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CONGENITAL HEART DEFECTS, EXAMINATION METHODS AND MANAGEMENT TACTICS IN THE NEONATAL PERIOD

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ABSTRACT:

The article under discussion reveals congenital heart defects examination methods and management tactics in the neonatal period. The authors of the article consider that the right standard for congenital heart defects diagnosis is the echo-CG and the main challenge is the development of prenatal diagnosis. But even without echo-CG available, an attentive and competent physician can make a preliminary diagnosis and provide the right and timely help to a patient with congenital heart defects.

Keywords: congenital heart defects, the anatomical structure, pathology, infants' death, embryopathies, cardiovascular system, dystrophic changes, body tissues.

INTRODUCTION:

Heart defects - defects in the anatomical structure of the heart, affecting its function. Congenital and acquired heart defects are distinguished.

Congenital heart defects are rather widespread pathology among cardio-vascular diseases, which is one of the main reasons of infants' death during their first year of life.

The main cause of death in infants during their first year of life is a cardiac defect that occurs during embryonic development (embryopathies), resulting in haemodynamic disturbances, which can lead to heart failure and dystrophic changes in body tissues. Congenital heart defects are a rather common pathology among diseases of the cardiovascular system, i.e. defects in the structure of the heart or large vessels arising from the heart. Congenital heart defects are one of the main causes of death in children in their first year of life, which form during embryonic development (embryopathy) and result in haemodynamic abnormalities that can lead to heart failure and dystrophic changes in body tissues [1].

MAIN PART:

Congenital malformations with abnormal division heart cavity malformations:

Interventricular septal defect (ISD): this is common and depends on the growth of one of the septal structures, which results in a pathological communication between the ventricles. In the early stages of embryogenesis, the ventricular septum is absent and grows from below (from the apex) upwards. This type of defect reflects a variety of incomplete septal formation, ranging from complete absence to a small defect in the upper, membranous, part of the septum, which is the most common. Blood flow through the defect is from left to right, so there is no cyanosis or hypoxia (white type). A significant defect leads to hypertrophy of the right

ventricle, while a minor defect does not cause significant hemodynamic changes.

Atrial septal defect (ASD) as an isolated defect is rare. It occurs either with abnormal development of the primary atrial septum in the 5th week of embryogenesis or later in the formation of the secondary septum and the oval opening. The oval orifice is a normal structure of the heart during the intrauterine period, as the lack of lung activity makes blood circulation in the small circle vessels redundant. After birth the oval orifice gradually closes. A primary septal defect has the appearance of an opening directly above the ventricular valves; a secondary septal defect has a wide open oval opening devoid of a flap.

In both cases the blood flow is from left to right and there is no hypoxia or cyanosis (white type of malformation). The overflow of blood to the right side of the heart is accompanied by hypertrophy of the right ventricle and dilatation of the trunk and branches of the pulmonary artery. Complete absence of interventricular or interatrial septum leads to the development of tricuspid heart disease, a severe malformation. However, during compensation there is no complete mixing of arterial and venous blood, as the main flow of one or the other retains its direction, and therefore the degree of hypoxia increases with decompensation progresses.

Congenital heart defects with division disorders arterial trunk Common arterial trunk in complete absence of division of the arterial trunk. In this malformation there is one common arterial trunk originating from both ventricles, the outlet has 4 semilunar valves or less; the defect is often combined with an interventricular septal defect.

The pulmonary arteries branch off the common trunk close to the valves, before the branch of the great vessels of the head and neck, they may be absent altogether and then the lungs receive blood from the dilated bronchial arteries. Severe hypoxia and cyanosis (blue type of malformation) are observed with this malformation.

Complete transposition of the pulmonary artery and aorta occurs when the septum of the arterial trunk grows in the opposite direction to the rest, normally developing parts of the heart. In this malformation the aorta is placed in front and to the right of the right ventricle of the heart, the pulmonary artery lies behind the aorta and branches off from the left ventricle. Arterial blood can only enter the systemic circulation if there is a defect in the septum of the heart or if the arterial (botallic) duct and the oval orifice are not closed. The defect is accompanied by severe hypoxia and cyanosis (blue type of defect).

The myocardium suffers significantly as the coronary arteries do not receive arterial blood. The children are not viable.

Pulmonary artery stenosis and atresia are seen when the septum of the arterial trunk is displaced to the right, often combined with an interventricular septal defect and other malformations. With significant pulmonary artery narrowing, blood enters the lungs via the arterial.

The arterial (botallic) duct and dilated bronchial arteries allow blood to enter the lungs via the arterial (botallic) duct and dilated bronchial arteries. The defect is accompanied by hypoxia and pronounced cyanosis (blue type of malformation).

Aortic stenosis and atresia are a consequence of displacement of the arterial trunk septum to the left. They are less common than septal displacement to the right and are often accompanied by hypoplasia of the left ventricle of the heart.

There is a sharp degree of right ventricular hypertrophy, enlargement of the right atrium and severe general cyanosis. Narrowing of the aortic isthmus (coarctation),

including atresia, is compensated by the development of collateral circulation through the intercostal and thoracic arteries and severe left ventricle heart hypertrophy.

Non-dilation of the arterial (botallic) duct can be considered a malformation when present with simultaneous dilation in children older than 3 months of life. The blood flow is from left to right (white malformation).

An isolated malformation is amenable to surgical correction, in which the duct is ligated while the heart is working.

Combined congenital heart disease. The most common combined malformations are triad, tetrad and pentad Fallot. Fallot's triad has 3 features: interventricular septal defect, pulmonary artery stenosis and consequent hypertrophy of the right ventricle. Fallot's tetrad has 4 features: interventricular septal defect, pulmonary artery stenosis, aortic dextraposition (displacement of the aortic orifice to the right) and right ventricular hypertrophy. Fallot's pentad, in addition to these four, includes the 5th feature - atrial septal defect.

Etiology:

Three groups of factors are important in the etiology of congenital heart defects (risk factors for birth of a child with CHD)

1. Primary genetic factors;
2. Teratogenic environmental factors: intrauterine infections (rubella viruses, cytomegalovirus, coxsackieviruses, influenza viruses, etc.), intake of drugs by a pregnant woman, alcohol consumption, contact with toxic substances (acids, alcohols, heavy metals, etc.), adverse environmental factors (contamination of water, soil, heavy metals, etc.), adverse environmental factors (contamination of water, soil, air with mutagenic substances), nutritional deficiencies in the mother during early pregnancy, parental age, and the mother's own medical conditions (metabolic disorders, severe toxicosis in the first half of pregnancy, diabetes, cardiovascular disease);
3. Interaction of genetic factors and adverse environmental factors

Diagnosis of congenital heart defects:

1. **Prenatal diagnosis**:

- Detailed collection of a pregnant woman's medical history and identification of risk factors for the birth of a child with developmental abnormalities;
- Prenatal ultrasound screening by the due date;
- If malformations are suspected, a guided ultrasound scan is performed on an expert apparatus;
- Prenatal counseling is conducted in order to determine the timing and site of delivery, to inform the parents about the existing malformation, the expected management of the child after birth, possible cardiosurgical correction, to make a preliminary prognosis.

2. **Medical history**:

When a child is born with a suspected congenital heart defects, a detailed medical history is collected: maternal, family, heredity, prenatally diagnosed cardiac abnormality.

3. **Objective examination of the newborn**:

- External examination of the newborn: identification of embryogenesis stigmata, assessment of skin color, crying pattern, activity of the baby, presence of edema;
- Heart rate and pulse rate;
- Presence and assessment of peripheral arterial pulsation: arteria radialis, arteria femoralis;
- Percussion: assess the borders of cardiac obtuseness, locate the heart in the chest cavity and the liver in the abdomen;

- Heart and lung auscultation: detect cardiac murmur, rales in the lungs;
- Palpation of abdominal organs: size of liver, spleen;
- Identification of syndromal abnormalities or other congenital malformations.

4. Laboratory examination:
- Clinical blood test;
- Blood chemistry;

5. Instrumental examination:
- Pulse oximetry: measurement of preductal and postductal saturation: measurement of saturation on the right arm and either leg, preferably at one time;
- BP: measurement on the right arm and either leg;
- ECG: position of the electrical axis of the heart, overload in the heart, coronary changes, arrhythmias;
- Chest X-ray: heart size, status of pulmonary vasculature pattern;
- Echo-CG: detailing the defect, determining the treatment tactics.

Clinical picture:

The main syndromes manifested by congenital heart defects (CHD) are:
- Syndrome of circulatory insufficiency;
- Arterial hypoxemia syndrome;
- A combination of arterial hypoxaemia and circulatory failure syndromes.

Some CHDs may be asymptomatic immediately after birth. The following factors contribute to the clinical picture.

First factor: in ductus-related heart disease, the patent ductus arteriosus is the main source of blood supply to the aorta or the pulmonary artery. Therefore, closure of the ductus leads to a deterioration, often incompatible with life.

Second factor: in the early neonatal period, the LSS, still relatively high, prevents blood flow from the left ventricle to the right ventricle and an increase in pulmonary blood flow.

After a drop in pulmonary vascular resistance (PVR), there is significant effusion, increased pulmonary blood flow and a murmur. In this regard, the following critical periods in the course of neonatal heart defects are distinguished.

I. Period of arterial duct closure: 3-5 days of life

II. Period of decreasing pulmonary vascular resistance: first few weeks of life (3-6 weeks).

The symptoms and signs of heart disease in the newborn depend mainly on the partial oxygen pressure of the arterial blood [3].

The leading signs of cardiac defects are cyanosis, cardiac murmurs and symptoms of heart failure.

Severe CHD in neonates can also manifest with distress syndrome, shock or collapse.

Symptoms indicating the possibility of critical CHD are:

1) Central cyanosis or grey, pale skin;
2) Absence or abrupt weakening of arterial pulsation in the extremities;
3) BP in the legs decreased by 10 mmHg or more compared to the right arm;
4) Pulse oximetry data show decreased saturation or oxygen saturation in the right arm is 5 % or higher than in the leg;
5) Dyspnea more than 60 per 1 minute;
6) HR greater than 180 beats per minute or less than 100 beats per minute;
7) Abnormal heart rhythm;
8) Hepatomegaly (lower edge protrudes more than 2 cm from underneath the edge of the rib arch);
9) Oliguria;
10) Heart murmur.

Heart murmurs

Heart murmurs occur in 66% of babies in the first 48 hours of life and even in 70% of

newborns during the first week of life, but in 60% of cases they do not indicate valve defects or functional insufficiency.

A significant percentage of physiological cardiac murmurs is due to postnatal restructuring of pulmonary circulation and change of right ventricular dominance to left ventricular dominance.

The same factors determine the patterns of pathological cardiac murmurs, the latter may indicate not only CHD, but also transient circulatory disturbances and cardiomyopathies.

The dependence on the postnatal formation of hemodynamics is manifested in a change in the correlation between the causes of murmurs in newborns of different ages.

In the first hours and days of life, the vast majority of noises have a physiological nature, or (less frequently) they are due to some anatomical forms of ventricular outlet obstruction. From day 2-3, murmurs are heard in cardiomyopathies and transient circulatory disorders.

Noises in intracardiac septal defects become more intense with the elimination of pulmonary hypertension, i.e. reaching a maximum by day 4-5 of life [4].

A murmur persisting beyond 3 days of age is likely to be associated with CHD, especially if it is combined with signs of heart failure.

Physiological murmurs are always systolic and of low intensity. The intensity and localisation of abnormal murmurs are of little help in the diagnosis of an anatomical anomaly, as for neonates, widespread spread of murmurs outside the heart is more the rule than the exception.

Cardiac murmurs in the absence of arrhythmia, cyanosis or cardiac:

In the absence of arrhythmia, cyanosis or heart failure, cardiac murmurs are not an indication for emergency diagnosis and medication, but indicate the need for a detailed follow-up examination.

Many critical CHDs are aphonic:

Therefore, the absence of a heart murmur does not exclude severe CHD at all; moreover, when a newborn with a clinical picture of CHD has no heart murmur, it is usually an extremely unfavourable prognostic sign [1].

General principles of stabilization and examination of newborns with CHD:

1. ABC algorithm: first aid resuscitation according to a common standard.

As recommended: primary respiratory support in the preterm infant should be administered using air, be aware of the possibility of ductus-related heart defects and refrain from administering high percentage oxygen!

2. Transferring the baby to the post intensive care unit.

3. Providing venous access: peripheral and central.

4. Monitoring: pulse oximetry (preferably right arm and any lower limb),
blood pressure (arms/feet), ECG.

5. Provide conditions to reduce oxygen uptake: temperature comfort, reduction of pain, tactile, auditory, light stimuli.

6. Oxygen is contraindicated: it stimulates contraction of the smooth muscle layer of the arterial wall.

Oxygen is contraindicated: it stimulates contraction of the smooth muscle layer of the arterial wall, which promotes ductal closure. But with an established prostaglandin E1 infusion, oxygen (30-35%) can be insufflated to prevent severe hypoxaemia.

7. Indications for transferring the child to ventilator ventilation: presence of metabolic and/or respiratory acidosis: pH < 7.28, pCO_2 > 60 mmHg.

8. Hyperventilation and alkalosis must be avoided.
9. Maintenance of mean arterial pressure > 40 mmHg.
If necessary, use of inotropes: dopamine, dobutrex, mesaton, norepinephrine, adrenaline.
10. Infusion therapy administered on general indications, zero or negative water balance.
11. Antibiotic therapy to be administered on general indication.
12. Diuresis is taken into account, if physiological rate of diuresis decreases: diuretics: lasix 0.1-0.2 mg/kg up to 4 times a day.
13. Maintenance of arterial duct function by administration of prostaglandin E1 as indicated: presence of confirmed ductus-related heart defect.
14. Determination of the detailed structure of the anatomical defect by Echo-Cardiography.
15. Diagnosis and correction of secondary organ and system dysfunctions (lungs, kidneys, liver, central nervous system).
16. Identification of malformations of other organs: a comprehensive ultrasound of the internal organs.
17. Genetic testing.

Conclusion:

The right standard for CHD diagnosis is the echo-CG and the main challenge is the development of prenatal diagnosis. But even without echo-CG available, an attentive and competent physician can make a preliminary diagnosis and provide the right and timely help to a patient with CHD.

REFERENCES:

1) Manual on Perinatology. Edited by D.O. Ivanov. - St. Petersburg: Informnavigator, 2015. P.34
2) Neonatology/ed. T.L. Gomella, M.D. Cunningham, F.G. Eyal. Translation from English. Edited by Prof. D.N. Degtyarev, 2015. P.59
3) Neonatology. Practical guidelines. Reinhard Roose, Orsola Genzel-Borowicz, Hans Prokitte. Moscow. Medical Literature. 2013. P.28
4) Richard A. Paulin, Alan R. Spitzer. Secrets of neonatology and perinatology. Translation from English. Edited by Prof. N.N. Volodin. Moscow.
5) Publishing house BINOM, 2011. P.13
6) N.P. Shabalov. Neonatology textbook in 2 volumes. M. MED press-Inform, 2009. P.7
7) N.P. Shabalov. Paediatric diseases. Textbook. Sixth edition, revised and supplemented in two volumes. “Peter” 2007. P.42.