Chylothorax: diagnostic approach

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Current Opinion in Pulmonary Medicine 2010, 16:387–393

Purpose of review

This review evaluates recent research findings and proposes an up-to-date diagnostic approach for patients with suspected chylothorax.

Recent findings

Typically, chylothorax is a milky exudate with high triglyceride content (>110 mg/dl). However, milky appearance is not always the case and triglyceride levels can be less than 110 mg/dl, especially in fasting or malnourished patients. Transudative chylothoraces have been reported when cirrhosis, nephrosis or heart failure co-exist. In addition, although the vast majority of the white blood cells in chyle are lymphocytes, chylothoraces can be neutrophilic, especially the postsurgical ones.

Summary

Chylothorax is the accumulation of chyle into the pleural cavity usually due to thoracic duct leak and should be suspected not only in patients with milky effusions but also in the presence of certain co-morbidities or history of chest/neck trauma. Fluid triglycerides more than 110 mg/dl or less than 50 mg/dl virtually establish or exclude the diagnosis, respectively; ambiguous cases with values 50–110 mg/dl require lipoprotein analysis for the demonstration of chylomicrons. In fasting or malnourished patients lipoprotein analysis is suggested even with triglycerides less than 50 mg/dl. Typical pleural fluid in chylothorax is a lymphocytic exudate with low lactate dehydrogenase; atypical fluid characteristics (i.e. transudative nature, neutrophil-predominance or high lactate dehydrogenase) may be a sign of additional causes of pleural fluid accumulation.

Keywords

chylomicrons, chylous, triglycerides

Curr Opin Pulm Med 16:387–393

1070-5287

Introduction

Chylothorax or chylous pleural effusion (CPE) is defined as a pleural effusion containing chyle. The presence of chyle in the pleural space is usually due to thoracic duct disruption. Clinically, chylothorax usually presents with progressive breathlessness of acute or subacute onset. Diagnostic thoracentesis usually results in the aspiration of a milky lymphocyte-predominant exudative fluid. Diagnosis is confirmed when pleural fluid is found to contain chylomicrons or when the pleural fluid triglyceride levels exceed 110 mg/dl [1–3]. However, most clinicians would order these tests only in patients with milky or turbid effusions. Are there any other clinical conditions in which pleural fluid lipid analysis could be indicated even in nonmilky effusions? Should the practitioner consider the possibility of chylothorax in any group of patients presenting with transudative or neutrophil-predominant pleural effusion? In the present article we try to address these questions based on recently reported clinical observations and we attempt to provide a comprehensive diagnostic approach for patients with suspected cylothorax.

Anatomy and physiology

Dietary triglycerides are degraded in the small intestine into free fatty acids which diffuse into the intestinal cells after a number of assimilation processes. Long-chain fatty acids (>12 carbons) incorporated into triglycerides combine with cholester-esters, retinyl-esters, phospholipids and cholesterol to form chylomicrons, which leave the intestine in the lymph channels that terminate in the cisterna chyli. The cisterna chyli contains a bacteriostatic, nonirritating, milky fluid called chyle which is characterized by T-lymphocyte predominance, protein concentration of 2.2–6.0 g/dl, high triglyceride levels (>110 mg/dl) and cholesterol levels (65–220 mg/dl) lower than those of serum [1,4–7].

Arising from the cisterna chyli at the level of the L2 vertebrae, the thoracic duct enters the chest cavity through the esophageal hiatus and ascends extrapleurally in the posterior mediastinum, along the right surface of the vertebral column. At the carina, it crosses to the left of the vertebral column and continues cephalad. After

exiting the mediastinum, it arches above the clavicle and descends anterior to the subclavian artery to terminate in the junction of the left internal jugular and left subclavian veins. This anatomic pattern is present in approximately 50% of individuals, whereas wide anatomic variations exist in the remainder [1,2,8–10].

Etiology and pathogenesis

The accumulation of chyle in the pleural cavity may be due to rupture of the thoracic duct and/or its tributaries, leakage from the pleural lymphatics and/or collateral vessels, or transdiaphragmatic flow of chyle from the peritoneal cavity in patients with chylous ascites. The main pathogenetic mechanisms of chyle leak are interruption of lymphatic wall integrity due to direct injury, straining or infiltration by abnormal tissue, and lymph vessel rupture due to elevated intraluminal pressure as a result of obstruction, increased intraluminal lymph volume, abnormal lymphatic lumen or increased venous pressure [10–23]. Situations evoking any of these mechanisms represent chylothorax causes (Table 1), which are grouped into four major categories: trauma, malignancy, miscellaneous and idiopathic [1].

Trauma of the large supra-diaphragmatic lymphatics is the most common cause of chylothorax, accounting for approximately 50% of cases [15,24••]. Iatrogenic trauma of thoracic duct and/or its tributaries results from surgical and minimally invasive procedures to the chest or neck. Although any chest or neck intervention (Table 1) can result in lymph vessel injury, esophagectomy is the operation most commonly complicated with chylothorax [15,19,24••,25•,26–31]. Other interventions, such as central venous catheter placement, tube thoracostomy, pacemaker implantation, pulmonary artery embolization and thoracic irradiation, are responsible for approximately 5% of chylothoraces [15]. Penetrating or blunt thoracic trauma, weight lifting, straining, coughing, vomiting, yawning, seat belt injury and childbirth are the main noniatrogenic causes of lymphatic injury [32–37]. Malignancy is the underlying cause in approximately 30% of chylothoraces, representing the second most frequent cause [1,15,24••,38,39]. Because lymphoma accounts for 70–75% of the malignancy-associated chylous pleural effusions, it should be always considered in chylothoraces that cannot be attributed to another obvious cause [1,15,40]. The 'miscellaneous' category includes a variety of diseases (Table 1) that can cause chylothorax by diverse pathogenetic mechanisms [15,21,24••,41–48]. If no cause is identified, chylothorax is termed idiopathic.

Diagnostic approach

The diagnostic approach of chylothorax (Fig. 1) consists of three sequential steps: first, one should suspect the diagnosis, second the presence of chyle in the pleural cavity should be verified and third, the underlying abnormality should be defined.

Chylothorax suspicion

Chylothorax is certainly suspected when milky fluid is aspirated. However, for reasons discussed below, this diagnosis could also be considered in patients with pleural effusions even when the fluid is nonmilky if certain diseases commonly associated with chylothorax are present, especially, but not exclusively, in fasting or malnourished patients.

Medical history and clinical presentation

The diseases reported to cause chylothorax are numerous (Table 1). Some specific morbidities though, such as recent chest or neck intervention, malignancy, superior vena cava or subclavian vein thrombosis, liver cirrhosis and lymphatic disorders, are more frequently related and should alert the clinician for the possibility of chylothorax [1,10–21,24••,25•,26,32,35,38,41,42,49••]. Symptoms of chylothorax namely dyspnea, nonproductive cough and chest discomfort are similar to that of pleural effusions of other etiologies and, therefore, are not really useful in the diagnostic approach. Typically, chylothorax is not accompanied by pleuritic chest pain and fever, as chyle does not cause pleural inflammation [1,2,38].

Pleural fluid appearance

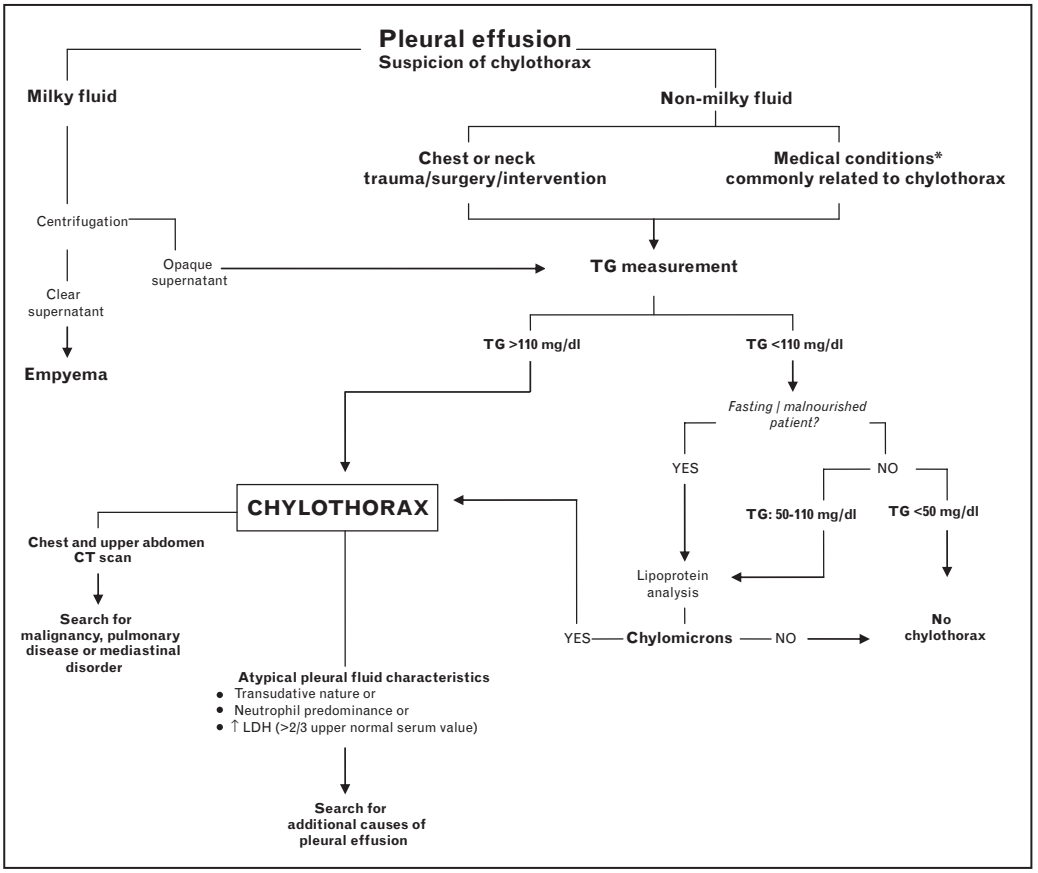
Although chylothorax has been traditionally linked to the aspiration of a white, odorless, milky fluid, this is not always the case [1,3]. In a recent study by Maldonado *et al.* [24••], out of 61 chylothoraces that had pleural fluid appearance documented, only 27 (44%) were milky, the rest being serous (26%), serosanguinous (26%) or bloody (3%). Similar results were reported by Agrawal *et al.* [49••] in a smaller sample ($n = 22$) of chylothorax patients, of which pleural effusion was milky or turbid in 23%, yellow in 55% and serosanguinous in 22%. Importantly, the appearance of chylothorax has not been linked to any specific etiology with the exception of hemorrhagic fluid, most commonly encountered in postsurgery settings [24••]. Contrary to the reasonable anticipation that chylothoraces of serous appearance would be mainly related to diseases causing transudative effusions, nonmilky chylous pleural effusions have been reported in patients with a variety of diagnoses [24••,49••].

Conversely, a milky pleural effusion does not guarantee the diagnosis of chylothorax as it can also be found with pseudochylothorax and empyema. Milky appearance in lipid pleural effusions, namely chylothorax and pseudochylothorax, is produced by high lipid levels, either chylomicrons or cholesterol and lecithin–globulin complexes, respectively. In empyema, milkiness is due to the suspended leukocytes, bacteria and cellular debris. Milky

Table 1 Etiopathogenetic classification of chylothorax

TRAUMA	MALIGNANCY	MISCELLANEOUS		IDIOPATHIC
A Iatrogenic (direct injury)	**Infiltration and/or lymphatic obstruction**	**A Increased lymph volume +/or abnormal lymphatic lumen**	**C Increased venous pressure**	**Unknown mechanism**
Surgical	lymphoma		Congestive heart failure	Exclude lymphoma
esophagogastrectomy	metastatic carcinoma	**Lymphatic disorders-congenital**	Cardiomyopathy	
CHD repair	lung cancer	Gorham disease	Constrictive pericarditis	
lung resection	Kaposi's sarcoma	Milroy disease	Mediastinal fibrosis	
mediastinal mass resection	chronic lymphocytic	Noonan syndrome	SVC/subclavian vein thrombosis	
mediastinoscopy		Down syndrome		
lymphadenectomy	leukemia		**D Chylous ascites (transdiaphragmatic chyle movement)**	
cardiovascular surgery		Turner's syndrome	cirrhosis (alcoholic/cryptogenic)	
pericardiectomy		lymphatic hypoplasia	hepatitis C	
heart/lung transplant		lymphangiectasia	non-alcoholic steatohepatitis	
spine surgery		tuberous sclerosis	primary billiary cirrhosis	
laparoscopic gastric banding		tracheoesophageal fistula	primary sclerosing cholangiitis	
endoscopic sympathectomy			cholangiocarcinoma	
		-acquired	pancreatic carcinoma	
Non-surgical		yellow nail syndrome	pancreatitis	
CVC placement		lymphangioma	biopsy of peritoneal lymph nodes	
pacemaker implantation		lymphangiomatosis	gastrectomy	
PA embolization		lymphangiectasis	hypothyroidism	
thracic irradiation		lymphangioleiomyomatosis		
tube thoracostomy				
		B Lymphatic obstruction -intraluminal obstruction		
B Non-iatrogenic (direct injury or straining)				
		thoracic duct thrombosis		
blunt thoracic trauma		lymphangiitis		
penetrating thoracic trauma		thoracic duct cyst		
weight lifting		malignancy		
straining				
coughing		**-external pressure**		
vomiting		pulmonary tuberculosis		
yawning		sarcoidosis		
seat belt injury		Castlemann's disease		
childbirth		Waldenstrom		
		macroglobulinemia		
		retrosternal goiter		
		Extramedullary hematopoiesis		
		amyloidosis		
		mediastinal fibrosis		

Figure 1 The diagnostic approach of chylothorax consists of three steps: (i) clinical suspicion of chylothorax (ii) verification of chylothorax by pleural fluid analysis and (iii) determination of chylothorax etiology and investigation for additional pleural fluid formation causes



*Malignancy, superior vena cava or subclavian vein thrombosis, liver cirrhosis or lymphatic disorder. LDH, lactate dehydrogenase; TG, triglycerides.

effusions should undergo centrifugation and if the supernatant becomes clear, empyema is the most probable diagnosis; otherwise, the differential diagnosis is between lipid pleural effusions and pleural fluid analysis is required [1].

Pleural fluid characteristics

Typical chylothoraces are considered to be lymphocytic exudates [1,2,38]. Agrawal *et al.* [49••] proposed that chylous pleural effusions are not only exudates with lymphocytes more than 50% but also present low lactate dehydrogenase (LDH) levels (<2/3 upper normal serum value), a feature that reflects the absence of pleural inflammation. Unfortunately, a notable number of chylothoraces are not typical.

Chylothorax does not always meet the exudative criteria. In a review of 74 chylous pleural effusions, Maldonado *et al.* [24••] reported 10 (14%) transudative cases using criteria proposed by Heffner *et al.* [50]. Cirrhosis accounted for 40% of these effusions; surgical procedures, lymphoproliferative disorders, pancreatic cancer, radiation injury and idiopathic were the etiologies in the remainder. It was assumed that in cirrhosis the coexistence of transudative ascites occurring secondary to portal hypertension and chylous ascites may result in the low protein and LDH levels in the fluid that moves from the peritoneal to the pleural cavity [24••]. Another study on 22 patients with chylothorax by Agrawal *et al.* [49••], identified seven (32%) transudative cases; heart failure, cirrhosis or constrictive pericarditis were the underlying abnormalities in these patients. A few years earlier, Diaz-Guzman *et al.* [18] had reviewed the English literature on transudative chylous pleural effusions and found 15 such cases. Importantly, in these patients chylothorax was attributed to diseases typically associated with pleural transudates, that is, cirrhosis, nephrosis or heart failure.

Maldonado *et al.* [24••] also demonstrated that lymphocyte-predominance is not always present in chylothorax. Out of 30 chylous pleural effusions with total and differential cell counts available, six (20%, 5 exudates and 1 transudate) were neutrophilic. All neutrophilic chylothoraces were postsurgical, whereas only two (25%)

of the eight postsurgical chylothoraces were lymphocytic. Intra-operative pleural trauma or infection is probably the cause of pleural fluid neutrophilia in these cases.

Chylothorax verification

Once chylothorax is suspected, one has to prove that the aspirated pleural fluid contains chyle to establish the diagnosis. This is achieved by pleural fluid analysis. Indirect methods such as detection of ingested dyed fats in the pleural space or localization of chyle leak point have also been described [1].

Triglyceride measurement

The easiest way to establish chylothorax diagnosis is to demonstrate pleural fluid triglyceride levels more than 110 mg/dl in pleural fluid [1]. The institution of this criterion was based on a study published 30 years ago by Staats *et al.* [3]. According to that study which examined the fluid features of 38 chylous and 103 non-chylous pleural effusions, the possibility of chylothorax in patients with pleural fluid triglyceride levels more than 110 mg/dl and less than 50 mg/dl was more than 99% and 5% or less, respectively; for the effusions with triglyceride values 50–110 mg/dl, lipoprotein analysis was suggested [3]. Along the same lines, Maldonado *et al.* [24••] reported that 10 (14%) of 74 chylous pleural effusions had triglyceride levels less than 110 mg/dl, including two (3%) presenting with triglycerides less than 50 mg/dl; eight (80%) of the above cases were observed in fasting or malnourished patients [24••]. Another case with pleural fluid triglyceride levels less than 110 mg/dl was also reported by Buttiker *et al.* [14] in a study of 39 chylothoraces in children. Simultaneous cholesterol measurement is suggested to examine the possibility of pseudochylothorax which can also be characterized by relatively high triglyceride levels. However, in pseudochylothorax pleural fluid cholesterol levels are usually more than 200 mg/dl, they are higher than pleural fluid triglyceride levels and the fluid to serum cholesterol ratio is more than one. In addition, the presence of cholesterol crystals is considered to be diagnostic of pseudochylothorax [1,51].

Lipoprotein analysis

The gold standard in chylothorax diagnosis is the demonstration of chylomicrons in pleural fluid by lipoprotein analysis. This finding is specific for chylothorax because normally chylomicrons should not be present in any body fluid but chyle [1,5]. It should, however, be noticed that no data exist about pleural effusions in patients with hyperlipoproteinemias who present high serum chylomicron levels. Although it seems reasonable to expect some false positive results in these patients due to extravasation of chylomicrons into the pleural space, the large size of chylomicrons makes it rather unlikely for them to diffuse through the vascular endothelium.

Lipophilic dye ingestion

The ingestion of a fatty meal with a lipophilic dye followed by a thoracentesis 30–60 min later to check for pleural fluid color change is an alternative method for the diagnosis of chylothorax [1,52]. However, this method is not practical and its diagnostic accuracy has not been validated.

Chyle leak localization

Upon strong suspicion of chylothorax, one could straightway localize the point of chyle leak to establish the diagnosis. Lymphangiography and lymphoscintigraphy, during which a radiographic contrast material or ^{99m}Tc-human albumin, respectively, is injected into the dorsum of the foot or hand followed by thoracic-duct imaging, are the main techniques for this purpose. However, thoracic-duct imaging approximately 80 min after the oral ingestion of ^{123}I-labeled iodophenyl-methyl-pentadecanoic acid may be preferred over the aforementioned methods due to the avoidance of difficult injections. Considering patient's pain and radiation exposure, we suggest chyle leak localization in chylothorax not to be included in the diagnostic approach unless thoracic-duct ligation is necessary [1,53–56].

Cause determination

In traumatic chylothorax, the cause is usually evident by the patient's history and clinical presentation. If a history of chest/neck injury does not exist, the exclusion of malignancy, especially lymphoma, is crucial. For this purpose, chest and upper abdomen CT scan is suggested to search for enlarged lymph nodes, signs of tumor or other lung parenchymal abnormalities (i.e lymphangioleiomyomatosis) [1,2,38]. Use of intravenous contrast would reveal thrombosis of thorax or neck central veins, if present. Bipedal lymphangiography is useful to unveil thoracic or diffuse lymphatic abnormalities. CT studies right after lymphangiography are expected to better depict their association with adjacent tissues [1,2]. The use of PET scanning in nontraumatic cases for identification of malignant tissue could be a reasonable choice, as malignancy represents the second more frequent cause of the disease; however, its role in the diagnostic approach for chylothorax has not been studied yet.

Once malignancy and pulmonary parenchymal diseases are excluded, the clinician needs to consider whether other causes of pleural effusion coexist. This may be the case if pleural fluid features are not compatible with a lymphocyte-predominant exudate [49••]. A transudative nature of the fluid is suggestive of the presence of heart failure, cirrhosis or nephrotic syndrome, whereas neutrophilic predominance is expected in posttraumatic cases and may also raise the possibility of a co-existing acute inflammatory disease such as infection or pulmonary emboli. A lymphocytic chylothorax with inappropriately

high LDH levels may be suggestive of infiltration of the pleura by tumor. The importance of the recognition of a coexisting disease underlies the need for these abnormalities to be corrected to permit accurate assessment of the rate of chyle flow which in turn, affects decisions on whether thoracic duct ligation is required to fix the leak [49••].

Conclusion

The typical feature of chylothorax, which is supposed to be a milky lymphocytic exudate with low LDH levels, is not the rule after all. Less than half of chylous pleural effusions have the distinctive milky appearance while up to 14% can be transudates. Even in the presence of milky fluid, the differential diagnosis can only be limited between lipid pleural effusions and empyema. The assertion that chylothorax should always be a lymphocyte-predominant effusion is also false; up to 20% of them can be neutrophilic, especially the postsurgical ones. Hence, chylothorax should be suspected not only in patients with milky pleural effusions but also in those with pleural effusion and chest/neck trauma or medical conditions that may cause chylothorax. As chylothorax may be due to a wide variety of conditions and the absence of pleural fluid milkiness does not rule out a chylous origin of the effusion, it could be postulated that the possibility of chylothorax should be examined in any patient with persistent effusion of undetermined cause. However, to the best of the authors' knowledge this issue has not been appropriately investigated so far.

Pleural fluid triglyceride measurement remains the recommended diagnostic method in chylothorax, values more than 110 mg/dl or less than 50 mg/dl virtually establishing or excluding the diagnosis, respectively; ambiguous cases with triglycerides 50–110 mg/dl require lipoprotein analysis for the demonstration of chylomicrons. In fasting or malnourished patients whose chylothorax may present low triglycerides, lipoprotein analysis is suggested even with values less than 50 mg/dl. Once chylous pleural effusion is diagnosed, atypical fluid characteristics should alert the clinician for the presence of additional pleural effusion causes.

Acknowledgements

There are no conflicts of interests.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 412–413).

1 Light RW. Chylothorax and pseudochylothorax. In: Light RW, editor. Pleural diseases, 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2007 pp. 346–361.

2 Hillerdal G. Effusions from lymphatic disruptions. In: Light RW, Lee YCG, editors. Textbook of pleural diseases, 2nd ed. London: Hodder Arnold; 2008. pp. 389–396.

3 Staats BA, Ellefson RD, Budahn LL, *et al.* The lipoprotein profile of chylous and nonchylous pleural effusions. Mayo Clin Proc 1980; 55:700–704.

4 Fauci AS. Disorders of lipoprotein metabolism. In: Fauci AS, Kasper DL, Longo DL, Braunwald E, Hauser SL, Jameson JL, Loscalzo J, editors. Harrison's principles of internal medicine, 17th ed. New York: McGraw-Hill Medical; 2008. pp. 2416–2428.

5 Pocock G, Richards CD. The digestive system. In: Pocock G, Richards CD, editors. Human physiology: the basis of medicine, 3rd ed. Oxford, New York: Oxford University Press; 2006. pp. 378–415.

6 Machado JD, Suen VM, Marchini JS. Is oral nutritional therapy effective for the treatment of chylothorax? A case report. Nutrition 2008; 24:607–609.

7 Panthongviriyakul C, Bines JE. Postoperative chylothorax in children: an evidence-based management algorithm. J Paediatr Child Health 2008; 44:716–721.

8 Bessone LN, Ferguson TB, Burford TH. Chylothorax. Ann Thorac Surg 1971; 12:527–550.

9 Platis IE, Nwogu CE. Chylothorax. Thorac Surg Clin 2006; 16:209–214.

10 Nair SK, Petko M, Hayward MP. Aetiology and management of chylothorax in adults. Eur J Cardiothorac Surg 2007; 32:362–369.

11 Fremont RD, Milstone AP, Light RW, Ninan M. Chylothoraces after lung transplantation for lymphangioleiomyomatosis: review of the literature and utilization of a pleurovenous shunt. J Heart Lung Transplant 2007; 26:953–955.

12 Ryu JH, Doerr CH, Fisher SD, *et al.* Chylothorax in lymphangioleiomyomatosis. Chest 2003; 123:623–627.

13 Kumasaka T, Seyama K, Mitani K, *et al.* Lymphangiogenesis-mediated shedding of LAM cell clusters as a mechanism for dissemination in lymphangioleiomyomatosis. Am J Surg Pathol 2005; 29:1356–1366.

14 Buttiker V, Fanconi S, Burger R. Chylothorax in children: guidelines for diagnosis and management. Chest 1999; 116:682–687.

15 Doerr CH, Allen MS, Nichols FC 3rd, Ryu JH. Etiology of chylothorax in 203 patients. Mayo Clin Proc 2005; 80:867–870.

16 Busquets JM, Rullan PJ, Trinidad-Pinedo J. Bilateral chylothorax after neck dissection. Otolaryngol Head Neck Surg 2004; 130:492–495.

17 Lin SH, Lin YF, Shih YL. An unusual complication of nephrotic syndrome: chylothorax treated with hemodialysis. Nephron 2001; 87:188–189.

18 Diaz-Guzman E, Culver DA, Stoller JK. Transudative chylothorax: report of two cases and review of the literature. Lung 2005; 183:169–175.

19 Chang PY, Chao TY, Dai MS. Early occurrence of chylothorax related to thoracic irradiation and concomitant chemotherapy. Clin Oncol (R Coll Radiol) 2005; 17:291.

20 Hillerdal G. Chylothorax and pseudochylothorax. Eur Respir J 1997; 10:1157–1162.

21 Amasyali B, Heper G, Akkoc O, *et al.* Chylous ascites and pleural effusion secondary to constrictive pericarditis presenting with signs of lymphatic obstruction. Jpn Heart J 2004; 45:535–540.

22 Diamond JM, Kotloff RM, Liu CF, Cooper JM. Severe venous and lymphatic obstruction after single-chamber pacemaker implantation in a patient with chest radiation therapy. Pacing Clin Electrophysiol (PACE) 2009 [Epub ahead of print].

23 Suri D, Gupta N, Morigeri C, *et al.* Chylopericardial tamponade secondary to superior vena cava thrombosis in a child with nephrotic syndrome. Pediatr Nephrol 2009; 24:1243–1245.

24 •• Maldonado F, Hawkins FJ, Daniels CE, *et al.* Pleural fluid characteristics of chylothorax. Mayo Clin Proc 2009; 84:129–133.
This is the largest study on pleural fluid characteristics of chylothorax patients in the literature. It was demonstrated not only that chylous pleural effusions can be neutrophilic, especially the postsurgical ones, but also that up to 14% of them may be transudates. Finally, it was confirmed that milky pleural fluid is found in less than 50% of chylothoraces, the rest of them being serous or serosanguinous in appearance.

25 • Limsukon A,Yick D, Kamangar N. Chylothorax: a rare complication of tube thoracostomy. J Emerg Med 2008. [Epub ahead of print]
This article points out that although traumatic chylothorax can be infrequently caused by chest tube insertion, it should always be included in the differential diagnosis of pleural effusions that remain despite tube thoracostomy.

26 Thomas R, Christopher DJ, Roy A, *et al.* Chylothorax following innominate vein thrombosis: a rare complication of transvenous pacemaker implantation. Respiration 2007; 74:338–340.

27 Repko BM, Scorza LB, Mahraj RP. Recurrent chylothorax after neck surgery: percutaneous thoracic duct embolization as primary treatment. Otolaryngol Head Neck Surg 2009; 141:426–427.

28 Milonakis M, Chatzis AC, Giannopoulos NM, *et al.* Etiology and management of chylothorax following pediatric heart surgery. J Card Surg 2009; 24:369–373.

29 Abdel-Galil K, Milton R, McCaul J. High output chyle leak after neck surgery: the role of video-assisted thoracoscopic surgery. Br J Oral Maxillofac Surg 2009; 47:478–480.

30 Araujo LF, Moreschi AH, de Macedo GB, *et al.* Chylothorax after surgical treatment of right-sided thoracic outlet syndrome. J Bras Pneumol 2009; 35:388–391.

31 Zerrweck C, Caiazzo R, Arnalsteen L, *et al.* Chylothorax: unusual complication after laparoscopic gastric banding. Obes Surg 2009; 19:667–670.

32 Agrawal V, Doelken P, Sahn SA. Seat belt-induced chylothorax: a cause of idiopathic chylothorax? Chest 2007; 132:690–692.

33 Thorne PS. Traumatic chylothorax. Tubercle 1958; 39:29–34.

34 Cammarata SK, Brush RE Jr, Hyzy RC. Chylothorax after childbirth. Chest 1991; 99:1539–1540.

35 Reilly KM, Tsou E. Bilateral chylothorax. A case report following episodes of stretching. JAMA 1975; 233:536–537.

36 Apostolakis E, Akinosoglou K, Koletsis E, Dougenis D. Traumatic chylothorax following blunt thoracic trauma: two conservatively treated cases. J Card Surg 2009; 24:220–222.

37 Kushwaha RA, Verma SK, Sanjay, *et al.* Bilateral chylothorax in a child after mild trauma. Indian J Chest Dis Allied Sci 2008; 50:355–357.

38 Agrawal V, Sahn SA. Lipid pleural effusions. Am J Med Sci 2008; 335:16–20.

39 Cigarral C, Montero A, Salas C, *et al.* Chylothorax due to metastatic prostate carcinoma: an unusual complication. Clin Transl Oncol 2009; 11:767–769.

40 Iqbal MH, Smith PR, Bande S. Chylothorax due to angioimmunoblastic T-cell lymphoma. Intern Med J 2009; 39:67–68.

41 Barker PM, Esther CR Jr, Fordham LA, *et al.* Primary pulmonary lymphangiectasia in infancy and childhood. Eur Respir J 2004; 24:413–419.

42 Tanaka E, Matsumoto K, Shindo T, Taguchi Y. Implantation of a pleurovenous shunt for massive chylothorax in a patient with yellow nail syndrome. Thorax 2005; 60:254–255.

43 Lin WY, Lin GM, Wu CC. Coexistence of non-communicated chylothorax and chylous ascites in nephrotic syndrome. Nephrology (Carlton) 2009; 14:700.

44 Fernandez FG, Denlinger CE, Patterson GA, *et al.* Massive bilateral chylothoraces complicating mediastinal granulomatous disease. Ann Thorac Surg 2009; 88:1012–1013.

45 Blawat P, Kowalewski J, Dancewicz M, Bella M. Chylothorax due to retrosternal goiter. J Chir (Paris) 2009; 146:324–325.

46 Benjilali L, Harmouche H, Alaoui-Bennesser H, *et al.* Chylothorax and chylopericardium in a young man with Behcet's disease. Joint Bone Spine 2008; 75:743–745.

47 Maayan H, Izbicki G, Heyd J, *et al.* Persistent chylothorax in Behcet's disease treated with a blood patch. Isr Med Assoc J 2008; 10 (8–9):588–589.

48 Karapolat S, Sanli A, Onen A. Chylothorax due to tuberculosis lymphadenopathy: report of a case. Surg Today 2008; 38:938–941.

49 •• Agrawal V, Doelken P, Sahn SA. Pleural fluid analysis in chylous pleural effusion. Chest 2008; 133:1436–1441.
This is the first study to demonstrate that atypical fluid characteristics in chylothorax may be a sign of additional pleural fluid formation causes. Typical pleural fluid in chylothorax was found to be a lymphocytic exudate with low LDH levels due to the absence of pleural inflammation. It was also confirmed that pleural fluid milky appearance is a rather insensitive criterion for chylothorax diagnosis.

50 Heffner JE, Sahn SA, Brown LK. Multilevel likelihood ratios for identifying exudative pleural effusions. Chest 2002; 121:1916–1920.

51 Hamm H, Pfalzer B, Fabel H. Lipoprotein analysis in a chyliform pleural effusion: implications for pathogenesis and diagnosis. Respiration 1991; 58 (5–6):294–300.

52 Klepser RG, Berry JF. The diagnosis and surgical management of chylothorax with the aid of lipophilic dyes. Dis Chest 1954; 25:409–426.

53 Stavngaard T, Mortensen J, Brenoe J, Svendsen LB. Lymphoscintigraphy using technetium-99m human serum albumin in chylothorax. Thorac Cardiovasc Surg 2002; 50:250–252.

54 Restrepo JM, Caride VJ. Lymphoscintigraphy and radionuclide venography in chylothorax. Clin Nucl Med 2004; 29:440–441.

55 Qureshy A, Kubota K, Ono S, *et al.* Thoracic duct scintigraphy by orally administered I-123 BMIPP: normal findings and a case report. Clin Nucl Med 2001; 26:847–855.

56 Ngan H, Fok M, Wong J. The role of lymphography in chylothorax following thoracic surgery. Br J Radiol 1988; 61:1032–1036.