Learning objectives

1-Infarction
- a-Definition
- b-Causes
- c-Factors effect development
- d-Types (red and white)

2-Shock
- a-Definition
- b-Causes
- c-Types
- d-morphology

Infract

Definition: An infarct is an area of ischemic necrosis caused by occlusion of the vascular supply to the affected tissue.
Infarction primarily affecting the heart and the brain is a common and extremely important cause of clinical illness.

Causes:

1. Arterial thrombosis or arterial embolism underlies the vast majority of infarctions.
2. Less common causes of arterial obstruction include:
   - Vasospasm.
   - expansion of an atheroma secondary to intraplaque hemorrhage.
   - and extrinsic compression of a vessel, such as by tumor, a dissecting aortic aneurysm.
   - Other uncommon causes of tissue infarction include vessel twisting (e.g., in testicular torsion or bowel volvulus).

Factors That Influence Infarct Development

• ***Anatomy of the vascular supply.*** The presence or absence of an **alternative blood supply** is the most important factor in determining whether occlusion of an individual vessel causes damage.

- ❖ The dual supply of the **lung** by the pulmonary and bronchial arteries means that obstruction of the pulmonary arterioles does not cause lung infarction unless the bronchial circulation also is compromised.
- ❖ Similarly, the **liver**, which receives blood from the hepatic artery and the portal vein.
- ❖ the **hand and forearm**, with its parallel radial and ulnar arterial supply, are resistant to infarction.
- ❖ By contrast, the **kidney and the spleen both have end-arterial circulations,** and arterial obstruction generally leads to infarction in these tissues.

• ***Rate of occlusion.*** Slowly developing occlusions are less likely to cause infarction because they allow time for the development of **collateral** blood supplies. For example, small interarteriolar anastomoses, which normally carry minimal blood flow, interconnect the three major coronary arteries. If one coronary artery is slowly occluded flow in this collateral circulation may increase sufficiently to prevent infarction—even if the original artery becomes completely occluded.

• ***Tissue vulnerability to hypoxia.*** **Neurons** undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. **Myocardial cells**, although hardier than neurons, still die after only 20 to 30 minutes of ischemia. By contrast, **fibroblasts** within myocardium remain viable after many hours of ischemia.

Morphology of infarcts

Types of infarcts

Infarcts are classified depending on:

A) the basis of their color (reflecting the amount of hemorrhage) into:

1. Hemorrhagic (Red) infarcts
2. Anemic (White) infarcts

B) the presence or absence of microbial infection into:

1. Septic infarcts
2. Bland infarcts

1. Red infarcts occur in:

a)Venous occlusions as in ovarian torsion

b) Loose tissues such as the lung which allow blood to collect in infarct zone.

c) Tissues with dual circulations (e.g. the lung), permitting flow of blood from unobstructed vessel into necrotic zone.
d) In tissues that were previously congested because of sluggish outflow of blood.
e) When blood flow is reestablished to a site of previous arterial occlusion & necrosis.

2. White infarcts occur in:

a)Arterial occlusion in organs with a single arterial blood supply.
b) Solid organs such as the heart, spleen, & kidney, where the solidity of the tissue limits the amount of hemorrage that can percolate or seep in to the area of ischemic necrosis from the nearby capillaries.

Gross: All infarcts are wedge-shaped with the occluded vessel at the apex and the periphery of the organ forming the base of the wedge. The infarction will induce inflammation in the tissue surrounding the area of infarction. Following inflammation, some of the infarcts may show recovery, however, most are ultimately replaced with scars except in the brain.

Microscopy:

The dominant histologic feature of infarction is ischemic coagulative necrosis. The brain is an exception to this generalization, where liquifactive necrosis is common.

Fig. 4.17 Red and white infarcts. (A) Hemorrhagic, roughly wedge-shaped pulmonary infarct *(red infarct)*. (B) Sharply demarcated pale infarct in the spleen *(white infarct)*.

Shock

Definition: Shock is a state in which diminished cardiac output or reduced effective circulating blood volume impairs tissue perfusion and leads to cellular hypoxia.

Its causes fall into three general categories:

- ***Cardiogenic shock*** results from low cardiac output as a result of myocardial pump failure. It may be caused by myocardial damage (infarction), ventricular arrhythmias, extrinsic compression (cardiac tamponade) or outflow obstruction (e.g., pulmonaryembolism).
- ***Hypovolemic shock*** results from low cardiac output due to loss of blood or plasma volume (e.g., resulting from hemorrhage or fluid loss from severe burns).
- ***Septic shock*** *is triggered by microbial infections and is associated with severe systemic inflammatory response syndrome(SIRS).* In addition to microbes, SIRS may be triggered by a variety of insults, including burns, trauma, and/or pancreatitis. The common pathogenic mechanism is a massive outpouring of inflammatory mediators from innate and adaptive immune cells that produce arterial vasodilation, vascular leakage, and venous blood pooling. These cardiovascular abnormalities result in tissue hypoperfusion, cellular hypoxia, and metabolic derangements that lead to organ dysfunction and, if severe and persistent, organ failure and death.
- Less commonly, shock can result from a loss of vascular tone associated with anesthesia or secondary to a spinal cord injury **(neurogenic shock).**
- **Anaphylactic shock** results from systemic vasodilation and increased vascular permeability that is triggered by an immunoglobulin E–mediated hypersensitivity reaction.

Pathogenesis of septic shock:

It results from the spread & expansion of an initially localized infection like pneumonia into the blood stream.

Most causes of septic shock (~70%) are caused by endotoxin-producing gram-posative bacilli, hence the term endotoxic shock

Septic shock acting on:

- The heart – causing decreased myocardial contractility which results in low cardiac output.
- Blood vessel – causing systemic vasodilation
- The mediators also cause widespread endothelial injury & activation of the coagulation system resulting in DIC.
- Lung – causing alveolar capillary damage resulting in adult respiratory distress syndrome (ARDS).

Stages of shock

Uncorrected shock passes through 3 important stages:

1) An initial nonprogressive phase

during which reflex compensatory mechanisms are activated and vital organ perfusion is maintained.

2)Progressive stage (Established shock)

- characterized by tissue hypoperfusion and onset of worsening circulatory and metabolic derangement, including acidosis.

3)An irreversible stage

- in which cellular and tissue injury is so severe that even if the hemodynamic defects are corrected, survival is not possible.

Morphology of shock:

All organs are affected in severe shock. In shock, there is widespread tissue hypoperfusion involving various organs such as the heart, brain, & kidney. Leads to widespread hypoxic tissue necrosis.

The widespread tissue necrosis manifests as multiple organ dysfunction [MODS]. And lungs may show ARDS.