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CASE REPORT

Early Detection of Isolated Transient Right Ventricular Hypertrophy in a Term Neonate: the Neonatologist's Role

Evgeniya BABACHEVA[a], Evangelia GIANNOUSIOU[a], Maria FLOROU[b], Apostolia STAMPOULI[a], Maria LITHOXOPOULOU[a*], Christos TSAKALIDIS[a*]

[a]2nd Department of Neonatology and NICU, Aristotle University of Thessaloniki, "Papageorgiou" Hospital, Thessaloniki, Greece

[b]2nd Department of Pediatric Surgery, Aristotle University of Thessaloniki, "Papageorgiou" Hospital, Thessaloniki, Greece

[*]Equally contributing authors

ABSTRACT

Right ventricular hypertrophy (RVH) is a rare finding in the neonatal age. Multiple pathological conditions are associated with the development of RVH, such as metabolic diseases, hereditary disorders and premature closure of the ductus arteriosus during the intrauterine life. Transient isolated RVH may be due to perinatal factors such as perinatal distress and corticosteroid treatment of preterm neonates with bronchopulmonary dysplasia. We report a case of a term neonate, appropriate for gestational age presenting transient hypertrophy of the right ventricle, who was diagnosed with echocardiography applied by neonatologists on the first day of life.

Keywords: transient, isolated right ventricular hypertrophy, acute pulmonary hypertension, neonate.

INTRODUCTION

Right ventricular hypertrophy (RVH) is a rare condition during the neonatal period. It could be a manifestation of various disorders, including metabolic diseases, genetic syndromes, chromosomal anomalies and the premature closure of the ductus arteriosus in fetal life (1-4). In extremely rare cases, the presentation of transient isolated hypertrophy of the right ventricle has been attributed to the temporary effects of perinatal incidents, such as a perinatal hypoxemic-ischemic event (4-6).

We present an uncommon case of transient isolated RVH, possibly caused by the impact of prenatal and perinatal factors. Our case was presented as cyanotic congenital heart disease in the absence of other structural cardiac anomalies and was accompanied by evidence of acute pulmonary hypertension. The diagnosis was set by a

Address for correspondence:
Maria Florou
Second Department of Pediatric Surgery, Aristotle University of Thessaloniki, Papageorgiou General Hospital of Thessaloniki, 56403, Greece
Tel.: +306945295552; email: flwrou.mar@gmail.com, ORCID: https://orcid.org/0000-0002-2166-4338

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neonatologist with experience in performing targeted neonatal echocardiogram (TnECHO) and confirmed by a pediatric cardiologist. ❑

CASE PRESENTATION

A term female infant was born at 37 weeks of gestational age by a 22-year-old mother. It was an appropriate-for-gestational-age infant with 2640 g birth weight who was delivered *via* caesarean section due to two previous deliveries by the same surgical procedure. The maternal and family history was clear, reporting no diabetes mellitus during pregnancy and hereditary disorders. The neonate did not require resuscitation and the Apgar score was 8 at both the first and fifth minute of life. No signs of perinatal stress were observed. At 30 minutes of life, a measurement of oxygen saturation was performed, with noted levels at 90-92%; also, there were no differences between upper and lower extremities and no signs of respiratory distress. When supplemental oxygen was applied with initial FiO_2 of 0.3, increased up to 0.5, oxygen saturation ($SatO_2$) levels at 95% followed. The common causing factors, including upper airway obstruction, electrolyte and metabolic disorders, perinatal infections and pneumothorax were excluded soon after birth. Subsequently, in the absence of respiratory distress and the need for constant oxygen supplementation, the cyanotic congenital heart disease was suspected and a hyperoxia test was applied. There is a slight difference in pO_2 levels before and after the hyperoxia test, supporting a possible cardiac origin of the hypoxemia. The neonate was transferred to the Neonatal Intensive Care Unit (NICU) at 12 hours of age for further investigation and management. Upon admission, the neonate remained hemodynamically stable without respiratory distress. Prostaglandin-E1 (PGE1) therapy was initiated empirically to maintain the ductal patency, since the cyanotic congenital heart disease had not yet been excluded. At the time of admission, before comprehensive cardiologic evaluation, maintaining ductal patency was considered a precautionary measure in case of an undiagnosed duct-dependent cardiac lesion. The TnECHO revealed a severe hypertrophy of the right ventricle, with surprisingly neither other structural cardiac anomalies, or right-ventricle output obstruction (Figure 1). The IVS-systolic was 1.1 cm and the IVS-diastolic 0.8 cm. The tricuspid regurgitation velocity was 3.5 m/s and the corresponding pressure in the pulmonary artery was 60 mm Hg. A small patent ductus arteriosus was detected in the first day of life with diameter of 0.1 cm and a right-to-left shunt. The shunt eliminated during the second day of life and the prostaglandin was then discontinued. Also, signs of pulmonary hypertension were detected, including: suprasystematic increased pulmonary pressure, a D-shaped left ventricleand right-to-left shunting through the foramen ovale and the ductus arteriosus due to high afterload in pulmonary circulation. A complete cardiological investigation by a pediatric cardiologist was performed and confirmed the diagnosis of isolated, right ventricular hypertrophy in association with pulmonary hypertension.

Treatment with sildenafil (phosphodiesterase-5 (PDE-5) inhibitor) was directly initiated as a pulmonary vasodilator, and continued for a total of 18 days. When the pulmonary hypertension was completely eliminated based on clinical and echocardiographic findings, sildenafil treatment was discontinued. Close monitoring and systematic cardiac evaluation by the pediatric cardiologist followed, showing gradual improvement of the echocardiographic findings, until complete resolution of the RVH and the complete decrease of pulmonary vascular resistance by the completion of four weeks of life (Figure 2). The neonate received adequate respiratory support, with the aim of maintaining adequate oxygenation in view of pulmonary hypertension. Respiratory support was provided with supplemental oxygen (FiO_2 45%) during the early neonatal period and was progressively tapered until day 10 of life. Following oxygen discontinuation, the neonate

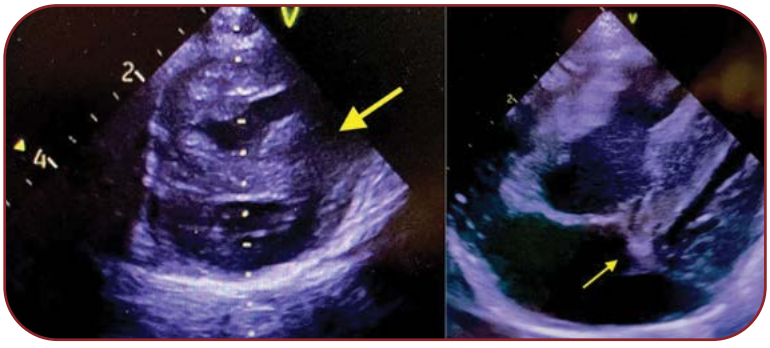

FIGURE 1. Targeted neonatal cardiac ECHO revealing severe hypertrophy of the right ventricle (arrows). The IVS-systolic was 1.1 cm and the IVS-diastolic 0.8 cm.

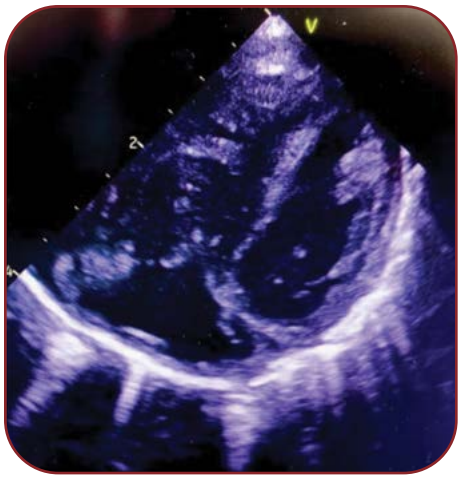

FIGURE 2. Four-chamber echocardiographic view demonstrating interval improvement of cardiac morphology and function during follow-up

maintained adequate respiratory function in room air and remained clinically stable. In consequence of the transient manifestation of the RVH, no screening tests for metabolic diseases and genetic disorders were performed due to the transient nature of the RVH with rapid resolution. ❑

DISCUSSION

Right ventricular hypertrophy (RVH) is a part of the congenital heart diseases such as tetralogy Fallot, pulmonary stenosis and truncus arteriosus (1). Often RVH is also observed in some metabolic disorders and some neonates born to mothers with diabetes mellitus, and may also result from intrauterine closure of ductus arteriosus due to maternal intake of prostaglandin-synthetase inhibitors such as glucocorticoids, non-steroid anti-inflammatory drugs (NSAIDs) and aspirin during pregnancy (2, 7-9). On the other hand, the transient isolated hypertrophy of the right ventricle is an uncommon condition in neonatal age with an unclear etiology (1).

In our case, echocardiographic findings were negative for congenital heart diseases and there was no information from maternal history regarding pathological glucose metabolism and prenatal prostaglandin-synthetase inhibitors intake. Additionally, the neonate had normal blood-glucose levels, no other metabolic and electrolytic disorder and no perinatal distress.

According to the literature, there are very few reports of isolated, transient RVH similar to our patient (1). The isolated presentation of RVH after birth is extremely rare and the causes remain uncertain. One possible explanation for RVH is the pulmonary vascular remodeling, during the transition from fetal to neonatal circulation, resulting in transient elevation of pulmonary vascular resistance, pulmonary hypertension and secondary right ventricular pressure overload (4, 6). The echocardiographic findings like suprasystemic pulmonary pressures, right-to-left shunting through both the patent foramen ovale and ductus arteriosus and septal flattening with D-shaped left ventricle, support this hypothesis (10).

Another alternative explanation represents a partial fetal ductal constriction occurring during late gestation. Prenatal ductal constriction may increase right ventricular afterload and promote right ventricular hypertrophy even in the absence of severe prenatal or perinatal clinical manifestations. Although no abnormalities were reported in mothers' personal history, the pregnancy was not carefully monitored and no prenatal ultrasounds were performed. Furthermore, no history of maternal exposure to non-steroidal anti-inflammatory drugs was identified; also, information regarding maternal consumption of polyphenol-rich foods and beverages was not systematically collected. Consequently, a prenatal contribution cannot be completely excluded (8-11).

Vaillant et al reported three cases of transient, hypertrophic cardiomyopathy in neonates because of myocardial ischemia, due to acute fetal distress. The authors explained the development of RVH with mechanical, metabolic and calcium based ionic hypothesis (5). In our case, there were no signs from perinatal history and clinical presentation of the infant to suggest perinatal distress or delayed postnatal adaptation. So, the exact pathophysiological mechanism in our patient remains unclear. Given the rapid regression of both pulmonary hypertension and right ventricular hypertrophy during follow-up, we hypothesize that a transient increase in right ventricular afterload, either during fetal life or the immediate postnatal transitional period, may have played a central role in the pathogenesis of this condition. The findings suggest that transient maladaptation of the pulmonary circulation during the early neonatal transition may have resulted in increased pulmonary vascular resistance, acute pulmonary hypertension and secondary right ventricular pressure overload, leading to transient RVH.

Based on the existing data, the isolated transient form of RVH is characterized by spontaneous resolution within 8-10 weeks, along with pul-

monary hypertension and hypertrophy improvement. Interestingly, in our case, the pathological echocardiographic findings fully resolved by four weeks of life. Serial echocardiographic examinations were performed during hospitalization and outpatient follow-up. Although treatment is not mandatory in many cases, our patient received a phosphodiesterase-5 inhibitor which was effective in reducing pulmonary vascular resistance. Sildenafil promotes pulmonary vasodilation by increasing intracellular cyclic-GMP in pulmonary smooth muscle and has been widely used for neonatal pulmonary hypertension. In this case, gradual weaning of oxygen supplementation and normalization of echocardiographic findings support its therapeutic benefit. The complete normalization of echocardiographic findings was confirmed at the four-week follow-up examination. ❑

CONCLUSIONS

Knowledge and training of neonatologists in performance of cardiac echocardiography (ECHO) play a crucial role in early identification and management of structural and functional cardiac anomalies, particularly when immediate evaluation by a pediatric cardiologist is not possible. Definitive diagnosis is established by a pediatric cardiologist. Therefore, close collaboration between the two specialties is essential. In our case, early detection of RVH helped in achieving cardiocirculatory stability and adequate oxygenation, leading to improvement and sooner resolution. ❑

Conflicts of interest: none declared.

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Ethics statement: Informed consent was obtained from the patient's parents.

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