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Commentary & Perspective

What's Good for the Heart Is Good for the Shoulder?

Commentary on an article by Chang-Meen Sung, MD, et al.: "Are Serum Lipids Involved in Primary Frozen Shoulder? A Case-Control Study"

Mark D. Lazarus, MD

There are countless jokes told about the supposed lack of general medical knowledge held by the typical orthopaedic surgeon. Perhaps studies such as this may slowly erode that source of humor.

One would have to be isolated from all lay press to be ignorant of the association between hyperlipidemia and heart disease. Less known is the correlation with orthopaedic conditions, specifically problems of the shoulder. For instance, the data on the relationship between hypercholesterolemia and rotator cuff tear have been mixed, with some studies¹ recognizing a connection and others² failing to demonstrate a link. In an animal model, hypercholesterolemia has been shown to negatively affect the mechanical properties of the rotator cuff tendon³.

Similarly, there has been some controversy with regard to the relationship between hypercholesterolemia and hyperlipidemia with frozen shoulder; some studies have suggested a strong association⁴ but others have not found any significant relationship⁵. Some of these differences may lie in which lipoproteins were measured and by what testing technique. Alternatively, the different conclusions may simply reflect differences in the patient populations. In their study, Dr. Sung and colleagues observed patients from a single practice, with an age and sex-matched control group, to attempt to rectify these differences. Also, in contrast with prior studies, the authors excluded patients with diabetes and thyroid abnormalities. Finally, the cholesterol and lipoprotein testing protocol was thorough. These authors demonstrated that total cholesterol, low-density lipoprotein, and high-density lipoprotein levels are significantly associated with idiopathic frozen shoulder. Similar associations were found between certain inflammatory lipoproteinemias. No connection was found with hypertriglyceridemia.

Case-control studies such as this clearly have inherent methodological limits that restrict drawing any conclusions other than associations. For instance, there may be dramatic differences between the case and control populations other than their serum lipid profile. Did the two patient populations have similar dietary and exercise habits? What percentage of the patients in each group was taking statin medications? We know that the use of certain medications is associated with frozen shoulder. Was there an association with the use of statin medications and the development of frozen shoulder? More importantly, was normalizing the serum cholesterol level by use of statin medications associated with a lower risk of developing frozen shoulder?

The authors tell us that serum high-density lipoprotein may be an acute-phase reactant, naturally elevated in response to inflammatory conditions. Because the initial phase of frozen shoulder is an intense inflammation (synovitis and capsulitis), are the findings of elevated serum high-density lipoprotein presented in this study merely an inflammatory marker responding to an inflammatory process? In the study patient population, there was a wide range in the duration of symptoms at the time of presentation (less than one to sixty months) and in the time from initial presentation to blood sampling (zero to 118 days), potentially further confusing the role of inflammatory response as a cause of higher serum high-density lipoprotein levels in the study population.

The authors include a large number of controls (three times the number of patients), so the differences in cholesterol and lipoprotein levels between patients and controls easily reach significance. However, the larger question is whether the differences are clinically important. Some of the absolute differences between cases and controls are small, and there is a large overlap in the range of findings between the groups. For instance, is an 8-point difference in the low-density lipoprotein level or a 3-point difference in the high-density lipoprotein level clinically important? Would a physician apply a treatment to reduce a low-density lipoprotein level from 126 mg/dL to 118 mg/dL? The authors do comment on this and suggest that, in the cardiology literature, these small numerical differences are still clinically important.

Finally, the conclusions in this article hold only for idiopathic frozen shoulder and only in patients with this specific nationality. Shoulder stiffness secondary to trauma, surgery, diabetes, or any of the other known underlying etiologies of frozen shoulder may not have the same correlation with hypercholesterolemia and hyperlipidemia. Similarly, patients of different nationalities may not demonstrate the same associations.

Despite these weaknesses, the demonstration of an association among cholesterol, lipoproteins, and frozen shoulder leads to some intriguing hypotheses. Early idiopathic frozen shoulder, prior to the freezing phase, can be a difficult diagnosis to make. Could

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demonstrating a specific pattern of serum lipid protein elevation differentiate frozen shoulder from other inflammatory diagnoses? Can the incidence or disease course of frozen shoulder be modulated by employing strategies to reduce hypercholesterolemia and hyperlipoproteinemia, such as exercise, diet change, and medical management?

The authors should be congratulated for a rigorously performed study that provides important conclusions. They have also given us a hospital cafeteria conversation starter with our medical colleagues. Maybe if we all alert our internal medicine and cardiology friends to the association between hypercholesterolemia or hyperlipidemia and shoulder pathology, we can slowly eliminate the use of the medically ignorant orthopaedic surgeon as the butt of so many jokes.

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