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A QUICK REVIEW BOOK ON GENERAL PATHOLOGY PART II

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CHAPTER-1 OEDEMA

Definition: Oedema is "abnormal and excessive accumulation of free fluid in the interstitial tissue spaces and serous cavities."

- Free fluid in body cavities: e.g. Hydrothorax, Ascites
- Free fluid in interstitial space: Commonly termed as oedema, the fluid present free in the interstitial space between the cells and can be displaced from one place to another.

Oedema is differentiated into two types

1. Localised oedema: Present in particular part of the body e.g. Pulmonary oedema, facial oedema.
2. Generalised oedema: Present all over the body in same distribution e.g. Anasarca.

Depending upon fluid composition, oedema fluid may be:

A. **Transudate** which is more common such as in oedema of cardiac and renal disease.

B. **Exudate** present in inflammatory oedema.

PATHOGENESIS OF OEDEMA

Oedema is occurs when there is interference in the normal distribution of the fluid in the body. There are well known 5 mechanisms by which oedema can be developed:

1. INCREASED CAPILLARY HYDROSTATIC PRESSURE

- A rise in the hydrostatic pressure can cause fluid to shift into extra cellular space. E.g. oedema of cardiac diseases, postural oedema

2. DECREASED PLASMA ONCOTIC PRESSURE

    - When total plasma protein level fall in body then the oncotic pressure which is responsible for maintain the fluid into in vascular system fails and it causes oedema. E.g. oedema of renal origin.

3. LYMPHATIC OBSTRUCTION

    - Occurs due to obstruction of the lymphatics which are also responsible for the removal of the excessive fluid from ECF to the vascular system via lymphatics. E.g. Occlusion of lymphatic channels by malignant cells may result in lymphoedema.

4. INCREASED CAPILLARY PERMEABILITY

    - When the endothelium of the capillary injured it causes “GAP FORMATION” in the inner layer of the vessels which causes excessive leaking of the fluid from vascular compartment to the ECS. E.g. oedema occur due to inflammation

5. SODIUM AND WATER RETENTION

    - When sodium concentration in the blood is not maintainted properly it causes oedema. By theses machenism it can be explained

        a. Intrinsic renal mechanism

        b. Extrinsic renal mechanism

        c. ADH mechanism

- The examples of oedema by these mechanisms are as under:

i) Oedema of cardiac disease e.g. in congestive cardiac failure.

ii) Ascites of liver disease e.g. in cirrhosis of liver.

iii) Oedema of renal disease e.g. in nephrotic and nephritic syndrome.

Some Important types of oedemas are enumerated here:

- ❋ Renal Oedema
- ❋ Cardiac Oedema
- ❋ Pulmonary Oedema
- ❋ Cerebral Oedema
- ❋ Hepatic Oedema
- ❋ Nutritional Oedema
- ❋ Myxoedema

CHAPTER- 2 SHOCK

Shock is defines as a life-threatening clinical condition of cardiovascular failure characterised by:

- an acute reduction of effective circulating blood volume (hypotension); and
- an inadequate perfusion of cells and tissues (hypoperfusion).

Definition "*true (or secondary) shock"* is a circulatory imbalance between oxygen requirements at the cellular level and oxygen supply.

- It is also called as circulatory shock and is the type which is commonly referred to as 'shock' if not specified.

CLASSIFICATION OF SHOCK

Major classification of shock are:

1. Hypovolaemic Shock

- This type of shock occurs due to amount of flowing blood reduced.
- It can occur due to loss of RBC or Plasma due to injury which causes haemorrhage or due to loss of plasma protein alone like in cases of burn.

2. Cardiogenic Shock

- This type of shock occurs due to sudden stoppage of the heart or decline in the cardiac output without reduction in the actual volume of the flowing blood.

3. Septic (Toxaemic) Shock

- This type of shock occurs due to the severe infection of the bacteria.

- It may be the result of Gram-negative septicaemia (endotoxic shock) which is more common, or less often from Gram-positive septicaemia (exotoxic shock).

4. Other types These include:

(i) Traumatic shock- occurs due to any severe trauma to the body.

(ii) Neurogenic shock- Occurs when there is injury to the spinal cord and dysregulation in the Autonomic activity.

(iii) Hypoadrenal shock- occurs when there is severe insufficiency of the adrenal harmoens especially cortisol.

PATHOGENESIS OF SHOCK

Shock involve following 3 derangements:

i) Reduction in the circulating blood volume.

ii) Reduction in the supply of oxygen to the cells and tissues which causes hypoxia and further leads to anoxia.

iii) Inflammatory mediators and toxins released from shock further causes cellular injury.

- These imbalances starts a compensatory mechanisms.
- But after some time a very dangerous non reversible cycle of cell injury and severe cellular dysfunction occurs which lead to failure of organ function.

PATHOGENESIS OF HYPOVOLAEMIC SHOCK

- Occurs due to inadequate circulating blood volume which can be due to various causes.

- Most common cause is loss of red cell mass due to haemorrhage thus also called as haemorrhagic shock.
- Due to loss of the blood volume there is decreased cardiac output and low intracardiac pressure which causes the clinical symptoms of shock.

PATHOGENESIS OF CARDIOGENIC SHOCK

- It is most commonly occurs due to the left ventricular failure.
- It can be due to myocardial infarction.
- Due to failure in the cardiac output there is transfer of fluid occurs from pulmonary vascular bed to pulmonary interstitial space.
- Which further leads to the fluid movement into the alveoli.

PATHOGENESIS OF SEPTIC SHOCK

- Septic shock occurs more commonly from Gram-negative bacteria and less often from Gram-positive bacteria.
- These bacteria can enter in the body from alimentary tract, respiratory tract, genitourinary tract or from skin.
- Due to this infection there is immune system activation and severe systemic inflammatory response.
- Due to that there is vasodilatation and increased vascular permeability.
- Due to that profound peripheral vasodilatation and pooling of blood occurs which causes ***hyperdynamic*** circulation.
- Increased vascular permeability is responsible for the development of inflammatory oedema.

- Disseminated intravascular coagulation (DIC) is most likely to develop in septic shock because the toxins causes endothelial cell injury.

PATHOPHYSIOLOGY (STAGES OF SHOCK)

- Deterioration of the circulation in shock is a progressive and continuous phenomenon.
- Compensatory mechanisms help in some extent but after sometimes it become gradually ineffective.
- For purpose of understanding shock is divided in 3 stages:

A COMPENSATED (NON-PROGRESSIVE, INITIAL, REVERSIBLE) SHOCK

- It is the earliest stage of the shock.
- In this stage body is trying to maintain adequate cerebral and coronary blood supply by increasing and redistribution of blood to the most important organs.
- Due to that the most vital organs which are brain and heart are sufficiently perfused and oxygenated.
- This is accomplished by activation of various neurohormonal mechanisms causing "*widespread vasoconstriction* and by *fluid conservation by the kidney*."

B PROGRESSIVE DECOMPENSATED SHOCK

- This is the second stage.
- This is a stage of progressive deterioration.
- If the patient is suffering from any heart condition it can be a risk factor for the early deterioration.
- Tissue hypoperfusion in progressive decompensated shock cause:

i) Pulmonary hypoperfusion

ii) Tissue ischaemia

C IRREVERSIBLE DECOMPENSATED SHOCK

- It is the last stage of shock.
- When the shock is so severe that compensatory mechanisms and therapy doesn't help the patient and no recovery takes place.
- Then, It is called decompensated or irreversible shock.
- Due to widespread cell injury following changes takes place in the body:

  i) Progressive vasodilatation

  ii) Increased vascular permeability

  iii) Release of myocardial depressant factor (MDF)

  iv) Worsening pulmonary hypoperfusion

  v) Anoxic damage to heart, kidney and brain

  vi) Hypercoagulability of blood

CLINICAL FEATURES AND COMPLICATIONS

- The classical features of decompensated shock are characterised by *depression of 4 vital processes:*

  i) Very low blood pressure

  ii) Subnormal temperature

  iii) Feeble and irregular pulse

iv) Shallow and sighing respiration

The patients in shock have

a. pale face,

b. sunken eyes,

c. weakness,

d. cold and clammy skin.

- Life-threatening complications in shock occurs due to insufficient oxygen supply to the cells of the body which causes cell injury and results in immuno-inflammatory responses and activation of various cascades (clotting, complement, kinin).
- These include the following:
    i. Acute respiratory distress syndrome (ARDS)
    ii. Disseminated intravascular coagulation (DIC)
    iii. Acute renal failure (ARF)
    iv. Multiple organ dysfunction syndrome (MODS)
    v. Stupor
    vi. Coma and death

CHAPTER-3 THROMBOSIS

Definition: - "Thrombosis is defined as a formation of solid mass in the circulation from the constituent of the flowing blood."

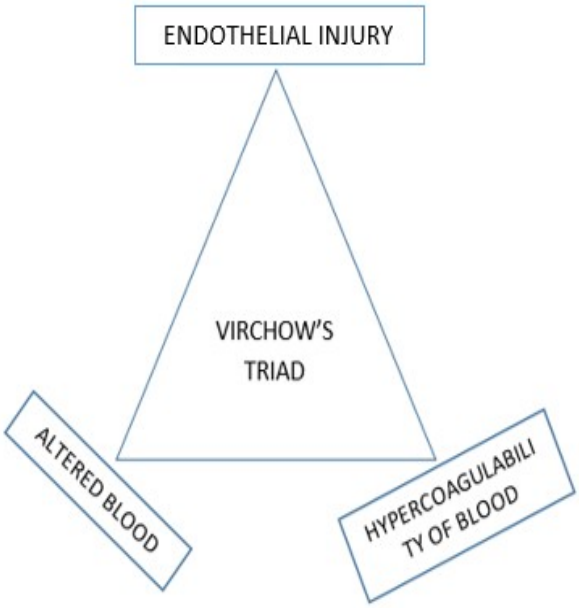
- The Flowing solid mass is called **Thrombus.**
- *Thrombosis* involves activation of platelets, While C*lotting* involves conversion of soluble fibrinogen to insoluble polymerised fibrin.
- *Haematoma* is Characterised by the extravascular accumulation of blood e.g. into the tissues.
- *Haemostatic plugs* are the blood clots formed due to the injury to the blood vessels.
- Thrombi in cardiovascular system may be life-threatening because it causes one of the following harmful effects:

1. Ischaemic injury -Thrombi may reduce or stop the blood supply to part of an organ or tissue and cause ischaemia which further leads to infarction.

2. Thromboembolism- Thrombus or its part may get detached and travel along in the blood vessel as embolus.

PATHOPHYSIOLOGY

- Virchow described three primary events which predispose to thrombus Formation.



- After that activation of platelets and of clotting system takes place and further process of thrombosis starts.

1. ENDOTHELIAL INJURY
    - Vascular injury causes subendothelial extracellular matrix or ECM to expose which contain collagen, elastin etc which works as thrombogenic agents and plays an important role in commencing thrombosis.
2. ROLE OF PATELETES
    - Platelets plays a main role in normal haemostasis as well as in thrombosis. The sequence that follows are:
        a. Receptor on Platelets recognises the site of injury and the circulating platelets stick to exposed subendothelial ECM. This process is also known as primary aggregation.

b. Now Activated platelets go through release reaction due to that platelet granules are released to the exterior. Two main types of platelet granules are released:

i. Dense bodies ii. Alpha granules

c. Due to primary aggregation of the platelets, these plateles release Adenosine diphosphate (ADP) which is a Strong platelet aggregating agent, it causes more platelets to aggregate and attached to the platelets. This is known as the secondary aggregation.

3. ROLE OF COAGULATION SYSTEM

- Coagulation is a mechanism in that plasma fibrinogen is converted into the solid mass of fibrin.
- The coagulation system is plays an important role in both haemostatic process as well as thrombus formation.

i) In the intrinsic pathway

ii) In the extrinsic pathway

iii) The common pathway

- ***Regulation of coagulation system*** innormal conditions the blood is kept in fluid state and the coagulation system is remains in controle by following:

i) *Protease inhibitors*

ii) *Fibrinolytic system*

4. ALTERATION IN BLOOD FLOW

- It is described in relation to normal blood flow.

a. In normal condition the flow of blood is axial.

b. Thrombosis causes turbulence and stasis due to that the normal axial flow of blood is disturbed.

5. HYPERCOAGULABILITY

- Thrombophilia or hypercoagulablility states is a condition in which the coagulation process is increased and due to that the patient is more susceptible to develop venous thrombosis.
- Thrombophilia occurs either due to hereditary or acquired causes.

ORIGIN OF THROMBI AT DIFFERENT SITES

A. CARDIAC THROMBI: -

- Thrombi may form in any of the chambers of the heart and on the valve cusps.
- They are more common in the atrial appendages, especially of the right atrium, and on mitral and aortic valves such as vegetations seen in infective endocarditis and non-bacterial thrombotic endocarditis.

ARTERIAL THROMBIe.g.

i) Aorta: aneurysms, arteritis.

ii) Coronary arteries: atherosclerosis.

iii) Mesenteric artery: atherosclerosis, arteritis.

iv) Arteries of limbs: atherosclerosis, diabetes mellitus, Buerger's disease, Raynaud's disease.

v) Renal artery: atherosclerosis, arteritis.

vi) Cerebral artery: atherosclerosis, vasculitis.

VENOUS THROMBIe.g.

i) Veins of lower limbs: deep veins of legs, varicose veins.

ii) Popliteal, femoral and iliac veins: postoperative stage, postpartum.

iii) Pulmonary veins: CHF, pulmonary hypertension.

iv) Hepatic and portal vein: portal hypertension.

v) Superior vena cava: infections in head and neck.

vi) Inferior vena cava: extension of thrombus from hepatic vein.

vii) Mesenteric veins: volvulus, intestinal obstruction.

viii) Renal vein: renal amyloidosis.

CAPILLARY THROMBI

In this condition minute thrombi are formed especially in the capillaries in acute inflammatory lesions, vasculitis and in disseminated intravascular coagulation (DIC).

FATE OF THROMBUS

1. RESOLUTION

- Completely dissolve the thrombus.

3. ORGANISATION

- In case body is unable to dissolve the thrombus it starts getting organise. After organisation the Phagocytic cells appear at the site and begin to phagocytose fibrin and cell debris.

4. PROPAGATION

- Due to more and more deposition from the constituents of flowing blood the thrombus starts to enlarge in size.

5. THROMBOEMBOLISM

- When thrombi are in in early stage and also when thrombi are infected they are quite friable and get easily detached from the vessel wall. These friable thrombi

release in the blood circulation either completely or in the part known as "*Emboli.*"

CLINICAL EFFECTS

1. CARDIAC THROMBI

- In case when thrombi are large they may sudden death by mechanical obstruction of blood flow or through thromboembolism to vital organs.

2. ARTERIAL THROMBI

- Due to that obstruction of the blood flow occurs and it can further leads to the necrosis of that particular part which may further leads to gangrene. In cases of the thrombosis of coronary artery sudden death can occurs.

3. VENOUS THROMBI (PHLEBOTHROMBOSIS)

- These may cause following effects:

  i) Thromboembolism

  ii) Oedema of area drained

  iii) Poor wound healing

  iv) Skin ulcer

  v) Painful thrombosed veins (thrombophlebitis)

  vi) Painful white leg (phlegmasia alba dolens) due to ileofemoral venous thrombosis in postpartum cases

  vii) Thrombophlebitis migrans in cancer.

4. CAPILLARY THROMBI

- Microthrombi in capillary circulation may give rise to disseminated intravascular coagulation (DIC).

CHAPTER-4 EMBOLISM

- Embolism is define as the "Partial or complete obstruction of some part of the cardiovascular system by any mass carried in the circulation."
- The intravascular mass detached from its site of origin in the circulation is known as embolus.
- Most usual forms of emboli (90%) are thromboemboli i.e. originating from thrombi or their parts detached from the vessel wall.

Emboli may be of various types:

A. Depending upon the matter in the emboli:

i) Solid

ii) Liquid

iii) Gaseous

B. Depending upon whether infected or not:

i) Bland

ii) Septic

C. Depending upon the source of the emboli:

i) Cardiac emboli

ii) Arterial emboli

iii) Venous emboli

iv) Lymphatic emboli.

D. Depending upon the flow of blood, two special types of emboli are mentioned:

i) Paradoxical embolus

- An embolus which is transferred from the Right side of heart or venous side of circulation to the left side of the heat or arterial side or vice versa, is called paradoxical or crossed embolus.

ii) Retrograde embolus

- Retrograde embolus travels against the flowing blood.

THROMBOEMBOLISM

- It is the most common type of embolism.
- These may arise in the arterial or venous circulation.

Arterial (systemic) thromboembolism e.g.

A. Causes within the heart (80-85%):

i. Mural thrombi in the left atrium or left ventricle
ii. Vegetations on the mitral or aortic valves
iii. Prosthetic heart valves
iv. Cardiomyopathy.

B. Causes within the arteries:

i. Emboli developing in relation to atherosclerotic plaques
ii. Aortic aneurysms
iii. Pulmonary veins
iv. Paradoxical arterial emboli from the systemic venous circulation.

If the blood vessels is obstructed by the emboli it may leads to the following results:

i) *Infarction* of the organ or its affected part.

ii) *Gangrene*

iii) *Arteritis and mycotic aneurysm* formation from bacterial endocarditis.

iv) *Myocardial infarction* may occur following coronary embolism.

v) *Sudden death* may result from coronary circulation obstruction by embolism or embolism in the middle cerebral artery.

Venous thromboembolism e.g.

i) Deep vein thrombosis (DVT) of the lower legs, the most common cause of venous thrombi.

ii) Thrombi in the pelvic veins.

iii) Thrombi in the veins of the upper limbs.

iv) Thrombosis in cavernous sinus of the brain.

v) Thrombi in the right side of heart.

- **Pulmonary embolism is the most significant effect of the venous emboli.**

CHAPTER-5 PULMONARY THROMBOEMBOLISM

Definition

- It is the most common and deadly form of venous thromboembolism.
- Due to the thromboemboli there is complete obstruction of the pulmonary arterial circulation.
- Pulmonary thrombosis is uncommon and may occur in pulmonary atherosclerosis and pulmonary hypertension.

Etiology

- Pulmonary emboli are commonly occurs in hospitalised or bed-ridden patients.
- The causes are as follows:

i) Thrombi originating from large veins of lower legs are the cause in 95% of pulmonary emboli.

ii) Less common sources include thrombi in varicosities of superficial veins of the legs, pelvic veins, uterine, periovarian and broad ligament veins.

Pathogenesis

- Stasis of the venous blood and hypercoagulable states is the risk factor of the thomboembolism.
- If the thrombus is Large, it is stuck at the bifurcation of the main pulmonary arteryor may be found in the right ventricle or its outflow tract.
- If there are *multiple emboli*, or a large embolus may get split into many smaller emboli which are then stuck in a number of vessels.

- Rarely, *paradoxical embolism* may occur by passage of an embolus from right heart into the left heart through atrial or ventricular septal defect. In this way, pulmonary emboli may reach systemic circulation.

CONSEQUENCES OF PULMONARY EMBOLISM

- The patients who are immobilised due to some disease has a more risk of developing Pulmonary embolism.
- Women in their reproductive period are at higher risk such as in late pregnancy, following delivery and with use of contraceptive pills.
- Occurrence of pulmonary embolism may leads to following consequences:

i) Sudden death

ii) Acute corpulmonale

iii) Pulmonary infarction

iv) Pulmonary haemorrhage

v) Resolution

vi) Pulmonary hypertension, chronic corpulmonale and pulmonary arteriosclerosis.\

CHAPTER- 6 SYSTEMIC EMBOLISM

- This is the type of arterial embolism that originates commonly from thrombi in the diseased heart, especially in the left ventricle.
- These heart diseases include myocardial infarction, congenital heart disease, cardiomyopathy, infective endocarditis etc.

FAT EMBOLISM

- In this type of embolism obstruction of arterioles and capillaries occurs by fat globules.
- It may occur from following causes:

Traumatic causes:

i) Trauma to bones is the most common cause of fat embolism e.g. in fractures of long bones.

ii) Trauma to soft tissue e.g. laceration of adipose tissue and in puerperium due to injury to pelvic fatty tissue.

Non-traumatic causes:

i. Extensive burns
ii. Inflammation of bones and soft tissues
iii. Fatty liver
iv. Diabetes mellitus
v. Hyperlipidaemia
vi. Pancreatitis
vii. Decompression sickness
viii. Sickle cell anaemia
ix. Extrinsic fat or oils introduced into the body

PATHOGENESIS

- Pathogenesis of fat embolism is explained by following mechanisms:

i) Mechanical theory: - Due to trauma to the bone or soft tissues fat fluid may get into the circulation and may case obstruction of the circulation.

ii) Emulsion instability theory: -Fat emboli are formed by accumulation of plasma lipids and fatty acids due to disturbance in natural emulsification of fat.

iii) Intravascular coagulation theory: - e.g. disseminated intravascular coagulation DIC and accumulation of fat emboli.

iv) Toxic injury theory According to this theory, the small blood vessels of lungs are chemically injured by high plasma levels of free fatty acid which may leads to the development of fat embolism.

CONSEQUENCES OF FAT EMBOLISM

- The effects of fat embolism depend upon the size and quantity of fat globules.

i) Pulmonary fat embolism: When a patient is died suddenly after fracture of a long bone at the time of autopsy numerous fat emboli are found in the capillaries of the lung.

ii) Systemic fat embolism Some of the fat globules may pass through the pulmonary circulation and may develop the consequences depending upon where the fat globules are obstructing the blood flow.

AIR EMBOLISM

- It occurs when air is introduced into either vein or artery.

Venous Air Embolism

- Air may get enter into the systemic venous circulation
- It may occur in following condition:

i) Operations on the head and neck, and trauma

ii) Obstetrical operations and trauma

iii) Intravenous infusion of blood and fluid

iv) Angiography

- Factors that affect venous air embolism depends upon:

i) Amount of air enters in circulation

ii) Rapidity of entry

iii) Position of the patient

iv) General condition of the patient

- ❋ Death occurs when air is trapped in the pulmonary arterial trunk in the right heart.

ARTERIAL AIR EMBOLISM

- Air may enter in the pulmonary vein or its tributaries in the following conditions:

  i) Cardiothoracic surgery and trauma

  ii) Paradoxical air embolism

  iii) Arteriography

- The *effects* of arterial air embolism have certain characteristic features:

  i) Marble skin due to blockage of cutaneous vessels.

  ii) Air bubbles in the retinal vessels seen ophthalmoscopically.

iii) Pallor of the tongue due to occlusion of a branch of lingual artery.

iv) Coronary or cerebral arterial air embolism may cause sudden death.

DECOMPRESSION SICKNESS

- It is also a special type of gas embolism.
- It is also known as caisson's disease, divers' palsy or aeroembolism.

Pathogenesis

- Decompression sickness occurs when person suddenly decompresses either from high atmospheric pressure to normal level or from normal pressure to low atmospheric pressure.
- Divers, workers in caissons (diving-bells), offshore drilling and tunnelers, when they *descend* to high atmospheric pressure there is increased amount of atmospheric gases mainly nitrogen; others are O2, CO2 which dissolved in blood and tissue fluids.
- When such individual ascends too suddenly i.e. comes to normal level suddenly from high atmospheric pressure, the gases come out of the fluid as minute bubbles.
- Persons who ascendto high altitudes or air flight in unpressurised cabins, the person are exposed to sudden decompression from low environmental pressure to normal levels.
- Pathologic changes are more marked in sudden decompression from high pressure to normal levels than in those individuals who decompress from low pressure to normal levels.

EFFECTS OF DECOMPRESSION SICKNESS

- Clinical effects of decompression sickness are of 2 types:

A **Acute form** occurs when there is acute obstruction of small blood vessels in the near the joints and skeletal muscles. The condition is clinically characterised by:

(i) *The bends*

(ii) *The chokes* and

(iii) C*erebral effects*.

B **Chronic form** when ischaemic necrosis occurs throughout the body, especially in the skeletal system. Ischaemic necrosis may occur due to embolism but other factors such as platelet activation, intravascular coagulation and hypoxia may contribute for the further enhancement of the necrosis. The features of chronic form are as under:

i) Avascular necrosis of bones

ii) Neurological symptoms

iii) Lung involvement

iv) Skin manifestations

AMNIOTIC FLUID EMBOLISM

- This is the most serious, unpredictable cause of maternal death.
- It is very serious and if it occurs it can't be prevented.
- While in labour and in the immediate postpartum period the contents of amniotic fluid may get enter in the uterine veins and then reach right side of the heart which results in fatal complications.
- The Components of the amniotic fluid which may be found in uterine veins, pulmonary artery and vessels of other organs are:

i. Epithelial squames,
ii. vernix caseosa,
iii. lanugo hair,
iv. bile from meconium, and
v. mucus.

- The characteristic features of the arterial air embolism are:

a. Sudden respiratory distress and dyspnoea
b. Deep cyanosis
c. Cardiovascular shock
d. Convulsions
e. Coma
f. Unexpected death

ATHEROEMBOLISM

- Sometimes atheromatous plaques may get dislodge from the aorta (where it forms) and form emboli which may lodged in medium-sized and small arteries.

TUMOUR EMBOLISM

"Malignant tumour cells invade the local blood vessels and may form tumour emboli to be lodged elsewhere, producing metastatic tumour deposits."

CHPATER-7 HYPERAEMIA AND CONGESTION

- When the blood volume is increased in the dilated vessels of an organ or tissue it is known as hyperaemia and congestion. When this condition develops rapidly it is called *acute*, and when this develops gradually it is known as *chronic*.

1. *ACTIVE HYPERAEMIA*

- When the arteries, arterioles and capillaries is dilated it is occurs either due to sympathetic neurogenic mechanism or by the release of vasoactive substances. The affected tissue or organ is pink or red in appearance e.g.

    i) Inflammation e.g. congested vessels in the walls of alveoli in pneumonia

    ii) Blushing i.e. flushing of the skin of face in response to emotions

    iii) Menopausal flush

    iv) Muscular exercise

    v) High grade fever

    vi) Goitre

2. *PASSIVE HYPERAEMIA (VENOUS CONGESTION)*

- When there is impaired drainage of the veins occurs it leads to the dilatation of the veins and capillaries which is known as passive hyperaemia or venous congestion, commonly known as *passive congestion*.
- Congestion may be acute or chronic.
- The chronic is more common and is called *chronic venous congestion* (CVC).

i. ***Local venous congestion*** occurs when there is obstruction of the venous outflow from an organ or part of the body e.g. portal venous obstruction in cirrhosis of the liver, outside pressure on the vessel wall as occurs in tight bandage, plasters, tumours, pregnancy, hernia or intraluminal occlusion by thrombosis, etc.

ii. ***Systemic (General) venous congestion***occurs when there is engorgement of veins e.g. in left-sided and right-sided heart failure and diseases of the lungs which interfere with pulmonary blood flow like pulmonary fibrosis, emphysema etc.

CHAPTER-8 HAEMORRHAGE

- Haemorrhage occurs when the blood comes out from the blood vessels.
- The bleeding of haemorrhage may occur either *externally or internally* into the serous cavities (e.g. haemothorax, haemoperitoneum, haemopericardium)or into a hollow viscus.
- Haematoma occurs when the blood escape from the blood vessel and come out in the tissue. Haematoma occurs when little amount of the blood comes out from the blood vessel.
- When large amount of blood comes out into the skin and mucous membranes it is known as *ecchymoses*.
- *Purpuras* are small areas of haemorrhages upto 1 cm into the skin and mucous membrane.
- *Petechiae*are minute pinhead-sized haemorrhages.
- When RBCs escape into the tissue e.g. in acute inflammation it is known as *diapedesis*.

The effects of blood loss depend upon 3 main factors:

i) The Quantity of blood loss;

ii) The speed of blood loss; and

iii) The Location of haemorrhage.

CHAPTER-9 ISCHAEMIA

- Ischaemia occurs when the blood supply to an organ in decreases due to any reason and the organ needs are not completely fulfilled.
- Decrease in of blood supply may be *complete* (complete ischaemia) or *partial* (partial ischaemia).
- The adverse effects of ischaemia may result from 3 ways:

  1. Hypoxia due to low oxygen to tissues.

  2. Malnourishment of cells due to inadequate supply of nutrients to the tissue.

  3. Inadequate clearance of metabolites which causes accumulation of metabolic wastes in cell.

ETIOLOGY

1. Causes in the heart

- Inadequate cardiac output resulting from heart block, ventricular arrest and fibrillation from various causes.

2. Causes in the arteries

- The commonest and most important causes of ischaemia are due to obstruction in arterial blood supply as under:

i) Luminal occlusion of artery (intraluminal):

a) Thrombosis

b) Embolism

ii) Causes in the arterial walls (intramural):

a) Vasospasm (e.g. in Raynaud's disease)

b) Hypothermia, ergotism

c) Arteriosclerosis

d) Polyarteritisnodosa

e) Thromboangiitis obliterans (Buerger's disease)

f) Severed vessel wall

iii) Outside pressure on an artery (extramural):

a) Ligature

b) Tourniquet

c) Tight plaster, bandages

d) Torsion.

3. Causes in the veins

- Blockage of venous drainage may lead to engorgement and obstruction to arterial blood supply resulting in ischaemia.

i) *Luminal occlusion of vein (intraluminal):*

a) Thrombosis of mesenteric veins

b) Cavernous sinus thrombosis

***ii) Causes in the vessel wall of vein (intramural)*:**

a) Varicose veins of the legs

iii) Outside pressure on vein (extramural):

a) Strangulated hernia

b) Intussusception

c) Volvulus

4. Causes in the microcirculation

- Ischaemia may result from occlusion of arterioles, capillaries and venules.

i) *Luminal occlusion in microvasculature (intraluminal):*

a) By red cells e.g. in sickle cell anaemia, red cells parasitised by malaria, acquired haemolytic anaemia, sludging of the blood.

b) By white cells e.g. in chronic myeloid leukaemia

c) By fibrin e.g. defibrination syndrome

d) By precipitated cryoglobulins

e) By fat embolism

f) In decompression sickness.

***ii) Causes in the microvasculature wall (intramural)*:**

a) Vasculitis e.g. in polyarteritisnodosa, Henoch-Schönlein purpura, Arthus reaction, septicaemia.

b) Frost-bite injuring the wall of small blood vessels.

***iii) Outside pressure on microvasculature (extramural)*:**

a) Bedsores.

CHAPTER-10 INFARCTION

- Infarction is occurred when there is ischemia to the tissue for log time which causes irreversible cell injury or necrosis.
- *Infarct* is the area where necrosis occurs.

ETIOLOGY

- All the causes of ischaemia can cause infarction.

  i) Most commonly, infarcts are caused by interruption in arterial blood supply, called *ischaemic necrosis*.

  ii) Less commonly, venous obstruction can produce infarcts termed *stagnant hypoxia*.

  iii) Generally, *sudden, complete, and continuous occlusion* e.g. thrombosis or embolism produces infarcts.

  iv) Infarcts may be produced by *nonocclusive circulatory insufficiency* as well e.g. incomplete atherosclerotic narrowing of coronary arteries may produce myocardial infarction due to acute coronary insufficiency.

TYPES OF INFARCTS

- Infarcts are classified depending upon different features:

1. According to their colour:

i) Pale or anaemic

ii) Red or haemorrhagic.

2. According to their age:

i) Recent or fresh

ii) Old or healed

3. According to presence or absence of infection:

i) Bland, when free of bacterial contamination

ii) Septic, when infected.

PATHOGENESIS

- The process of infarction takes place as follows:

i) Localised hyperaemia

ii) Oedema and haemorrhage.

iii) Cellular changes like degeneration occurs (reversible cell injury).

iv) Irriversible cell injury occurs.

v) Then here is progressive proteolysis

vi) Then An acute inflammatory reaction and hyperaemia occurs.

vi) Blood pigments start to deposit on Infarct.

vii) Finally, ingrowth of granulation tissue occurs.

viii) Infarct then replaced by the scar tissue.

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