

**RELATIONSHIP BETWEEN DIABETIC NEPHROPATHY AND CARDIAC DISORDERS  
IN PATIENTS WITH TYPE 2 DIABETES****Negmatova.G.Sh***Head of Endocrinology Department:**Scientific leader: assistant.***Salimova D.E****Bakhranov Akmaljon Alisherovich****Rahimov is the son of Sardar Erkin****Usarov is the son of Shakhboz Bektash***Students of Samarkand State Medical University*

**Abstract:** *Over time, high blood sugar can damage blood vessels and the nerves that control your heart. People with diabetes are also more likely to have other conditions that raise the risk for heart disease: High blood pressure increases the force of blood through your arteries and can damage artery walls.*

**Keywords:** *diabetic nephropathy, diabetes, kidney failure, microalbuminuria indicates, cardiovascular pathologies.*

The purpose of this study is to study the changes in the heart at different stages of diabetic nephropathy among patients with type 2 diabetes. Inspection materials and methods: During the inspection 30-6% of patients with type 2 diabetes suffer from kidney damage [1]. 5-10% of diabetic nephropathy ends with the terminal stage of kidney failure [5], the death rate from diabetes is 1.5-3% [3]. Diabetic nephropathy increases the mortality rate up to 5-8 times compared to people without diabetes and diabetic nephropathy [1]. Microalbuminuria is the first indicator of kidney damage in diabetes. Microalbuminuria indicates a high risk of developing pathologies not only in the kidneys, but also in the cardiovascular system [6]. Correlation between microalbuminuria and mortality the mechanism of development of heart damage in diabetic nephropathy is very complex and not fully understood. In most cases, all stages of diabetic nephropathy are complicated by cardiovascular pathologies, and most patients die of myocardial infarction, stroke, and life-threatening arrhythmias [1,2,4].

24 patients (16 men and 8 women) with type 2 diabetes mellitus, average age of  $56.85 \pm 2.93$  years, were treated in the General Endocrinology Department of the Samarkand branch of the Republican Center for Scientific and Applied Medicine of Specialized Endocrinology. patients were studied. 5 practically healthy people without diabetes with an average age of  $42.01 \pm 2.21$  were recognized and they formed the control group. Patients were divided into 3 groups. Group 1 included 8 patients with an average age of  $52.9 \pm 2.25$ , duration of diabetes  $7.86 \pm 0.87$ . Normoalbuminuria (NAU) was observed in them. YUIK to 3 patients from them. Stable tension angina FS I, II stage

was observed (Table 1). 8 patients were selected for the 2nd group, their average age was  $56.67 \pm 2.82$ , the duration of diabetes was  $10.18 \pm 0.91$  years, and they were diagnosed with microalbuminuria (MAU). YUIK to 5 patients from them. Stable tension angina FS I, III stage was observed (Table 1). 8 patients were selected for the 3rd group, their average age was  $59.9 \pm 2.0$ , the duration of diabetes was  $10.40 \pm 1.3$  years, proteinuria (PU) was detected in them. YUIK to 6 patients from them. Stable tension.

Patients with urinary tract infection not checked. Patients in all groups were compared according to age, body mass index, carbohydrate metabolism status. During the examination, the patients did not complain of heart palpitations or pain in the heart. Characteristics of the patient's clinical laboratory parameters are shown in Tables 1 and 2. Blood sugar levels.

It was determined by the method of K.H.Winterholter. The blood pressure of all patients was measured by the Korotkov method. The level of microalbuminuria was determined using "Urobel" test sticks. Diabetic nephropathy stage C.E. It was put according to Mogenson's classification. Glomerular filtration rate determined by CKD-EPI CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration): Normal 80-120 ml/min. Formula SKD-EPI KFT ( $\text{ml/min/1.73 m}^2$ ) =  $141 \times [\text{min plasma creatinine (mg/dl)/k or l}]^a \times [\text{max. plasma creatinine (mg/dl) / k or l}]^{-1.209} \times 0.993^{\text{age}} \times 1.018$  (for women)  $\times 1.159$  (for representatives of black race)  $K = 0.7$  for women and  $0.9$  for men,  $a = -0.329$  for women and  $-0.411$  for men  $\text{Creatinine } (\mu\text{mol/l}) = \text{creatinine (mg/dL)} \times 88.4$ . All patients underwent an echocardiographic examination using a multi-channel echocardiograph. The examination results were statistically analyzed using the EXCEL program, which includes variation statistical methods.

**Analysis of results and results:** General clinical indicators, indicators of carbohydrate metabolism, kidney and heart function were comparatively studied in all patients included in this investigation. YUIK was observed in group 1 - 25%, in group 2 - 75%, in group 3 - 95%. The mechanism of development of diabetic nephropathy is very complex and has not been studied to the end. In most cases, all stages of diabetic nephropathy are associated with cardiovascular pathologies. It is known that most diabetic patients do not reach the uremic stage of diabetic nephropathy and die from myocardial infarction, stroke, and life-threatening arrhythmias [1,2,4]. In patients with normoalbuminuria (group 1), the thickness of the interventricular septum was  $10.8 \pm 0.62$  mm, and the thickness of the posterior wall of the left ventricle was  $10.1 \pm 0.30$  mm. In patients with microalbuminuria (group 2), the interventricular septum was average thickness of the barrier (QTQ)  $10.73 \pm 0.93$  mm; the thickness of the back wall of the left ventricle (ChQODQ) is  $10.53 \pm 1$ , established 63 mm. In patients with proteinuria (group 3), the thickness of the ventricular septal barrier (SVS) was  $10.77 \pm 0.50$  mm, the thickness of the posterior wall of the left ventricle was  $11.38 \pm 0.97$  mm. Aggravation of diabetic nephropathy led to thickening of the walls of the left ventricle. Last diastolic blood pressure (ODO') in the 1st group ( $46.4 \pm 3.2$ ), in the second ( $55.78 \pm 0.85$ ) group, in

the third group ( $59.77 \pm 4.33$ ) compared to the control group ( $42, 07 \pm 2.86$ ) was relatively high. The last systolic size (OSO') was as follows: in the 1st group ( $30.6 \pm 2.3$ ), in the 2nd in group ( $41.57 \pm 8.48$ ) and in group 3 ( $32.73 \pm 1.92$ ), in the control group ( $28.91 \pm 1.89$ ). The progression of diabetic nephropathy led to hypertrophy of the left ventricle. The thickness of the wall of the left ventricle of the heart in patients with diabetic nephropathy compared to patients without diabetic nephropathy is reported in many literatures [2,3,6].

Diabetic nephropathy (PHD) decreased with increasing severity. The maximum speed of active charging (A) was higher than the maximum speed of fast charging (E) in groups 1, 2, 3 (Table 3). This indicates a violation of the diastolic function of the left ventricle.

**Summary:**

All information was thoroughly analyzed and it was observed that there is a pathogenetic relationship between kidney and heart damage in diabetes.

It was found that there is a correlation between the progression of diabetic nephropathy and the development of macroangiopathy, in particular, YUIK. The frequency of YUIK was 25% in patients with normoalbuminuria, 70% in patients with microalbuminuria, and 95% in patients with proteinuria.

It was found that there is a correlation between alburinuria and hypertrophy of the left ventricle of the heart. Left ventricular hypertrophy was observed in patients with normoalbuminuria - 8.91%, in patients with microalbuminuria - 12.82%, and in patients with proteinuria - 19.33% compared to the control group.

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