





The predictive value of various biomarkers of chronic inflammation associated with cardiometabolic disease

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An alarming increase in the development of cardiovascular (CV) disease and recent knowledge about the common pathophysiological background of cardiometabolic disease, with inflammation being in the center of the pathophysiological networks, has indicated that a more integrated approach is needed for classifying these disorders.¹ Chronic inflammation is considered to be a major force driving the development of cardiometabolic diseases such as myocardial infarction (MI), stroke, diabetes type 2 (T2D). It is known that changes in the body's shape and structure that occur with aging, followed by muscle loss and an increase in visceral fat, contribute to inflammation and the development of insulin resistance, which taken together increase the risk for metabolic and vascular disorders associated with them.² Currently, there is no consensus as to which markers of inflammation best represent which phases of chronic inflammatory response. Interleukin 6 (IL-6) and C-reactive protein (CRP, whose production is stimulated by IL-6) are arguably the two most commonly assayed biomarkers used to stratify risk in patients with CV risk factors. Nowadays, a more prevalent role has been given to the neutrophil-to-lymphocyte ratio (NLR) and new sets of markers of inflammation including interleukins IL-17A and IL-37, that have been explored for their potential use for risk stratification in everyday clinical practice.³ The evaluation of their circulating levels might provide new insights into the course of disease, and may guide the prognostics and emerging therapeutics in area of cardio- metabolic disease. In contrary to IL-17A, which plays a central role in the process of end-organ damage and is complementary to NLR, alternative anti-inflammatory treatment of IL-37, may turn out to be more effective, depending on genetic predispositions, duration, and manifestation of the disease.

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