Shorts

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Antiphospholipid Antibody Syndrome Linked with Chronic Fatigue Syndrome

Ken Lassesen, MS, developed chronic fatigue immune deficiency syndrome (CFIDS) in 1999. He considers himself lucky because he worked for Microsoft and, therefore, had health insurance that put little restriction on testing. He also had a family-practice doctor who "believed that CFS was very real." She didn't know how to treat it, but she was willing to learn from peer-reviewed research. Lassesen found a study by David Berg et al. that linked chronic fatigue syndrome (CFS) to antiphospholipid antibody syndrome (APS) (aka Hughes syndrome), a hypercoagulable condition. The characteristic "sticky" blood impedes the delivery of oxygen and nutrients throughout the body and removal of cellular waste and toxins.

In their October 1999 paper for *Blood Coagulation & Fibrinolysis*, David Berg and colleagues describe their blinded prospective study for testing the hypothesis that most people with CFS or fibromyalgia (FM) have antiphospholipid syndrome. (CFS and fibromyalgia share many physiological characteristics and symptoms.) Fifty-four people diagnosed with CFS or fibromyalgia and 23 controls were given five tests to evaluate their blood: fibrinogen (FIB), prothrombin fragment 1 + 2 (F1 + 2), thrombin/antithrombin complexes (T/AT), soluble fibrin monomer (SFM), and platelet activation by flow cytometry (PA) using CD62P and ADP. The object was to differentiate the controls from the patients simply by looking at the test results. People with two or more assays that indicated hypercoagulability were labeled patients. The researchers correctly identified 22 of the 23 controls and 50 of the 54 patients: "One control was positive in two assays for a false positivity rate of 4%. Of the 54 patients, 4 had normal values, for a false negative rate of only 7.4%. This shows that 92+% of CFS and/or FM patients had a demonstrable hypercoagulable state." In previous, unpublished research, Berg et al. found that "three out of four CFS &/or FM patients have a genetic deficiency [for thrombophilia or hypofibrinolysis]" – which may promote "sticky" blood. They also report that "[c]ertain pathogens induce the immune system generation of APL antibodies and can be a triggering mechanism for APS." Several pathogens have been linked to the onset of CFS and FM. In an editorial for *Annals of Rheumatic Diseases*, Y. Shoenfeld and colleagues "suggest that molecular mimicry mechanism between the pathogen and the [β2-glycoprotein I] molecule may be the cause of [antiphospholipid antibody syndrome]."

In Ken Lassesen's case, he and his doctor decided to focus on three goals: "eliminate the infection(s) that cause the antibodies; eliminate items that trigger coagulation [e.g., stress, chemical and perfume exposure]; get assistance in reducing coagulation." Protocols developed by Professor Garth Nicolson and Cécile Jadin, MD, gave them a place to start. Dr. Jadin, a South African surgeon, recognized the symptoms of CFIDS as being akin to Rickettsia infection. Her father investigated Rickettsia while working at the Pasteur Institute. As Lassesen improved, his family doctor turned attention to Lassesen's wife and two daughters, who also suffered with an array of puzzling symptoms. Tests found hypercoagulation problems in them, but their clinical symptoms differed. Lassesen's "For What's It Worth" or "Zero-Based Protocol" outlines the progression of his treatment. It is posted at http://lassesen.com/cfids/ZeroBasedProtocol.htm.

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Shoenfeld Y, Blank M, Cervera R, Font J, Raschi E, Meroni P-L. Infectious origin of the antiphospholipid syndrome. *Ann Rheum Dis*. January 2006; 65(1): 2–6. Available at: www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1797971. Accessed August 11, 2009.

Breathing Technique May Help Nervous System in Chronic Fatigue

People with chronic fatigue usually exhibit signs of autonomic nervous system dysfunction. Signs include rapid heart rate, reduced diastole speed (when blood flows into the heart), increased systole rate (when heart contracts, pushing blood out of the heart), and low heart rate variability. A September 2007 study reports that