Editor’s Message

Targeting Low-Density Lipoprotein Cholesterol in Cardiovascular Health

Kelly Anne Spratt, DO

For many years, heart disease prevention strategies have focused on primary prevention in those who have not had a manifest coronary event and secondary prevention, which is more aggressive, in those with known coronary artery disease (CAD) or an equivalent. Treatment strategies typically include lowering low-density lipoprotein cholesterol (LDL-C) levels and prescribing HMG-CoA reductase inhibitors (statins). However, new imaging modalities such as coronary artery calcium score (CACS) and biomarkers such as C-reactive protein have created a “gray zone” of patients who do not fit primary or secondary prevention—ie, patients with evidence of atherosclerosis but no history of a CAD event. Data now suggest that these patients also benefit from aggressive LDL-C lowering and statins.1,2

The contents of this supplement to JAOA—The Journal of the American Osteopathic Association were developed in part from a seminar held on Wednesday, November 4, 2009, during the American Osteopathic Association’s 114th Annual Osteopathic Medical Conference and Exposition in New Orleans, Louisiana. Michael B. Clearfield, DO, and Keith Ferdinand, MD, led the seminar, which was certified for continuing medical education.

In the first article of this supplement, Dr Clearfield discusses the pleiotropic benefits of statins beyond lowering LDL-C levels, including reducing plaque formation and decreasing inflammation. Advanced imaging such as intravascular ultrasonography has demonstrated a reduction in atheroma plaque volume after statin therapy.3 In addition, one study4 noted a decrease in cardiovascular events by targeting highest-risk patients to reduce LDL-C levels targets as low as 40 to 60 mg/dL. As a marker of vascular inflammation, C-reactive protein may potentially be a future target of treatment efficacy. Studies5-7 including primary and secondary prevention have noted that patients with higher levels of C-reactive protein achieved the greatest benefit from statins as well as the lowest LDL-C levels.

In the next article, Dr Ferdinand presents an illustrative case of a typical patient with metabolic syndrome. In his discussion, Dr Ferdinand highlights the complexity of risk stratification. For example, the Framingham Risk Score, a traditional tool in identifying at-risk patients, may underestimate risk in certain populations.8 Some patients who are placed in a low-risk category may benefit from further LDL-C reduction and initiation of statins. Newer risk models, including the Reynold’s Risk Score,9 which includes high-sensitivity C-reactive

Dr Spratt is clinical associate professor of medicine at the University of Pennsylvania School of Medicine in Philadelphia.

Financial Disclosures: None reported.

Address correspondence to Kelly Anne Spratt, DO, ACC, Penn Presbyterian Medical Center, 51 N 39th St, Philadelphia Heart Institute, Suite 2C, Philadelphia, PA 19104-2640.

E-mail: kelly.spratt@uphs.upenn.edu

This supplement is supported by an independent educational grant from AstraZeneca Pharmaceuticals LP.
protein and family history, may refine reclassification of patients with reclassification for a lower LDL-C target. Imaging modalities such as ankle-brachial index and CACS may also place an apparently low-risk “healthy” patient in a moderate-risk or high-risk group for earlier initiation of statins. Finally, Dr Ferdinand points out that certain higher risk populations such as African American patients may warrant more aggressive risk stratification and treatment targets by virtue that they have increased risk of CAD and chronic kidney disease, a potential surrogate marker of atherosclerosis.12

In summary, additional biomarkers such as C-reactive protein or imaging modalities such as CACS help refine—but do not replace—traditional risk stratification models and optimize reclassification of a “low-risk” patient to a more moderate-risk patient with a lower LDL-C target. Therefore, these biomarkers more accurately target patients who would benefit from an aggressive approach to preventive strategies, further reducing cardiovascular events in patients who meet neither primary nor secondary prevention strategies but represent a whole new group of patients for CAD risk reduction.

References


