Preface

Cardiovascular Comorbidities in Inflammatory Rheumatic Diseases

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Editors

Patients with inflammatory rheumatic diseases experience higher cardiovascular morbidity and mortality compared with age- and gender-matched individuals in the general population. Those include atherosclerotic, ischemic complications as well as nonatherosclerotic events, such as venous thromboembolism, myocardial involvement, and heart failure. Disease flares and high cumulative inflammatory burden are characteristic of the systemic autoimmune diseases and constitute major, independent contributors to cardiovascular comorbidity. Moreover, chronic inflammation synergizes with traditional cardiac risk factors in promoting cardiovascular comorbidity, facilitates the development and appearance of some, like insulin resistance, diabetes mellitus, and hypertension, while altering the impact of others, such as lipids, lipoprotein levels, and obesity.

Disease-specific or -associated immune responses further contribute to atherosclerosis development by affecting lipoprotein composition and function, cholesterol loading to arterial wall macrophages, foam cell formation, plaque instability, and rupture. Several of them serve as predictive and prognostic biomarkers, optimizing risk estimates for atherosclerotic plaque presence, progression, and cardiovascular events above and beyond traditional risk factor models. The evaluation of subclinical atherosclerosis across various vascular territories and using different, noninvasive imaging modalities, additionally optimize cardiovascular risk estimates. Future risk prediction models combining traditional risk factors, disease-specific characteristics, information from disease-associated biomarkers, imaging, and synthesized with the help of machine- and deep-learning modalities of artificial intelligence may further optimize risk recognition and allow the development of more effective and comprehensive cardioprotective strategies.

The identification of disease-associated cellular and molecular processes and their specific targeting with biological or newer synthetic disease-modifying agents has

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revolutionized our ability to achieve low-disease activity or remission and comprehensive inflammatory control. Above and beyond their potent anti-inflammatory effects, these agents may further optimize lipoprotein structure and function, cholesterol accumulation, and efflux from atherosclerotic plaques and influence body composition. Interestingly, most widely available analgesics, such as nonsteroidal anti-inflammatory drugs, used for short-term pain control can have long-term unintended cardiac risks. Overall, these treatment benefits should be judiciously weighed against the potential of harm in these patients. Beyond the development of highly efficient and disease-specific targeted therapies, the increasing and broader appreciation of accelerated risk, prompt and diligent clinical screening, and application of effective prevention campaigns have yielded significant improvements in inflammatory disease-associated and treatment-associated cardiovascular risk.

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