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We’ll talk today about ischemic heart diseases (IHD), and it’s a topic that concern every one of us. It’s a broad spectrum of diseases that affect the heart due to decreased blood flow to the myocardium; It’s the commonest medical cause of death worldwide (more than any other medical illnesses).

The doctor showed a slide that reviews the anatomy of heart arteries. We know that heart is a muscle that continuously pumping, starting early in the utero (in the 6-8 week) and continue all over life, and this muscle needs blood supply to provide energy (ATP), the size of this muscle (the heart) is about the size of your fist :’). Heart is supplied by two big arteries, one on the left and one on the right, the one on the left is a main stem that is divided into left anterior descending artery (LAD) and the circumflex artery and they mainly supply the left ventricle(LV). And on the other side we have the right coronary artery that supplies the right ventricle and the inferio-basal part of the left ventricle. so the largest arteries are left main and LAD.

Ischemia results when there is imbalance between supply and demand. Normally when myocardial blood demand increases this should be combined by increase in the coronary blood flow. Factors that determine the oxygen demand of the myocardium:

1. Heart rate (HR); the faster the HR the more oxygen required and vise versa. For that reason, a Pt. that have IHD would like to have his HR in a low rate, so we give him drugs to slow down the HR like Beta blockers (the corner stone in treating pts. with IHD, so if a pt. comes to you with IHD or not, you SHOULDN’T stop it abruptly to avoid rebound tachycardia which may precipitate angina or chest pain), Ca channel blocker.
2. Contractility; the stronger the contractility the more Oxygen requirement.
3. Wall tension in the ventricle in the diastolic phase(diastolic ventricular wall tension), the more the tension the more the oxygen requirement.
4. Ventricular muscle mass ( the bigger the muscle mass the more oxygen requirement).

Factors that determine supply:

1. Patency of the coronary artery (main factor); if patent= increase in blood flow during exercise( when the coronary blood flow can increase up to 8 times that during the rest), to meet this increase in the flow we need to have patent artery, else (if stenosed or diseased) the blood flow won’t increase(imbalance)>> the pt. will suffer IHD.
2. Hemoglobin levels; the lower the Hgb levels the less the oxygen supply, that’s why anemia can cause chest pain without a coronary artery disease because the oxygen carrying capacity of Hgb is decreased.
3. Myocardial oxygen extraction; normally, oxygen extraction of skeletal muscles like biceps or quadriceps… increases by exercise (at rest about 20% extraction up to 80% during exercise). In the myocardial muscle, the extraction is always high and fixed (55% whether in rest or exercise).
4. Arterial oxygen saturation (hypoxia).

What are the causes of IHD?

-the most common cause of IHD is atherosclerosis (تصلّب الشرايين)” 95%”. Atherosclerosis is a diffused disease that may affect in any artery in our bodies (carotid, vertebral, peripheral vascular, mesenteric arteries, renal …) and one of these arteries that is affected coronary arteries.

-less than 5% is a non-atherosclerotic causes of IHD like vasculitis (like rheumatoid arthritis ), and some vasculitis may affect the coronary artery and may lead to stenosis. Also, embolization, abnormality of the coronary arteries is a non-atherosclerotic cause; we know that the coronary artery arises from the aorta, if it arises from pulmonary trunk, this may lead to ischemia in early childhood (2-3 years old).

**KEEP IN MIND, the commonest cause of IHD is atherosclerosis.**

Atherosclerosis:

-it’s a process that starts early in life, and factors that affect it are the interaction between genetic predisposition and environmental factors (smoking, DM, HTN, stress, lack of exercise…).

-we have many factors that increase the incidence of atherosclerosis and we divide them into modifiable(reversible)and non-reversible.

# Modifiable are what we can treat and reverse, like smoking (passive or active), HTN(systolic or diastolic), hyperlipidemia(high LDL, low HDL and high triglycerides) and DM. (these are four classical risk factors of atherosclerosis), also dietary factor (intake of fatty acids, lack of exercise, obesity(specially truncal obesity ”metabolic syndrome and insulin resistance), homocystenemia, hyperuricemia.

# Non-modifiable risk factors; like a first degree relative that have IHD. We consider the family history is positive if the pt. has a positive family history of a first degree relative in a male less than 55 years old. (If a patient said that my father had it when he was 80, then it’s NOT a risk factor), and a first degree relative in a female less than 65 years old. Also, another non-modifiable risk factor is age (in males more than 55 and females more than 65).

# We need to concentrate more on the modifiable factors as those what we can modify :P and reverse.

# Presentations of IHD (how a pt. with IHD presents to us):

# -there is a broad spectrum of manifestations that range from asymptomatic to sudden death, and in between there is many presentations like stable or unstable angina, avute coronary syndrome or MI, HF, arrhythmias. All of these mentioned might be presentation of IHD.

# -Angina: as we said it’s a manifestation of IHD, and we have different types of angina:

# 1-stable angina (as we said, it results from imbalance between demand and supply, increase in demand without an increase in supply), this condition is characterized by: \**chest pain* that is precipitated by exertion or stress and anxiety (anything that increases the HR), the person says that he has chest pain when he walks and subsides when he rests or when he takes sublingual tablet.

# The commonest site of this pain is retro sternum (left side), and it may radiate to left shoulder, left arm, left forearm, little and ring finger, neck, throat, jaws, right shoulder, epigastric area or interscapular area, the pain might be firstly in these areas, like when a pt. says that he has pain in throat when he walks, or toothache when he walks (the doctor mentioned that he saw a pt. who had a toothache when he walks and the pain subsides when he is in rest, he consulted dentists but they just treated the tooth, then finally he came to our doctor who has found that this is angina). Sometimes the pain starts in the epigastric area and might mimic peptic ulcer disease.

# The character of this pain; in our countries the pt. might say في بلاطة, in foreign countries they say (elephant on my chest), or squeezing or burning sensation or dull aching (the pt. might say that he doesn’t feel ok when he walks, but he can’t describe the pain well).

# The pain lasts for 10 mins or less, and is relieved after about 5 mins of rest or by sublingual nitrate. The pain might be associated with nausea and rarely with vomiting and sweating, dyspnea and palpitation “less common”

# -angina is graded according to the activity that precipitate the angina, some will have pain when they walk for a long distance (class 2), some when they walk for 2 steps for ex.(class 3), some will have it during rest(class 4).

# -Examination of angina: there is no specific signs for stable angina, but we look for signs of risk factors (HTN, DM, hyperlipidemia) and we also look for signs and symptoms of atherosclerosis in other areas( like if he have atherosclerosis in carotid, we can ask about history of stroke, or he may have atherosclerosis in the lower limps arteries we ask about intermittent claudication “we ask if his legs hurt when he walks, he will describe the pain in muscles of his legs (muscle pain on exertion that is relieved by rest, which is a peripheral vascular disease mainly due to atherosclerosis).

# -Diagnosis of angina: is clinical diagnosis, history of chest pain during exertion which subsides at rest. There is a specific criteria to say it’s stable or unstable. Further investigations like ECG might be also used, but normal ECG will not exclude it or exclude any cardiac disease, for ex. A MI pt. might have a normal ECG. Sometimes we use exercise/stress/treadmill ECG to induce ischemia (by increasing the oxygen demand and see if any imbalance and changes happen ECG ) and we do him echo and see what segment is affected, contractility will be slow, diminished hypokinezia?, blood glucose, lipids, and finally conangiograph, and we can see coronary arteries from inside (we see the plaque and stenosis) and accordingly we treat.

# -Treatment; when you treat any disease you should have objective in your mind, you should look for the risk factors and correct them, there is no point of treating an ischemic patient as long as he continues smoking, so correct this risk factor (smoking), BP is another risk factor, control his diabetes, his weight and physical exercise. Also, correct other factors that increase the heart rate like: anemia or arrhythmias. Then, you give drugs that improve prognosis to prevent MI for ex. or to regress the atherosclerosis like: statins (a key role in the treatment, a drug “antilipid” that decrease the level of LDL and increase HDL ,our goal is to reach 100mg/dl LDL and nowadays this no. is being lowered 70mg/dl), aspirin (antiplatelet, any pt. with atherosclerosis should be on a lifelong aspirin), ACE inhibitor (that regress or decrease the progression of atherosclerosis). Then, we move to the symptomatic line (treatment of symptoms), drugs that relieve symptoms but have nothing to do with prognosis, like: nitrate, Ca channels blocker, triazidine? , Beta blockers; (relieve symptoms by decreasing HR we decrease the oxygen requirement, also Beta blockers have a negative inotropic effect “decrease the contractility thus decrease the oxygen requirement”, the third mechanism of Beta blockers is decreasing the HR by increasing diastolic time of cardiac cycle “remember, usually 80% of coronary perfusion happens in the diastolic phase. Other organs have perfusion during the systolic phase. but we are here talking about coronary perfusion; in systolic phase the muscle is contracted and there will be resistant of blood flow through this contracted muscle and during diastolic phase the muscle is relaxed and the resistance of blood flow to the heart is decreased”

# So, drugs like Aspirin is used to decrease the risk factor, nitrate sublingual, beta blockers, statins, if pt still symptomatic we can add CA channel blockers and we can do angiogram and correct accordingly otherwise extent or coverage

Prognosis; depends on many factors, such as:

1. The site and extent of lesion.
2. whichartery is affected ( is it in a small vessel or small branch or a big artery like LAD )
3. Ejection fraction (he lower the contractility/ or the lower the ejection fraction, the worse the outcome).

If the lesion is in the left main artery, 12 patients may die within 1 year if the problem is not corrected properly.

Other spectrum of IHD or other manifestation is what we call :

-**Acute coronary syndrome (ACS):** what is important to us is that ACSa MI or unstable angina, it’s a sudden ischemia to the myocardium due to rupture of the plaque or atheroma in the artery which will lead to total or subtotal closure of coronary artery, if it’s totally occluded then we’ll have MI (ST- segment elevation MI), but if the coronary artery is sub totally occluded you will have unstable angina.

In ST elevation coronary syndrome usually the artery **will be totally occluded** but how the artery will becomeoccluded**?** The first step in ACS is plaque rupture; atherosclerosis causes narrowing of the vessels by accumulation of lipids sub-intimal ,this accumulation is called *atheromatous plaque*,this plaque maybe ruptured, eroded, or fissured (80% of ACS is due to plaque rupture), this will lead to many sequences , the first one is about platelets**,** platelets are found normally circulating in vessels , when we have plaque rupture, they will recognize the ruptured rough area, trying to do healing by setting on that area, first there will be *platelet adhesion* by many mediators and factors (such as Von Willebrand (vWF), glycoprotein 1a2a which will act like a glue) , then we’ll have *platelet activation* by many mediators “e.g. thromboxane a2, thrombin, ADP, serotonin, histamine..” ,this activation will cause many projections to be presented on the surface and it will look “angry like”, this will cause the surface area to increase thousand times,

and this activation will lead to expression of new molecules on the surface, such as glycoprotein 2b3a receptors, these receptors are linked together by fibrinogen ,which will lead to*platelet aggregation .*

SO we have *platelet adhesion >>activation >>aggregation*>> the aggregation will lead to what we call *white thrombus*>> which will *activate the intrinsic coagulation system.* Any injury in any vessel tries to heal by this process

We have primary hemostasis (by platelet) and secondary hemostasis (by coagulation factor)

Then, the thrombus will start to form in the lumen of the artery by platelet plug (white thrombus), and then the activation of the intrinsic coagulation system which leads to the formation of fibrin clot and so on until it will lead to total occlusion of the coronary artery causing ST segment elevation MI.

**If the artery is subtotally occluded** ; non-ST segment elevation ACS, either MI if there is a leak in trobonin or unstable angina if there’s no leak of trobonin. Note: when thrombus form ,,if will increase in size within few min.

**MI is the commonest medical cause of death.**

-Presentation of patient with MI*;* it doesn’t depend on the type of MI whether its ST or non-ST elevation. It’s characterized by sudden severe prolonged chest pain that lasts more than 30 mins , it (MI) is the no.1 differential diagnosis in pt. with this characteristic.

-The site of pain is the same as angina and it could radiate to same areas also “e.g. left shoulder, left arm, forearm, leg, jaw, epigastric pain, interscapular area”, but its more severe, it’s the worst pain a person can suffer through life, he feels like he’s going to die, usually associated with nausea, vomiting, profuse sweating, and dyspnea.This is the classical presentation of MI. The Pain comes at rest, unlike angina. The highest risk period is in the early morning hrs. in most vascular accidents including MI, due to many factors; the platelets, coagulation system, thrombolytic system all are presented more in the early morning.

15% of pts. come with painless MI, they are presented with shock hypotension, they have low blood pressure without known reason, there is no bleeding, diarrhea, vomiting or any cause of volume decrease. Or they may be presented with pulmonary edema, heart failure, arrhythmias, ketoacidosis or syncope. Painless MI is seen more in diabetic or elderly patient.

-Examination:there is no specific sign for MI. pts look irritable, sweaty ,pain, vital signs, tachycardia is common due to pain, stress, catecholamine is high, bradycardia if there is heart block, blood pressure may be normal, hypo or hypertension “depending where the infarction happened”, we may listen to S3 S4 heart sounds, pain in the same side as angina.

-Diagnosis is by

1-History taking.

2-ECG (we have classical ECG changes, the dr. showed some examples how ECG of ST elevation and non ST elevation MI look like).

3-Cardiac markers (in the past we used CPK, nowadays we mainly use troponin t or troponin I, start detecting for 6 hrs. And now we have a new one, more specific, and positivity start detecting within 1 hr. Troponin lasts up to 2 weeks “10-14 days” after elevation, while CPK returns to normal after 3 days (continue to be elevated up to 72 hrs)).

-Treatment of MI ; should start at home, if pt. has severe chest pain for more that 30 mins, suspect MI and you should be quit, ask to be transferred to nearby hospital by ambulance, if the pt. is not taking aspirin you can give him aspirin at home, avoid any exercise or exertion to pt. because the pt may develop ventricular fibrillation and have cardiac arrest at any time, and there is no way to convert this ventricular fibrillation other than defibrillation which is not found at home, that’s why he should be transferred by well equipped ambulance to hospital, and there on the emergency room after quick history, assessment, and medical examination, they put IV canula and oxygen, take ECG, if the pt is not hypotensive they use sublingual nitrates, aspirin and other antiplatelets, morphine to relieve his pain We give the pt. something called **MONA:** morphine, oxygen, nitrate and aspirin, because there’s no contraindication on any of these drugs.Then you should think about opening the artery, and this is the only way that improve the prognosis, we have 2 ways for opening the artery:

1-PCI: primary coronary intervention, if you have it, it’s better than thrombolytics, and it’s used more nowadays. We do it by doing first catheterization, and we take shot to the blocked artery and open it (it’s the best way to open the artery).

2-thrombolytics**:** like streptokinase, TPAse (it was used more in the past), we use it if the pt. is far away from a hospital that can do catheterization or if there is no cath lab.

\*if you have the facility to do PCI and there will not be a delay to the pt for more than 90 mins after presentation, then it’s better to do it because it has less mortality rate and complications.

-Other form of ACS is **unstable angina**, it’s when there is new onset angina in 8 weeks, and pt will have chest pain with minimal exertion. Or it the pt. has angina in the past, but the pain start to increase, and lasts longer, and it’s onset is with less exertion (change in the frequency, pattern, or severity of already presented angina) then we also call it unstable angina.

The pt is with a plaque rupture due to subtotal occlusion or small thrombus in the coronary artery has unstable angina or he has non st elevation MI “have chest pain, ECG have no st elevation, but is troponin positive)

-Any pt with ischemic chest pain and positive cardiac marker>> has MI whether it’s ST segment elevation (totally occluded, long term for about 3 yrs) ,on non-ST segment elevation (subtotal occlusion, short term, better )

-Treatment; we treat them again by antiplatelets, anticoagulants, nitrate, beta blockers, statins (given to pts with any form of ischemic heart disease), no thrombolytics here. Depending on the situation, either we take the pt. to the cath. lab and do urgent catheterization, or we delay him to the second morning, this depend on many factors, vital signs, severity, recurrence, hypotension.

-You as a dentist should know how pts with IHD present, and what factors will precipitate angina or ischemic insult in your pt. You should put in your mind that pt with IHD should *avoid tachycardia*, because it’s not will tolerated in those patients (tachycardia increase the oxygen demand). So if you have a patient with history of angina, his heart rate should be low, and you give him beta blockers, and you avoid anything that would lead to tachycardia to this patient, so you give him good sedation and analgesia, avoid giving him adrenaline (because if you give adrenaline submucosally, some adrenaline may get into veins by mistake, and this induce tachycardia, arrhythmias, and patient may have chest pain).

The pt. shouldn’t discontinue drugs that lead to bradycardia like (diltiazem, adopten, dropamine, beta blocker specially ) .

We also should antiplatelets which should be given to any type of IHD, aspirin mainly if he has MI. a patient who has stent usually are kept on 2 antiplatelets; aspirin and other antiplatelets that work on at different side, like clopidogrel(trade name is Plavix) or ticagrelor (trade name Brilinta) (… “الباقي ما عرفناهم”

You ask the patient about drugs he is taken, and if your procedure will lead to bleeding “and the bleeding maybe difficult to control” it’s better not to do it while he’s taking antiplatelets, you delay it if your procedure will not lead to bleeding you can do it.

You should not stop the antiplatelets abruptly in patients who has recent MI, or has a recent stent (he should have 2 antiplatelets for at least 1 yr)

-rarely we use anticoagulants in patients with ischemic heart disease, commonly we use antiplatelets (because as we said those patients have atherosclerosis, which may rupture and cause platelet aggregation, so we prevent it or regress it by giving antiplatets).

-anticoagulants “like warfarin” is indicated mainly in patients with prosthetic heart disease (manufactured valve) , or patient who has atreial fibrillation, stroke, thrombus in left ventricle, MI

\*\*Sorry for being late. Some few words neither of us could hear.

Forgive us for any mistake ☺