CORONARY CIRCULATION DISORDERS

(CHAPTER 24)

QUESTION 1

What is the cause of angina pectoris? How is angina described?

Angina pectoris is defined as **ischemic cardiac pain** caused by an imbalance in the **supply and demand** of **oxygen** to the heart.

The **myocardium** (heart muscle) receives oxygenation through the four **coronary arteries** that run along the **outside** of the heart.

If these arteries are delivering **less oxygen** than the heart needs to operate, the result is **ischemia** and thus **angina** (pain.)

Angina falls into one of **three categories**:

Stable angina is **predictable**, usually occuring with **physical exertion** and thus increased oxygen **demand** by the heart.

As such, it is relatively easy to treat with **nitrate** vasodilators such as NTG, which can be taken **before** exertion to prevent the angina from occurring.

Unstable angina is **more serious** than stable angina and occurs **unpredictably**, even at rest. This suggests that the heart's **baseline** oxygen needs are not being consistently met.

Unstable angina can still be treated with nitrates, but they must be taken in **response** to the onset of chest pain, due to its unpredictable nature.

Variant angina (a.k.a. Prinzmetal angina) is caused by episodes of **vasospasm** which temporarily reduce blood flow to the heart.

It is **less common** than traditional occlusive angina, and often occurs in those who suffer from other vasospastic disorders such as Reynaud's disease.

QUESTION 2

Give some examples of cardiovascular disease.

cerebrovascular accident (CVA, a.k.a. "stroke") – failure of O_2 delivery to the **brain**, resulting in permanent neurologic damage

transient ischemic attack (TIA, a.k.a. "mini-stroke") – short episode of brain ischemia, resulting in symptoms < 1 hr and no permanent damage

myocardial infarction (MI, a.k.a. "heart attack") – failure of O_2 delivery to the **heart**, resulting in **necrosis**

angina – ischemic chest pain resulting from insufficient O_2 to the heart

claudication – pain, numbness, or tingling in the legs due to insufficient tissue perfusion (literally means "limping")

QUESTION 3

Distinguish between myocardial ischemia and myocardial infarction (MI.)

Ischemia and **infarction** both result from tissues being starved of the oxygen that they need to perform cellular metabolism.

The difference is that ischemia refers to the **general state** of having poor blood supply to an area, whereas **infarction** implies **permanent** tissue damage (necrosis.)

In the context of the heart, **myocardial ischemia** results in **angina**, whereas **myocardial infarction** can result in permanent **heart failure**, **conductive blocks**, or even **death** if not promptly treated.

QUESTION 4

Define thromboembolism.

Remember that the root "**thrombo**-" means **clotting**.

An **embolism** is a **moving object** or material that has become lodged in a blood vessel, causing an occlusion.

Combining these terms, a **thromboembolism** is an **embolism** (material lodged in the blood vessels) in which the object is a **blood clot** from elsewhere in the body.

Thromboembolisms represent a major source of acute vascular occlusions, including ischemic CVA, MI, pulmonary embolism (PE,) etc.

QUESTION 5

List the three key risk factors for coronary artery disease (CAD.)

hypertension – chronic high BP weakens the walls of the arteries, causing them to become stiff and narrower

hyperlipidemia – cholesterol is a major component of **atherotic plaques**, which occlude blood flow and can rupture causing acute clots

smoking – risk factor for pretty much everything; promotes clotting, encourages vasospasm and arrhythmias

QUESTION 6

What is C-reactive protein (CRP?) How is it linked to CAD?

C-reactive protein or CRP is an **inflammatory marker** that can be suggestive of the presence of atherotic lesions such as those seen in CAD.

It is worth noting that CRP is **not** a specific marker of vascular damage, and can be elevated for a variety of reasons from infection and inflammatory diseases to cancer, or even pregnancy.

There is a **known link** between increased baseline level of CRP and cardiovascular disease, but elevated CRP alone **does not** confirm a diagnosis of CAD.

CRP is **not** recommended as a screening test for CAD in healthy individuals without risk factors, due in part to its poor specificity.

QUESTION 7

Which of the plasma lipoproteins is protective against CAD? Why?

Lipoproteins are microscopic "bubbles" of protein responsible for carrying lipids, which are normally not water-soluble, through the bloodstream.

They are produced in the **liver**, and fall into categories based on their **size** and **contents**: from very-low density (VLDL) through intermediate (IDL) and high-density (HDL.)

These transport proteins start in the form of **VLDL** and travel through the bloodstream, where they gradually become LDL, then IDL, and finally HDL as the lipids they carry are taken up into the cells.

Having a **higher HDL** level is considered beneficial, because these "bubbles" are nearly empty and thus can **absorb** free-floating lipids to be returned to the liver, protecting against the formation of **fatty deposits** on the arterial wall.

QUESTION 8

Why would diabetes mellitus be a risk factor for CAD?

The link between **diabetes** and CAD is complex, but it is known that diabetes results in a **2-4 times greater** risk of CAD-related mortality.

Part of the process responsible for this increased risk involves an enzyme called **hormone-sensitive lipase** (HSL,) which is responsible for **mobilizing lipid stores** in situations where there is insufficient energy production from carbohydrates alone.

HSL is called "hormone-sensitive" because it is **inhibited** by insulin; when there is sufficient energy production from glucose, fat mobilization is **not needed**.

If insulin production is diminished, as occurs in DM, HSL activity is **increased** and serum lipid levels **increase**.

As we've mentioned previously, high lipid levels in the blood contribute to the formation of fatty deposits and plaques.

QUESTION 9

How can CAD be prevented?

Prevention of CAD is centered around promoting a **healthy lifestyle** and controlling **risk factors**:

- Control **blood pressure**, plus **lipid** and **glucose** levels
- Maintain a healthy, **balanced diet** and ideal **weight**
- Maintain an **active** lifestyle with adequate exercise
- Complete cessation of **smoking** in patients who smoke

QUESTION 10

Why would atherosclerosis of the coronary arteries compromise the heart?

Atherosclerosis of the coronary arteries **narrows the lumen**, creating a smaller passage for blood and thus **reducing the blood supply** to the heart.

This can result in **myocardial ischemia** and thus **unstable angina**.

Additionally, advanced lesions can **rupture**, triggering intravascular **clotting** which can suddenly occlude an entire coronary artery.

This results in severe oxygen depletion of the distal heart muscle, and ultimately **myocardial infarction**.

QUESTION 11

Does the decline of body organ systems with age relate to atherosclerosis?

Yes. Gradual narrowing of the arteries and the associated decrease in blood flow is believed to contribute to decreasing organ function with age.

QUESTION 12

How can advanced CAD be treated? What medications can help stop the progression?

Lifestyle modification is always a great place to start: **dietary changes** such as decreasing fat and cholesterol intake, controlling **body weight**, and **smoking cessation**.

In addition, some **medications** can help manage CAD:

Statin therapy (e.g. atorvastatin/Lipitor, simvastatin/Zocor) influences the liver to reduce blood lipid levels

Antihypertensive drugs such as CCBs, ARBs, ACE inhibitors, beta-blockers, and diuretics can slow the gradual weakening of the arterial wall from high BP

QUESTION 13

What are achievable goals for lipid-lowering therapy? When is it prescribed?

Statin therapy is prescribed to control lipid levels in those with **hyperlipidemia**, ultimately to reduce risk of cardiac-related morbidity and mortality such as MI.

Medication can be used to reduce lipid levels by **over 50%** if sufficient dosage is well-tolerated by the patient.

Remember the adverse effects of statins from pharm: **myalgias** (muscle pains) and occasionally **rhabdomyolysis** (breakdown of skeletal muscle.)

Be especially wary of severe side effects if a high dose is required for therapeutic effect.

QUESTION 14

Describe some of the tests used for acute coronary syndrome (ACS.)

Acute coronary syndrome (ACS) refers to a **group of symptoms** that occur when there is a disruption in oxygenation of the heart: **chest pain**, **dyspnea**, and sometimes **nausea**, **dizziness**, or **diaphoresis**.

Remember that a **syndrome** is a group of symptoms that tend to occur together, not necessarily linked to one specific cause.

In ACS, we want to determine **why** these symptoms are occurring as quickly as possible so that we can intervene appropriately—is it **angina** or an **infarction**?

Some tests used for this purpose:

electrocardiogram (EKG/ECG) – changes in electrical activity can reveal possible infarction

troponin I & T – elevated serum levels indicate cardiac muscle death, e.g. infarction

creatine kinase (CK/CK-MB) – non-specific marker for muscle (incl. skeletal muscle) death

C-reactive protein (CRP) – protein synthesized in the liver as a general response to inflammation

QUESTION 15

What is the goal of treatment in ACS?

The golden rule of ACS treatment: **"Time is muscle!"**

When blood flow to the heart is interrupted, we **must** restore oxygenation **as quickly as possible** to reduce or prevent permanent heart damage.

QUESTION 16

Describe percutaneous coronary intervention (PCI.)

Percutaneous coronary intervention is a minimally-invasive procedure for restoring and maintaining blood flow through the coronary arteries.

It is performed in a specialized unit called a **catheterization lab** ("cath lab") under continuous XR guidance with contrast.

In a PCI, a **guide wire** is inserted intravascularly through the radial or femoral artery, and advanced to the point of the blockage in the coronary arteries.

Once in place, a **balloon-tipped catheter** is passed over the guide wire and **inflated** within the narrowed vessel, physically **forcing the vessel open.**

In addition to this process, known as **balloon angioplasty**, PCI also involves the placement of a permanent **stent** which continues to hold the lumen of the vessel open after the catheter is removed.

Note that the plaque is **not removed** during a PCI. The obstruction is merely forced towards the walls of the vessel, and the stent prevents the vessel from collapsing back.

QUESTION 17

Describe a coronary artery bypass graft (CABG.)

A **coronary artery bypass graft**, CABG (pronounced "cabbage",) or "heart bypass" is a surgical procedure to permanently route blood **around** an obstruction in the coronary arteries.

The heart is either stopped or partially immobilized, and one or more veins taken from elsewhere in the body (such as the saphenous veins in the legs) are **grafted** to bridge the aorta to the distal side of the obstruction, allowing blood to **bypass** the obstruction and restoring perfusion of the distal tissue.

Multiple grafts can be performed during the same surgical procedure, providing routes around multiple obstructions. For example, a CABG x3 or "**triple bypass**" consists of grafts to **three branches** of the coronary arteries.

QUESTION 18

List some other therapies used for ACS.

"**MONA**" acronym:

- **Morphine** for acute pain relief
- Supplemental **oxygen** to offset poor perfusion
- Sublingual **nitroglycerin** to dilate coronary arteries and impove blood flow
- **Aspirin** to reduce platelet aggregation and prevent worsening of any clots

QUESTION 19

Describe some complications of ACS.

ACS is an **emergent**, **life-threatening** condition that demands **immediate treatment**.

Prolonged ischemia of the heart can cause necrosis (**infarction**) which can result in chronic **heart failure** due to muscle loss or **dysrhythmias** due to disruption of the heart's electrical pathways.

In severe or untreated cases, these dysrhythmias can include "**lethal rhythms**" such as ventricular fibrillation, which result in **cardiac arrest** (failure of O_2 delivery to the **entire body**.)

Once this occurs, there is a rapid onset of ischemic damage to every organ in the body, with the **brain** and **kidneys** being among the first affected.

Ultimately, prolonged disruption of the circulatory system can result in **neurological damage**, **organ damage**, and **death**.

Moral of the story: ACS is **serious** and there is **no margin** for wasting time in diagnosis and treatment.

QUESTION 20

Intraventricular blocks interfere with what function of the heart? What would you expect to observe in a patient with an intraventricular block?

Intraventricular blocks ("between the ventricles") interfere with the conduction of electrical signals from the **atrioventricular node** (AV node) to one or both **ventricles**, resulting in changes in **rhythm**.

Signs include a **widened QRS complex**, **QRS inversions**, or **wide, inverted T-waves**, indicating that the ventricles are not contracting **together**.

QUESTION 21

Are aneurysms only seen in blood vessels? Describe some types of aneurysms.

No. While aneurysms of the blood vessels, such as the aorta, are most common, they can occur **anywhere** there is vascular endothelium.

Atrial septal aneurysm (ASA) or **venticular septal aneurysm** (VSA) occur when the septum of the heart **bulges** towards the left or right side.

The heart wall can also bulge out into the surrounding tissue, such as in **left ventricular aneurysm**.

The **most common site** for aneurysms, however, is in the blood vessels, and particularly in the **abdominal aorta** (due to large diameter and lack of rigid support from abdominal organs.)

The **thoracic aorta** and blood vessels of the **cerebrum** are also relatively common sites.

Aneurysms typically fall into one of three categories:

- **Fusiform** – vessel wall bulges out in **all directions**
- **Saccular** – vessel wall bulges out in **one direction**, forming a "sac-like" protrusion
- **Dissecting** – blood infiltrates **between the layers** of the vessel wall, behind the endothelium

QUESTION 22

What is Dressler syndrome?

Dressler syndrome refers to a relatively rare **complication of myocardial infarction** in which there is **inflammation** of the pericardium (**pericarditis**) after the patient recovers from the MI.

When it does occur, symptoms usually begin within 2-3 weeks of the initial MI and include **fever**, **chest pain**, and **orthopnea**.

QUESTION 23

What are cardiac tamponade and pulsus paradoxus?

Cardiac tamponade is an **emergent** condition in which a **buildup of fluid** in the pericardium **compresses** the heart from the outside, interfering with its normal pumping action.

As mentioned in the unit on shock, tamponade can result in **obstructive shock** and ultimately **cardiac arrest** if left untreated.

Pulsus paradoxus is a diagnostic sign pointing to tamponade, defined as a **sudden decrease in systolic BP** that occurs **during inspiration**, due to the inflated lungs **exerting pressure** on the fluid-filled pericardium and **further constricting** the heart.

QUESTION 24

Describe mitral valve regurgitation. What conditions might lead to this disorder?

Mitral regurgitation occurs when the **mitral valve** between the left atrium and left ventricle **fails to close** completely after diastole, allowing blood to flow **back into the atrium** when the heart contracts.

The most common cause is **mitral prolapse**, in which the papillary muscles fail to hold the valve in place during systole, causing it to be forced open in the **opposite direction**, into the atrium.

It can also be caused by genetic disorders that affect the development of connective tissue, such as **Marfan syndrome**, or as a sequela of **rheumatic fever**.

Clinically, patients suffering from mitral regurgitation present with a **pansystolic murmur**.

QUESTION 25

Describe some of the consequences of mitral valve stenosis.

Mitral stenosis is a condition almost always associated with **rheumatic heart disease**, which results in decreased **forward** flow of blood from the left atrium to the left ventricle and thus **decreased diastolic filling**.

Unlike mitral regurgitation, mitral stenosis results in a **diastolic** murmur, although both cause a similar **decrease in cardiac output**.