

# Writer:019

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Sheet no. : 7

## Ischemic Heart Disease (Angina Pectoris) In this lecture we are going to talk about "Myocardial Ischemia". Note : Heart disease is the leading cause of morbidity and mortality worldwide Normally in our body, there should be some sort of *balance* between myocardial blood supply and oxygen demand. **SO**, myocardial ischemia develops when: 1) Blood supply is **decreased** or 2) Oxygen demand is **increased**. $\rightarrow$ Meaning that the disease develops when some sort of **imbalance** between blood supply and oxygen demand occurs. Myocardial Ischemia: **1**77 Blood Oxygen supply demand Factors that increase oxygen Factors that decrease blood demand in the heart: supply are: 1) Increase exertion (Physical activity) 1) Atherosclerosis of coronary vessels 2) Hypertension 2) Coronary vasospasm 3) Stress 3) Hypovolemia 4) Tachycardia 4) Shock Why do these things increase oxygen demand of the 5) reduction of oxygen content in the blood cardiac muscles? (There's blood, but there's no enough oxygen) Because they have to contract more often and with a stronger power (They need to work harder than usual) so they need more oxygen **Ischemic heart disease:** a group of related syndromes (conditions) resulting from myocardial ischemia (an imbalance between cardiac blood supply (perfusion) and myocardial oxygen demand). In many cases we can use "Coronary Artery Disease (CAD)" as an interchangeable term with Ischemic Heart Disease(IHD) because of the great association between them .

it was found that CAD is responsible for the majority of ischemic heart disease cases

Mechanisms of ischemia in the heart (causes):

the most important

1) Reduction in coronary blood flow: mainly due to atherosclerosis, (90% of all cases).

2) Increased oxygen demand like: Tachycardia, hypertension and other examples as we said above.

3) Diminished oxygen-carrying capacity (least common cause) ; it means that <u>blood supply</u> <u>is not really diminished and the myocardial oxygen demand is not greatly increased , but</u> <u>the imbalance is resulting from the *insufficiency of oxygen* supply inside the blood, and this may be caused by rare cases like: severe sudden anemia, OR carbon monoxide (co) poisoning .</u>

As we said, the IHD is not a single disease; it is actually a **group** of conditions that all are related to ischemia.

So, there are four basic clinical syndromes of IHD:

1) Angina pectoris: ischemia causes pain, but is insufficient to lead to death of myocardium

2) Acute myocardial infarction (MI): the severity or duration of ischemia is enough (sufficient) to cause cardiac muscle death is more than 20-30 min, or in very severe obstruction

3) **Chronic IHD**: form of progressive cardiac decompensation (congestive heart failure) following MI

4) **Sudden cardiac death (SCD):** form of death that can result from a **lethal arrhythmia** following myocardial ischemia.

\* Angina is usually caused by reduction of blood flow mainly (like in Atherosclerosis), or increased oxygen demand

Angina Rectoris: means that there is an ischemic chest pain in a form of crushing or squeezing substernal (central and usually more to the left side) chest pain. Sometimes, this pain radiates to: neck, jaw, upper abdomen, left shoulder and left arm.





 Possible areas of radiating pain: neck, jaw, upper abdomen, shoulders and arms

NOW, both angina pectoris and acute

myocardial infarction are related to ischemic chest pain, so how can we differentiate between them?

<u>Angina</u>: causes intermittent chest pain caused by transient reversible myocardial ischemia (ischemia is sufficient to cause pain but is insufficient to lead to death of mot enough to myocardium). Usually angina pectoris related pain time < 20 minutes and since it is caused by transient ischemia so it is relieved by rest (which reduces oxygen demand) or some medications like nitroglycerin (which is a vasodilator) so it will improve blood supply.</li>
Here we're trying to relief the ischemia so we need to increase the blood supply (perfusion) or reduce oxygen demand

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• <u>MI:</u> it's more severe, pain is caused by irreversible myocardial ischemia, in this case ischemia had developed 20 minutes before pain appeared, so pain lasts > 20 minutes to several hours and is not relieved by nitroglycerin or rest WHY? because we reached a level of irreversible cell damage (necrosis)



EXRTA: The term myocardial infarction indicates the development of an area of MYOCARDIAL NECROSIS caused by ischemia. Myocardial necrosis begins within 20-30 minutes of coronary A. occlusion. The infarct usually reaches its full size within 3-6 hours; thus the disease pain is longer than that in angina.

#### Types of Angina : most types of angina are because of a problem inside the coronary artery

- 1) Stable angina / Classic angina/ Effort angina/ Typical angina
- 2) Unstable angina / Crescendo angina/ Preinfarction angina

### 3) Variant angina/ Prinzmetal angina

NOW: we will discuss these types

 critical stensosi is one of the possible complications following atherosclerosis

• The pathogenesis of stable angina is <u>critical coronary stenosis</u>: means that there is a significant narrowing in the coronary artery (there's loss of a significant percentage of the surface area/diameter of the coronary artery) by an atherosclerotic plaque, so blood supply will be less than normal (50-75% or more of the blood supply is lost)

- It's associated with episodic pain only with increased oxygen demand.
- Examples that increase oxygen demand: **exertion**, tachycardia, hypertension, fever, emotional exertion (anxiety, anger or fear).
- So it should be associated with **critical** atherosclerotic narrowing.
- Because of the above factors, this angina could be relieved by rest (reducing o2 demand) or by certain medications/drugs (e.g. nitroglycerin).

\*\*\* \* Why it's called Effort Angina ??



## "Prinzmetal Angina" / "Variant angina"

#### This type is <u>rare</u>

• Pathogenesis of this type is severe coronary vasospasm The problem here is in the blood supply, there's a sudden and acute reduction in the blood supply by that coronary artery and this will lead to ischemic chest pain.

## Because of that, it can occur at rest or sleep (because it's not related to oxygen demand). (not related to effort).

- Coronary vessels without atherosclerosis can be affected.
- Etiology is not clear.
- But because it is caused by vasospasm, so it could be treated with vasodilators (nitroglycerin or calcium channel blockers)



## "Unstable Angina" / "Crescendo Angina" / "Preinfarction Angina" This is the WORST type of angina

Pathogenesis is: presence of critical stenosis with superimposed <u>Acute Plaque</u>
<u>Change</u>

\*acute plaque change means development of certain acute changes that are superimposed on already stenotic vessels.

#### This includes:

- 1- Plaque disruption (or rupture). Atherana disruption
- 2- Partial thrombosis (non-occlusive: not complete occlusion, not 100% of lumen is lost).
- 3- Distal embolization (The thrombus is moving in the coronary artery towards distal arteries, which are smaller in diameter so this will cause distal occlusion).

4- Superimposed vasospasm over the atherosclerosis.

all happens over the critically stenosed coronary vessel. (Acute on top of critical stenosis)

• Unstable angina (crescendo angina) is the *worst* type of angina and if it's left untreated, it'll eventually lead to infarction, that's why it's called preinfarction angina it's on the border line between viable myocardium and non-viable (necrotic) myocardium

it's the worst type because it's usually associated with:

- Increasing frequency of pain, precipitated by less exertion and the pain is more intense and longer lasting than stable angina. Don't respond to treatment used for stable angina
- Causes: plaque disruption; superimposed partial thrombosis; distal embolization; vasospasm. (*important*!!!!)
- Usually precedes more serious, potentially irreversible ischemia, thus it is called: preinfarction angina.

The most typical scenario is that the patient already has atherosclerosis, and he might already have attacks of stable angina in his medical history, but now the patient is developing ischemic chest pain even with a minor amount of physical activity and this pain is more intense and severe, and it lasts longer and it's less responsive to nitroglycerin and rest



Picture about types of angina , the doctor mentioned the names only .



قال النبي صل الله عليه وسلم: " أكثروا من قول : لا حول ولا قوة إلا بالله، فإنها كنز من كنوز الجنة "