

Isolated thoracic duct injury from blunt force trauma

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ABSTRACT

Isolated thoracic duct injury is an uncommon clinical event and is rare in the setting of trauma. We describe a case of an isolated thoracic duct injury resulting in the development of bilateral chylothorax following a motor vehicle collision in the absence of any other definable injury. We outline the initial patient presentation and diagnosis. After failing a trial of conservative management the patient underwent lymphangiography followed by thoracic duct ligation with pleurodesis. This case highlights the importance of recognizing thoracic duct injury following trauma.

CASE REPORT

CASE REPORT

A 29-year-old male was brought to the Emergency Department following a motor vehicle collision whereby his vehicle was rear ended. Cervical spine radiographs were normal. Initial chest radiograph revealed small bilateral pleural effusions (Figure 1). The patient was discharged home as he was asymptomatic and did not warrant hospital admission. The next day the patient presented to hospital following an episode of difficulty breathing accompanied by flushing, diaphoresis, and vomiting. Repeat chest X-ray identified increasing bilateral large pleural effusions (Figure 2). A follow up Computed Tomography (CT) scan of the abdomen and thorax confirmed large bilateral pleural effusions (Figure 3). There were no fractures and no intra-abdominal injuries. On physical examination, the patient was hemodynamically stable with no signs of significant respiratory distress. There was mild tenderness of the right anterior thorax and decreased bilateral air entry to the base of the lungs.

A chest tube was inserted in the right mid-axillary line; from which, 1500 mL of white to strawberry-coloured fluid was drained with no evidence of blood. The effusion sample was found to have a triglyceride level of 42.26 mmol/L and cholesterol of 2.86 mmol/L indicating the presence of chyle. A left chest tube was also placed for a large chylothorax.

Initially, the patient was treated conservatively for his chylothorax with octreotide, total parenteral nutrition and chest tube drainage. With little improvement in the chylothorax after 5 days, an intranodal ultrasound (US) guided lymphangiogram was performed. Lipiodol (30 mL total) was injected slowly into the left and right inguinal lymph nodes accessed with a 22-gauge needle under direct ultrasonic guidance. Intermittent fluoroscopy showed bilateral uptake into the lymphatic chain (Figure 4a). Over time, an amorphous area of Lipiodol began to accumulate over the 10th thoracic vertebra (Figure 4b). Increased attenuation to the left of the midline between the 11th-12th left ribs was suspected to be pooling of the Lipiodol in the left pleural space. A follow up non-contrast CT of the thorax confirmed an amorphous

collection of lipiodol spilling directly from the well opacified thoracic duct at the 10th thoracic vertebra with relative diminished caliber of the thoracic duct thereafter, in keeping with disruption of the thoracic duct (Figure 5).

After 9 days of non-operative treatment with no resolution of the patient's chylothorax, surgical management was offered. An open surgical approach was undertaken with a right sided posterolateral thoracotomy. The thoracic duct was ligated at the level of the identified thoracic duct leak. Pleurodesis was performed using a talc mixture to prevent reaccumulation of chyle within the pleural space. Bilateral chest tubes and a left sided pigtail pleural catheter were left in place. Following the procedure, a portable chest radiograph revealed small bilateral effusions (Figure 6). The patient was slowly transitioned back to an oral diet and the chest tubes were removed. A chest X-ray performed 6 weeks post-surgery demonstrated resolving chylothorax (Figure 7).

DISCUSSION

Etiology & Demographics:

Blunt force trauma is a rare cause of thoracic duct injury, particularly in the absence of any other associated injuries. Approximately 50% of thoracic duct injuries are the result of a traumatic injury [1]. Of those traumatic injuries 80% are iatrogenic [2]. The other 20% are caused by penetrating or blunt trauma. Although rare, isolated thoracic duct injuries have also been reported with hyperextension of the spine or forceful cough and emesis [3-7].

The thoracic duct is created by the joining of lymphatic vessels from the lower limbs at the point of the cisterna chyli within the retroperitoneum located at roughly the second lumbar vertebra. The duct ascends between the aorta and the azygos vein, anterior to the vertebral column. Caudally, the thoracic duct runs to the right of the aorta in the aorto-esophageal recesses. However, at the level of the fourth to sixth thoracic vertebrae the thoracic duct crosses over the midline to travel posterior to the esophagus. This anatomy is present in ~50% of the population [8]. In keeping with the common anatomy, injury below the level of the fifth thoracic vertebra often leads to a right sided chylothorax, while injury above the fifth thoracic vertebra is associated with effusion on the left [9]. Damage below the diaphragm results in chylous ascites.

Given the location of the thoracic duct within the body it is seldom injured in isolation. It is understandable how an isolated thoracic duct injury can occur following penetrating trauma and there are numerous cases reported in the literature describing this event [10-12]. Many cases of thoracic duct injury following blunt force trauma do not happen in isolation, often these cases also involve fractures within the thorax [13-21]. These fractures may be associated with direct trauma to the thoracic duct leading to a tear in the lymphatics. Damage to the thoracic duct in which there are no other injuries present are quite rare. To our knowledge there have only been 9 cases of chylothorax resulting from an isolated thoracic duct injury following blunt trauma reported in the literature since 1990

[19,22-28] with 13 cases of isolated chyloperitoneum or chyloretroperitoneum [29-41].

In general, chylothorax is a relatively uncommon clinical event. It is most often associated with malignancy or postoperative complication of thoracic surgery. A retrospective case review performed by Doerr et al. (2005) examined 203 patients at the Mayo Clinic that developed a chylothorax over a period of 21 years. Almost half of the reported cases at their institution were the result of surgery or trauma. The vast majority within this group were iatrogenic in nature with the highest rates being due to esophagectomy, cardiovascular surgery or mass resection in the mediastinum [1]. Despite the most common cause of chylothorax being due to surgical complications, it is only seen in roughly 0.4% of surgical procedures in the thorax [42]. Outside of surgical trauma and malignancy, chylothorax is exceedingly rare. Valentine and Raffin found that only 3% of cases of chylothorax were due to non-surgical trauma after reviewing 191 cases from 1964-1986 [43]. Doerr et al. (2005) found only a single case of chylothorax due to blunt force trauma out of the 203 cases studied [1].

There are case reports of spontaneous chylothorax in which there is no apparent trauma. It is postulated that these spontaneous events have the same etiology as blunt trauma in which the thoracic duct is damaged through increased intrathoracic pressure and/or hyperextension of the spine [3-7]. In 1952, Meade studied 5 such cases of spontaneous chylothorax in which no direct cause could be identified. Upon further review, it was noted that 4 out of the 5 patients experienced sudden hyperextension of the spine prior to the development of chylothorax. Reviewing the patient histories, all 5 patients had evidence of fixation of the thoracic duct to the vertebrae through prior trauma, infection or congenital anomaly [7].

One of the first reviews in the literature regarding traumatic chylothorax was done by McNab and Scarlett in 1932. They reported 34 cases of traumatic chylothorax with 28 cases being due to blunt trauma. Of the 28, there were 19 cases of isolated thoracic duct injury, with no external wounds or fracture to the ribs or vertebrae. Most injuries were the result of automobile accidents or falls from moderate heights [44]. This early report formed a large base of our knowledge regarding traumatic thoracic duct injury. However, it does not appear to reflect the cases reported in the current literature. Perhaps this is due to increased force of impact in modern automobile accidents resulting in more severe trauma than an isolated thoracic duct injury. Additionally, in the early 1900s there was a decreased ability to detect underlying medical conditions and/or malignancies that could have made the thoracic duct more prone to rupture. Due to these limitations there is potential that some of the cases of isolated thoracic duct injury presented by McNab and Scarlett (1932) were truly due to underlying medical conditions and may have been misattributed to blunt trauma.

Clinical & Imaging findings:

Chylothorax is difficult to differentiate from other causes of pleural effusion on most imaging modalities. On US a chylothorax will appear as a homogeneous region of

echogenicity [45]. There should not be any areas of loculation or separation [45]. On chest X-ray a pleural effusion is characterized by blunting of the costophrenic and cardiophrenic angles. For a chylothorax the area of effusion will appear homogeneous [45]. The use of CT may provide a degree of differentiation of the underlying etiology of the pleural effusion, although its utility is limited. A chylous effusion will have a lower attenuation value than other forms of pleural effusion [45]. Conclusive diagnosis requires a laboratory analysis of the pleural fluid with triglyceride level >1.24 mmol/L with a cholesterol level <5.18 mmol/L [1]. Once the diagnosis of chylothorax has been reached, lymphangiography is the gold standard to determine the precise location of the thoracic duct injury. The American College of Radiology recommends only using CT and Magnetic Resonance Imaging (MRI) when the precise anatomic details required for surgical planning are not obtained from lymphangiography [46]. Proper visualization of the site of injury within the thoracic duct using MRI requires gadolinium contrast injected in a similar fashion to lymphangiography [47]. Lymphangiography remains invaluable to identify anatomic details.

Treatment & Prognosis:

If lymphatic leakage goes unnoticed serious complications can develop. Large losses of chyle can lead to hypovolemia if the fluid volume is not adequately replaced [48]. Chyle is dense in nutrients and lymphatic cells and continuous loss of these biologically important molecules and cells can lead to malnutrition and immunosuppression [48]. There is typically a delay between the time of injury and the presentation of a chylothorax to medical attention. The delay is due to the time it takes for the chyle to leak into the mediastinum and the pressure to be large enough to enter the pleural space. Typically, this delay can be from 2-7 days [44,48]. Approximately 57% of patients initially present with dyspnea, however 37% of patients may experience no symptoms at the time of diagnosis [1]. If untreated the mortality rate for traumatic chylothorax is 50% [44]. The delayed presentation and potentially insidious onset of a chylothorax can cause difficulty in diagnosis, especially for a rare etiology. Therefore, it is important to recognize potential chylothorax following blunt force trauma.

There are currently no treatment guidelines for the management of lymphatic duct injury. Generally, a trial of conservative management is taken before offering definitive surgical treatment. The utility of conservative management is variable, with the literature quoting success rates between 20-80% [2]. There is currently no defined length of time that conservative management of chylothorax should be trialed, although consensus is not to go beyond 2 weeks [49].

Conservative management typically includes thoracentesis and nil per os (NPO) status while the patient is maintained with total parenteral nutrition. The administered nutrition should be low in fat to minimize the body's production of chyle. Thoracentesis is done for symptom management of the developing chylothorax. NPO status is required to reduce the volume of chyle that is lost and allow the thoracic duct injury to heal [17]. Octreotide can be given as it can slow the production of chyle [2].

If a trial of conservative therapy is unsuccessful, lymphangiography may provide both therapeutic and diagnostic benefit. A contrast agent, typically lipiodol, is injected into the lymphatic system by either pedal or inguinal nodes under US guidance. Targeting of the inguinal nodes has become common practice as the transit time to the site of leak is reduced, therefore reducing time, volume of lipiodol used and radiation dose. Serial fluoroscopic images are then obtained approximately every 5-10 minutes to track uptake into the thoracic duct. Traditionally, lymphangiography was performed prior to surgery to identify the site of leak and aid in surgical planning. However, recent data has shown that lymphangiography alone is enough to correct thoracic duct injuries in 50-90% of instances [50-53]. With the recent introduction of US guided intranodal lymphangiography at our institution, as performed by the senior author, the local rate of therapeutic benefit is currently at 50%. The exact mechanism in treating chyle leakage is unknown. Matsumoto et al. (2009) hypothesizes that lipiodol accumulation causes an inflammatory reaction that leads to therapeutic embolization [50]. Lymphangiography carries with it the standard risks of bleeding, infection, and pain. In rare cases serious complications such as lipiodol related pulmonary embolisms or anaphylaxis can occur [54].

If the chylous leak persists following the lymphangiogram, treatment can be escalated to more definitive options such as thoracic duct embolization or thoracic duct ligation. Thoracic duct embolization (TDE) is an interventional technique done immediately after lymphangiography. The cisterna chyli is targeted through percutaneous transabdominal catheterization while the thoracic duct and site of injury are visible due to the injected contrast. The catheter is advanced to the point of the leakage under fluoroscopic guidance. Metallic coils and/or adhesive substances are then deployed to cause embolization at the site of injury. If the embolization procedure is completed it has a 90% rate of success [55]. Unfortunately, TDE is only successfully completed in approximately 67% of cases cannulated [55]. If TDE is unsuccessful a technique known as thoracic duct disruption can be used in which a needle is repeatedly passed through the duct in an effort to interrupt the lymphatic vessel and prevent further leakage. TDE carries a risk of developing protein-losing enteropathy, lymphedema, and chylous ascites [56]. Alternatively, thoracic duct ligation (TDL) is the act of repairing the point of thoracic duct leakage with sutures. This may be performed by thoracotomy or more commonly using video assistance. A high degree of surgical accuracy is required as the thoracic duct travels alongside critical blood vessels as well as the esophagus. To reduce the risk of perforation of the esophagus an orogastric tube is often used, which can be filled with methylene blue stained creams to improve recognition [57]. Overall, TDL is a relatively safe procedure with a high overall success rate of 90% [2]. The major postoperative complication is lymphedema, however over time symptoms usually decrease as collateral lymphatics develop [45]. Pleurodesis is a technique to cause scarring within the pleural space to prevent reaccumulation of fluid. This can be done alone or in conjunction with thoracic duct ligation.

Differential Diagnosis:

Radiographs and CT are the mainstay of detection for the presence of pleural effusion. US can play a role in narrowing down differential diagnoses in settings with limited resources or increasing utilization of point of care US. Despite the importance of imaging, to reach a conclusive diagnosis of the underlying etiology, a sample of the pleural fluid must be drawn for laboratory analysis.

Transudative effusions occur in the context of systemic processes causing an imbalance in the Starling forces. Therefore, on imaging they often present bilaterally in the basal portion of the lungs. These pleural effusions will have a homogenous, fluid-like appearance. MRI has limited use in delineating the etiology of a pleural effusion aside from distinguishing transudative from exudative effusions. On diffusion weighted MRI transudative effusions will appear isointense while exudative effusions are hyperintense [58]. The Light's Criteria is used to define a transudative effusion: a) pleural fluid/serum protein ratio <0.5 , b) pleural fluid LDH/serum LDH <0.6 , or c) pleural LDH $< 2/3$ the upper limit of normal serum LDH [59].

Pleural effusions may be seen alongside pneumonia. The pleural space can be filled with simple fluid or pus. These parapneumonic pleural effusions or empyemas occur in approximately 2-3% of all cases of pneumonia [60]. The presence of typical symptoms or radiologic evidence of pneumonia may help direct your differential diagnosis. For a simple parapneumonic effusion, the pleural fluid will collect in the dependent areas of the lungs. US examination may show evidence of consolidation. If an empyema develops adhesions may be visible on chest radiographs as areas of loculation with tapered borders. On contrast enhanced CT there will be a high degree of enhancement of both the visceral and parietal pleura creating the split pleura sign [61]. Pleural thickening may also be present [62]. In an empyema the retrieved sample of pleural fluid will contain pus with a positive bacterial culture.

Tuberculosis (TB) related effusions may be visible on US as complex septated effusions [63]. TB typically presents on X-ray and CT as areas of focal infiltration in either the upper or lower lobes [64]. Approximately 24% of patients with TB will develop a pleural effusion [65]. Other signs of TB such as cavitation, consolidation, inflammation, as well as enlarged hilar and mediastinal lymph nodes may be present [64]. However, unilateral pleural effusion may be the sole manifestation [63]. The heightened resolution of contrast enhanced CT may reveal rim enhancing collections, loculations and septations in the case of pleural TB [66]. The diagnosis requires inoculation of *Mycobacterium tuberculosis* and a positive acid-fast bacilli stain from a sample of bodily fluid.

Primary pleural malignancy, hematological malignancy, or metastases to the pleura may lead to the development of pleural effusion. US may reveal heterogeneous echogenicity within the pleural fluid, thereby delineating the entity from a simple effusion. Indicators such as pulmonary masses or nodules, atelectasis from bronchial obstruction, or lymphadenopathy may be seen on radiographs. On CT pleural irregularities, nodules, or thickening of the pleura may be noted. In order to conclusively diagnose a malignant pleural

effusion positive cytology on laboratory analysis must be obtained.

In the case of hemothorax, US has little utility beyond the detection of pleural effusion. Blood in the pleural space will appear as a homogeneous density, with little characterization of the fluid or the source. Large hemothoraces greater than 500 mL can be detected on upright chest X-ray [67]. To detect hemothoraces less than 500 mL, CT imaging is required [68]. On CT fresh blood will have an elevated attenuation of greater than 35 Hounsfield Unit (HU), while clotted blood can have a HU of approximately 70 [69]. Hemothorax can be conclusively identified if the ratio of pleural fluid/blood hematocrit is found to be ≥ 0.5 [70]. However, pleural fluid/blood hematocrit in the range of 0.25-0.5 may be related to a hemothorax that has undergone some form of secondary dilution [70].

Conclusion:

Isolated thoracic duct injury is a rare outcome of blunt force trauma. Presentation is often delayed until the development of a symptomatic chylothorax. Thoracic duct injury and subsequent chylothorax should be considered in patients experiencing late onset dyspnea following a traumatic injury. Conservative management can be trialed. Lymphangiography is a useful tool to aid in the diagnosis, localization, and management of thoracic duct injury. However, definitive surgical management should be offered if the chylothorax fails to resolve within a reasonable period of time.

TEACHING POINT

Isolated thoracic duct injury resulting in chylothorax due to blunt trauma is exceptionally rare and may be difficult to detect due to the delayed and insidious presentation. Once the presence of a chylothorax is detected, lymphangiography can be useful for localization and surgical planning, it may also treat the thoracic duct leak in approximately 50-90% of cases.

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FIGURES

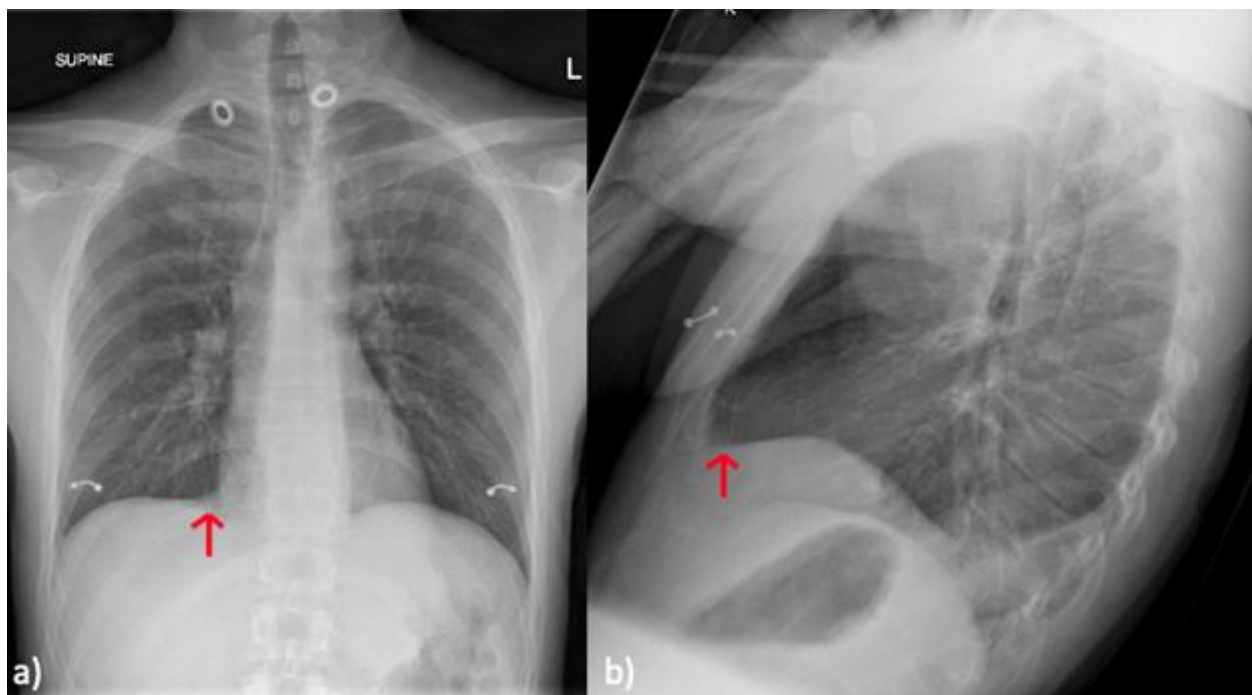


Figure 1: Chest radiographs of a 29-year-old male with bilateral chylothorax.

FINDINGS: a) Posterior-anterior view and b) lateral view of the chest reveals bilateral blunting of the costophrenic and costocardiac angles suggestive of a small bilateral pleural effusion.

TECHNIQUE: Plain film of the chest taken at 120 kVp.

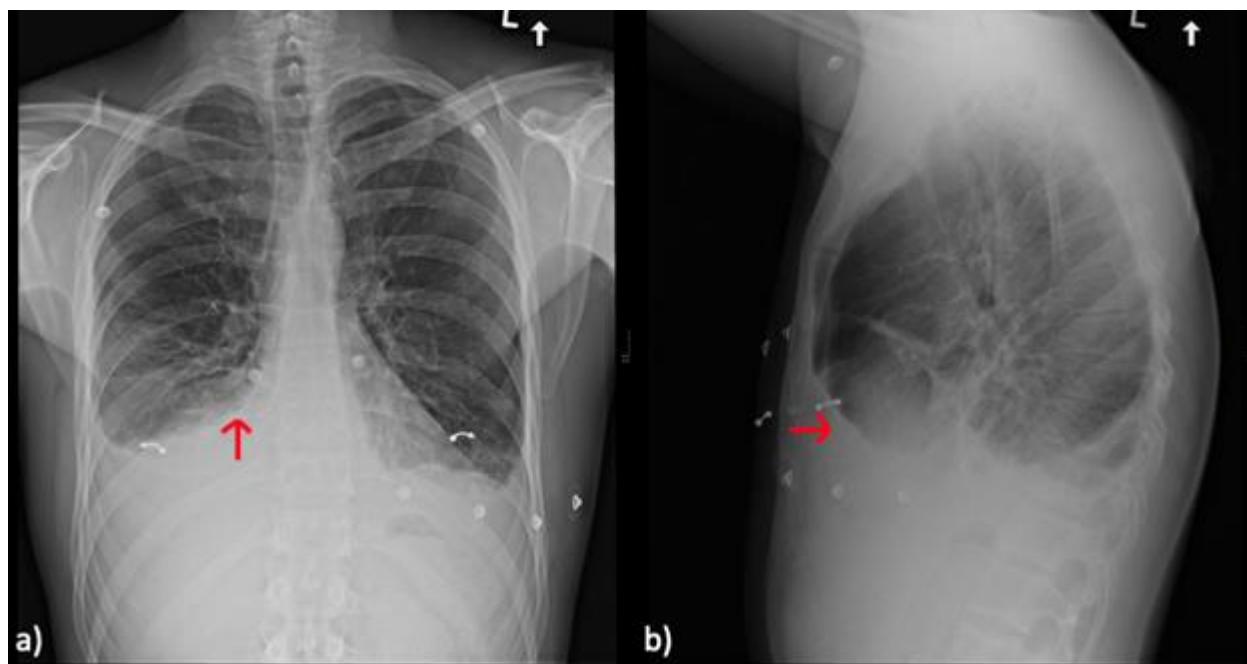


Figure 2: Chest X-ray of a 29-year-old male with bilateral chylothorax.

FINDINGS: a) Posterior-anterior view and b) lateral view of the chest reveals enlarging bilateral pleural effusions now moderate in volume on the right and small to moderate in volume on the left.

TECHNIQUE: Plain film of the chest taken at 120 kVp.



Figure 3 (left): Initial CT of a 29-year-old male with bilateral chylothorax.

FINDINGS: Large right and a moderate left pleural effusion with low attenuation.

TECHNIQUE: Contrast enhanced axial CT thorax with multiplanar reformats. A CT GE LightSpeed VCT was used. Images were taken at 120 kVp, mA of 225, with 5 mm slice thickness. 120mL of Omnipaque 350 were provided.

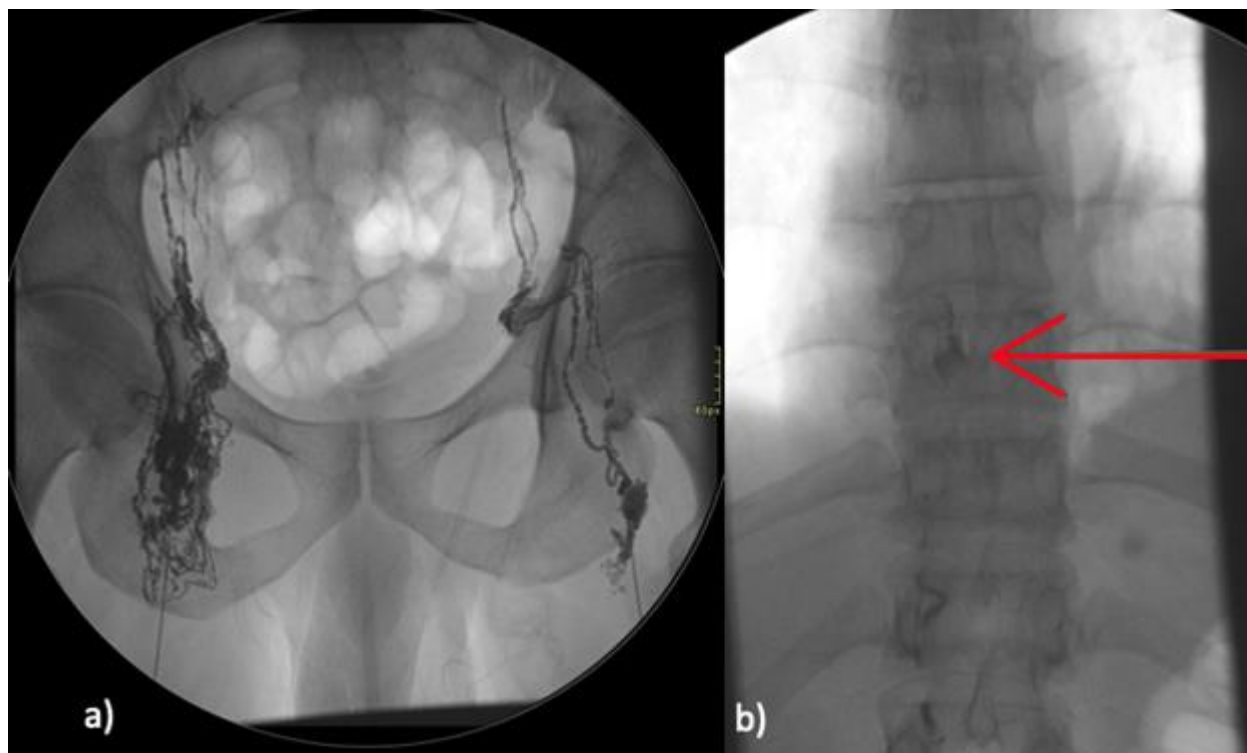


Figure 4: Bilateral pelvic lymphangiogram on a 29-year-old male presenting with bilateral chylothorax.

FINDINGS: a) Following infusion of Lipiodol excellent uptake can be seen in the pelvic lymphatic chain bilaterally. b) An amorphous pool of Lipiodol begins to form over the 10th thoracic vertebral level as indicated by the yellow arrow.

TECHNIQUE: Bilateral inguinal lymph nodes were targeted with a 22-gauge needle under direct ultrasound guidance and 30 mL of Lipiodol was slowly infused into the lymphatic system. Intermittent fluoroscopy was subsequently performed.

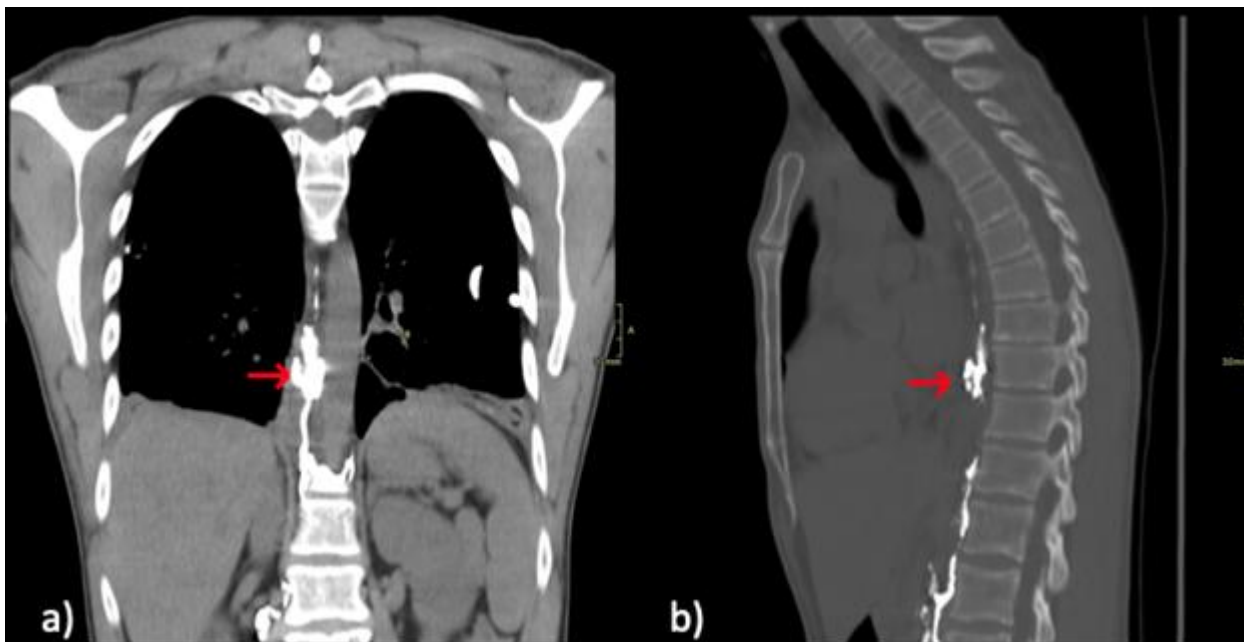


Figure 5: Non-contrast CT following percutaneous lymphangiogram on 29-year-old male presenting with bilateral chylothorax.

FINDINGS: Follow up CT confirmed the presence of an amorphous accumulation of Lipiodol to the right of the thoracic aorta centered at the 10th thoracic vertebral body as indicated by the red arrow in a) the coronal plane and b) the sagittal plane. The intact thoracic duct can be seen below this and the diminished caliber thoracic duct is shown above the disruption/transection

TECHNIQUE: Non-contrast axial CT with multiplanar reformats. A CT GE LightSpeed VCT was used. Images were taken at 120 kVp, 210 mA, with 3 mm slice thickness.

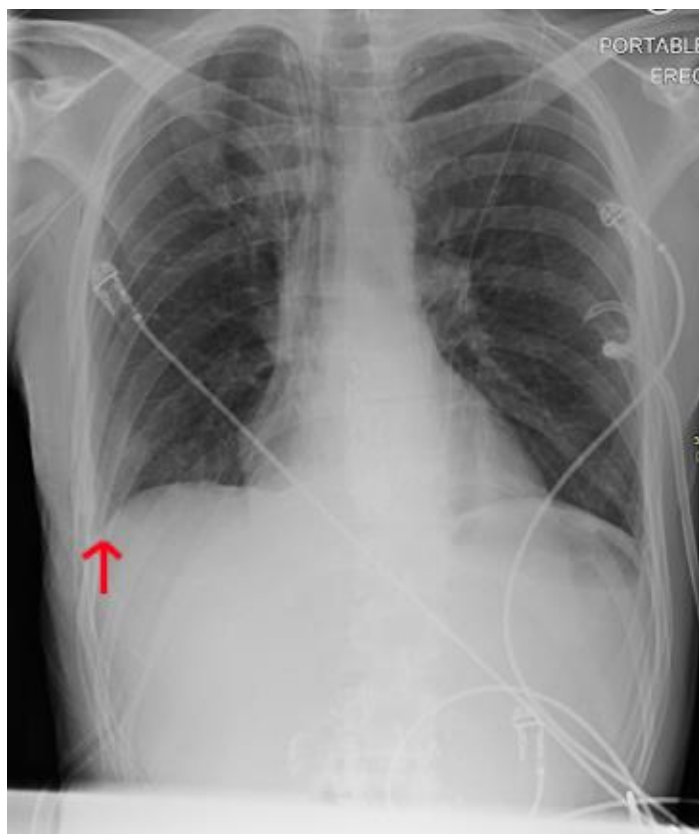


Figure 6 (left): Postoperative portable chest radiograph following thoracic duct ligation and talc pleurodesis on a 29-year-old male presenting with bilateral chylothorax.

FINDINGS: First postoperative portable chest radiograph. There are bilateral chest tubes and a pigtail pleural catheter on the left side. There is mild blunting of the costophrenic recess bilaterally, in keeping with small pleural effusions.

TECHNIQUE: Plain film of the chest taken at 101 kVp.

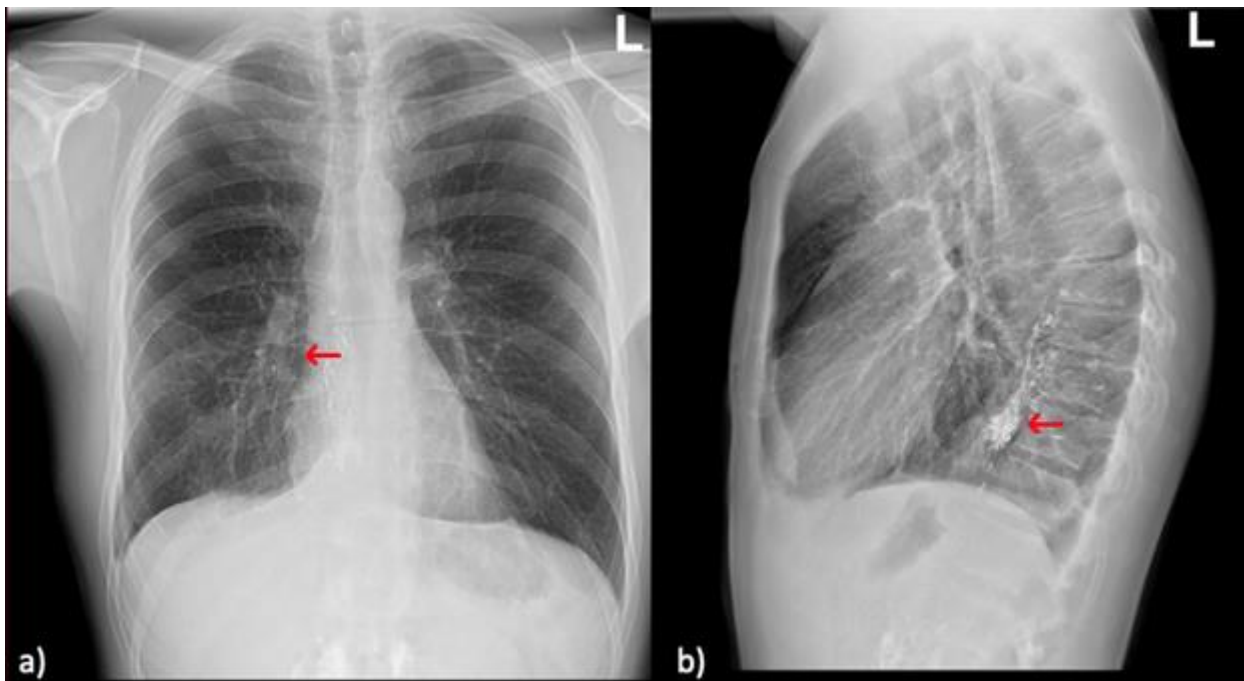


Figure 7: Chest radiographs of 29-year-old male 6 weeks post thoracic duct ligation with talc pleurodesis for bilateral chylothorax.

FINDINGS: Irregular dense material in the middle/posterior mediastinum and epigastric region represents resolving retained lymphangiogram contrast. Trace residual right pleural effusion.

TECHNIQUE: Plain film of the chest taken at 120 kVp.

Etiology	Increased intrathoracic pressure and/or hyperextension of the spine leading to damage of the thoracic duct
Incidence	Insufficient evidence to determine incidence
Gender Ratio	No predilection
Age predilection	No predilection
Risk Factors	Prior trauma to thoracic duct, infection, congenital anomaly, and malignancy
Treatments	Conservative management: NPO status, total parenteral nutrition, octreotide, thoracentesis Definitive management: lymphangiogram, thoracic duct embolization/disruption, thoracic duct ligation ± pleurodesis
Prognosis	If untreated up to 50% mortality rate. Thoracic duct ligation has a 90% success rate with minimal long-term sequelae.
Findings on Imaging	Presence of pleural effusion: Homogeneous opacities causing blunting of the costophrenic and costocardiac angles.

Table 1: Summary table of isolated thoracic duct injury and chylothorax due to blunt trauma.

	US	X-Ray	CT	Laboratory Analysis
Chylothorax	<ul style="list-style-type: none"> • Homogeneous anechoic collection • No areas of loculation or separation 	<ul style="list-style-type: none"> • Blunting of the costophrenic and costocardiac angles • Homogeneous density 	<ul style="list-style-type: none"> • Low attenuation compared to other causes of pleural effusion • Evidence of damage to thoracic duct 	<ul style="list-style-type: none"> • Triglyceride >1.24 mmol/L • Cholesterol < 5.18 mmol/L
Transudative Effusion	Anechoic fluid collection.	Bilateral blunting of the costophrenic and costocardiac angles	Homogeneous, fluid attenuation collection	Negative Light's Criteria
Parapneumonic Pleural Effusion/ Empyema	<ul style="list-style-type: none"> • ±Evidence of consolidation • Adhesions may be detectable on US if empyema develops 	<ul style="list-style-type: none"> • Presence of lobar, bronchial, or interstitial pneumonia • Parapneumonic effusion will localize to the dependent areas of the lungs • Empyema will initially be freely layering but may become loculated 	<ul style="list-style-type: none"> • Presence of split pleural sign due to elevated enhancement of pleural lining • Pleural thickening 	Empyema will have a positive culture
Tuberculosis	±Septated effusions	<ul style="list-style-type: none"> • Focal infiltration of upper or lower lobes • Cavitation and inflammation • Enlarged hilar and mediastinal lymph nodes 	<ul style="list-style-type: none"> • Variable appearance • Pleural TB may show rim enhancing collection, loculations/septations 	<ul style="list-style-type: none"> • Positive acid-fast bacilli stain • Positive culture of Mycobacterium tuberculosis
Malignant Pleural Effusion	May demonstrate a lesion in the pleural fluid, may be heterogeneous echogenicity	Potential indicators for malignancy such as pulmonary masses, nodules, atelectasis from bronchial obstruction or lymphadenopathy	Irregularities, nodules or thickening of the pleura present	Positive cytology
Hemothorax	Homogenous echogenicity pleural collection	<ul style="list-style-type: none"> • Hemothoraces greater than 500 mL are detectable on upright chest X-ray • Blunting of the costophrenic and costocardiac angles 	<ul style="list-style-type: none"> • Can detect smaller hemothoraces than X-ray • Fresh blood has attenuation of approximately 35 HU while clotted blood may have an attenuation of approximately 70 HU 	Ratio of pleural fluid/blood > 0.25 - 0.5

Table 2: Differential diagnosis table for chylothorax.

ABBREVIATIONS

CT = Computed tomography
 HU = Hounsfield Unit
 MRI = Magnetic resonance imaging
 NPO = Nil per os
 TB = Tuberculosis
 TDE = Thoracic duct embolization
 TDL = Thoracic duct ligation
 US = Ultrasound

KEYWORDS

lymphangiogram; thoracic duct; chylothorax; trauma; lymphatics

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