Устойчивая желудочковая тахикардия у пациента с единственным желудочком сердца и феохромоцитомой

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Феохромоцитома – редкая опухоль, развивающаяся из адреномедуллярных хромаффинных клеток и продуцирующая один или несколько катехоламинов (адреналин, норадреналин, дофамин), редко – гормонально неактивная. Цианотические пороки сердца также являются относительно редкой патологией, и одним из наиболее редких их вариантов является единственный желудочек сердца. Предположительно, у пациентов с цианотическими пороками сердца частота встречаемости феохромоцитом и параганглиом может быть выше вследствие наличия определенных герминативных и соматических мутаций. При цианотических пороках сердца развитие злокачественных аритмий является одной из частых причин смерти. Казуистически редко сочетание феохромоцитом с единственным левым желудочком сердца: в литературе описано всего восемь таких случаев. Нами описан уникальный случай сочетания единственного левого желудочка сердца, феохромоцитомы и устойчивой желудочковой тахикардии у молодого пациента. Причиной желудочковой тахикардии, по всей видимости, в данном случае являлась неадекватная медикаментозная терапия (назначение верапамила). Проведение хирургического лечения феохромоцитомы и направление пациента на кардиохирургическое лечение стало возможно только после коррекции антигипертензивной и антиаритмической терапии: замены верапамила на комбинацию доксазозина и амиодарона с достижением синусового ритма и относительно удовлетворительных показателей АД.

Ключевые слова: клинический случай, феохромоцитома, единственный левый желудочек сердца, желудочковая тахикардия.

Sustained ventricular tachycardia in a patient with a single ventricle of the heart and a pheochromocytoma

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A pheochromocytoma is a rare tumor that develops from adrenomedullary chromaffin cells and produce ones or more catecholamines, including adrenaline, norepinephrine, and dopamine. On rare occasions a pheochromocytoma is hormonally inactive. Cyanotic heart disease is also a relatively rare pathology. One of its least frequently occurring variants is the single ventricle of the heart. Presumably, in patients with cyanotic heart defects, the occurrence of pheochromocytes and paragangliomas will be higher due to the presence of certain germinative and somatic mutations. In cyanotic heart defects, the development of malignant arrythmias is one of the frequent causes of death. A combination of a pheochromocytoma with a single ventricle of the heart is extremely rare: only eight such cases have been described in the literature. This article describes a young patient with a unique case of a single ventricle of the heart, pheochromocytoma and sustained ventricular tachycardia. The cause of the ventricular tachycardia, in all likelihood, was inappropriate medical care – in this case, a prescription for verapamil. The surgical excision of the pheochromocytoma and the referral of the patient for cardiac surgery became possible only after correcting the antihypertensive and antiarrhythmic therapy. Verapamil was replaced with a combination of doxazosin and amiodarone, resulting in relatively satisfactory blood pressure readings and sinus rhythm.

Keywords: case report, pheochromocytoma, single ventricle of the heart, ventricular tachycardia.

Background

Cyanotic cardiac diseases (CCD) are a subgroup of congenital disorders of the cardiac anatomy that manifest shortly after birth by signs such as systemic hypoxia and hypoxemia, which, in turn, cause intracardiac hemodynamic disorders. The incidence rate of congenital heart defects among newborns is 7.05 per 1,000 newborns in Europeans and 13.08 per 1,000 newborns in Asians. In adults, congenital heart defects occur with a frequency of 4.09 cases per 1,000 individuals [1, 2].

Pheochromocytoma (PC) is a tumor that develops from adrenomedullary chromaffin cells, producing one or more catecholamines (adrenaline, norepinephrine, and dopamine), and is rarely hormonally inactive [3]. The incidence of PC in patients with arterial hypertension or adrenal incidentalomas is 0.1% and 4%, respectively [4]. The prevalence of PC in the general population is estimated to be ⩽1 per 100,000 individuals per year [5, 6].

Among the CCD, one of the rarest is the presence of only a single ventricle of the heart. According to a study by Coats et al. (2015), this defect occurs in 16 per 100,000 newborns and 2 per 100,000 adults [7]. Its combination with PC is even rarer, with only eight such cases described in the literature [8-14].

Remarkably, 70%–80% of patients with PCs and paragangliomas (derivatives of nonadrenal chromaffin cells of the neural crest) have a certain combination of specif-

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ic germinal and somatic mutations. According to the Cancer Genome Atlas, the genes involved in these mutation complexes are divided into three clusters, namely a pseudohypoxia group, including *SDHA, SDHB, SDHC, SDHD, SDHAF2, VHL,* and *HIF2*α; a group of Wnt-signaling pathways (*CSDE1* and *MAML3*); and a group of kinase signaling pathways (*RET, NF1, TMEM127, MAX*, and *HRAS*). Mutations in genes of the pseudohypoxia group are associated with dysregulation of the factor induced by hypoxia (HIF), an oxygen-dependent factor that is involved in the cellular and tissue response to hypoxia (comprising neoangiogenesis, modulation of apoptosis, proliferative reactions, and changes in the levels of cell metabolism) [15].

These genetic data enable us to take a second look at many classical works devoted to tissue and cellular changes under true hypoxic conditions. For example, carotid sinus glomal cells as well as chromaffin cells of the adrenal glands during the postnatal period are known to be sensitive to oxygen levels in the blood. Moreover, chronic hypoxemia leads to hyperplasia of the carotid sinus, and people living in highlands have a higher risk of developing PC and PG than those living in lowlands [16]. Furthermore, the mitochondrial structure (the main organelle involved in cell respiration) is disturbed in PC and paraganglioma cells, and a previous study described a case of paraganglioma regression after achieving remission of chronic hypoxia. Almost 25% (5/21) of patients with PC diagnosed at Johns Hopkins University from 1901 to 1962 had CCDs, and several clinical cases have been published that describe a combination of these pathologies. Moreover, based on autopsy findings, the incidence rate of congenital heart defects with peripheral tumors from cells derived from the neural crest is significantly higher than the average of the population [16].

Accordingly, it was suggested that the incidence of PCs and paragangliomas may be higher in patients with CCD [16].

Despite a casuistically rare combination of cyanotic heart defects and PCs, Opotowsky et al. (2015) reported that the incidence of PCs and paragangliomas in patients hospitalized due to CCD was significantly higher than in other patients [17]. Furthermore, this difference did not correlate with other known risk factors for developing tumors from chromaffin cells (the study was conducted retrospectively based on data from the American database Nationwide Inpatient Sample, and almost 17,000 cases were analyzed) [17].

A retrospective study by George et al. [18] also showed a significantly higher incidence of heart defects in patients with neuroblastomas compared with patients with leukemia (20% and 3.6%, respectively). However, evidence for a relationship between chronic hypoxia and the development of PCs and paragangliomas remains uncertain.

Recently, the survival rate of patients with congenital CCD has continued to increase as a result of the development of surgical care for these patients and, possibly, through this process, data that enable validation of the above patterns will be acquired [16].

Many cases of ventricular tachycardia (VT) in patients with PC have been described in the literature [19-25]. Stenstorm et al. (1988) reported 31 patients with PC in whom the average QTc interval was normal at the time of diagnosis (440±40 ms), somewhat shortened after administration of phenoxybenzamine (430±20 ms), and decreased to 410±20 ms after surgical treatment. Moreover, in 35% of patients, the QTc was more than 440 ms at the time of diagnosis, and these phenomena persisted postoperatively in only two patients [25]. Another study states that an increase in the QT interval is also found in 35% of patients with PC; however, in isolated cases this leads to the formation of ventricular rhythm disorders [26].

In CCD, the development of malignant arrhythmias is one of the common causes of death. In a major multicenter study of patients with various forms of congenital heart defects, arrhythmias were the cause of 14% of lethal outcomes; in the vast majority of cases, the cause of death was ventricular fibrillation [27].

The mortality rate of patients with a history of Fontan surgery for the formation of a total cavopulmonary junction is due to sudden cardiac death in only 9.2% of cases (in particular, ventricular arrhythmia). This indicator is significantly inferior to the mortality rate after the Fontan surgery itself (68%) and is comparable to mortality from chronic heart failure (CHF) (6.6%) and thromboembolism (7.2%) [28]. To the best of our knowledge, there are no epidemiological data on the frequency of monomorphic VT in patients with a single left ventricle of the heart.

Therefore, the patient described in this article is of particular interest because of the rarity of the combination of his pathologies. The case presented herein is characterized by the presence of recurrent VT and is compared to all patients described previously in the literature.

Case description

Patient *D.*, 21 years old, born and living in the mountainous regions of the Caucasus northeastern part, was hospitalized at the National Medical Endocrinology Research Center to determine the hormonal activity of incidentaloma of the right adrenal gland (detected by ultrasound in a random examination at a primary care facility). Upon admission, he complained of shortness of breath, dizziness, increased blood pressure (BP) of 170/100 mmHg, and palpitations. The anamnesis revealed that soon after birth, a congenital heart defect was revealed in the form of a double-inlet single anatomically left ventricle, combined pulmonary stenosis, an extra superior vena cava draining into the coronary sinus, and aortopulmonary collaterals from the descending aorta to both lungs. At the age of 1 year, the patient underwent bidirectional cavopulmonary anastomosis. In 2016, Fontan surgery was recommended due to the formation of anas-

tomosis between the inferior vena cava and pulmonary arteries. In 2017, in preparation for surgery, an asymptomatic increase in BP up to 220/110 mm Hg was detected, and an adrenal ultrasound revealed a massive formation of the right adrenal gland of 46 × 36 mm with clear contours. The level of blood cortisol was 464.2 nmol/l; no further examinations have been conducted. During Holter ECG monitoring, atrial pacemaker migration, supraventricular tachycardia runs, and idioventricular rhythm episodes with a heart rate of 60–70/min were recorded; as a result, the patient was referred to inpatient examination at the National Medical Endocrinology Research Center. Two weeks before hospitalization, to reduce the likelihood of erroneous examination results and determine the hormonal activity of the volumetric formation of the adrenal gland, beta-blocker therapy was canceled. In the period of hospitalization, the patient received verapamil 240 mg per day, doxazosin 0.2 mg per day, and his BP was monitored irregularly.

Results of physical, laboratory, and instrumental studies

Upon admission on the ECG, VT was registered **(Fig. 1)**, which was accompanied by arterial hypotension up to 90/70 mmHg.

Tachycardia lasted from several minutes to 1 hour, stopped spontaneously, but recurred again. To control the rhythm, verapamil was discontinued. Amiodarone in saturating doses (200 mg twice a day) and beta-blocking agents (metoprolol succinate 12.5 mg twice a day, followed by an increase to 25 mg twice a day) were prescribed orally, and therapy with alpha-adrenoblocker administration was continued (doxazosin 6 mg twice a day). Within 2 days, a steady sinus rhythm was achieved; at the same time, his BP increased to 200/100 mmHg; as a result, a prolonged form of nifedipine 30 mg was administered at night, as well as ACE inhibitors (enalapril 5 mg 2 twice a day).

Clinical and laboratory examination

Echocardiography **(Fig. 2)** revealed II-III degree mitral and tricuspid regurgitation as well as a functioning oval window with a diameter of 4–5 mm, a single double-inlet left ventricle, and a left ventricular ejection fraction of 75%.

Computed tomography revealed the formation of the right adrenal gland with clear and even contours, dimensions of 39×37×43 mm^3, and a native density of 44 HU.

Daily urinalysis revealed an isolated increase in normetanephrines to 15,367 mcg/day (35-445). Laboratory data suggesting hyperaldosteronism and hypercorticoidism were not obtained.

Differential diagnosis

To rule out multiple endocrine neoplasia, an ultrasound of the thyroid and parathyroid glands was performed. No additional formations were detected, and functional disorders of the thyroid gland were also ruled out; thus, PC was diagnosed.

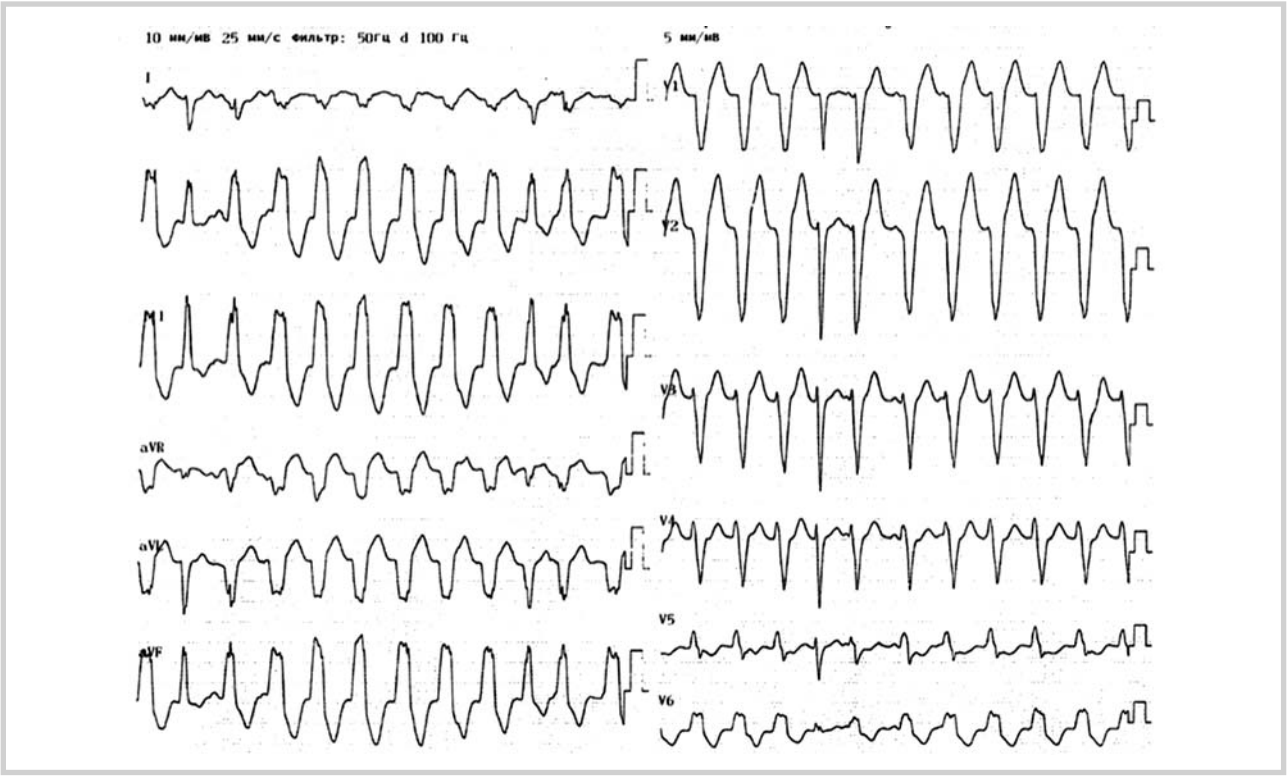

Fig. 1. **Electrocardiogram on admission. Monomorphic ventricular tachycardia is recorded.**

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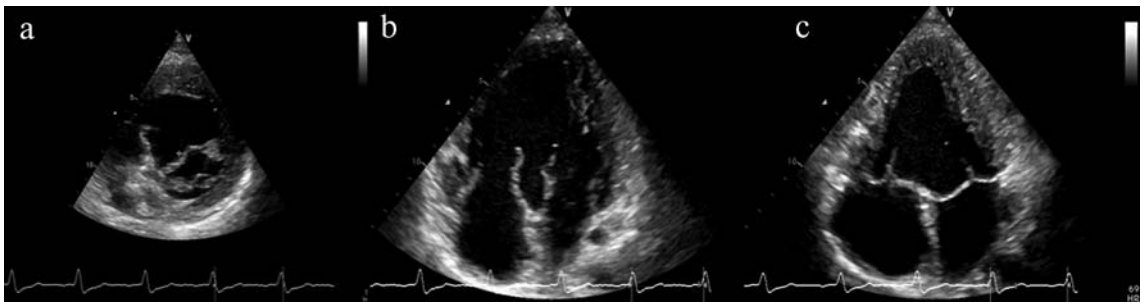

Fig. 2. **Echocardiography data.**
a – short parasternal position at the level of the mitral valve; b – apical 4-chamber position, diastole; c – apical 4-chamber position, systole.

Treatment

The results of an additional examination of the space-occupying lesion of the right adrenal gland facilitated diagnosis of PC; as a result, doxazosin was prescribed at an initial dose of 6 mg per day, and surgical treatment of right-sided adrenalectomy was recommended.

Additionally, considering the frequent recurrence of VT and heart failure, therapy with amiodarone (400 mg per day) and metoprolol (50 mg per day) was continued. To achieve optimal BP, enalapril 10 mg per day and nifedipine 30 mg per day were prescribed.

Outcome and results of the follow-up

After 5 months, the patient was hospitalized again at the National Medical Endocrinology Research Center. The patient adhered to the therapy prescribed previously, and the doxazosin dose was increased to 16 mg per day. As a result, BP indicators were achieved in the range of 170/100–100/70 mmHg. On ECG series, sinus tachycardia with a heart rate of 90–100/min was recorded, subjective complaints of cardiac arrhythmias were not present, and the patient did not experience syncopal or presyncopal conditions.

At the time of hospitalization, CHF symptoms were moderate, at a level of functional class 2, according to the NYHA. No ventricular arrhythmias were detected by Holter ECG monitoring.

Considering the persistent increase in blood levels of potassium (up to 5.8 mmol/L) and creatinine (up to 123 μmol/L, eGFR according to CKD-EPI 72 ml/min/1.73 m^2), ACE inhibitors were discontinued. Because of the high resting heart rate of 90–100 beats per min, the metoprolol succinate dose was increased to 50 mg per day. The calcium channel blocker (nifedipine) was withdrawn owing to the optimal control of BP during therapy with alpha and beta adrenoceptor blocking agents, and amiodarone therapy was continued.

The patient underwent laparoscopic right-sided adrenalectomy at the surgery department at the National Medical Endocrinology Research Center. The histological material obtained corresponded to alveolar type PC of histological structure with fibrous degeneration at the center. During the postoperative period, no abnormalities and no relapse of VT were noted, and BP was optimally controlled. The patient was discharged on day 10 after surgery and was referred to cardiac surgeons for surgical treatment of heart disease.

Discussion

Despite the extremely rare combination of PC and congenital heart defects, such patients can be found in the clinical practice of cardiologists, endocrinologists, or surgeons.

The treatment of such patients requires a particularly careful approach because some drugs prescribed routinely for one pathology may be contraindicated in the presence of concomitant diseases. In the present patient (with a single ventricle of the heart, CHF, and PC), it was most likely the prescription of verapamil to the patient along with morphological changes in the myocardium and the withdrawal of beta-blockers that led to the development of stable VT. Cases of VT initiated by verapamil have already been described in the literature [29]. Following the withdrawal of verapamil and large doses of alpha- and beta- adrenoceptor blocking agents and amiodarone, it was possible to achieve normalization of the heart rhythm, which facilitated surgical treatment of PC to be performed and further stages of the congenital heart disease treatment to be planned.

It is possible that the lengthening of the QT interval with PCs (with high concentrations of blood catecholamines) in some cases can be regarded as a manifestation of the elongated QT syndrome of the first type, which is a condition associated with the simultaneous presence of two populations of potassium channels in the myocardium: dysfunctional and intact. This type of syndrome is characterized by a paradoxical response to the administration of catecholamines such that the QT lengthens by more than 30 ms, whereas in healthy individuals, with an increase in blood levels of catecholamines, QT is shortened [30, 31].

Notably, in addition to the prescription of verapamil, both the weakness of the sinus node and the withdrawal of beta-blockers in a patient with PC could contribute to the development of VT.

In our opinion, urgent surgical treatment of VT in a patient with CCD and PC was not justified and should only be considered if antiarrhythmic therapy is ineffective after normalization of catecholamines. Furthermore, the implantation of a cardioverter defibrillator was not recommended because the cause of VT development was most likely reversible. However, the frequency of development of life-threatening cardiac arrhythmias in patients with PC remains unclear and requires further research.

Conclusion

This clinical case describes a complex combination of potentially life-threatening diseases, namely a single left ventricle of the heart and PC in a young patient. The prescription of a non- dihydropyridine calcium blocker (verapamil) led to the development of recurrent stable VT. Surgical treatment of PC and referral to cardiac surgery was only possible after the adjustment of antihypertensive and antiarrhythmic therapy, namely replacing verapamil with a combination of doxazosin and amiodarone and achieving sinus rhythm and relatively satisfactory BP values.

Additional information

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