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Ефименко Оксана Владимировна,
доцент кафедры
«Госпитальной и неотложной педиатрии»
Андижанского Государственного Медицинского института
Город Андижан, Республика Узбекистан
Хайдарова Лола Рустамовна,
старший преподаватель
кафедры «Госпитальной и неотложной педиатрии»
Андижанского Государственного Медицинского института
Город Андижан, Республика Узбекистан
Тешабоев Умиджон Махаммаджонович,
ассистенткафедры «Госпитальной и неотложной педиатрии»
Андижанского Государственного Медицинского института
Город Андижан, Республика Узбекистан
Курбанов Нурулло Кузибоевич
студент магистратуры
кафедры «Госпитальной и неотложной педиатрии»
Андижанского Государственного Медицинского института
Город Андижан, Республика Узбекистан
Якубов Ойбек Базарбаевич
студент магистратуры
кафедры «Госпитальной и неотложной педиатрии»
Андижанского Государственного Медицинского института
Город Андижан, Республика Узбекистан

КЛИНИКО-ИНСТРУМЕНТАЛЬНАЯ ОЦЕНКА ТЕЧЕНИЯ ВПС У ДЕТЕЙ РАННЕГО ВОЗРАСТА В ЗАВИСИМОСТИ ОТ СТЕПЕНИ ТЯЖЕСТИ АРТЕРИАЛЬНОЙ ГИПОКСИИ

Аннотация. *Врождённые пороки сердца являются самой распространённой аномалией развития у детей и остаются ведущей причиной инвалидизации и смертности. Наиболее распространенными врожденными пороками сердца у детей являются ДМЖП, ДМПП и тетрада Фалло. Естественное течение этих пороков сопровождается развитием ряда тяжелых осложнений, вследствие артериальной гипоксии. Высокая летальность у детей с септальными пороками обусловлена застойной сердечной недостаточностью и легочной гипертензией, у детей с ВПС синего типа - диффузной гипоксемией. Низкая обеспеченность артериальной крови кислородом вызывает тяжёлые клинические проявления порока и способствует формированию нарушений ритма сердца, которые могут служить дополнительным фактором риска в прогнозе ВПС.*

Ключевые слова: *врожденные пороки сердца, артериальная гипоксемия, ДМЖП, ДМПП, тетрада Фалло, ЭКГ, нарушения ритма сердца.*

Efimenko Oksana Vladimirovna,
Candidate of Medical Sciences,
Associate Professor of the Department
"Hospital and Emergency Pediatrics"
of Andijan State Medical Institute
Andijan City, Republic of Uzbekistan
Khaydarova Lola Rustamovna,
Senior lecturer Department of
"Hospital and Emergency Pediatrics"
of Andijan State Medical Institute
Andijan City, Republic of Uzbekistan
Teshaboev Umidjon Mahammadzhonovich,
Assistant of the Department of

"Hospital and Emergency Pediatrics»
Andijan State Medical Institute
Andijan City, Republic of Uzbekistan
Kurbanov Nurullo Kuziboevich , Master's student
of the department "Hospital and emergency pediatrics"
of Andijan State Medical Institute
Andijan City, Republic of Uzbekistan
Yakubov Oybek Bazarbayevch, Master's student
of the department "Hospital and emergency pediatrics"
of Andijan State Medical Institute
Andijan City, Republic of Uzbekistan

CLINICAL AND INSTRUMENTAL ASSESSMENT OF THE CURRENT CONGENITAL HEART DEFECT IN EARLY AGE CHILDREN DEPENDING ON THE SEVERITY OF ARTERIAL HYPOXIA

Annotation. *Congenital heart defects are the most common abnormalities in children and remain the leading cause of disability and mortality. The most common congenital heart defects in children are DMZHP, DMPP, and tetrad Fallot. The natural course of these defects is accompanied by the development of a number of severe complications, due to arterial hypoxia. High mortality in children with septal defects is due to congestive heart failure and pulmonary hypertension, in children with CHD of the blue type - diffuse hypoxemia. Low supply of arterial blood with oxygen causes severe clinical manifestations of the defect and contributes to the formation of heart rhythm disorders, which can serve as an additional risk factor in the prognosis of CHD.*

Key words*: congenital heart defects, arterial hypoxemia, DMZHP, DMPP, tetrad Fallot, ECG, cardiac arrhythmias.*

Relevance: Congenital heart disease (CHD) is a congenital defect in the structure of the heart or large vessels, present from birth. Currently, the concept

of CHD has a broader meaning, namely, this pathology should be considered as an anatomical deformity, regardless of when it was detected. (1,2)

Current literature data indicate that most children with CHD have a normal birth weight for gestational age, but may develop significant lags behind their peers at an early age. (3,6)

The prognosis of children with CHD is determined by the type of defect, the degree of arterial hypoxia, the timeliness of detection, and the presence of concomitant pathology. (3,5,8)

The most common are the so-called “big six” defects: VSD, ASD and tetralogy of Fallot. (1,4,6). The natural course of these defects is accompanied by the development of a number of severe complications. Thus, the main causes of death in children with CHD of the pale type are congestive heart failure and the development of pulmonary hypertension. In children with CHD of the blue type, high mortality is due to severe hypoxemia and thromboembolic complications. (5,6,8)

The main mechanisms of violation of cardiac hemodynamics in these defects is the overload of the heart by volume or resistance, the long existence of which depletes the compensatory mechanisms, which contributes to the dilatation and hypertrophy of the heart with the subsequent development of heart failure. Heart failure is accompanied by a violation of systemic hemodynamics, which causes plethora or anemia of the pulmonary circulation with the appearance of clinical manifestations of circulatory hypoxia in VSD and ASD, and diffuse - in Fallot's tetralogy. (1,3,7).

Excessive plethora (hypertension) in VSD and ASD is a risk factor for recurrent pneumonia, and diffuse hypoxia leads to the development of multiple organ dysfunctions. Changes in the structure of the heart chambers due to dilatation and hypertrophy, as well ashypoxic, ischemic and metabolic disorders in the myocardium can contribute to disturbances in the conduction system and

be accompanied by various arrhythmias that aggravate the clinical course of CHD. (1,3,7,8)

In this regard, we have set **purpose** to study the effect of arterial hypoxia on the course of CHD in young children.

Results: Research and collection of material was carried out on the basis of the Andijan Regional Children's Multidisciplinary Center. To solve the tasks of this work, we examined children who received treatment in the cardio-rheumatology department for the period of 2020, and also conducted a retrospective study of case histories.

We included 71 children from 6 months to 3 years old in the research program, of which 72% were children from 1 to 3 years old. By gender, boys predominated (55% versus 45%), both among children under 1 year old and older.

From CHD, depending on the hemodynamic regimes of the functioning of the circulatory system, we selected children with septal defects and hypertension of the pulmonary circulation: VSD (49.4%) and ASD (25.3%). A defect of the cyanotic type in our studies is represented by the multicomponent tetrad of Fallot (25.3%).

Upon admission to the hospital in 81.7% of children, the condition was assessed as severe, due to symptoms of heart failure. II degree. The level of oxygen saturation of capillary blood in all children was determined upon admission to the hospital. So among children, the lowest level of oxygen saturation of capillary blood - 91-84% - was detected in Fallot's tetralogy. In children with septal defects, this indicator was: 94-88% for VSD and 93-91% for ASD.

Frequent respiratory diseases and repeated hospitalizations occurred in all the children we examined. Our results are consistent with literature data.

The nature of complaints in all examined children with CHD depended on the degree of hypoxia. The common and most common complaint, regardless of

hemodynamic disorders, was dyspnea, stated in 88.6% of cases in children with VSD, in 77.8% of patients with ASD and in all children withFallot's notebook. The frequency of dyspnea in children with septal defects was associated with excessive pulmonary blood flow, and in Fallot's tetrad - with arterial hypoxemia due to right-left shunt.

Hemodynamic disturbances in these malformations were reflected and during theand in these children electrocardiographic examination. The results of an electrocardiographic examination, regardless of the degree of arterial hypoxemia, violation of automatism is represented by sinus tachycardia (76%) and sinus tachyarrhythmia (24%), and high electrical activity of the right ventricle was registered in children with defects in the interventricular (20%) and interatrial (50%) septa. Conduction disturbance with different frequency occurred in all examined children. Incomplete blockade of the right bundle branch of His was found in 80% of children with VSD; in 33.3% of children - with ASD; in 50% of children with Fallot's tetrad. Complete blockade of the right bundle branch of His occurred only in children with Fallot's tetrad (16.7%). First-degree atrioventricular block was registered in 3 children (8.6%) with a large defect in the interventricular septum and in one child with Fallot's tetralogy.

Electrocardiographic signs of right ventricular hypertrophy occurred with a frequency of 48.5% in children with VSD; 61.1% - with ASD and in all children with Fallot's tetralogy. Signs of left ventricular hypertrophy were noted in children with VSD (51.5%).

Evaluation of structural changes in the parts of the heart, as well as the size of congenital defects, was carried out according to the results of echocardiography. So, with VSD (35 children), the largest proportion was children with a defect size of 4-8 mm (48.6%), up to 4 mm - 34.3% of children and more than 8 mm - 17.1% of children. The size of the defect ininteratrial septum (18 children), the distribution of children was as follows: up to 5 mm -

61.1% of children, from 6 to 10 mm - 38.9% of children. By the size of the defect in the interventricular septum in children with Fallot's tetrad - 66.7% with a defect size of 6-10 mm, in the rest - more than 10 mm. With the localization of the narrowing of the outlet in the indubular section, 72.2% of children and in 27.8% - the narrowing was located at the level of the pulmonary artery valves.

Pulmonary hypertension in children with septal malformations on radiography was manifested by an increase in the pulmonary pattern along the arterial bed, and in Fallot's tetrad, increased transparency of the pulmonary fields was associated with depletion of the pulmonary pattern. Cardiomegaly was detected in all examined children, and in 74.6% of cases due to an enlarged right ventricle and in 25.4% of cases due to the left ventricle exclusively in children with VSD.

Thus, we have established a relationship between the effect of arterial hypoxia on the course and severity of clinical manifestations of CHD. The lower the supply of arterial blood with oxygen, the more severe the clinical manifestations of the defect and the more pronounced cardiac arrhythmias, which can serve as an additional risk factor in the prognosis of CHD.

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