CIRCULATORY DISTURBANCES

Dr Eman MS Muhammad

HAEMORRHAGE

- **Definition:**
- Escape of blood outside the blood vessels or cardiac chambers.

Causes of hemorrhage:

A. ***Traumatic hemorrhage:***

- Caused by mechanical injury to the vascular wall either accidental or surgical.

B. ***Spontaneous hemorrhage:*** due to:

(a) Diseases of the vascular wall e.g. atheroma and aneurysm.

(b) Inflammatory injury to the vascular wall as in phlebitis.

(c) Destruction of the vascular wall by tuberculosis, malignancy or peptic ulceration.

(d) Increased intravascular tension e.g. chronic venous congestion and hypertension.

(e) Hemorrhagic blood diseases as hemophilia and purpura.

(f) Vitamin C and K deficiency.

Types of hemorrhage:

I. **External hemorrhage:**

- Escape of blood outside the body.

(a) *Epistaxis:*

- Bleeding from the nose.

(b) *Hemoptysis:*

- Coughing of blood.
- The source of blood is the lung or the bronchi.
- The blood is red, frothy and alkaline.

(c) ***Hematemesis:***

- Vomiting of blood.
- The source of blood esophagus, stomach and duodenum.
- The blood is digested, brown in color, acidic and mixed with food particles.

(d) ***Melena:***

- Presence of dark digested blood in the stools.
- The blood originates from the esophagus, stomach and duodenum.

(e) ***Hematuria:***

- Blood in urine.
- The blood originates from the kidney, urinary passages and bladder.

(f) ***Menorrhagia:***

- Excessive or prolonged menstrual bleeding.

(g) ***Metrorrhagia:***

- Irregular uterine bleeding not related to menstruation.

(h) ***Hemorrhage from the skin.***

II. Internal hemorrhage:

- Escape of blood inside the body cavities (serous sacs).

a) ***Hemothorax:*** Hemorrhage into the pleura.

b) ***Hemopericardium:*** Hemorrhage into the pericardium.

c) ***Hemoperitoneum:*** Hemorrhage into the peritoneum.

d) ***Hematocele:*** Hemorrhage into the tunica vaginalis.

e) ***Hemoarthrosis:*** Hemorrhage into a joint cavity.

III. **Interstitial hemorrhage:**

- Escape of blood into the interstitial spaces, it may be:

(a) ***Petechial hemorrhage:***

- Small amount of blood of capillary origin.

(b) ***Ecchymosis:***

- Moderate amount of blood.

(c) ***Hematoma:***

- Large amount of blood causing a swelling.
- At first the site of interstitial hemorrhage is dark red (arterial blood) or bluish (venous blood).

- The extravasated red cells liberate hemoglobin which breaks down into biliverdin and hemosiderin.
- Biliverdin gives the area a green color but is soon absorbed in the blood.
- The hemosiderin left gives the area a brown color and is gradually removed by macrophages, so the color changes to yellow and gradually fades away.

Natural arrest of hemorrhage (hemostasis):

1. Fall in the blood pressure as a result of decrease in the blood volume decreases further blood loss.
2. Curling up of the endothelium and contraction of the muscular layer of the vessels.
3. Clotting of the blood within and around the vessels.
4. Serotonin released from the platelets at the site of clotting causes local vasoconstriction of the arterioles.
5. Permanent closure of the arterial wound is by organization of the formed clot, so the vessel is closed by fibrous tissue.

- In a septic wound the clot may undergo softening by the proteolytic enzymes liberated from the dead leucocytes, this results in secondary hemorrhage.

Effect of hemorrhage:

1. ***Small amount:*** No effect.
2. ***Small amount repeatedly (chronic hemorrhage):***

- Causes microcytic hypochromic anemia, e.g. in piles and peptic ulcers.

3. ***Moderate amount:*** (less than *750* cc.).

- This is compensated by:

a) Immediate fall in the blood pressure stimulates the aortic arch and carotid sinus reflexes which increase the heart rate.

b) Reflex vasoconstriction in the skin, muscles and splanchnic area to maintain the blood pressure.

- Also increased secretion of adrenaline elevates the blood pressure.

c) The decreased hydrostatic pressure inside the vessels allow the colloid osmotic pressure to withdraw tissue fluids into the blood.

d) Proteins are added from the liver and reticulo-endothelial system.

e) Red cells and white cells are added by the hyperplastic bone marrow.

- however a state of normocytic anemia persists for sometimes.

4. ***Massive amount:***

- This causes hemorrhagic shock.
- The venous return decreases, cardiac output decreases and blood pressure falls causing hemorrhagic shock and death.
- **Postmortem picture of hemorrhage:**
  The postmortem picture of shock, but the organs are pale anemic and dry.

SHOCK

- **Definition:**
- A widespread hypoperfusion of cells and tissues occurring due to inadequate effective circulating blood volume.

Clinical picture:

1. The patient is often restless and confused.
2. The skin is pale, cold and covered by sweat.
3. The pulse is rapid and weak, blood pressure is low, respiration is shallow and rapid.
4. The urine volume is decreased (oliguria).

Types of shock:

(1) Hypovolemic shock

(2) Cardiogenic shock

(3) Septic (endotoxic) shock

(4) Rare types are neurogenic and anaphylactic shock

HYPOVOLEMIC SHOCK

- **Causes:**
- Acute reduction of blood volume due to:

1. ***Severe hemorrhage:***

- Caused by trauma, surgical procedures, involvement of blood vessels in disease processes and hemorrhagic disorders.

2. **Severe burns:**

- Hypovolemia results from inflammatory exudation of plasma fluid from the damaged blood vessels in the vicinity of extensive burns.

3. ***Severe acute dehydration:***

- Due to severe vomiting and diarrhea in gastroenteritis, cholera ... etc.

Pathogenesis:

- Reduction of the effective circulating blood volume leads to decreased venous return to the heart, decreased cardiac output, reduced blood flow, reduced oxygen supply to the tissue (anoxia).
- If compensatory vasoconstriction does not correct the shock, tissue anoxia will cause more dilatation of the blood vessels and the shock becomes irreversible.

CARDIOGENIC SHOCK

- **Causes:**
- Marked reduction in the cardiac output due to:
  1. Coronary artery occlusion
  2. Rupture of a valve cusp
  3. Major arrhythmias
  4. Cardiac tamponade (hemopericardium).

SEPTIC (ENDOTOXIC) SHOCK

- **Causes:**
- Severe bacterial infections by gram negative organisms as E.coli (endotoxic shock) or gram positive bacteria as streptococci and meningococci.
- These bacterial infections include septicemia, infected burns, surgical procedures in infected genito-urinary, biliary and gastrointestinal tract lesions.
- Septic shock occurs also in patients with immunodeficiency states as leukemia and lymphomas and as a complication of cytotoxic drugs or immunosuppressive therapy.

Pathogenesis:

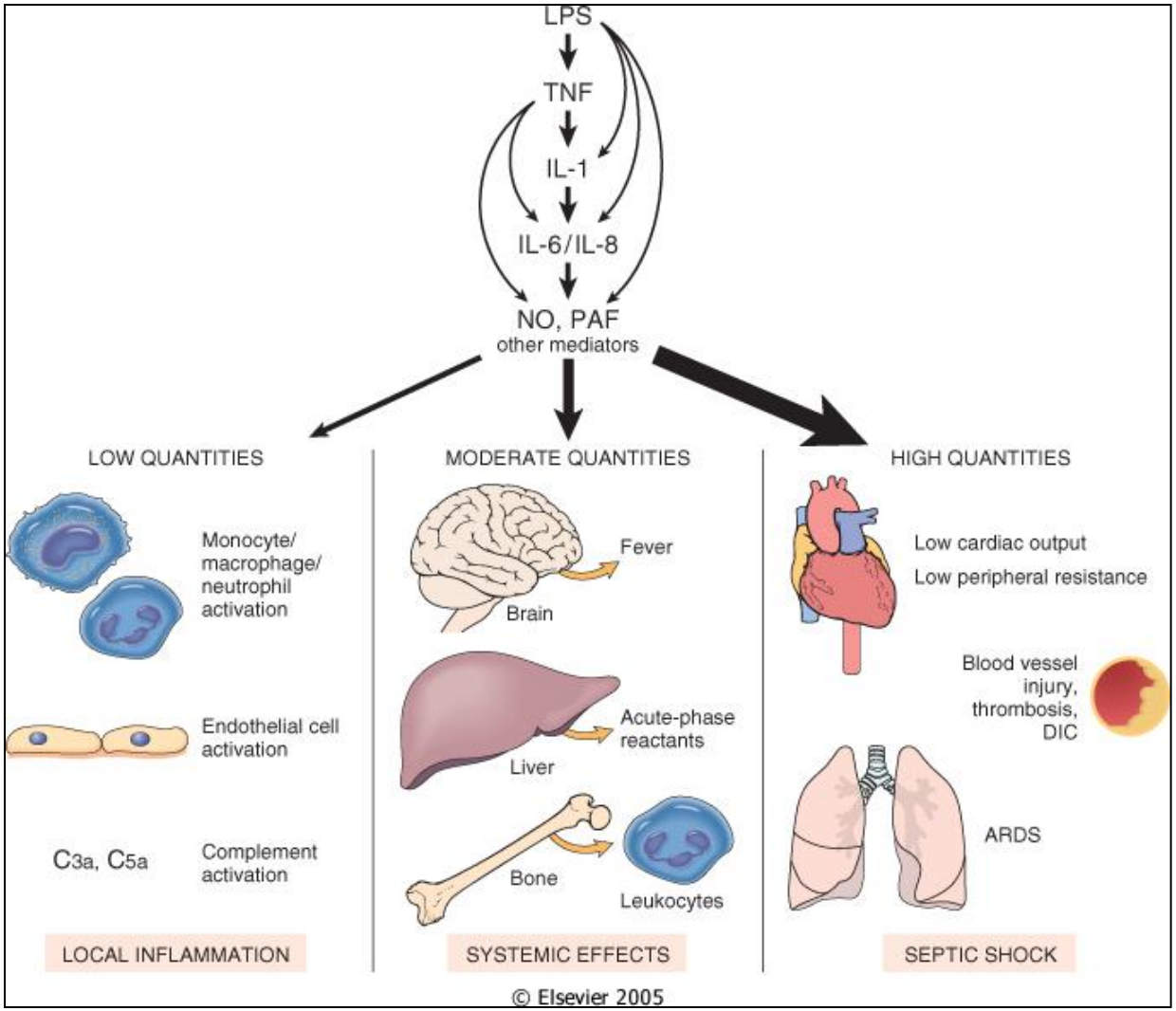
1. Dilatation of the capillaries and venules by the chemical mediators as histamine, kinins ... etc. results in pooling of the blood in the peripheral vessels and reduction in the effective circulating blood volume.
2. Toxic cell injury.
3. Endothelial cell injury with disseminated intravascular coagulation (DIC).

Postmortum picture of shock:

1. Congestion of the viscera due to vasodilatation.
2. Petechial hemorrhage easily detected in the serous membranes.
3. Edema of soft tissue and transudate in the serous cavities.
4. Absence of lipids from the adrenal cortex as they have been used in the formation of cortical hormones.
5. Degeneration and necrosis in the heart, liver and kidney.

- The kidney lesion may be extensive (acute tubular necrosis) causing oliguria and acute uremia.

Shock


LPS
TNF
IL-1
IL-6/IL-8
NO, PAF
other mediators
LOW QUANTITIES
MODERATE QUANTITIES
HIGH QUANTITIES
Monocyte/ macrophage/ neutrophil activation
Endothelial cell activation
C3a, C5a
Complement activation
Fever
Brain
Acute-phase reactants
Liver
Bone
Leukocytes
Low cardiac output
Low peripheral resistance
Blood vessel injury, thrombosis, DIC
ARDS
LOCAL INFLAMMATION
SYSTEMIC EFFECTS
SEPTIC SHOCK
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