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CLINICAL AND LABORATORY FEATURES OF STAGES OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Annotation: A growing body of evidence indicates an increasing prevalence of the normoalbuminuric variant of chronic kidney disease (NA-CKD) in patients with type 2 diabetes mellitus (T2DM), while the proportion of the albuminuric variant is decreasing. Diabetes mellitus (DM) is the leading cause of chronic kidney disease (CKD) in countries with industrial and post-industrial economies. Although the prevalence of CKD among adults with diabetes remains consistently high, there is a trend towards changes in its natural history. This trend is a decrease in the prevalence of increased urinary albumin excretion (EAU) and an increase in the number of patients who develop a decrease in glomerular filtration rate (GFR) without a previous or concomitant increase in albuminuria [1,2,3].

Keywords: diabetic nephropathy; chronic kidney disease; diabetes mellitus type 2; albumin to creatinine ratio in urine.

Relevance. A growing body of evidence indicates an increasing prevalence of the normoalbuminuric variant of chronic kidney disease (NA-CKD) in patients with type 2 diabetes mellitus (T2DM), while the proportion of the albuminuric variant is decreasing. Diabetes mellitus (DM) is the leading cause of chronic kidney disease (CKD) in countries with industrial and post-industrial economies. Although the prevalence of CKD among adults with diabetes remains consistently high, there is a trend towards changes in its natural history. This trend is a decrease in the prevalence of increased urinary albumin excretion (EAU) and an increase in the number of patients who develop a decrease in glomerular filtration rate (GFR) without a previous or concomitant increase in albuminuria [1,2,3]. This trend is most pronounced in type 2 diabetes (T2DM). According to generalized data, the proportion of normoalbuminuric CKD (NA-CKD) among patients with T2DM with GFR <60 ml/min/1.73 m2 varies from 43 to 71% [5,6]. The increase in the prevalence of AN-CKD is associated with the widespread use of blockers of the renin-angiotensin system, increased effectiveness of hypoglycemic, antihypertensive and lipid-lowering therapy, and smoking cessation. The growing popularity of glucagon-like peptide (GLP-1) agonists, dipeptidyl peptidase type 4 (DPP-4) and glucose-sodium cotransporter (SGLT-2) inhibitors, which have a glucose-independent antialbuminuric effect, is also considered as one of the reasons for the decrease in the prevalence of albuminuria among patients with T2DM [7 ,8,9]. Risk factors and mechanisms for the development of different variants of the course of CKD in T2DM are currently poorly studied. It has been shown that the CKD variant with increased albuminuria is associated with typical morphological changes in the renal glomeruli, while in the normoalbuminuric variant, changes in the interstitium and arteriolosclerosis may prevail over the signs of glomerulopathy [10,11,12]. In

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this regard, urgent tasks are to search and evaluate the diagnostic value of biomarkers of glomerular and tubulointerstitial changes in different variants of the course of CKD. Patients with chronic kidney disease (CKD) and type 2 diabetes mellitus (T2DM) are at high risk of progression to CKD and cardiovascular disease (CVD) [14,15]. The prevalence of CKD in patients with T2DM is currently about 40% and continues to rise. The growing number of people with CKD and T2DM will ultimately have a significant impact on the use of health care resources and the costs of treating people with T2DM[16,17]. Management of CKD in patients with T2DM is aimed at preserving renal function to reduce the risk of end-stage renal disease, cardiovascular complications and mortality. Evidence-based treatment guidelines for patients with CKD and T2DM are provided by several international and national organizations and recommend several lifestyle and pharmacological approaches, helping to prevent or delay the progression of CKD in patients with T2DM[18,19]. Recommendations include routinely screening patients with T2DM for CKD using urinary albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (at least annually and at least twice annually if UACR >300 mg/g) [28,29,30]. In addition, vascular complications should be assessed in conjunction with interventions aimed at improving glycemic control and lipid levels, maintaining healthy body weight, and optimizing blood pressure[20,21]. Drugs that slow the progression of CKD include renin-angiotensin system inhibitors, sodium-glucose cotransporter-2, glucagon-like peptide 1 receptor agonists and, more recently, selective non-steroidal mineralocorticoid receptor antagonists[22,23]. This review highlights current challenges faced by primary care providers in managing CKD in patients with T2DM, including managing comorbidities, implementing new treatment options, and implementing personalized care[26]. Achieving consensus on the optimal treatment of this disease is critical to ensure consistent and appropriate care for all patients. Strategies to improve outcomes should also include the use of clear referral criteria, the use of a multidisciplinary approach, and patient education[24,25].

Purpose of the study. To determine clinical and laboratory parameters associated with various types of CKD in patients with T2DM.

Research methods. The observational, single-center, cross-sectional study included 360 patients with T2DM with a duration of at least 10 years. Patients with urine albumin/creatinine ratio (ACR) <3 mg/mmol and estimated glomerular filtration rate (GFR) >60 ml/min/1.73 m^2entered the T2DM group without CKD (n=89). Patients with ACM <3 mg/mmol and GFR <60 ml/min/1.73 m^2formed the AN-CKD group (n=111). Patients with GFR ≥60 ml/min/1.73 $m^2$and AKM ≥3 mg/mmol were classified into the group of albuminuria with preserved renal function (A-CKD–, n=87). Patients with GFR <60 ml/min/1.73 $m^2$and AKM ≥3 mg/mmol formed the albuminuric CKD group (A-CKD+, n=73). Urinary excretion of podocyte slit diaphragm proteins (nephrin, podocin) and WAP-type core domain stabilized by four disulfide bonds-2 (WFDC-2), considered a marker of tubulointerstitial fibrosis, was determined using enzyme-linked immunosorbent assay (ELISA) and compared with healthy controls (n=20).

RESULTS. The presence of AN-CKD was associated with age ≥65 years (p=0.0001), duration of T2DM ≥15 years (p=0.0009), female gender (p=0.04), diuretic use (p=0.0005). Risk factors for CKD-A were male gender (p=0.01), smoking (p=0.01), waist-hip index >1 (p=0.01) and HbA1c concentration >8.0% (p=0.005). Duration of T2DM ≥15 years (p=0.01) and use of dihydropyridine calcium antagonists (p=0.01) were associated with A-CKD+. In T2DM, the excretion of nephrin and podocin increased (all p<0.001 compared with controls), especially in patients with increased albuminuria (p<0.01 compared with the NA-CKD group). Excretion of WFDC-2 increased in men of all “diabetic” groups (p<0.05 compared with control) and in women with reduced GFR (p<0.05 compared with control or AN-CKD).

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CONCLUSION. Variants of CKD in T2DM are heterogeneous in clinical and laboratory characteristics. Changes in nephrin and podocin excretion indicate an association of albuminuric variants with podocyte damage. A decrease in GFR in women with T2DM is associated with an increase in urinary excretion of the tubulointerstitial fibrosis marker WFDC-2.

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