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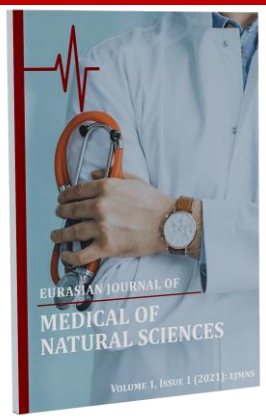

CONGENITAL HEART DEFECTS

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ABSTRACT

Congenital heart defects (TYN), also known as congenital heart anomaly and congenital heart disease, are defects in the structure of the heart or great vessels present at birth. Congenital heart defects are classified as cardiovascular diseases. Signs and symptoms depend on the specific type of defect. Symptoms can be harmless or life-threatening. If present, symptoms may include rapid breathing, bluish skin (cyanosis), low weight, and fatigue. Congenital heart defects do not cause chest pain. Congenital heart defects are often not associated with other diseases. A complication of congenital heart defects is heart failure.

Congenital heart defects may require surgery and medication. Medications include diuretics, which help remove water, salts, and digoxin from the body to make the heart contract harder. This slows the heart rate and removes some fluid from the tissues. Some defects require surgical procedures to restore normal circulation, and in some cases require multiple operations.

Many people need lifelong specialized heart care, first with a pediatric cardiologist and then with an adult congenital cardiologist. More than 1.8 million people live with congenital heart defects.

Heart defects are persistent defects, defects and changes in the anatomical structure of the heart; interferes with normal blood flow. A distinction is made between congenital and acquired heart disease. Congenital heart defects occur as a result of incorrect formation of the fetal heart and large vessels of the heart during embryonic development. In the early period of pregnancy, poisoning of the mother's body, suffering from some diseases, biological effects of ionizing rays, hereditary diseases, etc. cause. In infancy (up to 1 year of age), incomplete development of the cardiovascular system (for example, open arterial passages or incomplete completion of the foramen ovale) is also included in Heart defects.

The most common types of Congenital Heart Diseases are: various combinations of abnormal pathways between the large and small circulatory circles, as well as narrowed or occluded areas in the large vessels of the heart (eg, pulmonary artery and aorta) or incorrect

location of these vessels; mixed vices; defects related to the number and structure of heart chambers.

Depending on the degree of mixing of arterial and venous blood, some congenital heart defects occur with cyanosis (blue defects) and some without cyanosis (white defects). It depends on the direction in which the blood flows through the inappropriate holes connecting the large and small circulation circles (the direction of the shunt), the degree of pressure increase in the pulmonary artery, and the condition of the heart muscles. Physical maldevelopment of the child, paleness or blueness, shortness of breath, changes in the size and position of the heart, heart murmurs, etc. are typical signs of congenital heart defects.

Acquired heart disease is a disease of the heart during life, mostly rheumatic carditis, sometimes atherosclerosis, septic endocarditis, wounds, etc. appears as a result of diseases. Acquired heart defects: non-tight closing of the heart valves (at the time of closing); narrowing (stenosis) of the opening between the ventricles (right and left ventricles) or the outlet of the main vessels; a combination of these defects, a defect in one or more valves, etc. enters. There are mitral (opening between the left ventricle and the ventricle and bicuspid valve), aortic, mitral-aortal and other heart defects. In heart defects, due to valve defects, blood partially flows back or as a result of straining through a narrowed opening, the muscular wall of the heart thickens (hypertrophy), then the force of contraction decreases, and its cavities expand (dilatation). As a result, blood circulation is derailed - circulatory failure occurs. Acquired heart disease can occur quickly or slowly over a long period of time. Depending on the location, type, and degree of the disease, the clinical symptoms of the disease are different. Sometimes it can go without any symptoms for a long time. Pregnancy, colds, influenza, as well as strong physical stress, etc., cause the symptoms of the disease to intensify and manifest themselves. Symptoms of heart failure, such as rapid pulse, swelling of the legs, shortness of breath, and a murmur in the heart indicate the presence of heart attack. The clinical appearance of heart defects depends on the nature of the defect, as well as the course of the main disease causing it, the patient's work and rest regime. Timely therapeutic and surgical treatment can alleviate the patient's condition and prolong his life.

The treatment is carried out under the doctor's instructions and under his constant supervision. Some types of heart disease can be treated with medication and diet. For this reason, the specialist doctor decides not to operate the patient. Surgical treatment forms the basis of treatment of heart defects.

In order to prevent it, it is necessary to treat diseases that cause heart disease in time.

GENETIC FACTORS

The most studied causes of congenital heart disease are point mutations or chromosomal mutations in the form of deletions or duplications of DNA segments. Major chromosomal abnormalities such as trisomy 21, 13, and 18 account for approximately 5–8% of TYP cases. Trisomy 21 is the most common genetic cause. Some genes are associated with certain diseases. Mutations of the cardiac muscle protein α-myosin heavy chain (MYH6) are associated with intercompartmental barrier defects.

Some MYH6-interacting proteins have also been linked to heart defects. GATA4 forms a complex with the TBX5 gene, which interacts with the transcription factor MYH6. Another factor, NKX2-5 protein homeobox, is also affected by MYH6. Mutations of these proteins are

associated with the development of intercompartmental and interventricular barrier defects. In addition, NKX2-5 is associated with heart conduction defects, and TBX5 is associated with Holt-Oram syndrome. Another T-box gene, TBX1, is associated with DiGeorge syndrome, the deletion of which causes a wide range of symptoms, including heart-bleeding defects, including tetrad of Fallot.

RELATIONSHIP OF CONGENITAL HEART DEFECTS WITH GENDER

The study of the relationship of congenital heart defects to gender was carried out in the early 1970s, based on data collected in several large cardiac surgical centers and information from the literature. As a result of the analysis of 31,814 patients with congenital heart and trunk vessel defects, it was found that there is a clear connection between the type of defect and the gender of the patient. According to the gender ratio, congenital heart defects can be divided into three groups: "male", "female" and "neutral". In the table, E is male, A is female.

THEORIES

Rokitansky (1875) believed that the cause of congenital malformations is the cessation of heart development at various stages of ontogenesis. Spitzer (Spitzer, 1923) considers them to be a return to one of the stages of phylogenesis. Krimsky, synthesizing the previous two points of view, considers congenital heart defects to be the cessation of development at a certain stage of ontogenesis, corresponding to one or another stage of phylogeny. Only atavistic heart defects (feminine and neutral) can be accommodated within these theories, all male defects have no explanation, since none of the male elements of congenital heart defects correspond to similar formations in normal embryos or human phylogenetic generations. Application of "teratological rules of sexual dimorphism" allows to explain all three groups of defects.

The division of congenital heart and large vessel defects into male, female and neutral types makes it possible to use the patient's gender as a diagnostic symptom. At the same time, the coefficient of diagnostic value of defects of male and female type is very important. For example, taking into account information about the patient's gender increases the probability of diagnosing patent ductus arteriosus by 1.32 times.

PATHOGENESIS

Two mechanisms are leading:

1. Disturbance of cardiac hemodynamics → tension of the heart sections by volume (valvular insufficiency and septal defect type defects) or resistance (arterial or orifice stenosis type defects) → exhaustion of involved compensatory mechanisms (homeometric Anrep to resistance and heterometric Frank-Starling to volume) → development of hypertrophy and dilatation of heart chambers → development of heart failure (and, accordingly, disruption of systemic hemodynamics).
2. Disturbance of systemic hemodynamics (fullness/anemia of the small circulatory circle, anemia of the large circulatory circle) → development of systemic hypoxia (mainly, circulatory in white cells, hemic in blue cells, as well as ventilation and diffusion in acute left ventricular failure may also be hypoxia).

CLASSIFICATION

There are many classifications of birth defects. Congenital heart disease is conditionally divided into 2 groups:

1. White (arterial and venous blood do not mix, with left-right flow of blood). It includes 4 groups:

• With enrichment of the small blood circulation circle (open arterial tube, intercompartmental barrier defect, interventricular barrier defect, AB-communication, etc.);

• With weakening of the small blood circulation circle (isolated pulmonary stenosis, etc.);

• With weakening of the circle of large blood circulation (isolated aortic stenosis, coarctation of the aorta, etc.);

• Without a significant disturbance of systemic hemodynamics (dispositions of the heart - dextro-, sinistro-, mesocardia, cardiac dystopia - neck, chest, abdomen).

2. Blue (with right-left flow of blood, mixing arterial and venous blood). Includes 2 groups:

• With enrichment of the small blood circulation circle (complete transposition of the trunk vessels, Eisenmenger complex, etc.).

• With weakening of the small circle of rotation (tetrad of Fallo, Ebstein's anomaly, etc.).

In 2000, the International Nomenclature was developed to create a general classification system for birth defects.

HYPOPLASIA

Hypoplasia can damage the heart, usually leading to right or left ventricular failure. This means that only one side of the heart can effectively pump blood to the body and lungs. Cardiac hypoplasia is rare, but it is the most serious form of TYP. Such cases are called left heart hypoplasia syndrome when the left side of the heart is damaged, and right heart hypoplasia syndrome when the right side of the heart is damaged. In both cases, the presence of a patent ductus arteriosus (also a patent oval window if the hypoplasia affects the right side of the heart) is essential to keep the child alive until cardiac surgery, because without these techniques the blood cannot circulate in the body (or in the lungs, depending on the side of the heart damage). Cardiac hypoplasia is usually a blue heart defect.

OBSTRUCTION DEFECTS

Obstruction defects occur when heart valves, arteries and veins are stenosed or atretized. The main defects are stenosis of the pulmonary valve, stenosis of the aortic valve, and coarctation of the aorta. Bicuspid valve stenosis and subaortic stenosis are rare. Any stenosis or atresia can cause heart enlargement and hypertension.

BARRIER DEFECTS

The septum is a tissue wall that separates the left heart from the right. In intercompartmental or interventricular barrier defects, blood begins to flow from the left side of the heart to the right side, reducing the efficiency of the heart. Interventricular septal defect is the most common type of TYP.

BLUE PORKS

Blue heart defects are called so because they cause cyanosis, where the skin turns bluish-blue due to lack of oxygen in the body. Such malformations include a persistent arterial core, total anomaly of the union of the pulmonary veins, tetrad of Fallot, transposition of the main vessels, as well as congenital stenosis of the tricuspid valve.

PORKS

- Aortic stenosis;
- Intercompartmental barrier defect;

- Ventricular barrier defect;
- Two-way valve stenosis;
- Dextrocardia;
- Duplication of the left ventricular outlet;
- Duplication of the right ventricular outlet;
- Ebstein anomaly;
- Hypoplasia syndrome of left heart chambers;
- Right heart hypoplasia syndrome;
- Mitral valve stenosis;
- Pulmonary artery atresia;
- Congenital stenosis of the pulmonary artery valve;
- Transposition of trunk vessels:
- dextro-transposition
- senistro-transposition
- Congenital stenosis of the tricuspid valve;
- Persistent arterial core;
- Interventricular barrier defect.

1. Some conditions only affect the large vessels immediately near the heart, but these are often classified as TYP:

- Coarctation of the aorta;
- Atresia of the aorta;
- Open arterial tube;
- Partial anomaly of pulmonary vein connection;
- Total anomaly of connection of pulmonary veins.

2. Some of the vices usually occur together:

- Tetrad of Fallo;
- Cantrella pentad;
- Schon's syndrome / Schon's complex / Schon's anomaly.

SYMPTOMS AND SIGNS OF CONGENITAL HEART ATTACK

Clinical manifestations depend on the type and severity of the heart defect. Symptoms often appear early in life, but some TYPs may go unnoticed throughout life. Some children have no symptoms, while others have shortness of breath, cyanosis, fainting, heart murmurs, underdeveloped limbs and muscles, poor appetite or short stature, and frequent respiratory infections. can be. In congenital heart defects, heart murmurs appear due to its incorrect structure. They can be detected during auscultation, but not all heart murmurs are caused by congenital heart defects.

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