Cholesterol

Cholesterol is needed for cells to exist. The problem is that too much cholesterol leads to the development of plaques, the things that cause atherosclerosis. There is **bad cholesterol** (**LDL**) that is responsible for bringing the cholesterol to the periphery. Too much of it makes plaques form. The **good cholesterol** (**HDL**) clears those plaques and brings the cholesterol back to the liver for processing. It makes sense that we'd want to decrease the LDL and increase the HDL. Turns out that's not true. The goal is to get people on a statin.

Why Statins

Although controversial, the most recent lipid guidelines have said that targeting a specific number did not improve outcomes. What improved outcomes (stroke, heart attack, and death) was to be on a high-intensity statin. It's empiric.

Who Gets a Statin

There are four groups of people who should be on a **high-intensity statin** (atorvastatin or rosuvastatin). [1] Anyone with any vascular disease (**stroke**, **coronary artery disease**, **peripheral vascular disease**, or **carotid stenosis**) or [2] **LDL ≥ 190**. If a patient has vascular disease or an LDL ≥ 190, regardless of anything else, they're on a statin. It gets a bit trickier when one of these two criteria isn't present.

If the patient has an **LDL < 70** they do **NOT** need a statin. Vascular disease and LDL ≥ 190 trump this statement, though the intensity will likely be decreased.

So groups [3] Diabetes and [4]Calculated risk go something like this. If the patient has an **LDL 70-189** AND are **Age 40-75** AND are either a diabetic or have a 10-year calculated risk, they get a statin. That 10-year calculated risk thing just means "do you have 2 or more vascular risk factors" but in a more convoluted way you shouldn't memorize.

What Statin Should You Give?

The goal is to be on **high-intensity statin**. Start a moderate-intensity and increase the dose to high-intensity.

If there's **liver disease** or **renal disease**, start at and stay on a moderate-intensity statin.

If there are signs of **statin-toxicity** during treatment, **stop the statin** until the signs go away. Then restart the statin at a lower dose. Anaphylaxis would be an exception, though the incidence is so low it essentially doesn't count.

Evaluation of Statins – What and When

Baseline values of Lipids, A1c, CK, and LFTs are required before starting a statin. You want to know what their baselines are to allow comparison if something happens.

Lipids are assessed **annually** (NOT q3months).

CK and **LFTs** are **NOT** checked routinely. Assess them only when there's evidence of disease. **Myositis** presents with soreness, weakness, or muscle pain. Hepatitis presents with right upper quadrant abdominal pain or jaundice.

When it is an option, the right answer is always:

1. Lifestyle = Diet / Exercise
2. Adherence = Medication and Lifestyle

Who Needs a Statin?

1. Vascular Disease = MI, CVA, PVD, CS
2. LDL ≥ 190
3. LDL 70-189
   + Age
   + Diabetes
4. LDL 70-189
   + Age
   + Calculated Risk = "Risk Factors"

Risk Factors for Coronary Artery Disease

1. Diabetes
2. Smoking
3. Hypertension
4. Dyslipidemia
5. Age > 55 for women, > 45 for men

High-Intensity	Moderate-Intensity	Low-Intensity
Atorva 40, 80	**Atorva 10, 20**	----------
Rosuva 20, 40	**Rosuva 5, 10**	----------
----------	Simvastatin 20,40	Simvastatin 5, 10
----------	Pravastatin 40, 80	Pravastatin 10,20
----------	Lovastatin 40	Lova 20

Monitoring Statin Therapy

Baseline	**Routine**	**Symptoms**
Lipids	q1y	----------
A1c	DM = q3mo	----------
CK	----------	Muscle Sxs
LFTs	----------	Hepatitis

Statin-Myositis	Stop Statin… Start a lower dose
Statin-Hepatitis	Stop Statin… Restart at a lower dose

So You Can't Use A Statin

The way the test will go after your knowledge of other lipid medications (which is still viable test fodder) is by giving a patient who, for whatever reason, CAN'T take a statin. Then the door opens to follow the previous lipid guidelines of reducing the LDL to < 100 with medications. Of course, lifestyle modifications and adherence become paramount, but the test is going to get you to answer questions about medications.

Some highlights:

Fibrates are the **second line** to statins. They have the same side effect profile but are also really good at getting the LDL down and the HDL up. They make sense.

Niacin is a board favorite because it causes flushing. While Niacin has not been shown to have mortality benefit, it makes for a great test question. Treat the flushing with **Aspirin** prophylaxis.

Bile Acid resins and **Ezetimibe** block absorption of fatty-related stuff. That leads to a fatty stool. Fatty stool causes osmotic diarrhea.

Drug	*Effect*	*Mechanism*	*Side Effect*
Statins	↓LDL ↓TG	HMG-CoA reductase	**Myositis** **LFT ↑**
Fibrates	↓TC ↑HDL	Lipoprotein Lipase	**Myositis** **LFT ↑**
Ezetimibe	↓LDL	Cholesterol Absorption	**Diarrhea**
Niacin	↑HDL ↓LDL	↓ Fatty Acid Release ↓ LDL Synthesis	Flushing (treat with **ASA**)
Bile Acid Resins	↓LDL	Bile Acid Reabsorption	**Diarrhea**