

SHOCK

→ Systemic hypotension

→ caused by diminished cardiac output / reduced effective circulating blood volume.

→ Consequences → Impaired tissue perfusion and cellular hypoxia.

Three pathogenic categories.

- cardiogenic shock.

- hypovolemic shock.

- Septic shock.

- Neurogenic and anaphylactic shock.

OR

Cardiogenic, hypovolemic, obstructive, distributive shock.

Cardiogenic shock

low cardiac output due to myocardial pump failure.

→ Myocardial damage (infarction)

→ Ventricular arrhythmias.

→ extrinsic compression (cardiac tamponade)

→ outflow obstruction (eg: pulmonary embolism).

Hypovolemic shock

→ low cardiac output due to loss of blood or plasma volume.

(eg: hemorrhage or fluid loss from severe burns, vomiting)

→ hypovolemia - low cardiac

output → hypotension → inadequate tissue perfusion.

Septic shock

→ severe sepsis with hypotension.

→ systemic immune response to microbial infection.

→ Bacterial and fungal.

Factors contributing to the pathophysiology of septic shock

1. Inflammatory responses
2. Endothelial cell activation and injury.
3. Metabolic abnormalities.
4. Immune suppression.
5. Organ dysfunction.

① Inflammatory responses.

A) Engagement of receptors:

Microbial components engage receptors on cells of innate immune system.

↓
production of proinflammatory mediators: TNF, IL-1, IL-12, IL-18, HMGB1, reactive oxygen species and lipid mediators (prostaglandins & PAF)

↓
→ activate endothelial cells → expression of adhesion molecules.

(1) → secondary wave of cytokine storm

B. complement - cascade activated
 → Anaphylatoxins (C3a, C5a),
 Chemotactic fragments (C5a)
 and opsonins (C3b)
 → pro-inflammatory state.

C. Activation of coagulation
 FXII / Endothelial function
 alteration.

(2) Endothelial cell activation
 and injury.

1. Thrombosis.
2. Increased vascular permeability
 - edema
3. vasodilation
4. vasoactive inflammatory
 mediators: systemic hypotension.
 (which causes relax smooth muscle)

(3) Metabolic abnormalities.
 → insulin resistance and hyper-
 glycaemia (cytokines & catecholamines)
 → hyperglycaemia suppresses neutrophil
 function - thereby decreasing
 bactericidal activity
 → adrenal insufficiency - glucocorti-
 coid deficit
 → Waterhouse-Friderichsen
syndrome (necrosis of adrenal gland)
 → lactic acidosis.

(4) Immune suppression
 → Anti-inflammatory mediators
 (IL-10)
 → widespread apoptosis of
 lymphocytes

(5) Organ Dysfunction
 → decreased oxygen and
 nutrient supply to tissues
 → multi organ failure.

Stages of shock

(1) Non progressive phase:

- Neurohumoral mechanisms -
 cardiac output & BP.
- Tachycardia, vasoconstriction
- Renal conservation of fluid.

(2) Progressive stage.

- tissue hypoxia
- hypoperfusion
- lactic acidosis
- peripheral pooling of blood.

(3) Irreversible stage

- severe cellular & tissue injury
- Vasodilation, increased vascular
 permeability.
- Leakage of lysosomal enzymes.
- Abnormal myocardial contractile
 function
- Anoxic organ damage.

Morphology

- Adrenal: cortical cell lipid depletion, haemorrhagic necrosis.
- GIT: mucosal and mural infarction.
Diffuse haemorrhage & necrosis.
- Lung: diffuse alveolar damage
- Kidney: acute tubular necrosis.

Clinical features

- blue or grey fingernails / lips.
- shallow & rapid breathing
- distress
- fast pulse.

Ischaemia → ↓ blood flow.

Shock → ↓ BF → circulatory failure. → cellular damage.