PBL lecture #1
Dr Hanna Makhamreh Cardiovascular System Dina Shaban

In this lecture, we are going to discuss two topics; Myocardial Infarction and Heart Failure, in terms of how the patient is presented, symptoms, what to do etc …

At first, we'll talk about Acute Coronary Syndrome (ACS), which is classified into:

1. ST elevation MI (STEMI): ST segment is a part of the electrocardiogram (ECG) that in this case is elevated … High emergency; because it means that there's a coronal artery that is 100% occluded & we must open this occluded artery.
2. Non- ST elevation MI (NSTEMI): ST segment isn't elevated on ECG –normal ECG-, however troponin is high.
3. Unstable Angina: ST segment isn't elevated on ECG –normal ECG- & troponin is normal and here we mainly focus on symptoms for diagnosis. For instance, when a patient presents with severe chest pain and after ECG analysis no specific changes were observed and troponin is normal, hence this patient is labeled as having an unstable angina. Despite the normal ECG and the normal troponin, the patient is sent to have a cardiac catheterization.

What is Troponin test? It's a test that's more accurate than CPK & CK-MB & its high accuracy will indicate whether the case is NSTEMI or unstable angina.

So, STEMI is diagnosed via ECG, whereas, NSTEMI & unstable angina can't be diagnosed by ECG; since both will have a normal ECG, so how to differentiate between them? By Troponin test, which will be high in case of NSTEMI & normal in case of unstable angina.

Note: in ECG, V1, V2, V3, V4, V5, V6 represent the anterior wall of the heart (V1,V2 are anterioseptal … V3,V4 are anterior … V5,V6 are anteriolateral) & are supplied by LAD; i.e. if LAD is occluded, then these V1-V6 will be elevated… the ECG will be explained fully in the following lecs of the system, so don't worry about the aforementioned info..

Acute Coronary Syndrome is the single leading cause of death, statistics show that in the US there are almost 500 000 deaths in the year 2004 and almost 1,200,000 new cases of ACS per year.

40% of those with acute coronary syndrome die within a year of having the event, mostly immediately after the chest pain or during the transfer to the hospital.

Risk Factors:
-Smoking (Major risk factor)

-Hypertension

-Diabetes

-Dyslipidemia
Elevated LDL is the most effective risk in Dyslipidemia; low HDL comes next and thirdly high triglycerides.

-Family History
Here we are looking for first degree relatives only (mother, father, sister, brother, daughter, son) who have been affected at a young age, for males we consider this age to be 55 and for females it is less than 65 and so there's a 10 years difference.

- Age: males are usually at risk at the age of 45 and females at 55.

Other risk factors include: presence of chronic kidney disease, sedentary life style –lack of exercise- and obesity but the previous ones are more dangerous.

Age and gender are non-modifiable risk factors whereas smoking, dyslipidemia hypertension and diabetes are modifiable risk factors that can be controlled with treatment by drugs and lifestyle changes.

- "The presence of risk factors suggests that you have a problem": if a patient presents with chest pain & he doesn't have any of the prev. risk factors, then our suspicion that he has an acute coronary event is much less than in the case of a patient with 20 years of D.M on insulin & was presented with chest pain … so, we have to pay an attention to the great role of risk factors in ACS along with the results of ECG & Troponin test so that not all patients presented with chest pain will be sent for catheterization.

How to know whether a chest pain is concerning or not? 1) When it's retrosternal 2) The chest discomfort is usually described as "heaviness".

The onset of the chest pain could be at rest, with exertion or with emotional upset.

If the pain is 1) Described as heaviness. 2) Brought on by exertion. 3) Relieved with rest or sublingual nitroglycerine --- then, this is typical angina (3/3), however, if 2 of the previous 3 features of chest pain are present but not the third, then it's atypical angina (2/3) & if only 1 out of the 3 features is only present, then it's a non-cardiac chest pain (1/3).

When a patient having an acute coronary event arrives at the emergency room we follow the same procedure regardless of the time the patient arrived at, and we consider the time of arrival of the patient to the ER to be time zero, time is then calculated as from this point. Within 10 minutes after arrival time an ECG must be done and interpreted by an experienced and professional physician. Now, if there was an ST elevation in the ECG for example, which is the most lethal, a cascade of events must start immediately. If however the hospital doesn't provide cardiac catheterization –not seen in Amman for sure- then the patient should be given a life saving thrombolytic agent which is usually TPA – tissue plasminogen activator-(in the past streptokinase was given). Nevertheless, cath & interventions are worldwide known to be better than the thrombolytic agents.

Within 90 minutes into the ER, the patient must be given anticoagulants (heparin), aspirin, oxygen, morphine (to relieve pain) & IV access until the team of catheterization arrives, which should be within 30 minutes, the team consists of 4 people a doctor, two nurses and a radiologist. The team is responsible for preparing the groin for the catheter if the access is going to be from the femoral artery (but also the radial artery can be used) and the patient is connected to an ECG monitor. Blood flow must be resumed within those 90 minutes.

Why do we use the arteries for inserting the axis for catheterization?

This is because we are aiming for the coronary arteries which are the sites for ACS/MI and so inserting a catheter in the vein is of no use since it'll go back to the right atrium (via SVC or IVC) → right ventricle → pulmonary trunk (and this is of no concern to us). By anatomy, coronaries branch off the aorta, the left main coronary branches from the left coronary cusp, whereas the right coronary comes off the right coronary cusp, both of course at the root of the aorta just above the semi-lunar valve. In 80% of the people, there are 3 coronary arteries; the right coronary artery & the left main coronary which gives off 2 arteries: LAD (left anterior descending) and the left circumflex … so 3 coronary arteries.

In the other 20% of the people, there are 4 coronary arteries; the right coronary & the left main coronary which in this case gives off 3 branches: LAD, left circumflex and ramus intermedius,

In the right coronary we have a dominance issue were in 85% of the people we find the right coronary to be dominant (larger) than the left circumflex, in 10% the left circumflex is dominant and in 5% co dominance.

Usual sites of access for catheter: brachial, femoral or radial arteries through which we reach the aorta & then to the coronaries.

The urgency of this situation must be realized, we say that time is muscle → the more time you lose, the more muscle cells will die. Wherever the artery is blocked, necrosis of the tissue downstream will start within/at 90 minutes and this is why 90 minutes is considered to be the golden time to rescue the patient before necrosis starts in the heart muscle. And of course whatever damage occurs after that is irreversible since like the brain the cells don't regenerate.

Those were the main guidelines for PCI (Percutaneous Coronary Intervention).

As for the TPA (the thrombolytic agent), it must be given within 30 minutes from the time of administration into the ER.

Another important guideline: within 24 hours of patient's presentation of symptoms (this time has nothing to do with time zero that we mentioned before) we are allowed to go in and fix the artery; for example: if a patient calls you at 1 pm, telling you that he has chest pain since 12 am the last night & you administered him to the ER & he had ST elevation on his ECG (which in this case lasted for about 13 hours), then, you're allowed to do cath for him within 90 minutes of his administration in addition to giving him TPA within the first 30 minutes of his arrival to the ER … Of course, it's more preferable in cases of ACS to present as early as possible, but patients differ & some would wait before rushing to the hospital.

However, if more than 24 hours have passed upon the chest pain, the guidelines DO NOT support to administer the patient as an urgent case, why? Because if the artery has been occluded for that long, the benefits of catheterization will be much less & you'll gain nothing in turn but increasing the risks on the patient; because generally speaking, in emergency scenario, the medical team will be under-staffed, you're status at 2 am at night as a physician to treat this patient won't be optimal as your status at morning, also, if things went bad during cath or it turned up that the patient needs an open heart surgery, the surgeons won't be available this late … Actually, emergency procedures can't be without

risks, in addition to the fact that any procedure done at night won't be as the one scheduled to be done at morning!

-Does the thrombolytic agent given prevent other clots from forming?

No, it only takes care of the clot that's there and doesn't prevent other clots formation.

The success rate with the thrombolytic agent is 60 to 70% whereas with the catheter it is 90 to 95%. And so even if a patient did receive a thrombolytic agent, a catheterization must be done within 24 hours of patient's presentation. The results of thrombolytic agent's use aren't obtained immediately; 60 minutes are needed to see success of therapy, furthermore success *isn't* defined by complete resolution of the ST segment elevation, 50% of the baseline ST elevation is enough to call it successful along with the improvement of symptoms.

Factors that increase mortality for a patient with MI:

Hypotension (–like 80/50- that is because the patient is in cardiogenic shock, they are tachycardic), pulmonary crackles, elevation in jugular venous pressure, pulmonary edema, mechanical complication related to the MI (mitral regurgitation, VSD, diminished pulses and signs of stroke).

The Dr pointed out the ST segment on the ECG but we're not required to know how to read ECG of course yet, but this is how we localize the infarction according to which lead is elevated (ECG has 12 leads).

Again, for patients presented with chest pain with no ST elevations, the diagnostic markers that are used are cardiac enzymes mainly troponin (since it rises 4 to 8 hours after the injury and may remain elevated for 2 weeks). CKMB – Creatine Kinase- rises in 4 to 6 hours and peaks at 24 hours and remains elevated for 48 hours).

Pathogenesis of MI: upon the presence of risk factors → lipid rich plaque formation occurs in the sub-endothelial space (oxidized LDL goes in the sub-endothelial space) → this will attract macrophages → formation of foam cells → more oxidized LDL and more cytokine attraction for lymphocytes and monocytes… so now, there is a plaque with lipid core & lymphocytes in the sub-endothelial space. This plaque is surrounded by a very thin capsule (in nanometers) → rupture or fissuring of that plaque will occur at one time whether the plaque is triggered or not →leaking of the lipid core of the plaque which is highly thrombogenic → activation of coagulation cascade (intrinsic/extrinsic) → thrombus formation in-situ –at the site of rupture/ fissuring of the plaque, i.e. the thrombus didn't come from anywhere else - (not arterial-arterial or veno-arterial)

Characters of patient's presentation: classical retrosternal chest pain caused by ischemia (by classical we mean that the chest pain is described as heaviness), nausea, vomiting... The patient would be looking healthy most of the time (dry patient) with no signs of volume overload and the pain would occur suddenly.

If a patient has 70% occlusion/stenosis in the coronaries, then chest pain will be felt during physical activity such as climbing the stairs etc & relieved by rest or nitroglycerine... Why the pain? It's an issue of supplying demands, i.e. because not enough blood is reaching the cardiac muscle that's working and needs ATP & O2 → so there'll be an anaerobic respiration as a compensation due to lack of O2 → lactic acidosis → leading to anginal pain.

What are some other causes of chest pain? Reflux – heart burn, esophageal spasm, costochondritis –at the level of the ribs & the sternum-, pulmonary embolus, aortic dissection, hiatal hernia, pneumonia with pleurisy… but again, chest pain of angina is classical (described to be compressing & felt as heaviness).

2nd topic: Heart Failure

Symptoms are associated with volume overload, it is more of a congestive state: lower extremity edema, increase in abdominal girth, hepatomegaly, in males scrotal edema, crackles in chest when you use the stethoscope, dyspnea usually with minimal exertion (can happen at night: paroxysmal nocturnal dyspnea), orthopnea (is worse because it occurs right away when the patient lies flat as opposed to PND were it occurs *after hours* of laying flat on the back and so in PND, you are not extremely overloaded like in case of orthopnea).

Why does the heart failure occur?

Due to weakness in the cardiac muscle, which could be due to problems in systolic contractility or due to diastolic dysfunction, how can we tell which of which? Depending on the case scenario & the results from the echo test. However generally, regardless of the etiology we treat all the patients with heart failure the same way; if a patient has crackles, has dyspnea and has low oxygen saturation, then we give him oxygen & upon O2 supply the patient will be tachypnic to compensate and we also give him *Lasix* – made of furosemide- *which is a loop diuretic that functions upon the thick ascending loop of Henley, dumping 2Na & 2Cl, resulting in fluid drain-out from the lungs & other parts of the body (since water follows the dumped salts in urine that were blocked from reabsorption)… so lasix is a diuretic for fluid retention.*

Note: the use of diuretics is the clue to improve the clinical picture in patients with decompensated heart failure.

Clinical Cases:

1. 59 year old diabetic male with hypertension presented with 4 hour history of retrosternal chest pain associated with nausea and vomiting, on examination his blood pressure is 90/50 mmHg, heart rate is 75 bpm the rest of his examination is unremarkable. ECG shows ST elevation in LAD area.

-What are the risk factors? Hypertension, diabetes, age and gender.

-Presentation clearly shows that this patient has MI

- Destination management: (cath and PCI stent) or (thrombolytic agent). In the mean time, we give oxygen, aspirin, Plavix (antiplatelet agent), heparin, morphine, saline for his blood pressure if he is not dyspnic, if he is dyspnic we give vasopressors … So the official treatment of MI: dual anti-platelet therapy (aspirin and plavix) and anti-coagulant. Other treatments will come later such as statin and beta-blockers etc …

- Warfarin is no longer approved for coronary events, whereas for atrial fibrillation it is approved.

Question: all of the following statements are true except?

A. The occluded artery is the LAD.
B. Thrombolysis is inferior to PCI (true; because the success rate for thrombolytics is 60% – 70%, whereas in PCI, it's 90% - 95%)
C. Low pump pressure is associated with worse outcomes.
D. Waiting for a cardiac marker is a must before further management. **FALSE**
E. Caution in the use of nitroglycerin in the case of hypotension (true because nitroglycerine is a venodilator & coronary vasodilator)

2. 63 year old male with long history of hypertension, Dyslipidemia and long standing tobacco abuse presented to the ER with 4 hours history of severe chest pain, nausea, dyspnea and diaphoresis (profuse sweating). On examination the patient is in moderate distress, his skin is warm and dry, blood pressure is 95/60 mmHg, pulse is 70 bpm, neck veins are distended and there is a systolic murmur, the patient was given nitroglycerin which significantly lowered his blood pressure, ECG shows that the right coronary is occluded.

-Right coronary supplies the inferior surface of the heart and the right ventricle.

- Here, the infarction involved the right ventricle, but why the distention of the neck veins?

We know that the jugular veins drain into the S.V.C, which drains into the right atrium, which in turn, pumps the blood to the right ventricle, here, in this case, there's an infarction in the right ventricle leading to it's improper function, i.e. the blood will accumulate inside it & then regurgitates back to the right atrium back to S.V.C & then back to the jugular veins causing their distention.

Such patients in particular with right ventricular infarctions, can be given up to 10 liters of fluid with no problems, why? Because they need to receive more fluids in addition to the fact that when they're given nitroglycerine, it will cause a significant drop in their B.P

Which of the following represents the most like cause of this hypotension?

A. Papillary mucle dysfunction.
B. Aortic dissection with the involvement of the right coronary.
C. Cardiogenic shock.
D. Acute inferior wall MI with right ventricular infarction. ANSWER

3. 67 year old woman presents with substernal chest pressure, has lasted for 3 hours, the pressure has not gone back to normal despite administration of nitroglycerin, the nearest hospital with PCI is 45 minutes away, and the patient has history of hypertension and hyperlipidemia. On examination, temperature is 37, blood pressure is 146/92 mmHg, has tachycardia and respiratory rate is 18. She is uncomfortable, she has crackles in her chest, S1 is normal S2 is paradoxical, ECG illustrates normal sinus rythum…

   Most appropriate management in this case? This patient needs to be transferred to a PCI center.

The point of this question is to show that in a good percentage of patients presented with acute MI, the MI will be associated with heart failure, so due to the MI → acute heart failure, acute volume overload & pulmonary edema will present.

What is pulmonary edema: Normally: alveoli are gas chambers that have capillaries on their surfaces for gas exchange … what happens in pulmonary edema is that these chambers are going to be filled with water/ fluid –which should never happen- & by that, there'll be no gas exchange & the patient feels as if he's drowning in his own water!

The patient has to be supported by giving him diuretics.

To how extent is pulmonary edema bad? To the extent that the patient can't lay flat on his back during cath, so the cath team will intubate the patient & put him on a ventilator in the cath lab.

Note that the mortality rate is very high up to 80% in a case of combination of MI, decompensated heart failure & hypotension.