Circulatory disorders

Thrombosis

Definition: formation of a compact mass composed of elements of circulating blood inside a vessel or heart cavity during life. This compact mass called thrombus.

Causes of thrombosis:

1- **Damage to vascular endothelium:** by trauma, pressure, ligation, canulation, inflammation or atheroma.

2- **Slowing of blood stream:** in normal blood stream, the blood cells occupy central part and plasma in peripheral part. In stasis, platelets cross plasmatic zone and come in contact to vascular endothelium. Stasis occurs in the following conditions:

- In heart failure especially in veins of the leg.
- In the auricles of the heart in association with valvular diseases.
- In aneurysmal sacs, varicose veins and in the portal vein secondary to liver cirrhosis.
- In tissues showing acute inflammation.

3- Disordered blood stream:

Distortion of the vascular lumen as in aneurysm, atheroma, varicose veins and compression of the vessel wall from outside. All these allow platelets to come in contact with the vascular endothelium and initiate thrombosis.

4- **Change in blood composition:**

- After operations, platelets increase in number and become more sticky. They agglutinate in small masses and adhere to vascular endothelium. Their lysis release thromboplastin which starts thrombosis.
- Fibrinogen also increase in pregnancy and lobar pneumonia.

- RBCs increase in polycythaemia causing increase blood viscosity and stasis.
- WBCs increase in leukaemia.
- Plasma decrease in dehydration.

Formation of thrombus and Types of thrombi:

1) **Pale thrombus (platelet thrombus):** the initial thrombus is composed only of platelets. The platelets deposit on the site of endothelial damage. They adhere to the exposed subendothelial collagen by help of von Willebrand factor (factor VIII). This factor produced by endothelial cells and acts as a bridge between platelet surface receptors and collagen. The adherent platelets release ADP and serotonin which promote further adhesion and aggregation of platelets forming a small white amorphous mass. Such platelet is fragile and can be washed easily by blood stream except if stasis occurs.
2) **Mixed thrombus**: stasis allows clotting factors to accumulate in the area. Thromboplastin released from the platelets and damaged tissues promote fibrin formation which deposit on the primary platelet thrombus. Next the depositedfibrin encourage further platelet accumulation. The platelets deposit as laminae at a right angle to the blood stream. Between these laminae, there is complete blood stasis and fibrin is deposited entangling the red and white blood cells. the formed thrombus is a mixed one and it is composed of both platelet masses and blood clots.
3) **Propagating thrombus**: the propagating thrombus extends in the direction of the heart.

Classification of thrombi:

I- According to color:

- Pale thrombus: formed mainly of platelets and fibrin. It is small, greyish-white, firm and adherent to intima as cardiac vegetations.
- Red thrombus: formed mainly of RBCs and fibrin. It is dark-red, soft and loosely attached to the vessel wall. It is very rare.
- Mixed thrombus: most thrombi has red and pale components.

II- According to presence or absence of bacteria:

- Infected thrombus: the thrombus contains bacteria. If the bacteria are pyogenic, the thrombus is called septic thrombus.
- Non-infected thrombus (aseptic): it doesn't contain bacteria.

Sites of thrombus formation:

1) **Thrombosis in veins:** it is the most common due to slow blood flow and thin venous walls. Two types occur:

Thrombophlebitis: thrombosis is initiated by inflammation of the venous wall.

Phlebothrombosis: thrombosis caused by factors other than inflammation.

- As what occurs in veins of the feet and calf in chronic cardiac patients due to stasis of blood and compression of calf muscles against bed mattress.
- Thrombosis of varicose veins due to stasis.

2) **Thrombosis of the arteritis:** less common than venous thrombosis due to rapid blood flow and thick elastic arterial wall which resist injury. Arterial thrombosis cause ischaemia.
   - Thrombosis may occur in arteries affected by atherosclerosis, due to roughness of the intima.

3) **Thrombosis of the heart:** more common in the left side, the following types may occur;

- **Mural thrombi:** it occurs over infarcts, commonly on the endocardial surface of the left ventricle near the apex.
- **Vegetations:** pale thrombi over the valves in rheumatic and bacterial endocarditis.

4) **Thrombosis of capillaries:** generally are rare, but may occur in acute inflammation and sever cold.

Fate of thrombi:

1) **Septic thrombus:** it is fragmented by proteolytic enzymes into septic emboli causing pyaemic abscess.
2) **Aseptic thrombus:** its elements disintegrate and form a pale red structureless mass. If the mass is small it dissolves by fibrinolysis. If it is large; it undergoes one of the followings:

1. Organization: the thrombus get invaded by capillaries and fibroblasts from the vascular wall and change to fibrous mass. This cause permanent vascular occlusion.
2. Organization with subsequent canalization: some of the invaded capillaries dilate and allow passage of blood through the thrombus. Or the fibrous thrombus shrinks from the vascular wall leaving a space which is then get lined by endothelium.
3. Dystrophic calcification: may occur giving a phlebolith.
4. Detachment: forming aseptic emboli causing infarction.
5. Propagating thrombus.

I- Emolism

Definition: impaction of an embolus in a narrow vessel.

Embolus: an insoluble solid, liquid or gaseous mass circulating in the blood stream.

Sites of embolism:

1. Systemic arteries.
2. Pulmonary arteries.
3. Intrahepatic branches of portal vein.

Types of emboli:

1. Detached thrombi and vegetations.
2. Tumor emboli.
3. Parasitic emboli as bilharzial ova.
4. Air emboli.
5. Fat emboli.
6. Clumps of bacteria amniotic fluid emboli.

Effect of emboli of thrombotic origin: It dependes on

1. Size of the impacted embolus.
2. Nature of the embolus; septic or aseptic.

Aseptic embolus: cause transient ischaemia if the collateral circulation is good and infarction if poor collaterals.

Septic embolus: produces pyaemic abscesses at site of its impaction.

3. State of collateral circulation.

Air emboli: results from:

- Injury to large veins in the neck. Air is sucked by negative pressure in the thorax.
- Faulty technique in doing artificial pneumothorax and in blood transfusion.
- In criminal abortion, air is sucked into uterine veins.
- Caisson's disease (Decompression sickness): in deep diving, compressed gases are inhaled. High pressure increases amount of gases dissolved in blood. If decompression is done rapidly, gases especially Nitrogen form emboli in the blood vessels.

Small amount of air doesn't cause pathological insult. However, 50-100cc of air interfere with cardiac contraction and cause acute heart failure.

Fat embolism: resulting from:

- Bone fracture and crush limb injury.
- Cutaneous burns.
- Inflammation of fatty tissues.
- Severe fatty change in the liver.

Amniotic fluid embolism: rare, resulting from strong uterine contractions cause tears in fetal membranes. Amniotic fluid pushed in opened veins. Such condition cause fatal pulmonary embolism to the mother.

Gangrene

Definition: It is massive tissue necrosis followed by putrefaction.

Causes:

1. **Necrosis**: caused by sudden ischaemia or bacterial toxins.
2. **Putrefaction**: is caused by saprophytic bacteria which break down the protein of the necrotic tissue liberating hydrogen sulphide which gives the tissues the foul odour. Hydrogen sulphide then unites with iron of the haemoglobin forming iron sulphide that stains the gangrenous tissue black.

Types of gangrene:

- Dry gangrene.
- Moist gangrene.

Classification of gangrene into dry and moist types depends on amount of blood and tissue fluids in the affected part at time of its death. Dry gangrene occurs in parts with less blood supply and tissue fluids. While moist gangrene occurs in area rich in blood and tissue fluids.

Dry gangrene

Dry gangrene of a limb: when the main arterial supply of a limb is suddenly cut off with poor collateral circulation. Arterial supply is the only occluded while venous and lymphatic drainage continue together with surface evaporation. The gangrene will be of dry type. The commonest example of dry gangrene is Senile gangrene of the lower limb.

Senile gangrene of the lower limb:

Usually affecting old aged men.

Predisposing factors:

- Atherosclerosis which predispose to arterial thrombosis and poor collateral circulation.
- Weak heart action and low blood pressure causing vascular stasis.
- Low body resistance due to nutritional disturbances and anaemia.

Pathological features:

- Arterial occlusion occurs either spontaneously or as a result of slight injury as tight shoes.
- Distal to occlusion, massive tissue necrosis occurs. The affected part is pale, cold with loss of sensation. Later on the necrotic tissue stains red by blood escaped from necrotic vessels. Venous and lymphatic evaporation and surface evaporation continue, so the necrotic tissue become dry. It becomes shrunken and mummified.
- Saprophytic bacteria invade the necrotic tissue cause putrefaction. saprophytic bacteria break down the protein of the necrotic tissue liberating hydrogen sulphide which gives the tissues the foul odour. Hydrogen sulphide then unites with iron of the haemoglobin forming iron sulphide that stains the gangrenous tissue black.
- The gangrenous process advances slowly along the limb because the gangrenous part irritates the living one and initiate inflammation of the living tissue with thrombosis of its vessels and further tissue necrosis and extension of the gangrene.
- When the gangrene reach a level with good blood supply, it stops. The toxic products of putrefaction acts as an irritant and cause a zone of acute inflammation in the neighboring healthy part. It appears as a narrow red line between healthy and gangrenous part called line of demarcation.
- From the healthy side, granulation tissue grows towards the gangrenous part with formation of a groove on the surface called line of separation. This groove may slowly deepen until it separate the gangrenous part leaving a conical stump. The stump is conical as the gangrene spread higher up in the skin and subcutaneous tissue than in the muscles and bone as the blood supply of the skin and subcutaneous tissue is less abundant.

Moist gangrene:

It is caused by sudden arterial and venous occlusion. It occurs mainly in internal organs as intestine from which no evaporation of fluids can occur. Presence of tissue fluids aid rapid putrefaction. Gangrene spreads rapidly. The line of demarcation is poor and line of separation is absent. Toxaemia is sever.

Moist gangrene of the intestine: May occur in strangulated hernia, intussesception and volvulus. Venous obstruction occurs first. The affected loop shows congestion and oedema. It appears dark red and swollen. Then arterial occlusion occur and the intestinal loop undergo necrosis. The necrotic loop get invaded by putrefactive bacteria from the intestinal lumen. It stains black by hydrogen sulphide . Putrefaction spreads rapidly. Toxaemia is sever. The patient suffer from intestinal obstruction and may be complicated with peritonitis. Moist gangrene may occur due to occlusion of mesenteric arterial occlusion.

Diabetic gangrene: More common in diabetic women above age of 45 years old.

Diabetic gangrene is initiated by mild injury. It usually starts on the big toe or the sole of the foot. At first, gangrene is of dry type, but soon it become moist and oedematous as the tissue hyperglycaemia and poor body resistance help multiplication of bacteria followed by excess inflammatory fluid exudate. This type of gangrene spreads rapidly, poor line of demarcation. Usually with no tendency of self separation. Toxaemia is sever.

Item	Dry gangrene	Moist gangrene
Cause	Gradual arterial occlusion	Sudden venous and arterial occlusion
Site	Exposed parts as limbs	Internal organs as intestine.
Putrefaction	Slow Putrefaction	Rapid Putrefaction
Affected tissue	Mummified	Oedematous
Spread	Slow	Rapid
Line of demarcation	Marked	Faint/ poor
Self separation	May occur	Absent separation
Toxaemia	Mild Toxaemia	Severe Toxaemia

Haemorrhage

Definition: Haemorrhage means escape of blood outside blood vessels or cardiac champers.

Causes of Haemorrhage:

I- Traumatic causes.

II- Spontaneous haemorrhage as in:

- Diseased vascular walls as atherosclerosis or aneurysms.
- Inflammatory injury to vascular wall.
- Destruction of the vascular wall by tuberculosis, malignancy or peptic ulcer.
- Increased intravascular tension as in hypertension and chronic venous congestion.
- Haemorrhagic blood diseases.
- Vitamin deficiencies as vit. K,C.

Types of haemorrhage

1- **External Haemorrhage:** It is escape of blood outside the body

- Epistaxis: bleeding from the nose.
- Haemoptesis: coughing of blood. The source of the blood is lung or bronchi. The blood is red, frothy and alkaline.
- Haematemesis: vomiting of blood. The source of blood is the eosophagus, stomach and duodenum. Blood is digested, brown in color, acidic and mixed with food particles.
- Melena: dark digested blood in the stool. The source of blood is the eosophagus, stomach and duodenum.
- Haematuria: blood in the urine.
- Menorrhagia: excessive or prolonged menstrual bleeding.
- Metrorrhagia: irregular uterine bleeding not related to menstrual cycle.
- Haemorrhage from the skin.

2- **Internal Haemorrhage:** escape of blood inside body cavities (serous sacs).

- Haemothorax: Haemorrhage in the pleura.
- Haemopericardium: Haemorrhage in the pericardial sac.
- Haemoperitoneum: Haemorrhage in the peritoneal cavity.
- Haematocele: Haemorrhage in the tunica vaginalis.
- Haemoarthrosis: Haemorrhage in the joint cavity.

3- **Interstitial Haemorrhage:** escape of blood into the interstitial tissue spaces.

- Petechial Haemorrhage: small amount of blood of capillary origin.
- Ecchymosis: moderate amount of blood.
- Haematoma: large amount of blood causing swelling.

Natural arrest of Haemorrhage (haemostasis):

- Fall in blood pressure due to decrease blood volume leading to further blood loss.
- Curling up of endothelium and contraction of muscularis layer of the vessel.
- Serotonin released from platelets cause local vasoconstriction of the arterioles.
- Clotting of blood within and around the vessels.
- Organization of the formed clot causing permanent closure of the vessel by fibrous tissue.

In septic wounds; the clot may undergo softening by the proteolytic enzymes liberated from dead leucocytes resulting in secondary Haemorrhage.

Effects of Haemorrhage:

1- Small amounts and once: no effect.
2- Small amounts but repeatedly as in piles, peptic ulcers causing microcytic hypochromic anaemia.
3- Moderate amount (up to 750 cc): is compensated by

- Immediate fall in blood pressure.

- Decrease hydrostatic pressure inside the vessels allow withdrawal of tissue fluids to blood vessels.
- Increased secretion of adrenaline help in increasing blood pressure.
- Reflex vasoconstriction of blood vessels in skin, muscles and splanchnic area to maintain adequate blood supply to vital organs.
- Proteins are added from the liver.
- WBCs and RBCs are added from bone marrow.

4- Massive amount: cause haemorrhagic shock and death.