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Tetrad of Fallot: Modern Perspectives on Origin and Treatment

Xudoyberdiyev Davlat G‘ayrat o‘g‘li
Botirov Sanjar Shuxrat o‘g‘li
Narziqulov Shaxboz Baxodir o‘g‘li
Raxmonqulov Doniyorbek Davron o‘g‘li
Samarkand State Medical University
1st year ordinators of the Department of Cardiology,
Therapy and Functional Diagnostics

Abstract: Tetralogy of Fallot is a combined congenital heart anomaly characterized by right ventricular outflow tract stenosis, ventricular septal defect, aortic dextroposition, and right ventricular myocardial hypertrophy. Clinically, tetralogy of Fallot is manifested by early cyanosis, developmental delay, shortness of breath and dyspneic-cyanotic attacks, dizziness and fainting. Instrumental diagnosis of tetralogy of Fallot includes PCG, electrocardiography, cardiac ultrasound, chest X-ray, catheterization of cardiac cavities and ventriculography. Surgical treatment of tetralogy of Fallot can be palliative (intersystemic anastomosis) and radical (complete surgical repair of the defect).
Keywords: Features of hemodynamics in tetralogy of Fallot, symptoms of tetralogy of Fallot, diagnosis of tetralogy of Fallot, treatment of tetralogy of Fallot, prognosis of tetralogy of Fallot

Tetralogy of Fallot is a complex congenital heart defect of the "blue" type, whose morphological basis consists of four signs: right ventricular outflow tract obstruction, wide VSD, right ventricular hypertrophy, and aortic displacement. . In cardiology, tetralogy of Fallot occurs in 7-10% of all congenital heart defects and accounts for half of all cyanotic-type defects. A detailed anatomical description of the defect as an independent nosological form was first given in 1888 by the French pathologist E. LA Fallot, who later received his name.

In terms of the structure of the defect, the closest to the tetralogy of Fallot are triad of Fallot (stenosis of the pulmonary artery, atrial septal defect and hypertrophy of the right ventricle) and pentad of Fallot (tetralogy of Fallot and ASD). Tetralogy of Fallot can be combined with other anomalies of heart and blood vessels: right-sided aortic arch, anomalies of coronary arteries, stenosis of pulmonary artery branches, open arteriosus, complete form of patent atrioventricular canal, accessory. left superior vena cava, partial anomalous drainage of pulmonary veins.

Causes of tetralogy of Fallot

Tetralogy of Fallot is formed due to a violation of the cardiogenesis process for 2-8 weeks. embryonic development. Infectious diseases encountered by a pregnant woman in the early stages of pregnancy (measles, rubella, rubella) can cause the development of the defect; taking medications (hypnotics, sedatives, hormonal drugs, etc.), drugs or alcohol; exposure to harmful production factors. Heredity affects the formation of congenital heart diseases.

Tetralogy of Fallot is often accompanied by Cornelia de Lange syndrome (Amsterdam dwarfism), including mental retardation and many developmental anomalies ("clown face", choanal atresia,

auricular deformity, Gothic palate, strabismus, myopia, astigmatism, optic atrophy). occurs in children. , deformation of the sternum and spine, syndactyly of the legs, reduction of the number of fingers, malformation of internal organs, etc.).
The causative mechanism of tetralogy of Fallot is malrotation of the conus arteriosus (counter-clockwise), which causes the aortic valve to shift to the right relative to the pulmonary valve. In this case, the aorta is located above the interventricular septum ("riding aorta"). Incorrect positioning of the aorta leads to displacement of the pulmonary trunk, which is slightly elongated and narrowed. The rotation of the conus arteriosus prevents the connection of its septum with the interventricular septum, which leads to the formation of VSD and subsequent expansion of the right ventricle.

Classification of tetralogy of Fallot

Taking into account the nature of obstruction of the right ventricular outflow tract, anatomical variants of tetralogy of Fallot are divided into four types: embryological, hypertrophic, tubular and multicomponent.
Tetralogy of Fallot type I is embryological. Obstruction occurs as a result of anterior and left displacement of the conus septum and/or its low position. The zone of maximum stenosis corresponds to the level of the limiting muscle ring. The fibrous ring of the pulmonary valve is practically unchanged or moderately hypoplastic.
Type II tetralogy of Fallot is hypertrophic. Obstruction is based on an anterior and left displacement of the conical septum and/or its low location, as well as obvious hypertrophic changes in its proximal segment. The zone of maximum stenosis corresponds to the level of the right ventricular outflow tract and the dividing muscle ring.

Tetralogy of Fallot type III is tubular. Obstruction occurs as a result of uneven division of the common arterial trunk, as a result of which the pulmonary cone is sharply hypoplastic, narrowed and shortened. With this type of tetralogy of Fallot, hypoplasia of the annulus fibrosus or valvular stenosis of the pulmonary trunk may occur.
Tetralogy of Fallot type IV is multicomponent. The cause of the obstruction is a significant elongation of the conical septum or a high expansion of the septal-marginal trabeculae of the moderator cord.
Based on the specific characteristics of hemodynamics, three clinical and anatomical forms of tetralogy of Fallot are distinguished: 1) with atresia of the pulmonary artery; 2) cyanotic form with various degrees of stenosis of the orifice; 3) form of cyanosis.

Characteristics of hemodynamics in tetralogy of Fallot

The degree of hemodynamic disturbances in tetralogy of Fallot is determined by the severity of obstruction of the right ventricular outflow tract and the presence of a defect in the interventricular septum.
Significant stenosis of the pulmonary artery and the presence of a large septal defect determine the predominant flow of blood from both ventricles to the aorta and less to the pulmonary artery, which is accompanied by arterial hypoxemia. Due to a large septal defect, the pressure in both ventricles is equal. In the extreme form of tetralogy of Fallot associated with pulmonary atresia, blood enters the pulmonary circulation from the aorta through a patent ductus arteriosus or collaterals.
With moderate obstruction, the total peripheral resistance is higher than the resistance of the stenotic outflow tracts, so a left-to-right shunt develops, which leads to the development of the cyanotic (colorless) form of tetralogy of Fallot. However, as the stenosis progresses, first a cross, then veno-arterial (right-left) blood flow appears, which means that the defect changes from a "white" form to a "blue" one.

Symptoms of tetralogy of Fallot

Depending on the time of appearance of cyanosis, five clinical forms and, accordingly, the same number of periods of manifestation of tetralogy of Fallot are distinguished: early cyanotic form (appearance of cyanosis from the first months or first year of life), classic. (appearance of cyanosis in the second or third year of life), severe (occurs with shortness of breath-cyanotic attacks), late cyanotic (appearance of cyanosis at 6-10 years old) and cyanotic (pale) form.

In severe forms of tetralogy of Fallot, cyanosis of the lips and skin appears from 3-4 months and is stable up to 1 year. Blue color increases with eating, crying, tension, emotional stress and physical activity. Any physical activity (walking, running, playing outdoors) is accompanied by increased shortness of breath, weakness, tachycardia, and dizziness. The usual position for patients with tetralogy of Fallot after exercise is to lie down.

The most severe form of the clinical manifestation of tetralogy of Fallot is dyspneic-cyanotic attacks, which usually appear at the age of 2-5 years. The attack develops suddenly and is accompanied by anxiety of the child, increased cyanosis and shortness of breath, tachycardia, weakness and loss of consciousness. Apnea, hypoxic coma, convulsions may develop subsequent events of hemiparesis. Dyspnea-cyanotic attacks develop as a result of a sharp spasm of the infundibular section of the right ventricle, which causes the entire volume of venous blood to enter the aorta through a defect in the interventricular septum and increase central nervous hypoxia. system.

Children with terada of Fallot may lag behind in physical (stage II-III malnutrition) and motor development; they often suffer from repeated acute respiratory viral infections, chronic tonsillitis, sinusitis and recurrent pneumonia. In adult patients with tetralogy of Fallot, it may be associated with pulmonary tuberculosis.

Diagnosis of tetralogy of Fallot

In the objective examination of patients with tetralogy of Fallot, attention is paid to paleness or bluishness of the skin, thickening of the finger phalanges ("drumsticks" and "hour glasses"), forced posture, adynamia; less often - deformation of the chest (heart tails). Percussion reveals that the borders of the heart are slightly expanded in two directions. Typical auscultatory symptoms of tetralogy of Fallot are coarse systolic noise in the 2nd-3rd intercostal space on the left side of the sternum, weakening of the 2nd tone over the pulmonary artery, etc. The complete auscultatory picture of the defect is recorded using phonocardiography.

A chest X-ray shows moderate cardiomegaly, a typical shoe-shaped heart, and an attenuated lung pattern. The ECG picture is characterized by a significant deviation of EOS to the right, hypertrophic changes in the myocardium of the right ventricle, and incomplete blockade of the right bundle.

With the help of ultrasound of the heart, all anatomical components of tetralogy of Fallot are directly determined: the degree of pulmonary stenosis, the amount of displacement of the aorta, the size of VSD and the severity of right ventricular hypertrophy.

During the examination of the heart cavities, high pressure in the right ventricle, decreased oxygen saturation of arterial blood, catheter passage from the right ventricle to the aorta can be detected. During aortography and pulmonary arteriography, the presence of collateral blood flow, PDA and pulmonary artery pathology is determined. If necessary, left ventricle, selective coronary angiography, MSCT and cardiac MRI are performed.

Differential diagnosis of tetralogy of Fallot is carried out by transposition of great vessels, bilateral origin of the aorta and pulmonary artery from the right ventricle, from a single-ventricle heart, from a two-chambered heart.

Treatment of tetralogy of Fallot

All patients with tetralogy of Fallot undergo surgical treatment. For the development of dyspneic-cyanotic attacks, drug therapy is indicated: inhalation of humidified oxygen, intravenous injection of reopoliglucin, sodium bicarbonate, glucose, aminophylline. If drug therapy is ineffective, an aortopulmonary anastomosis is needed immediately.

The method of surgical correction of tetralogy of Fallot depends on the severity of the defect, its anatomical and hemodynamic variant, and the age of the patient. Newborns and young children with severe tetralogy of Fallot in the first stage require palliative operations, which reduce the risk of complications during subsequent radical correction of the defect.

Types of palliative (bypass) operations for tetralogy of Fallot include: subclavian-pulmonary anastomosis Blalock-Taussig, intrapericardial anastomosis of the ascending aorta and right pulmonary artery, central aortopulmonary anastomosis using a synthetic or biological prosthesis, aortotomy and disinfection. left pulmonary artery, etc. Open infundibuloplasty and balloon valvuloplasty are used to reduce hypoxemia.

Radical correction of tetralogy of Fallot includes plastic surgery of the VSD and elimination of obstruction of the right ventricular outflow tract. It is usually performed between six months and 3 years of age. Specific complications of operations performed for tetralogy of Fallot can be anastomotic thrombosis, acute heart failure, pulmonary hypertension, right ventricular aneurysm, AV block, arrhythmia, infectious endocarditis.

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