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FEATURES OF DAILY BLOOD PRESSURE IN PATIENTS WITH NEPHROGENIC ARTERIAL HYPERTENSION

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Abstract
Cardiovascular complications caused by nephrogenic arterial hypertension are the leading cause of death in patients with chronic renal failure. The most significant factor in the pathogenesis of hypertension in diffuse kidney diseases is considered to be sodium retention, accompanied by an increase in the volume of extracellular fluid and cardiac output. The adverse effect of uncontrolled hypertension on renal outcomes has been demonstrated by numerous studies. The data presented in the article indicate that in patients with nephropathies, in order to slow down and prevent the deterioration of kidney function, it is desirable to achieve target blood pressure values. Daily blood pressure monitoring in nephrology makes it possible to determine the severity of hypertension and its daily fluctuations, select adequate antihypertensive treatment, and evaluate its effectiveness.

Keywords: nephrogenic arterial hypertension, circadian rhythm of blood pressure, method of 24-hour blood pressure monitoring.

Introduction

Arterial hypertension is one of the most significant medical, economic and social problems of the 21st century. It has been established that arterial hypertension affects 20-30% of the adult population, and among people over 65 years of age, 50% or more. There are syndromes of primary and secondary arterial hypertension. The syndrome of primary arterial hypertension (essential hypertension) is observed in 90-95% of patients with high blood pressure, the remaining 5% have diseases syndromicly combined as secondary hypertension of renal and other origins [1] .

The syndrome of secondary arterial hypertension of renal origin is observed both with damage to the renal arteries (vasorenal) and with parenchymal hypertension: IgA nephropathy, chronic glomerulonephritis, secondary glomerulonephritis, complicating acute glomerulonephritis, chronic pyelonephritis, nephropathy of pregnancy, diabetic nephropathy, gout, collagenosis, vasculitis, kidney tumors, urolithiasis. But mostoften there is a hypertonic form of chronic glomerulonephritis. As is known, the kidneys are the most important organ for maintaining metabolic homeostosis of the body. Various kidney diseases are manifested and complicated by a number of syndromes.

One of the most significant, widespread and dangerous is nephrogenic arterial hypertension (NAH) [2]. According to statistics, NAH is the most common cause of symptomatic arterial hypertension, which causes up to 5% of all cases of NAH [13]. Its development is recorded in 50-80% of patients with nephrological diseases. The great significance of NAH is associated with the high frequency of the disease in young working age, often malignant in nature and resistance to treatment. Today,

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there is a lot of literature devoted to the increased cardiovascular risk in patients with NAH, the high incidence of hypertensive encephalopathy with seizures, plasma and retinal hemorrhages [3,5,8] .

Cardiovascular complications caused by NAH are the leading cause of death in patients with chronic renal failure. NAH is dangerous because it contributes to the progression of renal pathology. Increased cardiovascular risk and risk of overall mortality have been reported in patients with chronic renal failure. including those receiving hemodialysis, if they continue to have high blood pressure [15] .The main group of diseases that lead to the development of renal hypertension are renal parenchymal diseases. Separately, there is renovascular hypertension, which occurs as a result of renal artery stenosis. When In chronic glomerulonephritis, the frequency of hypertension is on average 50-60% and largely depends on the morphological variant of kidney damage. Most often (up to 70-85%) hypertension is detected in the mesangiocapillary variant of GN and FSGS, less often found in memranous, mesangioproliferative and Ig A -GN (from 40 to 50%). Most rarely, hypertension is recorded in cases of GN with minimal changes (34,45). At the present stage, several factors in the pathogenesis of renal hypertension are identified: sodium and water retention, dysregulation of pressor and depressor hormones, increased formation of free radicals , kidney ischemia, gene disorders.

The most significant factor in the pathogenesis of hypertension in diffuse kidney diseases is considered to be sodium retention, accompanied by an increase in the volume of extracellular fluid and cardiac output. This is the most common mechanism for the development of renal hypertension. Volume-dependent hypertension is detected in 80-90% of patients with acute GN and chronic renal failure. As a result of sodium retention, changes occur in the content of electrolytes and the vascular wall (accumulation of sodium and calcium ions in it), its swelling, which leads to an increase in the sensitivity of blood vessels to the pressor effects of vasoconstrictor hormones (angiotensin II, catecholamines, vasopressin, vasoconstrictor endothelial hormones) . acts as the main development factor for high peripheral resistance (OPR) and total renal vascular resistance [5]. Thus , sodium and water retention by the kidneys affects both factors of blood pressure regulation - cardiac output and TPR. The main causes of sodium retention in kidney disease - damage to the renal glomeruli with a subsequent decrease in the mass of active nephrons, inflammation in the renal parenchyma, increased reabsorption in the proximal, distal tubules and collecting duct, primary tubulointerstitial disorders. The presented data on the role of sodium in the mechanism of development of hypertension and the existence of many factors leading to sodium retention determine the need for the treatment of renal hypertension to limit sodium in the diet and, if necessary, prescribe diuretics. Dysregulation of pressor and depressor systems.

Renal hypertension, independent of volume, is detected in 5-10% of patients. In this variant of hypertension, blood volume and cardiac output, as a rule, remain within normal values. In this case, the reason for increased blood pressure is an increase in vascular tone due to dysregulation of pressor and depressor hormonal systems, which leads to an increase in peripheral blood pressure. Physiological regulators of vascular tone are vasoactive hormones: vasoconstrictor (angiotensin II, catecholamines, endothelins) and vasodilating (kinins, prostaglandins, endothelins) and vasodilating (kinins, prostaglandins, endothelium-relaxing factor, calcitonin-gene-associated peptide, etc.). In case of kidney diseases, an imbalance in the physiological balance in the vasoconstrictor-vasodilator system is detected in favor of vasoconstrictors. In kidney diseases, activation of one of the strongest vasoconstrictors, angiotensin II , occurs when renal hemodynamics are impaired as a result of the development of acute immune inflammation or sclerotic processes. In addition to the enhanced formation of systemic angiotensin II , the local RAAS is activated in the

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kidneys with the production of a vasoconstrictor hormone directly in the renal tissue. Combined effect activated systemic and renal angiotensin II , in the kidneys the local RAAS is activated with the production of a vasoconstrictor hormone directly in the renal tissue. The combined effect of activated systemic and renal angiotensin a II occurs when renal hemodynamics are impaired as a result of the development of acute immune inflammation or sclerotic processes. In addition to increased formation of systemic angiotensin II , in the kidneys the local RAAS is activated with the production of a vasoconstrictor hormone directly in the renal tissue. The combined effect of activated systemic and renal angiotensin II provokes a narrowing of both resistive vessels (medium-diameter arterioles), which mainly determine OPS, and intrarenal vessels, which leads to increase in OPS. In the pathogenesis of NAH, disturbances of intracardiac hemodynamics, activation of the juxtaglomerular apparatus (JGA), sympathoadrenol system (SAS), and increased activity of the renin-aldosterone-angiotensin system (RAAS) play a role .

Numerous studies have demonstrated the adverse effects of uncontrolled hypertension on renal outcomes and cardiovascular events. According to Shulman et al, over a 5-year period, an increase in serum creatinine from the initial normal level to 2.0 mg/dL was observed in 1.5% of patients with DBP from 90 to 104 mmHg. Art., 3.5% with DBP from 105 to 114 mm Hg. and 55% for DBP over 115 mmHg. Art. According to Perry et al. who summarized the results of a survey of 6182 patients and 5730 black veterans with hypertension, observed for 15 years, with SBP from 165 to 180 mmHg. the risk of developing ESRD increased 2.8 times and with SBP 180 mm Hg. st-6 times. It is SBP and vascular stiffness that are currently given the main importance in damage to target organs, including the kidneys. According to Mule et al, arterial stiffness is inversely correlated with GFR. Thus, the relationship between blood pressure and the risk of developing both cardiovascular events and kidney failure is typical for patients with NAH. There are not enough randomized controlled trials (RCT) on BP targets in patients with NAH. Jafar et alconducted a meta-analysis covering 1860 patients with nondiabetic nephropathy treated with and without ACEI. Treatment effectiveness was assessed by preventing a doubling of serum creatinine and the initiation of renal replacement therapy. During the observation period (2.2 years) progression of nephropathy was observed in 311 patients. The main determinants of progression in multivariate analysis were SBP and proteinuria. The slowest deterioration in renal function was observed with SBP from 110 to 129 mmHg. Art. and proteinuria less than 2.0 g/day. With SBP > 130 mm. Hg. The risk of progression increased by 1.83 times, but was maximum when SBP was less than 100 mmHg. With an increase in protein excretion to 2.9 g/day, the risk of progression increased to 2.54 and to 4.77 with proteinuria more than 6.0 g/day. At the same time, in the M DRD and AASK studies (The African American Study of Kidney Disease and Hypertension) in patients with both target and higher blood pressure values, the rate of decrease in glomerular filtration did not differ[]In the REIN-2 study (Ramipril Efficacy In Nephropathy) among 338 participants, terminal uremia during 36 months of observation developed in patients with blood pressure <130/80 mmHg and DBP <90 mmHg. The data presented indicate that in patients with nephropathy, in order to slow down and prevent deterioration of kidney function, it is desirable to achieve target blood pressure values .[13, 15] However, traditional one-time measurements do not always reflect true blood pressure, leaving open the question of the correctness of diagnosing increased blood pressure (for example, in many patients, when visiting a doctor, blood pressure may be 30-40 mmHg higher than when measured at home), do not provide an idea of the daily blood pressure curve and do not allow a full assessment of the antihypertensive effectiveness of drugs. normal human life opens up additional diagnostic possibilities: the results of daily blood pressure monitoring more accurately reflect the severity of

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hypertension and its prognosis [7,16]. Blood pressure is considered definitely elevated if its average daily values exceed 140/90 mm, during the day -150/90 mm. Hg at night - 130/80 mm Hg. During daily monitoring, a biphasic blood pressure rhythm is observed. Two peaks of blood pressure are recorded during the day - the morning one, when blood pressure reaches its maximum values, and a less pronounced evening one. During sleep, between 2 and 4 a.m., a nocturnal minimum blood pressure is observed, after which blood pressure rises sharply, reaching the daytime level by 6 a.m. The method of 24-hour blood pressure monitoring (ABPM) has been widely used in clinical practice for more than two decades. This method can be considered one of the most important achievements in cardiology, especially in the field of management of patients with arterial hypertension (AH). Undoubtedly, ABPM provides clinically valuable information due to the ability to assess blood pressure (BP) levels during the patient's normal activity, a large number of measurements throughout the day, including at night, analysis of the nature of circadian fluctuations and blood pressure variability.

Threshold values of hypertension for ABPM are different; they are slightly lower than clinical blood pressure (measured at a doctor's appointment) and depend on the time of day [4,8,11]. BP according to ABPM data and their significant advantage compared to traditional clinical BP measurements were demonstrated back in 1966 by M. Sokolow et al [2,9].

In clinical practice, blood pressure measurements are usually taken at 15-minute intervals during the day and at 30-minute intervals at night. Most often in practice, average daily, average night and average daily blood pressure values are used. Average daily and average nightly blood pressure readings can be calculated from a diary, taking into account the time of waking up and going to bed. The ratio of nighttime to daytime BP reflects the ratio between average nighttime and average daytime BP. Normally , at night, blood pressure decreases (≪dipping≫) . Based on the ABPM results, a number of additional indices can be calculated. These include: BP variability, morning peak BP, pressure load and ambulatory arterial stiffness index[1,6,10].

The daily index (DI) is calculated using the formula: CI = (avg ADD - mean ABP) x100%/avg d. The daily index in 82% of healthy individuals ranges from 10 to 20%, however, some normotensive patients experience disturbances in the circadian rhythm of blood pressure: insufficient reduction or higher - at night, excessive drop - during sleep. The progression of diseases leads to various changes in the daily blood pressure profile. The following groups of patients with hypertension are distinguished: dippers - patients (52-82%) with a normal decrease in blood pressure at night and a daily index of 10 -22%, non - dippers - patients (16-26%) with an insufficient nighttime drop in blood pressure, whose daily index is less than 10%, over - dippers - patients (about 19%) with an excessive drop in blood pressure at night, whose daily index exceeds 22% and night - peakers are patients (about 3%) with nocturnal hypertension, in whom night-time blood pressure exceeds daytime blood pressure, and the daily index has negative values. In non-dipper and night - peaker patients, LVH more often develops, which is an independent factor in the unfavorable course of hypertension [5,8,12]. A close connection has also been established between insufficient reduction in blood pressure at night and microalbuminuria. This dependence is especially pronounced in patients with night - peaker, in whom SI < 10%. Circadian rhythm disturbances with insufficient reduction in blood pressure at night are associated with a higher incidence of strokes, frequent development of LVH, and the frequency and severity of microalbuminuria. Daily blood pressure monitoring in nephrology allows you to determine the severity of hypertension and its daily fluctuations, select adequate antihypertensive treatment, and evaluate its effectiveness. Research conducted by G.A. Ignatenko et al (2002) showed that patients with CGN without chronic renal

failure belong predominantly to the night category - reactions - the prognostically most unfavorable variant of the course of hypertension.[6,13,14]

At present, many factors influencing the dynamics of blood pressure levels in humans remain unknown. However, simultaneous measurement of blood pressure and determination of the secretion of some biologically active substances into the blood made it possible to identify a correlation between the level of blood pressure, the activity of creatinine in the blood plasma, the level of norepinephrine and angiotensin. There is no doubt that the level of vasoactive hormones is important for increasing blood pressure in the early morning hours. It is believed that night - peak patients are prognostically the most severe contingent in terms of the effectiveness of treatment and prognosis. This type of circadian rhythm of blood pressure is often found in renoparenchymal hypertension. It is believed that it is based on hyperactivation of the RAAS and sympathetic nervous system.

Summarizing the data on the influence of daily changes in blood pressure on the course of the disease and the prognosis of nephrogenic arterial hypertension, determining the daily circadian rhythm of blood pressure is important when assessing the effectiveness of treatment in this category of patients.

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