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The ASCVD Journey With Lp(a): An ApoB-Family Lipoprotein

Announcer:
Welcome to ReachMD.

This medical industry feature, titled “The ASCVD Journey With Lp(a): an ApoB-Family Lipoprotein,” is sponsored by Novartis Pharmaceuticals Corporation.

Announcer:
Close your eyes and picture a patient with atherosclerotic cardiovascular disease, or ASCVD. What do they look like? Now open your eyes. The patient you see may not be the usual suspects.

Atherosclerosis is a lifelong process that begins from early childhood and ultimately results in cardiovascular disease morbidity and mortality.

Children can have aortic fatty streaks by the age of 3, which increase after 8 years of age and older, heightening the risk of cardiovascular events such as myocardial infarction or ischemic stroke in adulthood.2 Elevated lipoprotein (a), or Lp(a), is the most common inherited dyslipidemia that can drive the atherosclerotic process. Long-term exposure to elevated Lp(a) increases cardiovascular risk.

The impact of Lp(a) starts early in life, with the Lp(a) gene fully expressed by 1 to 2 years of age, reaching adult levels by 5 years of age. Levels of Lp(a) vary by ethnicity, with people of African descent, followed by those of South Asian descent, having the highest levels. 20% of the world’s population, or 1.43 billion people globally, have elevated Lp(a).

So, how does Lp(a) drive atherosclerosis? Through multiple processes, including proatherogenic, proinflammatory, and prothrombotic mechanisms.

Lp(a) is an apolipoprotein B, or apoB-100-containing low- density lipoprotein-like particle.8 Attached to apoB-100 is apo(a), the defining pathognomonic component of Lp(a). Lp(a) is also the primary carrier of oxidized phospholipids, which are found in apo(a) and the lipid core. Lp(a), as an apoB-100-containing lipoprotein, shares the atherogenic characteristics of low-density lipoprotein cholesterol, or LDL-C, such as the propensity to enter arterial vessel walls. Apo(a) further adds to the atherogenic, proinflammatory, and prothrombotic nature of Lp(a). Lysine-binding sites in apo(a) increase accumulation in the vessel wall. Oxidized phospholipids found in apo(a) and the lipid phase of Lp(a) further promote inflammation and progression of lesions. These pathophysiological changes from elevated Lp(a) eventually lead to an increased risk of cardiovascular events.

For example, the likelihood of peripheral artery disease increases by 2-fold. The likelihood of myocardial infarction increases by 3- to 4-fold. And the likelihood of stroke increases by 1.6-fold.

Lp(a) shares all the atherogenic properties of LDL-C and, due to the additional effects of apo(a) and oxidized phospholipids, is considered more atherogenic on an equimolar basis. In patients with elevated Lp(a), an increased risk of cardiovascular events and premature ASCVD persists, irrespective of LDL-C levels, even if patients are optimally treated with approved LDL-C-lowering therapies. In summary, elevated Lp(a) is an inherited, independent, and causal risk factor for ASCVD that is not modified by optimizing LDL-C levels.

So, how can we truly see and help the unusual suspects?

We can:

1. Incorporate LP(a) Incorporate Lp(a) guideline/consensus, testing recommendations to inform clinical practice
2. Stratify cardiovascular risk through assessment of Lp(a) levels
3. Optimize and/or intensify current lipid-lowering treatment per Lp(a) clinical practice guidelines and consensus recommendations to mitigate associated risks of ASCVD

Ultimately, taking these actions for patients with elevated Lp(a) may help them mitigate their risk of cardiovascular events, thereby improving their health and quality of life.

Announcer:
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