Update on Cardiovascular Disease Risk in Patients with Rheumatic Diseases

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KEYWORDS

• Rheumatoid arthritis • Cardiovascular • Lipids • Lipoproteins • Myocardial fibrosis

KEY POINTS

- Cardiovascular disease (CVD) risk calculators underestimate CVD risk in rheumatoid arthritis (RA) and should be multiplied by 1.5 to reflect the greater than 1.5 times higher risk of CVD among adults with RA, even with no traditional CVD risk factors, although risk increases substantially with the number of CVD risk factors.
- Current CVD risk factors, particularly total and low-density lipoprotein (LDL)-C, likely underestimate the extent of subclinical atherosclerosis.
- LDL or high-density lipoprotein (HDL) particles, or apolipoprotein (apo)-B or ApoA1, may be more reliable CVD risk factors than cholesterol (total, LDL, or HDL) concentrations because of chronic inflammation.
- Reduction in inflammation may prevent or reduce myocardial injury and heart failure.
- Disease activity is a strong risk factor for CVD and mortality, and a key target for CVD risk reduction.

INTRODUCTION

Cardiovascular disease (CVD) risk is increased in rheumatoid arthritis (RA) and other inflammatory autoimmune rheumatic diseases, which have a lifetime risk of adult onset of 1 in 12 for women and 1 in 20 for men.[1] This review focuses on the most common RA, which occurs 2 to 3 times more often in women than men. The risk for CVD and total

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mortality is greater than 1.5 times higher in RA patients and 10-year CVD risk scores underestimate risk. CVD is more likely to be fatal, and unrecognized myocardial infarction (MI), sudden death, and heart failure (HF) are increased. More aggressive primary and secondary prevention of CVD is needed in RA patients,[2,3] many of whom are postmenopausal women. The current review focuses on the following (1) the role of dyslipidemia in RA-related CVD risk, (2) the risk of inflammation-related myocardial disease and eventual HF, and (3) the emergence of RA disease activity as a key focus for CVD risk prediction and CVD risk reduction in RA.

RA is associated with greater than 1.5-fold higher risk of coronary heart disease (CHD), CVD, HF,[4,5] venous thrombosis,[6,7] fatal CVD, total mortality,[8–10] and other CVD outcomes (**Box 1**). Unrecognized MI, sudden death,[11] and asymptomatic HF[12] are all increased among RA patients. The greater than 1.5-fold higher risk of CVD exists at most levels of traditional CVD risk factors, even among individuals with no smoking, diabetes, hypertension, or history of hypercholesterolemia, as shown in the Women's Health Initiative (WHI)-RA Study (crude relative risk is 10.75/6.35 = 1.69) (**Table 1**).[13] CVD risk in RA is strongly related to traditional CVD risk factors; for example, cigarette smoking, hypertension, diabetes, and hyperlipidemia.[13–16] The risk factor profile in RA (**Box 2**) includes higher prevalence of smoking, hypertension,[17] diabetes,[14] and obesity, although some RA patients have low body mass index (BMI).

In contrast, the role of dyslipidemia in RA has been questioned, due to a lipid paradox. RA patients have lower levels of total cholesterol (TC) and low-density lipoprotein (LDL) cholesterol (LDL-C) than adults without RA.[18] Increased CVD risk is associated with low levels of TC and LDL-C.[19] TC and LDL-C levels decrease before RA diagnosis,[20] often increase in response to antiinflammatory medications, and decrease in response to flares of RA disease activity. The paradoxically low TC and LDL-C levels in many RA patients contribute to underestimation of CVD risk by CVD risk scores (eg, Framingham Risk Score, Reynolds Risk Score,[21] and the Systematic Coronary Risk Evaluation [SCORE][22]). These have been shown to incorrectly classify as low risk approximately one-third of patients who subsequently had CVD events[22] and approximately 60% of RA patients with coronary artery calcification greater than 300.[23]

WHAT EXPLAINS THE EXCESS CARDIOVASCULAR DISEASE RISK IN RHEUMATOID ARTHRITIS?

Active RA is characterized by systemic inflammation that is credited with much of the excess risk of CVD and mortality in RA. The contribution of inflammation to

Box 1
Cardiovascular diseases increased in rheumatoid arthritis

- MI (often unrecognized)[11]
- Sudden death[11]
- Stroke[8]
- Venous thrombosis[6,7]
- HF[4,5]
- Diastolic dysfunction[63]
- Peripheral vascular disease[78]
- Subclinical atherosclerosis[52,54,55]
- Endothelial dysfunction[79]

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