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The time-course effect of high-grade inflammation on left ventricular function and morphology in the collagen-induced arthritis model

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Statement of the Problem: High-grade inflammation plays a key role in the pathogenesis of heart failure in patients with long-standing rheumatoid arthritis (RA). However, the time-course effect of inflammation on left ventricular (LV) function and morphology in experimental models of RA is unknown. This study aimed to determine the time-course effects of high-grade inflammation on LV function and morphology in collagen-induced arthritis (CIA). Methodology & Theoretical Orientation: Thirty-nine, Sprague Dawley rats were divided into the control group (n=18) and CIA group (n=21). Rats in the CIA group received subcutaneous injections of bovine type-II collagen emulsified in incomplete Freund's adjuvant. Echocardiographic measures were assessed at week 3, week 6 and week 9 after CIA induction. At termination, circulating inflammatory markers were measured and the collagen content in the LV was determined by picro-sirius red staining. Findings: Compared to controls at week 9, CIA rats had increased tumor necrosis factor α (TNF-α) and C-reactive protein (CRP) ($p<0.05$). Compared to controls at week 9, CIA rats had reduced myocardial relaxation (e' and E/A) and increased filling pressures (E/e') ($p<0.05$). The collagen area fraction in the CIA group was significantly higher than the control group at week 9 ($p=0.001$). Increased TNF-α and CRP were associated with reduced e' ($p= 0.02$; $r= -0.84$ and $p= 0.02$; $r=-0.71$, respectively) and increased CRP was associated with increased E/e' ($p=0,01$; $r= 0,77$) at week 9. Increased TNF-α and CRP were associated with increased collagen area ($p=0.03$; $r=0.74$ and $p=0.02$; $r=0.81$ respectively). Conclusion & Significance: High-grade inflammation causes diastolic dysfunction in the late stages of arthritis without any changes in CIA rats. In addition, myocardial fibrosis worsens with an increased duration of exposure to inflammation.

Biography

Dr Lebogang Mokotedi is a member of the Cardiovascular Pathophysiology and Genomics Research Unit (CPGRU) which carries out research over wide areas, each designed to increase the understanding of the mechanisms of cardiovascular disease, and the roles of various lifestyle factors in either the development or prevention of hypertension and cardiovascular disease. Within the CPGRU, Dr Mokotedi's research focuses on the role of chronic inflammation on cardiac and vascular function in rheumatoid arthritis patients and a rodent model of arthritis. She is also involved in projects looking at modifiable metabolic, cardiovascular, and lifestyle markers that contribute to cardiovascular disease in different ethnic groups and experimental models.

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