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COMBORIDITY OF CONGENITAL HEART DISEASES IN CHILDREN

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ABSTRACT

The authors studied the condition of sick children aged 1 month to 18 years with congenital heart defects with comorbidity. In 77.5% of the examined children, concomitant diseases were diagnosed along with the underlying disease (CHD). The authors argue that frequent acute respiratory infections lead to a decrease in immunity and the formation of foci of chronic infections, the exacerbation of which is one of the reasons for late surgical correction, resulting in a high risk of postoperative complications, mortality and a decrease in the quality of life of patients with CHD. The natural comorbid course of CHD reduces the effectiveness of conservative treatment of heart failure.

KEYWORDS: Comorbidity, congenital heart defects, immunity.

INTRODUCTION

Despite significant advances in the development of theoretical and practical aspects of congenital heart defects (CHD), the prevalence of CHD in the population does not decrease. Among congenital developmental anomalies leading to disability, CHD is 15–20%.[2] In recent years, the number of operated patients on an open heart has been increasing. And accordingly, the group of patients in need of repeated operations is naturally increasing. According to various authors, the number of re-operated for CHD is 10-40% of the number of primary operations.[9]

In the structure of child mortality associated with malformations, CHD occupy the first line and among all those who died with heart defects, in all cases, the pathology of the thymus gland (TG) was diagnosed.[1]

It is known that in children with CHD there is often an increase in the size of VL.[5] In most cases, this technically complicates the correction of CHD and, as a result, there is a need for complete or partial removal of TG.[3]

In children, TG increases with age, and reaches its maximum weight in the age period of 6-14 years. Physiological involution of the gland begins from 1 year, and at the same time, a special change in its structure occurs.[7] By the expression of cytokeratin's in thymic epithelial cells in CHD, the relationship between the state of the thymic reticuloendothelial stroma and the number of receptor excision rings (REC) in the population of peripheral T-lymphocytes was traced.[4]

In children with CHD, the morphological picture of premature "aging" of the thymus with signs of its functional dysfunction has been established. Activation of apoptosis, an imbalance between proliferative and apoptotic processes, violation of the subpopulation ratio of lymphocytes are prerequisites for the formation of an immunodeficiency state.[6]

After improving the surgical treatment of CHD, complications from the nervous system acquired not only academic interest, but also practical significance. In children with CHD and convulsive syndrome, disorders associated with chronic cerebral vascular insufficiency and hypoxemia were noted.[2]

In pediatric cardiac surgery, critical CHD requires early surgical correction and, without intervention, a very high mortality and survival rate with significant disability.[8]

Purpose of the study: clinical characteristics of congenital heart defects with comorbidity.

MATERIALS AND METHODS

The study included 160 sick children with congenital heart defects (CHD) under the age of 18 years. The control group consisted of 30 healthy peers (14 boys and 16 girls).

When distributing sick children with CHD by gender, the structure determines the predominance of the male contingent. There are slightly more boys -88 (55.0 ± 0.3%) than girls-72 (45.0 ± 0.3%).

All sick children with CHD were distributed according to M.F. Zinkovsky into 3 groups:

1. A group of patients with critical heart defects. Surgical intervention in such children should be performed in the first hours or days of life - 3 (1.8%);
2. A group of patients for whom early surgical correction of CHD is not indicated due to minor hemodynamic disturbances - 13 (8.2%);
3. A group of patients with inoperable CHD or inoperable patients by somatic condition - 11 (6.8%).

The observed group consisted of the remaining 133 sick children who were shown planned surgery. Of these, 78 (58.6%) sick children after surgical correction of CHD, 44 boys (56.4 ± 0.2%) and 34 girls (43.6 ± 0.5%).

The structure of CHD of the observed group was 77- "white" (57.9%) and 56- "blue" malformations (42.1%): interventricular septal defect (BMD) - 30 (22.5%), tetralogy of Fallot (TF) -30 (22.5%), transposition of the great vessels (TMS) -26 (19.5%), atrial septal defect (ASL) -30 (22.5%), open aortic duct (OAI) -11 (8, 4%), pulmonary stenosis (ALS) -6 (4.6%).

Sick children with CHD, in order to correct cardiac hemodynamic disorders due to the underlying disease and other concomitant diseases, such as acute bronchopneumonia, obstructive bronchitis, have repeatedly received conservative treatment. Treatment of children with CHD for colds in a hospital setting included the appointment of antibiotics, anticoagulants, furosemide, followed by switching to verospiron. In the observation group, all sick children were prepared for surgery as planned. Surgical correction of CHD was performed at the TashSPMI cardiac surgery center and the Vakhidov`s Republican Center for Thoracic Surgery, as well as abroad: in the Russian Federation, Kiev and India.

DISCUSSION

It was found that in children with CHD an early formation of foci of chronic infections is observed. So, in our studies, along with the main disease (CHD), 77.5% of children were diagnosed with concomitant diseases: chronic tonsillitis, sinusitis, otitis media, urinary tract infection, anemia, dental caries, thyroid hyperplasia, lag in physical and mental development.

The analysis of the surveyed contingent depending on the place of residence showed the prevalence of CHD among the villagers. Characteristic was an increase in the incidence of cerebrospinal fluid and other complex (blue) malformations (TF, TMS) among children living permanently in the countryside.

The age-gender structure of the observation group is the largest number of boys under the age of 10 years, and girls are more often registered at the age of over 11 years. The manifestation of such a pattern in CHD is associated: firstly, with the peculiarities of the functioning of immunity in children in critical age periods (according to D.V. Stefani, D.E. Veltishchev: the first 30 days of life, 3-6 months, 2nd year of life , 4-6 years of life, adolescence: in girls, 12-13 years old; in boys, 14-15 years old), regardless of the type of defect; secondly, with early puberty (sexual) development of girls.

The most important factor in CHD is a hereditary predisposition. According to our data, hereditary burden was observed in 65 (48.8%) sick children, on the paternal side - in 13 (20.0%), maternal - in 50 (76.9%), was not observed in both lines. In 2 cases (3.1%), a family form of CHD was established. Parents suffered from various forms of CHD. The cardiac pathology of parents and children did not always coincide.

When analyzing pregnancy parity, there were 48 children (36.1%) born from the first pregnancy, 35 (26.3%) from the second, 50 (from the third and more pregnancies) (37.6%). The age structure of parents of sick children shows that more often parents were in the average reproductive age of 20-29 years (Table 1).

Table 1: Age structure of parents of sick children with CHD.

Place of residence	17-19 years old		20-29 years old		30-39 years old		40-49 years old	
	Abs.h	%	Abs	%	Abs	%	Abs	%
City	6	50,0	36	48.0	22	51,2	1	33.3
Village	6	50,0	39	52.0	21	48.8	2	66.7
Total	12	9.0	75	56.4	43	32.3	3	2,3

All examined patients were born full-term, 27 patients of them weighing up to 2500 g. (20.3%), 106 sick children - over 2500 gr. (79.7%). In the history of 75 (56.4%) mothers of sick children with CHD, a pathological course of pregnancy was observed, which was associated with extra genital (48.0%) and genital (52.0%) diseases. In mothers of examined sick children with CHD in 44% of cases, TORCH infection, in particular CMV, was anamnestically established.

The clinical manifestations of CHD, depending on the species, were diverse. With "blue" defects (TF, TMS, OAVK) and ALS, clinical symptoms appeared in the first 6 months of life, but some "white" CHDs, such as

OAP, DMSP, DMS, were undetected and diagnosed throughout their lives.

Objectively, when examining patients with "blue" CHD, cyanosis (92.6%), pulsation and swelling of the cervical vessels (36.8%), "heart hump" (89.3%), "drumsticks" (90.4%) were observed), a symptom of "watch glasses" (90.4%), a lag in physical development (81.4%). Characteristic symptoms were shortness of breath and cyanosis. In 32% of patients with "blue" CHD, a very pronounced cardiac hump was observed, and in patients with "white" CHD, there was no cardiac hump in 31% of patients with DMS.

In patients with DMS, the clinical picture was manifested in 2 versions: with high pulmonary hypertension and without pulmonary hypertension. With age, all children with CHD have complaints of pain in the heart, shortness of breath, palpitations, interruptions in heart function, loss of appetite, and frequent respiratory infections.

With TF and TMS, frequent dyspnea-cyanotic attacks with characteristic manifestations were observed, and a gradual development of symptoms of heart failure and chronic hypoxemia was also characteristic.

Physical development lag was found in 59 sick children with CHD (44.4%). Of these, 48 patients with "blue" CHD (81.4% of cases) in whom growth and developmental lag was found against the background of chronic hypoxemia.

CONCLUSION

Features of the functioning of immunity in critical periods of life, impaired cardiac hemodynamics and chronic hypoxemia are the cause of frequent acute respiratory infections and the development of an immunodeficiency state. Comorbidity significantly worsens the condition of patients with CHD, reduces the effectiveness of conservative treatment of heart failure, and causes a delay in the necessary surgical correction.

Throughout life with age, the formation of a vicious circle is characteristic of patients with complex (blue) types of CHD: the presence of cardiovascular anomaly contributes to the violation of hemodynamics, as a result of which tissue hypoxia develops. The latter, in turn, helps delay physical development and the development of frequent acute respiratory infections. Frequent acute respiratory infections lead to a decrease in immunity and the formation of foci of chronic infections, the exacerbation of which is one of the causes of late surgical correction, resulting in a high risk of postoperative complications, mortality and a decrease in the quality of life of patients with CHD.

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