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Abstract

Objective: It was proposed that proinflammatory cytokines can modulate cardiac structure and function through mechanisms resulted in heart hypertrophy, fibrosis, cardiomyocytes apoptosis, left ventricular diastolic and systolic dysfunction. Tumor necrosis factor- α (TNF- α) – proinflammatory cytokine can cause negative inotropic effects on the heart, activity of which can inhibits by soluble forms of TNF receptors type 1 (sTNF-R1).

The aim of our clinical study was to investigate circulating plasma TNF- α , sTNF-R1 levels in patients with arterial hypertension depend on left ventricular hypertrophy presence.

Design and methods: TNF- α and sTNF-R1 plasma levels were measured by ELISA in 72 hypertensive patients. Left ventricular myocardium mass (LVMM) and function by 2D echocardiography have been examined. Patients were divided into 2 groups depend on left ventricular hypertrophy (LVH) presence: 1st group – 22 patients (30.56%) with normal LVMM (LVMM index 103.59 ± 2.41 kg/m²; EF $63.14 \pm 1.33\%$), 2nd group – 50 patients (69.44%) with LVH (LVMM index 137.38 ± 2.75 kg/m²; EF $58.81 \pm 1.14\%$).

Results: Plasma TNF- α (139.52 ± 2.11 pg/ml) and sTNF-R1 (2.15 ± 0.11 ng/ml) levels were higher in hypertensives with LVH as compared with those without LVH (121.24 ± 26.04 pg/ml; $p < 0.05$ and 2.13 ± 0.15 ng/ml; $p > 0.05$). We calculated TNF- α /sTNF-R1 ratio which reflect degree of immunoinflammatory activity. This ratio was statistically elevated (64.89) in hypertensives with LVH vs hypertensives without LVH (56.92; $p < 0.05$) that indicate increased proinflammatory response in this group. Positive correlation between TNF- α and LVMM index were found in 2nd group ($r = 0.64$; $p < 0.05$) and negative with EF ($r = 0.53$; $p < 0.05$).

Conclusions: The result of our clinical study suggest that elevated levels of proinflammatory cytokine – TNF- α may be one of several different maladaptive mechanisms responsible for the progression of cardiac decompensation that occur in arterial hypertension. This study revealed increased levels of sTNFR1 that might reduce or in some cases completely inhibits TNF- α activity. Measurement of sTNFR1, in addition to TNF- α , could provide us with some additional and more complete information about activation of this cytokine in arterial hypertension.