mixed GGO lesions (80%). Moreover, the sensitivity is lower for smaller lesions (35%-67% for those ≤ 10 mm) than larger lesions. False-negative rates were reported to be about 20% to 30%. The rate of concordance between core needle and surgical biopsies in malignant and premalignant lesions was only 73%, and core biopsy failed to identify the area of invasion.

We recommend that persistent GGO lesions ≥ 10 mm be surgically resected for histologic examination and treatment, based on the high probability (42.9%) of growth during follow-up in our study; although there remains the possibility of overdiagnosis in a portion of the patients who undergo surgery. For pure GGO lesions measuring <10 mm, a strategy of long-term follow-up and selective surgery for growing lesions seems reasonable considering the low probability of growth. However, we agree that CT-scanned guided biopsy could be considered in pure GGOs of ≥ 10 mm for which surgery is contraindicated or in patients who refuse surgery.

To the Editor:

I read with interest the article by Nadolski and Itkin in CHEST (January 2013) on thoracic duct embolization (TDE) for non-traumatic chylothorax effusion. The authors are to be applauded for approaching a difficult topic with poorly established standards and a high mortality rate. This is an important condition for chest physicians to understand and manage. Unfortunately, and despite the authors’ vast experience in the management of > 160 patients with this frequently devastating disorder, the etiology of spontaneous or idiopathic chylothorax remains unexplained. What are the mechanisms for the thoracic duct undergoing obstruction in patients with no previous disease and normal MRI thoracic examinations? Up to 20% of patients with nontraumatic chylosis effusions present with lymphatic anomalies (lymphatic malformations, generalized lymphatic disease [lymphangiomatosis], or Gorham-Stout disease). Children with massive lymphatic malformations in the neck, axilla, or groin do not develop lymphatic duct obstruction with lymphedema. We do not have information regarding compared outcomes of TDE in patients with or without associated lymphatic malformations.

It is evident that TDE will soon definitively replace surgical thoracic duct ligation (TDL) because morbidity is significantly reduced by using this technique. To better understand the benefits of this procedure, we need information about the routes of chyle recanalization after thoracic duct occlusion. Apparently, post-operative recurrent chylothorax or proximal chylosis effusions are significantly reduced in patients undergoing TDE compared with those treated with TDL. This complication is common in patients with generalized lymphatic anomalies undergoing TDL. Are lymphatic-venous communications more easily opened and better functioning after TDE?

Nontraumatic chylothorax in children aged < 10 years remains an unsolved problem. Because successful TDE has been reported in the treatment of post-cardiac surgery chylothorax in small children, there are optimistic expectations of TDE improving the mortality rate in the pediatric population affected by pulmonary or disseminated lymphangiomatosis. We encourage pediatric interventional radiologists and thoracic surgeons involved in the management of children with chylothorax in the context of life-threatening lymphatic anomalies to develop and promote noninvasive techniques in the management of these patients.

Thoracic Duct Embolization, Unexplained Thoracic Duct Occlusions, and Lymphatic Anomalies

To the Editor:

I read with interest the article by Nadolski and Itkin in CHEST (January 2013) on thoracic duct embolization (TDE) for non-traumatic chylothorax effusion. The authors are to be applauded for approaching a difficult topic with poorly established standards and a high mortality rate. This is an important condition for chest physicians to understand and manage. Unfortunately, and despite the authors’ vast experience in the management of > 160 patients with this frequently devastating disorder, the etiology of spontaneous or idiopathic chylothorax remains unexplained. What are the mechanisms for the thoracic duct undergoing obstruction in patients with no previous disease and normal MRI thoracic examinations? Up to 20% of patients with nontraumatic chylosis effusions present with lymphatic anomalies (lymphatic malformations, generalized lymphatic disease [lymphangiomatosis], or Gorham-Stout disease). Children with massive lymphatic malformations in the neck, axilla, or groin do not develop lymphatic duct obstruction with lymphedema. We do not have information regarding compared outcomes of TDE in patients with or without associated lymphatic malformations.

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Boksoon Chang, MD
Sang-Won Um, MD, PhD
Seoul, South Korea

Affiliations: From the Division of Pulmonary and Critical Care Medicine, Department of Medicine, Sungkyunkwan University School of Medicine.

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Correspondence to: Sang-Won Um, MD, PhD, Division of Pulmonary and Critical Care Medicine, Department of Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, 50 Irwon-Dong, Kangnam-Gu, Seoul 135-710, South Korea. E-mail: umsnw@skku.edu

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Correspondence to:

Juan Carlos López-Gutiérrez, MD, PhD
Madrid, Spain

Affiliations: From the Division of Surgery, Vascular Anomalies Center, La Paz Children’s Hospital.

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Correspondence to: Juan Carlos López-Gutiérrez, MD, PhD, Department of Surgery, Vascular Anomalies Center, La Paz Children’s Hospital, Puente de la Castellana 261, 28046 Madrid, Spain; e-mail: queminfantil.hulp@salud.madrid.org

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REFERENCES


Response

To the Editor:

We thank Dr López-Gutiérrez for his interest in our recent work in CHEST. The etiology of idiopathic chylothorax has indeed been hardly investigated and in most parts remains unsolved. We suggest that all cases of idiopathic chylothorax can be divided into two major categories: (1) occlusion of the upper part of the thoracic duct (TD) with development of compensatory collaterals and (2) chylosus leak in the presence of a lymphatic malformation.

We believe that the cause of TD occlusion is subclinical trauma. In these cases, multiple lymphatic collaterals develop as a new route to the venous system. If one of these collaterals abut a serious surface (pleural, pericardial, or peritoneal), it can rupture and then result in a chylous leak. Traditional lymphangiogram can easily diagnose TD occlusion.

MRI ductography, which uses noncontrast fluid-weighted MRI sequences, allows visualization of not only the TD but also other abnormal lymphatic structures such as congenital lymphatic malformations. We hypothesize that an unidentified insult can result in a rupture of these structures and leakage of the chyli. If a leak from these structures happens proximal to the cisterna chyli, TD embolization (TDE) can be devastating, diverting all the flow from the TD into the leak.

Over the years, we have developed an algorithm to diagnose and treat idiopathic chylous effusions. We first perform magnetic resonance ductogram to identify TD and other lymphatic structures/malformations. Then, we perform conventional or intranodal lymphangiography. In cases of occlusion of the upper portion of the TD or demonstration of the leak, TDE usually cures chylothorax. In cases where TD is patent and the flow is normal, TDE should be avoided. Occasionally, oily-based contrast introduced during lymphangiogram can facilitate closure of the leak.

More distal occlusion of the TD leaves a longer segment of the TD intact, creating more opportunities for development of new lymphovenous anastomosis. The advantage of the TDE vs thoracic duct ligation is the ability to visualize the leak and occlude TD close to the leakage point (comparing to proximal supradiaphragmatic ligation of the TD in thoracic duct ligation). This potentially can result in development of more lymphovenous communications after TDE and reduction of the chylothorax recurrence.

Finally, future development of new imaging lymphatic agents may provide us with deeper insight into anatomy, physiology, and flow dynamics in the lymphatic system. Hopefully, these new insights will trigger further research and understanding of this vital yet overlooked “human plumbing” system.

Maxim Itkin, MD
Gregory J. Nadolski, MD
Philadelphia, PA

Affiliations: From the Department of Radiology, Section Interventional Radiology, Hospital of the University of Pennsylvania.

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Correspondence to: Maxim Itkin, MD, Department of Radiology, Section Interventional Radiology, Hospital of the University of Pennsylvania, 1 Silverstein, 3400 Spruce St, Philadelphia, PA 19104; e-mail: itkinmax@gmail.com

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Arrhythmias in COPD

Consider P-Wave Dispersion and Pulmonary Hypertension, Too

To the Editor:

In a recent issue of CHEST (August 2012), Wilchesky et al. noted an increase in cardiac arrhythmias in patients with COPD and concluded that these were due to long-acting bronchodilator therapy after adjusting for known confounding factors. However, two other potential contributory factors should be considered before attributing the observations to bronchodilators alone.

First, secondary pulmonary hypertension attributable to COPD or other lung disease can affect right atrial function without causing gross enlargement in the early stages leading to secondary atrial dysfunction and consequent atrial arrhythmias. Indeed, recent data suggest that pulmonary hypertension alone in COPD is associated with increased COPD exacerbations, and one wonders whether this is partly explained by an increase in cardiac arrhythmias. Was secondary pulmonary hypertension (without necessarily right atrial or ventricular enlargement) included in the comorbidity criteria of congestive heart failure and cardiac enlargement and, hence, adjusted for in the analysis?

Second, P-wave dispersion (reflecting heterogeneous atrial depolarization) has been reported to predispose to the development of atrial arrhythmias (especially atrial fibrillation) in COPD (as well as in other scenarios, eg, hypertension or postoperatively after coronary artery bypass graft) independent of lung function, blood gas and electrolyte levels, and atrial function. Was this variable measured and accounted for in the study?

Andrew R. L. Medford, MBChB, MD, FCCP
Bristol, England