
Chylothorax in Hematologic Malignancies

To the Editor:

I read with interest the excellent article by Alexandrakis et al., in which current knowledge regarding the hematologic entities associated with pleural disease is extensively reviewed. The authors mention that the pleural fluid may be chyloous (chylothorax) in some disorders such as non-Hodgkin lymphoma, Castleman disease, and as a late complication of thoracic irradiation. However, a reference to chylothorax in other associated disorders such as chronic lymphocytic leukemia (CLL) and Waldenström macroglobulinemia is absent. I would like to make some comments about these specific subjects.

Chylothorax, a milky white fluid from a pleural space, usually results from disruption of the thoracic duct or its tributaries. This fluid contains a high level of triglyceride (>110 mg/dL), an essential feature for its diagnosis. The presence of chyomicrons is also indicative of chylothorax. More than 50% of chylothorax is due to malignancy, and lymphoma accounts for 75%, followed by lung carcinoma.2,3

CLL of the B-cell type is the most common leukemia affecting adults, and may infiltrate any organ. Parenchymal infiltrates and pleural effusion are frequent manifestations in the lung, with chylothorax being less usual (Table 1). The rarity of chylothorax in CLl has been attributed to the very uncommon mediastinal lymphadenopathy although the biological features of some tumor may contribute to their appearance.5

Waldenström macroglobulinemia is a rare lymphoproliferative disorder (up to 2% of hematologic malignancies) affecting mostly elderly people, and the lung could be involved in up to 5%.4 A chyloous effusion is an infrequent and usually late complication. Few cases of initial or evolutive chylothorax associated with Waldenström disease appear in MEDLINE (Table 1).

Management of chylothorax includes therapy of the underlying disease associated with other conservative measures, such as drainage of pleural effusion, maintenance of nutritional condition, and chemical pleurodesis. Surgical therapy is proposed when conservative treatments have failed.3 Since the rarity of these conditions demand a higher index of suspicion, their inclusion in reviews could help to direct diagnostic work.

Enrique Antón, MD, PhD
Hospital of Zumárraga
Guipúzcoa, Spain

Table 1—Reported Cases of Chylothorax Associated With CLL and Waldenström Macroglobulinemia (MEDLINE)

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<thead>
<tr>
<th>Chylothorax and CLL</th>
<th>Waldenström Macroglobulinemia</th>
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<tr>
<td>Ampil et al1993</td>
<td>Rizzo and Campagnoli1984</td>
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<td>Zimhony et al1994</td>
<td>Marti et al1987</td>
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<td>Fernandez-Escribano et al1997</td>
<td>Monteagudo et al1987</td>
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<td>Aranda and Aguinaico2001</td>
<td>Antón2001</td>
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To the Editor:

We thank Dr. Antón for his interest in our review. He is right that pleural disease in patients with hematologic malignancies should also include chylothorax in patients with chronic lymphocytic leukemia and Waldenström macroglobulinemia. However, one additional case of chylothorax in a patient with Waldenström macroglobulinemia also should be included in Table 1 of the letter from Dr. Antón. A case of primary macroglobulinemia associated with pseudochylothorax in a Japanese patient also has been reported.2 Pseudochylothorax is a chyliform fluid in the pleural space.3 Chyomicrons are absent, and this effusion has nothing to do with lymphatic vessels or chyle. Most pseudochylothoraces have cholesterol levels of >250 mg/dL and triglyceride levels of <110 mg/dL. The addition of 1 to 2 mL ethyl ether

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clears the opalescence caused by high cholesterol levels. These cases further expand the array of pleural manifestations in hematologic malignancies.

Demosthenes Bouros, MD, FCCP
University Hospital of Alexandroupolis
Alexandroupolis, Greece

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CO₂ Retention in Acute Severe Asthma

To the Editor:

I applaud the authors for writing a comprehensive review on asthma (March 2004). However, I believe that further clarity needs to be given to their section on gas exchange (page 1083). The first paragraph states that the occurrence of hypercapnia is “a result of muscle fatigue and inability to maintain adequate alveolar ventilation.” While muscle fatigue is undeniably a major cause of hypercapnia in acute severe asthma, it has to be pointed out that there are other factors that cause CO₂ retention in these patients. I will limit this letter to the processes that create increase in dead space.

In addition to the low ventilation/perfusion (V˙/Q˙) areas (which cause hypoxia), uneven distribution of ventilation gives rise to regions of alveolar hyperinflation and high V˙/Q˙ ratios, which cause increase in pulmonary dead space and CO₂ retention. Alterations in regional perfusion from the increased intra-alveolar pressure and hypoxic pulmonary vasoconstriction are believed to contribute to this “dead space” effect. I had two patients with severe asthma in my ICU last year who had persistently high PaCO₂ despite being intubated and receiving minute ventilation of > 10 L. In one of these patients, I even went to the extent of ordering a CT angiogram to eliminate the possibility of pulmonary embolism (which showed only hyperinflation). Needless to say, the PaCO₂ came down to normal levels after the bronchosuspens was corrected.

Vijo Poulose, MD, FCCP
Singapore

References

Negative Pressure Pulmonary Edema as an Unsuspected Imitator of Acute Lung Injury/ARDS

To the Editor:

Further to the recent review article by Schwarz and Albert (April 2004), we would like to add negative pressure pulmonary edema (NPPE) as a further important imitator. NPPE may rarely fall into the category of diffuse alveolar hemorrhage (DAH) through damage of the pulmonary capillaries by mechanical disruption of the alveolar-capillary membrane, resulting in diffuse alveolar injury. More usually, pulmonary edema is manifest, with fulfillment of the clinical, physiologic, and radiographic criteria for acute lung injury (ALI)/ARDS. Several hypotheses have been postulated to explain the pathophysiology sequelae of NPPE. 1–5 While early recognition and specific treatment of an underlying condition 6–9 may rule in NPPE or rule out other causes of DAH, an obvious underlying cause may be absent particularly after emergence from anesthesia or unnoticed upper airway obstruction. Indeed, symptoms of NPPE may be considerably delayed after extubation. 9 Although symptoms usually resolve rapidly with restoration and/or maintenance of a patent airway and supplemental oxygen, positive end-expiratory pressure and mechanical ventilation may be required for a prolonged period of time. 10 Failure to consider NPPE in the differential diagnosis of acute clinical, physiologic, and radiographic changes that fit the criteria for ALI/ARDS may lead to unnecessary and potentially deleterious iatrogenic complications.

Gareth L. Ackland, PhD
Michael G. Mythen, MD
University College London
London, UK

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