A neonate with bilateral refractory chylothorax – a case report

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Summary

A thirty six week gestation male baby weighing three kilogram was born to a twenty five year old mother by spontaneous vaginal delivery. At four hours of life, the baby developed respiratory distress with cyanosis and was admitted to the neonatal intensive care unit. There was clinical and radiological evidence of bilateral pleural effusion. Thoracentesis revealed a transudate. Repeated thoracentesis was necessary to relieve the respiratory distress. Subsequently, multi resistant *Klebsiella aerogenes* was isolated from the blood. The baby expired due to gram negative sepsis.

Key words: Chylothorax, Thoracentesis

Introduction

Chylothorax is due to escape of chyle from the thoracic duct into the thoracic cavity. The pleural fluid is serious before the onset of feeding and becomes white or chylus (due to the presence of chylomicrons) after milk feeding is commented[1,2,3] Neonatal chylothorax is typically reported to be unilteral and occurs more commonly on the right side.[1,3] We report here a rare case of bilateral chylothorax.

Case Report

A thirty six weeks gestation baby weighing three kilograms was born to gravida two, para one mother by spontaneous vaginal delivery at home. Mother had history of leaking liquor for forty eigth hours prior to delivery. Baby cried at birth and was apparently doing well. At four hours of life, the baby was admitted for increasing respiratory distress. On examination the baby was cyanosed, dysnoeic, heart rate 140/minute and respiratory rate 60/minute. There was no dysmorphic facies. Diminished breadth sounds were heard on both lower lobes of the lung. Heart sounds were normal. Liver was enlarged measuring 4cms.

On admission, chest xray showed bilateral pleural effusion. Hb 15.3 gm/l, platelet count 139x109/1, P.C.V 48%, Total leucocyte count: 15.9/109/1. Blood group was O positive. Random blood sugar was 15.0 mmol/l. Subsequent random blood sugar on the second and third day was 8.8 and 5.1mmol/l respectively. On the second day of life, thoracentesis was done on the right side during which yellowish fluid was aspirated. The concentration of the protein and sugar in the pleural fluid was 26.7 grams/l and 9.3mmol/l. respectively. Multiresistant type of *Klebsiella aerogenes* was isolated from the blood culture.

Baby was managed conservatively with intravenous infusion 10% Dextrose and intravenous benzyl pencillin 100,000 units/kg and gentamicin 2.5mg/kg/ dose twice daily. Intermittent positive pressure ventilation was instituted.

Arterial blood gas analysis after initial stabilization showed pH 7.503, PCO 2 29.3, PO 2 114.5, HCO 3 22.2, Base Excess 1.2, O2 - Saturation 98.6%. Seventy two hours after admission, repeat thoracentesis on the left side revesled twenty ml yellowish fluid and on the right side of the chest, four ml of blood stained fluid was aspirated. Gram staining of the pleural fluid showed occassional pus cells. *Pseudomonas aeuroginosa* was isolated from the pleural fluid and was sensitive to cefotaxime and gentamicin. Baby was treated with intravenus cefotaxime 100mgm/kg/day and amikacin 15mgm/ kg/day in divided doses. Sequential thoracentesis was necessary on fourth and fifth day fo admission due to rapid reaccumulation of the pleural fluid. At this stage, the baby showed signs of clinical improvement. serial arterial blood gases were within normal limits. Subsequently, the baby was weaned off gradually from the ventilator. However, on sixth day of life, baby was not active and found to be scleremic over the extremities. Within twenty four hours after the onset of sclerema, the baby expired due to gram negative sepsis of multiresistant *Klebsiella aerogenesa* associated with severe hypoxia and acidosis.

Discussion

Chylothorax in the newborn period was first reported by Pisek in 1917.[4] Spontaneous chylothorx may occur due to anomalous lymphatic channels or failure of normality developed lumphatic channels to drain into the thoracic duct.[1] Infrequently, spontaneous chylothorax may also be associated with either foetal hydrops or hydraminos in the mother or chromosomal disorders like Downs, Noonan and Turner syndrome.[4] Chylothorx may also occur due to acquired causes like birth trauma or chest injury involving the thoracic duct[5] or thrombosis of superior vena cavae secondary to central venous catheter or post cardiac surgery or following repair for diaphragmatic hernia.[6]

The mode of therapy for chlothorax is usually conservative involving repeated thoracentesis. The nutrition of the baby is maintained by adding medium chain triglycerides to the diet in addition to the daily requirements.[2,7,8] Medium chain triglycerides has the advantage of bypassing the chyle for its absorption[2,7] In refractory cases, thoracentesis can be repeated for several months. In the majority of cases, chylothorax is selflimiting in nature due to development of new lymphatic channels[2,3]

The current mode of management of chylothorax is primarily supportive which involves provision of adequate nutritional support. The indications for surgical insertion of Denver Double valved shunt system under general anaesthesia are recurrent pleural effusions, inability to maintain the nutritional requirements of the baby, and difficulty in securing the thoracostomy tube.[9] Early pleuroperitoneal shunting is preferred because it is simple, safe, easy to perform and to prevent nutritional depletion.

Babies with refractory chylothorax are prone to develop infection due to depletion of lymphocytes from the chyle[6] Longaker et al reported in their series of thirty two cases of primary foetal hydrothorax that the following features were associated with poor prognosis: hydrops, gestational age less than thirty five weeks and bilateral pleural effusions.[10]

In this neonate, respiratory distress was appropriately managed with mechanical ventilation and repeated thoracentesis. However, baby acquired nosocomial infection most probably from the neonatal nursery. Subsequently, baby expired because of overwhelming gram negative sepsis due to multiresistant *Klebsiella aerogenesa.*

Acknowledgement

We would like to thank the Dean, (Prof) Roslani Abdul Majid, Universiti Sains Malaysia for encouragement and also permitting us to publish this paper.

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