CLINICAL VIGNETTE

Dyspnea and Pleural Effusion after a Motor Vehicle Accident: A Case of Traumatic Chylothorax

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Chylothorax occurs when chyle accumulates in the pleural space usually as a result of disruption of the thoracic lymphatics. Chyle, a milky white fluid, is formed when long-chained triglycerides in the diet are transformed into chylomicrons and secreted into the intestinal lacteals. Eventually, these lymphatic channels meet to form the thoracic duct that courses through the thoracic cavity and drains into the left subclavian vein. Any injury to the thoracic duct along its path can lead to a chylous effusion. Trauma, including surgical trauma, and lymphoma are the main causes of chylothorax, but numerous other etiologies exist. Diagnosis depends on direct analysis of the pleural fluid for triglyceride content and chylomicrons. Management is individualized and includes conservative options such as dietary modification, total parenteral nutrition, and octreotide or surgical management to attempt to repair or ligate the thoracic duct. Complications can include malnutrition and immune dysfunction due to loss of nutrients and immune cells in the chyle. We present a case of traumatic chylothorax that occurred after a motor vehicle accident that was successfully managed with conservative measures including dietary modification and octreotide treatment.

Case Report

A 73-year-old male with a history of hypertension was involved in a single car rollover motor vehicle accident. The patient was taken emergently to an outside hospital and found to have a twelfth thoracic (T12) vertebral body burst fracture with a retro-pulsed fragment and cord compression with paraplegia, several rib fractures, and a right sided hemothorax. A chest tube was placed and subsequently removed at the outside hospital. Given the patient's complex spine fracture, he was transferred to the Neurosurgery Spine service at our hospital four days after the accident to undergo spinal surgery. Chest radiography (CXR) performed on admission at our hospital showed "clear lungs" with "no pleural effusions" (**Figure 1A**). Nine days following the accident, the patient developed tachycardia and hypoxia and repeat CXR revealed "interval development of a large right sided pleural effusion with underlying atelectasis with complete whiteout of the right hemi thorax" (**Figure 1B**). Chest computed tomography (CT) scan showed a "large right and small left pleural effusion without loculation with significant atelecatasis in the adjacent right lung, thought to be due to the large effusion" (**Figure 2**). Pulmonary consultation was obtained and an ultrasound-guided thoracentesis was performed. The pleural fluid that was removed was a milky brown color. Cell count showed 204,000 RBC/cmm, 340 WBC/cmm with 70% lymphocytes. The glucose level was 137 mg/dL and lactate dehydrogenase was 252 U/L. "Gross lipemia" was seen and subsequent testing detected chylomicrons and showed the triglyceride level to be 1769 mg/dL. Pleural fluid bacterial cultures were negative. The patient was diagnosed with traumatic chylothorax related to thoracic duct injury associated with his T12 vertebral body burst fracture injury. A chest tube was placed, the patient was made 'nothing by mouth', total parenteral nutrition (TPN) was started and octreotide therapy was initiated at 100 µg subcutaneously every 8 hours. The patient subsequently underwent spinal stabilization surgery. The patient's hospital course was complicated by prolonged pleural drainage, hospital acquired pneumonia, and central line related bloodstream infection. After 17 days, pleural fluid drainage stopped and the chest tube was removed and the patient was transferred to a neurological rehabilitation facility.

Discussion

Each year in the United States, approximately 1.5 million people develop a pleural effusion. There are numerous causes of pleural effusions with congestive heart failure, parapneumonic effusion, malignant pleural effusion, pulmonary embolism, and viral disease being the most common[1]. Chylothorax, is a rare cause of pleural effusion and is defined as the accumulation of chyle in the pleural space usually due to disruption to the thoracic duct within the

thoracic cavity. Chyle is described as having a white, milky appearance, but this classic appearance is seen in only half the patient of patients with chylothorax making chemical analysis essential for the correct diagnosis[2]. Key to the diagnosis of chylothorax is suspecting its presence and ordering the correct tests from the pleural fluid. Management requires an understanding of lipid metabolism and the anatomy of the thoracic duct[3].

Humans normally consume 50 to 100 grams of fat per day. Dietary fat consists mainly of triglycerides. Digestive enzymes in the stomach and small intestine hydrolyze triglycerides. The hydrolyzed products are absorbed across the intestinal mucosal cells and then resynthesized into triglycerides. Medium-chain triglycerides are transported directly into the blood. Long-chain triglycerides, which constitute 95% of triglycerides in the typical daily diet, are water-insoluble and require special 'packaging' in the form of lipoproteins to be transported in the blood[2]. Most triglycerides are transported to the blood by lipoproteins called chylomicrons and very-low-density lipoproteins by way of the lymphatic system. They are initially secreted into the intestinal lacteals, which eventually coalesce and lead to the cysterna chili and the thoracic duct. Chyle also contains fat-soluble vitamins and lymphocytes, primarily T cells[3]. Thus, loss of chyle in a chylothorax can rapidly lead to both malnutrition and immunologic compromise. In fact, inanition and infection are thought to be the main causes of mortality in persisting chylothorax. Fatty acids with fewer than 10 carbon atoms in the chain are absorbed directly by the portal venous system. This fact forms the basis for the use of medium-chain triglycerides as an oral diet in the conservative management of chylothorax[4].

The primary function of the thoracic duct is to transport digestive fat to the venous system. While considerable anatomic variation exists, the thoracic duct typically ascends on the anterior surface of the vertebral bodies between the aorta and azygos vein. The duct crosses from right to left near the fifth thoracic (T5) vertebrae and ascends behind the aortic arch and ultimately empties into the junction of the left jugular and subclavian veins. Although there is significant anatomic variability, the typical route of the thoracic duct explains why injury to the duct below T5 often produces a right-sided chylothorax, while injury above T5 results in a left-sided effusion[4,5]. Chyle flow ranges widely, from 15 mL/hour during fasting up to 100 mL/hour after a meal. The content of the meal is also important with a higher chyle flow rate in proportion to higher fat content[3]. Dietary manipulation in an attempt to reduce chyle flow is an important part of the conservative management of chylothorax[4].

Clinically, chylothorax usually presents with worsening dyspnea as the lung is compressed by accumulating chyle in the pleural space. Pleuritic chest pain and pleural thickening are uncommon as chyle is non-irritating to the pleural surface. Thoracentesis with direct analysis of the pleural fluid is the most important diagnostic step and key for definitive diagnosis[4]. Appearance of the pleural fluid is typically milky but may be misleading and can be confused with empyema. Chylothorax may not have the classic white milky appearance in over half of the cases, especially if the patient is fasting or malnourished[5]. Thus, regardless of the pleural fluid appearance, chemical analysis, including triglyceride and chylomicron levels, should be ordered if the history suggests possible chylothorax or the diagnosis is unclear. Chylothorax is typically an exudate using Light's criteria with a lymphocytic predominance, but cases of transudate with neutrophil predominance have been described[1,5]. Pleural fluid LDH level is elevated, but typically no to the levels seen in infection or malignancy. Pleural and serum triglyceride and cholesterol measurements are the initial screening tests for chylothorax. A pleural fluid triglyceride level greater than 110 mg/dL, with a ratio of serum to pleural triglyceride level <1 and a serum to pleural fluid cholesterol level > 1 is consistent with chylothorax. A very low pleural fluid triglyceride level of less than 50 mg/dL makes chylothorax unlikely.[2] Fasting patients may have lower than expected levels of triglyceride in the pleural fluid.

There is also a less common disease entity called "pseudochylothorax" or "chyliform pleural effusion" which can develop in chronic pleural effusions (such as those seen after tuberculosis infection) and mimic chylothorax in appearance and chemistry. These chronic effusions have elevated cholesterol and triglyceride levels related to lipid complexes forming from chronic inflammation. In equivocal and uncertain cases of chylothorax, chylomicron analysis is indicated. The presence of chylomicrons in the pleural effusion verifies the diagnosis of chylothorax[2,4,5].

After the diagnosis of chylothorax is made by pleural fluid analysis, lymphangiography may be useful in helping to locate the leak in the thoracic duct if surgery is being considered. However, it should be noted that while some small studies have supported the use of this imaging technique in locating the area of leak, there are no large prospective studies demonstrating the benefits of lymphangiography in

managing patients with chylothorax. In addition to its limited availability, the sensitivity for detecting thoracic duct leaks is questionable. Thus, the exact role of lymphangiography remains unclear[3].

Determining the underlying cause of chylothorax is essential. The major causes of chylothorax can be divided into four major categories: tumors, trauma, idiopathic, and miscellaneous (**Table 1**)[3,5]. Clinical history may provide clues such as recent thoracic surgery, blunt trauma, or known lymphoma. Often, CT scan of the chest is done as part of the workup and this may reveal an occult tumor or other cause. Trauma (surgical, penetrating, or blunt traumatic injuries) is the most common cause of chylothorax. The thoracic duct lies close to the spine, esophagus, and aorta and postoperative chylothorax has been described following almost every known cardiothoracic surgical procedure as well as neck surgery[3]. Postoperative chylothorax is a well known complication of surgeries involving the posterior mediastinum, such as esophagectomy. Of note, postoperative or trauma related chylothorax can be delayed up to one or two weeks after the initial injury[4,6]. It is thought that it takes some time for chyloma to build up an ultimately rupture into the pleural cavity and become clinically apparent. Malignancy, especially lymphoma, is the second most frequent etiology accounting for approximately 30% of chylothoraces[5]. If no other cause of chylothorax is readily apparent, many advocated searching for underlying lymphoma. Chylothorax is the most common cause of congenital pleural effusion and most cases are idiopathic[6]. Numerous miscellaneous causes of chylothorax have been described[3].

The optimal management of chylothorax is controversial and remains challenging. The main goals of treatment are to alleviate dyspnea by draining the pleural effusion, to maintain nutrition and to reduce chyle flow[3]. If chyle flow is reduced, there is a reasonable chance that spontaneous closure of the thoracic duct leak will occur[7]. Some clinicians advocate a trial of conservative therapy for several weeks while others adopt an early surgical approach[4,8]. There is no doubt that the persistent loss of chyle containing essential proteins, immunoglobulins, fats, vitamins, electrolytes, and water can lead to severe malnutrition, electrolyte disturbances, and immunological compromise over time. Mortality is high if chyle loss persists beyond several weeks[6,9]. The underlying cause, the quantity and duration of the chylothorax, and the patient's nutritional status are all significant factors to consider. Many advocate conservative management for a maximum of 2-3 weeks and then surgical treatment if this fails[9].

Treatment may be categorized into nonoperative (conservative) and operative measures. Therapeutic thoracentesis and chest tube placement for drainage is typically the initial step in all cases. Patients are usually advised nothing by mouth or a diet rich in low-fat, medium chain triglycerides in an effort to reduce chyle flow. Recall that medium-chain triglycerides are absorbed directly into the portal circulation. Nutritional support with TPN is typically administered[4]. Due to immunologic compromise that develops with loss of chyle; high vigilance for infectious complications is warranted.

Several case reports and series have shown that octreotide injections are safe and likely effective in reducing chyle production and chyle leak and are an important adjuvant treatment in the conservative management of chylothorax[4,7]. It is unclear how long the clinician should wait when the patient in on dietary fat restriction before administering octreotide. Typical doses given are 50-100 μg every 8 hours. The exact mechanism by which octreotide works is unclear but it is thought to inhibit absorption from the intestine and decrease splanchnic blood flow. In successful cases, lymph drainage usually decreases significantly in the first few days after octreotide is initiated and the drug is typically administered for 1-2 weeks[7]. Treatment failure has been described and when no substantial reduction of lymph flow is evident after the first 7-10 days of treatment, alternative therapeutic measures should be considered[7,10].

If medical management fails, surgery should be undertaken. Of note, radiotherapy is often effective in controlling chylothorax associated with malignancy and is typically the treatment of choice. Surgical intervention for chylothorax is often effective but the mortality rate may be around 10% postoperatively. It is also worth mentioning that percutaneous embolization of the thoracic duct is available at some specialized institutions[7, 11]. Aggressive surgical therapy is recommended for post-surgical chylothorax in many cases. However, the clinical parameters that prompt surgical intervention vary between centers. In general, if the daily chyle leak persists at > 1.5 L/day, surgery is often undertaken[4]. Lymphangiography can help to delineate the lymphatic anatomy, though this test can be difficult to perform. Enteral administration of a high fat source like cream can be given with the addition of methylene blue to highlight the source of the chyle leak. If the chyle leak can be identified, direct

ligation should be performed. Mass ligation of all tissues between the aorta, spine, esophagus, and pericardium above the diaphragmatic hiatus is performed if the leak is unable to be identified[4, 8].

In conclusion, traumatic chylothorax related to blunt trauma and vertebral fracture is rare. Patients typically present with progressive dyspnea but the diagnosis is often delayed several days after the injury. Thoracentesis with direct analysis of pleural fluid for triglyceride content and chylomicrons is required for definitive diagnosis. Management is controversial and challenging and typically involves an extended hospital stay for patients. Severe complications such as malnutrition and immune compromise due to chyle loss are frequently encountered. Individualized treatment plans that include chyle drainage via chest tube and other conservative measures such as dietary modification, total parenteral nutrition, and octreotide are usually attempted for several weeks in hopes that chyle flow will abate and the thoracic duct injury will heal. Surgical management to attempt to repair or ligate the thoracic duct can be done in those who fail a trial of conservative therapy. The patient described above had a long hospital course complicated by several hospital-acquired infections. The chylothorax eventually resolved with conservative management including nothing by mouth, TPN, and octreotide and the patient was subsequently discharged to a rehabilitation facility.

REFERENCES

1. **Light RW**. Pleural effusions. *Med Clin North Am*. 2011 Nov;95(6):1055-70. doi:10.1016/j.mcna.2011.08.005. Epub 2011 Sep 25. Review. PubMed PMID: 22032427.
2. **Staats BA, Ellefson RD, Budahn LL, Dines DE, Prakash UB, Offord K**. The lipoprotein profile of chylous and nonchylous pleural effusions. *Mayo Clin Proc*. 1980 Nov;55(11):700-4. PubMed PMID: 7442324.
3. **Doerr CH, Miller DL, Ryu JH**. Chylothorax. *Semin Respir Crit Care Med*. 2001 Dec;22(6):617-26. PubMed PMID: 16088705.
4. **Nair SK, Petko M, Hayward MP**. Aetiology and management of chylothorax in adults. *Eur J Cardiothorac Surg*. 2007 Aug;32(2):362-9. Epub 2007 Jun 18. Review. PubMed PMID: 17580118.
5. **Skouras V, Kalomenidis I**. Chylothorax: diagnostic approach. *Curr Opin Pulm Med*. 2010 Jul;16(4):387-93. doi: 10.1097/MCP.0b013e328338dde2. Review. PubMed PMID: 20410823.
6. **Johnstone DW, Feins RH**. Chylothorax. *Chest Surg Clin N Am*. 1994 Aug;4(3):617-28. Review. PubMed PMID: 7953487.
7. **Kalomenidis I**. Octreotide and chylothorax. *Curr Opin Pulm Med*. 2006 Jul;12(4):264-7. Review. PubMed PMID: 16825878.
8. **Chalret du Rieu M, Baulieux J, Rode A, Mabrut JY**. Management of postoperative chylothorax. J Visc Surg. 2011 Oct;148(5):e346-52. doi:10.1016/j.jviscsurg.2011.09.006. Epub 2011 Oct 26. Review. Erratum in: *J Visc Surg*. 2012 Feb;149(1):e80. Baulieux, J [added]; Rhode, A [added]. PubMed PMID:22033151.
9. Valentine VG, Raffin TA. The management of chylothorax. Chest. 1992 Aug;102(2):586-91. Review. PubMed PMID: 1643953.
10. **Markham KM, Glover JL, Welsh RJ, Lucas RJ, Bendick PJ.** Octreotide in the treatment of thoracic duct injuries. *Am Surg*. 2000 Dec;66(12):1165-7. PubMed PMID: 11149591.
11. **Cope C**. Management of chylothorax via percutaneous embolization. *Curr Opin Pulm Med*. 2004 Jul;10(4):311-4. Review. PubMed PMID: 15220758.

Submitted on October 3, 2013

Table 1. **Selected causes of chylothorax***

- A. Trauma
  - a. Thoracic surgery
  - b. Penetrating and blunt trauma
- B. Malignancy
  - a. Lymphomas (70% of malignant causes)
  - b. Other malignant or benign tumors
- C. Idiopathic
  - a. Congenital
  - b. Other
- D. Miscellaneous
  - a. Infection
  - b. Sarcoidosis
  - c. Lymphangioleiomyomatosis (LAM), yellow nail syndrome, and many others.

***** adapted from Doerr CH, Miller DL, Ryu JH. Chylothorax. Seminars in Respiratory and Critical Care Medicine. 2001;22(6):617-26.

Figure 1A: Admission chest x-ray showed clear lungs

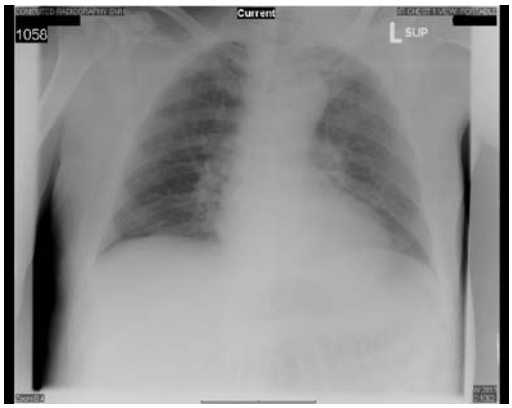

Figure 1B: Chest x-ray obtained few days later showed right sided pleural effusion

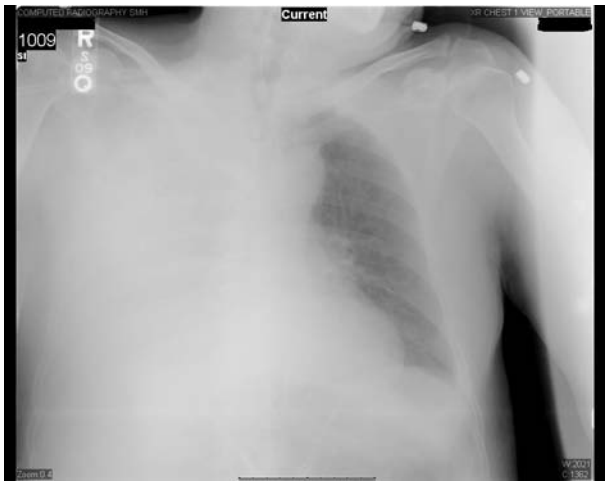

Figure 2: Chest computed tomography showed large right and small left pleural effusion