Embolism

Q definition, types, etiopathogenesis, morphology, complications.

definition.

Q) An embolus is an intravascular solid, liquid, or gaseous mass that is carried by the blood to a site of distant from it's point of origin.

- Vast majority of emboli derive from a dislodge thrombus - hence the term — thromboembolism

TYPES

① pulmonary thromboembolism
② systemic thromboembolism.
③ Fat embolism
④ Air embolism
⑤ Amniotic fluid Embolism.

pulmonary embolism (m/c)

Origin

→ 95% of cases → venous thrombi from deep leg vein proximal to popliteal fossa.

→ thrombi from lower leg → rarely embolize.

Question - H/o

if PE → bedridden patient suddenly due to DVT. developed dyspnoea

Fat embolism → fracture of femur & other long bone.

Systemic thromboembolism → pt with h/o chest pain, palpitation, dyspnoea - later develops
→ hemiplegia (or)
→ infarction of major organ (any)

in consequence of systemic embolization ↓ is ischemic necrosis (infarction).

for pulmonary embolism. ↓ ⇒ hypoxia, hypotension, right sided heart failure

pathophysiology

. thrombi {usually DVT} ↓ fragment. ↓ travel via venous system. ↓ right heart ↓ pulmonary arteries.

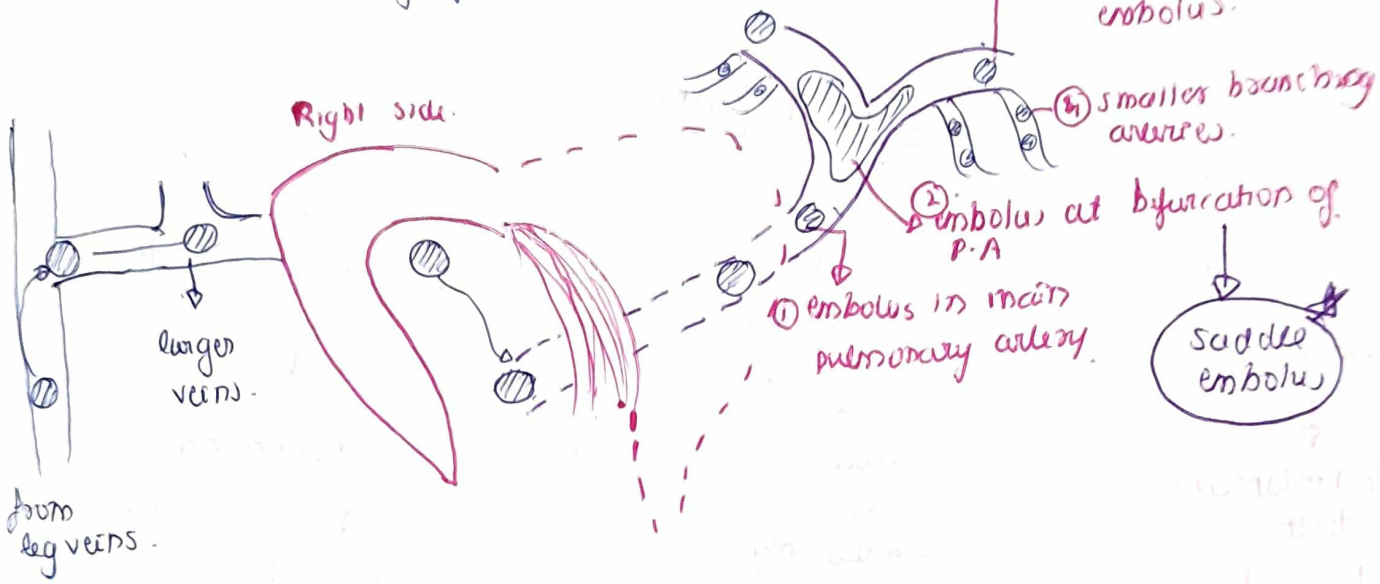
Sites of Embolus lodging.

① Occlude main pulmonary artery

② straddle the pulmonary artery bifurcation

③ pass out into smaller branching arteries.

④ left or right P.A embolus.



paradoxical embolism: Rarely Embolus bypass lung via cardiac defect → Enters systemic circulation.

Functional Consequences.

① most emboli are clinically silent
∵ they are small
↓
with time they organize & incorporate into vessel wall

② Sudden death, acute right heart failure, {cor pulmonale} or collapse.
} }
if emboli obstruct 60% or more of pulmonary circulation.

③ Embolic obstruction of medium-sized arteries and subsequent vascular ~~damage~~ rupture causes pulmonary hemorrhage
But no infarction ✗ due to dual blood supply to lungs.
↓ ↓
But in case of ♡ Failure and reduced Bronchial artery supply
↓
infarct.

④ Embolic obstruction of small end arteriolar pulmonary branches. does not often produce hemorrhage or necrosis.

⑤ multiple emboli overtime may cause pulmonary HTN & Right ventricular failure.

Clinical features

large PE
- Instantaneous death
- If patient survives
  - Severe chest pain
  - dyspnoea
  - shock

{mimic acute MI}

Small P.E
- Silent or
- Induce only transient chest pain & Cough.

In rest of the cases.
- dyspnoea.
- pleuritic pain
- cough
- calf/thigh swelling, pain.

Diagnosis
- d-dimer: screening.
- CT pulmonary Angiogram → definitive.
- for DVT → duplex USG.
- chest x-ray – wedge shaped infiltrates.

Rx
- anticoagulants
- thrombolytics.

Complications
- multiple small emboli
  - pulmonary Hypertension & Cor pulmonale.

preventive measures

Prevention
- Early ambulation post-op & postpartum.
- Elastic stockings.
- Anticoagulants in high risk patients.
- mandatory stocking – bed ridden patients.

Systemic thromboembolism (UQ) Essay.

due to atrial fibrillation.

→ H/o - chest pain, dyspnoea, palpitation.
↓
hemiplegia developed.

Q) Explain pathogenesis of dyspnoea.
Q) Why caused hemiplegia.

• outcome → tissue infarction

Systemic Thrombo Embolism / Arterial Emboli

80% arises from.
↓
Intra cardiac mural thrombi.

- (2/3) – arises in relation to left ventricular wall defect
- (1/4) – arises due to left atrial dilatation & atrial fibrillation.
- remaining –
  - aortic aneurysm
  - atherosclerotic plaque
  - valvular vegetation
  - Venous thrombi {paradoxical}

10-15%
↓
unk origin.

venous Emboli

↓
deposits in lung. {mainly}

arterial ~~thrombus~~ Embol

↓
travel to various sites
↓
m/c – lower extremity (75%)
& Brain (10%)
other: kidney, spleen, intestine.

pathogenesis of dyspnea in STE

① Cardiac origin of Emboli

LV infarct, Left atrial thrombus.
↓
↓ CO
↓
Congestive heart failure.
↓
pulmonary congestion
↓
Edema.

② Paradoxical Embolism

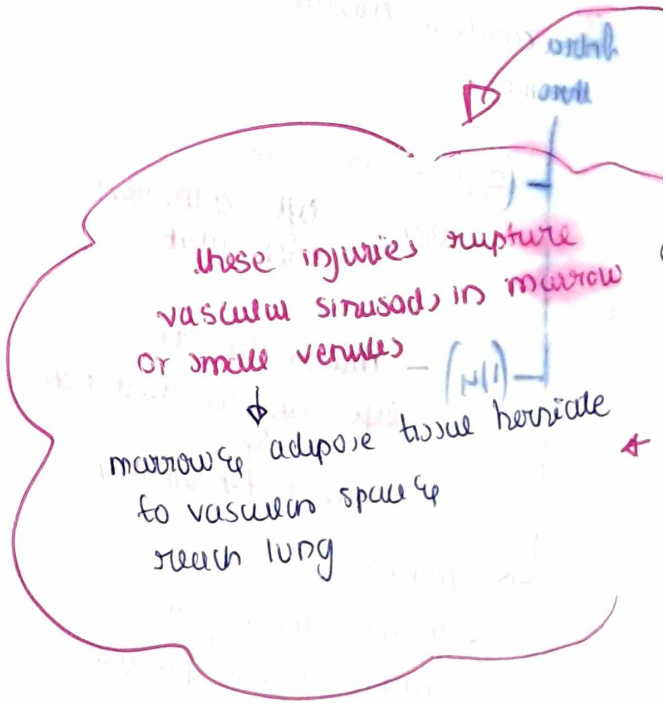
- embolus from venous
↓
arterial system
↓
via patent foramen ovale.
↓
blood pulmonary arteries.
↓
dyspnoea.

Hemiplegia in STE

Likely cause: Cerebral Infarction.
↓ due to
Embolus lodging in middle cerebral artery

pathogenesis.

- Embolus from LV thrombus, LA thrombus, aortic plaque.
↓
via carotid artery.
↓
MCA. : supplies motor cortex controlling opp face & upperlimb.
↓
Infarction
↓
Sudden onset of hemiplegia → paralysis of opposite side.



* autopsy finding
* fat emboli demonstration

Fat Embolism

UQ essay.

{H/o femur fracture}.
↓
collapse & died.

Q) diagnosis?

Q) etiopath

Q) morphological & postmortem findings.

* Fat Embolism refers to the presence of microscopic fat globules (sometimes with associated hematopoietic Bone marrow) in vasculature.
after fracture of long bones
or rarely in the setting of soft tissue trauma or burns.

Etiopathogenesis

Etiology

- long bone fractures (m/c)
- severe soft tissue trauma/burns
- vigorous CPR
- occurs in 90% of severe skeletal injuries. {but <10% have CF}.

pathogenesis

- mechanical obstruction.
- Biochemical injury.

pathogenesis

Mechanical obstruction:
- fat micro-emboli and associated red cell & platelet aggregates can occlude pulmonary & cerebral microvasculature.

Biochemical injury.
release of FFA from fat globules
↓
local toxic injury to Endothelium
↓
platelet ⊕ & granulocyte recruitment
↓
& exacerbate the situation.

* term "fat embolism syndrome" is used for minority of patients who become symptomatic

Symptoms

① pulmonary insufficiency
- typically presents 1-3 days after injury
↓
sudden onset of tachypnoea
dyspnoea
tachycardia

② Neurological symptoms
- Irritability
- restlessness
- delirium
- coma.

③ thrombocytopenia
- platelet adhere to fat globule
- splenic sequestration.

④ Anaemia.
- due to red cell aggregation & hemolysis.

Air Embolism (S.N)

definition

air embolism refers to the presence of gas bubbles within circulation, which can coalesce into frothy masses, obstruct vascular flow, and cause distal ischemic Injury.

Etiopathogenesis

Etiology

Entry of Air

air can Enter the vasculature when there is a negative pressure gradient

Small amount of air

- coronary artery during bypass.
- cerebral vessels during Neurosurgery in Sitting position.

→ Dire Consequences
- → Mental Impairment (CF) ✓
- → Sudden onset coma. (CF) ✓

large volume >100 cc.

↓

Necessary to produce C/F in pulmonary circulation

⇓

during obstetric / laparoscopy procedures.

⇓

OR consequence of chest wall injury.

~ 350 - 500ml

↓

fatal.

(CF) respiratory distress ✓

Decompression Sickness

definition

- Occurs when Individuals experience sudden decrease in atmospheric pressure, leading to gas bubble formation in blood & tissues.

Causes

- seen in Scuba divers
  deep sea divers.
  underwater construction workers

mechanism

air is breathed
* at high pressure → ↑ amnt of gas especially ↑ N_2 dissolves in blood & tissue
* rapid ascent (depressurization) → N_2 comes out of solution → forming gas bubbles.

Types & features

① Acute decompression Sickness.

① The "Bends"

painful condition
↓ due to.
rapid formation of gas bubbles within skeletal muscle and supporting tissue in & about joints.

② The "Chokes".

In lungs.
↓
bubbles in pulmonary vasculature
↓
causes
↓
- pulmonary edema
- Hemorrhage.
- focal atelectasis or emphysema.

↓
leads to Respiratory distress.

Treatment - Acute dec sickness.

{for acute I think ?}

- high pressure oxygen chamber
  → forces gas bubbles Back into solution.

+ Subsequent Slow decompression.
  → allows resorption & Excretion of gases.
  → prevents bubble formation

CHRONIC DECOMPRESSION SICKNESS.

CAISSON DISEASE

* persistent gas emboli in skeletal system.
  ↓
  - leads to multiple foci of ischemic necrosis.

M/C site → femoral head, tibia, humerus.

Amniotic fluid Embolism

Definition

- It is the 3rd m/c cause of maternal mortality worldwide.

⇒ It is an ominous complication of labor or the immediate postpartum period.

⇒ mortality rate can be as high as 80%.

Pathogenesis

{features of AFE differ from PE as it occurs due to.}

much of Morbidity & mortality is due to.

- Biochemical activation of:
  - coagulation factors.
  - innate immune system.
- ⊕ of innate immune system
- release of vasoactive substance. {rather than mechanical obstruction by amniotic debris}

Clinical feature.

Onset is characterized by:

- Sudden Severe dyspnea.
- Cyanosis
- Shock.

followed by Neurologic Impairment:

- Headache
- Seizures
- Coma

If the patient survives the initial crisis:

→ pulmonary edema.

→ DIC.

Vasoactive substances causes.

1. Acute pulmonary HTN
2. Right heart failure.
3. Left Heart failure
4. Pulmonary edema
5. diffuse alveolar damage.

Autopsy findings:

1. Squamous cells shed from fetal skin
2. Lanugo hair
3. Fat from vernix caseosa
4. mucin from fetal GIT / RT

→ in maternal pulmonary microvasculature.

Amniotic fluid Embolism

Definition

- It is the 5th m/c cause of maternal mortality worldwide.

⇒ It is an ominous complication of labor or the immediate postpartum period.

⇒ mortality rate can be as high as 80%.

Pathogenesis

{features of AFE differ from PE} as it occurs due to.

much of Morbidity & Mortality is due to.

- Biochemical activation of:
  - coagulation factors.
  - innate immune system.
- ④ of innate immune system
- release of vasoactive substance. {rather than mechanical obstruction by amniotic debris}

Underlying cause

↓

Effusion of amniotic fluid or fetal tissue.

↓

into maternal circulation.

↓

via

↓

- tear in placental membrane
- Rupture of uterine veins.

Clinical feature.

Onset is characterized by:

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↓

in maternal pulmonary microvasculature.

Other findings:

- marked pulmonary edema.
- diffuse alveolar damage
- fibrin thrombi in many vascular bed due to DIC.

Q). 50y - altered sensorium, cyanosis, oliguria, weak rapid pulse, tachypnea, cool clammy extremities.

a) provisional diagnosis ?

✗ Q) etiopathogenesis. what u mean? of what

Q) mention organs involved.

discuss pathology in lung.

Q) mention 4 types of shock?

Q) morphological changes in organs.

Q) septic shock - pathogenesis.

⇒ Shock is defined as a state of systemic hypoperfusion due to ~~what~~ - diminished cardiac output. or reduced effective circulating blood volume → impairs tissue perfusion and leads to cellular hypoxia.

Types

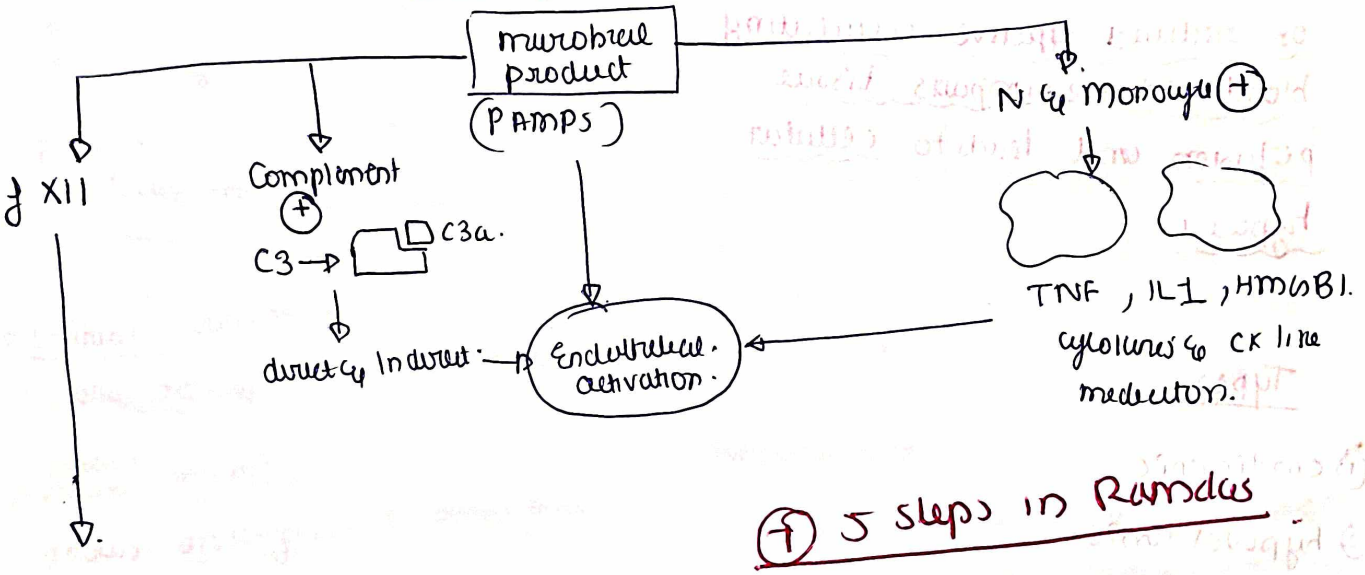
① cardiogenic.
② hypovolaemic.
③ Septic
④ Neurogenic & anaphylactic. shock

	Cardiogenic.	hypovolaemic	anaphylactic & Neurogenic.
Mechanism	• failure of myocardial pump due to intrinsic myocardial damage; Extrinsic compression / obstruction to outflow.	• fluid loss ↓ • inadequate blood or plasma volume. eg. • Haemorrhage • vomiting • diarrhoea • Burns • trauma.	Spinal cord injury to ~~RS~~ anesthetic accident ↓ N.S IgE mediated HSR ↓ A.S. ↓ vasodilation ↓ hypotension. ↓ tissue hypoperfusion.
Examples	: MI, Arrhythmia valvular rupture. Cardiac tamponade PE.		

Septic Shock.

Septic shock is caused by host responses to. Bacterial, viral or fungal injection;
It is a systemic Inflammatory condition characterized by endothelial cell activation, tissue edema, DIC. and metabolic derangements. that often lead to organ failure and death.

pathogenesis



(+) 5 steps in Ramdas

figure in Robbins – Screenshot -

Basic pathogenesis

- massive outpouring of Inflammatory mediators from Innate & adaptive Immune cells.

- peripheral vasodilation
- vascular leakage
- venous blood pooling.
- Endothelial activation

✓ DIC
WBC mediated injury.

- Tissue hypoxia.
- persistent
- death.

Morphology of Organs.

→ Changes in Cardiogenic / hypovolemic Shock.

Adrenal.

- Lipid depletion in cortical cell:
- due to conversion of relatively inactive vacuolated cell to metabolically active cells.
- active cells used it to make steroids.
- focal hemorrhage - in adrenal cortex. {in severe shock}
- Massive hemorrhagic necrosis of entire adrenal gland.

↓

Waterhouse - Friderichsen Syn.

↓

as with meningococcal septicemia.

Kidney

- Acute tubular necrosis.

{acute renal failure}.

GROSS:

Enlarged, swollen, congested, pale cortex.

- Cut section - blood pooling in outer section of medulla.

MC

- acute tubular necrosis - dilated tubules, epithelial necrosis.
- pigmented cast - {from Hb, myoglobin}.
- Interstitial edema & mononuclear infiltration.

Lung

- Lungs are relatively resistant to hypovolemic shock & hypoxic injury

GROSS

- But in shock due to bacterial sepsis/ trauma, it shows

↓

diffuse alveolar damage

↓

that can lead to ARDS.

GROSS

- Firm, congested lungs
- Cut surface: oozes frothy fluid

MC

- Edema - Interstitial & alveolar.
- Necrosis - of alveolar epithelium & endothelium.
- Intravascular microthrombi
- Hyaline membrane

Heart

GROSS: petechial hemorrhages on epicardium & endocardium.

MC

- myocyte necrosis: Light microscopy ✓
- contraction bands → Microscopy ✓

Liver, Brain, GIT

↓

In Ramadan table.