FEATURES OF NATURALLY PROGRESSING CONGENITAL HEART DEFECT — DEFECTS OF THE INTERGENEOUS PARTITION IN CHILDREN

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RELEVANCE

Cardiovascular diseases today are one of the prevailing causes of morbidity and mortality in children. In the structure of the cardiovascular pathology of children of newborns and the first year of life, 65–70% are occupied by CHD. In Europe, the incidence of CHD in children is 6.65 per 1000 live births. About 50% of children with CHD have critical defects of the neonatal period, which provides specialized care in the first hours and days of life. The level of diagnosis of CHD, including prenatal, in recent years has improved significantly due to the increase in cardiosurgery in Tajikistan. According to statistics, the defect of the interventricular septum is one of the leading places among the CHD.

The purpose and objectives of the study

the features of the natural course of CHD in children — the defect of the interventricular septum.

MATERIALS AND METHODS OF RESEARCH

Under supervision were 76 children with CHD–VCD at the age of up to 5 years. All patients underwent general clinical research methods, including chest x-ray, ECG and ultrasound of the heart. Depending on the severity and prognosis of the disease, patients are divided into 3 groups. In 1 group, defects with relatively free flow and outcome (28) are classified. According to the ultrasound of the heart, the size of the defect was up to 0.6 mm, the second group consisted of patients with a more severe course of the defect, whose defect size was more than 0.7 mm (28). The third group of patients consisted of 26 patients in whom the defect of the septum was within 10–30 mm.

RESULTS OF THE STUDY

At small defects (1–3 mm), there were usually no signs of circulatory disturbance. Children developed normally and did not need treatment. Clinically, he heard a rough systolic murmur in the 4–5 intercostal space. To the left of the sternum. With moderate defects (more than 7 mm), signs of circulatory disturbances appeared from 1.5–2 months of life. There was rapid fatigue in feeding, sweating, mild dyspnea, tachycardia, and a history of frequent respiratory diseases.

There was a systolic tremor, the 2nd tone split and strengthened in the pulmonary artery, followed by cardiac arrest for 3 years, heart failure progressed, children lagged behind in physical development. In patients of the third group, signs of circulatory disturbances were manifested from 1 month of life. Pronounced dyspnea and tachycardia, coughing, stagnation wheezing in the lungs, enlargement of the liver. In the anamnesis all children of this group had recurrent bronchitis, pneumonia. The cardiac hump, the 2nd tone accent on the pulmonary artery, and a part of the patients except for systolic murmur in 3–4 intercostal spaces to the left of the sternum were marked by mesodial tolicular noise in the apex associated with a relatively mitral stenosis, due to a large discharge or diastolic noise , associated with the relative failure of the pulmonary artery valve. In children with a large defect, signs of total heart failure were noted. Pulmonary hypertension developed as a rule, by the second half of the year, the pressure gradient between the right and left ventricles was more than 35 mm Hg.

With a small defect, the ECG is normal. The clinical symptoms of combined myocardial hypertrophy of both ventricles are combined with an average or large defect of the septum. Deep Q tooth in right thoracic leads in combination with signs of myocardial hypertrophy of both ventricles, rSR form; rR; R or RS in leads V1.

Electrocardiographically, the signs of hypertrophy of the right and left ventricles, and the overload of the left atrium were noted in the 2nd and 3rd groups of patients. On the roentgenogram of the chest with the average especially large defects of the interventricular septum — cardiomegaly due to the left atrium, left ventricle, less than the right. Arterial overload of pulmonary blood flow.

When rentgenographing chest organs in patients with small dimensions of VSD, there are no abnormalities. With large defects of the partitions, the pulmonary pattern is strengthened, the waist is smoothened, the ventricles are enlarged, and the arch of the pulmonary artery is often swollen.

When echocardiographic study revealed dilatation of the left atrium, dilatation and hypertrophy of the left and right ventricles, increased pressure in the pulmonary artery and right ventricle.

An increase in the right ventricle index, an increase in the rate of dynamic movement of the mitral valve, associated with accelerated blood flow through it, an increase in the end-diastolic diameter of the left ventricle.

At follow-up in patients of the 1st group, the VCD spontaneously closed in 8 children, which was confirmed by ultrasound of the heart, in 1 patient developed bacterial endocarditis, in the remaining children of this group, the cardiac insufficiency of 1–2 degree was gradually developed. In the 2nd and 3rd group, on the background of pulmonary hypertension, cardiac insufficiency developed in different degrees, so in 32–2A degree, in 9–2B degree, in 7–3 degree. During 3 years of observation, heart failure progressed in 10 patients, followed by Eisenmenger syndrome, i.e. sclerotic stage of pulmonary hypertension, leading to an inverse venous-arterial discharge, characterized in children by the appearance of cyanosis of the cheeks, lips, and fingertips. In 38 children, hemodynamic disturbances did not change for several years.

In 12 patients with a small and medium defect of the interventricular septum in the anamnesis, the size of the defect was reduced. Conservative therapy consisted in the appointment of diuretics, cardiac glycosides, potassium preparations, metabolites, vitamins and the treatment of concomitant diseases.

31 patients with VSD were operated with a favorable outcome.

CONCLUSIONS

In children with CHD of VSD with a defect size of up to 7 mm, signs of circulatory disturbance were 1–2 A degree and developed gradually. At moderate and large defects of partitions more than 7 mm, signs of ND of different degree were noted. With large defects of the partitions, cardiac insufficiency rapidly progressed and pulmonary hypertension developed, which necessitates earlier diagnosis and timely surgical correction of the defect.

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