

IMMUNE MECHANISMS OF SUBCLINICAL MYOCARDIAL INJURY IN ARTERIAL HYPERTENSION: THE ROLE OF CYTOKINES AND REMODELING FACTORS

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Relevance. Cardiovascular diseases remain the leading cause of morbidity and mortality worldwide, with arterial hypertension being one of the major modifiable risk factors for ischemic heart disease. In recent years, increasing attention has been paid to subclinical myocardial injury, which precedes the clinical manifestation of coronary pathology. Immunoinflammatory mechanisms, including cytokine imbalance, complement activation, and myocardial remodeling, play a key role in these processes; however, their prognostic significance remains insufficiently studied.

Aim. To evaluate the contribution of inflammatory mediators and angiogenesis/fibrosis-related factors to the development of subclinical myocardial injury in patients with arterial hypertension.

Materials and methods. The study included 78 patients with arterial hypertension who were examined in a specialized cardiology setting. Depending on the clinical characteristics, patients were divided into two groups: Group I (n=37) — patients with isolated arterial hypertension; Group II (n=41) — patients with arterial hypertension combined with ischemic heart disease. The control group consisted of 20 conditionally healthy individuals.

Serum levels of high-sensitivity cardiac troponin (hs-cTn), VEGF A, TGF β , interferons (IFN- α , IFN- γ), as well as pro-inflammatory markers (IL-6, C-reactive protein) and complement component C3a were assessed. Statistical analysis included evaluation of intergroup differences and correlation analysis ($p < 0.05$).

Results. It was established that progression of myocardial injury was associated with pronounced activation of immunoinflammatory mechanisms. Increased hs-cTn levels were accompanied by a stepwise elevation of VEGF A and TGF β , reflecting activation of angiogenesis and fibrotic remodeling. VEGF A and TGF β levels increased up to 6.2-fold and 6.9-fold, respectively, in patients with manifest ischemic heart disease. At the same time, a significant increase in pro-inflammatory markers was observed: IL-6 increased up to 4.9-fold and CRP up to 6.5-fold, indicating activation of systemic inflammatory response. Complement activation (C3a) demonstrated the most pronounced dynamics, increasing up to 36.7-fold at advanced stages of subclinical myocardial injury. The dynamics of interferons were multidirectional: IFN- α levels increased at early stages of myocardial injury but decreased in patients with manifest ischemic heart disease, while IFN- γ remained persistently elevated, reflecting chronic inflammation. Correlation analysis revealed a significant association between hs-cTn levels and IL-6 ($r \approx +0.62$), CRP ($r \approx +0.58$), and C3a ($r \approx +0.71$), confirming the relationship between myocardial injury and activation of inflammatory and complement pathways.

Conclusion. Subclinical myocardial injury in arterial hypertension is accompanied by a staged activation of immunoinflammatory mechanisms, including cytokine imbalance, complement activation, and remodeling processes. VEGF A, TGF β , IL-6, CRP, and C3a can be considered reliable biomarkers of disease progression and may be used for early risk stratification and prediction of ischemic heart disease development in patients with arterial hypertension.

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