Cholesterol Not a Complete Picture of Heart Disease Risk

Lisa, a 43-year-old wife and mother of two teenage children, works as a technologist at a local hospital. She became increasingly concerned about her medical future after her father died without warning of a fatal heart attack at age 59. More recently, her brother, who at 45 is just two years older than Lisa, had his second heart attack. Fortunately, he survived and had a stent placed in one of his coronary arteries.

Lisa has an upbeat, pleasant personality. She is a non-smoker and is thin...
and physically active. She has followed a low-fat diet for a number of years. At the start of the program, her LDL ("bad") cholesterol was 68 and her HDL ("good") cholesterol was a healthy 74—all without any treatment. Her primary-care physician had advised Lisa that on the basis of her lipid values, she is not at risk for a coronary "event" and has somehow escaped the genetic patterns of her father and brother.

Lisa expressed a desire to avoid a heart attack or having to undergo any kind of heart procedure. She therefore had a heart scan (performed using an ultra-fast electron beam tomography, or EBT, scanner) to obtain a calcium score, an indirect measure of silent coronary plaque. Her score of 447 placed her in the worst 1% (99th percentile) of all women her age. In fact, Lisa had the highest score I have ever seen in a woman under 50 years of age. After recovering from the initial shock of discovering her exceptionally high coronary calcium score, she sought to better understand why she had such extensive coronary plaque despite "normal" lipids.

Through a more thorough lipoprotein analysis, Lisa was found to have an abnormally high lipoprotein (a), or Lp(a), level of 85 mg/dl. This, in fact, was the only abnormality in her otherwise perfect profile. (Lisa's testing included lipoprotein analysis performed via nuclear magnetic resonance (NMR) spectroscopy to assess LDL particle number and particle size, HDL sub-fractions, VLDL and its sub-fractions, C-reactive protein, and homocysteine.)

Lisa's treatment program therefore focused on lowering her Lp(a) using:

- L-carnitine—1000 mg twice a day; lowers Lp(a) 7-10%
- Flaxseed—2 tbsp per day, ground; lowers Lp(a) 7%
- Raw almonds—1/4-1/2 cup per day; lowers Lp(a) 7-10%
- Fish oil capsules—4000 mg/day (to yield 1200 mg of omega-3 fatty acids); lowers Lp(a) up to 14%.

Lisa also was advised to add to her diet 6000 mg of l-arginine taken twice a day on an empty stomach. Considering her excellent LDL and HDL scores (and their accompanying sub-fractions), Lisa was advised to continue the low-fat, high-fiber, unprocessed-food diet program that she had begun on her own.

After a year on this program, Lisa's Lp(a) was a very safe 28 mg/dl. Her HDL was 93 and her LDL was 47—notable improvements over her already excellent starting values. A heart scan was repeated to re-assess how much her plaque had grown, and the resulting coronary calcium score was essentially unchanged. (Untreated, the score increases by an average of 30% per year.) Our hope is to achieve a reduction in
score in Lisa’s second year of participation in order to essentially “turn off” Lisa’s coronary plaque.

Discussion
Lisa’s high heart scan score indicates that her coronary plaque was well established and extensive. Lisa’s risk for heart attack over the next 10 years was a significant 45%, with virtually a 100% likelihood of heart attack, bypass surgery, or stents over a longer timeline. Lipids provide a measure of potential coronary disease, but only in a statistical sense; they are not a measure of coronary plaque itself. Lisa’s case is an excellent example of how conventional lipid values can fail to identify risk for coronary disease even when plaque is present to an extreme degree. (By the way, Lisa’s stress test was normal as well. Had we relied on a stress test to determine whether Lisa was at risk, we would have completely “missed the boat.”)

High Lp(a) is an inherited abnormality that has implications for plaque growth, blood clotting, and abnormal artery constriction (“endothelial dysfunction”), and it is frustratingly unresponsive to many lifestyle modifications. Specific approaches are therefore required to treat it effectively. Because high Lp(a) can be very difficult to treat, the treatment program used in this case included both niacin and nutritional adjustments. The flaxseed, raw almonds, L-carnitine, and omega-3 fatty acids prescribed in Lisa’s case have been shown in clinical studies to provide a measurable benefit in reducing Lp(a).

L-arginine, though it does not lower Lp(a), can be a powerful adjunct to facilitate plaque shrinkage, reduce plaque inflammation, and reverse the abnormal arterial constriction that is a particularly difficult problem when high Lp(a) is present.

Lp(a) can manifest as an excessive constrictive behavior in coronary arteries that permits “spasm,” or a vise-like pinching shut of a segment of artery provoked by emotional upset, exposure to cold, or exercise. This leads to chest pain, or angina. Excessive arterial constriction also can manifest as high blood pressure, which can be especially troublesome in people afflicted with high Lp(a), who often require the use of three, four, or five anti-hypertensive medications to lower their blood pressure. L-arginine has blood-pressure-lowering effects that, in my experience, will often permit the subtraction of one or more anti-hypertensive medications when used consistently over a period of three to six months.
Omega-3 fatty acids (as fish oil) can lower Lp(a), particularly when triglycerides are greater than 150 mg/dl. Even in the absence of elevated triglycerides, however, the benefits of omega-3 fatty acids are so broad—a 30% reduction in coronary “events” and sudden death, a mild HDL-raising effect, and reduced fibrinogen (a blood-clotting protein that can lead to heart attack), not to mention the cancer-preventing effects—that they are easily justified in a case like this.

**Conclusions**

High Lp(a) is a potent cause of heart disease that is not evident through standard cholesterol testing but can lead to heart attack in women as early as in their mid-50s and in men as early as in their late 40s. A combination of niacin and several nutritional supplements—especially flaxseed, raw almonds, l-carnitine, l-arginine, and omega-3 fatty acids—can help control this potent and often unrecognized cause of heart disease.

**Editor's note: Some people cannot tolerate high amounts of niacin because of the resulting "niacin flush" or excess stomach acidity. Those with liver disease should not take niacin.**

**References**

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