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FEATURES OF COMORBIDITY WITH CONGENITAL HEART DEFECTS IN CHILDREN

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✓ Resume

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

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

Resume: 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.

Key words: immunity, comorbidity, conservative therapy, congenital heart defects, foci of infection.

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√ Резюме

1 ойликдан 18 ёшгача, тугма юрак нуқсони (ТЮН)билан тугилган болаларда коморбидлик холати ўрганилди. Текширилган беморлардан 77,5% болаларда асосий касаллик (ТЮН) фонида хамрох касаллик сифатида ўткир респиратор касаллик (ЎРК) аниқланган. Муаллифлар такидлашича, ТЮН фонида ЎРК билан тез-тез касалланиш иммунитет пасайиши билан бир қаторда сурункали инфекция ўчоқларини шаклланишига хам олиб келади, касалликнинг қўзголиши ТЮНни хирургик даволашни кечикишига сабаб бўлади. Натижада оператив даводан сўнги асоратлар хавфи ошиши, ўлим кўрсатгичлари ўсиши ва хаёт сифати даражаси камайиши кузатилади. ТЮН коморбидлик фонида кечиши юрак етишмовчилигини консерватив даволаш самарадорлигини пасайишига олиб келади.

Тадқиқод мақсади: Туғма юрак нуқсонларининг коморбид холатлардаги клиник хусусиятларини ўрганиш.

Материал ва услублар: Тадқиқотда туғма юрак нуқсонлари билан туғилган 18 ёшгача бўлган 160 та бола қатнашди. Назорат гурухига 30 та мос ёшдаги соглом болалар кирди. (16 ўгил 14 та қиз). ТЮН билан бемор болалар жинс бўйича структураси ўрганилганда ўгил болалар қизларга нисбаттан кўпчиликни: ўгил болалар -88 (55,0±0,3%), қиз болалар-72 (45,0±0,3%) ташкил этишди.

Хулоса: Хаётнинг критик даврларида иммунитетнинг фаолият юритиши, юрак гемодинамикасининг бузилиши, хамда хроник гипоксемия иммунодефицит холатлар ва қайталаниб турувчи ЎРКнинг асосий сабабчиси сифатида келтириш мумкин. Коморбид

холатлар ТЮН бор беморлар ахволини сезиларли даражада огирлаштиради, юрак етишмовчилигидаги консерватив давони самарасини пасайтириб, керакли хирургик коррекциянинг кечиктирилишига олиб келиши мумкин.

Калит сўзлар: комрбидлик, консерватив даволаш, иммунитет, инфекция ўчоги, тугма юрак нуқсони.

ОСОБЕННОСТИ КОМОРБИДНОСТИ ВРОЖДЕННЫХ ПОРОКАХ СЕРДЦА У ДЕТЕЙ

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✓ Резюме

Изучено состояние больных детей в возрасте от 1 месяца до 18 лет с врожденными пороками сердца (ВПС) на фоне коморбидности. Из обследованных у 77,5% детей наряду с основным заболеванием (ВПС) диагностированы сопутствующие заболевания – острые респираторные заболевания (ОРЗ). Авторы утверждают, что частые ОРЗ могут привести к снижению иммунитета и к формированию очагов хронических инфекций, обострение которых являются одним из причин поздней хирургической коррекции ВПС, в результате чего возникает высокий риск послеоперационных осложнений, смертности и снижения качества жизни больных. Естественное течение ВПС на фоне коморбидности способствует снижению эффективности консервативной mepanuu недостаточности.

Цель: клиническая характеристика врожденных пороков сердца при коморбидности.

Материалы и методы: исследованию были включены 160 больных детей с врожденными пороками сердца (ВПС) в возрасте до 18 лет. Контрольную группу составили 30 здоровых сверстников (14 мальчика и 16 девочек).

При распределении больных детей с ВПС по полу, структура определяет преобладание мужского контингента. Мальчиков несколько больше -88 (55,0±0,3%), чем девочек-72 $(45,0\pm0,3\%).$

Заключение: особенности функционирования иммунитета в критические периоды жизни, нарушения сердечной гемодинамики и хроническая гипоксемия причиной частых ОРЗ и развития иммунодефицитного состояния. Коморбидность существенно ухудшает состояние больных с ВПС, уменьшают эффективность консервативной терапии сердечной недостаточности, становятся причиной отсрочки необходимой хирургической коррекции.

Ключевые слова: коморбидность, консервативная терапия, врожденные пороки сердца, иммунитет, очаги инфекции.

Relevance

espite significant advances 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. Accordingly, the group of patients in need of repeated operations is naturally increasing. According to various authors, the number of reoperated 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 TG [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.

Material 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 \pm 0.3%) than girls-72 (45.0 \pm 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 ($56.4 \pm 0.2\%$) and 34 girls ($43.6 \pm 0.5\%$).

The structure of CHD of the observed group was 77- "white" (57.9%) and 56- "blue" malformations (42.1%): ventricular septal defect (VSD) - 30 (22.5%), tetralogy of Fallot (TOF) - 30 (22.5%), transposition of the great arteries

(TGA) -26 (19.5%), atrial septal defect (ASD) -30 (22.5%), patent ductus arteriosus (PDA) -11 (8, 4%), pulmonary stenosis (PS) -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.

Results and Discussions

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 (TOF, TGA) 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.

Analyzing pregnancy parity, showed that 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	%	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.

Clinical manifestations of CHD, depending on the species, were diverse. With "blue" defects (TOF, TGA, ACD) and PS, clinical symptoms appeared in the first 6 months of life, but some "white" CHDs, such as PDA, VSD, ASD were undetected and diagnosed throughout their lives.

Objectively, when examining patients with "blue" CHD, cyanosis (92.6%), pulsation and swelling of the cervical arteries (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 VSD.

In patients with ASD, 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 TOF and TGA, frequent dyspneacyanotic with characteristic attacks 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.

Conclusions

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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