Hyperlipidemia

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Objectives

- Discuss the incidence of hyperlipidemia alone and the effects of diabetes, obesity and metabolic syndrome on lipid levels
- Explain the mechanisms as to why cholesterol levels increase and describe the characteristic atherogenic dyslipidemia found in diabetic patients
- Distinguish lifestyle interventions that improve cardiovascular health and address the effects of diabetes, obesity and metabolic syndrome
- Discuss and describe OTC, nutritional, herbal, ayurvedic, homeopathic and essential oil therapies for lipid control
Objectives

- Appraise treatment recommendations and evidence for screening, diagnosing and treating hyperlipidemia in both pediatric and adult patients (including women of childbearing potential and during pregnancy).
- Discuss mechanisms of action and the safety profile of lipid altering drugs including but not limited to statins, niacin, fibrates, bile acid sequestrants, and omega-3-acid ethyl esters to encourage the appropriate utilization of therapies for enhanced patient compliance.
- Discuss a patient treatment plan and then develop a systematic management plan to reduce risk factors in patients with pre-diabetes, abdominal obesity, dyslipidemia and other metabolic syndrome criteria.

Hyperlipidemia Facts

Three major classes of lipoproteins are found in the serum of a fasting individual:

- Low-density lipoproteins (LDL)
  - Make up 60-70% of total cholesterol and is the major atherogenic component.
  - Contains apolipoprotein-B (apo B)

- High-density lipoproteins (HDL)
  - Make up 20-30% of total cholesterol.
  - Contains apolipoprotein-A (apo A)

- Very low density lipoproteins (VLDL)
  - There are triglyceride rich and compose 10-15% of total cholesterol

Elevated total cholesterol, LDL cholesterol and reduced HDL cholesterol are associated with the development of coronary artery disease.

Diabetes effect on Lipids

- Up to 97% of patients with diabetes have dyslipidemia, which puts them at a extremely high risk for atherosclerosis.
- Diabetic patients have a characteristic pattern to their lipid panel:
  - Increased triglycerides
  - Decreased HDL levels
  - Abnormalities in the structure of the LDL cholesterol, they are usually small dense particles
  - Small dense LDLs are more atherogenic in nature than the larger LDL particles
  - Small dense LDLs easily penetrate the arterial wall, plus their adherence is much stronger.

References:
Diabetes and Hyperlipidemia

Cardiovascular disease is the main cause of morbidity and mortality for individuals with diabetes resulting from coexisting conditions such as dyslipidemia.

- Diabetes without CHD raises a persons risk to that of the risk level of a CHD risk equivalent.
- Diabetic patients have a >20% chance of developing coronary artery events within 10 years.
- Diabetes usually will lower HDL levels and increase both triglyceride and LDL levels (Diabetic Dyslipidemia).

Obesity and Hyperlipidemia

- More than one-third of the adults in the US are obese (35.7%)
- Studies show that being overweight, obese and significant weight gain are associated with an increase in cholesterol levels.
- The pattern of fat distribution appears to affect cholesterol, independently of the total weight.
- Total cholesterol are the highest in people with predominant abdominal obesity (waist-to-hip ratio).
  - Women ≥ 0.8
  - Men ≥ 1.0

Obesity cont..

- HDL levels are lower in both men and women with high BMIs.
- A BMI change of 1 unit is associated with a HDL of 1.1 mg/dl for young adult men and 0.65 mg/dl for young adult women.
- Triglycerides increase with BMI ≥ 30.
- LDL levels are higher by 10-20 mg/dl in relationship to a 10 unit difference in BMI, from levels of 20-30 kg/m².
- A 10 mg/dl rise in LDL-cholesterol corresponds to about a 10% increase in CHD risk over a span of 5 -10 years.
- However regardless of the high association between obesity and the risk for CHD, the ATP III guidelines do not have it as a risk factor that modifies LDL treatment goals.
Metabolic syndrome and Lipids

Metabolic syndrome risk factors are:

• Dyslipidemia
  - Triglycerides ≥ 150 mg/dl
  - Reduced HDL ≤ 40 in males, ≤ 50 in females
• Hypertension SBP ≥ 130 or DBP ≥ 85
• Hyperglycemia ≥ 100 mg/dl or on treatment for diabetes
• Abdominal obesity (>40" for males and >35" for females)

Atherogenic dyslipidemia consist of increased triglycerides and apolipoprotein B (apoB), increased small particle LDL and decreased HDLs.

The Role of Cholesterol

- Used in the formation of bile acids to aid in the breakdown of fats
- Cholesterol is the structural backbone of many hormones
- Cholesterol is essential for cell membrane function

Three Primary Sources of Cholesterol

- Liver by way of hepatic synthesis (Primary)
  - Accounts for slightly more than half of the cholesterol found in the body
- Intestinal absorption from one’s diet
- Extrahepatic synthesis (Minor)
Levels of Cholesterol

- HMG CoA Reductase is the rate limiting enzyme in the synthesis of cholesterol
- Three factors regulate HMG CoA Reductase and in turn cholesterol levels:
  - Rate of synthesis of HMG CoA Reductase
  - Rate of degradation of HMG CoA Reductase
  - Levels of plasma LDL and the number of LDL receptors
- This explains the importance of HMG CoA Reductase Inhibitors (Statins)

Three Major Classes of Lipoproteins

- Low Density Lipoproteins (LDL)
  - Also known as the “Bad Cholesterol”, when in excess can be deposited on the endothelium
- High Density Lipoproteins (HDL)
  - Also known as the “Good Cholesterol”, helps return free cholesterol back to the liver
- Very Low Density Lipoproteins (VLDL)
  - The major lipoprotein associated with triglycerides and lipoprotein remnants
  - Used as a marker to identify lipoprotein remnants which are also candidates for atherogenic deposition

What Factors Lead to Increased Cholesterol Levels

Genetic Factors
- Familial combined hyperlipidemia
  - Often resulting in elevated LDL and triglycerides which can result in premature atherosclerosis

Polygenic hypercholesterolemia
- Thought to be a combination of nutritional and genetic factors resulting in a loss of the LDL receptor
  - Often resulting in elevated LDL and consequential premature atherosclerosis

Familial hypercholesterolemia
- Defective gene for the LDL receptor
  - Often resulting in severe elevations of LDL, premature atherosclerosis, and tendon xanthomas

What Factors Lead to Increased Cholesterol Levels

Secondary hyperlipoproteinemia
- Hypercholesterolemia
  - Caused by but not limited to hypothyroidism, liver disease, nephrotic syndrome, and particular medications

Low HDL
- Caused by but not limited to malnutrition, obesity, and particular medications

Hypertriglyceridemia
- Caused by but not limited to obesity, diabetes mellitus, pregnancy, acute hepatitis, and particular medications

Diabetic Dyslipidemia
- Often is comprised of elevated triglycerides, minimally elevated LDL, and lower HDL levels

Typical characteristics of patients with diabetic dyslipidemia include:
- Obesity
- Abdominal obesity
- Insulin resistance
- Physical inactivity
- Raised blood pressure

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**Diabetic Dyslipidemia**

According to the ATP III guidelines, a diabetic patient’s LDL goal should be less than 100 mg/dl. LDL >130 mg/dl will require lifestyle changes, more intensified glycemic control, and drug therapy. Seeing that lowering LDL is the goal, statins tend to be the drug of choice. However, fibric acids lower VLDL and triglycerides while increasing HDL and improving glucose tolerance. During the Helsinki Heart Study, gemfibrozil was seen to be the most effective agent in the treatment of diabetic dyslipidemia.

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**Lifestyle Modifications for Patients with Type 2 Diabetes**

ATP III guidelines recommend therapeutic lifestyle changes (TLC). The first step of TLC includes maximum reduction of LDL cholesterol. Includes reducing intake of saturated fat and cholesterol. Adding LDL-lowering options include:

- Increasing soluble fiber
- Adding plant sterols/stanols

Plant sterols/stanols are found in small quantities of many fruits, vegetables, vegetable oils, nuts, seeds, cereals and legumes.

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Lifestyle Modifications for Patients with Type 2 Diabetes

Next step of TLC, after lowering LDL through dietary changes, is management of metabolic syndrome through increased physical activity and modifying associated lipid factors.

- Increasing HDL cholesterol
- Lowering triglycerides
- Increasing physical activity to 30 minutes per day on most, if not all, days of the week.

A study showed overweight and obese women with metabolic syndrome that added physical activity to a reduced calorie diet improved metabolic risk factors about 3.5 times more than diet alone.²

Over-The-Counter treatments

Niacin
50mg BID or 100mg QD
Take with meal
Lowers LDL, TG, and increases HDL
Significantly lowers Lipoprotein A levels
Immediate release niacin may cause itching, flushing, NA
May take ASA 30-60 minutes before dosing for flushing

Fish oil
4g/day in divided doses
Lowers TGs
Increases lipoprotein lipase activity
Avoid with fish allergies, ALT increase, may increase LDL, increase bleeding time.
If no response after 2 months in TGs, discontinue

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Herbal Remedies

Flaxseed and Flaxseed oil
In 2009 review found that cholesterol-lowering effects were more apparent in postmenopausal women and in people with high initial cholesterol concentrations.
Alpha-linolenic acid a substance in flaxseed may benefit people with heart disease, but more studies are needed to determine its effects for heart conditions.

Red Yeast Rice
There is evidence that red yeast rice may lower low-density lipoprotein in statin intolerant people.
In a study funded partly by NCCAM, it was concluded that red yeast rice significantly decreased LDL over a 12-week period without increasing incidence of myalgia.

Natural products: Work similar to HMG-CoA Reductase inhibitors

- Artichoke extract
  - Recommended dose: 1800mg/day into 2-3 divided doses
  - Flatulence
  - Avoid if allergic to aged wine

- Garlic
  - Inhibitor of CYP 3A4
  - Caution with warfarin, aspirin or other antiplatelet and anticoagulant agents
  - NCCAM funded a study and concluded there was no effect on LDL levels

- Policosanol
  - Use cautiously in patients who take aspirin or other antiplatelet/anticoagulant agents
  - Red yeast rice

- Policosanol
  - Use cautiously in patients who take aspirin or other antiplatelet/anticoagulant agents

- Nicotinamide
  - Using the long acting formulations to decrease the flushing

Nutritional choices that lower cholesterol

- Oatmeal, oat bran and high fiber foods (i.e. apples, prunes, kidney beans)
- Fish and omega-3 fatty acids (i.e. salmon, mackerel)
- Walnuts, almonds and other nuts
- Olive oil
- Foods with plant sterols
- Decrease saturated and trans-fat consumption
Ayurvedic therapies

The Aim of Ayurvedic medicine, which originated in India is to use products and techniques that cleanse the body to restore balance.

- **Alfalfa**
  - May lower cholesterol and triglycerides without altering HDL.
  - Saponins are found in alfalfa and it’s suggested that they decrease the intestinal absorption and increase fecal excretion of cholesterol.

- **Garlic**
  - Garlic’s lipid lowering ability may result from inhibition of HMG-CoA reductase.
  - NCCAM funded a study and found that three different preparations (fresh, dried powdered garlic) and found no effects on lowering cholesterol.

3. NCCAM Garlic Fact Sheet.

Risk Assessment for CHD

The detection and evaluation of cholesterol and lipoprotein should coincide with the patients overall risk assessment for CHD.

- **Measurement of LDL**
- **Identify co-existing risk determinants**
- **ATP III guidelines focus on primary prevention in people with multiple risk factors for CHD and to use more intensive LDL-lowering treatment**


Identification of persons with CHD and CHD risk equivalents

- **Coronary Heart Disease (10 year risk > 20%)**
  - History of MI
  - Myocardial ischemia
  - History of unstable or stable angina
  - Atherosclerotic diseases (CHD risk equivalent)
  - Peripheral arterial disease
  - Transient ischemic attack or stroke
  - Diabetes (CHD equivalent)
Risk Assessment in people without CHD or CHD risk equivalents

ATP III’s guidelines standard approach to evaluate the person’s risk for CHD is to count the number of major risk factors for CHD.

People that have a 2+ risk factor, the next step would be to evaluate the 10-year risk assessment for CHD using the Framingham scoring:

- > 20% (CHD risk equivalent)
- 10-20%
- < 10%

Electronic calculators to determine the 10-year risk are available at: www.nhlbi.nih.gov/guidelines/cholesterol.

Major Risk Factors (excluding LDL cholesterol) That modify LDL Goals

- Cigarette smoking
- Hypertension (BP ≥140/90) or on antihypertensive medication
- Low HDL < 40
- Family history of premature CHD
- CHD in male first degree relative < 55 yo
- CHD in female first degree relative <65 yo
- Age
  - Men ≥55 yo
  - Women ≥65 yo
- If the HDL ≥ 60 then one risk factor is subtracted from the total risk.

Who should be tested for cholesterol and lipoproteins?

Adults aged 20 years and older should obtain a fasting lipoprotein profile at least once every 5 years in low risk candidates.

1. NCEP, Circulation. 2. Defeat High Cholesterol, website (image)
General Approach to Treatment

Intensity of treatment is directly related to the patient’s risk for a CHD event. CHD or CHD equivalent have the lowest LDL goal and the most intensive treatment.

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>LDL Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD/CHD equiv.</td>
<td>&lt; 100mg/dl</td>
</tr>
<tr>
<td>Multiple (2+) risk factors</td>
<td>&lt; 130mg/dl</td>
</tr>
<tr>
<td>0-1 risk factor</td>
<td>&lt; 160mg/dl</td>
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</tbody>
</table>


Two step approach to cholesterol management

First Step:
- After goal for LDL-cholesterol is obtained:
  - The patient is started on dietary therapy and instructed on necessary lifestyle modifications.
  - If the patient has a CHD or CHD risk equivalent, LDL-lowering drug treatment and dietary plan can be initiated.

Second Step:
- After 3 months of dietary and lifestyle modifications have been tried with unsatisfactory results, other methods can be used.
  - If LDL goal was not met: initiate drug treatment.
  - If metabolic syndrome is present: enhanced lifestyle modifications must be added to reduce weight and increase physical activity.

Management of LDL-C for a person with CHD/CHD risk equivalent

Baseline LDL-C ≥ 130mg/dl
- Initiate LDL-lowering drug therapy to achieve LDL goal < 100mg/dl along with TLC.
  - If metabolic syndrome is present: increase weight loss and physical activity.
  - Elevated TGs or low HDL levels: consider nicotinic acid or fibrinic acid in combination with LDL-lowering drug.

Baseline LDL-C 100-129mg/dl
- TLC as initial therapy.
  - Increase dietary plant sterols and fiber.
  - If after 3 months LDL is not <100mg/dl, start LDL-lowering drug.
  - Elevated TGs or low HDL: consider nicotinic acid or fibrinic acid.

Screening pediatric population

The AACE recommends to screen children older than 2 years every 3 to 5 years:
- if they have CAD risk factor or a family history of premature CAD
- if they have dyslipidemia
- overweight, or obese
- Insulin resistance syndrome

Screen adolescents older than 16 years every 5 years or more often if the have CAD risk factors

1. AACE Lipids and Atherosclerosis Guidelines, Endocr Pract. 2012;18(suppl1)

Treatment for pediatric population

AACE recommends drug therapy for children older than 8 years old, who are not responding to lifestyle modification therapy

Must satisfy the following criteria for pharmacotherapy:
- LDL > 190mg/dl or
- LDL > 160mg/dl and having one or more of the following:
  - Presence of two or more cardiovascular risk factors after intense intervention
  - Family history of premature CAD (< 55yo)
  - Overweight, obese or insulin resistance syndrome

Drug options:
- Colesevelam: approved for patients > 8yo
- Statins (atorvastatin, lovastatin, pravastatin, simvastatin or rosvastatin): approved for patients >10 yo
- Cholestyramine: is also approved for treatment in children

AACE Lipid and Atherosclerosis Guidelines, Endocr Pract. 2012; 18(suppl 1)

Managing dyslipidemia for women of childbearing age

Fasting lipid panel should be obtained annually for women with diabetes and more often if needed to achieve goals

Lifestyle modifications have been shown to improve lipid profile of women with diabetes
- Reduction of saturated fat (<7% of energy), trans fat (as little as possible), and cholesterol intake (<200 mg/day)
- Increased physical activity
- Statin therapy is contraindicated during pregnancy and should be discontinued if pregnancy is anticipated
- For hyperlipidemia during pregnancy, bile acid sequestrants are not absorbed (pregnancy cat B)

1. AACE Lipids and Atherosclerosis Guidelines, Endocr Pract. 2012; 18(suppl 1)
Treatment Options

**HMG-CoA Reductase Inhibitors**

<table>
<thead>
<tr>
<th>Bile acid sequesterants</th>
<th>Nicotinic acid</th>
<th>Fibrinic acids</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simvastatin</td>
<td>Cholestyramine</td>
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<tr>
<td>Pravastatin</td>
<td>Colesevelam</td>
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<tr>
<td>Atorvastatin</td>
<td>Colestipol</td>
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<td>Fluvastatin</td>
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<td>Lovastatin</td>
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<td>Pitavastatin</td>
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<tr>
<td>Rosuvastatin</td>
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**HMG-CoA Reductase Inhibitors (Statins)**

- **Mechanism of Action:**
  
  Competitively inhibits HMG-CoA reductase, the enzyme responsible for the rate-limiting step of cholesterol biosynthesis.

- **Most effective drug class for lowering LDL cholesterol:**
  
  LDL = 18-55%  
  HDL = 5-15%  
  Triglycerides = 7-30%

**Contraindications**

- Absolute: acute or chronic liver disease
- Relative: Most statins are major substrates of CYP 3A4 (take into consideration when being used with CYP 3A4 inhibitors)

**Major side effects/adverse effects:**

- Myopathy/Rhabdomyolysis
- Increased liver transaminases

**Pregnancy category:** X

**Monitoring Parameters:**

- Baseline CPK (re-check if patient has symptoms of myopathy)
- Baseline Liver Function Test (discontinue if ALT/AST is >3 times normal limit)

**Administered in the evening**
HMG CoA Reductase Inhibitors

1) LDL Uptake
   Cholesterol
   Bile Acids
2) Synthesis
   HMG CoA
   Cholesterol
   Bile Acids

Characteristics of various Statins

Atorvastatin (Lipitor®)
Metabolized by CYP 3A4 but less than lovastatin and simvastatin
Decrease in LDL
10mg= 35-39%
20mg= 43%
40mg= 50%
80mg= 55-60%
No dose adjustment necessary for renal function
Avoid with Gemfibrozil

Fluvastatin (Lescol®)
Metabolized primarily by CYP 2C9, fewer interactions
Decrease in LDL
20mg = 22%
40mg=35%
80mg XL=35%
In severe renal impairment: caution in doses > 40mg

Lovastatin (Mevacor®)
Metabolized by CYP 3A4
Decrease in LDL
10mg=21%
20mg=24-27%
40mg=30%
80mg=40-42%
If CrCl < 30ml/min use doses > 20mg with caution
Avoid gemfibrozil
Don’t use with protease inhibitors

Pitavastatin (Livalo®)
Not significantly metabolized by P450
Decrease in LDL
1mg=32%
2mg=36-39%
4mg=41-45%
Characteristics of Statins

Pravastatin (Pravachol®)
- Not significantly metabolized by P450
- Decrease in LDL
  - 10mg=22%
  - 20mg=32%
  - 40mg=34%
  - 80mg=37%
- In significant renal impairment: start with 10mg
- Avoid gemfibrozil
- Reduce dose w/Niacin >1g/d

Rosuvastatin (Crestor®)
- Not significantly metabolized by P450
- Decrease in LDL
  - 5mg= 45%
  - 10mg= 46-52%
  - 20mg= 47-55%
  - 40mg=55-63%
- CrCl <30ml/min: max dose 10mg/day
- Avoid with gemfibrozil
- 40mg dose contraindicated with Niacin and fibrate

Simvastatin (Zocor®)
- Metabolized by CYP 3A4
- Decrease in LDL
  - 5mg = 26%
  - 10mg=30%
  - 20mg=38%
  - 40mg=29-41%
  - 80mg=36-47%
- In severe renal impairment: starting dose is 5mg with close monitoring
- Do not exceed 10mg/d with diltiazem or verapamil
- Do not exceed 40mg with Niaspan
- Contraindicated with gemfibrozil
- Do not use with protease inhibitors

Characteristics of Statins

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Bile Acid Sequestrants

Mechanism of Action:
- Forms a nonabsorbable complex with bile acids in the intestine, inhibiting enterohepatic reuptake of intestinal bile salts. Therefore increasing the fecal loss of bile salt-bound low density lipoprotein cholesterol Major use to lower LDL cholesterol
- LDL ê 15-30%
- HDL ê 3-5%
- Triglycerides: increase (with the exception of colesvelam)
Bile Acid Sequestrants

Contraindications: biliary obstruction, gastroparesis, dysphagia
- Absolute: dysbetalipoproteinemia TG > 400mg/dl
- Relative: TG > 200mg/dl

Side effects/Adverse effects: gastrointestinal symptoms (mainly constipation), chronic use may be associated with bleeding

Decrease absorption of other drugs so avoid taking simultaneously (1 hour before or 4 hours after)

Pregnancy category: No studies done, use with caution

Monitoring parameters: Triglyceride levels at baseline and periodically

Colestipol (Colestid®)

Formulations:
- Tablets
- Granules/packet

Colestipol (Colestid®) is indicated for the treatment of high cholesterol and triglycerides in adults. It is available without a prescription. Always consult with your healthcare provider before taking any new medication.
Cholestyramine

- Formulation: oral powder for suspension
- Start: 4gm QD or BID
- Maximum: 24gm/day & 6 doses/day
- Must mix in at least 3 oz of liquid, soup, cereal, pulpy fruits (pineapple, pears or peaches)
- Best time of administration is at meal time
- Advise patient not to sip or hold resin in mouth: can cause discoloration of teeth & erosion of enamel
- Have patient report signs/symptoms of bleeding

Colesevelam (Welchol®)

- Formulation: oral powder for suspension & Tablets
- Tablets: 3 tablets PO BID or 6 Tablets PO QD
- Powder: one single (1.875g packet BID or one single 3.375g packet QD)
- Must mix with 4-8 oz of water, fruit juice or diet soft drink.
- Administer with a meal
- Oral suspension is recommended in children 10-17 years
- Advise patient to report signs of pancreatitis, or severe abdominal pain
- FDA approved as an adjunct to treat Diabetes type 2

Nicotinic Acids

- Mechanism of Action:
  - Inhibits the synthesis of very low density lipoproteins (VLDL), and low density lipoprotein (LDL)
  - May also increase the rate of triglyceride removal
- Lipid/Lipoprotein effects:
  - LDL ê 5-25%
  - HDL ê 15-35%
  - Triglycerides ê 20-50%
**Nicotinic acids**

**Contraindications**
- Absolute: chronic liver disease, severe gout
- Relative: Hyperuricemia, high doses with Diabetes type 2 (>3g/day)

**Side effects/adverse effects**
- Flushing, hyperglycemia, hyperuricemia or gout, upper GI distress, hepatotoxicity (especially with sustained release formulation >2g/day)

**Monitoring parameters:**
- Blood glucose, CPK, serum K+ (if on concurrent HMG-CoA reductase inhibitor), LFTs, platelets (if on anticoagulants), Uric acid

**Fibrinic acids**

**Mechanism of Action:**
- Down regulates apoprotein C-III (an inhibitor of lipoprotein lipase) and up-regulates the synthesis of apoprotein A-I, fatty acid transport protein, lipoprotein lipase resulting in an increase in VLDL catabolism and triglycerides are reduced.

**Lipid/Lipoprotein effects:**
- LDL ≤ 5-20%
- HDL ≥ 10-35%
- TGs ≥ 20-50%
Fibrates

- Contraindications: severe hepatic or renal insufficiency
- Side effects/Adverse effects: dyspepsia, upper GI distress, gallstones, myopathy
- Monitoring parameters: Lipid panel (if no change in 6-8 weeks, discontinue), LFTs (discontinue if levels increase by >3 times upper normal limit), renal function, myopathy


Comparison of Trilix and Antara
- Trilix: can be taken without regard to food
- Antara: given with meals to increase bioavailability

Comparison of fenofibric Acid and Fenofibrate

- Fenofibric acids: Trilix, Antara
- Fenofibrate: given with meals to increase bioavailability

Triglide: can be taken without regard to food

Comparison of fenofibric Acid and Fenofibrate. 2009; 25(2):250-211
Ezetimibe (Zetia®)

Mechanism of Action:
- Inhibits absorption of cholesterol at the brush border of the small intestine, therefore there is a decrease of delivery to liver of cholesterol
- Lipid/lipoprotein effects: as add on therapy
  - Further LDL ≤ 25%
  - Further HDL ≥ 3%
  - Further TG ≤ 14%
- Monitoring parameters: LFTs, signs & symptoms of cholelithiasis

Contraindications:
- Concomitant use with an HMG-CoA reductase inhibitor in patients with active hepatic disease
- Renal and hepatic insufficiency use with caution
- Used with fibric acid derivatives – can increase risk of cholelithiasis
- Side Effects: Diarrhea, arthralgia, myalgia, increase in LFTs
What can Pharmacists do?

Even though evidence suggests aggressive lipid lowering therapy should be initiated in at risk patients with CHD, many patients are not being screened or started on appropriate therapy to obtain target levels.

A Pharmacist’s knowledge of lipid-lowering medications and optimal dosing, indications, and safety allow pharmacists a unique opportunity to enhance patient’s treatment success.

Through patient education and monitoring, pharmacists can ensure patient adherence.

Pharmacist led interventions have been shown to help patients reach treatment goals.

Summary

Cardiovascular disease is a major cause of mortality and morbidity for patients with diabetes.

Dyslipidemia increases risk of coronary artery disease and patients with diabetes are at an extremely high risk for atherosclerosis.

Diabetes predisposes patients with smaller and more dense LDL particles.

Pharmacists have the opportunity to counsel patients about hyperlipidemia and medication adherence for lipid lowering medication and therapeutic lifestyle changes.