
Response

To the Editor:

We thank Dr López-Gutiérrez for his interest in our recent work in CHEST.1 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) chylous 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 serous surface (pleural, pericardial, or peritoneal), it can rupture and then result in a chylous leak.2 Traditional lymphangiogram can easily diagnose TD occlusion. MRI ductography,3 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 ductography to identify TD and other lymphatic structures/malformations. Then, we perform conventional or intranodal lymphangiography.4 In cases of occlusion of the upper portion of the TD or demonstration of the leak, TDE usually cures chylous thorax. 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.5

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 chylorhexis 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.

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References


Arrhythmias in COPD

Consider P-Wave Dispersion and Pulmonary Hypertension, Too

To the Editor:

In a recent issue of CHEST (August 2012), Wilchesky et al1 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.2 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.3 Was this variable measured and accounted for in the study?

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