TETRA OF FALLO - CAUSES, MORPHOLOGY, CLASSIFICATION, CLINICS, DIAGNOSIS, TREATMENT

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https://doi.org/10.5281/zenodo.7303969

Abstract. *Tetrad of Fallo blue heart powder. Tetralogy of Fallot consists of a violation of hemodynamics, a decrease in blood flow to the lungs and the transfer of venous blood from the right ventricle to the aorta.*

Stenosis of the right ventricular outflow tract (valvular, subvalvular, pulmonary trunk and/or pulmonary artery stenosis or a combination thereof);

Upper (subaortal) defect of the interventricular septum;

Dextraposition of the aorta (shifting to the right);

Right ventricular hypertrophy.

Key words: *Causes of tetrad of Fallot, Morphology, Classification of tetrad of Fallot, Clinic, Diagnosis, Treatment, Prognosis of tetrad of Fallot*

ТЕТРА ФАЛЛО - ПРИЧИНЫ, МОРФОЛОГИЯ, КЛАССИФИКАЦИЯ, КЛИНИКА, ДИАГНОСТИКА, ЛЕЧЕНИЕ

Аннотация. *Тетрада синего сердечного порошка Фалло. Тетрада Фалло состоит из нарушения гемодинамики, уменьшения притока крови к легким и перехода венозной крови из правого желудочка в аорту.*

Стеноз выходного тракта правого желудочка (клапанный, подклапанный, стеноз легочного ствола и/или легочной артерии или их сочетание);

Верхний (субаортальный) дефект межжелудочковой перегородки;

декстрапозиция аорты (смещение вправо);

Гипертрофия правого желудочка.

Ключевые слова: *Причины тетрады Фалло, Морфология, Классификация тетрады Фалло, Клиника, Диагностика, Лечение, Прогноз тетрады Фалло.*

INTRODUCTION

CAUSES OF THE TETRA OF FALLO

Tetrad of Fallo is formed during the 2-8th week of embryonic development as a result of a disturbance in the process of cardiogenesis. The mother experiences infectious diseases (measles, scarlet fever, rubella) in the early stages of pregnancy; taking drugs (sleeping pills, sedatives, hormones, etc.), alcohol and narcotics; Harmful production factors can lead to the development of defects. A genetic factor also plays a role in the development of congenital heart disease.

Tetrad of Fallot is often seen in children with Cornelia de Lange syndrome, which includes oligophrenia and multiple developmental abnormalities.

The tetrad of Fallot is triggered by incorrect rotation of the arterial cone (counter-clockwise), which causes the aortic valve to shift to the right relative to the pulmonary valve. In this case, the aorta is located on the interventricular septum. Incorrect positioning of the aorta leads to displacement of the pulmonary trunk, which is slightly elongated and narrowed. The rotation of the arterial cone prevents its barrier from connecting with the interventricular barrier, as a result of which the CA is formed and the right ventricle expands.

MORPHOLOGY

Four anatomical components:

Ventriculoseptal defect - ventricular septal defect (VSD) - unites the right and left parts of the heart. QATN is always large and non-restrictive in tetrad of Fallot. As a rule, this is perimembranous CAD, muscular CAD or supraarterial CAD.

Obstruction of the right ventricular outflow tract occurs due to one or a combination of the following anatomical components. These include infundibular (subvalvular) stenosis of the right ventricular outflow tract, pulmonary artery stenosis, obstruction due to hypertrophied right ventricular myocardium, hypoplasia of the pulmonary artery core and/or branches.

Dextraposition of the aorta - the aorta is partially displaced from the right ventricle, or the blood flow in it remains dominant due to the activity of the left ventricle.

Right ventricular hypertrophy — hypertrophy of the right ventricular muscle component develops with age.

METHOD AND METHODOLOGY

ASSOCIATION WITH OTHER CONGENITAL HEART DISEASE

Tetrad of Fallot can be associated with pulmonary artery atresia, absence of pulmonary artery valve plates, atrioventicular septal defect, coronary artery defect.

ASSOCIATION WITH CHROMOSOMAL ANOMALIES

47,XX/XY+13 (Patau syndrome);

47,XX/XY+18 (Edwards syndrome);

47,XX/XY+21 (Down syndrome).

CLASSIFICATION OF THE TETRAD OF FALLO

According to the nature of obstruction of the outflow tract of the right ventricle, anatomical types of tetrad of Fallot are divided into four: embryological, hypertrophic, tubular and multi-component.

Embryological. Obstruction is caused by the forward and left and/or low position of the conical barrier.

Hypertrophic. Obstruction is formed due to forward and left and/or low location of the cone barrier, as well as significant hypertrophy of its proximal segment.

Tubular. Obstruction is caused by uneven division of the common arterial trunk, as a result of which the pulmonary cone is sharply hypoplastic, narrowed and shortened.

Multi-component. The cause of obstruction is a significant elongation of the conical barrier or a high beginning of the moderator belt barrier-marginal trabeculae (trabecula septomarginalis).

RESEARCH RESULTS

CLINIC

Cyanosis is the main symptom of tetrad of Fallot. The degree of cyanosis and the time of its occurrence depend on the severity of stenosis of the pulmonary artery. The gradual development of cyanosis is characteristic: first cyanosis of the lips is observed, then cyanosis of the mucous membranes, fingertips, face, hands and feet, body skin.

The first symptoms of the disease appear in the first hours after the birth of the child in the form of light cyanosis during the crying and sucking. Later, cyanosis and shortness of breath (panting) become more pronounced and become permanent by 1-2 years of age and do not disappear even when the child is at rest. Elderly patients complain of shortness of breath, rapid fatigue, headache, physical incapacity.

The pathogenesis of a wheezing attack is associated with a sharp spasm of the infundibular part of the right ventricle, as a result of which all the venous blood begins to flow into the aorta, which leads to severe hypoxia of the central nervous system. During an attack, blood oxygen

saturation decreases by 35%. In this case, the intensity of the noise will decrease sharply, and it may even disappear completely. The child is restless, his facial expression looks scared, his pupils are dilated, panting and cyanosis are increasing, his arms and legs are shaking; after that, fainting, convulsions are observed, as well as hypoxic coma and the possibility of death. Attacks are variable in severity and duration (from 10-15 seconds to 2-3 minutes).

By the age of 4-6, the frequency and intensity of attacks significantly decreases or disappears completely. This is related to the development of collaterals, and over time, the lungs begin to be more or less adequately supplied with blood.

Most patients are unable to get up. The situation is getting worse. The skin of the patients becomes bluish in color, when pressed with a finger, a liquid spot remains in this place, which gradually disappears. Most often, moderately enlarged veins are visible on the patient's body, they are especially noticeable in the area of the chest and on the skull. Nail phalanges look like "drum sticks". The muscular system develops slowly. When examined, in most cases, a "heart attack" is visible.

Depending on the characteristics of the clinic, the course of the wart is divided into three phases:

Phase I — relatively good (from 0 to 6 months), in which the patient's condition is relatively satisfactory, there is no lagging behind in physical development;

Phase II — wheezing-cyanotic attacks (6-24 months), which are characterized by more brain complications and deaths;

Phase III is transient, in which the clinical picture of the wart begins to take on adult characteristics.

IDENTIFY

During auscultation, it is revealed that the I tone is shortened at the apex of the heart, and the pulmonary artery tone is weakened. A systolic murmur is heard at the left edge of the sternum between II-III ribs.

In the general blood analysis, the amount of erythrocytes has increased, the amount of hemoglobin has increased to 130-150 g/l.

In the phonocardiogram, there is a murmur over the pulmonary artery and a murmur over the area of the defect and in the interventricular septum.

X-ray examination reveals the following: the heart is moderately enlarged, the cardiac belt is well defined, and the apex is shifted up. The contour at the level of the pulmonary artery is flat and sunken; the ascending part and arch of the aorta are enlarged; the image of the lungs is blurred, the shade of the roots is reduced.

During probing, the catheter passes directly from the right ventricle to the ascending part of the aorta. When the contrast material is injected into the right ventricle, the pulmonary artery and the ascending aorta are filled at the same time, pulmonary artery stenosis is detected.

Ultrasound examination of the heart reveals all the anatomical components of the tetrad of

Fallot: the degree of pulmonary stenosis, the value of the aortic displacement, the size of the coronary artery and the severity of the right ventricular hypertrophy.

The most characteristic sign in the ECG is a significant deviation of the electrical axis of the heart to the right (120-180°).

DISCUSSION

TREATMEN

Only surgical method is used. Surgical operations in tetrad of Fallot can be palliative and radical.

In palliative surgical procedures, a connection (pathway) is created between the aorta and the pulmonary artery, and weakly arterialized blood is ensured to flow from the aorta to the pulmonary artery, bypassing the narrowed part. Radical surgical operations consist of elimination of stenosis of the pulmonary artery, patching of the defect of the interventricular barrier. Such a patch is sewn to the cross section of the right ventricular wall in order to eliminate possible narrowing of the outflow tract.

The most common procedures include Blaylock-Taussig surgery. In this case, an anastomosis is placed between the right subvertebral artery and the right branch of the pulmonary artery. Usually, a palliative method is used in the first stage before radical surgery.

CONCLUSION

TETRAD OF FALLO PROGNOSIS

The natural progression of the disease mainly depends on the degree of pulmonary stenosis. A quarter of children with a severe form of tetrad of Fallot die in the first year of life, and half - in the newborn period. If surgery is not performed, the average life expectancy is 12 years, and less than 5 percent of patients reach 40 years. The cause of death of patients with tetrad of Fallot is often thrombosis of cerebral vessels (ischemic stroke) or brain abscess.

The long-term results of the radical correction of the tetrad of Fallot are good: patients are able to work and are socially active, they can bear physical loads satisfactorily. However, the later the radical operation is performed, the worse its long-term results are. All patients with tetrad of Fallot should be under the supervision of a cardiologist and cardiac surgeon, and should receive antibiotic prophylaxis against endocarditis before carrying out dental or surgical procedures that are potentially dangerous in terms of the development of bacteremia.

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