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"STUDYING THE PECULIARITIES OF INCREASING THE EFFICIENCY OF PREDICTION AND PREVENTION OF RENAL FAILURE IN PATIENTS WITH HYPERTENSIONAL NEPHROPATHY»

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ABSTRACT

In recent decades, along with cardiovascular manifestations, the incidence of renal lesions has increased in arterial hypertension (AH). It follows that the development and implementation of measures to combat the complications of hypertension, in particular, the prevention of kidney damage, is a priority in healthcare. The system of measures for predicting the risk of developing hypertensive nephropathy is a strategic basis for developing a prevention system, which is the purpose of this study.

KEYWORDS: *Arterial Hypertension, Kidneys, Diagnostics, Treatment.*

INTRODUCTION

A feature of the pathology of the kidneys is that with the development of a detailed picture of the disease or complications, such as chronic renal failure, it is not possible to radically affect the progression of the disease. And the only thing in this case is a decrease in the rate of progression of renal failure **[1]**. Kidney damage in hypertension is an example of this. Often, the risk of development and progression of kidney pathology in AH depends on the factorial environment **[4]**. Thus, the discovery of risk factors is a key step in understanding the pathogenesis pathways leading to the development of pathology and the identification of effective strategies to prevent the development and progression of the disease (Brenner BM, 2007). Impaired kidney function is one of the most important risk factors for cardiovascular complications. (SSO). The results of epidemiological studies indicate that early subclinical impairment of kidney function is an independent risk factor for CV events and death. The kidneys are part of the microcirculatory system of the body, they affect the formation of arterial hypertension, especially in combination with diabetes mellitus, heart failure and other renal diseases [1, 6, 8]. The appearance of microalbuminuria (MAU) and a decrease in the glomerular filtration rate (GFR) are considered as markers of an unfavorable prognosis for common cardiovascular diseases and are a reflection of the concept of cardiorenal relationships that has recently become widespread **[2, 3, 7]**. that early subclinical renal dysfunction is an independent risk factor for CV events and death. The kidneys are part of the microcirculatory system of the body, they affect the formation of arterial hypertension, especially in combination with diabetes mellitus, heart failure and other renal diseases [1, 6, 8]. The appearance of microalbuminuria (MAU) and a decrease in the glomerular filtration rate (GFR) are considered as markers of an unfavorable prognosis for common

cardiovascular diseases and are a reflection of the concept of cardiorenal relationships that has recently become widespread [2, 3, 7]. that early subclinical renal dysfunction is an independent risk factor for CV events and death. The kidneys are part of the microcirculatory system of the body, they affect the formation of arterial hypertension, especially in combination with diabetes mellitus, heart failure and other renal diseases [1, 6, 8]. The appearance of microalbuminuria (MAU) and a decrease in the glomerular filtration rate (GFR) are considered as markers of an unfavorable prognosis for common cardiovascular diseases and are a reflection of the concept of cardiorenal relationships that has recently become widespread [2, 3, 7].

For a long time, the only variant of kidney damage in arterial hypertension (AH) was considered hypertensive nephroangiosclerosis (damage mainly to the glomerular apparatus of the kidneys in AH). Concomitant hypertension also predisposes to the development of hypertensive nephroangiosclerosis - type 2 diabetes mellitus (DM), hyperuricemia, atherosclerotic stenosis of the renal artery - PA (ischemic disease).

kidney disease), embolization of PA with cholesterol crystals (CS), in which chronic renal failure (CRF) increases. Thus, the concept of "kidney damage in hypertension", or hypertensive nephropathy, combines several nosological forms:

1. Classical hypertensive nephroangiosclerosis (often in combination with concomitant chronic urate tubulo-interstitial nephritis and / or urate nephrolithiasis, diabetic nephropathy).

2. Atherosclerotic stenosis of the renal artery - ischemic kidney disease (IBD) and / or cholesterol embolism of the intrarenal vessels.

Let us consider these nosological forms in more detail.

Hypertensive nephroangiosclerosis is a lesion mainly of the glomerular apparatus of the kidneys in hypertension

Tubulointerstitial nephropathy is a primary lesion of the tubules and interstitial tissue of the kidneys due to immuno-inflammatory (autoimmune diseases), toxic (infections) and metabolic (impaired calcium, potassium, uric acid metabolism - urate nephrolithiasis) diseases. Diabetic nephropathy - kidney damage

in diabetes mellitus, the morphological basis of which is nephroangiosclerosis of the renal glomeruli.

Ischemic kidney disease (IBD) is an atherosclerotic stenosis of the renal arteries. UPS occurs predominantly in patients with widespread and often complicated atherosclerosis. Cholesterol embolism of the intrarenal vessels is a special variant of IBS, characterized by embolism of the intrarenal arteries by cholesterol crystals, the source of which is an atherosclerotic plaque localized in the abdominal aorta or in the main renal arteries.

Pathogenesis of hypertensive nephroangiosclerosis and ischemic kidney disease. A key determinant of deterioration in kidney function is an increase in systolic blood pressure (SBP). In hypertensive nephroangiosclerosis, damage to glomerular endotheliocytes is considered the primary link, which occurs as follows. With an increase in SBP, activation of the sympathoadrenal (SAS) and renin-angiotensin-aldosterone systems (RAAS), constriction of the afferent glomerular artery occurs with a decrease in effective renal blood flow (EPC) and the formation of angiotensin A11 (the leading factor), which causes spasm of the efferent glomerular

artery and contributes to the development of intraglomerular hypertension with subsequent hyperfiltration and increased protein permeability. In the future, hyperfiltration is aggravated, blood flow to the glomerular capillaries decreases,

Currently, hypertension is very often combined with other risk factors (RFs) for cardiovascular diseases: obesity, hyperuricemia, dyslipidemia, i.e. metabolic syndrome (MS), insulin resistance (IR) and type 2 diabetes mellitus (DM). Thus, an excess of insulin in the blood stimulates the proliferation of smooth muscle cells (SMC) of vessels, mesangial cells and renal tubulointerstitium, inducing the processes of local renal fibrogenesis. In addition, the end products of glycosylation cause the development of the phenomenon of persistent hyperfiltration, a fundamental component of the pathogenesis of diabetic kidney disease.

The consequence of impaired uric acid metabolism is urate nephrolithiasis and chronic tubulointerstitial nephritis (uric acid and its salts lead to tubulointerstitial fibrosis), which are already formed at the stage of hyperuricosuria. Thus, urate dysmetabolism, almost always associated with high blood pressure (BP), leads to urate nephropathy.

In obesity, the phenomenon of persistent hyperfiltration is well known. But the "nephrotoxic" hormone of adipose tissue, leptin, is still of decisive importance, as well as the transforming growth factor B-factor (TGF-B) and interleukin-6 produced by adipocytes.

As one of the likely mechanisms of kidney damage in AH, there may be a genetically determined insufficiency in the formation of endothelial vasodilators (primarily nitric oxide) in the renal microcirculatory bed.

Major Progression Factors

Kidney damage:

• Systemic hypertension;

• intraglomerular hypertension;

• proteinuria;

• Increased intake of protein from food;

• tubulointerstitial fibrosis (nephrosclerosis);

• hyperlipidemia;

• Hyperglycemia.

Ischemic kidney disease (IRD) develops with atherosclerotic stenosis of the renal artery (RA) and is determined by global hypoperfusion of the kidney tissue. In response to a decrease in the volume of blood entering the renal tissue, hyperactivation of the RAAS is observed, which makes it possible to keep the glomerular filtration rate (GFR) relatively constant. The renal tubulointerstitium is the most ischemic. As hypoperfusion increases, atrophy of the tubulointerstitium and its fibrosis increase. Complete obliteration of PA is always accompanied by atrophy of the corresponding kidney. Embolism of the intrarenal arteries is considered to be a special variant of the UPS with cholesterol crystals (CS), the source of which is an atherosclerotic plaque. Simultaneous massive embolism with crystals of cholesterol in the intrarenal vessels leads to a sharp ischemia of the renal tissue and acute renal failure (ARF),

accompanied by an intractable rise in blood pressure. If the embolism grows slowly (chronic variant), then cholesterol crystals injure the vessel wall, enter the renal tubulointerstitium, activate complement components and cause eosinophilic tubulointerstitial nephritis.

Diagnosis of kidney damage in arterial hypertension is based on the use of the following differential diagnostic features:

1. The duration of the existence of hypertension (history: arterial hypertension in young people, severe hypertension in people over 55 years of age).

2. Damage to other target organs (LVH, CHF, cerebrovascular disease).

3. The presence of risk factors: uric acid, glucose, insulin, dyslipidemia, obesity, abuse of non-steroidal anti-inflammatory drugs.

4. Presence of microalbuminuria — MAU, no changes in urinary sediment, stable renal failure (moderate hypercreatininemia, Table 1).

The clinical picture of kidney damage in AH (hypertensive nephroangiosclerosis) is nonspecific, for a long time this form of kidney damage remains almost asymptomatic [3, 5]. The changes relate to the appearance of "trace" microalbuminuria (MAU).

1. MAU (30-300 mg/day) is a “renal” sign of endothelial dysfunction.

2. Hypercreatininemia is moderate, its rate of increase is low (>115-133 μmol/l or>1.3-1.5 mg/dl).

3. To detect signs of kidney damage, the albumin / creatinine ratio should be used, at which values exceeding 30 mg of albumin per 1 g of creatinine are considered a deviation from the norm.

4. Changes in the urinary sediment are uncharacteristic of kidney damage in arterial hypertension.

5. Hypertensive nephroangiosclerosis appears later than the defeat of other target organs.

Ischemic kidney disease is diagnosed based on the following data (A.N. Mukhin, V.V. Fomin, 2005) [3]:

1. Risk factors for the development of atherosclerosis (smoking, hypercholesterolemia, hypertriglyceridemia, decreased levels of HDL, DM 2, hyperhomocysteinemia).

Table 1.

Urinary protein excretion (GNOK, 2008)

Albuminuria

Method for determining the norm of MAU or proteinuria

Proteinuria

Day. excretion<300 mg/day - >300 mg/day

Test strips <30 mg/dl - >30 mg/dl

Protein/Cr ratio <200 mg/g - >200 mg/g

Albuminuria Sut. excretion Test strip Protein/Cr ratio <30 mg/day <3 mg/dL 30-300 mg/day >3 mg/dL>300 mg/day

<17 mg/g (M) <25 mg/g (F) 17-250 mg/g (M) 25-355 mg/g (W) >250 mg/g (M) >355 mg/g (W)

Table 2.

Clinical action plan depending on the stage of CKD (WHO, 2008)

CKD stage Description GFR (mL/min/1.73m2) Actions

- Risk group, risk factor CKD > 90 Screening, risk factor correction

1 Normal or elevated GFR > 90 Diagnosis and treatment of causes of kidney damage

2 Slight decline in GFR 60-89 Assess progression

3 Moderate decrease in GFR 30-59 Detection and management of complications

4 Severe decrease in GFR 15-29 Preparing for renal replacement therapy

5 End-stage renal failure <15 (or hemodialysis) Renal replacement therapy

2. Features of arterial hypertension (high levels of blood pressure, ISAH, onset of hypertension in old age, low efficiency of combined antihypertensive therapy).

3. The prevalence of atherosclerosis (presence of coronary artery disease, cerebrovascular disease, intermittent claudication syndrome, abdominal aortic aneurysm).

4. Features of renal insufficiency (long-term history of moderate creatininemia, a sharp deterioration in renal function when prescribing ACE inhibitors and ARBs).

5. Doppler ultrasound data of the renal arteries, multislice computed tomography, magnetic resonance imaging.

Cholesterol embolism is characterized by changes in the retina (Holenhorst plaques), the results of a urine test (eosinophiluria), an increase in the serum level of CRP and ESR. Skin biopsy from areas of livedo reticularis (detection of cholesterol crystals) and kidney biopsy have a certain diagnostic value. Extrarenal manifestations of cholesterol embolism depend on the location of the emboli.

Treatment of patients with hypertensive nephropathy

The goal of treating patients with hypertension and kidney disease is to minimize cardiovascular and renal morbidity and mortality as much as possible. To achieve this goal, it is necessary

we go to control risk factors (smoking, dyslipidemia, diabetes mellitus), treatment of concomitant clinical conditions, treatment of elevated blood pressure itself (antihypertensive therapy).

Tasks of therapy; adequate control of blood pressure (<130/80 mm Hg), nephroprotection, especially in diabetic nephropathy, reduction of proteinuria or MAU to values close to normal.

In the presence of MAU or proteinuria, ACE inhibitors or angiotensin receptor blockers (ARBs) II with extrarenal elimination are the drugs of choice. To achieve the target level of blood pressure in kidney damage, combination therapy is often required, including a diuretic (in case of

impaired nitrogen excretion of the kidneys - a loop diuretic) and / or a calcium antagonist. With diabetes, taking into account the increased risk of cardiovascular complications, complex therapy is indicated: antihypertensive drugs, statins, antiplatelet agents, etc.

The main methods of treatment for lesions of the renal arteries. In case of vasorenal arterial hypertension (when atherosclerosis of the renal arteries is the cause of hypertension in 75% of cases), the main methods of treatment are symptomatic drug therapy, angioplasty and stenting of the affected renal arteries, and surgical treatment. Long-term combination therapy includes calcium antagonists, diuretics, statins, and low-dose aspirin. ACE inhibitors and ARBs are contraindicated in patients with bilateral hemodynamically significant renal artery stenosis or renal artery stenosis in a solitary kidney.

General principles for the management of patients with chronic kidney disease (CKD). CKD and CVD share modifiable risk factors, the most important of which are hypertension and diabetes. Strict control of hypertension is key to preventing the progression of CKD. The target BP level in patients with stage 3-4 CKD is BP < 130/80 mm Hg. Art. The management plan for patients with CKD takes into account the stage of renal dysfunction (Table 2) [4].

ACE inhibitors or angiotensin II receptor antagonists. All patients with CKD should receive ACE inhibitors or angiotensin II receptor antagonists that have been shown to slow the progression of CKD (without regard to blood pressure). However, in patients with hypotension (SBP <90 mmHg), potassium levels >5mmol/l, serum Cr >221 μmol/l (2.5 mg/dl), ACE inhibitors and ARA II should be administered with caution. With a decrease in GFR <30 ml / min / 1.73 m2, adjustment of the starting dose and ACE, and some angiotensin receptor antagonists (ARA) II is required. Diabetic nephropathy has special indications for the appointment of captopril, irbesartan, losartan. New blockers of the renin-angiotensin-aldosterone system (RAAS) are being studied - direct renin inhibitors (aliskiren).

Diuretics. Diuretics are required in most patients with CKD to achieve BP goals. Patients with GFR >30 ml/min/1.73 m2 should be given thiazide diuretics once a day; those with GFR <30 ml/min/1.73 m2 should receive loop diuretics (furosemide, torasemide) 1-2 times a day. Potassium-sparing diuretics should be used with caution in stage 4-5 CKD.

Other drugs. The antiproteinuric effect of calcium antagonists has been shown, for which dose reduction is not required with reduced renal function. Beta-blockers can be prescribed only if there are indications: myocardial infarction, stable angina, heart failure. In addition to standard therapy, moxonidinecan be used in CKD. In CKD stages 2-3, a decrease in LDL cholesterol <100 mg / dl is recommended (prescription of simvastatin, atorvastatin, rosuvastatin). However, with a more severe degree of CKD, the advisability of prescribing statins needs to be clarified.

anemia correction. The presence of anemia is established when the concentration of hemoglobin (Hb) <130 g/l in men and <120 g/l in women. All patients with an Hb level <110 g/l are indicated for treatment with erythropoiesis/stimulant drugs, individually for each patient, depending on the stage of CKD, efficacy, safety, and the class of erythropoiesis/stimulant drug.

Thus, hypertensive nephropathy includes several nosological forms, united by the presence of hypertension syndrome, microalbuminuria, and stable renal failure. Timely diagnosis of kidney damage in arterial hypertension and targeted

naya therapy can prevent the occurrence and further spread of cardiovascular complications.

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