Angiotensin-converting Enzyme and Neurosarcoidosis

To the Editor:

I read the case report by Godwin and Sahni with great interest and found it thought provoking. However, it warrants further comment.

Neurologic features are present in 5 percent of patients with sarcoidosis. However, neurologic dysfunction is frequently the presenting symptom, as illustrated by this patient. Therefore, neurosarcoidosis should not be considered rare, since the published literature reveals hundreds of cases of neurosarcoidosis.

Diagnosis of neurosarcoidosis can present a diagnostic challenge. Nervous system sarcoidosis might be found more often if the cerebrospinal fluid (CSF) were examined routinely in suspicious cases. Pleocytosis, elevated CSF protein concentration, and low CSF glucose level (hypoglycorrhachia)—the latter being characteristic with leptomeningitis—that CSF formula is specific to sarcoid neuropathy.

Cerebrospinal fluid immunoglobulin (Ig) levels were not measured in this patient, although an elevated IgG index is found in 30 percent of patients with neurosarcoidosis due to intrathecal IgG synthesis. Lymphocyte function and angiotensin-converting enzyme in both the serum and the CSF have provided evidence of intrathecal angiotensin-converting enzyme production, thereby pointing to the tremendous usefulness of CSF angiotensin-converting enzyme in diagnosing disease activity.

Oksanen et al not only supported their observations; he went further and emphasized that the presence of angiotensin-converting enzyme in the CSF is very important in the diagnosis and follow-up of neurosarcoidosis. Since high-dose steroid treatment is being contemplated, it must be emphasized that adverse effects of such therapy frequently induce psychiatric changes. Therefore, this iatrogenic effect should not be confused with the primary disease.

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Loculated Hemopneumothorax

To the Editor:

I would like to comment on the article “Loculated Hemopneumothorax of a Major Fissure in Patients with COPD and Associated Pleural Disease,” by M. H. Kollef.

The author describes two patients with presumed loculated hemopneumothorax in the major fissure. I would like to point out that we are dealing with two cases of subpleural infected bullae: (1) Both patients had severe bullous emphysema documented by CT. These subpleural bullae can assume an oblong, elliptical configuration and mimic an intrafissural collection. (2) The infected subpleural bullae were surrounded by pleural plaques. In both cases, the asbestos-related pleural plaques should involve only the parietal pleura and should not lead to pleural adhesions. In both cases, there is no convincing evidence of significant pleural adhesions on the plain films—the costophrenic sulci are not obliterated, the apical pleura is not thickened, and there is no diaphragmatic pleural tenting. (3) The CT scans, on which the diagnosis appears obvious, are not shown—a serious flaw. (4) We, as well as other authors, have pointed out that such infected, fluid-containing bullae clear slowly over 4 to 8 weeks. In the process of clearing, the bullae can obliterate. (5) A conservative approach is mandatory in order not to endanger patients’ lives. (6) It is not surprising that a Medline search does not yield literature on this topic. It would be highly unlikely to observe a loculated hemopneumothorax without a trace of pneumothorax through the rest of the pleural space, particularly without major pleural adhesions. (7) In view of the small amounts of gas and fluid, it is unlikely that the respiratory failure was contingent on these collections. (11) The course of action in these two cases depends on whether or not on interpretation of imaging studies that a radiologist should have been a coauthor on this article.

I agree with the author’s conclusion—a noninvasive approach is of paramount importance in such a situation.

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1 Kollef MH. Loculated hemopneumothorax of a major fissure in patients with COPD and associated pleural disease. Chest 1990; 97:873-76
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To the Editor:

The article by Kollef, “Loculated Hemopneumothorax of a Major Fissure in Patients with COPD and Associated Pleural Disease” (Chest 1990; 97:873-76), reminds us again of the potential danger of percutaneous needle drainage in patients with COPD. It should be brought to the reader's attention that needling of the