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A life-saving approach for fetal pleural effusion: A case report

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Abstract

Early recognition and management are crucial for fetal pleural effusion. Therefore, this case report focuses on the etiology and management. A pregnant mother was referred because of suspected fetal congenital anomalies. The fetal required amnioreduction and two intrauterine thoracocentesis, performed before cesarean delivery. After the baby was born, it was found positive on TORCH test and the analysis of pleural fluid showed serous hemorrhagic fluid. In the third day, the baby had fluids reaccumulated requiring chest tube insertion and receiving antibiotics, albumin, vitamin K, Sildenafil and dobutamine. The baby's condition is getting better every day and leading to discharge on day 16th. Early recognition of the etiology and intervention is the key factor for achieving good outcomes in fetal with pleural effusion.

Keywords: Fetal pleural effusion, Intrauterine drainage, Preterm, Neonatal

Introduction

Fetal pleural effusion is rare, occurring in 2.2 to 5.5 per 1,000 births^{1,2}. It may be asymtomatic or cause respiratory distress. The management is varies based on gestational age and severity³. This case report focuses on the etiology and management of fetal pleural effusion with intrauterine drainage.

Case report

A 37-year-old mother, in her third pregnancy, referred from Tuban Hospital because of suspected fetal congenital anomalies, with no history of illness, infections, or family disorders. Ultrasonography (Figure 1.) showed polyhydramnios with fetal bilateral pleural effusion without pericardial effusion or ascites. No other fetal anomalies were observed and fetal lung maturation was performed.

Amnioreduction and intrauterine thoracocentesis were performed before cesarean delivery, evacuating 85 ml of serous hemorrhagic fluid was drained from the left chest and 100 ml from the right.

However, fluid had reaccumulated within 72 hours, requiring a second thoracocentesis, with 115 ml drained from the left and 100 ml from the right. A 34-week, 2567-gram baby boy was born (Apgar scores 1-3-5 at 1, 5, and 10 minutes). He was gasping and had a heart rate of 80 bpm, requiring resuscitation and intubation. The baby was on positive pressure ventilation during transportation to the NICU.

In the NICU, the baby was ventilated on PCV mode, Pcontrol 15, PEEP 5, rate 60 tpm and 80% of FiO₂ with peripheral oxygen saturation (SpO2) of 96 %. Vital signs were stable. The laboratory results were within normal limits, except that albumin was slightly decreased (2.4 g/dL). Congenital TORCH screen was positive for IgG Rubella, Toxoplasma, and CMV. Analysis of pleural fluid showed serous hemorrhagic fluid, sterile culture, and no pathological karyotyping. The babygram showed bilateral pleural effusion, worse on the right (Figure 2.). A chest tube was placed and 80 ml of serous hemorrhagic fluid were drained. Severe tricuspid regurgitation (maximal pressure gradient 51.9 mmHg) and an ejection fraction of 68% were seen on Echocardiography.

Based on the history, clinical manifestation, laboratory, and babygram finding, the first working diagnosis were preterm infant with neonatal asphyxia, bilateral pleural effusion post chest tube insertion and persistent pulmonary hypertension of the newborn (PPHN).

The baby was treated with injection of cefoperazone sulbactam 125mg and amikacin 19mg for the infection, albumin 20%, vitamin K 1mg, and also Sildenafil 1mg and dobutamine ~5mcg/kg/minutes for the treatment of PPHN.

A babygram performed on the third day showed that the left chest had reaccumulated fluid (Figure 2.). The baby developed respiratory distress with decreased of vesicular sound, and underwent second chest tube insertion, drained 85 ml of serous hemorrhagic fluid. The baby's condition improved day by day, and was extubated on day 4th with the use of CPAP, and then weaned on day 7th to nasal cannula, and transferred to the neonatal intermediate ward. Antibiotics injection was continued for 14th days, except for amikacin injection.

The chest tube was removed after 9th day, when oxygen support was no longer needed. On day 11th, echocardiogram showed improved heart function, and the dobutamine was stopped. The baby maintained stable condition, and babygram's showed no pleural fluid reaccumulation, leading to discharge on the 16th day (Figure 2.).

Discussion

This case presents a baby, born at 34 weeks gestation due to polyhydramnios. The mother had no prior illnesses or infections, and her previous pregnancies were healthy. Ultrasound showed bilateral fetal pleural effusion with no other anomalies. the mother was treated initially with amnioreduction and intrauterine thoracocentesis, but a second procedure was required due to fluid reaccumulation.

Antenatal management of fetal pleural effusion depends on the use of ultrasound, amount of effusion, and gestational age at diagnosis⁴. conservative management of primary, small, nonhydropic effusions, as spontaneous regression is reported to be possible⁵. If the effusions increases rapidly or

hydrops develops, prenatal intervention, such as repeated thoracocentesis, thoraco-amniotic shunting, and pleurodesis, needs to be considered as it can improves the survival of the fetus^{3,6}

The gestational age has to take into account when deciding on the treatment: pleuro-amniotic shunts are better before 32 weeks, whereas thoracocentesis is preferred between 32-37 weeks^{7,15}. Isolated pleural effusion that presented before 32 weeks has a poor prognosis and almost 100% if hydrops fetalis associated³.

Differentiating primary and secondary pleural effusions is important, with primary cases being associated with frequently fetal structural malformations. A Thoracentesis would help confirm diagnosis. Ultrasonography and echocardiography must be meticulous. The content of the isolated pleural effusion is mostly chylous, which is serous initially and turns into chylous after milk feeding. Distinguishing features of chylous effusion are milky-white or yellow bloody color, with high triglyceride level and lymphocytosis8. Based on the pleural analysis and laboratory finding, the likely etiology of in this case is chylothorax.

After birth, the baby condition initially was poor and gasp, which required intubation. Respiratory distress is the common clinical presentation of spontaneous pleural effusion in the newborn and occurs in the first day of life in 50% cases and within the first week⁹. In severely ill patients with chylothorax, assisted ventilation may be necessary. positive end-expiratory pressure ventilation may tamponade the injured duct and can help to decrease chyle flow^{10,14}.

In this case, the baby's echocardiography showed PPHN and cardiomyopathy, which were treated by sildenafil and dobutamin to maintain the blood pressures and adequate cardiac output. PPHN is characterised by the failure of the pulmonary vasculature to adapt to the extra-uterine environment resulting in persisting high pulmonary vascular resistance (PVR) thus leading to right-to-left shunting¹¹. PPHN is caused by mal-development, under-development or maladaptation. Maladaptation is as a consequence of lung parenchymal disease, infection or perinatal asphyxia. in this case, which the patient was congenitally infected (in utero), is well known that it can lead to congenital malformation ¹². Administering the potent vasodilator and oxygen, is key to reducing PVR with target oxygen saturations no less than 94%. The baby's second echocardiography revealed the improvement and showed that the PPHN is associated with the baby's lung condition.

The initial treatment for pleural effusion is antibiotics, which target common pneumonia bacteria suitable for the child's age or the underlying infection. The early drainage of pleural effusion being performed to prevents loculation and fibrous peel. The chest tube was thus removed when drained amount was under 30-50 mL/day and symptoms improve. Management of chylothorax aims to relieve respiratory symptoms, prevent recurrence, and address malnutrition or immunodeficiency.

Conclusion

This case reports a rare case of fetal pleural effusion and were associated with chylothorax. Identification of the primary etiology based on pleural fluid analysis is crucial for figuring out the cause and in making management decision. Early intervention is the key factor for achieving good outcomes in baby with fetal pleural effusion.

Disclosure

The authors declared no conflicts of interest. Written consent was obtained from the patient/kin of the patient.

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Appendices



Figure 1. The ultrasonography of the patient demonstrated fetal hydrothorax



The first babygram of the patient (first day of hospitalization)



The babygram evaluation after the chest tube insertion in the left chest (third day of hospitalization)



The babygram evaluation after the chest tube removal (10th day of hospitalization)

Figure 2. The babygram of the patient