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These Risk Factors for Terminal Renal Failure in Patients Suffering From Diabetes Were Studied

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Abstract: The article examines these risk factors for end-stage renal failure in patients with diabetes mellitus. The purpose of the study was Early prediction of renal failure in patients with diabetes mellitus. Assess kidney function at a stage when it is not yet affected by the pathological process. The materials and methods of the study included 71 patients receiving hemodialysis with an approved main diagnosis of diabetes mellitus, the complications of which were end-stage renal failure. In all studied patients, urea (diamide acetic acid), creatinine, indican and cystatin C in the blood were examined. In all patients, the primary medical documentation of the outpatient card was analyzed, and concomitant diseases were also studied. Taking into account concomitant diseases, patients were divided into groups. There were 16 patients in the first group, 17 in the second group, 21 patients in the third group, 6 patients in the fourth group, and 11 patients with other diseases were included. In patients before the development of end-stage renal failure, the contents of urea and creatinine were studied. When studying the outpatient records of patients before the development of end-stage renal failure, it was revealed that in patients from 11 years of age after diabetes, there was an increase in urea content from 9.2 to 12.1 mmol/l and creatinine from 0.189-0.237 mmol/l in the blood of sick.

Keywords: Diabetes, end-stage renal failure, urea, creatinine, indican, cystatin.

Relevance. Diabetes mellitus is one of the most common endocrine diseases and is an acute medical and social problem. Diabetes mellitus is prevalent in all countries of the world. The prevalence of this disease is growing every year, as is the number of patients with severe vascular complications and chronic renal failure, leading to early disability and mortality. The greatest threat to the life of patients with diabetes mellitus is diabetic kidney damage - diabetic nephropathy, which develops within 10-15 years after the onset of diabetes mellitus and extremely quickly leads to the development of chronic renal failure [1.2.3.4.5.6].

Literature data [4.7.8.9.10] indicate that the prevalence of diabetes mellitus in developed countries is 4-5%, and in developing countries it reaches 10-15%. Every year, the number of new cases increases by 5-7%, and every 10-15 years, the number of people with diabetes doubles [4.9]. WHO experts have predicted that by 2030, every 15-20th inhabitant of the planet will have diabetes. The greatest threat to the life of patients is diabetic kidney damage, which quickly leads to the development of chronic renal failure (Diabetic Nephropathy, 1996; Balabolkin M.I., 2000; Sivous G.I. et al.).

The danger of diabetic nephropathy is that it develops slowly and gradually, remaining unnoticed for a long time. Only at the advanced (often terminal) stage of kidney damage do patients begin to complain of symptoms associated with nitrogenous waste intoxication, but at this stage it is not always possible to help the patient radically. Among all patients undergoing hemodialysis, 30% have diabetes mellitus [11.12.13]. This raises the important task of timely diagnosis of diabetic nephropathy and the need for adequate pathogenetic therapy for this complication.

The origin of diabetes mellitus mainly depends on the development of a number of complications, including diabetic nephropathy. Most authors associate the pathogenesis of kidney damage in diabetes mellitus with insulin deficiency and neurohumoral shifts against its background, which lead to hemodynamic dysfunction (hyperperfusion and hyperfiltration of the glomeruli) and metabolic stress on the renal parenchyma (Locatelli F. et al., 2003).

In recent years, it has become known that after the manifestation of diabetes, structural and functional changes are detected in the kidneys that cannot be detected by routine diagnostic methods. At the same time, it is precisely in the early stages of nephropathy that pathogenetic treatment methods (angiotensin-converting enzyme inhibitors, low-protein diet, etc.), whereas the appearance of proteinuria and high blood pressure—the first clinical signs of diabetic nephropathy—indicates pronounced and irreversible changes in the kidneys [4.9.14].

Low rates of diagnosis of diabetic nephropathy in the preclinical stage are associated with insufficient knowledge of the pathogenesis of angiopathy in diabetes mellitus and nephropathy. To date, little is known about how the excretion and reabsorption of sodium and potassium ions, urea nitrogen, and creatinine are disrupted [15.16.17].

According to the literature (Locatelli F. et al., 2003), diabetes mellitus is one of the most common diseases. The outcome of this pathology mainly depends on the development of a number of complications, including diabetic nephropathy. Most authors associate the pathogenesis of kidney damage in diabetes mellitus with insulin deficiency and neurohumoral shifts against its background, which lead to hemodynamic dysfunction (hyperperfusion and glomerular hyperfiltration) and metabolic stress on the renal parenchyma.

The incidence of acute renal failure (ARF) varies widely, from 5 to 36.6%, and its development is associated with high mortality (60–90%). The results of cohort studies involving 17,391 patients with COVID-19 showed that the total prevalence of pre-existing chronic kidney disease (CKD) and end-stage renal failure (ESRF) was 5.2% (2.8–8.1) and 2.3% (1.8–2.8), respectively [18.19].

Urinary syndrome is considered a possible damage to podocytes, renal tubules, and interstitium during SARS CoV 2 infection. This is justified by the detection of viral RNA in kidney tissue in 60% of cases and in urine. In addition, in severe cases of SARS CoV 2, collapsing focal segmental glomerulosclerosis develops in combination with acute tubulonecrosis [20.21.22]. This allows us to conclude that there is a correlation between the extra-respiratory and, in particular, renal tropism of SARS CoV 2 and the severity of COVID 19. Kidney damage in COVID-19 is caused by the nephrotropic effect of the virus and its cytopathic effect on the tubular epithelium in parallel with the pulmonary epithelium [17.20.22.23.24].

In severe and extremely severe cases of COVID-19, signs of proximal tubular dysfunction were detected, which at the structural level corresponded to acute tubulonecrosis with loss of the brush border and a significant decrease in megalin expression in it. Transmission electron microscopy identified particles resembling coronavirus in the endoplasmic reticulum of the proximal tubules, which may indicate direct parenchymal infection of the tubule epithelium and podocytes [18.19.22.24].

Research objective: Early prediction of renal failure in patients with diabetes mellitus and its clinical and prognostic significance. To assess kidney function at a stage when it is not yet affected by the pathological process. All this makes it important not only to develop methods for early diagnosis and prevention, but also to achieve pathogenetically justified, stable compensation for diabetes itself and all accompanying vascular complications, which can lead to a reduction in the risk of disability and mortality in patients with this pathology and an improvement in the quality of life of patients with diabetes.

Materials and methods: The study was conducted at the Bukhara Multidisciplinary Center in the nephrology and hemodialysis department. The study involved 71 patients undergoing hemodialysis with a confirmed primary diagnosis of diabetes mellitus. Of the total number of patients, 33 were women and 38 were men aged 41 to 58 years. The complications of the primary pathology were confirmed as end-stage renal failure. In addition to general clinical tests, all patients underwent tests for urea (acetic acid diamide), creatinine, indicin cystatin C in the blood before and after the development of end-stage renal failure. All patients underwent an analysis of their primary medical records and outpatient cards, and their comorbidities, harmful habits, dietary factors, etc. were studied. In all groups of patients, the content of indicin and cystatin C before the development of ESRF was not determined.

The outpatient records of all patients with a history of diabetes mellitus and concomitant pathologies were analyzed. The first group included 16 patients with diabetes mellitus without concomitant pathology. The second group included 17 patients with diabetes mellitus and concomitant anemia, the third group included 21 patients with diabetes mellitus and obesity, the fourth group included 6 patients with diabetes mellitus and coronavirus infection, and 11 patients with diabetes mellitus and other concomitant diseases.

Results and discussion: The studies showed that in all patients after the diagnosis of diabetes mellitus (DM) between the ages of 11 and 14, the concentration of urea, creatinine, indican, and cystatin C increased.

Thus, a study of the outpatient records of patients with DM showed that, starting from 11 to 14 years after the onset of the disease, urea levels began to rise from 9.4 to 11.9 mmol/L before admission to the hospital. With the development of end-stage renal failure, the blood urea levels of patients ranged from 19.9 to 57.1 mmol/L. The increase in blood urea levels prior to the development of renal failure is associated with the destruction of the basement membrane of the capillaries and their endothelium supplying blood to the kidneys. Hyalinosis and sclerotic changes in the capillary wall contribute to a decrease in capillary volume and even plasma extravasation from the capillary wall, which ultimately leads to fibrinoid necrosis and the development of renal capillary thrombosis. All of this has led to the development of renal failure.

The blood creatinine levels in patients with diabetes mellitus prior to the development of end-stage renal failure ranged from 0.189 to 0.204 mmol/L (normal range 0.088-0.176 (1.3)), When examining patients after the development of ESRD, this indicator exceeded the norm many times over, ranging from 0.867 to 2.3 mmol/L. Elevated creatinine levels indicate a significant impairment of renal excretory function as a result of damage to the renal parenchyma.

In patients with diabetes mellitus and the development of acute renal failure, we observed an increase in the content of indican and cystatin C in the blood from 3.9 to 4.6 μmol/L and from 1.6 to 3.1 mg/L (normal range 3.18 and 0.52–0.98), respectively. Elevated indican levels indicate a significant impairment of renal excretory function, which develops as a result of damage to the renal parenchyma.

In a study of patients with diabetes mellitus and anemia, urea levels prior to hospitalization ranged from 9.7 to 12.3 mmol/L, which is associated with renal tissue ischemia. With the development of end-stage renal failure, it increased from 26.7 mmol/L to 58.8 mmol/L.

Elevated blood urea levels prior to the development of renal failure are associated with ischemia and destruction of the capillary basement membrane and its endothelium.

A study of the medical history of these patients revealed that the blood creatinine content in patients with diabetes mellitus before the onset of end-stage renal failure ranged from 0.199 to 0.257 mmol/L (normal range 0.088-0.176 (1.3)), and with the development of ESRD, this indicator exceeded the norm many times over, ranging from 0.883 to 2.9 mmol/L, which is associated with impaired renal excretory function.

In patients with diabetes mellitus and the development of TPN, we observed an increase in the content of indican and cystatin C in the blood from 4.4 to 4.9 μmol/L and from 1.9 to 3.6 mg/L (normal range 3.18 and 0.52–0.98), respectively. Elevated indican levels indicate significant impairment of renal excretory function as a result of damage to the renal parenchyma.

Studies have shown that analysis of outpatient records of patients with diabetes mellitus and concomitant pathological obesity revealed an increase in urea levels from 9.2 to 9.8 mmol/L prior to hospitalization. In this group of patients with terminal renal failure, blood urea levels ranged from 30.4 to 56.6 mmol/L. The increase in blood urea levels is associated with sclerotic changes in the walls of the renal capillaries, which leads to impaired blood supply to the renal parenchyma, resulting in ischemia and further development of renal failure.

When reviewing the outpatient records of patients, it was found that the blood creatinine levels in patients with diabetes mellitus and morbid obesity before the onset of end-stage renal failure ranged from 0.198 to 0.251 mmol/L (normal range 0.088-0.176). Along with this, in the same patients, after the development of ESRD, this indicator exceeded the norm many times over, ranging from 0.649 to 2.9 mmol/L. Elevated creatinine levels indicate a significant impairment of the excretory function of the kidneys as a result of glomerulosclerosis of the renal capillaries.

When examining the blood of this group of patients, we observed an increase in the content of indican and cystatin C on average from 4.1 to 5.2 μmol/L and from 1.8 to 3.4 mg/L (with normal values of 3.18 and 0.52–0.98), respectively, which is associated with impaired excretory and metabolic function of the kidneys and its absorption into the blood. Normally, cystatin C in the blood and kidneys is metabolized but not reabsorbed into the blood, and indican should be excreted in the urine, which the studies conducted prove to be impaired in these kidney functions.

A study of patients suffering from diabetes with concomitant coronavirus showed that an analysis of their medical history revealed an increase in urea levels from 9.7 to 12.6 mmol/L prior to admission to the hospital. In patients with end-stage renal failure, blood urea levels ranged from 31.3 to 57.1 mmol/L. The increase in blood urea levels prior to the development of renal failure is directly related to the cytopathic effect of COVID-19 on the renal parenchyma, which was accompanied by severe renal complications with the development of AKI. Vascular damage in

diabetes and the damaging effect of the coronavirus on the renal vessels leads to the activation of macrophages and the development of microembolisms and microthrombi in the renal capillaries and conditions of hypercoagulation and endotheliitis. All this led to the development of renal failure.

When reviewing the outpatient records of patients with diabetes and coronavirus infection, it was found that the blood creatinine levels in patients with diabetes before the onset of end-stage renal failure ranged from 0.201 to 0.298 mmol/L (normal range 0.088-0.176), After the development of ARF in these same patients, creatinine levels were elevated from 0.857 to 2.93 mmol/L, indicating impaired renal excretory function as a result of damage to renal tissue structure.

This proves the increase in the content of indican and cystatin C in patients with diabetes mellitus with coronavirus infection complicated by AKI. Thus, the levels of indican and cystatin C in the blood increased from 3.8 to 4.4 μmol/L and from 1.5 to 3.2 mg/L (normal values are 3.18 and 0.52–0.98), respectively. Elevated indican levels indicate a significant impairment of renal excretory function.

Thus, the increase in the levels of the substances studied is directly related to the cytopathic renal effect of SARS CoV 2, which is accompanied by severe renal complications with the development of AKI. This is associated with kidney infection by viruses and a combination of immune and inflammatory responses with the development of cytokine storm, hypercoagulation, and hemodynamic changes accompanied by ischemia.

Studies of patients with diabetes and other diseases showed that an analysis of outpatient records revealed an increase in urea levels from 9.2 to 11.6 mmol/L prior to hospitalization. With the development of end-stage renal failure, the blood urea levels of patients ranged from 18.9 to 37.2 mmol/L.

Along with these, an increase in blood creatinine levels was observed in patients with diabetes mellitus and various diseases prior to the development of end-stage renal failure, ranging from 0.196 to 0.239 mmol/L (normal range 0.088-0.176). When the same patients were examined after the development of ESRD, this indicator exceeded the normal range many times over, ranging from 0.877 to 2.2 mmol/L.

In patients with diabetes mellitus with various pathologies complicated by ESRD, an increase in the content of indican and cystatin C in the blood was observed, ranging from 3.8 to 4.7 μmol/L and from 1.5 to 3.2 mg/L (normal range 3.18 and 0.52–0.98), respectively.

Conclusions. Thus, determining the concentration of urea, creatinine, indican, and cystatin C in patients with diabetes mellitus allows for early prediction of AKI at an early stage, which leads to a decrease in the number of patients receiving hemodialysis or allows for the detection of renal pathology at an early stage.

Impaired kidney structure in these pathologies leads to impaired cystatin C metabolism and its accumulation in the blood of patients.

An increase in cystatin C concentration indicates kidney damage, which can be determined in patients with diabetes mellitus for early diagnosis of kidney disease. An increase in cystatin C levels in the blood indicates early prediction and impaired kidney function.

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