A CASE OF RIGHT ATRIAL THROMBOSIS IN A NEWBORN INFANT

UDC 616.125.3–005.6–053.31
Received 15.05.2013

K.A. Kopylova, Postgraduate, the Department of Hospital Pediatrics¹;
A.V. Prakhov, D.Med.Sc., Professor, Head of the Department of Obstetrics, Gynecology and Emergent Pediatric Care²;
T.A. Zapevalova, Head of the Newborn and Premature Infant Pathology Department³;
L.E. Egorskaya, Head of the Department of Functional Diagnostics³;
O.V. Khaletskaya, D.Med.Sc., Professor, Head of the Department of Hospital Pediatrics¹

¹Nizhny Novgorod State Medical Academy, Minin and Pozharsky Square, 10/1, Nizhny Novgorod, Russian Federation, 603000;
²Military Medical Institute of Federal Security Service of the Russian Federation, Nizhne-Volzhskaya naberezhnaya, 1/1, Nizhny Novgorod, Russian Federation, 603001;
³Children city clinical hospital No.1, Gagarin Avenue, 76, Nizhny Novgorod, Russian Federation, 603081

The risk of neonatal thromboses consists in the development of severe complications (pulmonary artery thromboembolia) and a patient’s death. The intensity of complications can vary from asymptomatic to fatal thromboses. With the implementation of ultrasound technique into diagnostics, thromboses in newborn infants are recorded more frequently. We described a case of the right atrial thrombosis in a newborn infant. Heparin therapy every 7 days under echocardiography and hemostasis indices control was chosen as the treatment modality. Heparin was administered for 11 days till complete thrombolysis.

Key words: newborn thrombosis; heparin therapy; thrombolysis.

Venous and arterial occlusions of the vessels in childhood are very rare events and occur spontaneously, mainly, in the neonatal period with the following increase of incidence at the beginning of the pubertal period. As a rule, a combination of a number of congenital and acquired factors is necessary for thrombosis to occur in children. Presently a variety of hereditary factors are certain or likely to lead to thrombosis development: inherited decrease of physiological anticoagulant activity — proteins C, S, antithrombin III and others [1].

A large number of gene polymorphisms of the hemostasis system has been studied. The role of the following polymorphisms has been proved: mutation of factor V (Leiden) (G1691A), factor II (2021A) [2]. Gene polymorphism of methylenetetrahydrofolate reductase (MTHFR) (C677T) synthase methionine reductase (MTRR) (A66G) involves homocysteine metabolism disorder and elevation of its concentration in the blood [3, 4]. High concentration of homocysteine damages endothelium and causes thrombosis. The role of procoagulant activity increase — factors VIII, Willebrand, fibrinogen — in the processes of pathological thrombus formation is being studied [2].

Besides, acquired factors such as heavy somatic and infectious diseases, malignant tumors, systemic inflammatory diseases of the connective tissue, hemolytic anemias, hyperhomocysteinemia, increase of lipoprotein level in the blood — contribute greatly to thrombus formation in children [5]. Lately, great attention in the pathogenesis of thrombosis in children is being paid to connective tissue (mesenchymal) dysplasias [6], and others.

No less important in the development of thrombosis are vascular catheters. Intravascular manipulations are certain to be a powerful stimulus of thrombus formation, the triggering mechanism, which in the absence of the sufficient limiting potential leads to the excessive thrombus growth, occlusion of the vessels, and disturbance of the organ blood flow [7]. Due to the commonly used catheterization of the umbilical and subclavian veins and implementation of diagnostic ultrasound examinations, thrombosis in the first weeks of life is registered more and more often.

The intensity of neonatal thrombotic disorders may vary considerably — from asymptomatic thrombosis to fatal ones. Diagnosis of thrombosis in newborns is rather difficult, since there are no reliable criteria in the clinical...
A Case of Right Atrial Thrombosis in a Newborn Infant

US scanner Sim-7000 Chelenge (Russia), echocardiography was performed using 7-th day of life.

Cerebral ischemia of moderate degree, exudative pericarditis. Functioning arterial duct. Hydropericardium across the anterior wall of the right ventricle — up to 5.5 mm, up to 4 mm — at the top. In the right atrium cavity there was an echogenic formation sized 11x9x8 mm with irregular, clear contour near the upper third of the atrium, which did not float into the cavity.

It was necessary to differentiate a thrombus from the tumor process in the right atrium. The following facts evidenced in favor of the thrombus: the changed rheological properties of blood in the patient (during 2 weeks it was impossible to make coagulogram due to the formation of the blood clot in the needle), long-term application of the umbilical catheter, and availability of ischemic damage of the brain as a high risk factor of thrombus formation, which may be accompanied by the changes in rheological blood indexes.

Thus, on the basis of the history-taking data (hypoxia, long-term application of the catheter), objective examination and investigations (changed rheological blood properties, echocardiography findings) the following diagnosis was made: “Cardiopathy. Right atrium thrombus. Closing arterial ligament. 0 degree of blood circulation insufficiency”.

The danger of this situation is in the possibility of pulmonary embolism development, appearance of heart insufficiency signs and fatal outcome for the patient.

Because of the hypercoagulation hemostasis status, on the 12-th day of life it was decided to start heparinotherapy subcutaneously in the dose of 50 units/kg/day. Echocardiography and control of blood coagulation indexes were made every 7 days (See Figure).

Heparinotherapy was carried on for 11 days and completed by a full lysis of the thrombus and reduction of hydropericardium. Indexes of hemostasis system after heparinotherapy returned to normal values.

Echocadiogram of newborn Sch.: in the cavity of the right atrium an echogenic formation 9x6x4 mm in size with irregular, clear contours, without floating into the cavity, is visualized (at the 2-nd day of heparin treatment)
The infant was discharged from the in-patient unit at the age of 1 month in the satisfactory condition.

The study complies with the Declaration of Helsinki (the Declaration was passed in Helsinki, Finland, June, 1964, and revised in October, 2000, Edinburg, Scotland). Written informed consent was obtained from every patient.

The success in the treatment of this case was due to two factors: correctly and timely diagnosing thrombosis of the right atrium and performing the proper therapy for thrombus resolution.

The experience gained allows us to recommend the following tactics of examination, prevention and management of local thrombosis in newborns to avoid similar situations:

- the condition of hemostasis system in newborns can be determined on the basis of the following informative minimum: blood clotting time and bleeding duration, thrombocyte count, active partial thromboplastin time, thrombin time, prothrombin time and prothrombin index, fibrinogen count;
- taking into account a high occurrence of hereditary determined thrombosis it is necessary to examine close relatives (first of all, parents, sisters and brothers) for protein C, S activity, antithrombin activity, concentration of serum homosteine, and if needed, to carry on molecular genetic investigations;
- after the introduction of any central catheter to add 3–5 units/kg/day of heparin into the infusion medium;
- on the 3–5th day after each catheterization to perform control US examination of the cardiovascular system and hepatic vessels;
- in case of local thrombosis and absence of data on the syndrome of disseminated intravascular blood coagulation, heparin in the dose of 10–50 units/kg/day should be administered subcutaneously, if there is impaired microcirculation — in combination with pentoxifylline;

US control of the heart is to be made once in 5–7 days; heparin should be discontinued gradually during 3 days after thrombus lysis;

average thrombolysis time in adequate heparin dose takes 7–10 days.

**Study Funding and Conflict of Interest.** This study was not supported by any financial sources and there is no topic specific conflict of interest related to the authors of this study.

**References**