Cardio-Vascular System

2

Pathology

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Embolism

An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site *distant* from its point of origin.

❖ Types of emboli

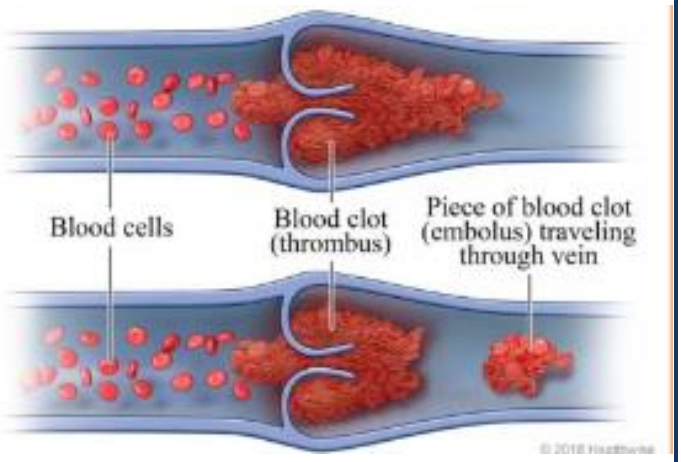
Emboli are classified according to their composition into 4 major types

I. Thromboembolism, which makes 99% of all cases,
II. Fat embolism,
III. Air embolism,
IV. Amniotic embolism.

These make the remaining 1%

❖ Thrombus vs Thromboembolus

- Thrombus is a blood clot that occurs inside the cardiovascular system.
- Thromboembolus is a piece that is dislodged of the original thrombus and is now travelling inside the circulation, it can go to distant sites away from its site of origin and once it's there it can produce the main clinical symptoms.



- Thromboembolism is the first type of emboli according to the composition of the embolus.

❖ Clinical significance

- The clinical significance of a thromboembolus is exactly the same as a thrombus.
- They could result in partial or complete vascular occlusion consequently leading to infarction (ischemic necrosis) of downstream tissue.

❖ Circulation

There are two types/sides of circulation:

- **Venous:**
  The direction of blood in the venous side of circulation is from tissues toward the heart specifically the right side of the heart, and then towards the lungs.
- **Arterial:**
  It starts with the heart and the direction of blood is from the heart towards different body tissues.

Emboli can also be classified according to site of their origin, meaning the location where they are first formed. Therefore, emboli can either be venous or arterial.

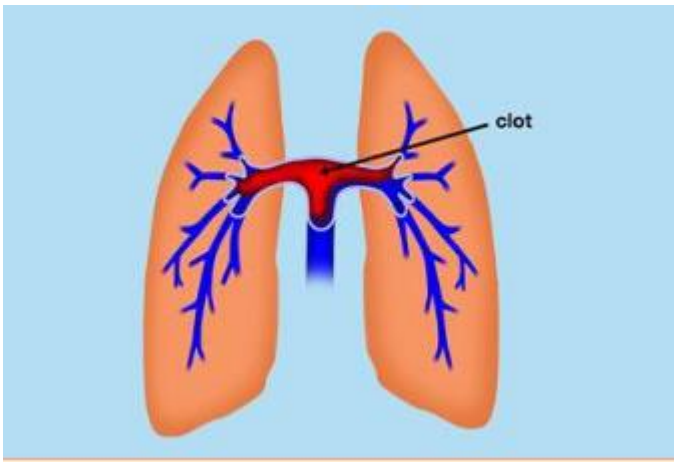
Venous thromboembolism

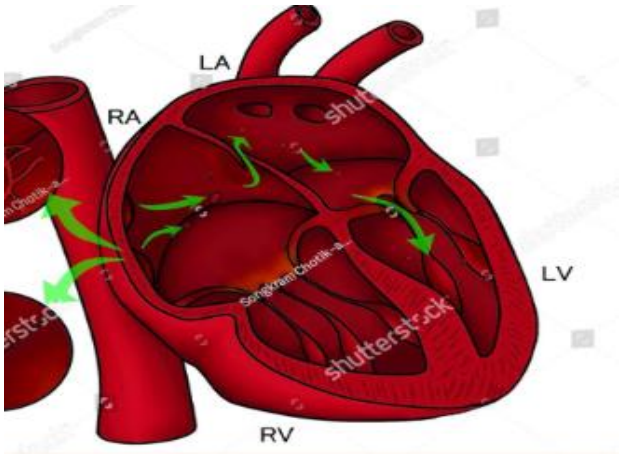
- A thrombus is formed inside the venous side of circulation and starts fragmenting. It's now forming a venous embolus. This venous embolus travels along with the blood toward the right side of the heart and from there it will pass through the pulmonary arteries into the lungs.
- The origin of most venous emboli is the *lower limbs*, specifically "the deep veins" causing what is known as deep vein thrombosis (DVT).
- The target of most venous emboli are lungs.
- The consequence of venous emboli depends on the *size* and exact *location* inside the lungs.

❖ Special terms regarding pulmonary thromboembolism

- saddle embolus is a **large** embolus capable of occluding **the bifurcation of pulmonary artery trunk,** leading to loss of blood supply to the lungs, which is usually fatal.

- paradoxical embolus is the passage of an embolus from the venous side of the heart to arterial side of the heart through a defect inside the heart, this defect could be a patent foramen ovale, atrial septal defect (ASD) or a ventricular septal defect (VSD). In this condition, the origin of the embolus is the venous circulation, but the target would be on the arterial side of the circulation (and that is the paradox)





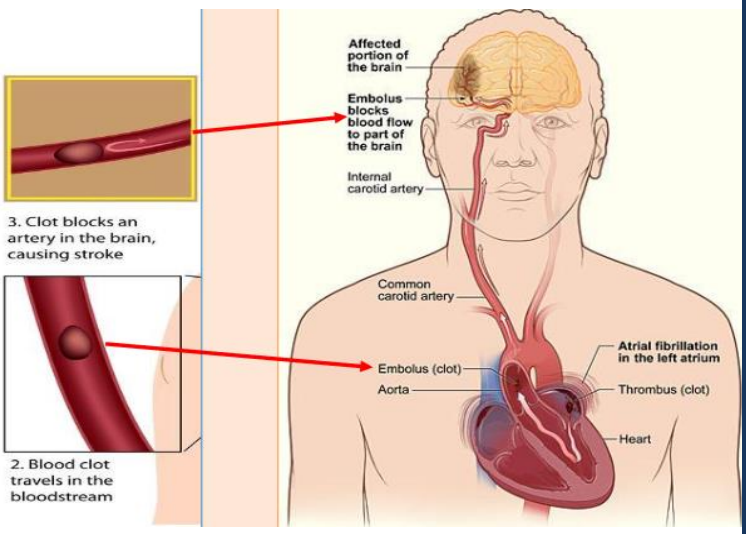
❖ Clinical consequences of pulmonary thromboembolism

- Mainly dependent on the size of the embolus and the exact location inside the pulmonary vasculature
- Most of these emboli are **very small** therefore would be totally asymptomatic. (but can lead to *organization* -this is not mentioned by the doctor-)
- **Larger** emboli cause symptoms including pulmonary infarction.
- Obstruction of **medium sized arteries** in the lung could lead to pulmonary hemorrhage.
- If more than 60% of pulmonary vessels are obstructed this could lead to **right ventricular failure (RVF)**, **Cardiovascular (CV) collapse** which ultimately leads to sudden death.
- If multiple small emboli keep forming over a long period of time, we call this "showers of emboli". This leads to **pulmonary hypertension** and **right ventricular failure.**

Arterial thromboembolism

- The origin of most arterial emboli are the heart chambers, from there it continues its flow with the direction of blood until it reaches its target, which could be any kind of tissue that receives blood from an arterial circulation.
- Target of arterial thromboembolism could be any organ that has arterial supply, but the major targets are:
  - Lower limbs (75%), Brain (10%), Intestines, Kidneys, Spleen. Etc.

- The figure to the right illustrates a thrombus formed inside the left atrium that fragments and sends an emboli, which goes with the direction of blood into the left ventricle and then through the aorta reaching anywhere in the arterial circulation. In this example, it goes through the common carotid -> internal carotid -> brain vasculature -> leading to occlusion of artery causing ischemic necrosis (stroke).



❖ Causes of Arterial thromboembolism

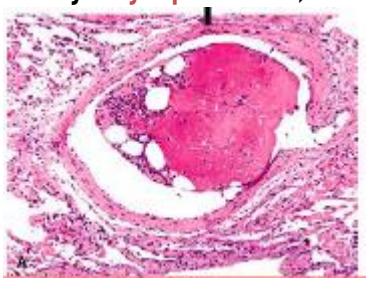
- Most of the time (80%), it is due to intracardiac mural thrombi (inside the heart).
- There are numerous causes for such thrombi (intracardiac). These include:
  - Left ventricular failure.
  - Left atrial dilatation.
  - Presence of complicated ulcerated atherosclerotic plaque.
  - Aortic aneurysm.
  - Valve vegetation. etc.
- The remaining 20% originate from arteries like the aorta for example.

Fat embolism

- This is the second type of emboli according to the composition of the embolus.
- Fat globules enter the circulation and go to different tissue targets, leading to occlusion of blood vessels and ischemic necrosis.

❖ Causes

- Most commonly due to Skeletal injuries such as fractures of long bones. Almost 90% of skeletal injuries result in fat embolisms but they are usually asymptomatic, only 10% or less have actual clinical findings called the fat embolism syndrome (will be discussed later).
- Rarely due to adipose tissue injury such as the massive fat necrosis happening after acute pancreatitis .



❖ Results

- Mechanical obstruction of blood vessels and ischemia.
- Release of free fatty acids from fat globules, which could cause local toxic injury to endothelial cells.

❖ Fat embolism syndrome

The term is used to describe clinically significant manifestations resulting from fat embolism.

Symptoms don't instantaneously appear after a bone injury. They're usually delayed and take 1-3 days to appear.

❖ Characteristics

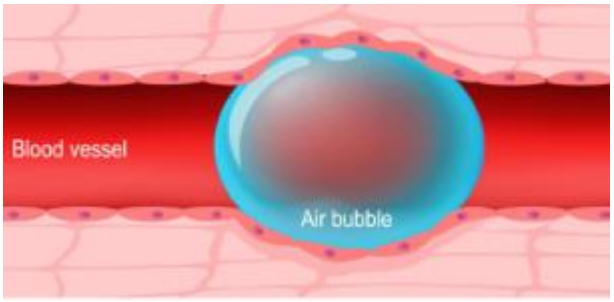
- Pulmonary insufficiency (rapid breathing and shortness of breath).
- Neurologic symptoms (mental confusion, lethargy, coma).
- Petechial rash (pinpoint rash, found on chest, head and neck area due to bleeding under skin).
- Fever.
- Anemia.
- Thrombocytopenia.
- Death in 10% of the cases with this syndrome.

Air embolism

This is the third type of emboli according to the composition of the embolus.

An air bubble is formed and is travelling inside the circulation. It can have a mechanical effect like any other thromboemboli, therefore it can lead to vascular occlusions and clinical symptoms.

❖ Causes



- Surgical and obstetric procedures,
- Vascular catheterization,
- Traumatic chest wall injury,
- Decompression sickness: it occurs in scuba deep-sea drivers and mainly due to nitrogen.

❖ Decompression sickness (nitrogen embolism)

Due to differences in atmospheric pressure and underwater pressure, when the diver goes deep into the sea, Nitrogen moves from high pressure in the lungs into the blood which has lower pressure and it gets dissolved there.

Now, if the diver quickly swims up to the surface, this wouldn't let nitrogen (which is now dissolved in blood) leave the blood and go to lungs resulting in painful air bubbles.

Instead, a professional diver would slowly swim up to the surface, giving enough time to the nitrogen dissolved in his blood to return back to the lungs, where it can be breathed out.

❖ Clinical consequences of air embolism

This depends on the *amount* of air that has been trapped inside the circulation in the form of bubbles. Based on that, it might be asymptomatic or could have serious complications.

- Painful joints due to rapid formation of gas bubbles within skeletal muscles and supporting tissues
- Focal ischemia in brain and heart
- Respiratory distress (chokes) occurs when massive amount of air enters the circulation. Leading to lung edema, hemorrhage, atelectasis, emphysema
- Caisson disease: another name for decompression sickness, where gas emboli in the bones leads to multiple foci of ischemic necrosis, usually in the heads of femurs, tibias, and humeri.

Amniotic fluid embolism

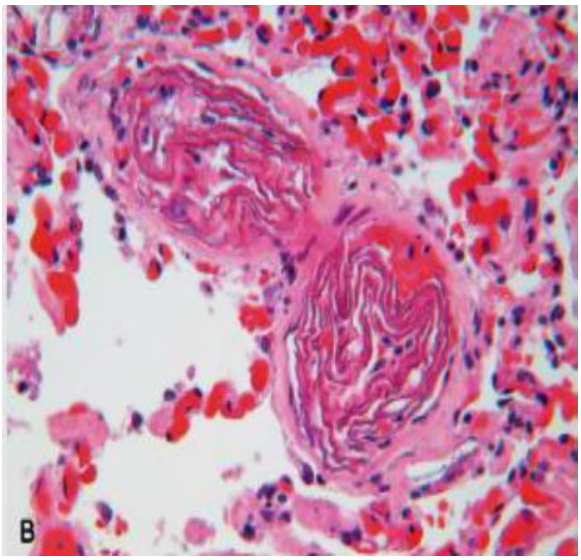
- It occurs as a result of the infusion of amniotic fluid into maternal circulation via tears in placental membranes and rupture of uterine veins.
- This is the fourth type of emboli according to the composition of the embolus.
- It is a very rare complication of labor.
- It has high mortality rate => around 20% - 40%.

❖ Symptoms

- Mechanical effects of vascular occlusion as in any other emboli.
- Associated with unique manifestations that are all related to DIC (Disseminated intravascular coagulation).
- Sudden severe dyspnea, cyanosis, ARDS, and hypotensive shock, followed by seizures, DIC and coma. These symptoms arise due to the allergic reactions that take place against the fetal components.

❖ Microscopic findings seen upon autopsy

- In this picture, we are looking into the pulmonary arterioles from the autopsy of a mother dying because of amniotic fluid embolism
- Presence of fetal components within the maternal pulmonary microcirculation is diagnostic of this condition, and by "fetal components" we mean **fetal squamous cells** and other components such as **lanugo hair**, **fat**, **keratin**, **mucin** Etc..
- In the image to the right, we can see keratin and fetal squamous cells.



- Note that this condition affects maternal and not fetal circulation.

Infarction

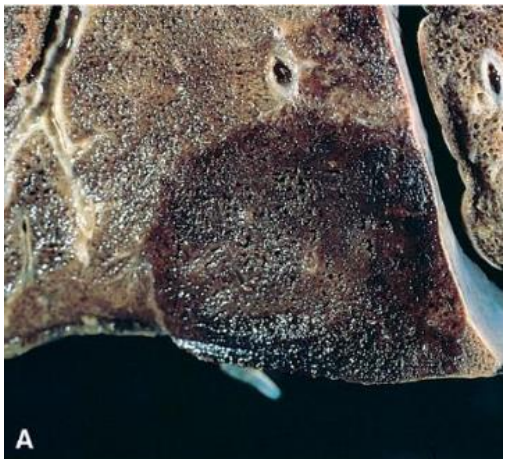
- The complications of thromboembolisms are mainly attributed to the ischemic necrosis of the tissue.
- Infarcts are the hallmark of tissue ischemia
- An infarct is an area of ischemic necrosis caused by the occlusion of arterial supply or venous drainage in a tissue.
- 99% of infarcts result from thrombi/emboli, but there are other mechanisms:
  - Vasospasm,
  - Extrinsic compression of the vessel (by a tumor for example),
  - Vessel twisting (examples include testicular torsion, volvulus),
  - Traumatic vessel rupture.

❖ Morphology of infarcts

- Infarcts may be either *red* (hemorrhagic, full of blood) or *white* (anemic), depending on the *amount of blood* left in the necrotic tissue. They also could be either septic or bland.
- Tend to be *wedge* shaped where the occluded vessel is at the apex and the periphery of the organ forming the *base*.
- Margins of the infarct tend to become better defined with time.
- histologic *hallmark* of infarction is **ischemic coagulative necrosis**. The **brain** here is an exception as it undergoes **liquefactive necrosis**.
- most infarcts are eventually replaced by *scar* tissue.

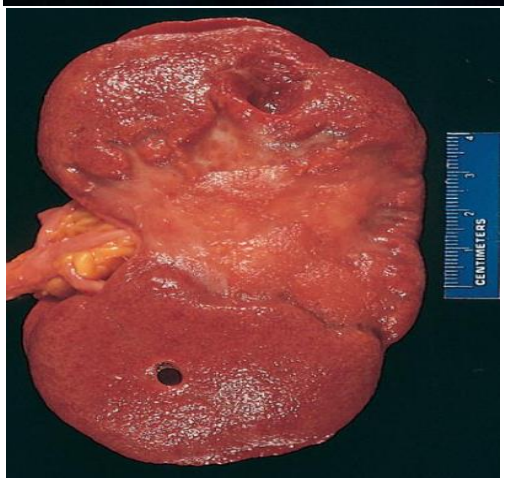
❖ Red infarcts

Here the necrotic tissue is filled with blood (like the picture on the right representing lung tissue with red infarct)and it occurs in the following scenarios:



- Venous occlusions (e.g. ovarian torsion).
- Loose tissues (e.g. lung) that allow blood to collect in the infarcted zone.
- Tissues with dual circulations, because one vessel is occluded while the other keeps the blood going to the necrotic area (e.g. lung and small intestine).
- Previously congested tissues because of sluggish venous outflow.
- When flow is re-established to a site of previous arterial occlusion and necrosis.

❖ White infarcts



Here, there is little to no amount of blood in the necrotic tissue, it occurs in:

- Arterial occlusions.
- Solid organs (such as heart, spleen, and kidney).

The image to the top-right shows white infarction in spleen, while the one on the bottom-right shows a white infarct in the kidney replaced by a fibrotic scar tissue.

- Remember that red and white infarcts are new descriptions of the color of the infarct. However, they both represent ischemic tissue necrosis.

❖ Septic infarctions

It is an area of infarcted tissue where the infarct is superimposed by an infection. examples:

- Infected vegetations on the cardiac valve.
- Microbes seed an area of necrotic tissue.
- Infarct is converted into abscess with a greater inflammatory response.

❖ Factors that influence development of an infarct

- Nature of vascular supply (whether the occluded vessel is an artery or a vein)
- Rate of occlusion development (presence of collateral circulation)
- Tissue vulnerability to hypoxia, some tissues are more resistant to hypoxia while other are very vulnerable. For example:
  - Neurons undergo irreversible damage within 3 to 4 minutes of ischemia.
  - Myocardial cells die after only 20 to 30 minutes of ischemia
- Oxygen content of blood

"That which does not kill us makes us stronger" -Friedrich Nietzsche

Good luck