory mechanisms, there is progressive decline in blood pressure &cardiac output.
Clinically observed increase in respiratory rate &decrease in urine output reflecting pulmonary &renal hypoperfusion.

3. **Irreversible Phase:** result from irreversible injury to the cell membrane as manifested by paralysis of sodium-potassium pump & defect in cell membrane so cell contents go to outside .The reduction in blood flow to the vital organs such as brain, heart, kidney lead to ischemic cell death in these organs.

Pathological Changes:

- Brain: Ischemic encephalopathy.
- Heart: coagulation necrosis.
- Kidneys: Extensive tubular ischemic injury (acute tubular necrosis) which lead to oliguria or anuria &electrolytes disturbances.
- Lungs: diffuse alveolar damage.
- GIT: patchy mucosal hemorrhage &necrosis.
- Liver: fatty change.

Clinical course:

- In hypovolemic &cardiogenic shock: patient present with hypotension, weak rapid pulse, tachypnea, cool &cyanotic skin.

- In septic shock: the skin may initially be warm &flushed because of peripheral vasodilatation.

Prognosis:

- In hypovolemic shock: 80-90 of young patients survive.
- Cardiogenic shock associated with extensive myocardial infarction & in gram-negative shock, 75% died.