Statins: friend or foe?

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Abstract

Statins (HMG-CoA reductase inhibitors) are drugs of choice for lipid lowering in patients at increased risk for cardiovascular disease (CVD) and for those with established atherosclerotic VD (secondary prevention). CV risk using risk calculators should be assessed prior to initiation of a statin for most patients being treated for primary prevention of atherosclerotic CVD. Guidelines vary regarding threshold for using statins for primary prevention: the American College of Cardiology/American Heart Association recommends statin therapy for patients in the following groups: patients with a 10-year risk of CVD ≥7.5% with consideration at risk 5% to 7.5%; patients with low-density lipoprotein (LDL) cholesterol ≥190 mg/dL; patients aged 40-75 years with diabetes (type 1 or 2) and LDL cholesterol ≥70 mg/dL. The National Institute for Health and Care Excellence recommends statin therapy if 10-year risk of CVD ≥10%. The European Society of Cardiology/European Atherosclerosis Society recommends statin therapy if estimated 10-year risk of first fatal atherosclerotic event ≥10%. Use of statin in intermediate-risk population also resulted in a significantly lower risk of CV events. Statins are well tolerated, but various statin-associated symptoms (SAS) might occur, including statin-associated muscle symptoms (SAMS), diabetes mellitus (DM), and central nervous system complaints. These SAS are rare in clinical trials, making their causative relationship to statins unclear. SAS are, nevertheless, important because they prompt dose reduction or discontinuation of these life-saving drugs. SAMS is the most frequent SAS, and mild myalgia may affect 5-10% of statin users. Clinically important muscle symptoms are rare, including rhabdomyolysis and statin-induced necrotizing autoimmune myopathy (SINAM). Antibodies against HMG-CoA reductase apparently provoke SINAM. Good evidence links statins to DM, but evidence linking statins to other SAS is not clear. The highest risk for incident diabetes with statins was found in older patients, independently of BMI at inclusion and changes in LDL cholesterol. The preventive effect on cardiovascular events did not change according to changes in HbA1c levels. These observations do not justify any change in clinical practice, except perhaps for a closer follow-up of HbA1c levels after initiating statin therapy. Management of SAS requires making the diagnosis, changing or adjusting the statin treatment, and using alternative lipid-lowering therapy.