Ischemic heart disease

- **Heart disease remains the leading cause of morbidity and mortality in industrialized nations.**

- **40% of all deaths in the U.S.A (nearly twice the number of deaths caused by all forms of cancer combined).**

- **The yearly economic burden of ischemic heart disease (IHD) alone is in excess of $100 billion.**

ISCHEMIC HEART DISEASE (IHD)

- IHD = coronary artery disease *–mainly atherosclerosis-* (CAD)

  Because 90% of all IHD is caused by abnormalities in the coronary arteries

- IHD is a generic description for a **group of related syndromes** ***(not one disease)*** resulting from myocardial *ischemia* (***an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand***.

Ischemia can result from:

1- reduction in coronary blood flow caused by obstructive atherosclerotic disease →90 % of cases

2- increased demand

→ increase the rate of contraction e.g. tachycardia , increased physical activity

→ increase the resistance the heart has to pump against e.g. hypertension

3-diminished oxygen-carrying capacity (e.g., severe anemia, carbon monoxide poisoning)

Not enough hemoglobin (no O2 carriers)

Has higher affinity to bind to hemoglobin than O2, thus replacing it.

There are four basic clinical syndromes of IHD:

1-Angina pectoris (الذبحة الصدرية)

ischemia causes pain but is insufficient to lead to death of myocardium

2-Acute myocardial infarction (MI) (احتشاء عضلة القلب)

the severity or duration of ischemia is enough to cause cardiac muscle death

3-Chronic IHD

progressive cardiac decompensation (heart failure) following MI

4-Sudden cardiac death (SCD)

can result from a lethal arrhythmia following myocardial ischemia.

2→4 will be discussed in the next lecture

There are four basic clinical syndromes of IHD:

1-Angina pectoris

- Angina pectoris is intermittent chest pain caused by transient, reversible myocardial ischemia (ischemia causes pain but is insufficient to lead to death of myocardium) ,whereas the acute myocardial infarction is chest pain that result from a prolonged , irreversible myocardial ischemia, in which the ischemia is sufficient to produce myocardial death.

If the ischemia persists for less than 20-30min , the patient will suffer from angina pectoris
But, if it stayed for more than 20-30 min, the patient will suffer from MI

-pain→ a crushing or squeezing substernal sensation

- radiate down the left arm or to the left jaw *(referred pain).*

The duration of pain is longer in MI patients than in angina pectoris

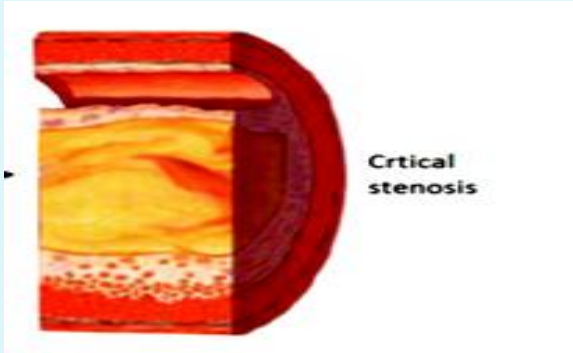
Pathogenesis of angina

- **atherosclerotic occlusion of coronary arteries and <u>new superimposed thrombosis and/or vasospasm</u>**

-lesion obstructing *<u>75%</u>* or more of a vessel lumen = critical stenosis → cause angina only <u>in the setting of *increased demand*</u>

-a fixed 90% stenosis can lead to inadequate coronary blood flow even *<u>at rest</u>* *(no increased demand)*.

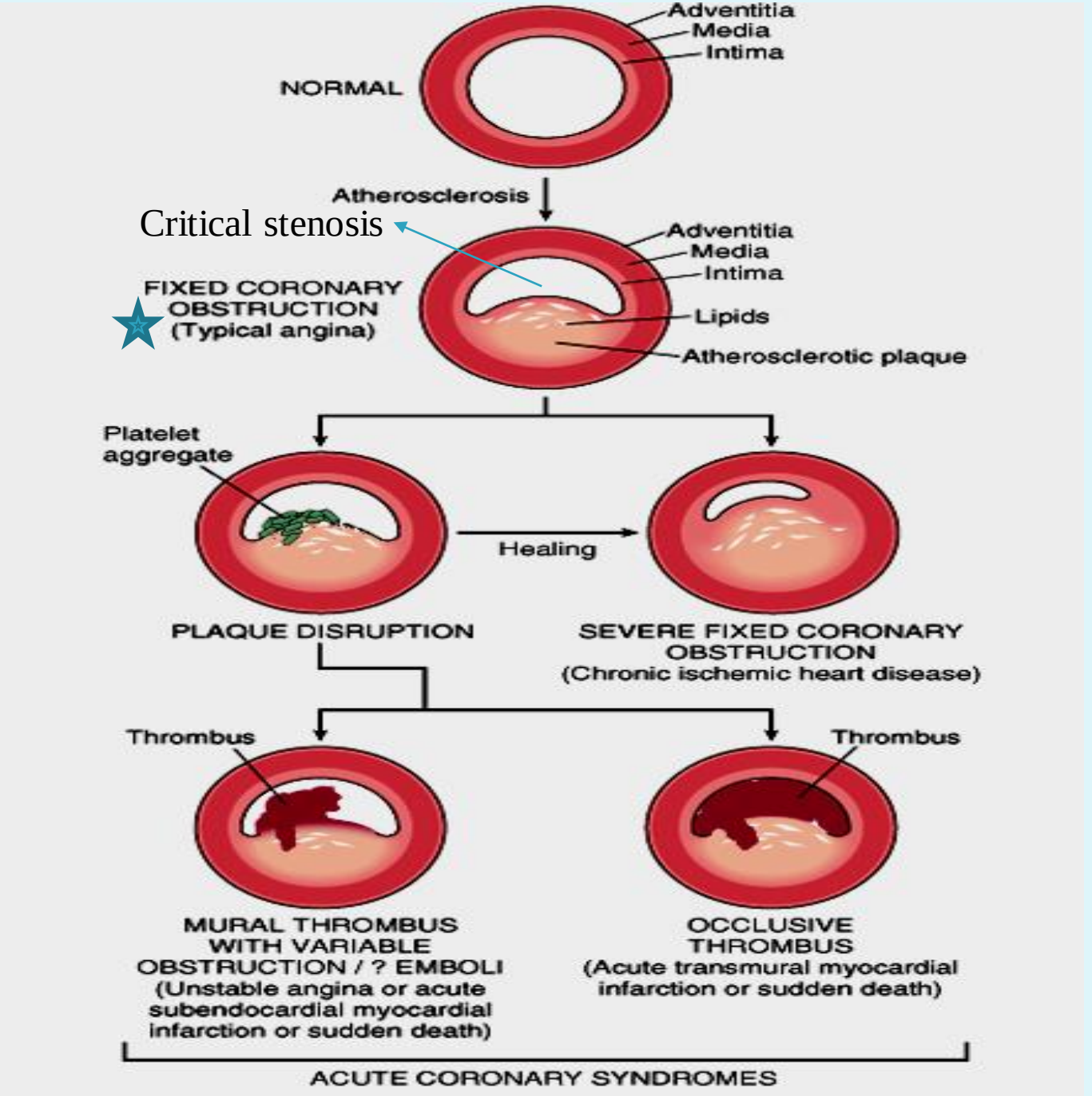
To develop clinical manifestations, the atherosclerotic occlusion must disrupt the balance between O2 supply and cardiac muscle demand



Pathogenesis

the progression to MI from critical stenosis involves increase the level of stenosis, or a superimposed thrombus on the atherosclerotic plaque caused by plaque disruption.

the doctor mentioned this, and u will see later that these complications lead to unstable angina the pre(MI) state.



Types of angina :

***1-stable* angina** (occur after certain levels of exertion)

2-variant angina or Prinzmetal angina *(* due to vessel spasm)

3-*Unstable* angina

occurring with progressively less exertion or even at rest.

1-Typical or *stable angina*

-is *episodic* *(the duration is less than 20mins)* **chest pain associated with exertion or some other form of increased myocardial oxygen demand (e.g., tachycardia or hypertension due to fever, anxiety, fear).**

1-Typical or *stable angina*

- **usually associated with critical atherosclerotic narrowing (≥75%) of one or more coronary arteries.**
- **the myocardial oxygen supply may be sufficient under basal conditions but cannot be adequately augmented to meet any increased requirements (exertion, emotional stress..etc)**

1-Typical or *stable angina*

- **The pain is relieved by *rest* (reducing demand) or by administering agents such as nitroglycerin;**
- **such drugs cause peripheral vasodilation and thus reduce venous blood delivered to the heart → reducing cardiac work.**
- **in larger doses, nitroglycerin also increases blood supply to the myocardium by direct coronary vasodilation**

2-Prinzmetal, or variant angina

- **Is angina occurring at rest due to coronary artery spasm** ***(not related to atherosclerosis)***
- **affect females more than males, and it occurs at rest or even during sleep**
- **completely normal vessels can be affected.**
- **The etiology is not clear, but might be associated with vasomotor hyperactivity (similar to what happens in raynoud phenomenon) .**
- **Treatment: administration of vasodilators such as nitroglycerin or calcium channel blockers.** ***(rest is not useful)***

In this type there is no permanent reduction in the blood flow, and the O2 demand isn't elevated

3-Unstable angina (crescendo angina)

- Progress from stable angina because of changes on the coronary atherosclerotic plaque. *(so the patient used to suffer from a stable angina)*

- characterized by *(the patient will start having these complications so he will seek for help)* **increasing frequency** of pain, precipitated by progressively **less** exertion, the episodes also tend to be more **intense** and **longer** lasting than stable angina, and its not relived by medications or rest .
- **associated with plaque disruption; superimposed partial thrombosis; distal embolization; vasospasm*(on the artery that already is atherosclerotic)*.**
- an indication of more serious, potentially irreversible ischemia (if complete luminal occlusion by thrombus)
- ***Called pre-infarction angina***

Acute vs chronic vascular insuffeciency

- **Chronic coronary occlusion**
  when a coronary artery develops atherosclerotic occlusion at a sufficiently slow rate, it may be able to stimulate collateral blood flow from other major epicardial vessels → protection against MI even in the setting of a complete vascular occlusion.
- **Acute coronary occlusions**
  cannot spontaneously recruit collateral flow and will result in infarction

The doctor didn't mention most of the things from this slide to the end :P
(The things that she mentioned from them are in the previous slides…)

Clinical Features of angina & MI

1) Severe, crushing substernal chest pain
2) Discomfort that can radiate to the neck, jaw, epigastrium, or left arm.

- angina pectoris→ pain < 20 minutes and relieved by rest or nitroglycerin
- MI→ pain lasts from 20 minutes to several hours and is not relieved by nitroglycerin or rest.

- 3) MIs can be entirely asymptomatic in 10% to 15% of the **cases (silent infarcts)➔** particularly common in patients with:

  1- underlying diabetes mellitus (due to peripheral neuropathies)

  2- in the elderly

4- the pulse is rapid and weak

5- patients nauseated particularly with posterior-wall MIs.

6- dyspnea is common (impaired myocardial contractility and dysfunction of the mitral valve apparatus, with resultant pulmonary congestion and edema).

7- massive MIs (>40% of the left ventricle)→ cardiogenic shock .