Ischemic Disease

Pathology Team

These are the companion notes
for the HANDOUTS given by Dr. Sufia Hussain
You can study from: Them + The Pathology Handouts

N.B. The" 🕮 "means it is an important MCQ

This was made by:
Dona Baraka - Nourhan Al-Shamma' - Dina Al-Kuhaimi - Sara Al-Husain.

In Cooperation with:
The Male's Pathology Team:
Raed Faraj – Abdullah Al-Faris – Aban Al-Bahebri – Muhammad Al-Harbi

Ischemic Heart Disease:

Mainly caused by "CORONARY Atherosclerosis" (stenosis in coronary arteries because of an atherosclerosis)
Stenosis: constriction or narrowing of flow.
Coronary: arteries that supplies the Heart

📖 Contributing factors of IHD:

Contributing Factors: the causes of it/ what makes it worst
(Hypertension: is a very important contributing factor of IHD**)**

Pathogenesis of IHD:

Stenosis is caused by: 75% or less of the lumen, is taken by "atherosclerotic plaque".

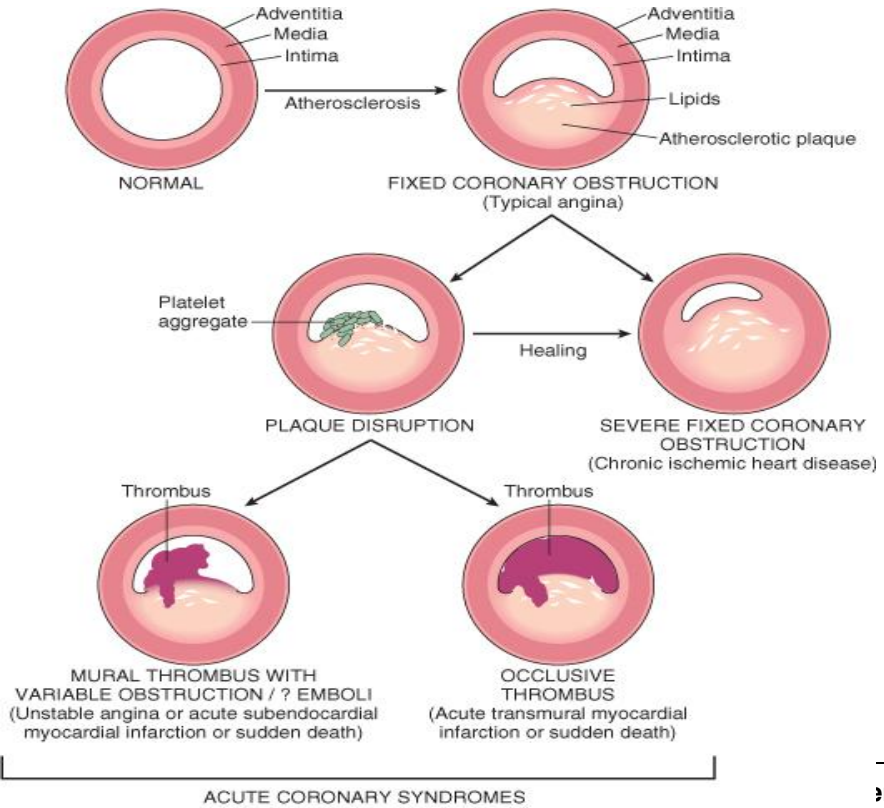
When plaque change (it rupture → hemorrhage in the atheroma → become a thromobosis)

Hypercoagulability (thrombophilia): the propensity to develop thrombosis due to an abnormality in the system of coagulation
Atheroma: the swelling of macrophages and fatty plaques (atherosclerotic tumor)
Thrombosis:
formation of a blood clot inside a blood vessel (due to injury to the endothelial lining of them or Hypercoagulabitiy)
Mural Thrombus: formation of a thrombus WITHOUT fully occlusion to the vessel wall.
Occlusive Thrombus: formation of a thrombus that occlude the vessel wall ENTIRELY.

Causing TOTAL occlusion (Occlusive thrombus) → it cause <u>Transmural MI</u>
Causing PARTIAL occlusion (Mural Thrombus) → it cause <u>subendocardial MI</u>

Transmural MI: infarction that involves WHOLE thickness of the cardiac muscle (from endo- to epicardium)
Subendocardial (non-mural) MI: infarction the involves ONLY the layer of the muscle adjacent to Endocardium

Thrombus in a coronary artery can <u>Embolize</u>
Emboli: a thrombus touring in circulation (could go to any where like the brain, lungs..etc)

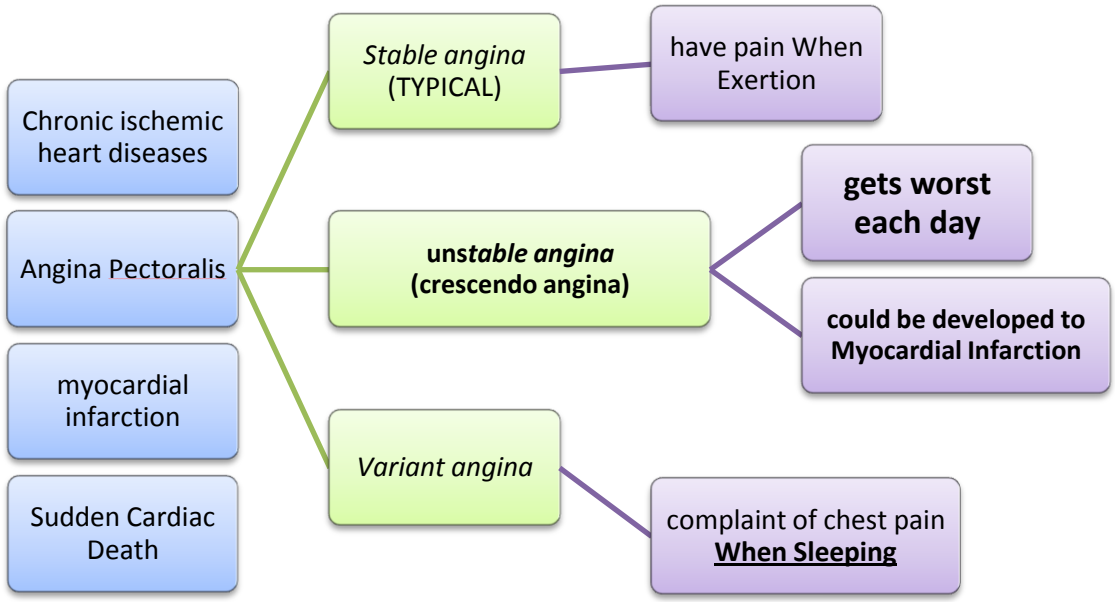


Stable Angina (classical):
not necessarily a rupture in the plaque (might be because of ↑ O_2 demand with exercise)
because it is RELIEVED by REST
Unstable Angina:
when sudden change in plaque's morphology occur (as described previously)
A sign of Myocardial Infarction

Spasm: vasoconstriction (ischemia).

Types of IHD:

Why is- We classify IHD?? Because each type has its own treatment way.



Myocardial Infarction

It's a Coagulative Necrosis (neither fibrinoid in blood vessels **or Liquefactive** in brain and lung**)**

The death of cardiac muscle – resulting from **ischemia** (imbalance between "myocardial oxygen" and "blood supply")

start in the sub-endocardial region (endocardium) → **We call it:** Transmural M.I.
Complicate into the whole muscle's thick region (endocardium→ myocardium → epicardium → pericardium)

- **Pathogenesis of Myocardial Infarction**
  Most common cause is **Thrombosis** on a pre-existing disrupted "atherosclerosis plaque".
  Initial Events of the RUPTURE:
  1- disruption-manifest as "intraplaque hemorrhage".
  2- Erosion (diminution) 3- Ulceration
  4- Rupture 5- Fissuring
  This irreversible cell injury occur within 20-30min.

- **Morphology:**
  1- Coagulative necrosis and inflammation
  2- Formation of granulation tissue (Granulation inflammation)
  3- Organization of the necrotic tissue to form a fibrous scar (inflammation → necorsis → fibrinous tissue)
  4- Dependent on age, size, recurrence, reperfusion.

Ischemic Cardiac Myocyte:

When ischemia occur, ATP is depleted in LESS THAN 2 MINUTES → There'd be Loss of contractility

ATP reduction sequence:

- ATP lower than normal (50% lower) → in 10 minutes } Irreversible cellular injury (20-40min)
- ATP lower than normal (10% lower) → in 40 minutes } Microvascular injury (1hour)

Location of the M.I:

Can be determined by the site if the occlusion
Anatomy of coronary circ.

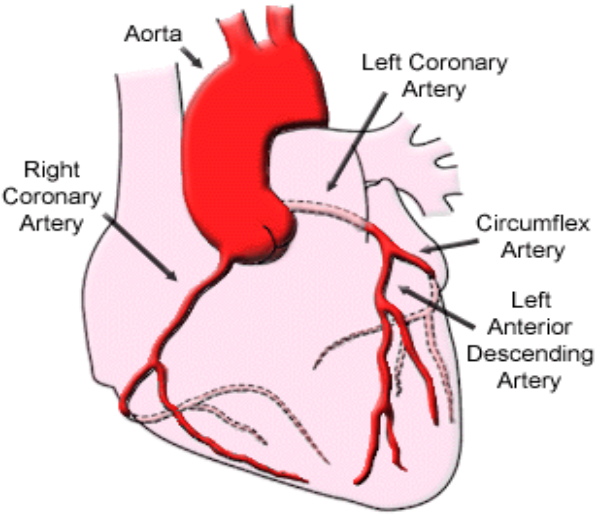
You may have to study "Anatomy of the Coronary Arteries" before This at the 1st lecture of *Anatomy*

Coronary Arteries Supply:

The heart has 3 major coronary arteries.

Two of these arteries → arise from a *common stem*, called the ***Left Main Coronary Artery.***
The third → is the ***Right Coronary Artery***

- The ***Left Main Coronary Artery***
  supplies the *left ,lateral and front side* of the heart, **by**:
  - The ***Left Anterior Descending (LAD)*** branch
    supplies the *front part of the heart* (with the **apex**).
    apex is the very front of the heart (it also means lt.ventricle)
  - The ***Left Circumflex (LCX)*** branch
    supplies *the left lateral and back side of the heart*.
- The ***Right Coronary Artery*** **(RCA)**
  supplies the *right and the bottom* parts of the heart.



Rate of development of "coronary atherosclerotic obstructions" = the rate of blockage (how fast is the block developing)
Collateral Blood Vessels: new vessels forming to induct when there's stenosis (angiogenesis).

What will you find in an MI (morphology):

1- **Inflammation + coagulative necrosis**
Inflammation include: neutrophils and macrophages

2- **Granulation tissue**
3- **Necrotic tissue becomes a** Fibrotic scar (contractility becomes hard –infarction- so → it diminishes –the muscle)

- **Complications**
  1- Angina pectoris (chest pain)
  2- Rupture.
  3- Arrhythmias
  4- Pericarditis (Pericardium inflammed)
  5- Mural thrombus, embolising to become "Thromboembolism" <<<VERY DANGEROUS.
  6- Ventricular aneurysms.

- **Clinical Features:**

1- Pain (characteristic of this pain)
a referred pain: due to inadequate perfusion = Pain felt in a part of the body at a distance from the area of pathology
epigastrium: upper middle region of the abdomin.

2- Pulse is weak.
3- Dyspnea (awareness of breathing)
4- Diaphoresis (perspiration = sweating)

~What is the Most common cardiac cause? → ***atherosclerosis***

if there's a **heart attack of "apex" of left ventricle**, which vessel would be blocked? → ***left anterior descending***

Why is- Long-term diabetic patients don't feel the Angina?? Because they'd lose their senses (nerves are weak).

JUST REMEMBER ECG abnormalities

OUTCOMES of MI

VERY IMPORTANT TO KNOW:
- Types of necrosis
- Types of enzymes affecting it
- Complications of MI
- Coronary Arteries (what they supply – if they are blocked)

- **Types of Necrosis**
  Coagulative Necrosis = in the heart
  Liquifactive Necrosis = in brain and lung
  Fibrinoid necrosis = of blood vessels

- **Enzymes Affecting it: (are elevated in MI – help in diagnosing)**
  2- **CK:** creatine kinase
  (enzyme in heart, skeletal ms. And little in brain – ↑when there's significant injury to them)

  3- **LD :**lactate dehydrogenase
  (Catalyses the conversion of lactate to pyruvate)
  Some of the organs relatively rich in LDH are the heart, kidney, liver, and muscle.
  As cells die, their LDH is released and finds its way into the blood

  4- **Troponin** (a complex of 3 regulatory proteins that regulate muscle contraction
  its levels can be used as a test of several different heart disorders, including MI

Thank you…. (pathology Team)