Subject: CVS- PATHOLOGY

Topic: EMBOLISM AND INFARCTION

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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: (according to composition)

1.Thromboembolism: 99% (from dislodged thrombus)

2. Fat embolism

3. Air/ Nitrogen embolism

4. Amniotic fluid embolism

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*** thrombus is a blood clot that occur inside the cardiovascular system.**

*** thromboembolus is a piece that is dislodged from the original thrombus and is now traveling inside the circulation, it produces its own clinical symptom.**

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Types: (according to SIDES OF CIRCULATION)

1. VENOUS: ***Origin of most venous emboli = lower limbs... *Target of most venous emboli = lungs**

   *** **thrombus inside the venous side of circulation >> fragmenting >> venous embolus >> travels along with the blood >> the right side of the heart >> pulmonary arteries >> lungs.**

   venous emboli occur specifically in the deep veins, causing what is known as **deep vein thrombosis(DVT)**

2. ARTERIAL (SYSTEMIC)**: *Origin of most arterial emboli = heart chambers (intracardiac mural thrombi)... Target of most arterial emboli = lower limbs 75% then brain 10%**

-The clinical significance of a thromboembolism exactly the same as a thrombus:

- Emboli result in partial or complete vascular occlusion.
- The consequences of thromboembolism: ischemic necrosis (infarction) of downstream tissue
- the consequence of *venous emboli* depends on the size and exact location inside the lung

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TERMS>>>

***SADDLE EMBOLUS:** large embolus occluding the bifurcation of pulmonary artery trunk (fatal)> leading to loss of blood supply to the lungs.

***PARADOXICAL EMBOLUS:** Passage of embolus from venous to systemic circulation through ASD or VSD>>this passage is 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.)

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CLINICAL CONSEQUENCE OF PULMONARY THROMBOEMBOLISM :

1. 60%- 80% pulmonary emboli are clinically ***asymptomatic*** (small)> Organization
2. If large> ***Pulmonary infarction***
3. 60% of pulmonary vessels are obstructed>>***RVF, CV collapse >> Sudden death***
   -right ventricular failure (RVF)
4. obstruction of medium sized arteries > ***Pulmonary hemorrhage***
5. If **multiple** emboli (showers of emboli) over a long time>***Pulmonary Hypertension and right ventricular failure***

Fat embolism:

Causes:

1. Skeletal injury (In skeletal injury, fat embolism occurs in 90% of cases, but only 10% or less have clinical findings = *Fat embolism syndrome*).
2. Adipose tissue Injury.

Results:

1- Mechanical obstruction of vessels

2- free fatty acid release from fat globules >>>local toxic injury to endothelium.

Fat embolism 'syndrome' is characterized by:

Pulmonary Insufficiency, Neurologic symptoms, petechial rash,Fever, Anemia and Thrombocytopenia.

- Death in 10% of the case

- Symptoms appear 1-3 days after injury

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

causes:

1. Surgical and obstetric procedures
2. Vascular catheterization
3. Traumatic chest wall injury
4. Decompression sickness: in Scuba deep-sea divers ((nitrogen))

****AIR EMBOLISM- CLINICAL CONSEQUENCE:** Painful joints, Focal ischemia in brain and heart, Respiratory distress, Caisson disease.

Amniotic fluid embolism (rare complication of labor): infusion of amniotic fluid into **maternal (NOT FETAL)** circulation via tears in placental membranes and rupture of uterine veins.

****Symptoms:** sudden severe dyspnea, cyanosis, ARDS, and hypotensive shock, followed by seizures, DIC and coma.

=DIC: disseminated intravascular coagulation=

****Microscopic Findings upon autopsy:** fetal squamous cells, lanugo hair, fat, mucin …..etc within the maternal pulmonary microcirculation. (**Diagnostic**)

infarct = an area of **ischemic necrosis** caused by occlusion of arterial supply or venous drainage in a tissue, 99% of infarcts result from thromboi/emboli…other mechanisms: Vasospasm, extrinsic compression,vessel twisting and traumatic vessel rupture.

Infarct: red or white/ septic or bland/ wedge shaped/ margins are better defined with time/ histologic hallmark of infarction is **ischemic coagulative necrosis** (The brain is an exception (liquefactive necrosis))/ replaced by scar.

FACTORS THAT INFLUENCE DEVELOPMENT OF AN INFARCT:

- nature of vascular supply
- rate of occlusion development
- tissue vulnerability to hypoxia (Neurons damage = 3 to 4 minutes of ischemia/ Myocardial cells die =20 to 30 minutes)
- oxygen content of blood

RED INFARCTS:

(1)venous occlusions (2) loose tissues (3) tissues with dual (4) previously congested tissues because of sluggish venous outflow (5) when flow is re-established to a site of previous arterial occlusion and necrosis

WHITE INFARCTS:

1) arterial occlusions 2) solid organs

Septic infarctions: - occur when infarct is superimposed by infection.

**- infarct is converted into abscess with a greater inflammatory response