

CCS PATHOLOGY

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Color code

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Additional info

Important

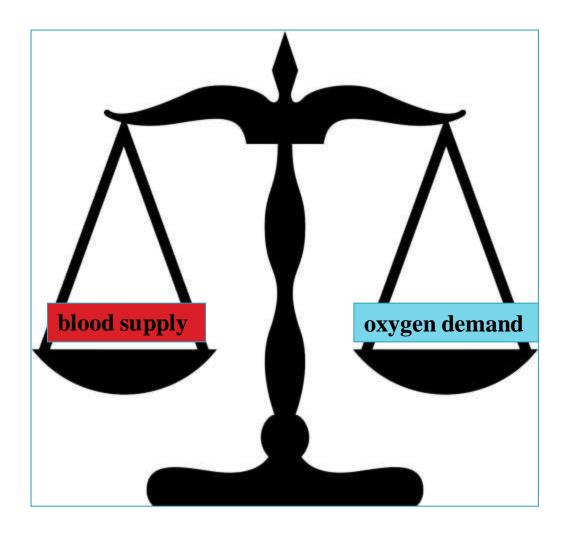


ISCHEMIC HEART DISEASE-1 Angina pectoris

Dr. Nisreen Abu Shahin Professor of Pathology Pathology Department University of Jordan • Heart disease is the leading cause of morbidity and mortality worldwide (Especially ischemic heart

disease).

Normally ...



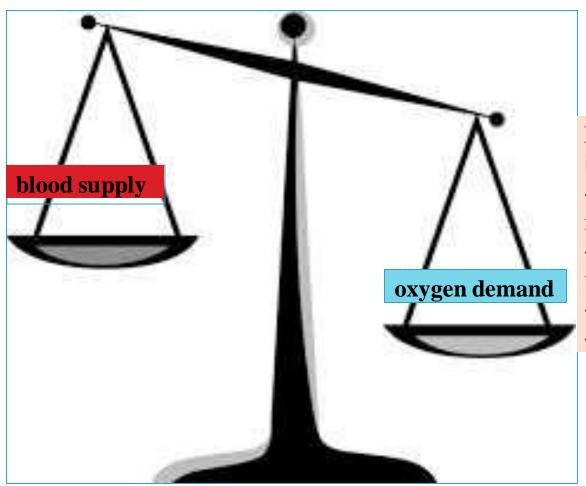
Normally there should be a balance between blood supply and oxygen demand for the cardiac muscles to function regularly.

myocardial ischemia occurs when:

Examples (Of things that reduce blood supply):

- -Atherosclerosis
- -Coronary Vasospasm
- -Hypovolemia (Massive bleeding)
- -Shock

Ischemia occurs whenever there is a reduction in blood supply or increase in oxygen demand.



Examples (Of things that increase oxygen demand):

- -exertion (Physical movement)
- -hypertension(Increase the volume of blood)
- -stress (Emotional)
- -tachycardia

Ischemic heart disease (IHD)

 a group of related syndromes resulting from myocardial ischemia (an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand)

• IHD \approx coronary artery disease (CAD)

The naming is usually interchangeable between IHD and CAD

Ischemia can result from:

- 1. reduction in coronary blood flow atherosclerosis (90 % of cases)
- 2. increased demand (e.g., tachycardia or hypertension)
- 3. diminished oxygen-carrying capacity (e.g., (severe) anemia, CO poisoning by competing with oxygen binding to hemoglobin)

There are four basic clinical syndromes of IHD: Ischemia is precent but not enough to

- 1. Angina pectoris (الذبحة الصدرية) cause cardiac muscle death (limited Ischemia) ischemia causes pain but is insufficient to lead to death of myocardium
- 2. Acute myocardial infarction (MI) (احتشاء عضلات القلب)
 the severity or duration of ischemia is enough to cause
 cardiac muscle death

 Myocardial ischemia of longer duration and
 more sever

3. Chronic IHD

progressive cardiac decompensation (heart failure) following MI. The viable myocardium becomes exhausted after attacks of myocardial ischemia.

4. Sudden cardiac death (SCD)

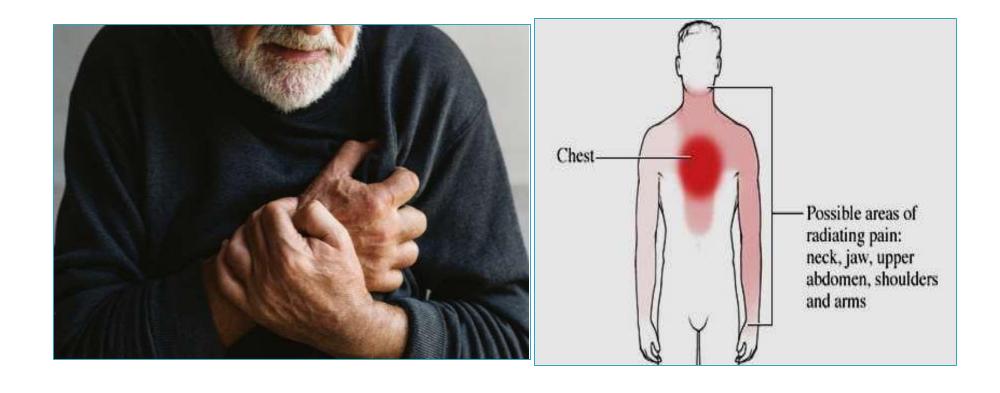
Death that either follows cardiac symptoms within less than 24 hours or without obvious symptoms.

Can result from a <u>lethal arrhythmia</u> following myocardial ischemia.

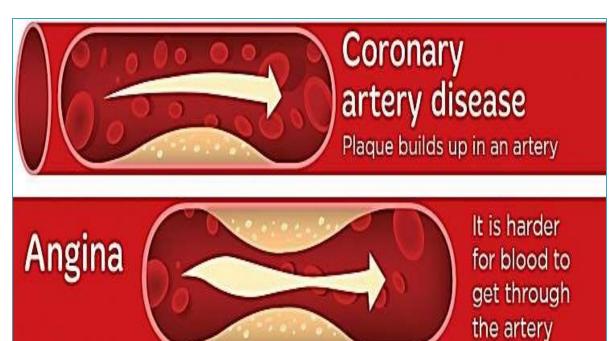
The underlying cause is cardia Thus called cardiac death

Today's lecture we are **Angina Pain**

A crushing or squeezing substernal chest pain that possibly radiates to left arm, left side of the neck, the jaw, the upper abdomen, and the shoulders. The pain of myocardial infarction is similar and has the same distribution.



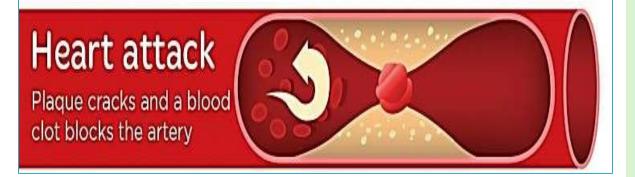
Angina pectoris vs MI



- Angina causes intermittent chest pain caused by transient reversible myocardial ischemia (ischemia causes pain but is insufficient to lead to death of myocardium).
- Angina pectoris: pain < 20 minutes and relieved by rest or nitroglycerin.
- MI: pain <u>lasts > 20 minutes</u> to several hours and is **not** relieved by nitroglycerin or rest.

Both angina and acute MI (heart attack) are a result of ischemia " a reduction in the blood supply "

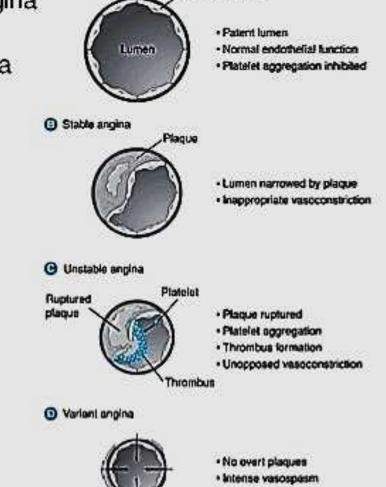
- Angina: The severity and duration of the ischemia is not enough to kill the cardiac muscle cell <20 minutes.
 Other cells are much more sensitive like the brain 3 minutes only.
- MI: death, necrosis of the cardiac muscle cells



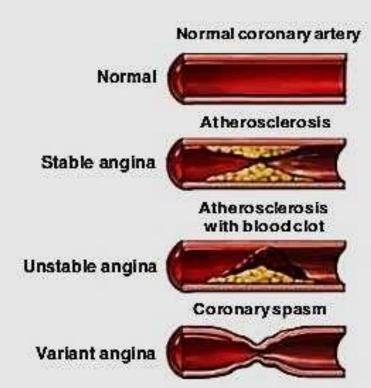
You must memorize all names for each type:

Three types of angina

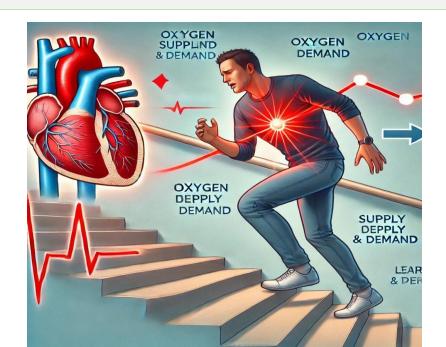
- Stable angina/Classic angina/Effort angina / typical angina
- Unstable angina/Crescendo angina
 / pre-infarction angina
 /
- Variant angina/Prinzmetal angina



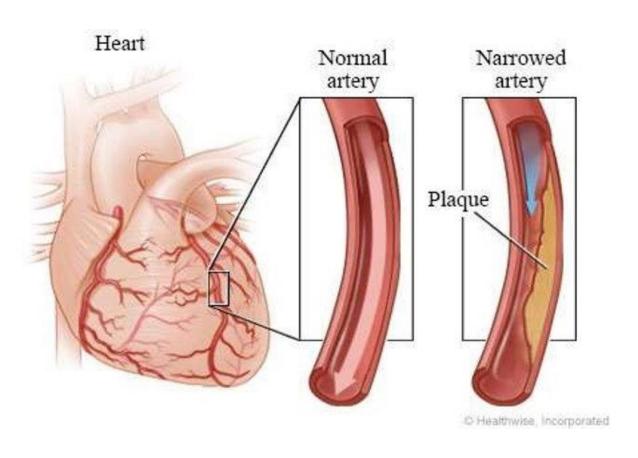
Endothelial cell



- Read this scenario that represent the classic scenario of typical (stable) angina:
- A person decided to take the stairs instead of the elevator. While climbing the three floors, he began to experience severe chest pain that reaches to his left arm <u>after reaching the second floor</u>. What caused the angina in this patient?
- The patient has underlying atherosclerosis, particularly severe coronary stenosis, but had not experienced pain while at rest. The pain occurred when he reached the second floor <u>due to an increased demand for oxygen from physical exertion</u>. With the <u>existing coronary stenosis</u>, the heart's ability to deliver enough oxygen to the muscles was impaired, leading to an imbalance between oxygen supply and demand, which triggered the chest pain.

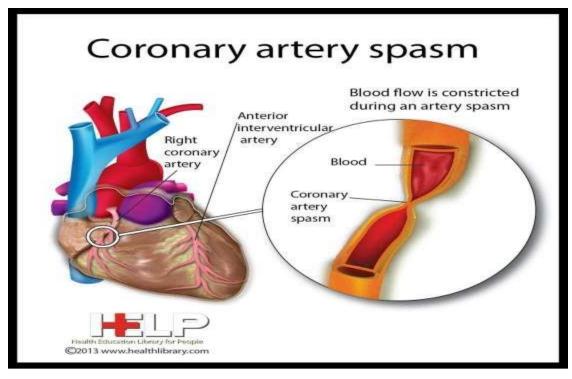


Pathogenesis of stable angina: critical coronary stenosis



- -Episodic pain only with increased demand
- -forms of \(\) myocardial oxygen demand (e.g. exertion; tachycardia; hypertension; fever; anxiety; fear).
- -Associated with critical atherosclerotic narrowing.
- -Relieved by rest (reducing demand) or by drugs (e.g. nitroglycerin ->> vasodilator)

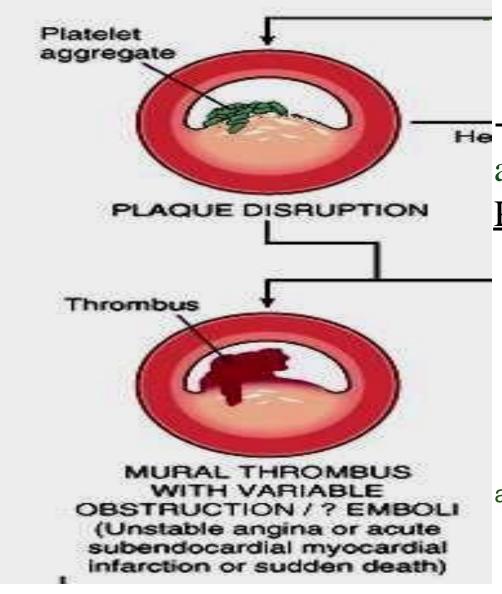
Pathogenesis of prinzmetal angaina (variant): Severe coronary vasospasm



- occur at rest or sleep (without increase oxygen demand)
- Vessels without atherosclerosis can be affected
- Etiology not clear
- Treatment: vasodilators (nitroglycerin or calcium channel blockers)
- Rest is not considered a treatment. I want to decrease the vasoconstriction, trying to make vasodilation.

- If there is no atherosclerosis and no increase in oxygen demand, What will cause angina?
- It happens due to severe coronary vasospasm. This vasospasm causes a sudden reduction in blood flow to the heart muscle, leading to ischemia. The ischemia then triggers the typical symptoms of angina, such as substernal chest pain that reaches to the left arm.

Pathogenesis of unstable angaina (pre-infarction angina or crescendo angina):



Unstable angina lies on the borderline between stable angina and acute myocardial infarction.

- Critical coronary stenosis (usually with atherosclerosis) with **superimposed** Acute Plaque Change:

- 1-plaque disruption
- 2- partial thrombosis (non-occlusive)
- 3- distal embolization
- 4- vasospasm over the already stenosed coronary artery.

• The patient has atherosclerosis and has experienced episodes of stable angina in the past, for which he was treated. However, now there are acute changes occurring at the site of the atherosclerosis (plaque disruption ,partial thrombosis (non-occlusive), distal embolization, vasospasm); In the past (before these changes) the blood supply was less than 20%, but with these changes, it became very low, almost zero. As a result, the patient is in a critical situation, between stable angina and myocardial infarction. For this reason, it is referred to as pre-infarction angina.

Unstable angaina (crescendo angina):

- increasing <u>frequency</u> of pain, precipitated by <u>less</u> exertion.
- more intense and longer duration lasting than stable angina
- <u>Causes</u>: plaque disruption; superimposed partial thrombosis; distal embolization; vasospasm.
- Usually precedes more serious, potentially irreversible ischemia, thus it is called: pre-infarction angina



VERSIONS	SLIDE#	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2 → V3			



امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!