HEMODYNAMIC DISORDERS

EMBOLISM:

An embolus is a detached intravascular solid, liquid, or gaseous mass carried by the blood to a site distant from its point of origin.

Forms of emboli

1-thromboembolism. 99% of all emboli represent a dislodged thrombus

Rare forms of emboli include:

2-Fat Embolism: Microscopic fat globules can be found in the circulation after fractures of long bones (which contain fatty marrow). Although fat and marrow embolism occur in some 90% of individuals with severe skeletal injuries, fewer than 10% of such patients show any clinical findings.

3-Air Embolism: Gas bubbles within the circulation. Air may enter the circulation during as a consequence of chest wall injury. Air bubbles can coalesce to form frothy masses sufficiently large to occlude major vessels.

4. Atherosclerotic emboli (cholesterol emboli): consisting of atheromatous debris

5. Tumour emboli: made up of fragments of a tumour.

6. Bone marrow emboli: consisting of bits of bone marrow.

Emboli lodge in vessels too small to permit further passage, resulting in partial or complete vascular occlusion end in ischemic necrosis (infarction) of downstream tissue.

Pulmonary Thromboembolism

In more than 95% of cases, venous emboli originate from deep leg vein thrombi above the level of the knee such as the femoral, or iliac veins. These emboli are carried through

progressively larger channels and pass through the right side of the heart before entering the pulmonary arterial circulation

Systemic Thromboembolism: This refers to emboli in the arterial circulation, (80%) arise from Intracardiac mural thrombi

The major sites for arteriolar embolization are:

1. The lower extremities (75%).

2. The brain (10%).

3. The intestines (mesenteric), kidneys, and spleen.

4. The upper limbs are the least common sites.

INFARCTION:

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

Nearly 99% of all infarcts result from thrombotic or embolic events, and almost all result from arterial occlusion.

Morphology:

Infarcts are classified on the basis of their colour (reflecting the amount of haemorrhage) and the presence or absence of microbial infection. Therefore, infarcts may be either red (haemorrhagic) or white (anaemic) and may be either septic or nonseptic.

Red infarcts: Occur in these situations:

(1) With venous occlusions.

(2) In loose tissues (such as lung) that allow blood to collect in the

infarcted zone.

(3) In tissues with double circulations such as lung and small intestine,

permitting flow of blood from an unobstructed parallel supply into a

necrotic area (such perfusion not being sufficient to rescue the ischemic tissues).

(4) In tissues that were previously congested because of sluggish venous outflow.

(5) When flow is re-established to a site of previous arterial occlusion and

necrosis e.g., fragmentation of an occlusive embolus.

White infarcts:

Occur with arterial occlusions or in solid organs (such as heart, spleen, and kidney), where the solidity of the tissue limits the amount of haemorrhage that can seep into the area of ischemic necrosis from adjoining capillary beds.

Septic infarctions:

Occur when bacterial vegetations from a heart valve embolize or when microbes infect an area of necrotic tissue. In these cases, the infarct is converted into an abscess, with a correspondingly greater inflammatory response.

Histological appearance:

The dominant histologic characteristic of infarction is ischemic coagulative necrosis. In stable or labile tissues, parenchymal regeneration can occur at the periphery. However, most infarcts are ultimately replaced by scar. The brain is an exception to these generalizations; ischemic tissue injury in the central nervous system results in liquefactive necrosis.

Factors That Influence Development of an Infarct

Vascular occlusion can have no or minimal effect, or can cause death of a tissue or even the individual. The major determinants of the eventual outcome include:-

1- Nature of the Vascular Supply:

The presence of an alternative blood supply is the most important determinant of whether occlusion of a vessel will cause damage.

Lungs, liver, hand and forearm have a double artery blood supply; are all resistant to infarction. Thus, obstruction of small arterioles does not cause infarction in healthy individual with an intact bronchial circulation. Renal and splenic circulations have end-arterial blood supply so obstruction of such vessels generally causes infarction.

2- Rate of Development of Occlusion:

Slowly developing occlusions are less likely to cause infarction because they provide time for the development of alternative perfusion pathways.

3- Tissues susceptibility to Hypoxia:

The susceptibility of a tissue to hypoxia affects the occurrence of infarction. **Neurons** undergo irreversible damage when deprived of their blood supply for only 3 to 4 minutes. **Myocardial cells** are also quite sensitive and die after only 20 to 30 minutes of ischemia. In contrast, **fibroblasts** within myocardium remain viable after many hours of ischemia.

4- Oxygen Content of Blood:

Partial flow obstruction of a small vessel in an anaemic or cyanotic patient might lead to tissue infarction, whereas it would be without effect under conditions of normal oxygen tension.

SHOCK:

Shock is the final step for a number of potentially lethal clinical events including: Severe haemorrhage. Extensive trauma or burns. Large myocardial infarction. Large pulmonary embolism. Microbial sepsis.

Regardless of the underlying pathology, shock gives rise to systemic hypoperfusion caused either by reduced cardiac output or by reduced circulating blood volume. The end results are hypotension, impaired tissue perfusion, and cellular hypoxia resulting in the death of the patient.

Types of shock:

1- **Cardiogenic shock**: Results from failure of the cardiac pump. This may be caused by myocardial infarction, ventricular arrhythmias, extrinsic compression to the heart, or outflow obstruction (e.g., pulmonary embolism).

2- Hypovolemic shock: Results from loss of blood. This may be caused by hemorrhage, fluid loss from severe burns, or trauma.

3- Septic shock: Caused by microbial infection. Most commonly this occurs in the setting of gram-negative infections but it can also occur with gram-positive and fungal infections.

4- Neurogenic shock: Less common shock may occur in the setting of an anesthetic accident or a spinal cord injury as a result of loss of vascular tone and peripheral pooling of blood.

5- Anaphylactic shock: Represents systemic vasodilation and increased vascular permeability caused by an immunoglobulin E hypersensitivity reaction. In these situations, acute severe widespread vasodilation results in tissue hypoperfusion and cellular anoxia (Absence of oxygen).

Morphology:

Shock will induce cellular and tissue necrosis due to hypoxia or combination of decrease blood supply and fibrin thrombi which may be identified in any tissue, mostly visualized in kidney glomeruli.

Clinical manifestations:

In hypovolemic and cardiogenic shock, the patient presents with hypotension; a weak, rapid pulse; tachypnea; and cool, clammy, cyanotic skin.

In septic shock, the skin may be warm and flushed as a result of peripheral vasodilation. The prognosis varies with the type of shock, its duration, age and general health of the patients.

Thus, 80% to 90% of young, healthy patients with hypovolemic shock survive with appropriate management, whereas cardiogenic shock associated with extensive myocardial infarction, or gram-negative sepsis carries a mortality rate of 75%, even with appropriate treatment.