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Echocardiographic Diagnostics of Myocardial Infarction in Newborns

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Timely and correct diagnosis of myocardial infarction in neonates is impossible without the state-of-the-art instrumental and highly informative methods of study, the key among which is echocardiography. The article presents a clinical observation of a 3-day-old child referred to examination due to insufficient blood flow requiring cardiotonic support. Echocardiography revealed a hyperechogenic dyskinetic locus in the apical segment of the right ventricle (post-infarction cicatrical change), local pericardial effusion in the same projection, a hyperechogenic floating mass (thrombus) in the apical segment of the right ventricle; together with electrocardiographic findings, this helped to establish presence of myocardial infarction and intensity of the hemodynamic disorder. Transthoracic echocardiographic findings helped to correctly interpret clinical presentation of heart failure and identify the cause of the patient's dependence on cardiotonic support.
Keywords: *myocardial infarction in neonates, transthoracic echocardiography, diagnosis, electrocardiography, functional diagnosis, ultrasound diagnosis.*
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INTRODUCTION

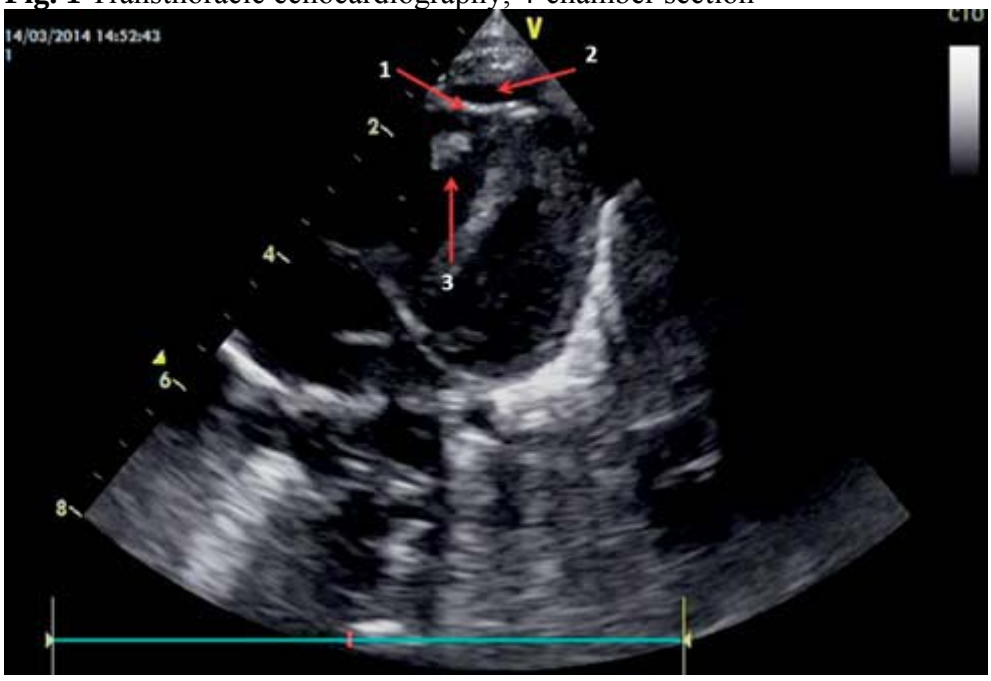
As of today, diagnostics of myocardial infarction in newborns is far from being perfect, as it is mostly based on electrocardiography-obtained data (ECG). However, in some cases the electrophysiological heart analysis cannot provide exhaustive data on the localization of the ischemic process and intensity of hemodynamics abnormalities.
Let us cite an example that shows why it is necessary to conduct echocardiographic analysis alongside with standard ECG and laboratory tests to obtain comprehensive data on the intensity of hemodynamics abnormalities and to assess the efficiency of treatment.

CASE MONITORING

Patient K. was admitted to the Scientific Center of Children's Health clinic at the age of three days. Postnatal conditions critical due to respiratory disorders, i.e. grade 3 respiratory failure as a result of severe asphyxia suffered upon birth. After born, pulmonary hemorrhage was identified twice in the child, hemodynamics stabilized by the third day of life. Until that age, mechanical ventilation had been applied. Prior to the encounter, the patient had been treated with dopamine, dobutrex, ceftriaxone, vancomycin, vicasol, dicynone, tranexam, contrical, Gabriglobin no 2, and

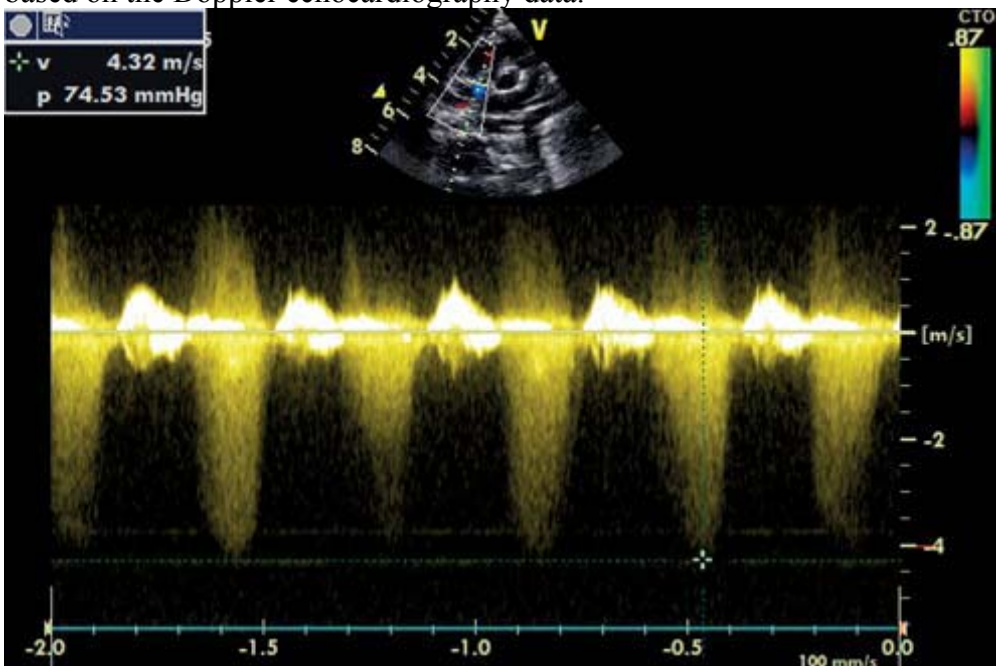
0(I) fresh frozen plasma transfusion. The patient had been fed artificially. *Auscultation* identified cardiac murmur.
Upon encounter, circulatory failure persisted and required cardiotonic support.
ECG data identified tachycardia of more than 170 bpm, right-ventricular myocardium potentials predominant, myocardium overload could not be excluded. The *Q* wave present in the III and aVF derivations as well as the *T* wave did not allow to exclude infarction-like alterations. There was noted non-specific intraventricular block and intense myocardium repolarization abnormalities.
Due to the circulatory failure that required cardiotonic support, pulmonary hemorrhage and insufficiency of instrumental data that did not allow for correct interpretation of the clinical picture, as well as due to the abnormalities of the intracardiac hemodynamics, we decided the patient should undergo echocardiographic study.
Transthoracic echocardiography was carried out using the portable ultrasonic scanner Vivid q(General Electric, USA). The following data were obtained by such analysis: local systolic function abnormal, with hyperechogenic dyskinetic locus in the apical segment of the right ventricle (post-infarction scar), local pericardial effusion in the same projection, hyperechogenic movable mass (thrombus) in the right-ventricular apical segment (Fig. 1). Coronary arteries outlets were normal. Persistent pulmonary hypertension of newborns. Open arterial duct, left-to-right shunt, open oval window. 1st degree tricuspid regurgitation (Fig. 2).
The data obtained enabled a correct interpretation of cardiac failure clinical picture and also allowed to identify why the patient relied on cardiotonic support.

Fig. 1 Transthoracic echocardiography, 4-chamber section



Note. 1 — hyperechogenic dyskinetic zone of the apical segment of the right ventricle; 2 — local pericardial effusion in the infarction locus projection; 3 — hyperechogenic movable mass in the apical segment of the right ventricle.

Fig. 2. Transthoracic echocardiography; hemodynamic parameters of tricuspid regurgitation, based on the Doppler echocardiography data.



DISCUSSION

Myocardial infarctions (MI) in newborns are rare. Causative factors that lead to the development of myocardial infarction in newborns and infants are various. Medical literature contains data on MI development in infants with innate hearth and coronary arteries abnormalities, severe perinatal hypoxia with the central nervous system damaged, respiratory distress syndrome, bronchopulmonary dysplasia, and primary pulmonary hypertension. Paradoxical embolism from the umbilical vein can also be a leading causative factor of myocardial infarction in newborns. Coronary arteries embolism may develop during the labor or upon the operation of blood replacement transfusion from the catheterized umbilical vein. In such cases, a thrombus may be formed inside the branch of the coronary artery as a result of coronary vessel inflammation or a viral infection. Most such situations had unfavorable outcomes, and the necrotic focus was determined morphologically. In newborns with normal chamber and coronary vessel structure, myocardial infarction may develop as a result of severe perinatal hypoxia survived. Severe hypoxia may sometimes cause fine-focal intramural infarctions, and disseminated intravascular blood-clotting is the leading factor thereof [1, 2].

In recent years, myocardial ischemia is increasingly often defined as transient or ischemic myocardial dysfunction. Specific ECG alterations like the *ST* segment depression or the *T* wave inversion do not allot to tell ischemia from the already present necrosis [3, 4].
Microscopically acute MI in newborns looks like an area of coagulatory necrosis with neutrophilic infiltration and marginal vasodilatation. During the recovery stage, the damaged myocardium is replaced with well-vascularized fibrotic tissue, around which hypertrophic muscular fiber is formed, which enables the required contractility of the cardiac muscle. Neonatal infarctions often end in myocardial calcification, which is clearly seen on the thoracic cage roentgenogram.

Clinical Picture

In the clinical picture of a neonatal myocardial infarction, pallor, tachypnea, tachycardia, and liver enlargement are the most prominent tokens. Cardiac murmur and peripheral endemas are

rather unusual. The intensity of clinical symptoms is determined by the damaged area of the cardiac muscle as well as by the extent to which the contractility of the ventricular myocardium is compromised. A major myocardial infarction is accompanied with the child's anxiety, cries of irritation, moaning, and cachexia. The skin becomes dead-like pale. In case of a severe myocardial infarction, the child's face looks like "dying". Sometimes, cyanosis of extremities is identified alongside with skin paleness. In some cases, skin paleness, acrocyanosis, or total cyanosis are manifested periodically, i.e. several times a day. Little to no suction. Physiological reflexes are either suppressed or cannot be triggered at all. Tachycardia of 160 to 200 bpm is noted almost all the time. Peripheral heart rate pulsations are weak or even not palpable. Cardiac arrhythmia in the form of extrasystoles or paroxysmal tachycardia is registered in some patients. The patient breathes at an increased rate, non-deeply, and with the mouth open; the rate reaches up to 90/min. In some cases, the respiratory rate does not exceed the age norm or is at the upper margin thereof. The liver is enlarged and stretched by 3-4 cm off the edge of the costal margin. In case myocardial infarction progresses quickly and is fatal, the liver does not enlarge and is palpable at the edge of the costal margin. There have been described cases of myocardial infarction, where heart failure symptoms were accompanied by temperature increase in the newborn [2, 6, 7].

Diagnostics

Ischemic heart disease is diagnosed by means of functional diagnostics, the leading one of which is ECG. If myocardial infarction is localized intramurally in the ECG, the amplitude of the main *QRS* waves is decreased as a result of decreased general contractility of the ventricular myocardium; or, a pathologic *Q* wave may appear with a duration exceeding 0.02s and amplitude exceeding 1/4 of the *R* wave amplitude in the same derivation. If the cardiac muscle is damaged severely, several adjacent thoracic derivations show a *QS*-type complex or a *Q*-type complex with no *R* wave, which indicates transmural damage with a portion of the muscular wall being excluded from the contraction process. Sometimes, such "exclusion" phenomenon is registered when the amplitude of the main *QRS* waves is decreased compared to the adjacent ventricular complexes, in one or more thoracic derivations. In this case, the morphology of the ventricular complex is preserved. The dynamics of myocardial infarctions, based on ECG data, resembles that in adults in many aspects. Over the first day since the onset of myocardial infarction in a newborn, ECG shows a rise of the *ST* segment above the isoline, whilst the amplitude of the *R* wave in the same derivations is decreased. In 36 hours, the following aberrations add up: ventricular qr-type *QRS* complexes emerge at the edge of the ischemic area, whilst the center of the damaged site shows a *QS*-shaped ventricular complex. After three days since the onset of myocardial infarction, the *ST* segment reverts to the isoline, whilst the *QS*-type ventricular complex is preserved at the center of the damaged site. After a month, the derivations reflecting the damaged area show the *ST* segment positioned at the isoelectic line, the ventricular complex becomes *rS*-shaped, the *T* waves decrease in amplitude and become positive [2, 6, 7].
Echocardiography is a very informative myocardial infarction diagnostics method. In case of MI, cardiac muscle contractility is disordered first. Echocardiography enables real-time identification of areas of abnormal myocardium contractility against the background of normal contractility areas; it helps measure contractility reduction in separate sites of the ventricular wall, which is manifested as hypokinesia, dyskinesia, or akinesia. Echocardiography allows to assess the global systolic function of the left ventricle as well as the intracardiac heart hemodynamics.
In case of left-ventricular MI, the roentgenogram of the thoracic cage taken in a few hours after the heart is damaged reveals cardiomegaly with the vascular bed overflown as a result of reduced left-ventricular contractility. In case of right-ventricular MI, it reveals hypoperfusion of pulmonary vessels due to right-ventricular failure.
myocardial infarction is accompanied by multiplication in the activity of myocardial creatine phosphokinase isozyme in blood serum. If myocardial infarction is not related to survived

hypoxia, determination of serum aspartate aminotransferase and alanine aminotransferase is significant for newborn diagnostics. Analyzing the activity of lactic dehydrogenase (LDG1, LDG2) fractions may add important data to other test results. The activity of LDG1 and LDG2 is multiplied in 48-72 hours after the cardiac muscle is damaged and is gradually reverted to its initial levels over a week, which can serve as a test for retrospective myocardial infarction assessment.

Since this pathology is rare in newborns, data on post-infarction ventricular arrhythmiae are limited. Complications described include ventricular tachycardia and ventricular fibrillation over the first 24 hours since the onset of myocardial infarction. Post-natal mortality rate among infant with acute MI is high (up to 60%) and does not depend on the cardiac pathology type. Post-infarction arrhythmia is accompanied by high mortality rates, reaching up to 80%. Ventricular arrhythmia is almost never recurrent in patients who survived myocardial infarction. In some cases, secondary mitral regurgitation develops after left-ventricular myocardial infarction that involved the posteromedial papillary muscle, which is due to the fibrosis of the damaged muscle with subsequent heart failure that progresses over several months. S. Kaminer described a case of myocardial infarction combined with hemorrhagic pericardial effusion and subsequent cardiac tamponade [2, 6, 7].

CONCLUSION

Thus, the role of echocardiography in assessing intracardiac hemodynamics, global and local systolic function of the ventricles, and anatomic peculiarities is undisputable, as this method enables quantitative and qualitative assessments of the current cardiovascular conditions, which allows to interpret the patient's status correctly and develop a proper treatment algorithm for the early neonatal period.

CONFLICT OF INTEREST

The authors of this article have declared absence of reportable financial support / conflict of interest.

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