

ENDOTHELIAL DYSFUNCTION IN CARDIORENAL SYNDROME

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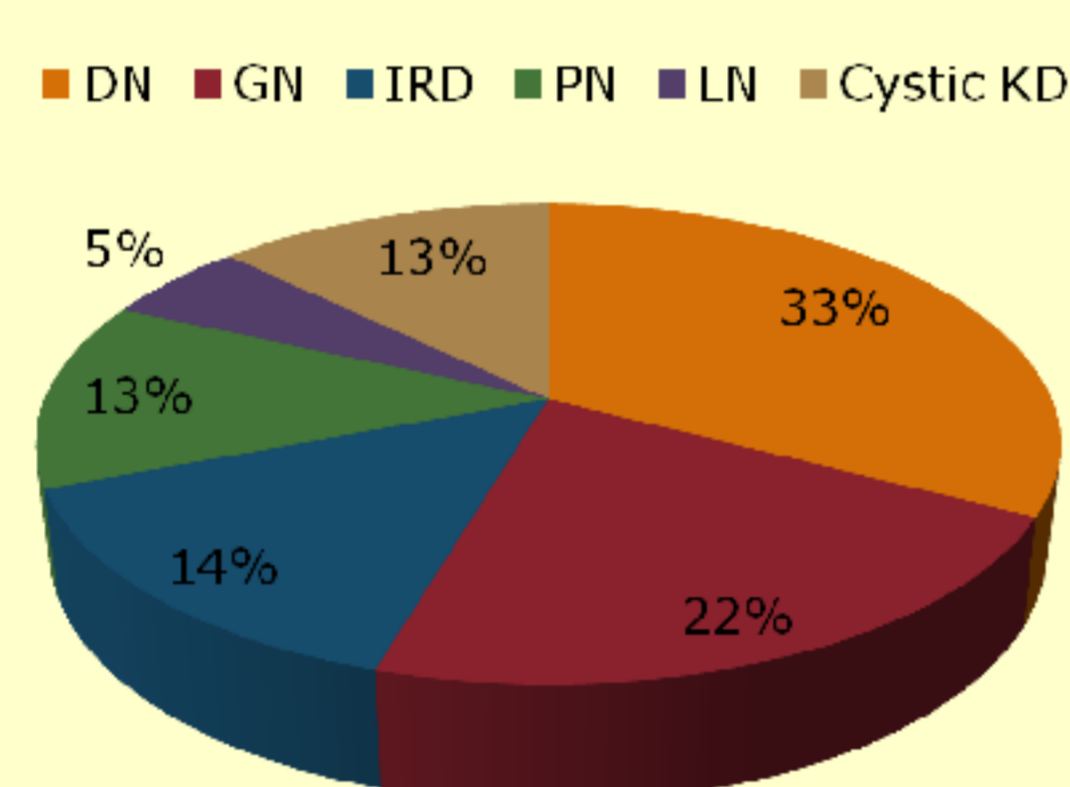
Objectives:

In cardiorenal syndrome, endothelial dysfunction (ED) contributes to the development of the pathological process and affects its progress [1, 2, 3, 4]. Increase in systemic blood pressure and disruption of intraglomerular hemodynamics damages the endothelium of glomerular vessels and increases the filtration of protein through the basement membrane, which in the early stages is manifested by microproteinuria, and subsequently – by the development of the hypertensive nephroangiosclerosis and chronic kidney disease (CKD). Endothelial dysfunction is a pathogenetic trigger mechanism for the development of nephropathy in patients with arterial hypertension. The most significant mediators in the development of nephroangiosclerosis are angiotensin II and precursor NO – abnormal dimethylarginine, contributing to the development of nitric oxide deficiency [5,6,7,8]. Hence, the restoration of glomerular endothelial function may have nephroprotective effect [9,10]. The study examines the peculiarities in the development of endothelial dysfunction in cardiorenal syndrome.

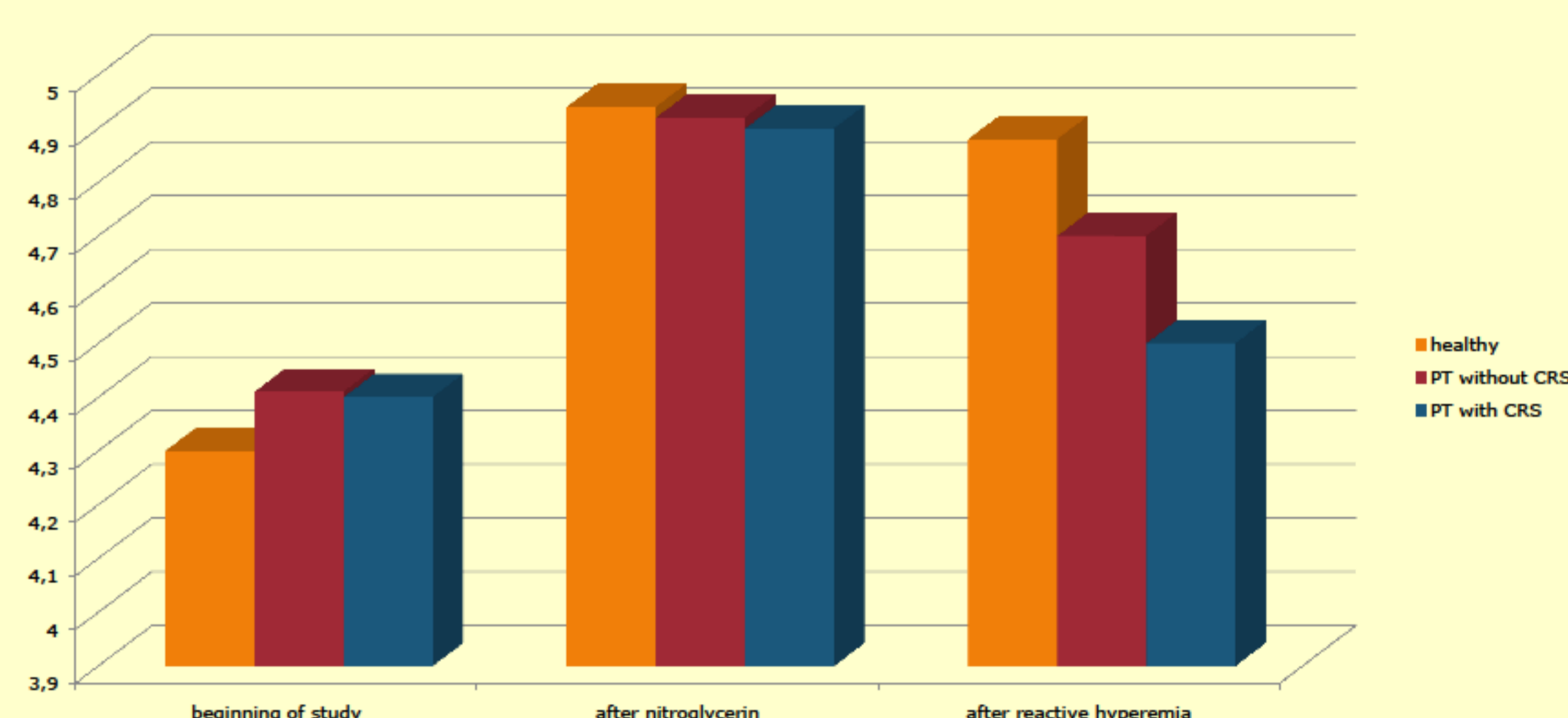
Methods:

We observed 50 patients (27 female and 21 male) aged 42-68 years (average age 54 ± 7.3 years). The cause of chronic kidney disease (CKD) in 18 cases was diabetic nephropathy, in 12 - chronic glomerulonephritis, in 8 - ischemic renal disease, in 7 - chronic pyelonephritis, in 3 - lupus nephritis, and in 2 - polycystic kidney disease (fig. 1). In 14 cases was determined the 2nd stage of CKD, in 16 - 3rd and in 18 - the 4th stage of CKD. All patients had 1-3 stages of heart failure (according to the NYHA). Functional state of endothelium was determined by the method of D.Celermajer (1992) by determining the change in brachial artery diameter after nitroglycerin and reactive hyperemia. Discovered disorders were more pronounced in patients with cardiorenal syndrome compared to those without it. With increasing stage of CKD, increases the degree of endothelial dysfunction, which can be explained by the increase of arterial hypertension, dyslipidemia, conducive to the development of atherosclerosis. Results were analyzed according to the presence of cardiorenal syndrome and the patients were divided into two groups respectively. The 1st group – patients without cardiorenal syndrome (22 people) and the 2nd group – patients with cardiorenal syndrome (28 people).

Causes of CKD (fig 1)



Changes in Brachial artery diameter depending on CRS, mm (fig 2)



Results:

Along with the clinical picture of renal and heart failure, hypertension, we observed hypoalbuminemia, hyperuricemia, dyslipidemia. The diameter of the brachial artery in patients with CKD was 4.41 ± 0.76 mm, which did not differ much from that of the control group (4.3 ± 0.78). In healthy people, after the application of nitroglycerin artery diameter increased by 10.23%, and the measurements were not significantly different from those obtained from patients (an increase by 10.21%). Along with this, endothelium-dependent vasodilation in healthy people increased by 13.4 ± 0.9%, while the increase in examined patients was only 6.7 ± 0.6%. Discovered dysfunctions were more pronounced in patients with cardiorenal syndrome compared to those without it (fig. 2). With the increase of CKD stage, we noted an increase in the degree of endothelial dysfunction, which can be explained by the increase of arterial hypertension, dyslipidemia, contributing to the development of atherosclerosis and impaired calcium-phosphorus metabolism.

Conclusions:

In CKD in the occurrence of complications in the cardiovascular system, the primary pathogenetic role belongs to the endothelial dysfunction that may occur due to metabolic disorders. On the other hand, the decrease in the level of relaxing, and the increase in the concentration of pressor agents in the body and decrease in sensitivity to vasodilator stimuli also contribute to endothelial dysfunction. Endothelial dysfunction can be considered the main pathogenetic mechanism for the development of cardiorenal syndrome.

References:

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