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Surgical Correction of an Atricular Border Defect in Adults

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Abstract: Interatrial septal defect is the most common heart defect diagnosed in adults. Its frequency is 5-15% of all congenital heart defects. It is precisely in the older age group that congenital malformations are often combined with complex rhythm disturbances, which not only worsens the quality of life of patients but also significantly exacerbates the natural course of the disease, leads to rapid decompensation of blood circulation, and is a risk factor that increases patient mortality. Timely diagnosis and treatment of the disease play a significant role in preventing the development of life-threatening complications. Studying the problem of atrial septal defect in adults is crucial for doctors of all specialties, as this pathology ranks first among congenital heart defects in terms of prevalence.

Keywords: atrial septal defect in adults, late diagnosis, surgical tactics.

Introduction. Atrial septal defect is the most common congenital heart defect diagnosed in adults. It is precisely in the older age group that congenital defects often combine with rhythm disturbances, which not only worsens the quality of life of patients but also significantly exacerbates the natural course of the disease, leading to rapid decompensation of blood circulation. Additionally, it is a risk factor that increases mortality in DMPP [1,2,6,9,12,14]. Timely surgical correction of the atrial septal defect in childhood and adolescence leads to the normalization of intracardiac hemodynamics, often preventing the development of arrhythmias in "adult life." The results of surgical correction of combined pathology, BPH, and arrhythmias, as well as optimal diagnostic methods and treatment approaches, remain relevant to date [3,5,13,20].

A congenital heart defect, in which there is a hole between the right and left atria, is called a defect of the interatrial septum. Through it, blood from the left parts of the heart is pumped into the right parts, leading to overload of the right parts and the pulmonary circulation. Symptomatology, course, and prognosis may vary depending on the severity. Studying the problem of atrial septal defect is crucial for doctors of all specialties, as this pathology ranks second in prevalence among congenital heart defects [7,8,16,19].

Scientists do not specify the exact cause of the atrial septal defect, but they note the genetic and external factors that cause it. Causes of atrial septal defect The formation of the defect is associated with underdevelopment of the primary or secondary atrial septum and endocardial valves during the embryonic period. Genetic, physical, ecological, and infectious factors can lead to organogenesis disruption. The risk of developing an atrial septal defect in the unborn child is significantly higher in families where relatives have congenital heart defects. Cases of familial atrioventricular septal defects combined with atrioventricular block have been described. In addition to hereditary predisposition, viral diseases of the pregnant woman (pox, rubella, herpes, syphilis, etc.), diabetes mellitus and other endocrinopathies, taking certain medications and alcohol during pregnancy, industrial hazards, ionizing radiation, and gestational complications can lead to the occurrence of DMPP [9,15,17].

The clinical picture of DMPD appears already in childhood, if there is a large arteriovenous discharge of blood through the defect. The disease can be combined with other abnormalities, which are often attributed to chromosomal mutations. The following symptoms are characteristic of the primary form of BCC: wet cough; swelling of the extremities; chest pain; bluish skin of the hands, feet, and nasolabial triangle. The symptoms of congenital heart disease depend on the patient's age, the size of the pathological opening, and the presence of other heart defects. With a large pathological opening,

pulmonary hypertension develops, characterized by elevated blood pressure in the pulmonary circulation [4,10,16].

In some cases, severe irreversible hypertension of the pulmonary circulation - Eisenmenger syndrome develops. One can name several other consequences of interatrial septal defect without treatment: heart failure; atrial fibrillation; stroke risk; high mortality. Patients with mildly expressed defects live up to 50 years, and some live longer. In adults, PE is diagnosed, more often by the age of 30, when symptoms of the disease appear. According to statistics, without treatment, approximately 50% of patients with moderate and large septal defects survive to 40-50 years. During a regular examination by a physician, therapist, or cardiologist, it is practically impossible to detect a septal defect, as pathological murmurs in the heart are often absent. Auscultation to the left of the sternum reveals a moderately intense systolic murmur in the II-III intercostal spaces, which, unlike a ventricular septal defect or pulmonary artery stenosis, is never harsh. Above the pulmonary artery, the cleavage of the second tone and the accentuation of its pulmonary component are observed. Auscultatory data are confirmed when performing phonocardiography. The pathology of the interatrial septum is established based on examination results, instrumental, and laboratory data. In secondary atrial septal defects in adult patients, ECG changes reflect the overload of the right heart chambers. Incomplete blockage of the right bundle branch can be recorded. Chest X-rays reveal increased lung pattern, pulmonary artery trunk bulging, and increased heart shadow due to right atrial and ventricular hypertrophy. Echocardiographic (EchoCG) - examination with color Doppler mapping reveals left-right blood discharge, presence of atrial septal defect, allows determining its size and location. If it is impossible to confirm the diagnosis by non-invasive methods, the patient undergoes invasive methods of cardiac examination. This method is used last. When the heart cavities are examined, an increase in pressure and oxygen saturation of the blood is found in the right parts of the heart and pulmonary artery. In cases of diagnostic difficulties, the examination is supplemented by atriography, ventriculography, jugular vein phlebography, angiopulmonography, and cardiac magnetic resonance imaging [1,7,9,11,18].

For the purpose of treating or preoperative preparation of patients with cerebral palsy in adults, all patient characteristics are considered before prescribing medications. The most common groups are considered to be the following: diuretics; beta-blockers; cardiac glycosides. Treatment of atrial septal defects is only surgical. Indications for cardiac surgery are the detection of significant hemodynamic arteriovenous blood discharge. The optimal age for correcting the defect in children is from 1 to 12 years. Surgical treatment is contraindicated for high pulmonary hypertension with veno-arterial discharge due to sclerotic changes in the pulmonary vessels and severe blood clotting disorders, acute infectious diseases. In cases of atrial septal defects, various methods are used to close them: suturing, plasty with a pericardial patch or synthetic patch in conditions of hypothermia and artificial circulation (IC). X-ray endovascular occlusion of the atrial septal defect allows closing openings no larger than 20 mm. In the last decade, the interventional treatment method has become the primary treatment method for secondary cerebral palsy. There are a number of alternative occluders, some of which minimize the amount of metal used. The "amplacer" device is most commonly used for occlusion. It consists of two fungi-like structures made of wire mesh, connected to each other by a central stem. When positioning, both halves of the device are placed on both sides of the atrial septum, then screwed. The use of this method allows avoiding open heart surgery, artificial blood circulation, as well as pain sensations and cosmetic inconveniences associated with surgical access [6,3,11,15,19].

With concomitant rhythm disturbances, successful surgical correction of the congenital defect without surgical removal of the arrhythmogenic focus does not always guarantee the absence of both early and long-term postoperative arrhythmias and associated disability and sudden cardiac death. The issue of indications for surgical treatment of secondary arrhythmias in adult patients with atrial septal defect is relevant. This article is dedicated to a detailed study of the specifics of surgical treatment of cerebral palsy in adults.

The aim of the study is to examine surgical tactics for atrial septal defect in adult patients.

Materials and methods of research. This article presents a clinical case of late diagnosis of atrial septal defect (ASD) in an elderly woman who had not been previously examined. Experience in correcting ASD in adult patients is limited to isolated operations. According to the literature, most of the adult population with ASD comes to the attention of cardiologists and cardiac surgeons between the ages of 15 and 50 [1,2,6,17]. Considering the rarity of ASD correction in adults, especially in combination with cardiac arrhythmia, we consider it possible to present our own clinical observation.

Research results. Clinical case description: A 61-year-old woman S. (Case No. 6447) was admitted to the cardiac surgery department of the AGMI clinic on January 9, 2025, with a congenital heart defect, complaining of shortness of breath and heart palpitations during physical exertion; symptoms resolve at rest. The defect was discovered recently; she has 5 children. She has noted worsening of her condition over the last 3 years, when she began to experience progressive shortness of breath. The first sensations of pathological heartbeat appeared at the beginning of 2025. Then shortness of breath appeared with minor exertion. She did not seek medical attention. Upon admission, the patient's general condition is of moderate severity. Moderate cyanosis of the skin is observed. The heart borders are extended to the right. A soft systolic murmur is heard over the heart in the 2nd-3rd intercostal space to the left of the sternum; the second heart sound over the pulmonary artery (PA) is accentuated and split. The liver and spleen are not enlarged. ECG shows atrial fibrillation with tachysystole, right axis deviation. Incomplete right bundle branch block. Signs of biventricular hypertrophy and myocardial ischemia. Radiologically, the pulmonary vasculature is enhanced, the cardiac silhouette is enlarged bilaterally, the PA arch is prominent, the atriovenous angle is raised, and the vascular pedicle is widened. Cardiothoracic ratio (CTR) is 65.2%. A transthoracic echocardiogram was performed, showing normal cardiac position: large secundum ASD (4x5 cm) with bidirectional shunting, predominantly right-to-left. Moderate to severe tricuspid regurgitation. Right ventricular systolic pressure is 70-78-82 mmHg. Left atrial size is 42 mm, left ventricular end-diastolic diameter is 54 mm, left ventricular end-systolic diameter is 37 mm. Left ventricular ejection fraction (LVEF) is 60%, LV end-diastolic volume index is 29. Right atrium measures 44 mm. PA diameter is 50 mm. Stroke volume is 86 ml. Pulmonary artery systolic pressure is 94 mmHg. Right ventricular ejection fraction is 60%. Right ventricular free wall thickness is 12 mm. The pericardial cavity is free of fluid (Fig. 1).

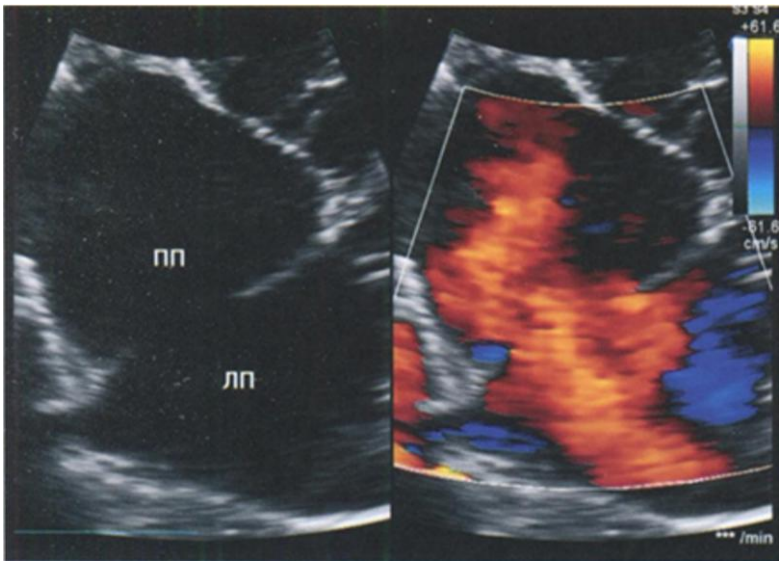

Fig. 1. Heart ultrasound.

In contrast multispiral computed tomography in 3D mode, a large gallbladder is visualized; an expanded gallbladder and prostate. The diameter of the aorta is 25 mm, the pulmonary artery trunk is 40 mm, the right pulmonary artery is 26 mm, and the left pulmonary artery is 28 mm. Selective angiography was performed: coronary artery entrance without stenosis. Anterior descending artery without stenosis. Bypass branch - without stenosis. Right coronary artery without stenosis. The posterior interventricular branch and posterior lateral branch pass through. Right blood supply type.

During catheterization, an increase in blood oxygen saturation is observed, starting from the PP; during angiocardiography, cross-discharge of blood is detected at the level of the atrium through the BPH, but predominates from left to right. Before surgery, capillary blood SaO2 was 71 - 80%. Diagnosed with a congenital heart defect, "secondary DMPP, with 3a-degree LH," the patient underwent midline sternotomy surgery on February 4, 2025. Canulation of the aorta and vena cava. Complete artificial circulation. Cardioplegia. The right atrium was opened with a horizontal incision. It can be seen that the secondary large BCC is 4x5cm in size; accordingly, a patch from the autopericardium was taken from this incision, then the defect was plasticated with a continuous wrapping suture, the hermetic seal was checked, after which a 1cm perforation was made in the center of the patch to loosen the left atrium. A semicircular plasty of the trikuspidal valve was performed using a fluoro-lavsan vascular prosthesis. Perfusion - 48 minutes. Myocardial ischemia - 36 minutes. The operation is usually completed, with drainage in the pericardial cavity and mediastinum. The patient was transferred to the intensive care unit with stable hemodynamic parameters against the background of inotropic support with dopamine (5 mcg/kg/min). The duration of mechanical ventilation was 4 hours and 45 minutes; dopamine infusion lasted 7 hours. The postoperative course is smooth. The patient was discharged home on the 8th day after surgery in satisfactory condition for outpatient treatment. During the control examination 3 months after the operation, the condition is completely satisfactory, no complaints. CaO2 in capillary blood - 90-94%. Auscultation of the heart reveals no pathological murmurs, the accent of the second tone over the LA remains, but to a lesser degree than before surgery. Electrocardiography - atrial fibrillation, normosistolic form, incomplete blockage of the right bundle branch, semi-horizontal position of the electrical axis of the heart, hypertrophy of both ventricles, but to a lesser degree than before surgery. Echocardiography - the linear dimensions of the cavities and the myocardial contractility are already approaching normal. X-ray - normal heart configuration, no hypervolemia in the pulmonary circulation, CTIs are normal. The result of the operation is good. The long-term results of DMP correction in adult patients are good both according to individual reports and large series [1,2,6,17].

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