



Disorders Function of the Heart and Kidney in Diabetes Mellitus

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Abstract: This article presents data on the prevalence of various types of heart remodeling in patients with type 2 diabetes with DN. It was found that the severity of structural and functional changes in the heart depends on the stage of diabetic nephropathy. New data are presented that establish a clear relationship between the indicators characterizing the structural and functional state of the heart and kidneys in patients with type 2 diabetes with DN. New facts have been obtained indicating that the basis of combined renocardial disorders in patients with type 2 diabetes is impaired vascular endothelial function.

Key words: diabetes mellitus, diabetic nephropathy, microalbuminuria, glomerular filtration rate, heart remodeling, endothelial dysfunction, cardiorenal syndrome.

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Background: Diabetes mellitus (DM) is a widespread disease. The number of people with diabetes in the world is 140 million people and, according to the World Health Organization, by 2025 this population will increase to 300 million [1,22]. Chronic kidney disease (CKD) affects 10-16% of the adult population in Asia, the USA, Australia and Europe and is a global health problem [3,5,17]. It increases the risk of all-cause mortality and cardiovascular disease, as well as the possibility of progression to end-stage renal disease [2,21,24]. According to the 2002 KDOQI guidelines, CKD is defined as kidney damage measured by albumin loss and decreased glomerular filtration rate (GFR), which is the basis for its staging [6,18,23].

The earliest marker of kidney damage in diabetes is microalbuminuria (MAU), the presence of which is closely associated with further progression of diabetic nephropathy. With the development of proteinuria, DN progresses to chronic renal failure. 5-10% of cases of DN end in end-stage renal failure [4,10], which in the structure of mortality in patients with type 2 diabetes is 1.5-3% [9,20]. All stages of DN are associated with cardiovascular pathology [7,12,14]. The appearance of DN leads to a 5-8-fold increase in mortality in these patients compared to the general population [13,17]. Diseases of the cardiovascular system to this day continue to be the leading cause of death in patients with type 2 diabetes who do not survive to end-stage renal failure [8,11]

Recent studies have described the relationship of GFR and albuminuria with clinical outcome in subjects in the general population, which makes it possible to present the threshold values of GFR

(<60 ml/min/1.73 m²) and MAU, which are risk factors that increase mortality [15,19,22]. Data from epidemiological studies examining more than 65 thousand patients support the view of MAU as a “biomarker” of adverse outcome even in patients with normal renal function [7,13]. This allows us to conclude that the concept of MAU should be included in the list of biomarkers indicating both the risk of development and progression of renal dysfunction.

Myocardial remodeling in CKD develops due to the influence of a number of factors: pressure and volume overload, anemia, exposure to a number of pressor hormones [6,9,16]. A number of indicators that determine the degree of renal dysfunction (GFR, creatinine) may have a role in hemodynamic disturbances and the progression of heart failure. Based on the above, we studied the relationship between EchoCG indicators of left ventricular (LV) remodeling, the level of MAU and the degree of renal dysfunction in patients with CRS types 2 and 4. The underlying mechanisms that determine the fact of combined damage to the heart and kidneys in type 2 diabetes are not fully understood.

Purpose of the study:

To study the features of cardiorenal syndrome in diabetic nephropathy in patients with type 2 diabetes mellitus.

Material and methods:

We examined 60 patients (32 men and 28 women) with type 2 diabetes with DN who were undergoing inpatient treatment at the Republican Scientific and Practical Center of Nephrology on the basis of the III-clinic of TMA. The average age of patients was 58.0±0.4 years, duration of type 2 diabetes was 10.6±0.3 years. Patients (n=60) were divided into 2 groups based on the duration of diabetes and the level of MAU: group I (n=25) MAU was noted within 12.8±4.65 µg/ml, in group II (n=35) it was 22.4±4.64 µg/ml. The study patients underwent general clinical and biochemical tests, and B-mode echocardiography.

An echocardiographic study was performed on a SONOSCAPE S20 ultrasound machine using a 3.5 MHz cardiac sensor in modes according to the generally accepted Simson method.

Glomerular filtration rate (GFR) was determined using the CKD-EPI formula (ml/min/1.73 m²)

Results and discussion:

In group I of patients, the level of creatinine and GFR were 76.4 ± 10.8 µmol/l and 79.2 ± 15.8 ml/min/1.73 m², and in group II - 78.2 ± 11.6 µmol/l and 73.4 ± 17.5 ml/min/1.73 m², respectively. Therefore, the higher the MAU level, the higher the creatinine and the lower the GFR, i.e. initial manifestations of renal dysfunction are noted (Table 1).

Table 1. Characteristics and paraclinical data of patients with CRS

Parameters	Group I (n=25)	Group II (n=35)	P
Age	58.0 ± 0.4	58.6 ± 0.9	
BMI (кг/м ²)	29.9 ± 4.6*	26.8 ± 3.5*	p<0.05
MBP (mm/m.c)	160/100	150/90	
Urea (µmol/l)	6.8 ± 1.4	5.6 ± 1.9	
Creatinine (µmol/l)	92.3 ± 11.8*	94.2 ± 12.6*	p<0.05
GFR (ml/min/1,73m ²)	74.3 ± 17.5	77.8 ± 16.8	

In patients of group I, EDR, TZS, TVR, EF and LV IMM were observed within the range of 5.18±0.33cm, 1.16±0.08cm, 0.47±0.03M, 9.5±2.9%, 135.9±24.1g/m², and in group II - 5.29 ± 0.48cm, 1.21±0.09cm, 0.48±0.04, 46.2±4.2%, 156.7±29.1g/m², respectively (Table 2).

Table 2. Structural and functional parameters of the myocardium in patients with CRS

Parameters	Group I (n=25)	Group II (n=35)
LV EDR (N=3.8-5.6 cm)	5.18 ± 0.33	5.29 ± 0.48
TIS (N=0.7-1.1 cm)	1.29 ± 0.11	1.21 ± 0.11
LV TSV (N=0.8-1.1 cm)	1.16 ± 0.08	1.21 ± 0.09
LV TVR (N< 0.45)	0.47 ± 0.03	0.48 ± 0.04
LV IMM (g/m ²)	135.9 ± 24.1	156.7 ± 29.1
LVEF (N- 53% >)	56.2 ± 3.7	46.2 ± 4.2

Comparative characteristics of the main structural and functional indicators of the myocardium according to echocardiography studies in patients with chronic CRS reveals in patients of group II (MAU-24.8 ± 5.03 µg/ml) a greater LV EDR (5.29 ± 0.48 cm) and a more pronounced decrease in EF LV (52.2 ± 4.2%). The degree of LVH is also significant (LV TSV -1.21 ± 0.09 cm) in patients of group II (1.16 ± 0.08 cm) than the same indicator in patients of group I (Table 3). Correlation analysis revealed a direct proportional relationship between the level of MAU and LV TVR, LV EDR and LV IMM, respectively r=0.2, r=0.3, r=0.3 (p<0.05) and an inverse relationship between LVEF and MAU (r=(-0.44) moderate correlation p<0.05).

Table 3. Renal function and indicators of structural and functional restructuring of the LV in patients with CRS.

Parameters	Group I (n=25)	Group II (n=35)	P
AGE	58.0 ± 0.4	58.6 ± 0.9	
MAU (mkg/ml)	14.7 ± 4.45	24.8 ± 5.03*	p<0.05
LVEDR (3.8-5.6 cm)	5.18 ± 0.33*	5.29 ± 0.48*	p<0.05
LVOTS	0,47±0,03	0,48±0,04	
LV TPW (0.8-1.1 cm)	1.16 ± 0.08**	1.21 ± 0.09**	p<0.01
TIS (0.7-1.1 cm)	1.26 ± 0.10*	1.31 ± 0.11*	p<0.05
LVEF (53% >)	49.5 ± 2.9**	46.2 ± 4.2**	p<0.01
LV IMM	135.9 ± 24.1	156.7 ± 29.1*	p<0.05
GFR (ml/min/1,73m ²)	89.4 ± 17.3	83.7 ± 19.3	
Creatinine (µmol/l)	96.1 ± 18.8	98.3± 17.3	

Higher or threshold levels of MAU are accompanied by increased creatinine and decreased GFR, are associated with increased LV myocardial mass, and are associated with preclinical impairment of LV systolic function.

The interaction we identified between increased blood pressure, increased mass of the LV myocardium, changes in its geometry and dysfunction increases the role of MAU as an early and reliable marker of cardiac, preclinical, structural and functional changes in the myocardium.

Data from a number of studies indicate levels of GFR and albuminuria used to define and stage CKD, taking into account complications, risk and assessing the effectiveness of management of these patients. Our results allow us to note the level of MAU (22.4 ± 4.64 µg/ml), at which a high probability of impaired LV contractile function is determined, which will make it possible to earlier predict and prevent the progression of CKD, and therefore CRS.

Conclusions:

Thus, there are common ethical factors and pathogenetic mechanisms of damage to the heart and kidneys in type 2 diabetes, parallelism in the stages of damage to these target organs.

The identified mechanisms of myocardial remodeling in chronic cattle can be the basis for early diagnosis of circulatory failure, as well as targeted and pathogenetically justified pharmacotherapy in this category of patients.

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