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CLINICAL CASE: FETO-FETAL TRANSFUSION SYNDROME IN A NEWBORN

This article presents a clinical case of feto-fetal transfusion syndrome (FFTS) in a newborn.

Feto-fetal transfusion syndrome (FFTS) is a severe complication of monochorionic multiple pregnancies that requires timely and accurate diagnosis, followed by fetoscopic laser coagulation of anastomoses to preserve the pregnancy. The presented clinical case of FFTS in a newborn highlights the importance of neonatologists and intensive care specialists being aware of the clinical features, diagnosis, and treatment of this condition. The development of FFTS in this case was due to a shared placenta, which likely led to the formation of transplacental vascular communications and circulatory imbalance between the placental vascular systems of the twin fetuses.

Keywords: Feto-fetal transfusion syndrome, newborn, donor, recipient, prematurity, twins, anemia-polycythemia syndrome.

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Introduction. Feto-fetal transfusion syndrome, or Twin-to-Twin Transfusion Syndrome (TTTS), is a severe complication of monochorionic multiple pregnancies associated with transplacental vascular communications and circulatory imbalance between the placental vascular systems of the twin fetuses [2].

All twin pregnancies should be considered at risk for FFTS until the type of placentation is determined [4]. The primary diagnostic method is fetal ultrasound. In the first trimester, signs indicating a high risk of developing this syndrome include discordance in nuchal translucency thickness, abnormal blood flow in the ductus venosus, and a size difference between the fetuses of more than 25% [2, 7].

In the presence of significant vascular anomalies in the placenta, hemodynamic relationships develop between the donor and recipient fetuses, leading to a disproportion in circulating blood volumes (CBV). Due to unbalanced transfusion, the donor fetus develops hypovolemia and anemia, along with growth restriction. A critical reduction in CBV is accompanied by progressive oliguria and anuria, severe oligohydramnios, which impedes normal lung maturation, and a high likelihood of antenatal fetal death. In the recipient fetus, the disproportionate circulation leads to a sharp increase in CBV, resulting in polycythemia and hypertrophic cardiomegaly. Hemodynamic decompensation leads to congestive heart failure. Increased renal blood flow and urine production lead to polyhydramnios, which increases the risk of premature rupture of membranes and preterm labor [2].

Although anastomoses and, consequently, blood shunting between fetuses occur in all monochorionic twins, FFTS typically develops only in diamniotic monochorionic twins. This is likely because monochorionic pregnancies have more bidirectional superficial anastomoses than diamniotic ones [3].

With expectant management, perinatal mortality in FFTS reaches 95% [1, 5, 6]. Treatment options for FFTS include fetoscopic laser coagulation of placental anastomoses, amnioreduction, selective reduction of one twin, or termination of pregnancy. Factors influencing the choice of treatment include gestational age, cervical length, and technical limitations for fetoscopy (placental location, cord insertion sites, and maternal anatomy).

Objective: To describe a clinical case of feto-fetal transfusion syndrome in a newborn.

Materials and methods: A retrospective analysis of the medical records of a patient in the neonatal intensive care unit (NICU) of the perinatal center of RBN№1-NCM was conducted.

Clinical case. A newborn boy from the 5th pregnancy and 4th delivery. In the first half of the pregnancy, the mother had a mild case of COVID-19 at 16 weeks without fever. She was treated on an outpatient basis with antiviral medications (Grippferon, Arpeflu).

At a routine ultrasound at 14.5 weeks, FFTS was suspected, and the mother was sent home with recommendations for weekly follow-up ultrasounds with fetometry.

At 18-19 weeks, she was hospitalized at the Central District Hospital. Ultrasound revealed a threatened miscarriage with a shortened cervix (<25 mm), abnormal blood flow in the uterine arteries, monochorionic diamniotic twins, and placental insufficiency: Doppler showed reversed diastolic flow. Oligohydramnios in the first fetus (amniotic fluid index <5 cm). Intrauterine growth restriction (IUGR) in the first fetus below the 10th percentile. The woman was referred to the Medical Genetic Center of GAU RS(Y) Republican Hospital №1, but she did not attend the consultation.

A telemedicine consultation was conducted with the National Medical Research Center for Obstetrics, Gynecology, and Perinatology named after Academician V.I. Kulakov. The recommendation was to monitor Doppler and maximum vertical pocket weekly until 27 weeks, with fetometry every 2

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weeks. No intervention was indicated at this stage.

At 23 weeks, ultrasound revealed: monochorionic diamniotic twins. IUGR in the first fetus below the 10th percentile. Placental insufficiency grade 1A. Marginal cord insertion in the first fetus. Pyelectasis in both fetuses. A tendency toward polyhydramnios in the second fetus. The woman was advised to have weekly ultrasounds, but she refused due to difficulty traveling from the district, citing icy conditions. She agreed to ultrasounds every 2 weeks.

At 31 weeks, a routine ultrasound revealed: monochorionic diamniotic twins at 31.2 weeks. Both fetuses in cephalic presentation. Placental insufficiency grade 2. Antenatal death of the first fetus. Tachycardia in the second fetus. Pericardial effusion in the second fetus. The woman was urgently referred to GAU RS(Y) Republican Hospital №1 for emergency delivery.

Histology of the placenta showed no inflammatory infiltration in the membranes or cords, trophoblastic dysfunction, and reduced vascularization. Vascular anastomoses were present.

Emergency delivery was performed at 31.2 weeks. The first twin, a boy, was delivered at 15:11, stillborn, weighing 1300g, length 41cm. On examination, the skin was burgundy-colored, with tight cord entanglement around the body. Autopsy findings: the cause of antenatal death of the first twin at 31.2 weeks was intrauterine hypoxia due to placental transfusion syndrome. Pathology of the fetus: first twin of monochorionic diamniotic twins. Tight cord entanglement around the body. Placental pathology: monochorionic diamniotic placenta. Trophoblastic dysfunction, reduced vascularization. Vascular anastomoses. Chronic placental insufficiency, mixed type. Edema of Wharton's jelly in the second cord.

The second twin, a boy, was born at 15:12, alive, premature, weighing 1540g, length 41cm, Apgar score 4/7. Condition was very severe at birth. He was admitted to the NICU from the operating room on nasal CPAP, accompanied by the NICU team in a transport incubator, with a preliminary diagnosis of respiratory distress syndrome (RDS) grade II, FFTS, donor? Prematurity 31.2 weeks. Congenital anemia at birth. Respiratory failure grade II. On admission, the infant was placed in a heated incubator and connected to an "IF" ventilator in "Biphasic" mode.

The infant's condition was severe due to severe RDS, FFTS (anemia-polycythemia syndrome), cardiovascular failure grade II A-B, respiratory failure grade III, severe congenital anemia, prematurity at 31 weeks, and very low birth weight.

Consciousness was moderately depressed. Pain score on N-PASS: 3. Anterior fontanelle was not tense. No seizures were observed. Neonatal reflexes were depressed. Muscle tone was reduced. Visible mucous membranes were pale pink, clean, and moist. Skin was pale pink, clean, and moderately moist. Warm to touch. Extremities were warm. Generalized edema was present. Breathing was assisted by mechanical ventilation; auscultation revealed uniform air-oxygen flow noise throughout all lung fields, with transmitted rales. Respiratory rate: 59/min. Silverman score: 3. Hemodynamics were relatively stable. Heart sounds were muffled and rhythmic. Heart rate: 114-118/min, with a tendency toward bradycardia. Microcirculation was not impaired. On total parenteral nutrition. Abdomen was soft, not distended, and accessible to palpation. Bowel sounds were present. Liver and spleen were not enlarged. No stool on admission. Urination was free into the diaper, with a diuresis of 13.4 ml/kg/h, indicating polyuria.

Echocardiography on day 0 revealed: congenital heart defect. Patent ductus arteriosus (0.29 cm), atrial septal defect (0.16 cm). Cardiac chambers were not dilated. Left ventricular contractility was normal. Ejection fraction: 86%. Abdominal ultrasound: a thin strip of free fluid near the spleen. Chest and abdominal X-ray: hypoventilation of the left lung.

On the first day of life, the infant's condition worsened due to respiratory failure, with a Silverman score of 5, increasing oxygen requirements, and tachypnea up to 90/min. The infant was switched to high-frequency oscillatory ventilation (HFOV) on a Leoni+ ventilator. HFOV parameters were adjusted: amplitude was reduced by 10%. A recruitment maneuver was performed. After 10 hours, due to the ineffectiveness of the recruitment maneuver and the infant's condition (hypotension, tachycardia), the infant was switched to S-IMV mode on the Leoni+ ventilator. Echocardiography revealed: congenital heart defect. Muscular ventricular septal defect (0.12 cm). Atrial septal aneurysm with shunt (0.36 cm). Patent ductus arteriosus (0.22-0.28 cm). Signs of pulmonary hypertension grade 1-2. Right ventricular hypertrophy. Mitral and tricuspid valve regurgitation grade 1. Mild dilation of the right ventricle, right atrium, and pulmonary artery. Ejection fraction: 75.4%. Neurosonography showed moderate periventricular hypoechogenicity.

Laboratory tests revealed: severe anemia with hemoglobin of 94 g/L. Blood transfusion with individual matching was indicated. Biochemical blood tests showed elevated liver enzymes and hyperbilirubinemia.

On the second day, heart rate increased to 200-215/min, and blood pressure reached 92/74/55 mmHg. Echocardiography showed: patent ductus arteriosus 0.18-0.21 cm. Left atrium: 1.2 cm, not dilated. Right ventricle: 0.92 cm, mildly dilated. Right ventricular anterior wall thickness: 0.34 cm. Mild right atrial hypertrophy: 1.4 cm, not dilated (Kushner). Estimated systolic pressure in the right ventricle: 40-42 mmHg. Pericardium: separation of pericardial layers up to 0.28 cm near the left ventricular apex, up to 0.27 cm near the right ventricular anterior wall, and up to 0.25 cm. Left ventricle (end-diastolic dimension 1.54 cm, end-systolic dimension 0.9 cm) was not dilated. Interventricular septal motion was normal. End-diastolic volume: 6.5 ml, end-systolic volume: 1.6 ml. Ejection fraction: 75.3%, fractional shortening: 40.2%.

Chest and abdominal X-ray: hypoventilation of the left lung. Uneven pneumatization of the intestines. Congenital heart defect. Cardiac shadow enlargement. Elevated high-sensitivity troponin: 74,200 ng/L.

On the third day, abdominal ultrasound revealed pyelectasis. After blood transfusion, red blood cell parameters improved, with hemoglobin of 121 g/L.

A telemedicine consultation was conducted with the National Medical Research Center for Obstetrics, Gynecology, and Perinatology named after Academician V.I. Kulakov. Recommendations included continuing the protective treatment regimen, respiratory therapy with monitoring of acid-base status and blood gases, analgesia, and antibiotic therapy with monitoring of inflammatory markers at 48-72 hours of life to confirm the diagnosis and decide whether to continue or stop antibiotics. Massive vasopressor and inotropic therapy was continued with blood pressure and echocardiography monitoring, considering pulmonary hypertension in the infant with FFTS. Therapy was adjusted with levosimendan 0.1-0.2 mcg/kg/min, dobutamine was gradually discontinued, and dopamine dose was reduced to 5-7 mcg/kg/min.

On the fifth day, blood pressure normalized, and heart rate decreased to moderate tachycardia of 180/min. Neurosonography revealed subependymal cysts in the left thalamo-caudal notch in the cyst formation stage. Periventricular hyperechogenicity.

On the sixth day, the infant was conscious, actively resisting the ventilator, and agitated. Inotropic therapy was discontinued, and the infant was extubated and switched to nasal CPAP on an IF ventilator in Biphasic mode. Red blood cell parameters normalized, with hemoglobin of 151 g/L, and high-sensitivity troponin decreased to 38,700 ng/L.

Echocardiography on day 9 showed: regurgitation grade 1. Left ventricle: not dilated. Atrial septum: 0.33 cm, hypertrophy, end-diastolic dimension 1.2 cm, posterior wall thickness 0.28 cm, end-systolic dimension 0.7 cm, ejection fraction 78%, fractional shortening 43%. Anterior wall thickness: 0.28 cm, mild hypertrophy. Tricuspid valve: thin leaflets. Minimal regurgitation. Estimated systolic pressure in the right ventricle: 20.0 mmHg.

On day 10, abdominal ultrasound showed improvement: parenchymal organs were normal, with mild meteorism.

On day 11, the infant was weaned off mechanical ventilation. Blood pressure and heart rate were within normal limits. On day 12, after stabilization, the infant was transferred to a specialized department for further care and treatment.

The infant received infusion therapy, respiratory support, and antibiotics (ampicillin-sulbactam). Central nervous system stimulation and apnea prevention were provided with caffeine citrate. Neurometabolic therapy included Cytoflavin. Inotropic therapy included levosimendan, dopamine, and dobutamine. Sedation and analgesia were provided with fentanyl and midazolam. Volume expander therapy included 0.9% sodium chloride solution.

Final diagnosis: Feto-fetal transfusion syndrome (anemia-polycythemia syndrome, edematous syndrome, severe congenital anemia (corrected), risk of cardiomyopathy). Respiratory distress syndrome: mild. Other cases of prematurity: Prematurity at 31 weeks. Second twin (monochorionic diamniotic twins). Complication: Respiratory failure grade 1. Associated conditions: Other cardiovascular disorders arising in the perinatal period: Congenital heart defect. Muscular ventricular septal defect 0.12 cm. Atrial septal aneurysm with shunt 0.36 cm. Intraventricular (non-traumatic) hemorrhage grade 1: subependymal cysts in the left thalamo-caudal notch in the cyst formation stage.

Conclusion. Feto-fetal transfusion syndrome (FFTS) is one of the most complex complications of monochorionic multiple pregnancies. A common complication in women with multiple pregnancies and antenatal death of one fetus is placental insufficiency, which inevitably leads to intrauterine growth restriction. In FFTS, significant vascular anomalies in the placenta lead to hemodynamic relationships between the donor and recipient fetuses, with a disproportion in circulating blood volumes.

According to studies by Logutova L.S. and Shilkina P.S., timely diagnosis and fetoscopic laser coagulation of anastomoses are the primary methods for preventing severe outcomes [2]. However, as noted by Mikhailov A.V. and colleagues, despite the presence of vascular anastomoses in all monochorionic twins, clinically significant FFTS does not always develop, which is related to the characteristics of blood circulation between the fetuses. Additional studies show that without treatment, mortality in FFTS can reach 95% [3]. Perinatal outcomes significantly improve with early detection and timely correction, such as fetoscopic laser coagulation of anastomoses, amnioreduction, or selective reduction of one fetus [3].

In this clinical case, FFTS was suspected at 14.5 weeks of pregnancy. The first twin was the recipient and died antenatally. The second twin was the donor. Clinical signs of FFTS appeared from the first day of life, including hypovolemia (elevated blood pressure on admission, tendency toward bradycardia), severe anemia, and cardiovascular abnormalities (tachycardia, hypertension, tachypnea).

Severe complications and fatal outcomes can be avoided with early diagnosis, optimal pregnancy management, and minimally invasive intrafetal correction methods such as laser coagulation of placental anastomoses, which can preserve the pregnancy, the lives of the mother and both fetuses, and reduce perinatal mortality. Management of such patients requires careful monitoring and control of fetal blood flow parameters, as recommended in modern clinical guidelines [1].

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