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EARLY ECHOCARDIOGRAPHIC MARKERS OF CHRONIC PULMONARY HEART DISEASE OF CHILDREN WITH A NEW FORM OF BRONCHOPULMONARY DYSPLASIA

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Abstract

The goal of this study was to improve early diagnosis of chronic pulmonary heart disease in children with a new form of bronchopulmonary dysplasia by identifying the marker role of the end-diastolic diameters of the right ventricle, the right ventricular free wall thickness and the mean pulmonary artery pressure.

The study was conducted at the the Regional centre of diagnosis and treatment of bronchopulmonary dysplasia in children, Regional children's hospital. The study involved 140 patients with a new form of bronchopulmonary dysplasia (main group) and 43 prematurely born children with respiratory disorders, who had not developed bronchopulmonary dysplasia (comparison group). The corrected age of the patients was in the range of 1-36 months.

The study found that the end-diastolic diameter of the right ventricle in children of the main group was significantly higher than that in the comparison group ($p<0.01$). In 67 (47.9±1.9%) patients of the main group the indicators of end-diastolic diameter of the right ventricle were measured over 90 percentile. The diameter of the right ventricle did not exceed 75 percentile ($p<0.001$) in the control group. The right ventricular free wall thickness did not exceed 90 percentile in 27 (13.1±2.3%) children of the main group. The right ventricular free wall thickness did not exceed 90th percentile ($p<0.001$) in the children of the comparison group. A characteristic feature of children with a new form of bronchopulmonary dysplasia was right ventricular dilatation ($KW(n=183)=17.3$; rank 4.1; $p<0.0001$) due to insufficient compensatory hypertrophy of the right ventricular wall in increasing the pressure in the pulmonary circulation ($r=0.165$; $p>0.05$). The correlation between end-diastolic diameter of the right ventricle and the mean pressure in the pulmonary artery was measured to be $r=0.413$; $p<0.05$. The higher the pressure in the pulmonary artery, the larger was the diameter of the right ventricle. Insufficient compensatory hypertrophy of the right ventricular wall in increasing the pressure in the pulmonary circulation, confirmed by the lack of a reliable correlative link between the right ventricular end-diastolic diameter and the right ventricular free wall thickness, was found in the children with a new form of bronchopulmonary dysplasia ($r=0.165$; $p>0.05$).

Persistent right ventricular dilatation can be considered the early markers of the development of the chronic pulmonary heart disease in children with a new form of bronchopulmonary (over 90 percentile) (λ Wilks=0.885; F sup 6.14; $p=0.019$) in the presence of persistent pulmonary hypertension ($\lambda=0.891$ Wilks; F sup 5.9; $p=0.021$).

Keywords: *bronchopulmonary dysplasia, echocardiography, markers, chronic pulmonary heart.*

Introduction

The signs of chronic pulmonary heart disease in an adult patient appear due to reduction of the pulmonary capillary surface area by 5-10%. Its reduction by 15-20% led to a significant right ventricular hypertrophy. Reduction of the pulmonary capillary surface area by more than 30% results in decompensation of chronic pulmonary heart and a relatively quick lethal outcome [*Lewandowski AJ et al., 2013; Moore BD et al., 2014*].

Bronchopulmonary dysplasia (BPD) is a chronic lung disease in children (ICD-10 P27.0).

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Patients with BPD develop chronic pulmonary heart disease. It is a dynamic process that becomes clinically significant only at the later stages of the disease [*Volodin NN, 2010*]. Pulmonary heart disease is precisely one of the main causes of death in children with BPD.

Chronic pulmonary heart disease usually develops in response to a progressive increase of the pulmonary vascular resistance, caused by hypoxia and inhibition of the alveolar capillary formation. The physiological reflex (Euler-Liljestrand reflex) of arterioles' contractions and impaired blood perfusion turns into a pathological one in patients with BPD due to long-term precapillary spasm. Eventually, the pressure in the pulmonary circulation and blood volume per minute increases, and biologically active substances are released. Prolonged spasm of the capillaries leads to anatomical reduction of the vessels, thrombosis and obliteration of arterioles. Hypertrophy, dilatation and myocardial degeneration and right ventricular failure develop in the right heart chambers [*Lemmer CE et al., 2014*].

Clinical manifestations of chronic pulmonary heart disease in children with a new form of BPD are nonspecific. It is often difficult to differentiate between the genesis of dyspnea, tachypnea, and cyanosis. Enlargement of the right heart chambers is determined by percussion at the later stages of the disease. Transthoracic Doppler echocardiography remains the most informative and the safest method of determining the state of the right heart chambers according to the recommendations of the European Association of Cardiologists [*Galie N et al., 2009*].

Patients with bronchial asthma, pneumonia, and tuberculosis are now widely studied to determine the state of their right heart chambers [*Galie N et al., 2009; Lemmer CE et al., 2014*]. Research on the condition of the right heart chambers in children with BPD is too little [*Lewandowski AJ et al., 2013; Vandenheuvel MA et al., 2013*]. And no research dedicated to a new form of BPD can be found at all.

The objective of the study is to improve early diagnosis of chronic pulmonary heart disease in children with a new form of BPD by determining the marker role of the right ventricular end-diastolic diameter (RVEDD), the size of the right ventricular wall thickness, and the mean pulmonary artery pressure.

Materials and Methods

The study was conducted at the Regional centre of diagnosis and treatment of BPD in children, Regional children's hospital. The study involved 183 patients with a new form of BPD at the corrected age of 1 to 36 months. They were divided into 2 groups. The main group was comprised of 140 patients with a new form of BPD, the comparison group included 43 children who had been born prematurely, had a respiratory disorder, but had not developed BPD.

Corrected age was calculated using the formula:

$$A(c)= \frac{-40+A(g) + A\ (ch)}{4}$$

where A (c) - corrected age, A (g) - the gestational age in weeks, and A (ch) - chronological age in weeks.

The measurement of the size of the right heart chambers and the pulmonary artery pressure was performed with the Doppler echocardiography "AU 3 Partner", (Esaote Biomedica, Italy). Dimensions of the right ventricle were compared with the standard indicators depending on the child›s weight [*Beloserov MY, 2004*]. Statistical processing of the data was performed by parametric and nonparametric methods. Early markers of the diagnosis of the chronic pulmonary heart disease in children with a new form of BPD were determined by the discrimination method with STATISTICA 6.0 software.

Results and discussion

The right ventricular diastolic diameter in children of the main group was significantly higher than that in the comparison group ($p<0.01$). Percentile distribution of final diastolic diameter of the right ventricle depending on the weight of the patients is presented in Figure 1.

The indicators of the end-diastolic diameter of the right ventricle were over 90 percentiles in 67 (47.9±1.9%) patients of the main group. The diameter of the right ventricle did not exceed the 75 percentile ($p<0.001$) in the comparison group. Right ventricular dilatation was typical in children with a new form of BPD (KW(n=183)=17.3; rank – 4.1; $p <0.0001$).

More than half of the patients with a new form of BPD had right atrial dilatation, as detected by echocardiography. Percentile distribution of the right ventricular free wall thickness (RVFWTh) based on weight is shown in Figure 2.

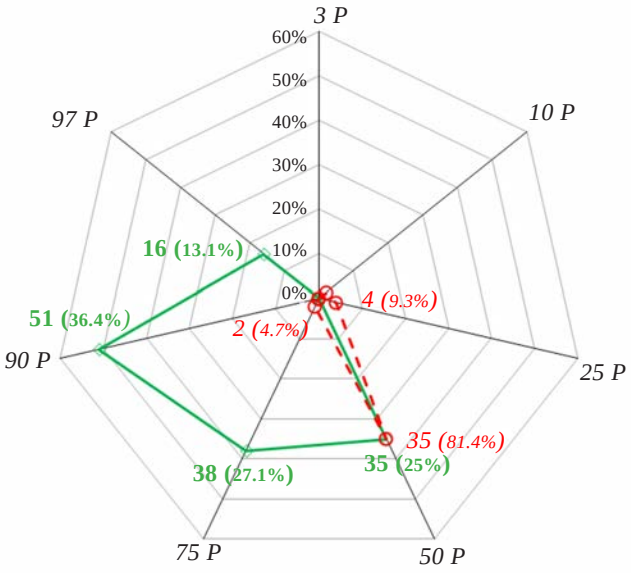

FIGURE 1. *Percentile distribution of right ventricular end-diastolic diameter indicators based on the weight of the subjects.*
NOTES: *main group (n=140) - solid line, comparison group (n=43) - dashed line, P - Percentile.*

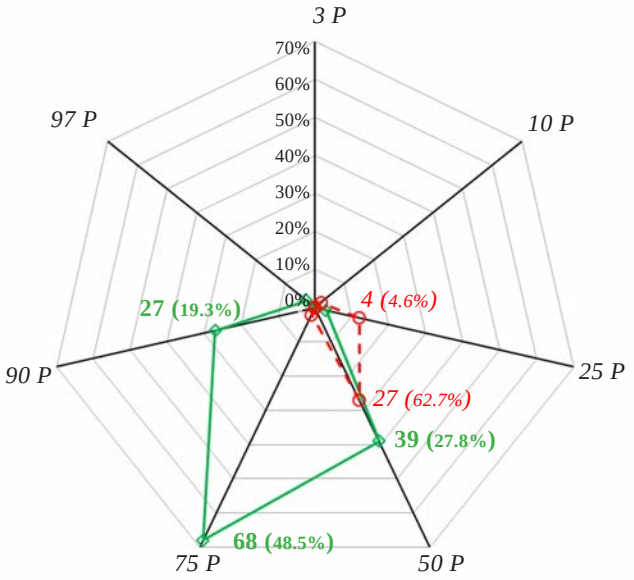

FIGURE 2. *Percentile distribution of the right ventricular free wall thickness depending on the subjects' weight.*
NOTES: *main group (n=140) - solid line, comparison group (n=43) - dashed line, P - Percentile.*

The RVFWTh was over 90 percentile in 27 (13.1±2.3%) children of the main group. The RVFWTh did not exceed the 90th percentile ($p<0.001$) in the comparison group. Since hypertrophy of the right ventricular free wall is inherent in BPD, we suppose that children of the main group had compensated chronic pulmonary heart disease. Meanwhile, development of myocardial hypertrophy of the right ventricle may be impossible in some children, since 40 (28.6±2.1%) patients of the main group with right ventricular dilatation have not developed compensated hypertrophy of the right ventricular free wall.

Analysis of the children's pulmonary artery mean pressure was performed and correlation of the latter with the parameters of the right heart chambers was determined. The mean pulmonary artery pressure in children of the main group was 32.37±1.7 *mmHg*. In the comparison group - 22.0±1.22 *mmHg*, it was significantly less ($p=0.033$).

The correlation between the RVEDD and the mean pressure in the pulmonary artery was determined ($r=0.413$; $p<0.05$). The higher the pressure was in the pulmonary artery, the larger was the right ventricular diameter. However, no reliable correlative link between the indicators of the RVEDD and the RVFWTh was found ($r=0.165$; $p>0.05$). It showed insufficient compensatory hypertrophy of the right ventricular wall in increasing the pressure in the pulmonary circulation.

Discriminant analysis showed that persistent right ventricular dilatation could be considered the early markers of chronic pulmonary heart disease (over 90 percentile) (λ Wilks=0.885; F sup 6.14, $p=0.019$), in the presence of persistent of pulmonary hypertension (λ=0.891 Wilks; F sup 5.9, $p=0.021$) during the first 2 years of life.

CONCLUSION

Thus, the right ventricular dilatation is a characteristic feature in children with a new form of BPD (KW (n=183)=17.3; rank 4.1, $p<0.0001$). Children with a new form of BPD have insufficient compensatory hypertrophy of the right ventricular wall in increasing the pressure in the pulmonary circulation, which is confirmed by the absence of reliable correlative link between the RVEDD and the RVFWTh ($r=0.165$; $p>0.05$).

We believe that persistent right ventricular dilatation is the early markers of chronic pulmonary heart disease in children with a new form of BPD (90 percentile) (λ Wilks=0.885; F SUP 6.14, $p=0.019$) in the presence of persistent pulmonary hypertension (λ Wilks=0.891; F SUP 5.9, $p=0.021$) during the first 2 years of life.

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