THROMBOSIS

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INTRODUCTION

Three primary influences predisposes to thrombus formation (Virchow's triad);

- endotherial injury
- stasis or turbulence of blood flow
- hypercoagulable state

Endothelial injury

- Important in thrombus formation in the heart and in the arterial circulation in cases of myocardial infarction, ulcerated atheromatous plaques and in vasculitis.
- Endothelial damage results in the exposure of the subendothelial collagen leading to platelet adhesion and exposure of the tissue factor and the depletion of prostacylin.

Alterations in the normal blood flow

- Turbulence causes endothelial injury and dysfunction.
- Stasis and turbulence;

- disrupts laminar flow and brings platelets into contact with the endothelium
- prevent dilution of activated clotting factors
- retards inflow of coagulation inhibitors
- promotes endothelial cell activation

Hypercoagulability

- Can be divided into primary and secondary disorders;
- *Primary (genetic)*

- Mutations in factor V
- Antithrombin III deficiency
- Fibrinolytic defects
- Protein C or S deficiency
- Homocysteinemia
- Variations in the prothrombin levels

SECONDARY(ACQUIRED)

- Immobilization
- Myocardial infarction
- Tissue damage
- Malignancy
- Prosthetic cardiac valves
- DIC
- Heparin- induced thrombocytopenia

…SECONDARY(ACQUIRED)

- Lupus anticoagulant syndrome
- Atrial fibrillation
- Cardiomyopathy
- Nephritic syndrome
- Hyperestrogenic states
- Oral contraceptive use
- Sickle cell anaemia
- smoking

WHOM TO CONSIDER FOR THROMBOPHILIA INV.

- Thrombosis occurring at a young age (i.e. < 45 yrs)
- Idiopathic VTE
- Recurrent VTE
- Thrombosis at an unusual site
- Family history of VTE or of inherited prothrombotic disorder
- Warfarin-induced skin necrosis
- Recurrence/extension of thrombosis while adequately anticoagulated.

CANCER AND VENOUS THROMBOEMBOLIC DISEASE

- There is a very strong association between cancer and venous thromboembolism. This association was first suggested in 1865 by Dr. Armand Trousseau, who later developed unexplained DVT and then died of a gastric carcinoma.
- Active cancer accounts for almost 20% of all new venous thromboembolic events occurring in the community.

- Patients presenting with unprovoked venous thromboembolism (VTE) have a 10% risk of developing cancer within the next two years.
- Occult malignancy is 3-4 times higher in patients who present with idiopathic thromboembolic disease versus patients with a secondary venous thromboembolic event.
- Patients who receive a diagnosis of cancer at the same time or within one year of an episode of a venous thromboembolic event have a shorter life expectancy than patients with cancer who do not have VTE.

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- Most common cancers in patients with VTE are lung, breast, colorectal and prostate. Certain malignancies are particularly associated with a high risk of venous thromboembolic disease. These include malignant brain tumours and adenocarcinoma, including ovary, pancreas, colon, stomach, lung and kidney.

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Risk factors for venous thromboembolic disease in patients with cancer include;

- immobility
- use of central venous access devices
- hormonal therapy
- chemotherapy
- surgery

THROMBOSIS IN PREGNANCY

- Pregnancy increases the risk of venous thromboembolism (VTE) but the true incidence is unknown. The risk of VTE continues in the post-partum period and is probably higher than during pregnancy.

EMBOLISM

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

90% of all emboli are part of the dislodged thrombus.

Other forms of emboli include;

- fat
- air
- atherosclerotic debris
- tumour fragments bone marrow
- foreign bodies

PULMONARY THROMBOEMBOLISM

- In more than 95% of the cases the emboli originate from the deep leg vein above the knee which passes through the right side of the heart to the pulmonary vasculature.
- Depending on the size of the embolus it may occlude the main pulmonary artery, impact across the bifurcation or pass out into the smaller branching arterioles.
- An embolus may pass through an interatrial or interventricular defect to the systemic circulation.

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- Most pulmonary emboli (60-80%) are clinically silent because they are small.
- Sudden death, right heart failure (cor pulmonale) or cardiovascular collapse occurs when 60% or more of the pulmonary circulation is obstructed with emboli.
- Multiple emboli over time may cause pulmonary hypertension with right heart failure.

SYSTEMIC THROMBOEBOLISM

Refers to an emboli traveling within the arterial circulation. 80% arise from the intracardial mural thrombi

The remainder originate from the;

- Aortic aneurysm
- Ulcerated atherosclerotic plaques
- From valvular vegetations
- Paradoxical emboli
- The major site for arterial embolization include the lower extremities and the brain.

FAT EMBOLISM

- Occur after fracture of the long bones, in soft tissue trauma and burns.
- Fat embolism syndrome begins 1 to 3 day after injury and it is characterized by tachypnea, dyspnoea, tachycardia, irritability, restlessness and petechial rush in the nondependent areas.
- Pathogenesis of fat emboli syndrome involves both mechanical obstruction and biochemical injury.
- Free fatty acids released from neutral fats are toxic to the endothelium.

AMNIOTIC FLUID EMBOLISM

It is a complication of labour and the immediate postpartum period.

Has a mortality rate in excess of 80%.

Onset is characterized by sudden severe dyspnoea, cynosis, and hypotensive shock followed by seizures and coma.

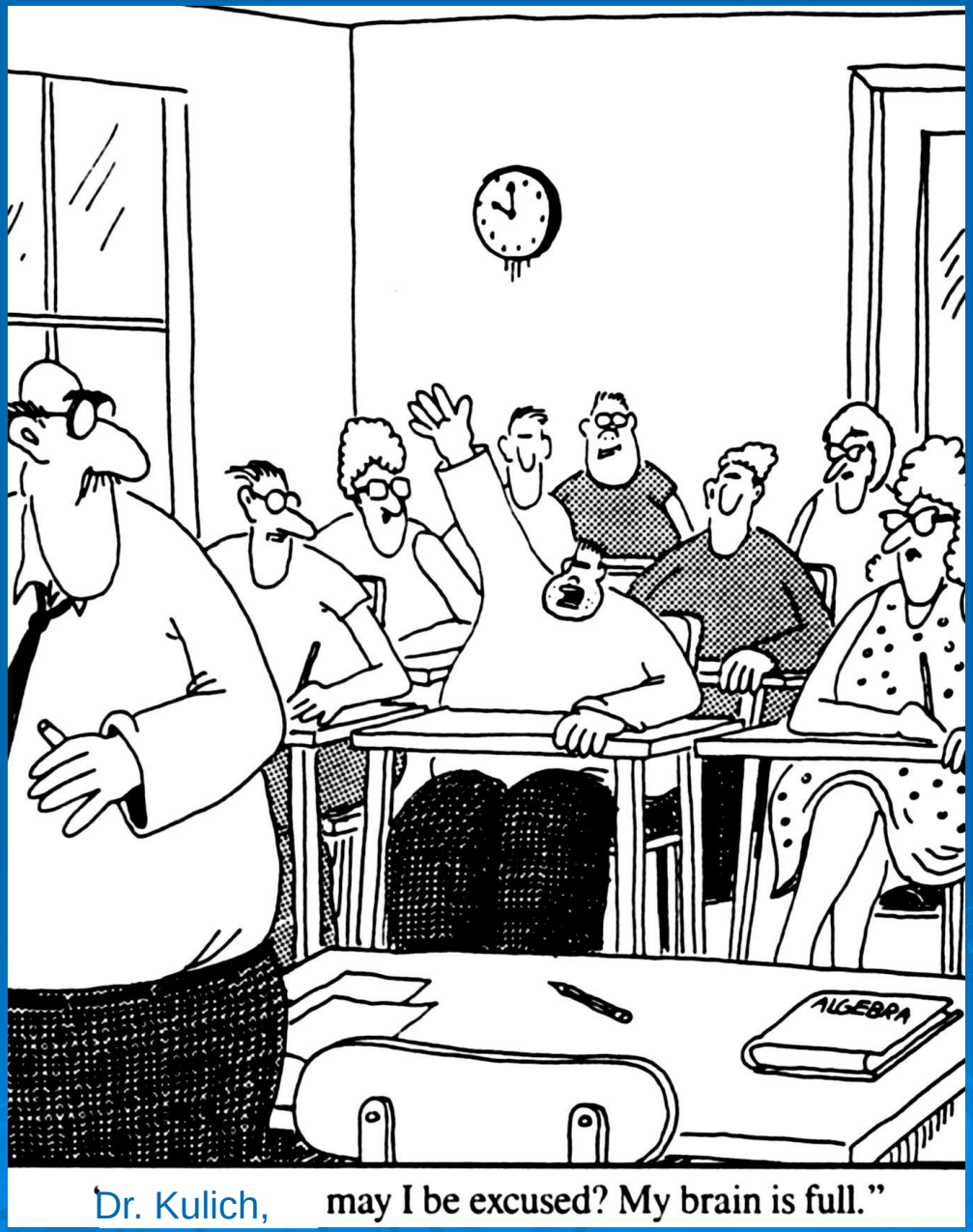
The classic findings are presence in the pulmonary microvasculature of;

- epithelial squamous cells
- lanugo hair
- fat
- mucin

AIR EMBOLISM

- Air may enter the circulation during obstetrics procedures and in chest wall injuries.
- Air in excess of 100cc is required to have a clinical effect.
- Decompression sickness occur when individuals are exposed to sudden changes in the atmospheric pressure.
- A more chronic form of decompression sickness is called caisson disease where persistent gas emboli in the skeletal system leads to multiple foci of ischaemic necrosis.

Questions?



Modified from The PreHistory of the Far Side: A 10th Anniversary Exhibit. 1989, p. 233. Andrews and McMeel

THANK YOU