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CHYLOTHORAX: A RARE COMPLICATION OF TUBE THORACOSTOMY

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□ **Abstract—Background: Chylothorax resulting from chest tube injury to the thoracic duct is very rare and underreported. Objective: The purpose of this case report is to exemplify this rare but potentially significant complication of chest tube thoracostomy. Case Report: An 86-year-old woman presented with sepsis and a massive right pleural effusion; she developed a chylous effusion with the pleural fluid triglyceride level of 158 mg/dL 2 days after a traumatic chest tube insertion. All investigations excluded common causes of non-traumatic chylothorax. The chylothorax improved after fasting and implementation of a medium-chain triglyceride diet. Conclusion: The optimal depth of insertion of the chest tube typically ranges from 5 to 15 cm, ensuring all sideports are within the chest and the proximal port is at least 2 cm beyond the rib margin. Traumatic chylothorax secondary to chest tube insertion should be included in the differential diagnosis of patients presenting with chylothorax after a thoracostomy tube.** © 2008 Elsevier Inc.

□ **Keywords—chylothorax; chylous effusion; chest tube; thoracostomy; thoracic duct**

INTRODUCTION

Chylothorax is the presence of lymphatic fluid in the pleural space resulting from disruption or obstruction of the thoracic duct. The thoracic duct is a continuation of the cisterna chyli, which lies just anterior to the first or second lumbar vertebra and passes through the aortic hiatus of the diaphragm to enter the posterior mediastinum. In the thorax, it continues cephalad in a rightward position where it lies to the right of the aorta, inclining to the left at approximately the level of the fifth thoracic vertebra, where it crosses over the vertebral column behind the esophagus and continues in the left posterior mediastinum. Entering the root of the neck, it turns laterally behind the carotid sheath and the vertebral artery, then downwards in front of the subclavian artery, entering the venous system at the junction of the left internal jugular and subclavian veins (1). The anatomy of the thoracic duct determines the location of effusion in case of disruption or obstruction of the duct. Because the thoracic duct crosses the mediastinum at the level of the fifth thoracic vertebra, lymphatic injury or obstruction below this level results in a right-sided pleural effusion. In contrast, lymphatic injury or obstruction above this level usually leads to a left-sided effusion (2).

Disruption or blockage of the thoracic duct represents the most common mechanism for the creation of chylothoraces. Chylous pleural effusions often appear turbid or milky due to high lipid content. A pleural fluid triglyceride level $>$ 110 mg/dL is highly suggestive of a chylothorax (3). The etiology of a chylothorax can be categorized as traumatic and non-traumatic. Among traumatic causes, chylothorax occurs after coronary artery bypass surgery with an incidence of 0.5% (4). Chylothorax secondary to chest tube insertion has rarely been reported. We are aware of only two reports describing

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this entity in the pediatric literature (5,6). We present a case of an elderly woman who developed a chylothorax after chest tube insertion.

CASE REPORT

An 86-year-old woman with Alzheimer's disease presented with dyspnea, hypotension, and altered mental status. Her vital signs were notable for systolic blood pressure of 66 mm Hg, fever to 40°C, and oxygen saturation of 96% on 100% non-rebreather mask. On physical examination, she appeared lethargic with labored breathing. The trachea was shifted to the left and there were decreased breath sounds and increased dullness on percussion of the right chest. Chest radiography showed large right-sided pleural effusion with shifting of the mediastinum to the left. Laboratory analysis was notable for a white blood cell (WBC) count of 13,700 cells/mm^3 with 76% neutrophils. Additional notable findings included evidence of acute renal failure, shock liver, and disseminated intravascular coagulation. Treatment with fluid resuscitation, antibiotics, and vasopressor support were initiated. In the Emergency Department, a 24F chest tube was inserted into the right pleural space via a guide wire by the Seldinger technique, with some initial resistance noted upon entry. Upon arrival into the intensive care unit (ICU), the chest tube was repositioned because it appeared too far advanced on initial chest radiography (Figure 1). The initial pleural fluid was serosanguinous, with the following cell count and differential: RBC-31,190/mm^3, WBC-171/mm^3 with 69% neutrophils and 31% mononuclear cells. The pleural fluid-to-serum ratios of total protein and lactate dehydrogenase (LDH) were 1.5 g/dL to 6 g/dL and 165 U/L to 744 U/L, respectively. The elevated serum LDH was predominantly LDH-5 isoenzyme. Despite continuously high chest tube output, there seemed to be marked radiographic improvement (Figure 2). On the third ICU day, upon initiation of enteral nutrition, the pleural fluid became milky (Figure 3) and the pleural fluid triglyceride level was noted to be 158 mg/dL. The patient was fasted overnight and, subsequently, the tube feeds were substituted with a medium chain triglyceride formula. As expected, the fluid characteristic changed from its milky appearance and became serous in nature. The pleural fluid cultures remained negative and the cytology

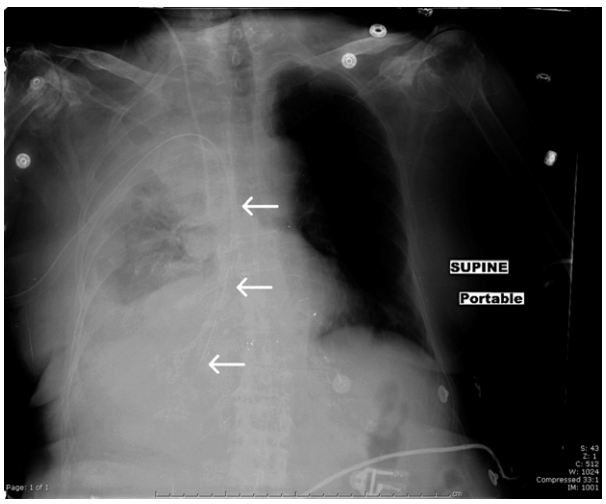

Figure 1. The chest X-ray study before chest tube reposition: the arrows outline the course of the chest tube along the right lower paravertebral region with the tip ending at the posterior costophrenic sulcus below the dome of diaphragm.

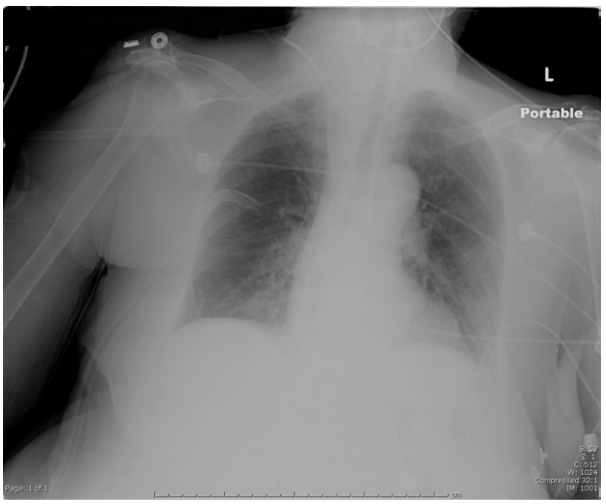

Figure 2. The chest X-ray study after the first day in the intensive care unit after chest tube reposition shows almost complete resolution of the pleural effusion and full expansion of the right lung.

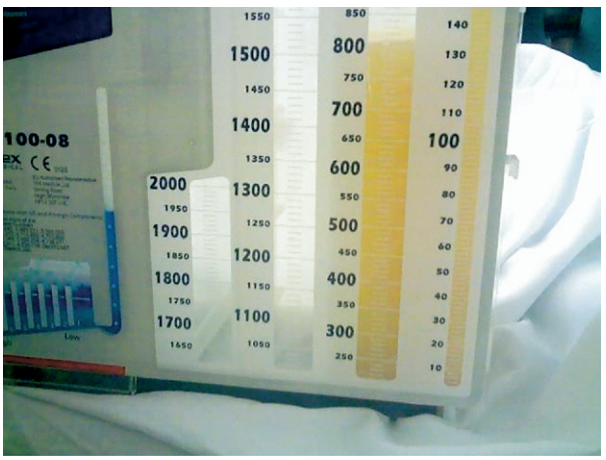

Figure 3. The milky appearance of the pleural fluid with the top white, creamy layer in the columns of the container.

showed abundant mesothelial cells with a cluster of atypical cells. Chest computed tomography (CT) scan showed no evidence of primary malignancy, metastatic disease, or lymphadenopathy in the lung or mediastinum. There was also no evidence of underlying parenchymal infiltration, cystic lung disease, or pleural thickening. The liver appeared lobulated with multiple hypodense lesions suspicious for metastatic disease, and a slightly enlarged spleen with ascites and findings suggestive of portal hypertension were noted. Due to persistent high pleural fluid output and the family's decision not to pursue aggressive interventions, closed pleurodesis was performed.

DISCUSSION

Our patient presented with severe sepsis and was found to have a massive right-sided pleural effusion. A chest tube was inserted emergently because the patient was deemed unstable. The first pleural fluid profile was consistent with an exudate based on Light's criteria (LDH > 2/3 of upper normal limit of serum) (7). However, there was no suggestion of empyema or a parapneumonic effusion, and repeat chemistries were consistent with a transudative effusion (Table 1). The initial serosanguinous appearance actually raised the concern of malignancy, but more likely was related to a traumatic chest tube insertion in a coagulopathic patient. The transudative component of the effusion was likely secondary to portal hypertension due to liver metastasis. Although the milky appearance is found in only 50% of the chylothoraces and its absence does not exclude the diagnosis, it was likely not the original etiology of our patient's pleural effusion, especially because it was transudative in nature (3). There were no findings on chest CT scan consistent with lymphoma, or other common non-traumatic etiologies for chylothorax such as other malignancies or lymphangioleiomyomatosis (7,8). We believe a traumatic chest tube insertion had injured or disrupted the lower part of the thoracic duct and the mediastinal pleura, resulting in a chylothorax. To minimize the risk of this complication, the depth of insertion should be determined before chest tube placement. The correct depth of insertion of the chest tube ranges from 5 to 15 cm, ensuring all sideports are within the chest and the proximal port is at least 2 cm beyond the rib margin (9).

Table 1. Pleural Effusion Profiles in Each Hospital Day

	Hospital Day			
Pleural Effusion	1	3	4	6
Appearance	SS	MK	S	S
RBC (cells/mm^3)	31,190	n/o	61	n/o
WBC (cells/mm^3)	171	n/o	82	n/o
-Neutrophils (%)	69	n/o	89	n/o
-Lymphocytes (%)	22	n/o	9	n/o
-Monocytes (%)	9	n/o	2	n/o
LDH (IU/L)	165	73	61	n/o
Total protein (g/dL)	1.5	n/o	<0.5	n/o
Glucose (g/dL)	n/o	n/o	189	n/o
Amylase (IU/L)	n/o	22	n/o	n/o
Triglyceride (mg/dL)	n/o	158	n/o	28
Cultures	NG	n/o	NG	n/o

MK = milky; NG = no growth; n/o = not obtained; RBC = red blood cells; SS = serosanguinous; S = serous; WBC = white blood cells; LDH = lactate dehydrogenase.

CONCLUSION

Traumatic chylothorax secondary to chest tube insertion should be included in the differential diagnosis of patients presenting with chylothorax after chest tube thoracostomy.

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