15- Hemorrhage & Pathophysiology of Shock

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Hemorrhage and shock

I- Hemorrhage

Definition: haemorrhage is the loss of blood from the cardiovascular system.

Types:

- *According to site*: To outside the body (external), into the body cavity (internal) or into the tissue spaces (interstitial).
- *According to onset*: 1- acute (sudden loss of blood)

  2- chronic (small and gradual loss e.g. peptic ulcer or piles which causes severe anemia and hypoxia).

- **According to prognosis:**
  - **If the amount of blood loss is less than 20% of total blood volume it can be compensated and the BP may return to normal level spontaneously.**
  - **If more than 20% rapid blood transfusion must be occurred to avoid the refractory shock.**

The Compensatory Homeostatic Mechanism For Hemorrhage

I- Immediate Compensatory Mechanisms

***Onset*: Very rapid within minutes**

***Types*:**

(A) Nervous control

(B) Hormonal mechanisms

(C) Thirst

(D) Respiration

II- Delayed Compensatory Mechanisms

The Compensatory homeostatic mechanism

[I] Immediate compensatory mechanisms:

Very rapid within minutes ,aim to restore the BP & blood supply to the vital organs.

(A) Nervous control

Stimulation of the pressor area of cardiovascular center as a result of:

a- Decrease ABP and CVP → ↓ inhibitory impulses from arterial baroreceptors and atrial baroreceptors.

b- Hypoxia of the peripheral chemoreceptors.

Stimulation of the pressor area and inhibition of depressor area leads to:

1- ↑ Sympathetic discharge to arterioles → VC →↑ BP towards normal (VC is mainly in skin and splanchnic BV) and VC of renal vessels →↓ urine volume.

2- VC OF venules →↑ VR →↑ COP →↑ ABP.

3- ↑ sympathetic to heart →

- ↑ Heart rate - ↑ power of contraction - coronary dilatation

So, the pulse is rapid and weak due to ↓ pulse pressure (thready pulse).

4- Contraction of the spleen: but this effect is little in man.

Liver causes:

5- Increase rate of blood coagulability to stop bleeding

6- Hyperglycemia to maintain fuel of the brain.

(B) Hormonal secretion

1) Secretion of Catecholamine:

Hemorrhage → stimulate sympathetic supply to adrenal medulla →↑ adrenaline secretion →↑ HR, VC, coronary dilatation, ↑ fibrinogen, increase blood coagulation and raising blood glucose level.

2) Secretion of renin-angiotensin system:

Low COP → renal ischemia → renin secretion → increase in angiotensin II → VC, sodium and water retention→ ↑ ABP.

3) Secretion of antidiuretic hormone:

↓ Venous return → decrease the central venous pressure → afferent from the right atrium to hypothalamus to stimulate secretion of ADH → VC & retention of urine

4) Cortisol hormone secretion:

Stress from hemorrhage → stimulate the hypothalamus to secrete releasing factor for cortisol secretion from the suprarenal cortex →↑ cortisol hormone as anti-stress hormone (↑ glucose, stabilize cell membrane, prevent cell damage).

(C) Intense thirst sensation

Hemorrhage → ↑ thirst sensation and water intake due to:

1- Hypovolemia → ↓ discharge of volume receptors in right atrium with stimulation of hypothalamus.
2- angiotensin II stimulates the thirst center

(D) Acceleration of respiration

By hypoxia of chemoreceptors and ↓ the inhibitory effect of arterial & atrial baroreceptors on respiration→ hyperventilation →↑ VR & COP →↑ ABP also ↑ Respiration →↑ O2 supply.

N.B.:

Clinical picture after hemorrhage:

-progressive ↓ ABP, rapid weak pulse (thready pulse), ↑ respiration, cold pale sweaty skin (sympathetic VC), intense thirst and nervous irritability (brain ischemia) and in severe cases coma may occur.

(II) Delayed (long-term) compensatory mechanisms:

Effective after 30 minutes and takes longer time:

1- Restoration of plasma volume: (12-72 hours)

a- ↓ ABP → ↓capillary hydrostatic pressure → less filtration to tissue fluid, but the oncotic pressure of plasma proteins is less changed → passage of fluids towards capillaries →↑ plasma volume.

b- ↑ ADH secretion → retention of water.

c- ↓ plasma volume & angiotensin II → thirst sensation → drinking of H2O →↑ plasma volume.

2- Restoration of plasma proteins: (3-4 days)

a- Rapid addition of preformed proteins (labile proteins) from the liver (fibrinogen at first).

b- New synthesis by the liver from the tissue proteins .

c- There are extravascular stores of albumin may be in interstitial tissue itself which mobilize rapidly to general circulation.

3- Restoration of cellular elements:(4-8 weeks)

a- Rapid addition from the spleen (little in human).

b- Activation of bone marrow to form new RBCs under effect of erythropoietin released by hypoxia.

c- Correction of WBCs by leuco-poietin & platelet by thrombo-poietin.

II- Shock

Definition: Circulatory shock means inadequate tissue perfusion with blood due to decreased COP & ABP.

* Types and causes of shock

(I) Low-resistance shock: (primary shock) (Normo volemic shock)

- It is caused by severe VD (bl. volume is normal) - e.g.:

(1) Neurogenic shock

Sever emotions (Vago-vagal syncope)→ vaso & veno-dilatation of skeletal blood vessels & bradycardia → ↓ ABP and shock.

(2) Anaphylactic shock

- Due to exaggerated antigen-antibody reaction with release of histamine or kinin causing vasodilation with drop in blood pressure.

(3) Septic shock

- Severe infection → bacterial endotoxin →depress the vasomotor center with resulted VD of arterioles and capillaries →↑ capillary permeability → decrease blood pressure → shock.

(II) Hypovolemic shock (Secondary shock) (Cold shock)

- **It is Caused by loss of blood or plasma or extracellular fluid. e.g.**

(1) Post hemorrhagic shock with failure of compensatory mechanisms.

(2) Burn shock: loss of plasma & VD.

(3) Traumatic shock: Hemorrhage, pain, loss of plasma to tissue & toxic substance.

(4) Dehydration: Severe vomiting, diarrhea or sweating.

(III) Cardiogenic shock

As in infarction, heart failure or arrhythmia → ↓ COP → shock.

(IV) Obstructive shock

- Due to obstruction of the blood flow at the centers of circulation which hinders blood flow to tissue:

In the lung: as in cases of the pulmonary embolism, thrombosis, and tension pneumothorax with marked elevation of the intra-thoracic pressure.

In the heart : as in cardiac tamponade (massive pericardial effusion) with fibrosis which prevent cardiac filling and contraction.

***Prognosis of shock:**

Its severity depends largely on the degree and rate of blood pressure drop and it may be either:

(A) Reversible (compensated) shock:

The compensatory mechanisms (immediate and delayed) gradually restore the ABP up to normal level .

(B) Irreversible (Refractory) shock:

This occurs in severe causes of shock and the patient not be treated for about 3-5 hours → progressive decrease in cardiac output and ABP in a positive feed back mechanisms (↓ ABP → loss of correction mechanism→ more ↓ ABP).

***Mechanisms that lead to death in refractory shock**

(1) Cardiac depression:

- **Severe decrease in ABP →↓ Coronary blood flow → myocardial ischemia → ↓ cardiac contraction → ↓ COP → ↓ABP and so on → myocardial infarction.**
- **Cardiac depression by myocardial toxic factor or other bacterial toxins released during shock.**

(2) Cerebral depression:

- **Severe decrease in ABP → ↓ Cerebral blood flow → depression of Vasomotor center → no correction of decreased ABP → more decrease in ABP & so on → cerebral damage.**

(3) Dilatation of precapillary sphincter:

- After hemorrhage → reflex sympathetic spasm of precapillary sphincters and venules especially in splanchnic area, after that dilatation of precapillary sphincter occurs by metabolites or toxins but venules remaining constricted →
- ↓ VR→ more decrease in ABP →more spasm of venules→ more ↓ VR .
- ↑ Capillary filtration due to ↑ in capillary hydrostatic pressure → ↑ loss of plasma in tissue space → ↓ bl. volume → ↓ VR → ↓ COP → ischemia of the capillary wall → more filtration.

(4) Release of toxins by ischemic tissues:

- **Myocardial toxic factor:** Extreme pancreatic ischemia → trypsin enzyme is released from pancreas → degeneration of pancreatic tissue → release of myocardial toxic factor → direct depression of the heart contractility.
- **Endotoxin:** released from intestinal bacteria under ischemia → absorbed to blood → severe VD and cardiac depression → severe shock.
- **Free radicals:** ↓ COP → tissue hypoxia→ injury of vessels→ adherence of granulocytes to vessels→ free radicals which causes more damage of vessels and more adherence of granulocytes and more free radicals and so on.

(5)Thrombosis of small vessels:

Due to sluggish circulation with activation of clotting factors and platelet aggregation.This leads to more tissue ischaemia.

(6) Acidosis:

↓O2 supply → lactic acid accumulation also ↑ CO2 → H2CO3. This acidosis leads to tissue damage and activation of intracellular proteolytic enzymes with auto-destruction.

(7) Acute respiratory failure:

Due to damage of capillary endothelium and alveolar epithelium in the lung with release of cytokines & Damage of respiratory center.

(8) Acute renal failure due to:

- Severe renal vasoconstriction causes renal ischemia and tubular necrosis.
- Muscular tissue damage leading to accumulation of myoglobin which enhance the damage in the kidney tissue with decrease renal plasma flow and glomerular filtration rate and the renal functions are severely impaired with uremia and anuria.

* Treatment of shock:

1) Warming the body (in hypovolemic shock) and raising the lower limb by 30 cm →↑ VR.
2) O_2 therapy and glucose injection.
3) Keep open air way and guard against pneumonia
4) Low resistance shock is treated by:
   - Antihistamincs, sympathomimetics.
   - Glucocorticoids (↓ permeability) to avoid anaphylaxis and resist cell damage.
   - Sedatives for pain (morphine).

5) Hypovolemic shock is treated by restoration of fluids according to types (blood, plasma or saline injection).

6) Septic shock needs proper antibiotics.

7) Correct electrolyte & acid base imbalance and dialysis in renal failure.

8) Treatment of the cause.


THANK
YOU