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Mini Review

Latest in High-Risk Atherosclerotic Cardiovascular Disease Lipid Guidelines-Mini Review

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Mini Review

Cardiovascular disease is the leading cause of death in the United States, accounting for 23.1% of all deaths in 2018 [1]. Established Atherosclerotic Cardiovascular Disease (ASCVD) is associated with a higher risk of recurrent cardiovascular events. A population-based study found that about 50% of patients with ASCVD experienced a cardiovascular event during the mean follow-up of 7.3 years [2]. Clinical studies established unequivocally that Low-Density Lipoproteins (LDL) cause ASCVD [3], and randomized clinical trials of cholesterol-lowering drugs confirm that lowering of LDL cholesterol (LDL-C) produces marked reductions in new ASCVD events [4]. However, despite awareness of the impact LDL-C levels reduction benefits in Acute Coronary Syndrome (ACS) patients, few of these high-risk patients attain their LDL-C target goals [5].

Lipoprotein(a), known as Lp (a), is a modified form of LDL-C and a well-established risk factor for CVD [6]. It is worth measuring in patients with a family history of premature ASCVD or a personal history of ASCVD that cannot be explained by major risk factors.

2019 European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) Guidelines

The guidelines stratify patients by CV risk, and any patient with documented ASCVD (eg, previous ACS, stable angina, coronary revascularization, stroke, transient ischemic attack, or peripheral artery disease) is considered to be at very high risk. The ultimate treatment goal for secondary prevention in very high-risk patients is an LDL-C reduction of ≥ 50% from baseline and a target LDL-C of < 55 mg/dL. Patients who do not achieve LDL-C goals on maximally tolerated statin therapy should receive combination therapy with a statin plus ezetimibe. In patients who have not achieved their LDL-C goal despite maximally tolerated statin in combination with ezetimibe, a proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor is recommended [5].

2018 American Heart Association/American College of Cardiology (AHA/ACC) Guidelines

Very high risk is defined as a history of multiple major ASCVD events or 1 major ASCVD event and multiple high-risk conditions (age ≥ 65 years, heterozygous familial hypercholesterolemia, history of prior coronary artery bypass surgery or percutaneous

coronary intervention outside of the major ASCVD event(s), diabetes mellitus, hypertension, chronic kidney disease, current smoker, persistently elevated LDL-C ≥ 100mg/dL on maximally tolerated statin and ezetimibe, and history of congestive heart failure). Patients with ASCVD who are at very high risk should receive high-intensity or maximally tolerated statin therapy. In those on maximally tolerated statin therapy who have not achieved LDL-C < 70mg/dL, adding ezetimibe is reasonable. In those on a maximally tolerated statin and ezetimibe therapy who have not achieved an LDL-C < 70mg/dL, adding a PCSK9 inhibitor is reasonable [4].

Pharmacologic Options

Statins and ezetimibe

Based on an extensive body of evidence from CV outcomes trials, statins are the first-line therapy, in addition to healthy lifestyle changes, for reducing LDL-C to reduce CV risk. An ACC/AHA Expert Panel found, in their review of multiple clinical trials, that initiation of moderate-intensity statin therapy (to lower LDL-C by 30% to < 50%) or high-intensity statin therapy (to lower LDL-C by ≥ 50%) is a critical factor to reduce ASCVD events. Ezetimibe is a selective cholesterol absorption inhibitor that lowers LDL-C by blocking the absorption of cholesterol in the intestines. Adding this drug to a statin regimen increases the magnitude of LDL-C lowering by 20% to 25% [4].

PCSK9 inhibitors

These agents are monoclonal antibodies that bind to the PCSK9 protein. Normally,

PCSK9 inhibits LDL receptor recycling: when PCSK9 binds to the LDL receptor, it targets the LDL receptor for degradation, which prevents the LDL receptor from returning to the surface of the hepatocyte to bind more LDL particles [6].

Inclisiran

Inclisiran is a first-in-class, cholesterol-lowering small interfering RNA-based drug that received approval from the European Medicines Agency in December 2020 for use in adults with primary hypercholesterolemia or mixed dyslipidemia as an adjunct to diet. It is not approved in the United States as of May 2021 [7].

Bempedoic acid

Bempedoic acid is an adenosine triphosphate-citrate lyase inhibitor approved by the US Food and Drug Administration (FDA) as an adjunct to diet and maximally tolerated statin therapy for the treatment of adults with heterozygous familial hypercholesterolemia or established ASCVD who require additional LDL-C lowering [8].

Icosapent ethyl

Icosapent ethyl is approved as an adjunct to maximally tolerated statin therapy to reduce the risk of MI, stroke, coronary revascularization, and unstable angina requiring hospitalization in adults with elevated triglycerides (≥ 150 mg/dL) who have either established CVD or who have type 2 diabetes (T2D) and ≥ 2 CVD risk factors [9].

Fibrates

Fibrates lower triglyceride levels by decreasing the liver's production of VLDL and by helping to remove triglycerides from the blood. Fibrates have variable effects on LDL-C levels, depending on the presence of other lipid abnormalities, and can also slightly increase HDL-C levels [10].

Niacin

Niacin is a B-complex vitamin that lowers triglycerides by 20% to 50%, lowers LDL-C levels by 10% to 20%, and raises HDL-C by 15% to 35% [11].

Bile acid sequestrants

Bile acid sequestrants in the intestine by binding to bile and blocking its reabsorption. Bile is made from cholesterol, so these medications reduce the body's total cholesterol to lower total cholesterol and LDL-C. Cholestyramine has been shown to reduce CV events modestly when used as monotherapy in hypercholesterolemic men [12].

Non-Pharmacologic Options

Lifestyle modifications are an essential part of secondary prevention ASCVD patients. Including, but not limited to smoking cessation, consuming a healthy diet composed of vegetables, fruits, whole grains, healthy proteins, and non-tropical vegetable oils, and limiting sweets, sugary beverages, and red meats. adjusting caloric intake to lose weight and performing moderate-to-vigorous aerobic physical activity 3-4 times a week for approximately 40 minutes per session. Whenever possible, patients should also complete cardiac rehabilitation programs [4,5].

References

1. Murphy SL, Xu J, Kochanek KD, Arias E, Tejada-Vera B (2021) Deaths: Final data for 2018. Natl Vital Stat Rep 69(13): 1-83.
2. Lindh M, Banefelt J, Fox KM, Hallberg S, Hui Tai M, et al. (2019) Cardiovascular event rates in a high atherosclerotic cardiovascular disease risk population: Estimates from Swedish population-based register data. Eur Heart J Qual Care Clin Outcomes 5(3): 225-232.
3. Ference BA, Ginsberg HN, Graham I, Ray KK, Packard CJ, et al. (2017) Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European atherosclerosis society consensus panel. Eur Heart J 38(32): 2459-2472.
4. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, et al. (2019) 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: A report of the American college of cardiology/American heart association task force on clinical practice guidelines. J Am Coll Cardiol 73(24): 3168-3209.
5. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, et al. (2020) 2019 ESC/EAS guidelines for the management of dysplipidaemias: Lipid modification to reduce cardiovascular risk. Eur Heart J 41(1): 111-188.

6. Rosenson RS, Hegele RA, Fazio S, Cannon CP (2018) The evolving future of pcsk9 inhibitors. J Am Coll Cardiol 72(3): 314-329.

7. Lamb YN (2021) Inclisiran: First approval. Drugs 81(3): 389-395.

8. Ann Arbor, NEXLETOL (bempedoic acid), Esperion Therapeutics, Michigan, USA.

9. Bridgewater NJ (2019) VASCEPA (icosapent ethyl), Amarin Pharma Inc, Dublin, Ireland.

10. Rubins H, Robins S, Collins D, Fye CL, Anderson JW, et al. (1999) Gemfibrozil for the secondary prevention of coronary heart disease in men with low levels of high-density lipoprotein cholesterol. Veterans affairs high-density lipoprotein cholesterol intervention trial study group. N Engl J Med 341(6): 410-418.

11. Robinson J (2009) Management of complex lipid abnormalities with a fixed dose combination of simvastatin and extended-release niacin. Vasc Health Risk Man 5(1): 31-43.

12. (1984) the lipid research clinics coronary primary prevention trial results. I. Reduction in incidence of coronary heart disease. JAMA 251(3): 351-364.