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Guidelines for conducting a practical lesson with 5th year students of the Faculty of Medicine on the topic:

DIFFERENTIAL DIAGNOSTICS OF BASIC PATHOLOGICAL SYNDROMES IN NEPHROLOGY

(the duration of the lesson is 12 hours, the third lesson is 4 hours)

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DIFFERENTIAL DIAGNOSTICS OF BASIC

PATHOLOGICAL SYNDROMES IN NEPHROLOGY.

PURPOSE OF THE LESSON: To improve the level (quality) of knowledge and skills of students in the differential diagnosis of the main pathological syndromes in the nephrological clinic, in particular, the syndrome of arterial hypertension.

Students should be able to:

1. to be able to identify the main syndromes in diseases of the urinary organs and the key points of the anamnesis, on the basis of complaints, anamnesis and objective status, make a preliminary diagnosis of the presence of kidney disease in a patient;

2. justify the need for additional research;

3. to highlight the syndrome of arterial hypertension; to determine the nosological basis of the identified syndrome;

4. to determine the scope of therapeutic assistance (pathogenetic, symptomatic treatment, treatment of complications).

MOTIVATION OF THE TOPIC RELEVANCE.

The kidneys play a major role in blood pressure regulation. The occurrence of hypertensive syndrome in kidney disease is due to sodium and water retention as a result of activation of the pressor (renin-angiotensin-aldosterone) system and a decrease in the function of the depressor (prostaglandin and kallikrein-kinin) systems. As a result, most kidney diseases are accompanied by hypertensive syndrome. In patients with end-stage renal failure, the incidence of arterial hypertension reaches 80-100%.

Determination of the level of training of students. The second level of knowledge: control methods - written survey (20 min.). Students should know the basic issues of etiology, pathogenesis, clinical picture and diagnosis of kidney and urinary tract diseases, highlight hypertensive syndrome. The main drugs used to treat kidney disease, their mechanisms of action; students should be proficient in propaedeutic skills.

Report of student curators in the chamber. When reporting a patient, students should pay attention to the main syndromes in the supervised patient, carry out differential diagnostics, i.e. to determine the nosological basis of the identified syndrome - arterial hypertension and its pathogenesis:

Retention of sodium and water

Renal dysfunction and decreased GFR are accompanied by a decrease in sodium and water excretion. The retention of sodium and water in most cases leads to an increase in the BCC, as well as to an increase in the sodium content in the vascular wall with its swelling and an increase in sensitivity to the pressor effects of angiotensin and catecholamines. Following the sodium retention, calcium accumulates in the vascular wall (in smooth muscle cells) with an increase in contractility and vascular tone, which leads to an increase in the general peripheral vascular resistance. This mechanism, with the leading role of overhydration, hypervolemia and increased cardiac output, is of primary importance in the development of arterial hypertension in OHN and chronic renal failure (especially in the terminal stage). It should be noted that the primary role of sodium and water retention leading to an increase in the volume of extracellular fluid and an increase in cardiac output, E.M. Tareev pointed out back in 1936, but this provision became generally recognized only in the 80s of the XX century.

Activation of pressor systems

The second mechanism causing the development of arterial hypertension in kidney disease is associated with the activation of the pressor system, including:

• renin-angiotensin-aldosterone system;

• sympathetic-adrenal system (which is judged by the content of serum catecholamines and their excretion in the urine);

• constrictor hormones - endothelin.

Renin is an enzyme produced in juxtaglomerular cells of the nephron. Under the action of renin, angiotensin 1 is formed from angiotensinogen (produced in the liver), which, under the influence of ACE, is converted into angiotensin II. The latter causes a systemic spasm of arterioles with an increase in OPSS, enhances sodium reabsorption (acting directly on the renal tubules, and also enhancing the secretion of aldosterone). Renin secretion is stimulated by a drop in pressure in the arterial system of the kidneys (blood loss, shock), hypovolemia, sodium deficiency in food, and taking diuretics. Aldosterone (a hormone of the adrenal glomerular zone) detains sodium, increasing its reabsorption in the collecting ducts, and increases the excretion of potassium. The release of aldosterone is regulated by the "renin - angiotensin" system and the concentration of Na + and K + ions. The pressor effect of aldosterone is associated with its effect on the cell membrane with an increase in its permeability to sodium. The accumulation of sodium in the walls of blood vessels leads to vasoconstriction.

Increased renin activity plays a role in the development of arterial hypertension in kidney diseases, characterized by the preservation of their function, but the presence of ischemia in the juxtaglomerular apparatus. This situation is most clearly expressed in renal artery stenosis. Renin-dependent hypertension is also observed in some patients in the terminal stage of renal failure. Carrying out hemodialysis in such patients does not reduce blood pressure, and only bilateral nephrectomy can lead to its decrease. Renin activity is increased in a number of patients with chronic glomerulonephritis, but this is not the only pathogenetic factor in arterial hypertension. Renin-dependent hypertension occurs with a significant increase in peripheral vascular resistance. At the same time, in patients with acute glomerulonephritis, renin activity is reduced and does not increase in response to the administration of diuretics. Increased secretion of aldosterone contributes to the development of arterial hypertension in all cases of activation of the renin-angiotensin system, as well as in hyperaldosteronism - primary (tumors of the glomerular zone of the adrenal cortex) and secondary. An increase in the activity of the sympathetic-adrenal system is associated with an increase in the formation of catecholamines (for example, with pheochromocytoma) or their delay in violation of the excretory function of the kidneys (for example, with chronic renal failure). Sodium retention increases the sensitivity of vascular wall receptors to the pressor effect of catecholamines. The role of catecholamines in the origin of arterial hypertension in kidney diseases is associated with vasoconstriction and increased peripheral vascular resistance, as well as with an increase in cardiac output.

Endothelial constrictor hormones also contribute to the development of arterial hypertension. When the vascular endothelium is damaged, the ratio between the mediator systems - vasoconstrictor (endothelin and thromboxane) and vasodilator (prostacyclin and nitric oxide) - shifts in favor of activation of vasoconstrictor components. In patients with diffuse kidney diseases, in violation of their function in the blood, an increased concentration of endothelin-l is found.

Reducing the influence of the depressor system

The depressor system that resists the action of pressor factors includes:

• prostaglandins;

• kallikrein-kinin system;

endothelial relaxing factor (nitric oxide - NO).

Prostaglandins reduce the tone of the arteries, reduce their response to vasopressor substances, and exhibit a powerful natriuretic and, consequently, diuretic effect. The end products of the kallikrein-kinin system, bradykinin and kallidin, also have pronounced vasodilating properties; excretion of kallikrein can serve as an indicator of vasodilation and activity of the natriuretic system. The defeat of the renal parenchyma leads to a decrease in the depressor function of the kidneys - a disorder of the "endothelin - nitric oxide" system, which contributes to the development of arterial hypertension due to a sharp increase in OPSS. Sodium retention in the body further enhances the imbalance of this system.

It should be emphasized once again that only in a few pathological conditions can we talk about one leading mechanism for the development of hypertensive syndrome. In most patients with chronic glomerulonephritis, hypertension has a mixed origin.

The development of hypertensive syndrome significantly worsens the prognosis of glomerulonephritis, as it is one of the

most important factors in the progression of the disease. Persistent arterial hypertension contributes to sclerotic changes in the vessels (arterioles) of the kidney with secondary activation of pressor mechanisms, as well as an increase in intraglomerular pressure with the subsequent development of glomerulosclerosis.

CLINICAL MANIFESTATIONS

The clinic of hypertensive syndrome in kidney diseases is determined by the degree of increase in blood pressure, the severity of damage to the heart and blood vessels. Patients complain of headache, blurred vision, pain in the heart, shortness of breath, with labile arterial hypertension (with hyperkinetic blood circulation) - fatigue, excitability, palpitations, less often headache. Malignant hypertensive syndrome is characterized by particularly high and persistent diastolic pressure, severe retinopathy (with foci of hemorrhage, edema of the optic nerve head, plasmorrhagia, often with reduced vision up to blindness), hypertensive encephalopathy, heart failure (first left ventricular, and then with congestion in the large circulatory system). In chronic renal failure, anemia also contributes to the development of heart failure. Compared with hypertension, complications (stroke, myocardial infarction) in a patient with renal hypertensive syndrome are less common.

With severe damage to the renal arteries, there is an increase in plasma levels of renin and aldosterone, a hyponatremic syndrome with hypokalemia, thirst, polyuria, and weight loss may develop.

Hypertensive crises (caused by the release of adrenaline), as a rule, are not frequent, they are manifested by a sharp headache, nausea, vomiting, visual impairment.

In the presence of a hypertensive syndrome, it is necessary to assess its severity and persistence, as well as (at least roughly) its hemodynamic variant. An approximate idea of the hemodynamic variant can give a measurement of the so-called basal pressure. When measuring blood pressure twice (lying and 5 minutes after being in a vertical position), a significant difference between the numbers (more than 20-30 mm Hg) indicates a hyperkinetic variant; the study is recommended to be carried out in a calm environment, in a warm room.

For differential diagnosis of high- and low-renin arterial hypertension, exclusion of vasorenal and high-renin arterial hypertension, a test with captopril is used. With the introduction of captopril (ACE inhibitor) to patients with high-renin arterial hypertension, a significant decrease in blood pressure is noted after 30-40 minutes; in patients with normo- or low-renin arterial hypertension, blood pressure does not change.

DIFFERENTIAL DIAGNOSIS

Renal hypertensive syndrome is observed in parenchymal diseases of the kidneys, damage to the renal vessels, as well as hyperproduction of aldosterone or catecholamines.

Hypertensive syndrome can be accompanied by almost all parenchymal kidney diseases. The hypertensive variant is observed in approximately 20% of patients with chronic glomerulonephritis. The disease proceeds with a moderate urinary syndrome (proteinuria usually does not exceed 1 g / day, erythrocyturia and cylindruria are minimal). Changes in urine are detected before registration of elevated blood pressure; the course is moderately progressive (10-year survival rate was 68%). The hypertensive variant of glomerulonephritis should be differentiated from hypertension and vasorenal arterial hypertension occurring in some patients with moderate urinary syndrome (ischemic nephropathy).

The hypertensive syndrome that does not come to the fore in the clinical picture is found in some patients with latent glomerulonephritis.

When a pronounced nephrotic syndrome is combined with severe hypertension, one should think about mixed CGN or RPGN (the latter is favored by the rapid deterioration of renal functions). In chronic interstitial nephritis, malignant arterial hypertension occurs in 25-30% of patients.

Hypertensive syndrome is characteristic of AGN, it is also often observed in chronic pyelonephritis. Chronic pyelonephritis can often occur with hypokalemia, which, in the presence of elevated blood pressure, forces differential diagnosis with primary hyperaldosteronism.

Persistent arterial hypertension, often malignant, is characteristic of polyarteritis nodosa, systemic scleroderma ("scleroderma kidney"), diabetic kidney; it is also observed in SLE, hemorrhagic vasculitis (purpura) of Schönlein-Genoch, gouty kidney, etc. With amyloidosis of the kidneys, persistent hypertensive syndrome is noted relatively rarely.

The causes of vasorenal arterial hypertension in 85-90% of cases are atherosclerosis of the vessels of the kidneys and fibromuscular hyperplasia. Much less often, the cause of vasorenal arterial hypertension is an aneurysm of the renal artery, aortoarteritis, embolism or thrombosis of the renal artery, and anomalies in the location of the vessels.

Atherosclerosis usually affects the proximal third of the renal artery, near the aorta; more often it develops in men older than 40 years. Fibromuscular hyperplasia is characterized by areas of fibrous and muscular hyperplasia, alternating with areas of media destruction. The middle and distal sections are affected more often. Most often women aged 20-40 years get sick. Aortoarteriitis often affects both the thoracic and abdominal aorta; young women get sick more often.

Vasorenal arterial hypertension should be considered in the presence of a pronounced increase in blood pressure, especially in diastolic or malignant arterial hypertension, resistant to ongoing standard antihypertensive therapy, in patients without a clear clinical picture of CGN or chronic pyelonephritis. An important clinical sign is considered systolic murmur (and sometimes diastolic), auscultated in the area of the projection of the renal arteries in 50% of patients. With atherosclerosis of the renal arteries, the noise is better heard in the midline above the navel, in the epigastric region (listen without pressing with a stethoscope); in fibromuscular hyperplasia, the murmur is heard slightly lateral and superior to the umbilicus. Sometimes the noise is heard better from the back. The sign is not absolute, since sometimes abdominal noise can be heard in patients without stenosis of the renal artery. Another clinical sign that makes one suspect vasorenal hypertension is the asymmetry of blood pressure in the extremities (with atherosclerosis or aortoarteritis).

The first stages of an additional examination in case of suspected vasorenal arterial hypertension include radioisotope renography, then excretory urology.

More convincing are the results of aortography and selective renal arteriography, which make it possible to identify the site of the lesion and its extent. Of great diagnostic value are new methods of examination - CT, MRI, color duplex sonography.

Additional diagnostic methods include determining the activity of renin in the blood plasma, as well as in the renal veins (increase on the side of stenosis); this study is especially informative when it is carried out before and after the administration of captopril. The results are more accurate with an additional (repeated) study of kidney function after taking 25-50 mg of captopril.Diagnosis of vasorenal arterial hypertension is important, since surgical treatment is possible (prosthesis of a stenotic area or elimination of stenosis) or intra-arterial expansion with a balloon catheter.It should be remembered that ACE inhibitors are contraindicated in patients with bilateral renal artery stenosis.

Particular attention has recently been paid to atherosclerosis as a cause of kidney damage, called ischemic nephropathy, leading to CRF in a fairly large number of patients.Clinically, ischemic nephropathy should be suspected if the patient has coronary artery disease or diseases (atherosclerotic) of peripheral vessels (in history or at the time of examination), noises heard during auscultation in the projection of the main arteries, inexplicable by other reasons, an increase in serum creatinine, often with minimal changes (or no changes) in a routine urinalysis, a significant increase in serum creatinine levels during treatment with ACE inhibitors. It is important to always keep these signs in mind during dynamic monitoring of patients, but especially elderly patients and people suffering from widespread atherosclerosis.Special attention should be paid to the importance of auscultation of the main arteries in these patients, as well as in elderly people with arterial hypertension - the detection of noise makes it possible to assume with a high probability the vasorenal nature of arterial hypertension and nephrosclerosis, which is also confirmed by Doppler ultrasound.Arterial hypertension associated with hyperproduction of pressor substances (endocrine hypertension) is observed in primary hyperaldosteronism and pheochromocytoma.Conducting classes in a thematic classroom. Analysis of the features of etiology, pathogenesis, clinic, diagnosis, treatment of a particular patient. Students should pay special attention to the main syndromes in the supervised patient, to conduct differential diagnostics, i.e. to determine the nosological basis of the identified arterial hypertension syndrome. Indicate the main methods of non-drug exposure (changing lifestyle, nutrition, giving up bad habits, doing physiotherapy exercises). The main groups of drugs and their mechanisms of action, the main indications and contraindications for use and the rationale for choosing a particular drug from pharmacological groups.The final part of the lesson: control of the acquired knowledge - solving situational problems without possible options for correct answers.

Summary.