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WISCONSIN CHAPTER

Rheumatoid Arthritis and Heart Disease

The natural course of rheumatoid arthritis (RA) is a chronic, progressive inflammation leading to articular destruction and increased morbidity and premature death. The leading cause of death in RA patients is cardiovascular disease (CVD). Compared to the general population, when controlling for traditional cardiovascular disease risk factors such as smoking, diabetes mellitus, hypertension, and hyperlipidemia, patients with RA were 2-3 times at increased risk for developing CVD.

Recent studies have suggested that part of the mechanism of CVD in RA is independent of the known risk factors, but can synergistically accelerate the disease process. It is thought that chronic inflammation in RA enhances the development of atherosclerosis. Proposed theories include the development of vascular damage from both endothelial dysfunction and impaired vascular repair caused by the chronic exposure to inflammatory mediators. The effects of cytokines, immune complexes, elevated levels of C-reactive protein, or rarely, coronary vasculitis has thought to play a role in the development of atherosclerosis. To further support the role of inflammation in the pathogenesis of CVD, studies evaluating RA patients treated more aggressively with DMARDs (disease modifying anti-rheumatic drugs) and biologic agents had a decreased incidence of cardiovascular events compared with less aggressively treated RA patients. In addition, RA patients with more severe disease, higher CRP values, and those that had positive anti-CCP antibodies had increased cardiovascular events.

The clinical manifestations of symptomatic coronary artery disease (CAD) in patients with RA are similar to those without RA. However, a larger number of patients with RA have clinically silent CAD than do individuals in the general population. Hence, patients with RA are less likely than those without RA to report chest pain during an acute coronary event. It is unclear why more RA versus non-RA patients have clinically silent CAD, but may be related to less physical activity due to structural joint damage or from an altered pain perception from anti-inflammatory and DMARD therapy.

Prevention and treatment of CVD in RA and non-RA patients are similar, however there are some unique issues related to RA patients. As in non-RA patients, it is important to address modifiable risk factors, such as smoking cessation, exercising, and implementing a healthy diet. However, it may be difficult for some RA patients to exercise depending on the amount of joint disease present, referring to a physical therapist for catered exercise regimens is important. Blood pressure and blood sugar control may be difficult to achieve in some RA patients who are being treated with glucocorticoids and non-steroidal anti-inflammatory drugs. Limiting the amount of glucocorticoids and NSAIDs helps in reducing CVD risk. Using glucocorticoids in the lowest dose for the shortest duration possible can help in risk reduction. Hyperlipidemia needs to be treated aggressively in this patient population. Currently, the guidelines for hypercholesterolemia are the same for RA and non-RA patients however, lipid lowering therapy as primary prevention of CVD has not been adequately studied in the RA population. From studies done thus far, physicians should have a lower threshold for starting lipid lowering agents and following strict LDL goals in RA patients.

Awareness of the heightened cardiovascular disease risk in RA patients is important for the internist and rheumatologist as well as other caretakers. Education and risk reduction measures should be aggressively implemented in this patient population.

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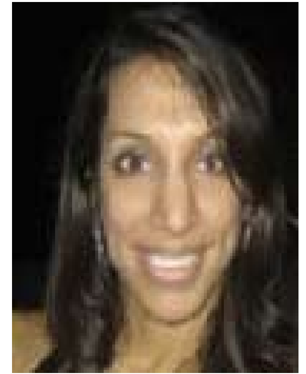

Ramona Raya is a board certified rheumatologist, currently practicing at the Medical College of Wisconsin. She obtained her Bachelor in Sciences at Case Western Reserve University in Cleveland, OH in 1999. She then completed medical school at University of Nebraska Medical Center in 2003. From 2003-2006 she underwent internal medicine residency training at Georgetown University Washington Hospital Center. She completed her fellowship in Rheumatology at the National Institutes of Health in Bethesda, MD, where she also served as the Chief Fellow in 2007-2008. She then joined MCW as an Assistant Professor of Medicine in 2008.

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