Antibodies linked to cardiovascular disease increase in patients with active lupus

25 February 2010

A study by researchers in Australia and the United Kingdom suggests that autoantibodies to fat binding proteins significantly increase in systemic lupus erythematosus (SLE) patients with active disease. This increase in anti-apolipoprotein (anti-Apo A-I), anti-high-density lipoprotein (anti-HDL), and anti-C-reactive protein (anti-CRP) may contribute to the development of atherosclerosis in SLE patients, placing them at risk for cardiovascular disease (CVD). Complete findings of this study are available in the March issue of *Arthritis & Rheumatism*, published by Wiley-Blackwell on behalf of the American College of Rheumatology.

Lupus is a chronic autoimmune disease where the immune system creates antibodies that attack an individuals' own cells, causing inflammation throughout the body. The inflammation leads to tissue and organ damage, affecting the heart, kidneys, lungs, brain, blood, skin and/or joints of those with SLE. According to a 2008 study for the National Arthritis Data Workgroup 322,000 Americans have a definite or probable SLE diagnosis. The Lupus Foundation of America's figures are much higher, with up to 1.5 million in the U.S. and close to 5 million worldwide reported having form (SLE, discoid, sub-acute cutaneous, drug-induced, or neonatal) of lupus.

In the current study serum levels of anti-Apo A-I, anti-HDL, and anti-CRP were taken from participants that included 39 SLE patients with high disease activity over the previous 2-year period; 42 SLE patients with low disease activity over the previous 2 years; 16 patients newly diagnosed with lupus nephritis (inflammation of the kidney caused by SLE); 25 patients with samples obtained at the time of a SLE flare and during inactivity of the disease; 24 SLE patients who had prior CVD events; and 34 healthy subjects in the control.

Researchers found that antibodies above the upper limit of normal (ULN) were higher in patients in the high disease activity group compared with the low disease activity group: anti-Apo A-I were higher in 35.9% vs. 12% of subjects; anti-HDL levels at 44.7% vs. 30.9%; and anti-CRP at 26.3% vs. 12.8%. Results further indicate that in 55% of the subjects, anti-Apo A-I levels were higher at the time of a disease flare compared with only 34.5% in preflare samples. "The main finding in our study was that levels of anti-Apo A-I and anti-HDL were significantly higher in patients with greater disease activity than in those with less active disease over the same period," said the authors.

In her editorial also published in Arthritis *& Rheumatism*, Bevra Hahn, M.D., from the David Geffen School of Medicine at the University of California Los Angeles, acknowledged that the study by O'Neill et al provided a novel method for studying association of autoantibodies with active disease by classifying SLE patients according to sustained chronic disease activity (or not) instead of the traditional approach of using a validated scoring system that identifies active disease at one point in time. "While this is an important step, measuring antibodies to Apo A-I, HDL or CRP in SLE patients has not yet reached the point where it can be used routinely to identify risk of accelerated atherosclerois," commented Dr. Hahn. "As risk prediction models emerge over the next few years, these antibodies may be included along with other predisposing variables."

More information: "Antibodies to Apolipoprotein A-I, High-Density Lipoprotein, and C-Reactive Protein Are Associated With Disease Activity in Patients With Systemic Lupus Erythematosus." Sean G. O'Neill, Ian Giles, Anastasia Lambrianides, Jessica Manson, David D'Cruz, Leslie Schrieber, Lyn M. March, David S. Latchman, David A. Isenberg,and Anisur Rahman. Arthritis & Rheumatism; Published Online: February 25, 2010 (DOI: 10.1002/art.27286); Print Issue Date: March 2010.

Provided by Wiley