Malgorzata Wislowska, Rheumatology 2017, 7:1(Suppl)
http://dx.doi.org/10.4172/2161-1149.C1.014

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Rheumatoid arthritis (RA) and heart diseases

RA is a chronic inflammatory disease affecting approximately 0.5-1% of the population, characterized by symmetric, erosive arthritis of the synovial joints and variable extra-articular features. The primarily affected site is the synovium where "pannus" is gradually formed. Typical articular symptoms include pain, stiffness and swelling. The progressive destruction of the articular cartilage may lead to deformation and loss of function of affected joints. The goal of treatment is to reduce mortality and to prevent joint damage and disability. RA imposes a substantial economic burden on both patients and society. The etiopathogenesis of RA is only partially understood. Its onset is triggered by environmental factors on the basis of a genetic predisposition in combination with altered immune responses. Tobacco and infection by Porphyromonas gingivalis during periodontal diseases favor ACPA production which has a crucial role in RA pathogenesis. Cytokine networks involving TNFα, IL-1β, 6, 15 and 17, matrix metalloproteinases and many other factors participate in disease perpetuation. Bone and cartilage destruction are primarily mediated by osteoclasts and fibroblasts – like synoviocytes, respectively. Although RA is primarily considered a disease of the joints, abnormal systemic immune responses can cause a variety of extra – articular manifestations. One of the current issues in rheumatology is the type and extent of pathological changes in the heart in the course of rheumatoid arthritis. The problems with diagnosis are due to a very limited number of clinical symptoms showing that cardiac muscle is affected by a disease process and that symptoms do not always appear. The results of epidemiological studies point to the presence of an increased risk of cardiovascular disease (CVD), particularly atherosclerosis and congestive heart failure (CHF) in RA. Necropsy examinations of RA hearts revealed a higher prevalence of clinically silent myocardial nodules, restrictive pericarditis and coronary vasculitis in RA compared to non-RA patients. At least 50% of abnormalities remained asymptomatic. Pathological conditions contributing to myocardial dysfunction such as high serum levels of IL-6, C-reactive protein and TNF alpha are present both in RA and CHF patients. The most common pathological mechanism leading to the development of heart failure is left ventricular diastolic dysfunction, which remains clinically asymptomatic for a long time. The presence of heart disease in RA patients is not only dependent on concurrent heart disease but is also caused by RA disease.

Biography

Margaret Wisłowska, Head of The Department of Internal Medicine and Rheumatology CSK MSW is a specialist in internal medicine, rheumatology, rehabilitation medicine, hypertention, and the author of over 200 scientific papers and books. She has participated in numerous scientific meetings and is a promoter of 12 PhD theses. She took trainings at Guy and St. Thomas' Hospitals in London, Charity Hospital in Berlin, Rheumatology Institutes in Prague and Moscow. In 2003, she started the Department of Internal Medicine and Rheumatology, and in 2010 the Clinic of Internal Medicine and Rheumatology CSK MSW. She is a Professor at the Warsaw Medical University.

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