University of Basrah
AL-Zahraa College of Medicine

Ministry of higher Education and Scientific Research

CardioRespiratory Care Block

Lecture title: Hyperlipidemia &Cardiovascular Disease

Date: 30/1/2022

Dr. Zainab M.Mohammed

CRC Block Staff:

Dr Firas Rashid Sayil (block leader)
Dr. Ahmed Bader
Dr Nawal Mustafa
Dr Zainab Almnaseer
Dr Farqad Alhamdani
Dr Ban M. Saleh
Dr Amani Namaa
Dr. Mohammed Adil
Dr Assad Hassan
Dr Ahmed Qassim
Dr Safa Asaad
Dr Mustafa Ghazi
Dr Ilham Mohammed
Dr Zainab Muzahim

References as in the workbook and classroom.

For more detailed instruction, any question, cases need help please contact block leader at the CRC Google classroom or email.

Hyperlipidemia

- Is an acquired or genetic disorders that result in a high level of lipids (cholesterol and triglycerides) circulating in the blood.
- **Lipids** are defined as organic compounds that are poorly soluble in water but miscible in organic solvents.

Lipids act as

1) Energy stores (triglycerides)

2) Structural components of cells (cholesterol and phospholipids).

They also have specialized functions (e.g. as adrenal and sex hormones).

Two important lipids, cholesterol and triglycerides, being insoluble in water are transported in the blood by lipoproteins.

A. Cholesterol present in the diet, but it is mainly synthesized in the liver and small intestine, the rate-limiting step being catalyzed by HMG-CoA reductase.

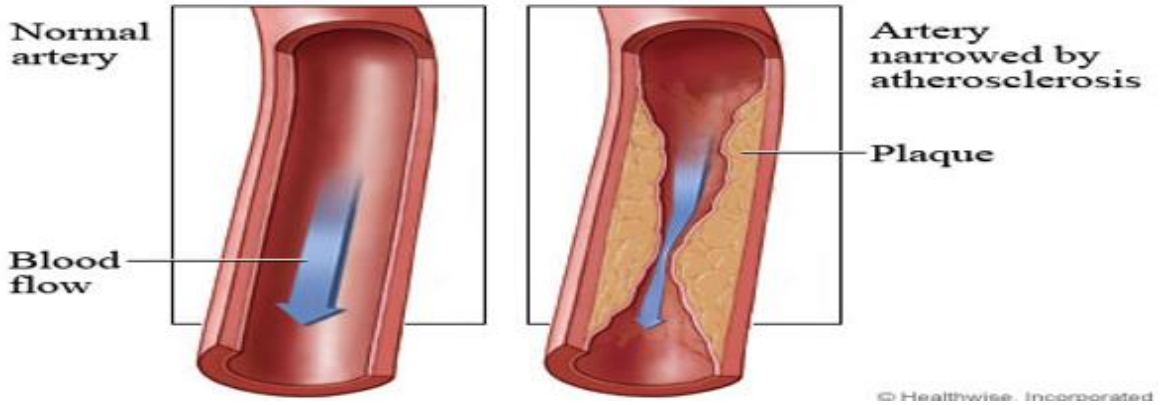
❖High-density lipoproteins **HDL** is called ‘good’ cholesterol, this is because it gets rid of the ‘bad’ cholesterol from blood.

❖ Low -density lipoproteins **LDL** is called 'bad' cholesterol. This is because its associated with increases risk of having a IHD or stroke.

Why is high cholesterol bad for the body?

LDL Transport cholesterol from the liver to the cells around the body.

Too much bad cholesterol (LDL) → plaque (deposit fatty material) on the inside walls of the arteries → narrowing the lumen of the artery → plaque disruption → Thrombus → MI or ischemic stroke.

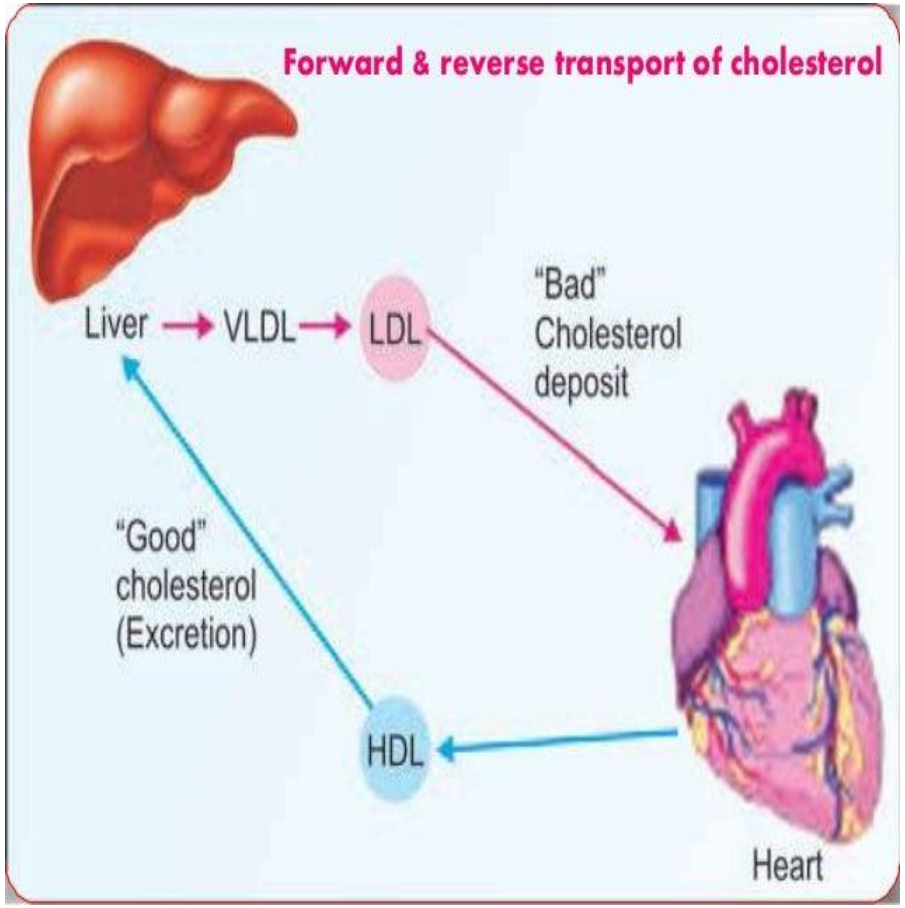


High-density lipoprotein HDL -cardio-protective

❖The transport of cholesterol from peripheral cell to the liver involves HDL particles, in a process called **reverse cholesterol transport**

❖Increased atherosclerotic plaque stability

❖ protection of LDL from oxidation, and maintaining the integrity of the vascular endothelium.



B. Triglycerides These are fatty acid esters of glycerol, and are the main lipids in the diet. Endogenous triglyceride synthesis occurs in the liver .

❖Triglycerides can also contribute to the narrowing of the of arteries, increasing the risk of CVD.

Factors affecting cholesterol

Diet : saturated fat, tend to raise plasma cholesterol.

Exercise: Regular exercise tends to cause a rise in plasma HDL

Age: plasma cholesterol rises with age.

Sex: In pre-menopausal women, plasma total cholesterol is lower than in men, and plasma HDL cholesterol is higher. These differences disappear after the menopause.

Increased cholesterol concentration (hypercholesterolemia) may be due to:

- genetic factors : Familial Hypercholesterolemia (FH).
- environmental factors:
- combination of the above:
- other diseases (secondary): hypothyroidism ,nephrotic syndrome, cholestasis e.g. primary biliary cirrhosis, drugs e.g. ciclosporin and

Why Get Tested?

1. To assess the risk of developing cardiovascular disease (CVD).
2. To monitor treatment of unhealthy lipid levels.

Test Preparation

❖ **Ideally fasting samples should be requested.**

The patient fasts overnight -for around 12 h (allowed only to drink water) .Although plasma cholesterol concentration is little affected by fasting **(as it mainly from endogenous source**), triglyceride concentrations rise and HDL cholesterol concentration

❖ **Diet**

The patient should be on usual diet for a couple of weeks preceding the test.

❖ **Timing**

Plasma lipids should not be assessed in patients who are acutely ill, for example acute myocardial infarction, as plasma cholesterol concentration may be decreased due to the acute-phase response. Wait for about 3 months after the event, unless the sample is taken within 12 h of an event, a ‘true’ result may be obtained.

What is being tested? Lipid profile :

1. **Total cholesterol:**
   measures the cholesterol in all the lipoprotein particles.
2. **High-density lipoprotein cholesterol (HDL-C):**
   measures the cholesterol in HDL particles.
3. **Low-density lipoprotein cholesterol (LDL-C):**
   measures the cholesterol in LDL particles.
4. **Triglyceride:**
   measures the triglycerides in all the lipoprotein particles. (most is in the very low-density lipoproteins (VLDL).

Some other information may be reported as part of the lipid profile. These parameters are **calculated from the results of the tests listed above.**

- **very low-density lipoproteins (VLDL-C):**
  calculated from triglycerides / 5
- **Non-HDL-C:**
  calculated from total cholesterol minus HDL-C

LDL cholesterol concentration to be calculated (Friedewald equation)

LDL cholesterol = total cholesterol – HDL cholesterol - [triglyceride]/5

This equation makes certain assumptions, that :

the patient is fasting

the plasma triglyceride concentration does not exceed 400 mg/dl

Coronary artery disease and prevention:

the importance of lipid lowering

❖Coronary artery disease remains one of the major causes of morbidity and mortality in world.

❖The major risk factors are :hyperlipidemia, hypertension ,smoking, diabetes mellitus, a family history of premature coronary heart disease and obesity.

❖All patients with established vascular disease **(secondary prevention)** should be treated with lipid-lowering drugs, in addition to lifestyle interventions (discontinue smoking, adopt a healthy diet and exercise)

- aim for a LDL cholesterol concentration of about **77 mg/dl**
- Total cholesterol concentration of about **155 mg/dl**

❖In terms of primary coronary heart disease prevention, the use of a cardiovascular risk factor assessment may be helpful to determine if a lipid lowering drug is indicated.

Drugs used to treat hypercholesterolemia:

1. Inhibitors of endogenous production (Statins) ⟹ inhibit the rate-limiting enzyme of cholesterol synthesis HMG-CoA reductase,
2. Inhibitors of intestinal cholesterol absorption (ezetimibe).
3. Inhibitors of bile reuptake.(cholestyramine).


Thank you