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The Role of Aldosterone in the Development of Chronic Heart Failure and the Effectiveness of Mineralocorticoid Receptor Antagonists in its Treatment

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ABSTRACT

Aldosterone is considered a steroid hormone and its prolonged increase in concentration affects blood pressure, causes complications and metabolic changes in diseases of the vessels, heart and kidneys. It is synthesized mainly from deoxycorticosterone under the influence of mitochondrial cytochrome R 450 aldosterone synthetase in the adrenal glomerular zone.

Introduction

It was named aldosterone because it contains an aldehyde group. Aldosterone secretion is controlled by RAT and, to a lesser extent, by adenocorticotropic hormone (ACTG). By reducing the volume and velocity of blood flow in the afferent arterioles, renin secretion increases, which then causes an increase in the production of angiotensin II. Angiotensin II, in turn, increases the production of aldosterone. Aldosterone acts on receptors in the epithelial cells of the distal nephron and transfers sodium from the space of the distal tubules to its cells in exchange for potassium and hydrogen. As a result, the amount of blood circulating in the body increases. Its similar action is observed in the distal colon, rectum and sweat glands. A number of data show that aldosterone is produced directly in the vessels, endothelial and smooth muscle cells of the myocardium, kidneys, including podocytes, and in a number of other organs. Currently, mineralocorticoid receptor antagonists are recommended as part of the standard CHD treatment plan to target aldosterone produced outside the adrenal glands. Aldosterone affects non-epithelial mineralocorticoid receptors in the myocardium, renal vessels, pituitary gland and hypothalamus.

It is one of the main participants in the cardiovascular continuum and increases the expression of angiotensin-converting enzyme RNA in cardiomyocytes, causing limited production of angiotensin II

and angiotensin II type 1 receptors. Aldosterone activates the sympathetic nervous system and induces apoptosis by stimulating the free radical response. As a result, with prolonged hyperadrenosteronism, remodeling processes in the heart and other organs are enhanced, as a result of which the course of coronary artery disease and the outcome of the disease are aggravated. The effects of this hormone on a number of target organs can be summarized as follows. It stimulates collagen synthesis by activating fibroblasts in the heart and causes interstitial myocardial fibrosis. It increases the reabsorption of sodium and water in the distal tubules of the kidneys, increases the excretion of potassium and magnesium, enhances collagen synthesis by stimulating fibroblasts and causes mesangial fibrosis, increases the volume of blood circulating in the body, and causes electrolyte imbalance.

It also acts on vessels, inhibits the production of vasodilators, enhances collagen synthesis, causes endothelial proliferation and dysfunction, leading to the formation of perivascular fibrosis and thrombi. The negative effects of coronary artery disease on aldosterone have long been associated with the fact that it causes only water retention in the body and changes the electrolyte balance. But at the end of the last and the beginning of this century, it was found that it causes fibrotic processes, stimulates apoptosis in cardiomyocytes, and causes life-threatening arrhythmias.

Key words: TGF-β1, comorbid diseases, aldosterone.

Material and methods. Under supervision there were 120 patients with coronary artery disease, which were divided into three groups. Their first group consisted of 40 patients with IHD II-III FS albuminuria and one comorbid disease. The second group consisted of 40 patients with IHD II-III FS, albuminuria and two comorbid diseases, their average age was 61.8 ± 4.7 years, including 19 men and 21 women. The third group consisted of 40 patients diagnosed with CAD II-III FS albuminuria, suffering from three or more comorbid diseases. In all cases, it was found that CKD, postinfarction cardiosclerosis and hypertension were the causes of IHD. All patients received β-blockers as standard treatment for coronary artery disease, azilsartan as an angiotensin II receptor antagonist, and eplerenone 25-50 mg of the latest generation of MKRA as an antifibrotic agent. IHD in one comorbid disease, aldosterone before and after three months of treatment increased from 563.1±28.3 pg/ml to 247.4±13.4 pg/ml, i.e. by 2.27 times, with two and three or more comorbid diseases, respectively, from 699.2±31.2 pg/ml. ml to 402.2 ± 23.4 pg/ml and from 708.5 ± 45.7 to 415.1 ± 29.4 pg/ml, i.e. decreased by 0.7 times.

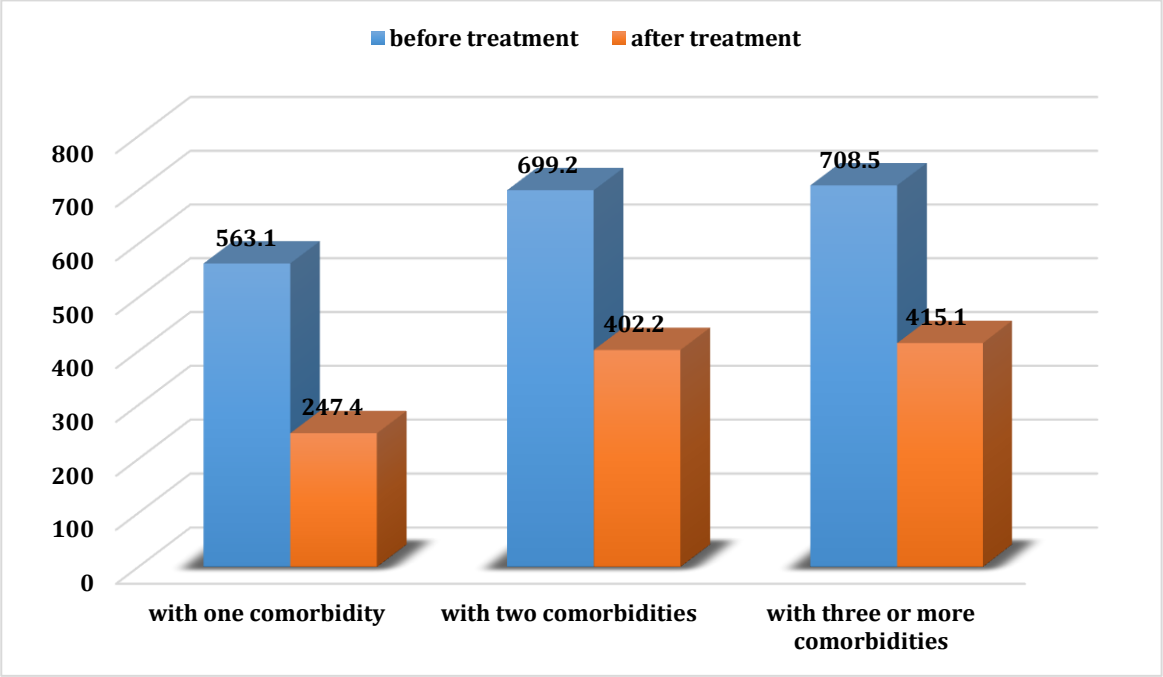

Figure 1. Aldosterone values (pg/ml) before and after treatment for chronic heart failure with various comorbidities

In all cases, after the treatment, there was a significant decrease in aldosterone in the blood serum of patients. Here, as mentioned above, this reduction factor (2.27; 1.74 and 0.7) is inextricably linked with the number of comorbid diseases, and as they increase, positive changes also decrease.

The levels of TGF-β1 before and after treatment in the observed patients also varied in proportion to the number of comorbidities. From 2390.8±98.3 pg/ml to 1092.9±78.4 pg/ml in one comorbid disease, (R<0.001) 2466.2±150.4 pg/ml in two or three or more comorbid diseases respectively ml up to 1859.8±103 pg/ml. ml and from 2735.8±190.2 to 2187.6±150.3 pg/ml decreased significantly (R<0.001) (Fig. 2).

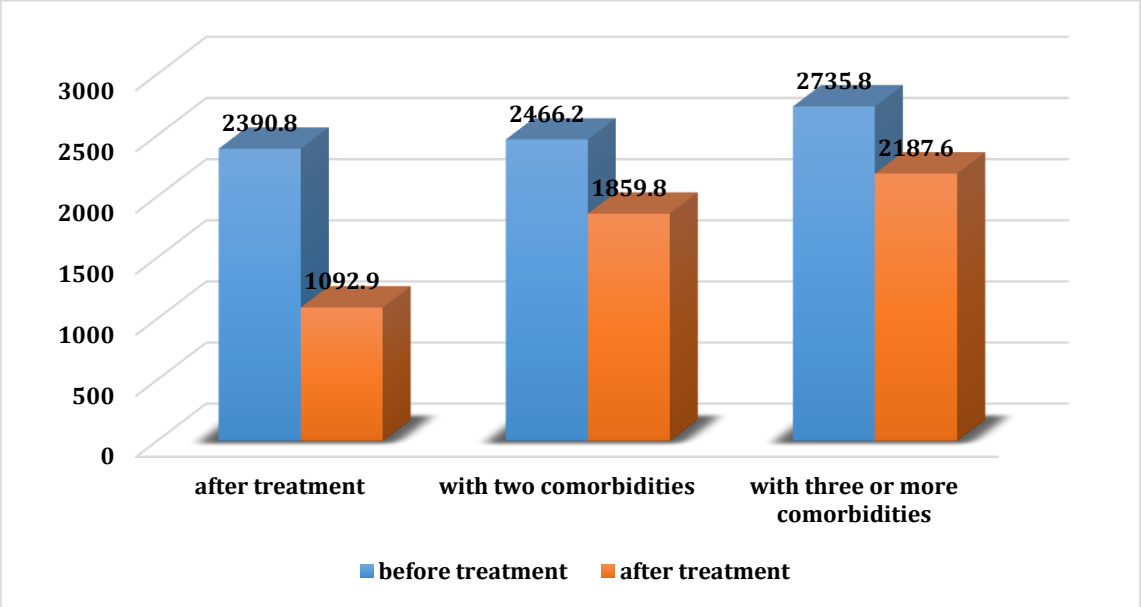

Figure-2. TGF-β1 levels (pg/ml) before and after treatment for chronic heart failure with various comorbidities

Thus, complex treatment containing eplerenone led to a significant decrease in fibrosis markers,

thereby stabilizing the process. But positive changes are more pronounced when IHD is accompanied by one comorbid disease.

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