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(REVIEW ARTICLE)



Overview of ideal antilipidemic drugs: Past, present and the future

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Abstract

Atherosclerosis, a disease which affects large and medium sized arteries, is now a leading cause of death in many developed countries. High serum LDL (low density lipoproteins) and VLDL (very low density lipoproteins) levels are considered as atherogenic, whereas high HDL (High Density Lipoproteins) over 60 mg/dl has a protective effect. Literature reviews were conducted by me from different articles on hypolipidemic drugs and the present review looking forward ideal hypolipidemic drugs with their target of action in the treatment of atherosclerosis. Statins are the first line antilipidemic drugs which possess dose dependent adverse effects like muscle pain, neuropathy, hepatic dysfunction and rhabdomyolysis. Reducing the statin dose, adding drug like proprotein convertase subtilisin/kexin type 9 inhibitors, supplements with coenzyme Q10 and L-carnitine are some of the approaches to reduce statin adverse effects. In future for those patients who cannot tolerate statin and for those who can't achieve LDL target, squalene synthase inhibitors are the best choice. We focused on current existing hypolipidemic drugs, their targets and mechanism of actions and also the new ideal antilipidemic drugs of future. Ideal antilipidemic drug is one which should produce target lipid level, cause fewer side effects and drug interactions.

Keywords: Statins; Atherosclerosis; LDL; Novel drugs to treat hyperlipidemia

1. Introduction

Dyslipidemic persons have high risk for atherosclerosis [1]. Atherosclerotic lesions form a localized plaque in intima and narrow the arterial lumen. High levels of triglycerides in plasma can cause pancreatitis. Accumulations of lipids in heart induce oxidative stress and inflammatory cardiac fibrosis leads to cardiac dysfunction [2].

Review starts with the different classes of lipoproteins with their role and normal and abnormal levels. Hyperlipidemia can be treated by life style changes, dietary modification, reducing the other risk factors of atherosclerosis and finally with effective and safe use of drug therapy.

Drug therapy start with statins monotherapy to reduce LDL-cholesterol. But in order to reach LDL and triglycerides and HDL target in high risk cardiovascular patients combinations of drugs like ezetimibe, fibrates with statins play a important role. In patients who are intolerant to statins and those have constant elevation of triglycerides non-statin therapy with drugs like CETP inhibitors (Cholesteryl Ester Transfer Protein inhibitors) and PCSK9 inhibitors (Proprotein Convertase subtilisin/kexin type 9 inhibitors) are useful to reduce cardiovascular risk [3]. Effective and safe use of drugs and lower the lipoproteins level to the target level should be the motive in drug prescribing and drug usage by the patients. Starting with established frontline older antilipidemic drugs and development of newer novel ideal drugs in present and the future focused with the help of reference articles in this review.

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2. Plasma lipoproteins

Water insoluble plasma lipids are transported in the following classes of lipoproteins. 1. Chylomicrons 2. Chylomicron remnants 3. Very low density lipoproteins (VLDL) 4. Intermediate density lipoproteins (IDL) 5. Low density lipoproteins (LDL) 6. High density lipoproteins (HDL) 7. Lipoproteins (a) [4]

Table 1 Serum lipid levels (mg/dl) and the risk of ischemic heart disease

Lipid	Desirable level (Low risk)	Abnormal level (High risk)
Total cholesterol	< 200	> 240
LDL cholesterol	< 130	> 160
HDL cholesterol	> 60	< 40
Triglycerides	< 200	> 400

3. Hyperlipidemia

An elevated level of lipids or lipoproteins in blood causes Hyperlipidemia. Hyperlipidemia is an important major risk factor for cardiovascular disease [5]. Reducing serum lipids can reverse this cardiac dysfunction.

4. Management of Hyperlipidemia

Stress reduction, dietary modification, reduce the risk factors of atherosclerosis like body weight, consumption of alcohol, smoking and treatment of diseases like hypothyroidism, diabetes mellitus, hypertension are also important while starting hypolipidemic drug therapy.

5. Drug therapy

5.1. HMG CoA reductase (HydroxyMethylGlutaryl Coenzyme A reductase) inhibitors

Statins by inhibiting HMG CoA reductase block HMG CoA to mevalonate conversion. This is the rate limiting step in cholesterol synthesis [6].

Reduced mortality rate and no tolerability issues are the main backbone reasons for statins's leading success rate over other non-statins in the treatment of atherosclerosis [7]. As per Scandinavian Simvastatin Survival Study statin reduced 35% LDL-cholesterol and 25% Triglycerides after 5.4 years follow-up. But muscle related side effects are common in clinical practice while on statin therapy [8], [11]

Simvastatin possess antibacterial activity against staphylococcus aureus which has resistance to methicillin. Simvastatin because of its antibiofilm activity can be combined with topical antimicrobials to treat above methicillin resistant staphylococcus infection [9], [10]. Simvastatin can be considered as novel antibacterial agent.

Table 2 Currently used statins and their daily dose [12]

Statins	Daily dose in mg
Lovastatin	20-40
Pravastatin	10-20
Simvastatin	5-20
Fluvastatin	20-60
Atorvastatin	10-40
Rosuvastatin	5-20
Pitavastatin	1-4

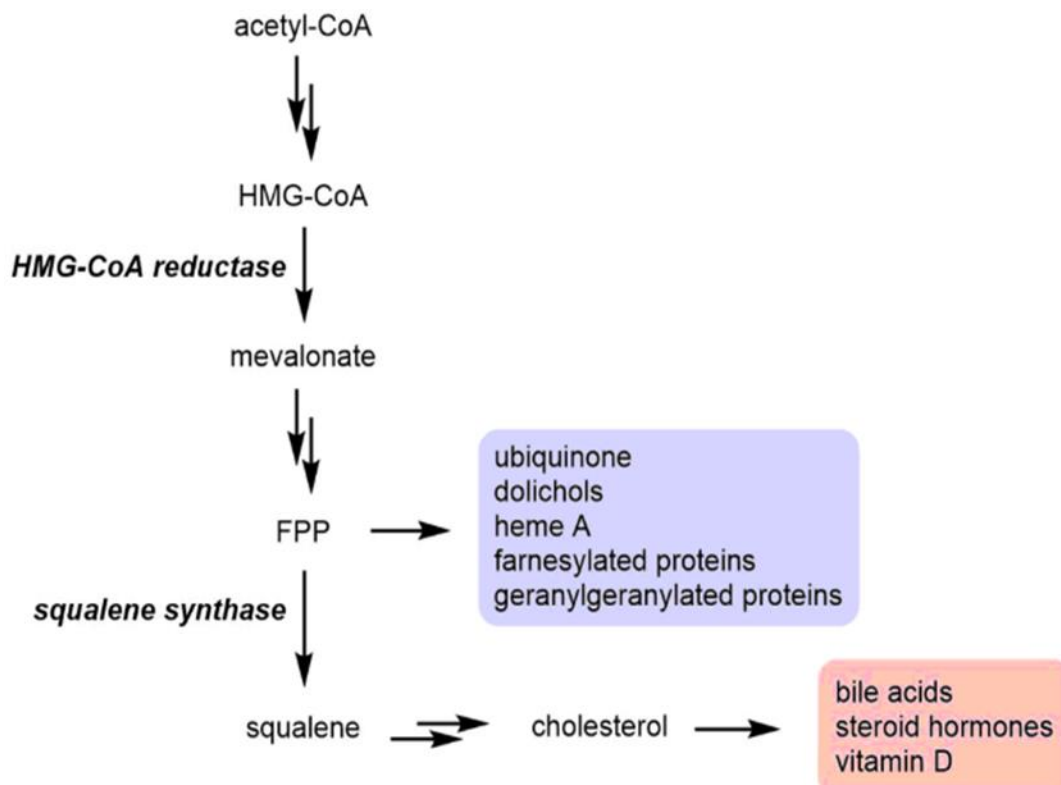


Figure 1 Cholesterol Biosynthesis

[HMG-CoA: 3-hydroxy-3-methylglutaryl-CoA, FPP: Farnesyl Pyrophosphate]

Adverse reactions of statins [12]

- Hepato toxicity-During statin therapy monitor transaminase level, if it reaches beyond more than three times of normal, discontinue statin therapy. Either shift to other drug or reduce the dose after transaminase level become normal.
- Muscle toxicity-Myopathy and muscular pains are caused by statins rarely. Elevated muscle enzymes level, rhabdomyolysis occur in few people. If patient get muscle pain it is better to check muscle enzyme levels.
- Diabetes- Before initiates statin therapy and during treatment with statin particularly with high dose checks blood sugar level.
- Cognitive impairment-Memory loss, mood changes and depression are some of the expected statin adverse reactions.

5.2. Cholesterol absorption inhibitors

Because of many patients have higher risk for coronary heart disease not reaching LDL goal after statin therapy alone, need a ideal second drug in addition with statins [13]. Also many patients meet intolerability issue when statins combined with fibrates, resins or niacin. Myopathy was severe when these drugs combined with statins. With combination of ezetimibe with statins no drug interactions are reported. Combination of ezetimibe and statin produces good cholesterol reduction than statins alone [16].

Ezetimibe decreases LDL cholesterol of plasma by 15-18%, as monotherapy. Ezetimibe exhibit good safety profile. In the intestine ezetimibe inhibit both biliary as well as dietary cholesterol absorption.. Ezetimibe used in the dose range of 5-10 mg/day. Avoid its use in pregnancy and children [14], [15].

5.3. PPAR α inhibitors

Fibrates stimulate peroxisome proliferator activated receptor-alpha receptor which controls genes expression that mediate metabolism of triglycerides. Fibrates increase lipoprotein lipase and hydrolysis of triglycerides. Plasma triglycerides reduced by 50%, cholesterol reduced by 15% and HDL increased by 20%. Commonly used fibrates are gemfibrosil, bezafibrate and fenofibrate [17]. The novel drugs which target both alpha and gamma PPAR receptors improve both glycaemic and lipid parameters Example: Tesaglitazar [18].

Table 3 LDL cholesterol lowering efficacy of drugs [19]

Drug	Percentage LDL cholesterol reducing efficacy
Statins	
Atorvastatin	39-60
Rosuvastatin	45-63
Bile acid sequestrants	15-30
Niacin	5-25
Fibrates	5-20
Ezetimibe	18

Table 4 Triglycerides lowering efficacy of drugs [19]

Drug	Percentage triglycerides lowering efficacy
Fibrates	30-50
Niacin immediate release	20-50
Statins	10-30
Ezetimibe	5-10
Omega-3 fatty acids	20-30

5.4. Niacin

Niacin in the dose of 2-8 g/day, decreases plasma triglycerides, LDL and raises HDL level. Niacin prevents lipolysis in adipose tissue. Slow down the fatty acids synthesis in liver. Side effect of niacin is flushing in therapeutic dose. But this can be minimized by taking niacin in smaller dose and increase gradually and administer with meals [20].

5.5. Omega-3 Fatty acids

Omega-3 fatty acids in fish oil decrease triglycerides and have no drug interaction with statins. Recommended dose is 2-4 gram/daily. Patients with very high triglycerides level above or equal to 500 mg/DL have more risk for acute pancreatitis [21].

5.6. ACAT inhibitor (Acyl- Coenzyme A: Cholesterol acyl Transferase inhibitor)

ACAT is the enzyme which synthesise cholesterol esters in tissues. ACAT inhibitor avasimibe reduce plasma cholesterol level and suppress atherosclerosis. Rall FJ et al enrolled twenty seven subjects in a double blind randomized 3-sequence cross over trial and administered atorvastatin 80 mg QD, avasimibe 750 mg QD and combined treatment of atorvastatin 80 mg QD and avasimibe 750 mg QD after washout period of four weeks. Six week treatment period allotted for each treatment [22]. After combination therapy Total cholesterol level was reduced by 22% instead of 18% by atorvastatin monotherapy.

5.7. Bile acid transport inhibitor

Metabolic effect of Elobixibat, a bile acid transporter inhibitor was evaluated by Rudling, M et al. [23]. After four week study on dyslipidemic patients drug effect on plasma lipids and bile acid synthesis was evaluated in samples. LDL cholesterol was reduced by 7.4% and bile acid synthesis was also induced.

Because elobixibat inhibit apical sodium dependent bile acid transporter, ileal bile acid absorption is reduced. Colon motility is increased due to more colon content of bileacid. Elobixibat reduces chronic constipation and the cardiovascular risk. Elobixibat should be taken before breakfast and tolerated upto 20 mg dose [24].

5.8. CETP inhibitor (Cholesteryl Ester Transfer Protein inhibitor)

CETP inhibitors reduce serum low-density lipoproteins and elevate high density lipoprotein cholesterol [25] and reduced the incidence of diabetes (12%). Cholesterol esters are transferred from HDL by CETP to LDL, forms lipoprotein apo-B which leads to more peripheral arterial deposition of cholesterol in walls. CETP inhibitors by increasing the HDL concentration reduced the incidence of diabetes. After anacetrapib treatment there is a modest cardiovascular benefit compared to the other two CETP inhibitors.

Table 5 CETP inhibitors and their properties [26]

Drug	Dose	LDL-C reduction	Increase in HDL
Torcetrapib	60 mg/day	25 %	72 %
Dalcetrapib	600 mg/day	No change	31 %
Anacetrapib	100 mg/day	40 %	138 %
Evacetrapib	130 mg/day	36 %	129 %

5.9. Lipoprotein(a) inhibitor

Elevation of lipoprotein(a), an atherogenic lipoprotein act as a genetic risk marker for atherosclerosis[27]. Manocha A and Srivastava LM explained benefit of lowering of lipoprotein (a) in treatment of atherosclerosis. Inhibition of Lipoprotein (a) by inhibiting lipoprotein (a) assembly is a better choice in therapy of future.

Niacin in high dose and PCSK9 inhibitors have the ability to lower lipoprotein (a) but not yet proved by trials. The ability of antisense oligonucleotides to lower lipoprotein (a) level near to normal level is a great scope in future to reduce cardiovascular risk in patients after the ongoing clinical trials [28].

5.10. Novel drugs with other mode of treatments to treat dyslipidemia

Other than the first line statins therapy the new treatments with novel drugs with other mode of actions are needed in order to improve efficiency to reach target LDL-cholesterol in high risk patients, or as alternative in statin intolerants or add on therapy with statins[29].

5.10.1. PCSK9 inhibitors [30]

PCSK9 (Proprotein Convertase subtilisin/kexin type 9) inhibitors

PCSK9 promote LDL receptor destruction on liver cells, prevent blood clearance of LDL.

Table 6 PCSK inhibitors and their brand names and dose [31]

Drug	Brand name	Dose
Alirocumab	Praluent (sanofil)	75 mg every two weeks, Subcutaneously
Evolocumab	Repatha (amgen)	140 mg every two weeks

Whenever we double the dose of statins to lower LDL, only 6 % additional lowering effect of LDL we are getting. But with high statin dose we get more increase in transaminase level and muscle toxicity as adverse effect. Achieving target LDL

level in cardiovascular risk patients have become increasingly complex. Addition of PSSK9 inhibitors with low dose statins avoiding the side effects and also reduces LDL effectively [32].

5.10.2. MTP (Microsomal Triglyceride transfer Protein) Inhibitor [33]

MTPI are able to reduce both triglycerides and LDL-cholesterol. Lomitapide from Aegerion, a MPTI able to reduce LDL-cholesterol. Lomitapide monotherapy at 5mg for four weeks reduced 19 % LDL and 30 % at 10mg. 60 mg is the maximum dose. Hepatic steatosis and increase in liver transaminase level are the serious side effects. Plasma concentration of statin and warfarin can be increased when they are used along with lomitapide.

Maximum Assembly of apolipoproteins, triglycerides and cholesterol in liver is the role of MTP [34].

5.10.3. AMPK/ATP-citrate lyase (AMP-activated protein kinase and ATP-citrate lyase)

ETC-1002 (8-hydroxy-2, 2, 14, 14-tetramethylpentadecanedioic acid), a novel investigational drug inhibit ATP citrate lyase. ATP citrate lyase is an enzyme coordinate in lipid synthesis. Combination of these two mechanisms regulates LDL [35].

ETC-1002 (bempedoic acid) and ezetimibe combination is a better option to reduce LDL in statin intolerant patients. Increased side effects with increase in statin dose, many patients not able to reach LDL target are the reasons for the clinical trials focusing ezetimibe and ETC-1002 combination or statin and ETC-1002 combination [36].

5.10.4. Apolipoprotein B synthesis inhibitor [37, 40]

Apolipoprotein B is important protein component of lipoproteins like VLDL and LDL. Mipomerson reduce atherogenic lipids and lipoproteins in patients with hypercholesterolemia by inhibit apolipoprotein B. Once in a week 200 mg/ml subcutaneously is the recommended dose. Elevation in plasma alanine aminotransferase level and fat accumulation in liver are the side effects. In severe liver or renal dysfunction avoid mipomerson.

5.10.5. Squalene synthase inhibitor

HMG CoA reductase inhibitors act by reducing mevalonate production. But non-sterols like coenzyme Q10 production depend on mevalonate. This causes elevation of hepatic transaminase level. Because squalene synthase inhibitors inhibit cholesterol synthesis (Figure-1) beyond mevalonate production, squalene synthase inhibitors become the best novel antilipidemic drug. Unlike statins hepatic transaminase level elevation is only moderate with squalene synthase inhibitors [38, 39 and 41].

6. Conclusion

Statin use is contraindicated in pregnant women and in acute liver disease. Ezetimibe monotherapy can be started but in active liver disorder avoid ezetimibe with statin combination. Bile acid sequestrants like colestipol should be avoided in biliary or bowel obstruction. Avoid fibrates in severe liver or renal disorders. For childrens below 10 years ezetimibe usage is not advisable. Among statins use Pitavastatin can be considered because of its less drug interaction. Colesevelam is the only lipid lowering drugs approved by FDA to use safely during pregnancy and also in children. PCSK9 inhibitors cannot be used in hypersensitivity patients. Cost of novel drugs PCSK9 inhibitors has their limitations in use. Novel drugs like CETP inhibitors and PPAR gamma inhibitors have apart from antilipidemic action also have anti-diabetic potential. Novel Squalene synthase inhibitors produce decreased rise in hepatic transaminase level as adverse effect as compare to statins. We need more novel drugs which reduce blood LDL and triglycerides, increase HDL and to reach lipid goal in risky patients. But research should go on not only targeting disease but also their safe usage in other comorbid conditions, in pregnancy, childrens and old people.

Compliance with ethical standards

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Disclosure of conflict of interest

This review article is completely devoid of conflict of interest.

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