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Review Article

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Prevalence, Etiology and Pathogenesis of Chronic Renal Failure

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ABSTRACT

Chronic glomerulonephritis, diabetic nephropathy, arterial hypertension are the main reasons for the increase in the number of patients with chronic renal failure. The main task facing nephrologists, urologists, endocrinologists, cardiologists and therapists is to postpone the onset of the terminal stage of chronic renal failure with the need for hemodialysis among patients with kidney pathology. the continuing high mortality rate among patients with the terminal phase of chronic renal failure, which, according to a number of authors, ranges from 52.1% to 67.9%. From these data, it follows that the issues of prevention of renal failure are important and relevant. This review article presents modern information on the prevalence, etiology and pathogenesis of chronic renal failure.

Keywords: *chronic renal failure, nephropathy, renal replacement therapy*

Chronic renal failure is a clinical syndrome characterized by secondary damage to the kidneys in relation to changes in the structure and its function in the form of irreversible and slowly progressive changes in this organ. Another important aspect is that chronic renal failure represents a higher risk of complications and mortality, especially among cardiovascular diseases [21].

The prevalence of chronic renal failure, according to the literature, has a large variation due to the use of different methods for assessing the functional state of the kidneys. Statistics confirm that there are from 3 million to 6 million patients with chronic renal failure worldwide [24].

Chronic renal failure is predominantly found among the adult population and its prevalence among the adult population of developed countries is up to 13% and continues to grow over the years [23].

According to J.R. Lugon [17] of the 2017 census conducted by the Brazilian Society of Nephrology (BSN), the total estimated number of patients on dialysis was 126583, and the national estimates of the prevalence and incidence of patients on dialysis treatment per 1 million population were 61,010.

In addition to its high prevalence, chronic kidney failure is associated with a higher risk of cardiovascular disease and death. In fact, global data from 2013 showed that a decrease in glomerular filtration rate was associated with 4% of deaths worldwide, i.e. 2.2 million deaths. In more than half of these deaths, cardiovascular disease was the leading cause of death, while 0.96 million were

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attributable to end-stage renal disease. At the same time, the total annual mortality rate on dialysis is 19.9% [12].

The main causes of chronic renal failure are diabetes mellitus, arterial hypertension, chronic glomerulonephritis, chronic pyelonephritis, kidney toxicity, chronic use of anti-inflammatory drugs, autoimmune diseases, polycystic kidney disease, congenital malformations, and long-term acute renal failure [22].

Only a few decades have passed to identify the role of nephropathy in the development of chronic renal failure. One of the common causes of such a lesion is diabetic nephropathy. It took only a few decades for this problem to be recognized as the leading etiological cause of chronic renal failure [2]. Since the development of diabetic nephropathy is based on microangiopathy, it can equally occur among patients with type 1 and type 2 diabetes mellitus [27, 32].

Chronic renal failure associated with diabetic nephropathy is a major but underrecognized factor contributing to the global burden of disease. Thus, according to R. Lozano et al. [11] Between 1990 and 2012, the number of deaths associated with diabetic nephropathy increased by 94%. This sharp increase is one of the highest among all recorded chronic diseases [4]. It is noteworthy that most of the excess risk of all-cause mortality and cardiovascular disease in patients with diabetes mellitus is associated with the presence of diabetic nephropathy [16].

Risk factors for the development of chronic renal failure as a result of diabetic nephropathy can be conceptually classified as susceptibility factors (age, gender, family history), initiation factors (e.g., hyperglycemia and acute renal failure), and progression factors (e.g., hypertension, dietary factors, and obesity) [31]. The two most well-known established risk factors are hyperglycemia and hypertension.

In patients with normoalbuminurian type 1 diabetes mellitus, poor glycemic control is an independent predictor of the progression of proteinuria (albuminuria) and/or chronic renal failure [3]. Two landmark studies conducted with patients with early-stage type 1 and type 2 diabetes mellitus have shown that intensive blood glucose control in the early stages of the disease has a long-term beneficial effect on the risk of developing diabetic nephropathy [7, 15]. This phenomenon has been dubbed by a group of scientists led by S. Tonna [19] as "metabolic memory", which suggests that early, intensive glycemic control can prevent irreversible damage, such as epigenetic changes associated with hyperglycemia.

In patients with newly diagnosed neuropathy with blood pressure up to 150/85 mm Hg. over 15 years led to a significant reduction in the risk of developing chronic renal failure by 37% compared to patients who received targeted therapy. Each increase in mean systolic blood pressure by 10 mm Hg. Art. was associated with a 15% increase in the risk ratio of micro- and macroalbuminuria and renal impairment [37]. In general, a baseline systolic blood pressure level above 140 mm Hg. in patients with nephropathy was associated with a higher risk of developing chronic renal failure and death [14, 25].

The development of chronic renal failure against the background of diabetic nephropathy is associated with changes in the structure of multiple renal elements. The earliest consistent change is a thickening of the glomerular basement membrane, which manifests itself within 1.5 to 2 years after the diagnosis of diabetic nephropathy. In parallel, the basement membrane of capillaries and tubules thickens [3, 10, 34].

Other glomerular changes include loss of endothelial fenestration, expansion of the mesangial matrix, and loss of podocytes. An increase in mesangial volume is detected within 5–7 years after the diagnosis of diabetic nephropathy [3, 6, 10, 26].

Segmental mesangiolysis is observed in the progression of nephropathy and is believed to be associated with the development of Kimmelstil-Wilson nodules and microaneurysms, which often appear together [18, 30].

Exudative lesions result from subendothelial deposits of plasma proteins, which form periodic acid-schiff-positive and electron-dense deposits and accumulate in small arterial branches, arterioles and glomerular capillaries, as well as microaneurysms. These deposits can lead to a disturbance of the lumen (for example, hyaline atherosclerosis). Similar subepithelial deposits are observed in Bowman's capsule (capsular guttate lesion) and proximal renal tubules. In the later stages of diabetic nephropathy, interstitial changes and glomerulopathy merge into segmental and global sclerosis. In the case of diabetic nephropathy, glomerular filtration rate, albuminuria, and hypertension have been shown to be strongly correlated with mesangial expansion and somewhat less strongly related to glomerular basement membrane width [28].

Critical metabolic changes that alter renal hemodynamics and promote inflammation and fibrosis in the early development of nephropathy include hyperamino acidemia, glomerular hyperfiltration, and hyperperfusion [8, 9, 13, 36].

According to E. Premaratne et al. [33] Glomerular hyperfiltration leads to early chronic renal failure. Glomerular hyperfiltration is seen in 40% to 75% of patients with diabetic nephropathy. However, as the authors themselves point out, the mechanisms underlying glomerular hyperfiltration in diabetic nephropathy remain not fully understood.

According to K.R. Tuttle [35], one of the likely mechanisms is an increase in the proximal tubular reabsorption of glucose through the sodium-glucose cotransporter, which reduces the distal delivery of solutes, especially sodium chloride, to the dense macula.

According to H.J.L. Heerspink et al. [29] The resulting decrease in tubuglomerular feedback may dilate the afferent arteriole to increase glomerular perfusion, while the high local production of angiotensin II in the efferent arteriole causes vasoconstriction. The overall effect is high intraglomerular pressure and glomerular hyperfiltration.

The clinical diagnosis of diabetic nephropathy is made based on the measurement of glomerular filtration rate and albuminuria, as well as clinical signs, such as the duration of diabetes mellitus and the presence of other signs of diabetic angiopathy, such as diabetic retinopathy [1, 5].

Diabetic nephropathy is clinically defined by a persistently high urinary albumin-to-creatinine ratio of ≥30 mg/g and/or a sustained decrease in glomerular filtration rate of less than 60 ml/min per 1.73 m2 [20].

Screening for diabetic nephropathy should be done annually for patients with type 1 diabetes mellitus starting 5 years after diagnosis, and annually for all patients with type 2 diabetes mellitus starting at diagnosis. In patients with albuminuria, the presence of diabetic retinopathy strongly suggests diabetic nephropathy. The preferred test for albuminuria is to determine the ratio of albumin to creatinine in the urine on a point sample, preferably in the morning [1, 5, 20].

The glomerular filtration rate is calculated based on the serum creatinine concentration. Although the Chronic Kidney Disease Epidemiological Outlook Initiative equation is more accurate, especially at glomerular filtration rate levels in the normal or near-normal range, the Kidney Disease Diet Change equation is commonly reported by clinical laboratories. Two abnormal measurements at least 3 months apart are required to confirm albuminuria or low glomerular filtration rate. If signs that are atypical of diabetic kidney disease are present, then other causes of kidney disease should be considered. Atypical signs include sudden onset of low glomerular filtration rate or a rapid decrease in glomerular filtration rate, a sharp increase in albuminuria or the development of nephrotic or nephritic syndrome, refractory hypertension, signs or symptoms of another systemic disease, and a >30% decrease in glomerular filtration rate within 2 to 3 months of initiation of a renin-angiotensin system inhibitor.

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SURUNKALI BUYRAK ETISHMOVCHILIGINING TARQALISHI, ETIOLOGIYASI VA PATOGENEZI

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ABSTRAKT

Surunkali glomerulonefrit, diabetik nefropatiya, arterial gipertoniya surunkali buyrak etishmovchiligi bilan og'rigan bemorlar sonining ko'payishiga asosiy sabab bo'ladi. Nefrologlar, urologlar, endokrinologlar, kardiologlar va terapevtlar oldida turgan asosiy vazifa buyrak patologiyasi bilan og'rigan bemorlar orasida gemodializ zarurati bilan surunkali buyrak yetishmovchiligining terminal bosqichi boshlanishini orqaga surishdan iborat. surunkali buyrak etishmovchiligining terminal bosqichida bo'lgan bemorlar o'rtasida o'lim darajasi yuqori bo'lib, bir qator mualliflarning fikriga ko'ra, 52.1% dan 67.9% gacha. Ushbu ma'lumotlardan buyrak etishmovchiligining oldini olish masalalari muhim va dolzarb ekanligidan kelib chiqadi.Ushbu ko'rib chiqish maqolasida surunkali buyrak yetishmovchiligining tarqalishi, etiologiyasi va patogenezi bo'yicha zamonaviy ma'lumotlar keltirilgan.

Tayanch iboralar: surunkali buyrak yetishmovchiligi, nefropatiya, buyrak almashtirish terapiyasi

РАСПРОСТРАНЕННОСТЬ, ЭТИОЛОГИЯ И ПАТОГЕНЕЗ ХРОНИЧЕСКОЙ ПОЧЕЧНОЙ НЕДОСТАТОЧНОСТИ

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АБСТРАКТ

Хронический гломерулонефрит, диабетическая нефропатия, артериальная гипертония – являются основными причинами увеличения количества больных с хронической почечной недостаточностью. Главное задачей, стоящей перед нефрологами, урологами, эндокринологами, кардиологами и терапевтами является отдалить наступление терминальной стадии хронической почечной недостаточности с потребностью в гемодиализе среди больных с патологией почек. Данный вектор стратегического направления обусловлен все еще сохраняющейся высокой летальностью среди больных с терминальной фазой хронической почечной недостаточностью, достигающую по данным ряда авторов от от 52,1% до 67,9%. Из этих данных следует то, что вопросы профилактики почечной недостаточности являются важными и актуальными. Данной обзорной статье представляются современные сведения относительно распространенности, этиологии и патогенеза хронической почечной недостаточности.

Ключевые слова: хроническая почечная недостаточность, нефропатия, заместительная почечная терапия