Povidone Pleurodesis In Bilateral Chylothorax Post Congenital Diaphragmatic Hernia Repair

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Abstract

Chylothorax is a known complication of postoperative congenital diaphragmatic hernia repair in neonates. We herein report a successful povidone pleurodesis for bilateral chylothorax post congenital diaphragmatic hernia repair for a 3.24kg term male baby. He was noted tachypniec and was subsequently intubated soon after birth. Diagnosis of left congenital diaphragmatic hernia was confirmed by chest x ray and corrective surgery was done after a period of stabilisation. Post operatively he made a poor recovery and serial chest images show features of effusion. Drains were inserted and fluids drained were confirmed to be chyle. Initially child was treated conservatively. However, due to persistent high output showing signs of refractory to conservative management we decided to use povidone as pleurodesis. During the course of povidone iodine instillation into both chest tubes, the amount of drain was reducing and subsequently child recovered well.

Keywords

- Congenital Diaphragmatic Hernia
- povidone pleurodesis
- chylothorax

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Introduction

Congenital diaphragmatic hernia (CDH) is a congenital anomaly in which abdominal content herniated through an orifice in the diaphragm due to failure of fusion of diaphragmatic components, particularly pleuroperitoneal membrane during first trimester of pregnancy. The most common type is posterolateral CDH, also known as Bochdalek's hernia, which comprises 80% of CDH cases. CDH occurs in 1 in 2500 newborns and is commonly associated with pulmonary hypoplasia and persistent pulmonary hypertension, which explains its high mortality rate ranging from 10 - 40%, depending on the size of defect and managing centers.[1-3]

Historically, CDH was repaired immediately. With better understanding of the pathophysiology and natural history of the disease, surgeons shifted towards delayed repair in favor of preoperative hemodynamic and cardiorespiratory optimization.[1-2]

Pleural effusion might develop post CDH repair especially in those with bigger defect with the need to utilize mesh, but overally less than 5% of patients develop chylothorax post CDH repair.[3]

The majority of chylothoraces are successfully treated conservatively. Surgical intervention is usually reserved for cases who do not respond to medical treatment.[1-5] We therefore discussed a case of neonate who developed bilateral chylothorax post CDH repair and underwent povidone iodine (PI) pleurodesis.

Case presentation

A case of full-term baby boy with birth weight of 3.42 kg was referred to our center for left congenital diaphragmatic hernia (CDH). Antenatally mother had iron deficiency anemia and antenatal scan was normal. Baby born via normal vaginal delivery and was intubated at birth for grunting with poor breathing effort. There was no dysmorphism, no murmur on auscultation, air entry into the left lung was reduced, and abdomen was scaphoid. Patient remained well, being able to maintain saturation on conventional ventilator until the operation on the day 4 of life.

Intraoperatively, there was left posterolateral defect, the content was small bowel and spleen, there was no hernial sac and left lung was hypoplastic. Left CDH

was repaired primarily with prolene 3-0 and it was a straight forward repair.

Patient remained well on conventional ventilator and hemodynamically was unsupported. He was started on total parenteral nutrition (TPN) and feeding was introduced after day 3 post-operative which later managed to step slowly till achieved a third of patient's requirement. However, on day 10 postoperative, patient had frequent desaturations and chest radiograph revealed bilateral pleural effusion. Hence, right chest tube was inserted. There was a transudative fluid and pleural fluid triglyceride was 0.7mmol/L which later turned milky on day 18 postoperative after feeding was restarted. Repeated pleural fluid triglyceride was 1.8mmol/L which was suggestive for chylothorax. Patient was kept NPO since then and full TPN administered. However, despite that, chest tube output was ranging 200 - 400 ml per day which was replaced with Hartmann solution.

On day 30 postoperative, left chest tube was inserted as patient had desaturations and chest radiograph showed worsening left pleural effusion with mediastinal shift to the right. By this time, each chest tube output was ranging around 600 - 800ml per day, which were replaced with Hartmann solution and 5% albumin. Initially, chylothorax was treated with dietary modification only; but after second chest tube insertion, octreotide infusion was added.

After a week on octreotide infusion, on top of nil by mouth, both chest tube outputs remained high. Patient developed oliguria and worsening metabolic acidosis. A Computed Tomography Scan of thorax was performed which ruled out thoracic lymphatic malformation and pulmonary lymphangiectasia.

Since there was no sign of improvement, we decided to try out PI pleurodesis. 10 ml of 10% povidone iodine was diluted into 15 ml, then instilled into right chest tube and clamped for 5 hours before releasing. The same procedure was repeated for left chest tube on the next day. Throughout the period post instillation, there was no episode of desaturation and chest tube output was decreasing even after we had restarted feeding. The left chest tube was removed after 9 days of povidone instillation (post op day 48) and 10 days for right chest tube (post op day 49).

Patient eventually remained stable, was extubated 3 weeks after chest tube removal and later discharge well home on day 80 of

life. At present, patient almost reached 2 years old, he is thriving well and has no readmission since he was discharged from neonatal care

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Figure 1: intra-operative image; posterolateral defect, repaired with Prolene 3-0

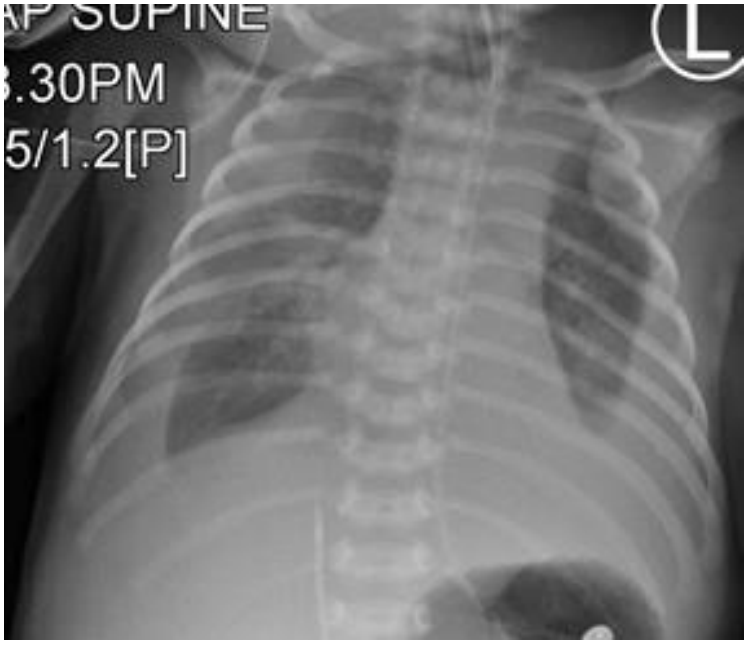

Figure 2: Bilateral pleural effusion developed after post op day 10

Figure 3: Right chest tube drained milky fluid

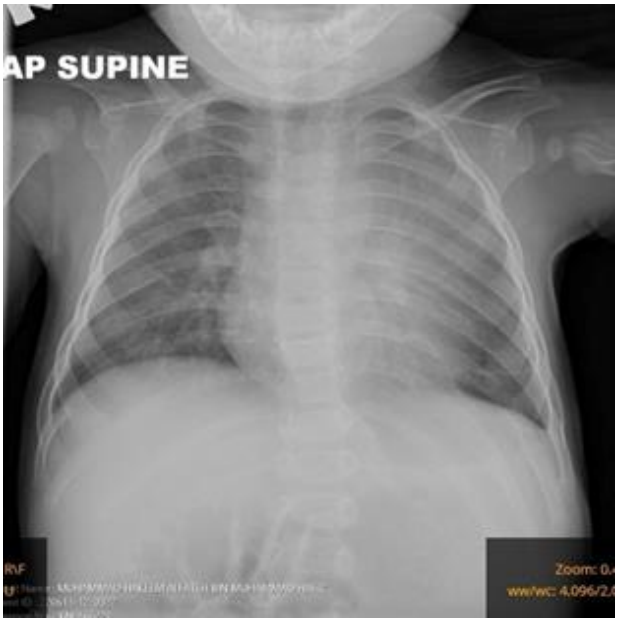

Figure 4: chest radiograph during follow-up at 8 months old. Both lungs well expanded. Some opacity over left middle zone is seen, which may be caused by pleural thickening post pleurodesis.

Discussion

Chylothorax is the accumulation of chyle in the pleural space that occurs as a result of damage to the thoracic duct by rupture, laceration, tear or compression. It can be life threatening as it may lead to hypovolaemia due to rapid fluid loss and respiratory distress as the fluids fill up the pleural space.[1-5] Patient can also develop severe malnutrition as protein, fat and lipid soluble vitamins are lost. Levy et al reported the incident of chylothorax post CDH repair is 4% and it is significantly related to patch repair and patient on extracorporeal membrane oxygenation (ECMO).[3]

Chylothorax is clinically suspected if milky color fluid noticed from chest tube as feeding was started. Biochemically, it is diagnosed by elevated triglyceride levels in the pleural fluid above the established cut-off limit of 1.24 mmol/L (110 mg/dL) and elevated lymphocytes >1000 cells/μL on the fluid microscopy.[1,4-5]

It is well documented that chylothorax can be successfully treated conservatively.[1,3-5] Ruangnapa et al reported 65 patients aged

0 - 15 years with chylothorax over 15 years, 89.3 of them are successfully treated medically. There were 10.7% required surgical correction which involved ligation of thoracic duct with or without pleurodesis.[4] Medical treatment mainly focus on maneuvers to reduce chyle production such as keeping the child nil by mouth, diet modification with either fat free, low fat or medium chain triglyceride (MCT) diet.

On top of that, medication such as octreotide, a somatostatin analogue, acts on vascular somatostatin receptor, may reduce lymphatic excretion via increasing splanchnic arteriolar resistance, resulting in decrease in gastrointestinal blood flow. Cessation of chyle leak post octreotide infusion is up to 62 to 72% with median use of 9 - 10 days.[3-4] However, it may cause GI upset, bradycardia and hyper/ hypoglycemia during the course of infusion which we need to monitor.

Ventilator strategy as such pressure-controlled ventilation with positive end-expiratory pressure or high frequency oscillatory ventilation (HFOV) also play some role in reducing chyle leakage.[4]

Unfortunately, our patients did not recover from chylothorax despite was keep nil by mouth with TPN for almost a month and on octreotide infusion. Hence, we decided to try PI pleurodesis. It was first reported by Brissaud et al in year 2003, they instilled PI via chest tube in 4 congenital chylothorax patients which chylothorax resolved in 3 patients in 6, 10, and 16 days. Pre- and post-instillation thyroxine levels were unchanged. The other one baby died of renal failure and post mortem assessment revealed pulmonary lymphangiectasia.[6]

As for our patient, 10 ml PI 10% was diluted to 15 ml and instilled into right chest tube, then clamped for 5 hours before release. The same procedure was repeated for left side on the next day. Throughout post-instillation period, patient remained stable on moderate setting ventilation, there was no spikes of temperature and both chest tube outputs were gradually reducing until it was removed on day 9 post instillation for left side and day 10 for the right side. During period post-instillation period, feeding was restarted and there was no increase in chest tube output.

There were other studies which used PI as pleurodesis with total of 13 chylothorax patients, but only our patient developed chylothorax post CDH repair. Out of 13 patients, 3 patients died post-instillation which related to presence of pulmonary lymphangiectasia. The concentration of PI

used for instillation, amount of PI instilled, and duration of clamping varied among the studies.[6-10]

PI is a bactericidal antiseptic solution which is commonly used for irrigation of open wound and preoperative skin preparation. Its exact mechanism in pleurodesis is unclear, but its pro-inflammatory properties is believed to enhance sclerosis, thus explaining its pleurodesis mechanism. PI is a low cost, readily available preparation, and has a biochemical marker to monitor toxicity.[6,10-11] It is in liquid form hence it is easier to administer into chest tube as compared to talc which is in powder form. Other agents such as OK-432 are not available in the market anymore.

The use of PI as chemical pleurodesis should be reserved as last option after exhausted initial treatment of diet modification and octreotide infusion. Pulmonary lymphangiectasia should be ruled out before embark on PI use, as it may lead to massive absorption of PI, causing PI toxicity.[6-8] A further study should be done to develop a standard protocol in installation of PI as chemical pleurodesis to ensure good and consistent outcome of chylothorax resolution and patient safety.

Conclusion

We reported a case of a successful chylothorax management post CDH repair using PI as pleurodesis agent. Further study is needed in order to come out with a standard protocol for management of chylothorax after failed medical management.

Ethical Consideration

Written consent for participation was obtained from the parent or guardian of the participant in the study**.** This study was approved by Wanita dan Kanak Kanak Sabah Hospital.

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Conflict of interests

There is no conflict of interest

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