Subject: Pathology

Topic: Thrombosis

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General information:

1- **CVS**: heart + blood vessels
2- **Circulation**: venous circulation + arterial circulation
3- **venous circulation**: it returns blood from tissues to the heart.
Venules >> medium veins >> large veins
4- **Arterial circulation**: from heart to the tissues.
Aorta>> smaller branches>> arterioles

- **Arterioles** and **Venules** connect inside tissues as capillaries (network of blood vessels)

- all types of blood vessels <u>are composed of</u>:
1-Tunica intima
2-Tunica media
3- Tunica adventitia
- both the heart and the blood vessels are covered from inside by the Endothelial cells

Layer of blood vessels from inside to outside

Tunica intima

(special type of Endothelium)

internal elastic lamina (special type of CT)

It gives thickness + contractility to the artery

Tunica Media

(smooth muscle cells)

+some elastic fibers and proteins

external elastic Lamina

Tunica adventitia

(connective tissue layer)

It contains **vasa vasorum**+ **Nervi Vasorum**

It supports the blood vessel from the outside

The major difference between arteries and veins is:

Tunica media: it's much thicker in artery than in vein.

So, artery have a good contractile ability

Hemostasis

it's the natural, physiological protective process against adverse consequences of blood loss after vessels injury.

It includes vascular spasm as well as recruitment and activation of certain constituents of the blood like platelets and fibrin in order to form the platelet plug.

pathological thrombosis

This happens whenever unnecessary blood clotting is activated

by the presence of at least one of 3 factors (together called **Virchow's triad**) sometimes 2 or 3 of these factors can be happening at the same time.

1-endothelial injury

2- normal blood flow

3- hypercoagulability

Under normal conditions

the endothelial cells will prevent or inhibit the process of coagulation inside the vascular space

(**basal or inactive state**)

(non-adhesive and non-thrombogenic surface)

Under abnormal conditions

-turbulence, hypertension, cytokines and bacterial products, cigarette smoke

(**activated state** or the **injury of the endothelial cell**)

increase the expression of certain pro-coagulants and adhesion molecules, and some pro inflammatory factors>> promoting coagulation.

Changes in the wall of blood vessels

1- Recruitment of smooth muscle cells into the Tunica intima.
2- smooth muscle cells will undergo mitosis
3- they will also elaborate extracellular matrix proteins
(thickening in the intima)

pathological effect>> is excessive thickening of the intima>> luminal stenosis>> blockage of the vascular flow.

Normal blood flow (laminar)

platelets will flow centrally in the vessel lumen and they will be separated from the surface of the endothelium by a slower moving clear zone of plasma

(fastest flow in the center)

Platelets are kept away from the endothelial cell surface to inhibit unnecessary coagulation.

turbulent blood flow

it leads to abnormal direction of the blood flow and sluggish movement of blood constituents>> increase the risk to develop unnecessary coagulation.

Causes:

1- Stenosis 2- dilation of BV

>> sluggish movement of blood>> formation of blood clots.

Stasis

- **major** factor in Venus thrombi formation.
- Stasis + turbulence can lead to:

1- Disrupt the normal blood flow.
2- prevent the dilution of the activated clotting factors.
3- retard the inflow of clotting factor inhibitors to the site of coagulation.
4- promote endothelial cell injury.

(**formation of the thrombus**)

Causes of stasis

1- Atherosclerosis
2- Aneurysms
3- Myocardial infarction
4- Mitral valve stenosis (atrial dilation)
5- Hyper viscosity syndrome.
6- Anything that leads to hypercoagulability of the blood

Blood hypercoagulability

(high tendency to develop blood clots)

- **Genetic(primary):** results from mutations in genes that are involved in coagulation
  (rare, most common mutation are factor five and prothrombin gene)
- **Acquired (secondary):** multifactorial + more complicated + (local or systematic).

1- **Arterial or Cardiac thrombi**:
   usually begin at sites of endothelial injury + superimposed on atherosclerotic plaque
2- **Venous thrombi:**
   are very common at sites of stasis or turbulence of blood flow
   (Most commonly are the lower extremity veins (90%))

Terms

1- **Lines of ZAHN:** laminations inside the lumen of the blood vessel (pale platelets and fibrin layers (alternating with darker erythrocyte rich layers) those lines are to distinguish between **Antemortem** (laminated) and **postmortem** (non- laminated) blood clots (don't contain lines of Zahn).

2- **Mural Thrombi**: thrombi inside the heart chambers or in the aortic lumen.

3- **Cardiac Vegetations**: overlying the heart valves (**infectious** > bacterial or fungal / **Non-infectious**> non-bacterial)

Fates of a thrombus

Resolution

removal of the formed thrombus

(perfect situation)

propagation of the thrombus

enlargement of the thrombus by accumulation of additional platelets, fibrin and red blood cells

(BV obstruction)

Thromboembolism

thrombi or fragments of the thrombi can dislodge which are transported elsewhere in the vasculature

organization

induce inflammation and fibrosis

might lead to complete occlusion of BV.

affects **chronic thrombi.**

recanalization

means that a new channel or a new lumen is developing inside or through the organized thrombus>>maintenance of certain amount of blood flow through the affected BV.

Mycotic aneurysm or superimposed infection

superimposed infection at the site of the thrombus formation.

Good luck