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ENDOTHELIN-1 IN PATIENTS WITH ISCHEMIC HEART DISEASE

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Abstract Ishemia or ischemic heart disease is the leading cause of disability and premature deaths worldwide. IHD is a condition in which the heartis starved of oxygen due to a redused blood spply. Deapite a large pool of interesting candidate biomarkers, endothelin-1 (ET-1) appears to be involvedin multiple aspecis of IHD pathogenesis that include neurohormonal activation, cardiac remodeling, endothelial dysfunction, inflammation, atherosclerosis and alteration of the renal function. ET-1 production and release can be upregulated by inflammatory factors, such as IFN-γ and IL-1β, during disease

Key words: ischemic heart disease, endothelin-1, inflammation, biomarkers, neurohormanal activation.

The purpose of this work was common medications in IHD and their effect on the serum level of ET1.

Materials and methods. We examined 45 patients with IHD who are among them are 30 men and 15 women.The duration of the disease ranged from 5 year to 15 , average age 50,1±8,6 years. There were also 10 practically healthy individuals were studied. 100% this group suffered from coronary artery disease, 90% from arterial hypertension. All patients underwent general clinical examination. we used different classes of medications in patients and observed changes in ET1 in the blood.

Results. Although ET-1 can have indirect effects on cardiac muscle by modulating coronary artery tone, it can also have direct effects on the muscle and affect cardiac output. Previous studies have indicated that ET-1 plays a role in neurohormonal activation mediated by angiotensin II. It is intriguing to consider whether patients with elevated ET-1 levels may experience enhanced benefits from angiotensin-converting enzyme inhibitor (ACEI) administration. We observed an inverse correlation between ET-1 levels and changes in ACEI dose. Beta-blockers such as talinolol, atenalol, metoprolol and propranolol can also decrease the production and release of ET-1 in human endothelial cells [12]. Patients with ET-1 values above the median tended to have lower baseline doses of captopril [11]. Perindopril is the only ACEI that has been shown to improve endothelial function. Notably, we demonstrated that after 12-month therapy with perindopril, ET-1

level decreased significantly in both HFpEF and HFmrEF patients [15]. In addition, other studies have shown that captopril and lisinopril caused a significant reduction in ET-1 production [5]. Other therapies have been proposed to reduce ET-1 concentrations. Furthermore, loop diuretics like furosemide and torasemide may help improve endothelial function and potentially lead to a decrease in ET-1 levels [17]. However, no significant correlations were found between changes in ET-1 levels and the use of angiotensin receptor blockers or mineralocorticoid receptor antagonists [9]

Conclusions: IHD is the most common cause of hospitalization and is associated with a high risk of readmission and mortality. ET-1 has a crucial role in the pathogenesis of IHD. Multiple studies have demonstrated that the severity of symptoms and cardiac dysfunction in IHD is correlated with the circulating levels of ET-1, which can be used as a strong prognostic indicator for these patients. Although significant progress has been made since the discovery of ET-1, further research is necessary before implementing this biomarker in clinical practice.

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