

СЛОЖНЫЙ СЛУЧАЙ ДИАГНОСТИКИ СИНДРОМА КОННА

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Первичный гиперальдостеронизм, известный также как синдром Конна, является редко диагностируемым заболеванием, зачастую протекающим под «маской» ишемической болезни сердца и первичной артериальной гипертензии (АГ). Тем не менее, распространенность данной патологии среди всех пациентов с АГ составляет почти 17%. В свою очередь, отсутствие специфических клинических проявлений затрудняет своевременную и правильную его диагностику, что приводит к возникновению серьезных осложнений. В данной статье описывается клинический случай синдрома Конна и особенность его диагностики.

Ключевые слова: синдром Конна; первичный гиперальдостеронизм; вторичные артериальные гипертензии; гипокалиемия.

A COMPLEX CASE OF DIAGNOSIS OF CONN'S SYNDROME

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The primary hyperaldosteronism also known as Conn's syndrome, is a rarely diagnosed disease that commonly runs under a 'mask' of ischemic heart disease and the primary arterial hypertension (AH). Nevertheless, the incidence of the given pathology among all patients with AH makes almost 17%. On the other hand, the absence of specific clinical manifestations of the disease makes its timely and correct diagnosis difficult which is fraught with serious complications. In the article a clinical case of Conn's syndrome and peculiarities of its diagnosis are described.

Keywords: Conn's syndrome; primary hyperaldosteronism; secondary arterial hypertension; hypokalemia.

Symptomatic arterial hypertension (AH) play a significant role in development of resistant forms of elevated arterial pressure (AP). Today, several etiologic factors are known leading to such conditions. One of the most common, but rarely identified conditions is primary hyperaldosteronism – Conn's syndrome.

The incidence of primary hyperaldosteronism (PHA), according to literature is

about 17%, here, 10% of cases of resistant AH are associated just with this pathology [1-3]. Conn's syndrome develops at the age of 30-60 years, women affected 3 times more often than men. A peculiarity of this disease is absence of specific symptoms that tremendously impedes the diagnosis [4,5].

The incidence of AH among such patients is almost 100%. Other symptoms such as hypokalemia, muscle weakness,



polyuria and polydipsia are observed in 50-70% of cases of this disease [1-5]. As an example a clinical case of diagnosis of this syndrome is given.

Clinical example. A female patient S, of 55 years of age, was admitted to the cardiology department with complaints of dizziness, muscle weakness, fast fatigue, sensations of palpitations and discomfort in the heart region.

Case history: According to the data of the outpatient card, within the previous ten years a significant elevation of AP has been observed in the patient with maximal figures reaching 300/160 mm Hg. The patient underwent several examinations and was on treatment in therapeutic and cardiologic hospitals with the diagnosis: II stage, 3 degree essential hypertension. The conducted therapy gave unstable effect. The patient was troubled by pronounced weakness which made her leave the job. In March 2019 the patient started to feel frequent palpitations and retrosternal discomfort. In ECG depression of ST segment was identified, and the patient was hospitalized to the cardiology department with suspicion on unstable angina.

On the second day of stay in the hospital episodes of brief loss of consciousness and weakness in the extremities appeared. After examination by a neuropathologist the patient was transferred to the neurology department with the diagnosis of ischemic stroke.

On top of therapy administered at neurology department, the patient developed paroxysm of ventricular fibrillation with drop of AP and loss of consciousness. In result of successive resuscitation measures the sinus rhythm recovered.

On examination at resuscitation department clinical blood analysis showed leukocytosis up to $17 \cdot 10^9/l$, ESR 26 mm/h. Biochemical analysis of blood showed increased transaminases (AsAT – 457 U/l, AlAt – 231 U/l), urea – up to 12.1 mmol/l, which normalized within a week. Electrolyte imbalance was also noted: reduction of potassium

to 1.48 mmol/l of 135-150 mmol/l), which rapidly leveled out after intravenous introduction of potassium. After stabilization of the condition, the patient was transferred to the department of emergency cardiology.

On examination: General condition moderately severe. Skin of normal color, warm. Peripheral lymph nodes not enlarged, non-palpable. Thyroid gland non-palpable. Respiratory rate 16 per minute. In percussion vesicular resonance was determined over lungs, in auscultation vesicular breathing was heard, additional breathing sounds and crackles were absent. Heart boundaries: the right boundary went along the right edge of the sternum, the upper boundary – along III rib, the left boundary – 1 cm outward from the left mid-clavicular line. The heart activity rhythmic, heart rate about 75 beats/min, heart sounds deadened. II sound accentuated on the aortic valve. AP 180/90 mm Hg. The abdomen soft, painless, the liver near the edge of the costal margin, kidneys and spleen non-palpable. No peripheral edema.

So, the first step in diagnosis was determination of the cause of high arterial pressure resistant to treatment. Since the history data (increase in the systolic AP to 300 mm Hg, resistance to therapy, negative hereditary history for AH) suggested just secondary AH, first of all it was necessary to exclude pathology of organs that might lead to symptomatic increase in AP.

First of all, pathology of the aorta should be considered, since coarctation of the aorta and aortic stenosis are accompanied by elevation of the pressure in systemic circulation [1]. The key diagnostic method was echocardiographic examination (EchoCG).

EchoCG data: minimal mitral insufficiency, I degree tricuspid insufficiency. An area of hypertrophy in the region of the middle third of the interventricular septum. Disorder of the diastolic function of the left ventricle (LV). Satisfactory contractility of LV, ΔS -37%, stroke volume 82 mm, ejection fraction of LV 66%. The aorta without pecu-

liarities. Pressure in the pulmonary artery 26 mm Hg. Thus, by the results of EchoCG, the role of the aorta in arterial hypertension could be excluded.

The next step in diagnosis was exclusion of the so called 'renal hypertension'. 50-70% Of all secondary hypertension are known to result from diseases of kidney [1,2]. The history of such patients usually contains an indication of a past or present at the moment renal disease. The functional condition of kidney is reflected by the level of urea and clearance of endogenous creatinine. Urine test did not show any stable changes. Renal tests exhibited a mild elevation of urea – 12.1 mmol/l and of creatinine – 135.7 μ mol/l that could not be the reason for such a high level of AP. Thus renoparenchymal AH was also excluded.

In auscultation in the region of projection of renal arteries no murmurs were heard which permitted to exclude vasorenal AH.

Thyrotoxicosis and hypothyroidism also run with elevation of AP in 50% of cases [1]. In these cases symptoms of the main disease are always present with a change in the parameters of thyroid hormones. In our patient no pathological changes on the part of thyroid were found.

At last, it should be noted that, *besides significant AH, a pronounced hypokalemia* was identified in the patient, which accounts for muscle weakness, and also *polyuria and polydipsia* in this category of patients. The cause for polyuria and polydipsia is development of hypokalemic tubular nephropathy, dystrophy of tubules and reduction of sensitivity to antidiuretic hormone.

On admission to the department, characteristic *signs of hypokalemia* were found in the ECG (Figure 1): *depression of ST segment, negative T wave, increase in the amplitude of U wave, prolongation of QT interval.*

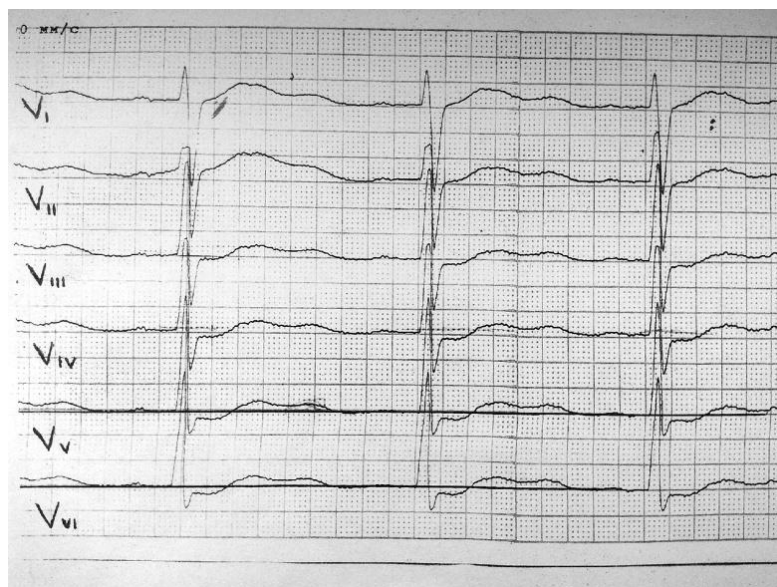


Fig. 1. V1-V6 leads of ECG of female patient S. with evident hypokalemia

From the history, no other etiology of hypokalemia was found – the patient did not take drugs leading to hypokalemia, vomiting and diarrhea were also absent. Thus hypokalemia could be most likely attributed to PHA. Aldosterone induces reabsorption of sodium in exchange for potassium and hydrogen ions

in the distal part of nephron. Increased secretion of this mineralocorticoid can induce hypertension with hypokalemia [2,3].

So the blood test was conducted for determination of the level of aldosterone which concentration was found to be 369.21 pg/ml against the norm 80.14 pg/ml.

The next diagnostic task was to elicit the cause of that high parameters of aldosterone. Increased secretion of this mineralocorticoid may be secondary to reduction of the level of fluid in the body [1,3] that leads to activation of renin-angiotensin system and stimulation of secretion of aldosterone; however, there were no clinical signs of insufficient amount of fluid in the patient. Besides, potassium could not be considered an acting factor in stimulation of aldosterone secretion since the patient had evident hypokalemia.

Absence of 'cushingoid' facial traits, of central type of obesity, normal body weight and optimal blood glucose level permitted to exclude any influence of excess adrenocorticotrophic hormone on the given process [2,3]. The patient did not take drugs possessing mineralocorticoid effect and did not use food stuffs containing licorice that is capable of inducing hypokalemia and AH.

Hypersecretion of aldosterone was most likely an independent process. The patient was sent to computed tomography (CT) of the abdominal organs and of the retroperitoneal space.

The results of *multi-slice CT of the abdominal cavity, of retroperitoneal space with preliminary per os contrasting*. The left adrenal was enlarged due to some additional formation of rounded shape originating from the left peduncle. The right adrenal of usual shape, no additional formations determined. Other organs of the abdominal cavity and of retroperitoneal space without changes.

In the given case the formation should be differentiated between pheochromocytoma and other tumors of adrenals. The diagnosis of pheochromocytoma could be rejected or confirmed by biochemical data – high content of catecholamines in blood [4] that was not observed in the patient.

In result, on the basis of the history data and the data of clinical examinations, the preliminary diagnosis was made: *Aldosteroma*, and PHA was suspected.

One of criteria that confirms PHA is the level of activity of renin in blood plasma [3,5].

In the given pathological condition it will be rather low. In turn, in the secondary hyperaldosteronism the opposite situation is observed – very high level of renin activity. However, it should be borne in mind that this examination is not always informative, therefore it is recommended that the ratio of the content of aldosterone to the level of activity of renin should be checked [3-5]. Because of some technical difficulties, this examination was not conducted in our patient. In parallel with determination of the activity of renin, additional diagnostic tests with sodium chloride and captopril should be conducted in all patients.

Thus, taking into account complaints, the data of the objective method and of additional methods of examination, the *final clinical diagnosis* was made:

Main disease: Aldosteroma. Conn's syndrome (primary hyperaldosteronism). Arterial hypertension, III stage 3^d degree, very high risk.

Complications of the main disease: Ischemic stroke in the right middle cerebral artery circulation with a mild paresis of the left arm. Paroxysm of ventricular fibrillation.

Due to this, the patient was administered the following medicinal treatment in the department: spironolactone 200 mg 3 times a day, Lisinopril 10 mg twice a day, amlodipine 5 mg once a day, atorvastatin 20 mg once a day, cardiomagnyl 75 mg once a day.

With conducted therapy, the condition of the patient improved: with stabilization of the electrolyte parameters of blood, the general weakness decreased, no episodes of loss of consciousness were noted, AP stabilized at 130/80 mm Hg. In 6 months the surgical treatment for adrenal tumor was recommended in the conditions of urology department.

Conclusion

It should be borne in mind that the given pathology may run under the mask of ischemic heart disease and of essential hypertension which creates difficulties in diagnostic search. The correct approach and clear order in the doctor's actions in management of

such patients will permit to make timely and correct diagnosis which in turn will provide

maximally effective therapy and will reduce the risks of development of complications.

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