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# RISK FACTORS FOR END-STAGE RENAL DISEASE IN PATIENTS WITH ARTERIAL HIPERTENSION

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**Abstract:**

These risk factors for terminal renal failure in patients suffering from hypertension were studied. In patients before the development of end-stage renal failure, the content of urea and creatinine was studied. When examining the outpatient card of patients before the development of terminal renal failure, it was found that in some patients, starting from 7 years after suffering hypertension, an increase in the content of urea from 9.2 to 12.1 mmol / l and creatinine from 0.189-0.237 mmol / l in the blood was observed sick.

**Keywords:** Hypertension, end-stage renal failure, urea, creatinine, indican, cystatin

**RELEVANCE OF THE TOPIC:** Diseases of the urinary system throughout the world are an urgent problem and are awaiting their solution. Renal failure (RF) is a violation of kidney function manifested by a decrease in the relative density of urine, an increase in the content of creatinine, urea and indican in the blood, as a result of the cessation of urine output less than 200 ml. With RF, the filtration capacity of the kidneys is impaired. Among the causes leading to renal insufficiency, a significant role is played by hypertension (AH), which follows dibetes mellitus (2.6.16).

Diseases of the genitourinary system are diagnosed late and often, patients remain untreated. Underestimated and progressive factors of diseases of the urinary system, such as diabetes mellitus and arterial hypertension, systemic diseases, anemia, proteinuria, etc., which contribute to an increase in the number of patients with chronic renal failure (CRF) (2.5.6.16.19).

With , HD destruction occurs in the basement membrane of the capillaries and their endothelium. Hyalinosis and sclerotic changes in the capillary wall occur, which leads to a decrease in the volume of capillaries, which ultimately leads to fibrinoid necrosis, and sometimes thrombosis of the kidney capillaries develops. Hyalinosis of capillaries leads to sclerotic changes in the glomeruli and is the main cause of glomerulosclerosis. As a result, the blood supply is disturbed, which leads to organ hypoxia. As a result of hypoxia, atrophy of the renal tissue and necrosis of the organ occurs. Such changes lead to the growth of connective tissue in the kidneys. The number of functioning nephrons decreases. The excretory and concentration function of the kidney is impaired (2.4.6.19)

According to (2.5.6.16), the prevalence of hypertension in Russia is about 40% of the adult population. In persons older than 60 years, the incidence of hypertension exceeds 50%.

According to Rugginenti P., Fassi A., Ilieva A.R., A 2004, an analysis of patients receiving replacement therapy, hemodialysis showed that arterial hypertension is the cause of end-stage chronic renal failure and ranks second after diabetes mellitus and exceeds primary kidney disease. In recent years, there has been an increase in the proportion of hypertensive nephrosclerosis, mainly due to older age groups (Bikbov B.T., Tomilina H.A., 2011).

The definitions of pathological hyperfiltration and microalbuminuria made it possible to reveal the mechanism of the pathogenesis of nephrosclerosis, to identify the initial signs of hypertensive nephropathy. AH occupies one of the leading places in the structure of the causes of end-stage renal disease (ESRD), and indicates the absence of effective nephroprotection at the stage of the course of AH without renal failure and in the initial period of CKD (1.4.6.16.18.).

Of particular interest are the results of several studies indicating that chronic kidney disease (CKD), along with traditional risk factors, should be considered as a predictor of cardiovascular disease. Thus, it has been established that CKD is a significant independent predictor of coronary heart disease (CHD), almost doubling the risk of the disease even when other risk factors for CAD are controlled [Reis S.E. et al., 2002].

The filtration capacity of the kidneys seems to be by and large the only factor that determines the concentration of cystatin C in the blood serum. Cystatin C, being a protein with a low molecular weight, is freely filtered in the renal glomeruli - accumulations of tiny blood vessels, through the pores in the wall of which liquid and low molecular weight substances dissolved in it are filtered. From the formed filtrate, cystatin C in the renal tubules is reabsorbed

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(reabsorbed) and completely metabolized, that is, it is destroyed in the kidneys and does not return back to the blood, and the filtrate enters the bladder and is excreted from the body in the form of urine (1.2.3.7.8.16. 17)

The frequency 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 a cohort study involving 17391 patients with COVID 19 demonstrated that the combined prevalence of pre-existing chronic kidney disease (CKD) and ESRD was 5.2% (2.8–8.1) and 2.3% (1.8 –2.8), respectively.

The authors explain that the urinary syndrome is considered as possible damage to podocytes, renal tubules and interstitium during infection with SARS CoV 2. This is justified by the detection of viral RNA in kidney tissue and urine. In addition, in severe SARS CoV 2, collapsing focal segmental glomerulosclerosis develops in combination with acute tubulonecrosis (5.9.11.13.15)

When examining kidney biopsies of patients who died from COVID 19, in 60% of cases they found the presence of SARS CoV 2 RNA in them. In acute kidney damage, virus RNA was more often detected. This allows us to conclude that there is a correlation between extra respiratory and, in particular, renal tropism of SARS CoV 2 and the severity of COVID 19. Kidney damage in COVID 19 is due to the nephrotropic effect of the virus and its cytopathic effect on the tubular epithelium in parallel with the lung (5.8.9.11.14.15 ).

In patients with severe and extremely severe course of COVID 19, signs of proximal tubular dysfunction were revealed, 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 revealed particles resembling coronavirus in the endoplasmic reticulum of the proximal tubules, which may indicate direct parenchymal infection of the tubular epithelium and podocytes (5.12.15.17).

According to (8.12.17.19), viral particles are present in the endothelial cells of the kidneys, which indicates viremia as a possible cause of endothelial damage in the kidneys and a likely factor contributing to the development of AKI. In addition, SARS-CoV-2 infects the renal tubular epithelium and podocytes through an ACE2-dependent signaling pathway and causes mitochondrial dysfunction, acute tubular necrosis, vacuole formation during protein reabsorption, collapsing glomerulopathy, and protein penetration into the Shumlyansky-Bowman capsule. Another potential mechanism of AKI involves dysregulation of the immune response associated with SARS-CoV-2, as evidenced by the observed lymphopenia and cytokine release syndrome (cytokine storm) in patients [10.15.18.19].

Several factors contribute to acute kidney injury (AKI), including rhabdomyolysis, macrophage activation syndrome, and the development of microemboli and microthrombi in conditions of hypercoagulability and endothelitis [11. 17]

Clarification of risk factors for the development of renal dysfunction will make it possible to predict the timing of development and the rate of progression of renal failure, provide individual assistance and influence the life expectancy of patients with kidney damage in hypertension.

**PURPOSE OF THE STUDY:** To study risk factors and early prediction of renal failure in patients with hypertension and its clinical and prognostic value.

Concomitant diseases G+renal diseases, G+obesity, G+anemia, G+coronavirus, G+ various diseases.

**MATERIALS AND METHODS OF RESEARCH:** The research was carried out in the Bukhara multidisciplinary center in the department of nephrology and hemodialysis. Studies were conducted in 59 patients receiving hemodialysis with an approved primary diagnosis of hypertension. Of the total number of sick women were 17 and men 42 aged 31 to 56 years. All patients were diagnosed with GB II and III degree. End-stage renal failure was approved as a complication of the underlying pathology. In addition to general clinical tests, urea (diamide acetic acid), creatinine, indican in the blood, urinalysis according to Nechiporenko, Zimnitsky, ECG and X-ray studies were examined in all the studied patients. Patients also determined the body mass index. In all patients, the analysis of data from the primary medical documentation of the outpatient card was carried out, concomitant diseases, bad habits, nutritional factors, etc. were studied.

The outpatient records of all patients with prior HD with concomitant pathology were analyzed. The first group included 12 patients with HD without concomitant pathology. In the second group, 16 patients with hypertension concomitant with anemia, in the third group, hypertension with obesity, 18 patients, the fourth group, with coronovirus infection, 6 patients, and hypertension with concomitant other diseases, 7 patients.

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**RESULTS AND DISCUSSION:** The conducted studies showed that in all patients, after the diagnosis of HD, starting from 7 to 11 years, the concentration of urea, creatinine, indican and cystatin C began to increase.

So, the study of the outpatient card of patients with HD showed that the urea content, starting from 7-11 years after the disease, began to increase the urea content from 9.2 to 11.5 mmol/l before admission to the hospital. With the development of terminal renal failure, blood urea in patients ranged from 27.6 to 36.3 mmol/l. An increase in the level of urea in the blood before the development of renal failure is associated with destruction in the basement membrane of the capillaries and their endothelium. Hyalinosis and sclerotic changes in the capillary wall contribute to a decrease in the volume of capillaries, even plasma protrusion of the capillary wall, which ultimately leads to fibrinoid necrosis, as well as the development of thrombosis of the kidney capillaries. All this became the reasons for the development of renal failure.

When reviewing the medical history of patients, it was found that the content of creatinine in the blood of patients with GB before the onset of terminal renal failure ranged from 0.189 to 0.204 mmol / l (normally 0.088-0.176 (1.3)), when examining those patients after the development of ESRD, this the indicator was many times higher than the norm, which ranged from 0.807 to 1.12 mmol / l. An increase in creatinine levels indicates a significant impairment of the excretory function of the kidneys as a result of a violation of the structure of the renal parenchyma.

The content of indican and cystatin C before the development of ESRD has not been determined. In patients with HD with the development of ESRD, 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 (normally 3.18 and 0.52 - 0 .98), respectively. An increase in the level of indican indicates a significant violation of the excretory function of the kidneys, which develops as a result of a violation of the structure of the kidney parenchyma.

In the study of patients suffering from hypertension with anemia, creatinine values before the stationary period ranged from 9.8 to 12.1 mmol/l, which is associated with ischemia of the renal tissue. With the development of terminal renal failure, it increased from 30.4 to 37.8 mmol / l.

An increase in the level of urea in the blood before the development of renal failure is associated with ischemia and destruction in the basement membrane of the capillaries and their endothelium.

When reviewing the medical history of patients, it was found that the content of creatinine in the blood of patients with HD before the onset of terminal renal failure ranged from 0.194 to 0.237 mmol / l (normally 0.088-0.176 (1.3)), when examining those patients after the development of ESRD, this indicator many times higher than the norm, which ranged from 0.871 to 2.1 mmol / l, which is associated with a violation of the excretory function of the kidney.

The content of indican and cystatin C before the development of ESRD has not been determined. In patients with HD with the development of ESRD, 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 (normally 3.18 and 0.52 - 0 .98), respectively. An increase in the level of indican indicates a significant violation of the excretory function of the kidneys as a result of a violation of the structure of the kidney parenchyma.

The conducted studies showed that when analyzing the outpatient card of patients suffering from hypertension with a concomitant disease of obesity, showed an increase in the urea content from 8.9 to 9.7 mmol/l before admission to the hospital. In this group of patients with the development of end-stage renal failure, blood urea ranged from 29.9 to 37.5 mmol/l. An increase in the level of urea in the blood is associated with sclerotic changes in the walls of the capillaries of the kidneys, which leads to disruption of the blood supply to the parenchyma of the kidneys leading to ischemia and further development of renal failure.

When reviewing the medical history of patients, it was found that the content of creatinine in the blood of patients with HD with obesity before the onset of terminal renal failure ranged from 0.196 to 0.248 mmol / l (normally 0.088-0.176). Along with these, in these same patients, after the development of ESRD, this indicator was many times higher than the norm, which ranged from 0.846 to 1.98 mmol/l. An increase in creatinine levels indicates a significant impairment of the excretory function of the kidneys as a result of glomerulosclerosis of the kidney 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 (at a rate of 3.18 and 0.52 - 0.98), respectively, which is associated with a violation of the excretory and metabolizing functions of the kidneys and its absorption into the blood. Normally, cystatin C, which is found in the blood in the kidneys, is metabolized but not absorbed back into the blood, and indican must be excreted in the urine,

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which studies have shown that these kidney functions are impaired.

In studies of patients suffering from hypertension with concomitant coronavirus, it was shown that when analyzing the medical history, an increase in the level of urea from 9.5 to 12.4 mmol / l was observed before admission to the hospital. In patients with terminal renal failure, blood urea of patients ranged from 29.2 to 37.4 mmol/l. An increase in the level of urea in the blood before 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 ESRD. Vascular lesions in hypertension and the damaging effect of coronavirus on the vessels of the kidneys leads to the activation of macrophages and the development of microembolism and microthrombi in the capillaries of the kidney under conditions of hypercoagulability and endothelitis. All this became the reasons for the development of renal failure.

When reviewing the medical history of patients suffering from hypertension with coronavirus infection, it was found that the creatinine content in the blood of patients with GB before the onset of terminal renal failure ranged from 0.192 to 0.213 mmol / l (normally 0.088-0.176), after the development of ESRD in these same patients creatinine values were increased from 0.837 to 2.31 mmol / l, which indicates violations of the excretory function of the kidneys as a result of a violation of the structure of the renal tissues.

This is also proved by the increase in the content of indican and cystatin C in patients with HD with coronavirus infection complicated by ESRD. So the content 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 (normally 3.18 and 0.52-0.98), respectively. An increase in the level of indican indicates a significant impairment of the excretory function of the kidneys.

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

Studies of patients with hypertension with other diseases showed that when analyzing the outpatient card, an increase in the urea content from 9.1 to 11.3 mmol / l was found before admission to the hospital. With the development of terminal renal failure, blood urea in patients ranged from 27.8 to 36.4 mmol/l.

Along with these, an increase in the content of creatinine in the blood was determined in patients with HD with various diseases before the development of terminal renal failure by 0.191 to 0.244 mmol / l (normally 0.088-0.176), in the study of these same patients after the development of ESRD, this indicator is much times exceeded the norm, which ranged from 0.887 to 2.1 mmol / l.

In patients with HD with various pathologies of complicated ESRD, an increase in the content of indican and cystatin C in the blood by 3.8 to 4.7 μmol/l and from 1.5 to 3.2 mg/l (normally 3.18 and 0.52 – 0.98), respectively.

**CONCLUSIONS.** Thus, the determination of the concentration of urea, creatinine, indican and cystatin C in patients suffering from HA for 7-11 years or more gives early prediction of ESRD at an early stage, which leads to a decrease in the number of patients receiving hemodialysis or allows to identify renal pathology at an early stage.

Violation of the structure of the kidneys in these pathologies leads to a violation of the metabolism of cystatin C and its accumulation in the blood of patients.

An increase in the concentration of cystatin C indicates kidney damage, which can be determined in hypertensive patients for early diagnosis of kidney disease. An increase in the level of cystatin C in the blood indicates an early prediction of impaired renal function

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