# SHOCK

EMAN KREISHAN, M.D.

15-12-2021.

# SHOCK

- Latency associated nuclear antigen

# Three Major Types of Shock

- 1. 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).
- outflow obstruction (e.g., pulmonary embolism).

cardiac tamponade: fluid or blood builds up between the heart and the  pericardium.

- 2. 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).

- 3. Neurogenic shock:
- result from a loss of vascular tone.

- 4. Anaphylactic shock:
- results from systemic vasodilation and increased vascular permeability that is triggered by an immunoglobulin E–mediated hypersensitivity reaction.

# 5. Septic shock

- life-threatening organ dysfunction due to dysregulated host response to infection, and organ dysfunction.

- Caused by massive outpouring of inflammatory mediators from innate and adaptive immune cells that produce:

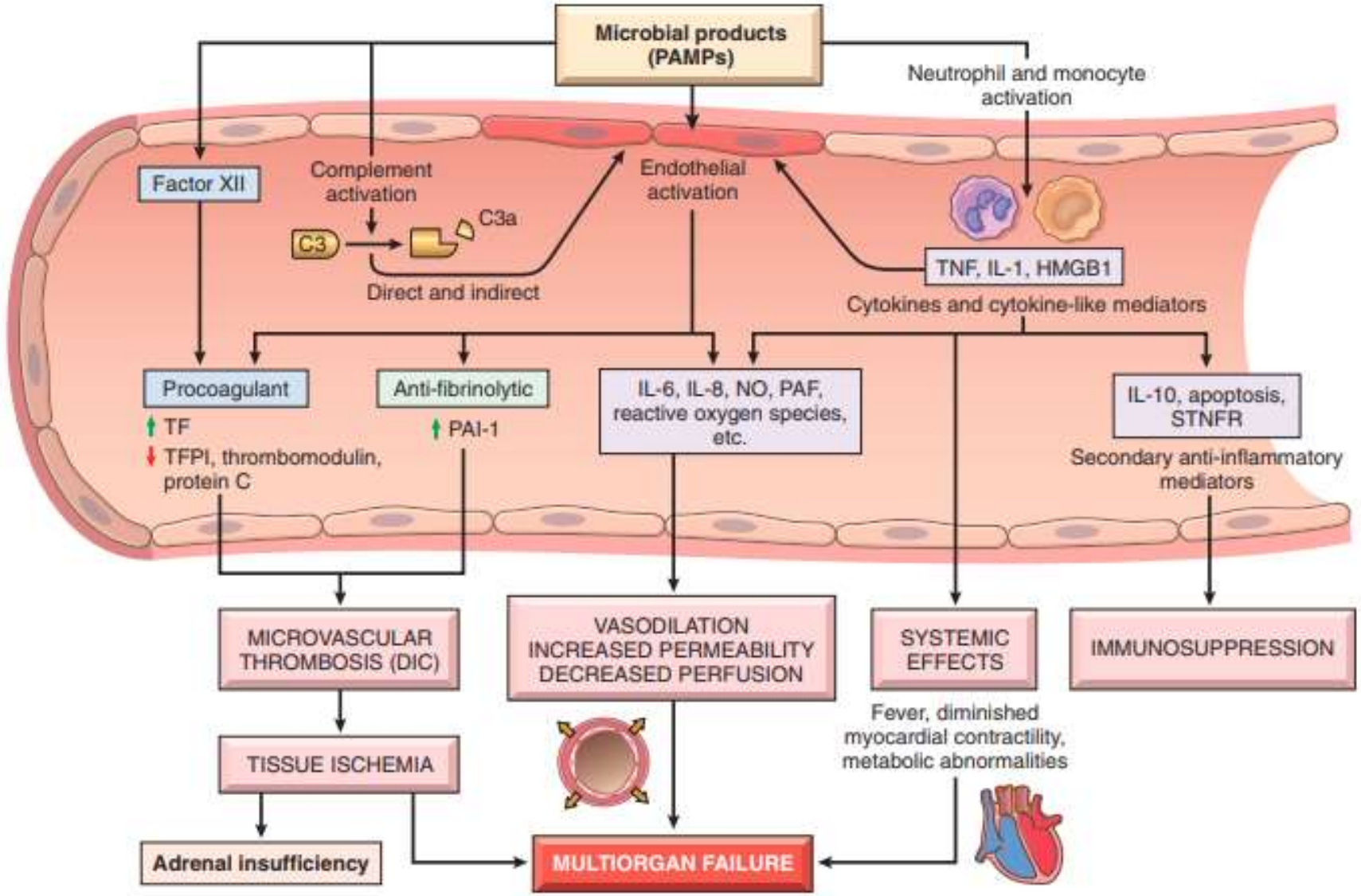
- arterial vasodilation.
- vascular leakage.
- venous blood pooling.

LEAD TO

- tissue hypoperfusion
- cellular hypoxia
- metabolic derangements

**Table 4.3 Three Major Types of Shock**

| Type of Shock | Clinical Examples | Principal Pathogenic Mechanisms |
|---|---|---|
| Cardiogenic | Myocardial infarction<br>Ventricular rupture<br>Arrhythmia<br>Cardiac tamponade<br>Pulmonary embolism | Failure of myocardial pump resulting from intrinsic myocardial damage, extrinsic pressure, or obstruction to outflow |
| Hypovolemic | Hemorrhage<br>Fluid loss (e.g., vomiting, diarrhea, burns, trauma) | Inadequate blood or plasma volume |
| Septic | Overwhelming microbial infections<br>Gram-negative sepsis<br>Gram-positive septicemia<br>Fungal sepsis<br>Superantigens (e.g., toxic shock syndrome) | Peripheral vasodilation and pooling of blood; endothelial activation/injury; leukocyte-induced damage; disseminated intravascular coagulation; activation of cytokine cascades |


Microbial products (PAMPs)
Neutrophil and monocyte activation
Factor XII
Complement activation
C3
C3a
Direct and indirect
Endothelial activation
TNF, IL-1, HMGB1
Cytokines and cytokine-like mediators
Procoagulant
TF
TFPI, thrombomodulin, protein C
Anti-fibrinolytic
PAI-1
IL-6, IL-8, NO, PAF, reactive oxygen species, etc.
IL-10, apoptosis, STNFR
Secondary anti-inflammatory mediators
MICROVASCULAR THROMBOSIS (DIC)
VASODILATION INCREASED PERMEABILITY DECREASED PERFUSION
SYSTEMIC EFFECTS
IMMUNOSUPPRESSION
Fever, diminished myocardial contractility, metabolic abnormalities
TISSUE ISCHEMIA
Adrenal insufficiency
MULTIORGAN FAILURE

# Pathogenesis of Septic Shock

- 1. Inflammatory and counterinflammatory responses:
- microbial cell wall constituents engage receptors , e.g: (TLRs).
- innate immune cells produce numerous cytokines, including TNF, IL-1, IFN-γ.
- the complement cascade is also activated by microbial components resulting in the production of anaphylotoxins (C3a, C5a).
- microbial components can activate coagulation directly through factor XII and indirectly through altered endothelial function.

# The hyperinflammatory state, triggers counterregulatory immunosuppressive mechanisms

- As a result, septic patients may range between hyperinflammatory and immunosuppressed states during their clinical course.

- mechanisms for the immune suppression include:

- shift from proinflammatory (TH1) to anti-inflammatory (TH2).
- production of anti-inflammatory mediators (e.g., soluble TNF receptor, IL-1 receptor antagonist, and IL-10).
- lymphocyte apoptosis

- 2. • Endothelial activation and injury:

- Cytokines loosen endothelial cell tight junctions, making vessels leaky and resulting in the accumulation of protein-rich edema fluid throughout the body.

- Activated endothelium also upregulates production of nitric oxide (NO) ,C3a, C5a, and PAF, which may contribute to vascular smooth muscle relaxation and systemic hypotension

- 3. Induction of a procoagulant state:

- 4. Metabolic abnormalities:

- Septic patients exhibit insulin resistance and hyperglycemia.
- TNF and IL-1, glucagon, growth hormone, glucocorticoids, and catecholamines all drive gluconeogenesis.*

- 5.Organ dysfunction:
- Systemic hypotension, interstitial edema, and small vessel thrombosis all decrease the delivery of oxygen and nutrients to the tissues.

# Stages of Shock

- 1.An initial nonprogressive stage:
- during which reflex compensatory mechanisms are activated and vital organ perfusion is maintained.

- 2. A progressive stage :
- 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.

- The cellular and tissue effects of shock are:

- those of hypoxic injury and are caused by a combination of hypoperfusion and microvascular thrombosis.

# Clinical Features

- The primary threat to life is the underlying initiating event.
- Hypotension.
- a weak rapid pulse.
- tachypnea,

- If patients survive the initial period, worsening renal function can provoke a phase dominated by :
- progressive oliguria.
- Acidosis.
- electrolyte imbalances