Pediatrics and Neonatology (2011) 52, 297–301

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CASE REPORT

Congenital Chylothorax in a Late Preterm Infant and Successful Treatment With Octreotide

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Received Sep 20, 2010; received in revised form Dec 13, 2010; accepted Dec 30, 2010

Key Words
hydrops;
lymph;
pleural effusion;
respiratory distress;
somatostatin analog

Chylothorax is defined as abnormal accumulation of lymphatic fluid in the pleural space and is a rare condition in neonates. Chylothorax causes respiratory and nutritional problems and a significant mortality rate. Octreotide is a long-acting somatostatin analog that can reduce lymphatic fluid production and has been used as a new strategy in the treatment of chylothorax. Here, we report a premature baby with severe bilateral pleural effusion diagnosed by prenatal ultrasound and subsequently confirmed to be congenital chylothorax after birth. This newborn baby was initially treated with bilateral chest tube insertion to relieve severe respiratory distress. However, the chylothorax recurred after a medium-chain-triglyceride-enriched formula was initiated. The accumulation of chylothorax diminished after the administration of octreotide. Therefore, octreotide may allow the patient to avoid invasive procedures, such as reinsertion of chest tubes or surgery.

1. Introduction

Chylothorax is defined as abnormal accumulation of lymphatic fluid in the pleural space and is a relatively rare condition in newborns. In neonates, chylothorax occurs in situations causing damage to the thoracic duct, such as cardiothoracic surgery, birth trauma, and great vessel thrombosis.[1] It also occurs in dysmorphic syndromes, such as Turner or Noonan syndrome. However, in many situations, the etiology of the chylothorax is uncertain and is believed to be caused by abnormality of thoracic or pulmonary lymphatic system. This is termed idiopathic congenital chylothorax.[2] Regardless of the underlying mechanism, chylothorax causes respiratory, nutritional,

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doi:10.1016/j.pedneo.2011.06.011

and immunological complications.[3,4] The mortality rate has been reported to be as high as 50% depending on gestational age, presence of abnormal karyotype, additional congenital anomalies, hydrops fetalis, and the duration and severity of the chylothorax.[3]

Octreotide is a long-acting somatostatin analog that acts on somatostatin receptors in the splanchnic vessels to inhibit lymphatic fluid production. Octreotide has been used in the treatment of postoperative or spontaneous chylothorax in infants and older children.[5,6] It has also been used for the treatment of congenital chylothorax in term neonates.[7,8] However, the experience of octreotide use in premature babies with congenital chylothorax is limited.[6,9–14]

Here, we report a female premature baby identified with prenatal severe bilateral pleural effusion and subsequently diagnosed with congenital chylothorax after delivery. She was initially treated with emergent chest tube insertion because of severe respiratory distress and was successfully treated with octreotide when the chylothorax reaccumulated. Octreotide avoided the baby needing reinsertion of the chest tube or surgery.

2. Case Report

This female neonate was delivered at 35 weeks of gestation to a primigravida mother by means of cesarean delivery for severe bilateral pleural effusion. The birth weight was 1904 g, and the Apgar scores were 1 at 1 minute and 4 at 5 minutes. The parents were nonconsanguineous, and the pregnancy was uneventful until 2 days before the delivery, when bilateral severe pleural effusion was detected by routine prenatal ultrasound (Figure 1). After delivery, the neonate was intubated because of severe respiratory distress and admitted to the neonatal intensive care unit. The patient had generalized edema but no dysmorphic features. A chest X-ray revealed severe bilateral whiteout of the lung field (Figure 2), and bilateral pleural effusion was confirmed by ultrasound.

Bilateral chest tube insertion was performed immediately because of severe respiratory distress and deterioration of O_2 saturation. The drained pleural effusion revealed straw-colored fluid. The cell counts of the pleural effusion revealed the following: red blood cells, 1750 cells/mm^3 and white blood cells (WBCs), 1950 cells/mm^3 with 100% lymphocytes. Biochemical analysis of the pleural effusion revealed the following: glucose, 54 mg/dL; total protein, 3.1 g/dL; lactate dehydrogenase, 97 IU/L; total cholesterol, 17 mg/dL; and triglyceride, 10 mg/dL (before feeding). The cell counts of the peripheral blood revealed the following: WBC, 12,300 cells/mm^3 with 42% neutrophils and 30% lymphocytes (absolute lymphocyte count, 3690 cells/mm^3). The albumin level of the plasma was 2.0 g/dL. Total parenteral nutrition (TPN) was initiated. Follow-up chest X-ray and ultrasound revealed no pleural effusion accumulation. On Day 4, the endotracheal tube was removed, and feeding was initiated. To observe the change of triglyceride level after feeding but avoid worsening of the chylothorax, we started a small amount of feeding with 2 mL of half-strength regular formula every 12 hours. The output of pleural effusion continued to decrease, but the color became turbid. The cell counts of the pleural effusion revealed the following: red blood cells, 4300 cells/mm^3 and WBC, 7029 cells/mm^3 with 66% lymphocytes and 34% neutrophils, and the biochemical analysis revealed the following: total cholesterol, 28 mg/dL and triglyceride, 170 mg/dL. The cell counts of the peripheral blood revealed the following: WBC, 9300 cells/mm^3 with 69% neutrophils and 10% lymphocytes (absolute

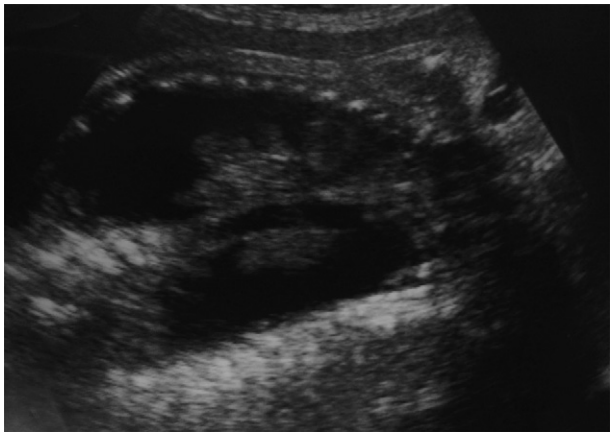

Figure 1 Prenatal ultrasound demonstrating the presence of bilateral large amount of pleural effusion.

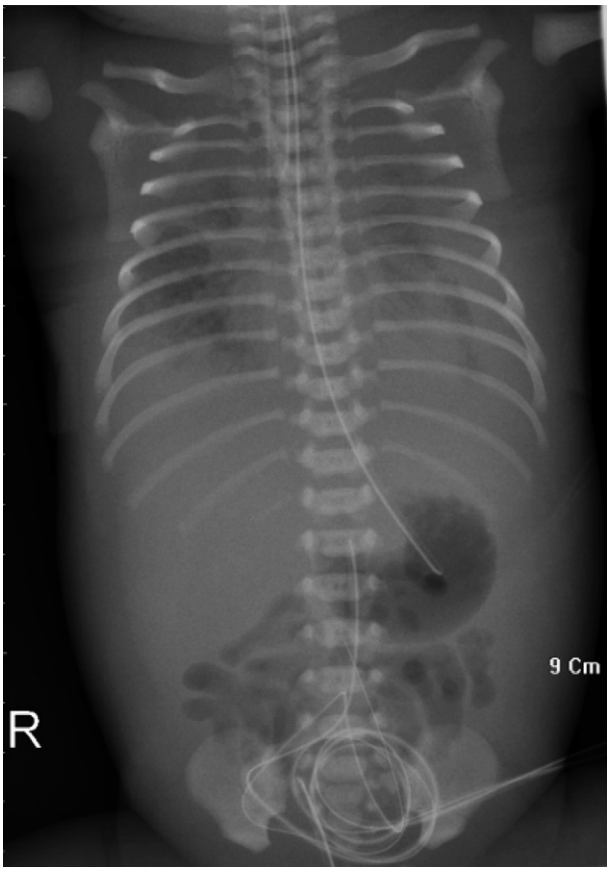

Figure 2 X-ray picture showing the presence of bilateral large amounts of pleural effusion, ascites (centralized bowel loops), and subcutaneous edema.