"He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all."
William Osler

Medicine

430

Acute Coronary Syndrome

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Acute coronary syndrome

Acute coronary syndrome (ACS) refers to any group of symptoms attributed to obstruction of the coronary arteries. The most common symptom prompting diagnosis of ACS is chest pain, often radiating of the left arm or angle of the jaw, pressure-like in character, and associated with nausea and sweating.

Acute coronary syndrome usually occurs as a result of one of three problems:

1- ST elevation myocardial infarction (STEMI)
2- Non-ST elevation myocardial infarction (NSTEMI)
3- Unstable angina

What Is Atherosclerosis?

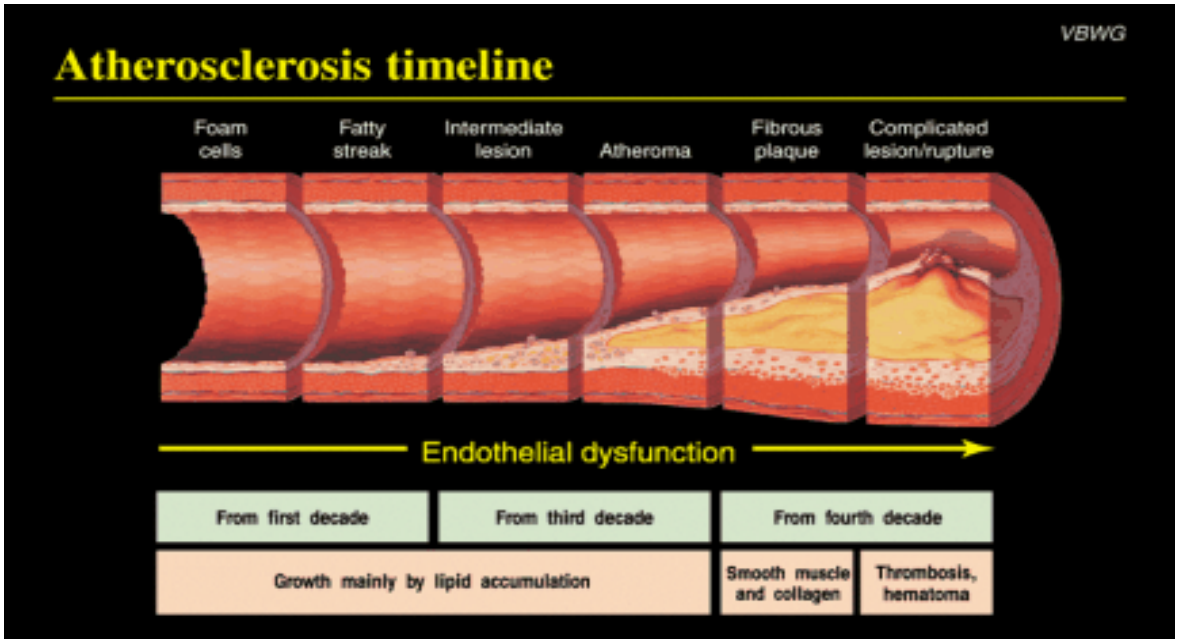
It's a disease in which plaque builds up inside your arteries (in the endothelium in the media of the artery to be precise)
Arteries are blood vessels that carry oxygen-rich blood to your heart and other parts of your body .A **Plaque** is made up of fat, cholesterol, calcium, and other substances found in the blood. Over time, the plaque hardens and narrows the affected artery. This limits the flow of oxygen-rich blood to your organs and other parts of your body.

Atherosclerosis is a major risk factor for ACS!

WHO'S AT RISK to have CORONARY ARTERY DISEASE?

Non-modifiable factors that influence risk for coronary artery disease include age, sex, family history, and ethnicity or race. Men have a higher risk than women.

Modifiable risk factors include elevated levels of serum cholesterol, low-density lipoprotein(LDL) , and triglycerides(TG); lower levels of high-density lipoprotein (HDL); and the presence of type 2 diabetes, cigarette smoking, obesity, a sedentary lifestyle, hypertension, and stress.



Atherosclerosis timeline

- The pathological effects of atherosclerosis occur over decades. A subtle injury to the endothelium initiates the atherosclerotic process. Endothelial dysfunction underlies many stages in the progression of atherosclerosis from earliest onset to the lesions that result in coronary heart disease (CHD).
- Foam cells may infiltrate the vessel, progressing to a fatty streak. As the lesion progresses, small pools of extracellular lipid form within the smooth muscle layers, disrupting the intimal lining of the vessel. Progression to an advanced lesion, or atheroma, occurs when accumulated lipid, cells, and other plaque components disrupt the arterial wall.

Further Explanation

- Progression of atheroma involves accumulation of smooth muscle cells that elaborate extracellular matrix macromolecules. Once the plaque becomes fibrous, the danger of rupture increases. This type of advanced lesion can be found beginning in the fourth decade of life.
- The clinically important complications of atheroma usually involve thrombosis. Arterial stenosis by itself seldom causes acute unstable angina or acute myocardial infarction. Indeed, sizable atheroma may remain silent for decades or produce only stable symptoms, such as angina, precipitated by increased demand.
- Thrombus formation usually occurs because of physical disruption of atherosclerotic plaque. The majority of coronary thromboses result from a rupture of the plaque's protective fibrous cap, which permits contact between blood and the highly thrombogenic material located in the lesion's lipid core.
- The endothelium participates in the atherosclerotic process and remodeling through secretion of specific compounds.

Types of plaques

STABLE PLAQUES	**UNSTABLE PLAQUES**
contain thick fibrous cap	Thin fibrous cap)due to the Low smooth muscle cell count(
No inflammation	collection of white blood cells (inflammation)
Small hard lipid core	Large soft lipid core
less likely to rupture and develop an embolus	**More** likely to rupture and develop an embolus

Factors contributing in Plaque vulnerability:

- Low smooth muscle cell count
- Thin Cap
- Large Lipid Core
- High Macrophage content

What is ischemia?

It's a restriction in blood supply to tissues, causing a shortage of oxygen and glucose needed for cellular metabolism (to keep tissue alive). Ischemia is generally caused by problems with blood vessels, with resultant damage or dysfunction of tissue.

"It's the imbalance between body demand and body supply of oxygen!"

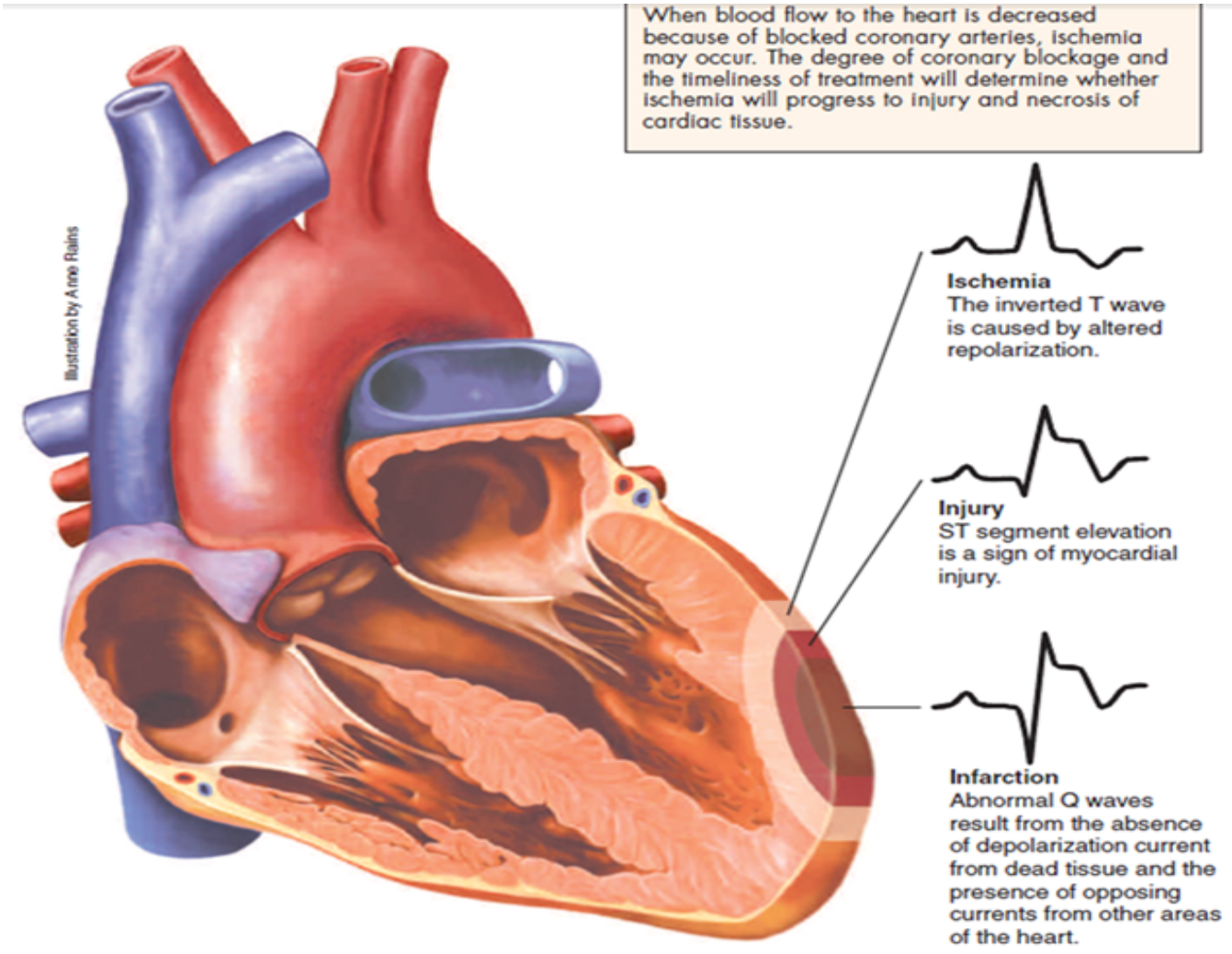
Acute Coronary Syndroms :

1- Unstable Angina
2- Non–ST-Segment Elevation Myocardial Infarction (NSTEMI)
3- ST-Segment Elevation Myocardial Infarction (STEMI)

How Do They Differ ?

	Unstable Angina	NSTEMI	STEMI
cause	Thrombus partially or intermittently occludes the coronary artery	Thrombus partially or intermittently occludes the coronary artery	Thrombus fully occludes the coronary artery
Signs and Symptoms	1- Occurs at rest or with exertion; limits activity	1- Occurs at rest or with exertion; limits activity 2- Longer in duration and more severe than in unstable angina	1- Occurs at rest or with exertion; limits activity 2- Longer in duration and more severe than in unstable angina (irreversible tissue damage [infarction] occurs if perfusion is not restored)
Diagnostic Findings	1-ST-segment depression or T-wave inversion on electrocardiography 2-Cardiac biomarkers not elevated	1-ST-segment depression or T-wave inversion on electrocardiography 2-Cardiac biomarkers are elevated	1-ST-segment elevation or new left bundle branch block on electrocardiography 2-Cardiac biomarkers are elevated

Acute Coronary Syndrome: From Ischemia to Necrosis



How ACS happens ?

- ACS begins when a disrupted atherosclerotic plaque (unstable, vulnerable plaque) in a coronary artery stimulates platelet aggregation and thrombus formation.
- That would lead to deacrease blood flow (oxyginated blood) to myocardial cells wich eventually leads to ischemia.

Clinical presentation :

The degree to which a coronary artery is occluded typically correlates with presenting symptoms and with variations in cardiac markers and electrocardiographic findings.

- unstable angina, chest pain normally occurs either at rest or with exertion and results in limited activity.
- Chest pain associated with NSTEMI is normally longer in duration and more severe than chest pain associated with unstable angina.
- In both conditions,the frequency and intensity of pain can increase if not resolved with **rest, nitroglycerin, or both** and may last longer than 15 minutes.
- **Pain** may occur with or without radiation to the arm, neck,back, or epigastric area. In addition to angina,patients with ACS also present with **shortness of breath**, diaphoresis, nausea, and lightheadedness.
- Changes in vital signs, such as **tachycardia, tachypnea,hypertension, or hypotension**, and decreased oxygen saturation (SaO2) or cardiac rhythm abnormalities may also be present.

Diagnosis of MI :

The patient's clinical history, presenting symptoms, biomarker levels, and electrocardiographic results are all evaluated.

Cardiac biomarkers:

Injured myocardial cells release proteins and enzymes known as cardiac biomarkers into the blood. These markers help practitioners determine whether the patient is having or has recently had an acute MI (either an NSTEMI or a STEMI).

The utility of various biomarkers is determined by the timing and duration of their elevation as well as by the extent of their cardiac specil levels **of troponins I and T** increase within **four to six hours of myocardial injury and last up to 2 weeks.**

Cardiac troponins are the preferred biomarkers for diagnosing acute MI because elevated levels correlate with a more accurate diagnosis, predict a high risk of future cardiac events even when levels of the myocardium-specific biomarker creatine kinase-MB (CK-MB) are normal or only mildly elevated

CK-MB is a cardiac-specific enzyme that's released within four to six hours of injury and remains elevated for 48 to 72 hours after injury.

Additional information: troponin I levels remain elevated for four to seven days, and troponin T levels remain elevated for 10 to 14 days

Other helpful investigations:

- CBC- Leucocytosis
- Elevated ESR
- Chest X-Ray **(Pulmonary Edema**)
- Echocardiography

Management

Aim of therapy	Drugs	
Improve oxygen supply	Supplemental O2	
	Antiplatelets drugs	**Aspirin** • Decreases mortality in MI and should be administered as early as possible and continued indefinitely in patients with ACS. • Chewable aspirin 160 to 325 mg at presentation, then 75 to 325 mg daily **Clopidogrel** • More potent than ASA • Irreversible ADP receptor blockers • Adjunct to reperfusion therapy
	Antithrombotics	**Heparin** - Unfractionated - Low molecular 1. Used for patients with NSTEMI and STEMI 2. Prevents further thrombosis and aids in insuring patency of the occluded artery.
	Coronary vasodilators	(**Nitroglycerine**)
	Reperfusion therapy: (**TIME IS MUSCLE!**) - Fibrinolytic therapy - Percutanous coronary intervention (**PCI**)	Note: early restoration of blood flow can limit necrosis. • improve left ventricular function • reduce mortality rate, especially in STEMI! • Should be given during a 12hr window, and given **ASAP**! 2 types of fibrinolytics: uNon Fibrin specific (**Streptokinase**) vFibrin specific <u>only used for STEMI</u> (NOT NSTEMI) • Reduces short and long term mortality • shown to be effective in numerous randomized trials . **Primary PCI**
Reduce O2 demand	Beta blockers → Analgesics →	(**Propranolol**, Metoprolol) (**Morphine**)
Other medications	ACE inhibitors(**Enalapril**, **Lisinopril**) - Statin therapy	**Fibrinolytics drugs :** • ACE inhibitors(Enalapril, Lisinopril) • Statin therapy (not only used here for reducing cholesterol level as it works as an anti-inflammatory agent

Myocardial Infarction's complications

It can be sub-categorized into Electrical complications, mechanical and functional.

Electrical complications

1. Tachyarrhythmias

a. Ventricular:
- Ventricular Tachycardia
- Ventricular Fibrillation (common in STEMI particularly with reperfusion)

b. Supraventricular:
- Atrial Fibrillation

*Patients with ventricular tachycardia should be treated with IV beta-blocker

2. Bradyarrhthmia

- 1st, 2nd, and 3rd degree AV blocks
- New LBBB (Left bundle branch block), or RBBB (Right bundle branch block)

* Patients treated initially with IV atropine and may require pacemaker insertion

Mechanical complications

1. Mitral regurgitation →
   - (2-7 days post MI)
   - Caused by papillary muscle rupture.

Sever MR can occur early in the course of STEMI, 3 mechanisms may occur:

1-sever left ventricular dysfunction and dilation, causing annular dilation of the valve and subsequent regurgitation

2- MI of the inferior wall, producing dysfunction of the papillary muscle that may respond to coronary intervention

3-MI of the papillary muscle causing a sudden severe pulmonary oedema and cardiogenic shock.

2. Free LV wall rupture
   - Rare
   - 1st 24hr up to 2 weeks

3. Ventricular septal defect
   - 1-3% (may occur in 1-2% of patient with STEMI, associated with delayed or failed fibrinolysis)
   - Occurs with inferior and anterior MI

*An intra-aortic balloon pump and coronary angiography may allow for patients optimization prior to surgery.

Pump failure (functional)

1. **Heart failure**
   - Bad prognostic sign
   - Reflects the size of the MI
   - ACE inhibitors (can be givin in <24-48 hours if the patient's blood pressure is satisfactory) and diuretics (IV furosemide to mild heart failure patients) is cornerstone therapy.

2. **Cardiogenic Shock**

(Cardiogenic shock is when the heart has been damaged so much that it is unable to supply enough blood to the organs of the body)

- Happens with major MI's
- Carries high mortality (>50% in 30 days)
- Should be rushed for cardiac catheter and either PCI or Coronary bypass grafting.