# Chylothorax after blunt trauma

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**ABSTRACT** Presented is a 50-year-old female who sustained a rare blunt traumatic chylothorax. Traumatic chylothoracies are usually the result of penetrating trauma and disruption of the thoracic duct. Diagnosis and treatment are discussed. The diagnosis is sometimes difficult in the trauma setting due to the possible presence of an underlying hemothorax or empyema and the usual delayed onset of chylothorax. Increased vigilance will allow physicians to properly diagnose and treat this condition early to avoid having to ligate the thoracic duct.

**KEY WORDS** Chylothorax; blunt trauma; thoracic

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## Introduction

Chylothorax is an accumulation of chyle in the pleural cavity due to a disruption of the thoracic duct. It was first described by Bartolet in 1633, and the first case was reported by Quincke in 1875 (1). Traumatic chylothoracies are usually the result of penetrating trauma and disruption of the thoracic duct. Thus, blunt traumatic chylothorax is a rare condition however, it should be considered in the differential diagnosis in patients who sustain chest trauma and subsequently develop a pleural effusion since it may lead to significant morbidity and mortality. This article reviews the diagnosis and treatment of this condition.

## Clinical summary

A 50-year-old female was involved in a motor vehicle collision and sustained a right acetabular fracture, and a left upper extremity fracture. Aside from hitting her chest in the car accident there were no skeletal fractures in her chest or spine. She was optimized for planned open reduction and internal fixation of her radius and acetabular fracture. The radius fracture was fixed first and the patient had some respiratory compromise, which delayed her acetabular repair. She had a past medical history significant for Wolf-Parkinson-White syndrome, for which she had an automatic implantable cardioverter-defibrillator (AICD) placed in 2005, atrial fibrillation, and cardiomyopathy. She had no effusion on admission. After 10 days of diuresis and conservative care for her respiratory compromise and congestive heart failure a left sided effusion presented itself on chest X-ray, which subsequently increased.

Tube thoracostomy drained 800 milliliters of white fluid. The surgical team began treatment for a suspected empyema with broad-spectrum antibiotics. Further evaluation was performed to evaluate for possible chylothorax. A triglyceride count was sent on the fluid and the patient was started empirically with total peripheral nutrition and nothing by mouth. The broad-spectrum antibiotics were continued due to concerns of an overlying empyema. This was a concern due to the appearance of the drained fluid and the rarity of chylothorax resulting from blunt trauma. The chest tube showed rapid decrease in drainage and the patient was placed on a low fat diet and subsequently had her acetabular fracture repaired. The chest tube was successfully removed 7 days after insertion. The patient recovered and the there was no radiographic evidence of the effusion 8 days after the chest tube was removed. The triglyceride count was 796 mg/dL, which lead us to the diagnosis of a chylothorax. This was the only test done to evaluate for chyle.

## Discussion

Chylothoraces can be classified as neoplastic, traumatic, congenital and miscellaneous (2). Neoplastic obstruction is the most common cause of non-traumatic chylothorax, where lymphoma accounts for 70% of cases (3). Traumatic cases of chylothorax are usually iatrogenic, secondary to operative procedures or from the complication of percutaneous placement

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of central venous catheters in 0.37% to 2% of thoracic procedures (4,5). Esophageal resection is the most common iatrogenic cause of chylothorax with an incidence of 4% (6). Central venous catheterization may directly damage the thoracic duct or cause venous thrombosis in the neck, which impedes the antegrade flow of chyle into the subclavian veins. Chylothorax also occurs after non-surgical trauma, including penetrating or blunt injuries to the chest or neck area, but this is usually an uncommon event (7). Nevertheless blunt injuries, especially to the vertebral bodies, as well as penetrating wounds can lacerate the thoracic duct. It is interesting to note that this case received an AICD pacemaker a year earlier. There is a described case of a patient who had a chylothorax 6 years after transvenous pacemaker implantation due to innonimate vein thrombosis, but our patient had no clinical evidence of this upon admission (8).

The most common form of blunt injury to the thoracic duct is produced by sudden hyperextension of the spine with rupture of the duct just above the diaphragm in the right thorax. Sudden stretching over the vertebral bodies may tear the thoracic duct, but this usually occurs in a duct previously affected by disease (9-12). The thoracic duct commonly originates from the cisterna chyli, which is located anterior to the second lumbar vertebra and posterolateral to the abdominal aorta. The thoracic duct ascends into the thorax through the aortic hiatus at the level of the tenth to twelfth thoracic vertebra and travels to the right of the vertebral column. At the level of the fifth or sixth thoracic vertebra the thoracic duct crosses posterior to the aorta and the aortic arch into the left posterior mediastinum. It courses anterior to the subclavian artery, within Poirier's triangle, and exits the superior thoracic aperture in the neck. After exiting the thorax, it forms an arch anterior to the anterior scalene muscle and turns inferiorly to terminate at the junction of the left subclavian and internal jugular veins (13). The pattern described is true only in approximately 65% of the population (14). Our patient encountered considerable trauma to her right hip & left upper extremity. From our review of blunt thoracic trauma and the mechanism of injury to the right side of her body, a right sided chylothorax would be a reasonable assumption however this patient developed a chylothorax on the right. Half of all chylothoraces are right-sided, one-third is left-sided, and the remainders are bilateral (15). Damage to her thoracic duct most likely occurred above the level of fifth or sixth thoracic vertebra. There is a slight possibility that the injury occurred at the junction between the thoracic duct and systemic circulation. Normally, the volume of chyle and lymph varies between 10 and 100 mL/kg body weight, depending on diet, intestinal absorption, and degree of physical activity (16). We would expect the amount of drainage from an injury around the entry site to be greater than the chest output seen in this patient, unless there was a minor injury. Although apparently rare, seat beltinduced thoracic duct injury should be considered as a cause of idiopathic chylothorax, especially when there is a history of a sudden increase in pressure to the thorax or upper abdomen that is temporally related (17).

The color of the pleural fluid is not always indicative of a chylothorax. Pleural fluid may not appear chylous if the patient is fasting or the pleural fluid is mixed with blood. In fact, 50% of patients may demonstrate bloody, yellow or green turbid, serous, or serosanguinous effusions (18). The typical milky appearance of the effusion may also be seen in pseudochylothorax or in an empyema where the purulent fluid contributes to the whitish color. Thus we employed a quantitative analysis of triglyceride in the pleural fluid for the diagnosis of a chylothorax. Staats *et al*. conducted a study in which triglyceride values were determined for 142 effusions defined as chylous or non-chylous by the gold standard test of lipoprotein electrophoresis. Using Gaussian distribution techniques, it was estimated that fluid with a triglyceride value of more than 110 mg/dL has less than a 1% chance of not being chylous and fluid with a triglyceride value less than 50 mg/dL has no more than a 5% chance of being chylous (19). When the triglyceride level is between 55 and 110 mg/dL, a lipoprotein analysis is indicated to detect the presence of chylomicrons in the pleural fluid. In our patient, the triglyceride level was greater than 110 mg/dL which lead us to believe that her pleural effusion was due to a chylothorax from blunt chest trauma.

There are several techniques used in the diagnosis of a chylothorax. The most definitive diagnosis is based on the presence of chylomicrons in the pleural fluid. Chylomicrons stain with Sudan III stain and cytological preparations of pleural fluid thus stained may help identify chylomicrons (12). Lipoprotein analysis is another method to confirm the presence of chylomicrons but many institutions do not have the capability. Bipedal lymphangiography is a special imaging modality that has been recommended to identify the cause and detect the site and size of a leak (20). Unfortunately, access to pedal lymphatics is painful and technically difficult. A meal with a high fat content mixed with methylene blue leads to a blue-green discoloration of pleural fluid. This can help localize a thoracic duct injury but in the setting of chest trauma the leak may be from an esophageal perforation (12). Other important characteristics of the fluid are pH 7.4 to 7.8, lymphocyte predominance in cell count, or a specific gravity of 1.012 or higher (21). A chylothorax may be suspected if the pleural fluid to serum triglyceride ratio is more than 1 and a pleural fluid to serum cholesterol ratio is less than 1 (22).

Symptoms of a chylothorax typically have a gradual onset. Often, a latency period of 2-7 days exists between the time of injury and clinical evidence of chylothorax if the injury is not a major one (12). The longest latency period reported in the literature was 20 years (23). Two possible explanations for the delay in presentation of a chylothorax have been summarized

by Milano *et al.* (24). Chyle and lymph slowly collect in the posterior mediastinum until it ruptures and leaks into the pleural space. Bilateral chylothoraces have been seen on rare occasions when the mediastinal pleura is ruptured on both sides. Furthermore, the interruption of a normal diet after trauma with a lower chylous production may also cause a delay in the recognition of a chylothorax.

The thoracic duct transports chyle & lymph from the intestines, liver, abdominal wall and lower extremities into the systemic venous system. Chyle consists of chylomicron, triglyceride, fat-soluble vitamins and cholesterol. It has an electrolyte composition similar to that of serum (12). The concentration of protein in chyle is 2.2-6 g/dL (25). The loss of chyle and lymph into the pleural space can lead to loss of water, electrolytes, proteins, immunoglobulins, fat, and essential vitamins. Patients are usually able to compensate in the early stages but in advanced cases there may be signs and symptoms of malnutrition and hypovolemia. Acidosis, hyponatremia, and hypocalcemia are the most common abnormalities (26). Continued loss of proteins, immunoglobulins, and B/T-lymphocytes into the pleural space can lead to immunosuppression (27). At no time during this hospitalization did our patient present with clinical symptoms of chyle loss.

The treatment for chylothorax depends on its etiology, the amount of drainage and the clinical picture. Most treatment regimens include a combination of tube thoracostomy, nothing by mouth (NPO), medium-chain triglycerides, total peripheral nutrition (TPN) and observation. Patients frequently require aggressive nutritional support to reverse protein loss, electrolyte abnormalities, hypovolemia, and immunosuppression. In addition, there are some institutions that use octreotide infusions as a way to decrease the output of chylous effusions (28,29). Conservative measures have achieved as high as an 88% success rate (30). Chyle has an irritating nature which actually promotes pleurodesis and in half of patients the leak will stop spontaneously. The chest tube allows the lung to expand to close any fistula and to improve the clinical status. Patients are kept NPO to decrease the amount of chyle production and allow the duct time to heal since lymph flow is about 14 mL/ h in the fasting state and over 100 mL/h after eating (31). Although the duration of conservative management varies in the literature anywhere from 1-4 weeks, most authorities recommend conservative management for no more than 2 weeks (32). In general, surgical intervention offers better results than conservative management when the daily chyle leak exceeds 1 L/day for a period more than 5 days (32) or 1.5 L/day in an adult or >100 mL/kg body weight per day in a child (33). The main surgical treatment option is ligation of the thoracic duct where there is a leakage. This may be approached through an open thoracotomy or video-assisted thoracoscopic surgery (VATS). Many surgeons prefer to ligate the thoracic duct at the diaphragmatic level because this procedure has the advantage of stopping flow from any accessory ducts that may not be recognized (34,35). When the thoracic duct cannot be identified, talc pleurodesis may be used. This traditional technique has a success rate of 95% and negligible morbidity (36). Before the introduction of surgical ligation of the thoracic duct, the mortality rate from a chylothorax was greater than 50%. With the advent of TPN and surgical ligation for persistent leaks, the mortality rate of chylothorax became less than 10%. Newer modalities in the management of chylothorax are on the horizon. Recent reports have shown some success with percutaneous CT guided drainage, percutaneous embolization, and robotic surgery for the treatment of chylothoraces (37-40).

In summary, a chylothorax should be considered in the differential in patients who sustain chest trauma and subsequently develop a pleural effusion. Analysis of pleural fluid is paramount for the management of chylothorax. Patients who are symptomatic from chyle loss or have a high output chylothorax will require prompt surgical intervention. The beneficence of early minimally invasive techniques in the treatment of chylothorax may become apparent in the near future.

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