INFLUENCE OF LIPID METABOLISM DISORDERS ON ENDOTHELIAL FUNCTION IN CHRONIC KIDNEY DISEASE

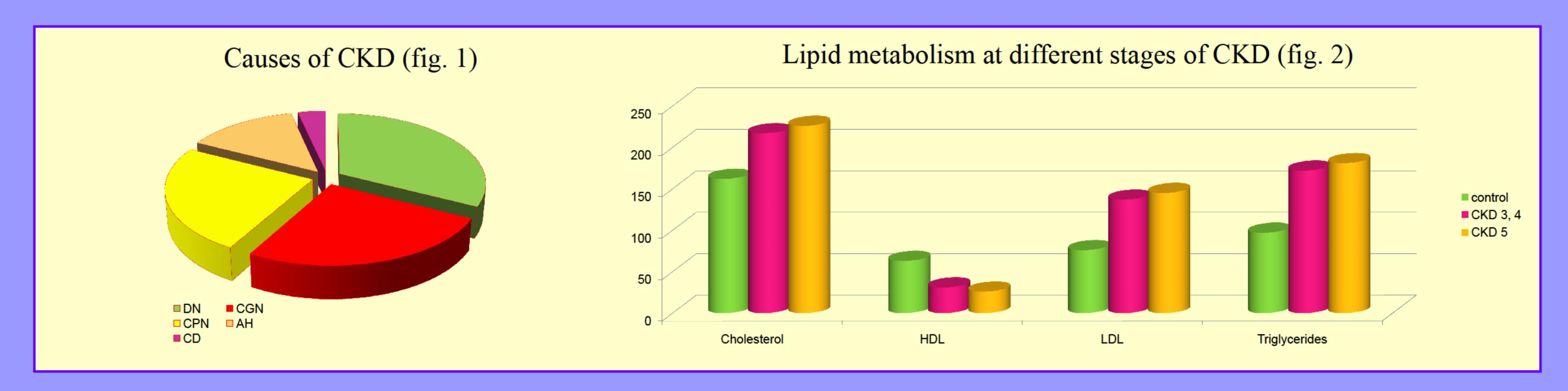
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Objectives:

In chronic kidney disease (CKD) lipid disorder[1], hypertension [2], diabetes [3], obesety [4], inflammation are risk factors for endothelial dysfunction and changes in the cardiovascular system. Lipid metabolism disorder in CKD often contributes to changes in the cardiovascular system. The observed cardiovascular complication often changes the clinical course of the disease and determines the prognosis [5, 6]. This study investigates the relationship between the level of blood lipids and the endothelial function in patients with different stages of CKD.

Methods:

The study involved 64 patients (36 women and 28 men) aged 41-69 years (mean age 55 7.2 years). The cause of CKD in 26 cases was diabetic nephropathy, in 15 cases - chronic glomerulonephritis, in 14 - chronic pyelonephritis, in 6 - lupus nephritis and in 3 - polycystic kidney disease (fig. 1). Patients were divided into 2 groups: one group consisted of 39 patients (16 patients with 3rd stage of CKD and 23 – with 4th stage of CKD) and the second group consisted of 25 patients with 5th stage of CKD. All patients were characterized by increased cholesterol level, low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and triglycerides in blood serum. Functional state of the endothelium was determined by the method of D.S. Selermajer (1992) [7] by detecting changes in brachial artery diameter after reactive hyperemia and nitroglycerin.



Results:

Indicators of total cholesterol, LDH-C, serum triglycerides in all patients with CKD was increased and the HDL-C was decreased (fig. 2). The study of endothelial function showed that the diameter of the brachial artery in patients of the first group with CKD was 4.41 0.16 mm, in the second group it was 4.11 0.13 mm which is little different from that of the control group (4.5 0.18 mm). In healthy people after nitroglycerin the diameter of the brachial artery increased (endothelium independent vasodilatation) by 10.23 0.16% and was not significantly different from the data obtained from patients (10.26 0.17% increase). Endothelium dependent vasodilatation in healthy individuals increased by 13.4 0.18%, whereas the increase in patients of the first group was 6.6 0.13% and in the second group it was 5.9 0.12%. The revealed disorders were more pronounced in patients with high cholesterol, LDL-C and serum triglycerides and they were lower in patients with high HDL-C. Increasing severity of CKD was accompanied by more pronounced impairment of the endothelial dysfunction which may be due to the lipid metabolism disorder.

Conclusions:

We can assume that disorders in metabolism of cholesterol, triglycerides and LDL-C are the factors contributing to the development of endothelial dysfunction in patients with CKD, whereas HDL-C has a protective effect on the endothelium and kidney. Timely correction of disorders of lipid metabolism will not only contribute to the correction of endothelial dysfunction but also slow down the growth of CKD.

References:

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