Review on Bronchopleural Fistula

Did a Surgeon Review It?

To the Editor:

We read with interest the recently published review by Lois and Noppen in CHEST (December 2005) on bronchopleural fistula (BPF) and found it confusing. Even though the article is focused mainly on the endoscopic treatment of BPF, the authors widely discussed all of the clinical aspects of the problem.

In the proposed classification, the subgroup “nonpostoperative” does not contain BPF patients but only those with pneumothorax. BPF and pneumothorax do not share etiology, clinical presentation, diagnosis, prognosis, or treatment. They are simply different things.

The main clinical signs of BPF are not precisely outlined in the article. When the fistula is small (on the order of a few millimeters), the symptoms are cough, particularly when the patient is turned on the side of the fistula, and, as the authors stated, a delay in cavity filling after pneumonectomy. When empyema is present, infectious symptoms are dominant.

Concerning diagnosis, it is universally accepted that the “gold standard” for the diagnosis of BPF is bronchoscopy. The authors mentioned it at the end of the first paragraph in the “Diagnosis” section after methylene blue staining, bronchography, $^{133}$Xe ventilation study, CT scanning, and gas concentration scintigraphy. This message is misleading.

In the “Prognosis” section of the article, the authors mainly discussed the problem of pneumothorax in intubated patients. This is not relevant in a discussion of BPF.

Concerning the treatment of patients with BPF, there are few rules that the authors did not discuss. The vast majority of these patients had undergone pneumonectomy. The first step in treatment is bronchoscopy and drainage of the chest cavity. Afterward, a decision on surgical treatment is made (e.g., Claggett procedure, direct repair, or thoracoscopy, depending on the dimension of the fistula and the time of onset). Moreover, in the “Treatment” section, mechanical ventilation is mentioned. This is not a therapy for patients with BPF.

The paragraphs on the endoscopic treatment of BPF (in the “Bronchoscopy” section of the article) are well-written and exhaustive. Unfortunately, BPF is a surgical problem that can sometimes be successfully treated by endoscopy to avoid performing an aggressive surgical treatment. But, it remains a surgical problem. Did a thoracic surgeon review this review?

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REFERENCES

1 Lois M, Noppen M. Bronchopleural fistulas: an overview of the problem with special focus on endoscopic management. Chest 2005; 128:3955–3965

To the Editor:

We appreciate Dr. Leo’s interest in our article and his comments. We agree that the clinical signs of bronchopleural fistula (BPF) vary pending on the size of the fistula.

Dr. Leo states that the conditions listed in our proposed classification of nonpostoperative etiologies do not cause BPF, but pneumothorax. This is not the case; as discussed in our article, necrotic lung complicating infection, chemotherapy or radiotherapy (for lung cancer), persistent spontaneous pneumothorax, inflammatory diseases, and other conditions have been associated with the appearance of BPF.1–10

We agree that the first step in treatment is bronchoscopy, as is mentioned several times in our article. Bronchoscopic exploration is important as a diagnostic and therapeutic intervention. It allows for the proper evaluation of the stump, attempts to localize the BPF as well as to exclude tuberculosis or other infectious etiologies, and, if possible, allows the introduction of sealants into the fistulous tract.11

Again, the comments of Dr. Leo and colleagues that the section on “Prognosis” is not relevant in a discussion on BPFs is erroneous. In our article, the literature pertaining to the persistence of air leak and mortality was addressed.11

We did not state that mechanical ventilation was a therapy for BPF. The section addresses the issue of how to best ventilate these patients without creating further problems or perpetuating the existence of the BPF

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1 Lois M, Noppen M. Bronchopleural fistulas: an overview of the problem with special focus on endoscopic management. Chest 2005; 125:3955–3965

Gastroesophageal Reflux Disease and Asthma

The Role of Proton Pump Inhibitors

To the Editor:

We read with interest the recent study in CHEST (September 2005) by Littner et al, who demonstrated a significant reduction in the number of exacerbations of asthma and improvement in quality of life using therapy with antireflux medication. Overall, however, there appeared to be little objective evidence of improvement in asthma. We wonder whether the generally negative findings of the study are due to the selection criteria used to define asthmatic patients with gastroesophageal reflux disease (GERD). Both asthma and GERD are common problems, and it is likely that they selected many patients with simple classic asthma and incidental coexisting acid reflux. In fact, reflux diseases related to airways symptoms are not simply defined, as in their study, by the presence of heartburn. Laryngopharyngeal reflux (LPR) is widely recognized as a cause of upper airways symptoms including cough and airway hyperresponsiveness. LPR differs significantly from the symptom complex in a GERD-related heartburn, and we suggest that had a more accurate clinical history of LPR been used to define reflux-related asthma, then the results of the study may have been more positive.

To illustrate the importance of a correct appreciation of reflux disease that is causally linked to asthma, we report the following example. A 31-year-old lifelong nonsmoker presented with worsening control of his asthma. He also gave a history of globus and repeated episodes of loss of voice, which suggested LPR. Heartburn was an occasional symptom. On treatment with omeprazole, 20 mg twice daily for a period of 2 months, the symptoms of LPR settled and his asthma improved. Methacholine challenge showed a provocative concentration of methacholine causing a 20% fall in FEV₁ of 0.3 mg/mL on presentation, improving to 9.6 mg/mL after 2 months of twice-daily therapy with omeprazole. As the patient’s symptoms had settled, the patient discontinued therapy with omeprazole. Relapse occurred that was associated with a fall in the provocative concentration of methacholine causing a 20% fall in FEV₁ to 1.31 mg/mL. Symptoms of LPR such as globus and dysphonia may be more discriminative of “asthma” responding to therapy with proton pump inhibitors.

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