Lipid Physiology & Disorders

435 medicine teamwork

[Important | Notes | Extra | Editing file]

Lecture Objectives:

- Introduction
- Lipoprotein Classes
- Physiology of Lipid Metabolism
- Dyslipidemia Types & Etiology
- Clinical Findings & Associated Risks with Types of Dyslipidemia
- Management of Dyslipidemia

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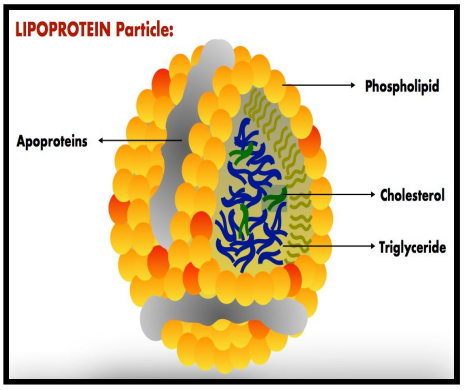
References: Doctors' Slides+Davidson+Step-up

Lipid Physiology

Introduction:



- Lipids, such as cholesterol & triglycerides, are insoluble in plasma.
- Circulating lipid is carried in **Lipoproteins** (main content of lipids) that transport the lipid to various tissue for:

1. Energy utilization
2. Fat deposition
3. Steroid hormone production main content of steroid is cholesterol, and steroids are secreted from the adrenal gland. so if you have a problem with the cholesterol you will have a problem with the adrenals.
4. Bile acid formation Essential for the absorption of 1) Vitamins 2)Lipids

- The lipoprotein consists of **esterified & unesterified cholesterol**, **triglycerides**, **phospholipids** and **proteins.**

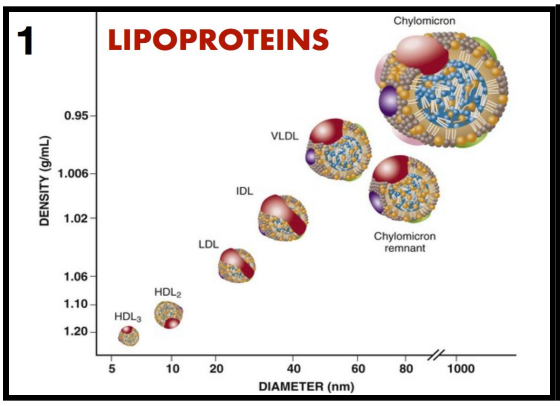
-LDL may deposit in the wall of blood vessels, thus causing atherosclerosis. This leads to IHD & Stroke.
-Hypertriglyceridemia can present as pancreatitis.

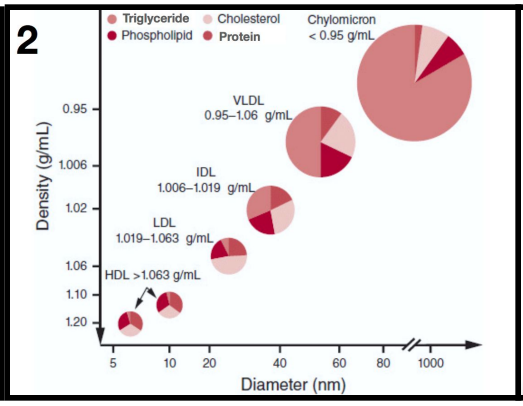
Classification of Lipoproteins:

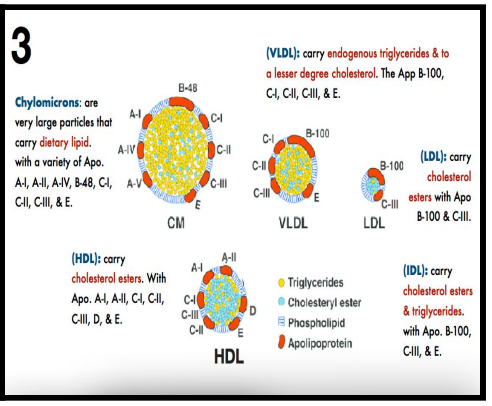
- The five major ***lipoproteins*** are presented below:	***Apolipoproteins*** are presented below:
The size gets smaller, Chylomicrons are the largest → HDL is the smallest. Note as you go down in size you decrease the amount of TAG. They differ in size, content and dimension. Ester Cholesterol is manufactured in the liver (IDL,LDL,VLDL) • **Chylomicrons: A) Source :** Gut **B) Major lipid :** are very large particles that carry **dietary lipid C) Apoprotein :** with a variety of Apo A-I, A-II, A-IV, **B-48**, C-I, C-II, C-III, & E. . \ Very raised triglycerides concentration (>6 mmol/l) cause a greatly increased risk of acute pancreatitis and retinal thrombosis . They are synthesized in the endothelium of intestines→ Lymphatics→ Blood. Chylomicrons has the highest level of TGA (70-80% TGA) • **Very Low Density Lipoproteins (VLDL): A) Source :** Liver. **B) Major lipid :** Carry **endogenous triglycerides & to a lesser degree cholesterol**. **C) Apoprotein :** The Apo **B-100**, C-I, C-II, C-III, & E. B100 is synthesized in the liver **D) Atherogenicity: (+)** • **Intermediate Density Lipoprotein (IDL): A)Source :** VLDL remnant. **B) Major lipid :** Carry **Cholesterol esters & triglycerides. C) Apoprotein :** Apo B-100, CIII & E. **D) Atherogenicity : (+)** • **Low Density Lipoproteins (LDL): A) Source:** VLDL, IDL . **B) Major lipid :** Carry **cholesterol esters. C) Apoprotein :** with Apo B-100 & C-III. **D) Atherogenicity : (+++) .** LDL has the highest Cholesterol (50% cholesterol ester) • **High Density Lipoproteins (HDL): A) Source:** Gut , Liver **B) Major lipid :** Carry **Cholesterol esters. C) Apoprotein :** with Apo. A-I, A-II, C-I, C-II,C-III, D, E. **D) Atherogenicity : (-)**	You must know what each one does: • Apolipoprotein A (I,II,IV) :ease the transport of free fatty acid to inside tissue. • Apolipoprotein B (48,100) • Apolipoprotein C (I,II,III) → inhibit E • Apolipoprotein E : Mediates remnant uptake. • Apolipoprotein D • Apo(A) ***ligand is a substance that fits into a receptor** ligand present on the particles → binds to receptors → activation

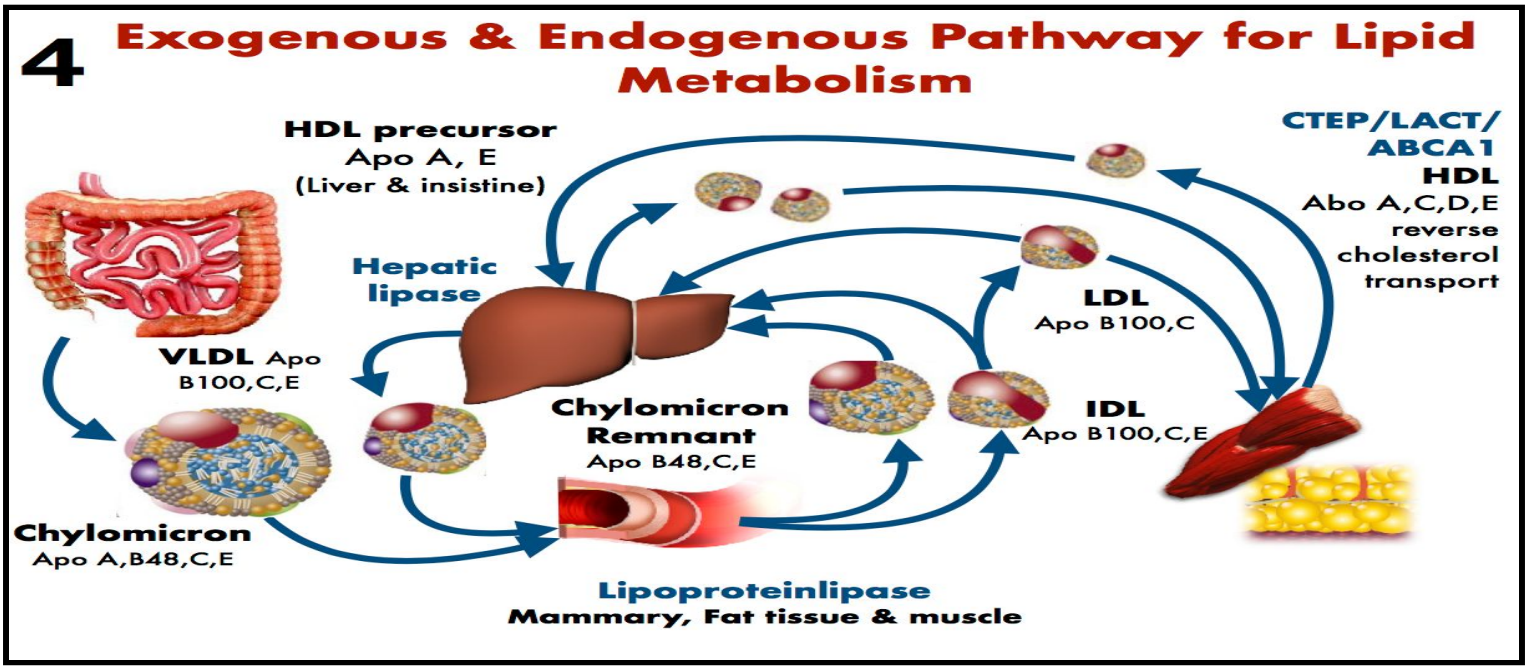
Anti-atherogenic . 1.lowest TAGs 2.Cholesterol is higher than TAG 3. high in Protein + Phospholipids. **It has No ApoB**.	

Physiology:









1- Chylomicrons are made in the intestine and then it goes via the lymphatic system to the blood. it circulates in the blood to go to the peripheral tissues and with the help of its ligand apo C-||, it well activate lipoproteinlipase and then it will give some triglycerides to the peripheral tissues and the result is chylomicron remnant.
2- this chylomicron remnant will go to the liver and with the help of hepatic lipase new particle will be formed which is VLDL (Endogenous lipid)
3- VLDL will go to the blood and with the help of ApoE it will activate lipoproteinlipase and then it will give some triglyceride to the peripheral tissues to be used in different things such as free fatty acids formation , steroids formation .
4- Then we will have IDL which has ApoB100 , Apo E and it either goes to the liver or to the peripheral tissues.
5- HDL has a very important function, It transport cholesterol from the peripheral tissues to the liver .HDL has a bit a complicated pathway in the bloodstream & has 3 types of particles some as precursors..& other mature

*insulin is a cofactor for lipoproteinlipase

Dyslipidemia

elevation of either triglycerides or cholesterol or combination

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dyslipidemia doesn't necessarily mean high serum lipids, it may be referred to low lipid levels e.g. fish eye syndrome ,IN Fish eye syndrome we have low HDL It happens secondary to Deficiency of LCAT (important enzyme to help HDL to attach the cholesterol, it is an autosomal recessive disease and it comes with vit E deficiency and neurological deficits .

- They are disorders of lipoprotein metabolism that result in the following abnormalities:

- They are categorized as follows:

Monogenic	*Secondary Dyslipidemia*	*Idiopathic*
Conditions to to a **single** gene defect. such as: - Familial Hypercholesterolemia - Familial Defective Apolipoprotein B - Familial Hypertriglyceridemia	Related to **specific diseases, conditions or exposures.** such as: - Obesity - Renal disorder : Nephrotic Syndrome , uremia - Endocrine disorder : hypothyroidism , T2DM , cushing syndrome - Drugs eg.glucocorticoids , estrogen , thiazide diuretics , B-blockers , Alcohol, isotretinoin - Pregnancy	Related to **polygenic** defects

Risk factors :

Diet	• Saturated fatty acids and cholesterol cause elevation in LDL and total cholesterol. • High-calorie diets do not increase LDL or cholesterol levels (are “neutral”) but do increase triglyceride (TG) levels. • Alcohol increases TG levels and HDL levels but does not affect total cholesterol levels.
Age	Cholesterol levels increase with age until approximately age 65. The increase is greatest during early adulthood—about 2 mg/dL per year.
Inactive lifestyle	abdominal obesity
Gender	Men generally have higher cholesterol levels than do women; when women reach menopause, cholesterol levels then equalize and may even be higher in women than in men.
Medication	• **Thiazides**—increase LDL, total cholesterol, TG (VLDL) levels • **β -blockers (propranolol)**—increase TGs (VLDL) and lower HDL levels • **Estrogens**—TG levels may further increase in patients with hypertriglyceridemia. • **Corticosteroids and HIV protease inhibitors** can elevate serum lipids
Genetic mutations	predispose to the most severe hyperlipidemias
Family history	—

Fredrickson's Classification:

Class	Name	Lipoprotein Elevated	Serum lipid pattern	Treatment
Type I	Exogenous hyperlipidemia	Chylomicrons	Elevated triglycerides	Diet
Type IIa	Familial hypercholesterolemia	LDL ممكن يجي سؤال	Elevated cholesterol	Statin Niacin Cholestyramine
Type IIb	Combined hyperlipoproteinemia	LDL + VLDL	Elevated triglycerides and cholesterol	Statin Niacin Gemfibrozil
Type III high levels of cholesterol and triglycerides "combined"	Familial dysbetalipoproteinemia	IDL	Elevated triglycerides and cholesterol	Gemfibrozil Niacin
Type IV	Endogenous hyperlipidemia	VLDL	Elevated triglycerides	Statin Niacin Gemfibrozil
Type V	Familial hypertriglyceridemia	VLDL + Chylomicrons	Elevated triglycerides and cholesterol	Gemfibrozil Niacin

Genetic Causes of Dyslipidemia:

Disease	Lipid Profile	Prevalence	Etiology
Primary Hypercholesterolemia			
Familial Hypercholesterolemia	↑ ↑ LDL	1:500 (+/-)	↓ LDL Receptor
Familial Defective ApoB100	↑ ↑ LDL	1:100	↓ ApoB100 binding to LDLR
Hypercholesterolemia	↑ Cholesterol	Common	Unknown
Primary Hypertriglyceridemia			
Familial Hypertriglyceridemia	↑ TG ↑ VLDL ↓ HDL المشكلة هنا تكون في lipoproteinlipase او الاشياء المتعلقة فيه	Common	↓ VLDL Breakdown ↑ VLDL Synthesis
Mixed Hyperlipidemia			

Familial Combined Hyperlipidemia	↑ TG ↑ LDL ↓ HDL	1:100	Unknown, Dominant inheritance
Disorders of HDL Metabolism			
Polygenic HDL	↓ HDL	Common	Obesity, DM, High carb diet
Familial Hypoalphalipoproteinemia	↓ HDL	1:100	Unknown, Dominant inheritance

Familial Hypercholesterolemia (FH):

FH is a genetically modulated clinical syndrome

Characterized by:

1. Elevated Low-Density Lipoprotein Cholesterol (LDL-C) level from birth
2. Xanthomata in untreated adults and patients with homozygous FH
3. Early onset Coronary Heart Disease (CHD)

Caused By:

- Mutation in LDL Receptor gene
- Mutation in the genes that code for Proportion Converts Subtilisin Kevin 9 (PCSK9)
- Mutation in Apolipoprotein B

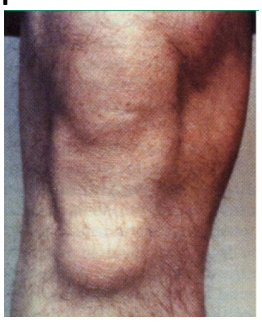
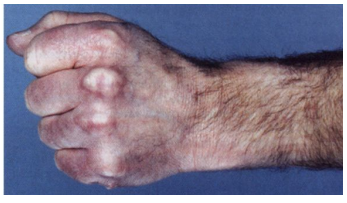
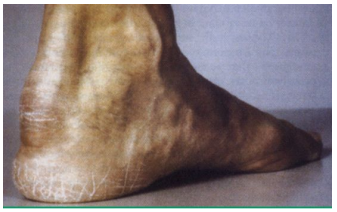
Diagnosis:

A Diagnosis of FH should be suspected in **children** with any of the following:

1. **Elevated Plasma LDL-C :** The level of LDL-C that warrants further evaluation depends upon whether additional family members have hypercholesterolemia and/or early cardiovascular disease (CVD):

Patients with a **Negative or Unknown family Hx**	An LDL-C level **of ≥190 mg/dL (4.9 mmol/L)** suggests FH
Patients with a **Positive family history** of hypercholesterolemia and/or early (CVD)	An LDL-C level **of ≥160 mg/dL (4.1 mmol/L)** is suggestive of FH.

2. Family member with known FH or *elevated* cholesterol ([TC] > 240 mg/dL [6.2 mmol/L] in either parent.
3. Tendon Xanthomas in the child or family member(s).
4. Premature CHD in the child or family member(s).
5. Sudden premature cardiac death in a family member.

Subperiosteal Xanthomata	**Tendon Xanthomata**	**Achilles Tendon Xanthoma** (hard,yellowish masses found on tendon)
		

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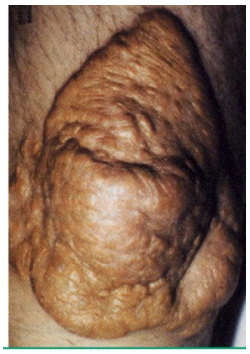
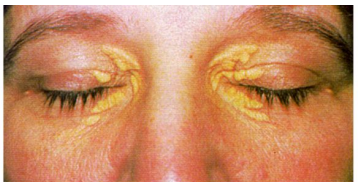
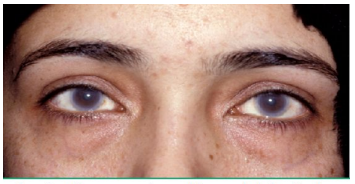
Planar Xanthoma	Xanthelasma (Yellow plaques on eyelids)	Corneal Arcus
 rarely seen		

Familial Hypertriglyceridemia:

The serum TG concentration can be stratified in terms of population percentiles and/or coronary risk:

Definition		Prevalence
<150 mg/dL (1.7 mmol/L)	Normal	33%
150 to 199 mg/dL (1.7 to 2.2 mmol/L)	Borderline High	18%
200 to 499 mg/dL (2.3 to 5.6 mmol/L)	High	1.7%
≥500 mg/dL (≥5.6 mmol/L)	Very High	0.4%

Causes:

Acquired Disorders	Hereditary Disorders
- Obesity - DM - Nephrotic Syndrome - Hypothyroidism - Pregnancy - Estrogen Replacement Administered Orally - Tamoxifen - Beta Blockers - Immunosuppressive medication such as glucocorticoids & cyclosporine. - Retinoids	- Chylomicrons - Familial hypertriglyceridemia - Familial Combined hyperlipidemia - Familial Dysbetalipoproteinemia

Clinical Manifestations:

- In patients with **acquired disorders** such as diabetes or obesity, clinical manifestations are usually due to an underlying disorder rather than the lipid abnormality.

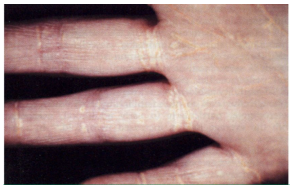
-In patients with **hereditary disorders,** skin lesions such as xanthomas & xanthelasmas may be present.
- In patients with **very high TG levels** (above 1000 mg/dL [11 mmol/L], pancreatitis may develop. Don't forget this value .

Triglycerides and CVD Risk:

- Studies generally show a positive relationship between hypertriglyceridemia & atherosclerotic burden.
- Abnormalities that predispose to atherosclerosis or are associated with increased CVD risk. **These include:**
    - Low levels of high density lipoprotein cholesterol (HDL)
    - Small, Dense low density lipoprotein particles (LDL)
    - Atherogenic triglyceride- rich lipoprotein remnants (VLDL)
    - Insulin resistance
    - Increase in coagulability & viscosity, triglyceride-meditated hyperviscosity may contribute to endothelial dysfunction, tissue ischemia and the chylomicronemia syndrome.

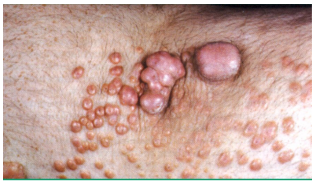
Familial Dysbetalipoproteinemia/ High TG (Hypertriglyceridemia)

Palmer Xanthomata



Striate xanthomata of the palmar creases in a patient with Type III hyperlipoproteinemia

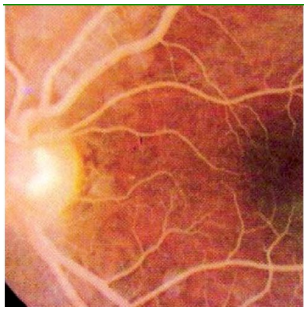
Tuberoeruptive xanthomata



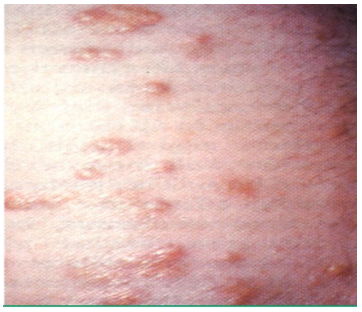
On the elbow and extensor surface of the arm in a patient with Type III hyperlipoproteinemia

Familial Hypertriglyceridemia

Lipemia Retinalis



Eruptive xanthomata



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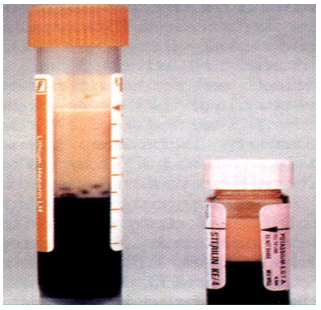
Milk Plasma



ATP III Classification of LDL, Total and HDL Cholesterol (mg/dL):

Why should the patient fast before doing a lipid blood test ?

Because we want to measure the endogenous VLDL production not chylomicrons.

LDL Cholesterol	
<100	Optimal
100-129	Near Optimal/ Above Optimal
130-159	Borderline high
160-189	High
≥190	Very High
Total Cholesterol	
<200	Desirable
200-239	Borderline High
≥240	High
HDL Cholesterol	
<40	Low
≥60	High

Management of Hyperlipidemia

(LDL-C) Lowering:

Rationale for LDL-C Lowering:

- LDL-C plays a **key role** in the pathogenesis & perpetuation of atherosclerotic CVD.
- Elevated level of LDL-C is associated with an increased risk of CVD events. lowering of LDL-C is associated with a reduction in the events.
- Mendelian randomization analysis has shown that a lifelong very low LDL-C is associated with a much lower risk of CVD.
- Randomized trials of many classes of LDL-C lowering drugs, include **Statins**, **Ezetimibe**, **PCSK9 inhibitors** & **Cholesteryl ester transfer protein CETP inhibitors**, have shown a reduction in CVD events.

Risk Groups:

- Patients with established CVD include those with stable or unstable coronary artery disease, ischemic stroke, transient ischemic attack, or peripheral arterial disease.
- Prevention of CVD events in these ***high risk group*** is referred to as **Secondary prevention.**
- We consider CVD patients with the following characteristics to be at ***very high risk:***
  1. Acute coronary syndrome within the past year.
  2. Familial hypercholesterolemia.
  3. DM, HTN, Smoking.
  4. CKD stage 3,4,5.
  5. Recurrent atherosclerotic CVD event, need for revascularization while on statins.

Management of (CVD) or High risk patients:

- Across a broad range of baseline CVD risk and LDL-C baseline levels, most therapies that lower LDL-C lead to a clinically important reduction in the risk of MI and ischemic stroke.
- Among these therapies are:
  - **Statins**
  - **Ezetimibe**
  - **PCSK9 inhibitors.**
- **Goal of therapy FH/ High risk group with CVD:**
  Targeted values of LDL-C depend on the presence of associated risk factors & are generally as follows:
  - Minimal Value, LDL-C <130 mg/dL (3.35 mmol/L)
  - Optimal Value, LDL-C <110 mg/dL (2.85 mmol/L)
  - Some providers target values of LDL-C <100 mg/dL (2.59 mmol/L) for high risk patients, such as those with DM or chronic renal insufficiency.

The use of three classes of LDL-C lowering drugs in **statin-treated patients** has been shown to lead an **additional reduction** in CVD events in study populations that achieved a mean LDL-C < 55 mg/dL:

- In the FOURIER trial → PCSKi9
- In the IMPROVE IT trial→ Ezetimibe
- In the REVEAL trial → Anacetrapib

<table>
<tr><th colspan="2">Hyperlipidemia Management</th></tr>
<tr><th colspan="2">First Line Therapy</th></tr>
<tr><td colspan="2">Lifestyle Modification:
- Weight loss in obese patients.
- Aerobic exercise.
- Avoidance of medications that raise serum triglyceride levels & alcohol overuse.
- Strict glycemic control in diabetics.
- Other risk factors for CVD, such as hypertension & smoking, should also be addressed.
- Dietary modification:
- Low saturated fat diet: limits total fat intake to 30% of total calories, saturated fat to 7-10%, T.C to 300 mg/day.
- Fiber, avoidance of concentrated sugars.</td></tr>
<tr><th colspan="2">Second Line Therapy</th></tr>
<tr><th>Hypercholesterolemia (LDL-C)</th><th>Hypertriglyceridemia Management</th></tr>
<tr><td>Pharmacotherapy:
- Statins
- Ezetimibe
- PCSK9 inhibitors</td><td>Indications for drug therapy:
- The two potential indications for pharmacologic therapy to lower triglyceride levels are prevention of episodes of pancreatitis & lowering of cardiovascular risk.
- If triglyceride levels persistently > 886 mg/dL (10 mmol/L) we start drug therapy to lower the risk for pancreatitis.
Pharmacotherapy:
- Fenofibrate
- - Statins should be considered even in patients with high triglyceride levels because of their cardioprotective effects.
- Niacin
- Fish oil</td></tr>
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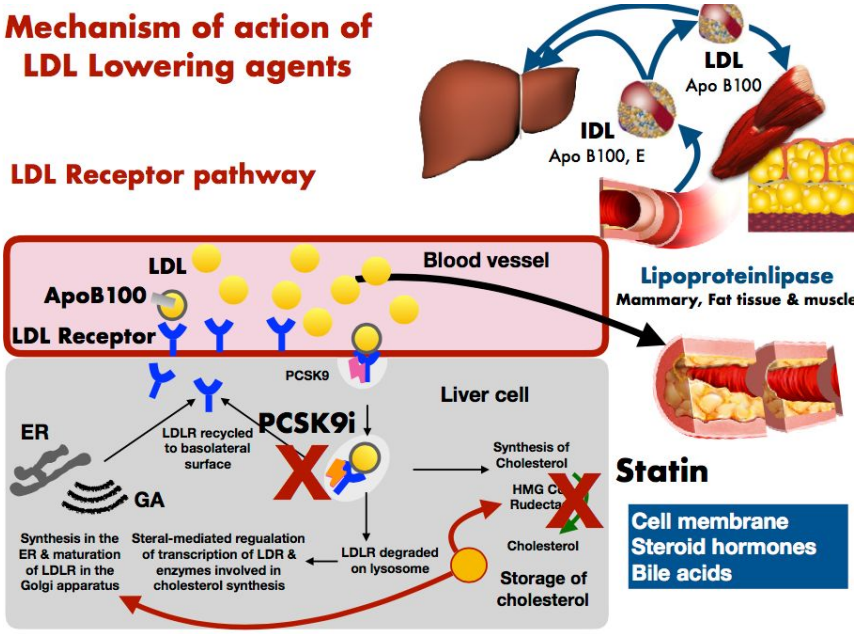

- LDL فيها lipoprotein Apo B100 وشغلته تسهيل و تدخل endocytosis الارتباط بالريسبتور بعدين بيصير بعدين بيصير تحلل لكل lysosome جوا الخليه تحديدا المكونات و بيطلع لنا كوليسترول بمساعدة HMG-COA

- PCSK9 هو فاكتر مرتبط بالرسبتر اهميته ان يسوي degradation for the combination of receptor and the particle so we can take the particle to use it for cholesterol formation.

when we have enough cholesterol, PCSK9i well be suppressed so there will be no formation of new receptors.

Average effects of different classes of lipid lowering drugs on serum lipids

Drug class	Serum LDL cholesterol (% change)	Serum HDL cholesterol (% change)	Serum triglycerides (% change)
Bile acid sequestrants	↓ 15 to 30	0 to slight increase	No change or increase
Cholesterol absorption inhibitors	↓ 17	↑ 1	↓ 7 to 8
Fenofibrate (micronized form)	↓ 6 to 20	↑ 5 to 20¶	↓ 41 to 53
Gemfibrozil◇	↓ 10 to 15	↑ 5 to 20¶	↓ 35 to 50
Neomycin	↓ 20 to 25	No change	No change
Nicotinic acid (niacin)	↓ 10 to 25	↑ 15 to 35	↓ 25 to 30
Omega 3 fatty acids△	↑ 4 to 49	↑ 5 to 9	↓ 23 to 45
PCSK9 inhibitors	↓ 38 to 72	↑ 4 to 9	↓ 2 to 23
Statins	↓ 20 to 60	↑ 5 to 10	↓ 10 to 33

important to know which drugs affect which type of lipids.

-just know the side effects of the drugs

Drug class	Dose	Dosing	Major side effects and drug interactions
Statins			
Atorvastatin	10 to 80 mg/day		Headache; nausea; sleep disturbance; elevations in hepatocellular enzymes and alkaline phosphatase. Myositis and rhabdomyolysis, primarily when given with gemfibrozil or cyclosporine; myositis is also seen with severe renal insufficiency (CrCl <30 mL/min). Lovastatin, atorvastatin, rosuvastatin, and simvastatin potentiate effect of warfarin; this interaction is not seen with pravastatin, fluvastatin, or pitavastatin. Most statins can also affect digoxin metabolism and levels.
Fluvastatin	IR: 20 to 80 mg/day	IR take in the evening. Divide dose twice per day (morning and evening) if dose >40 mg/day.	
	XR: 80 mg/day	XR take any time	
Lovastatin	IR: 20 to 80 mg/day	IR take with evening mea Divide dose twice per day with meals if dose >20 mg/day.	
	XR: 20 to 60 mg/day	XR take any time	
Pitavastatin	1 to 4 mg/day		
Pravastatin	10 to 80 mg/day		
Rosuvastatin	5 to 40 mg/day		
Simvastatin	5 to 40 mg/day	Take in the evening	
PCSK9 inhibitors			
Alirocumab	75 to 150 mg every two weeks	Subcutaneous injections	Injection site reactions
Evolocumab	140 mg every two weeks or 420 mg every month Homozygous familial hypercholesterolemia: 420 mg every month to 420 mg every two weeks		

Fibric acid derivatives			
Fenofibrate	Nanocrystal 145 mg/day Micronized 160 to 200 mg/day	Micronized taken with meals. Use lower doses with renal insufficiency.	Skin rash, gastrointestinal (nausea, bloating, cramping) myalgia; lowers blood cyclosporine levels; potentially nephrotoxic in cyclosporine treated patients. Avoid in patients with CrCl <30 mL/min.
Gemfibrozil	600 mg twice per day	30 to 60 minutes before meals	Potentiates warfarin action. Absorption of gemfibrozil diminished by bile acid sequestrants.
Nicotinic acid (niacin)	IR: 1 to 6 g/day	IR: Taken with meals. Start with 100 mg twice per day and titrate to 500 mg three times per day. After six weeks, check lipids, glucose, liver function, and uric acid. Increase dose as needed.	Prostaglandin-mediated cutaneous flushing, headache, warm sensation, and pruritus; hyperpigmentation (particularly in intertriginous regions); acanthosis nigricans; dry skin; nausea; vomiting; diarrhea; and myositis
	XR (Niaspan): 0.5 to 2 g/day	XR: Taken at bedtime; adjust dose every four weeks as needed.	
Bile acid sequestrants			
Cholestyramine	4 to 24 g/day	Take within 30 minutes of a meal. A double dose with dinner produces same lipid-lowering effect as twice per day dosing.	Nausea, bloating, cramping, and constipation; elevations in hepatic transaminases and alkaline phosphatase. Impaired absorption of fat soluble vitamins and coadministered medications including: Amiodarone, digoxin, warfarin, thiazides, beta blockers, levothyroxine, others; interaction can be minimized by taking other medications at least one hour before or four hours after bile acid sequestrant.
Colestipol	5 to 30 g/day		
Colesevelam	3.75 g/day	Take with meals once daily or in two divided doses.	Similar
Cholesterol absorption inhibitors			
Ezetimibe	10 mg/day		Increased transaminases in combination with statins
Neomycin	1 g twice per day		Ototoxicity; nephrotoxicity
Probucol (not available in United States)	500 mg twice per day		Loose stools; eosinophilia; QT prolongation; angioneurotic edema

Drug Therapy for Hyperlipidemia :

Drug	Effects	Comments	Side effects
HMG CoA reductase inhibitor (statins)	--Lower LDL levels (most potent for lowering LDL) -- Minimal effect on HDL and TG levels	Have been shown to reduce mortality from cardiovascular events and significantly reduce total mortality Drugs of choice for lowering LD	--Monitor LFTs (monthly for first 3 months, then every 3–6 months). -- Harmless elevation in muscle enzymes (CPK) may occur.
Niacin	--Lowers TG levels -- Lowers LDL levels --Increases HDL leve	--Do not use in diabetic patients (may worsen glycemic control) -- Most potent agent for increasing HDL levels and lowering TG levels	--Flushing effect (cutaneous flushing of face/arms; pruritus may be present) . --Check LFTs and CPK levels as with statin drugs.
Bile acid–binding resins (cholestyramine, colestipol)	--Lowers LDL -- Increases TG levels	Effective when used in combination with statins or niacin to treat severe disease in high-risk patients	Adverse GI side effects, poorly tolerated
Fibrates(gemfibrozi)	--Lower VLDL and TG -- Increase HDL	Primarily for lowering TG levels	GI side effects (mild) Mild abnormalities in LFTs Gynecomastia, gallstones, weight gain, and myopathies are other side effects.

MCQs

1) 25 years old male, not hypertensive and does not have DM and not a smoker. his brother dies from a cardiovascular disease at age 32.His lipid profile is: LDL: 6.1 mmol/L (=~196), HDL 0.1 and a 10 y risk of AVCAD .0,1%. What is the best management for him?

a. high sustained statin therapy.
b. low sustained statin therapy.
c. moderate sustained statin therapy.
d. dot give him anything.

2) Which one of the following is associated with cardiac protection?

a. Low TG.
b. High HDL.
c. High LDI.
d. Low HDL.

3) A 23-year old presented with chest pain. He has a brother who died from MI in his 30s. Which of the following is the most likely mutated receptor?

a. LDH receptor.
b. VLDL receptor.
c. Lipoprotein lipase.
d. LDL receptor.

4) 39 year-old man with prior history of myocardial infarction complains of yellow bumps on his elbows and buttocks. yellow-colored cutaneous plaques are noted in those areas. the lesions occur in crops and have a surrounding reddish halo. which of the following is the best next step in evaluation of this patient?

a. Biopsy of skin lesion.
b. Lipid profile.
c. Uric acid level.
d. Chest x-ray.

Answer key:

1 (a) | 2 (b) | 3 (d) | 4 (b)