Amyloidosis and Pleural Disease

To the Editor:

In the July 1990 issue of Chest, Kavuru et al\(^1\) reported the cases of five patients in whom the diagnosis of pleural amyloidosis was established by Cope needle biopsy during evaluation of pleural effusions of indeterminate cause. In their review of the English-language literature they found five additional cases of pleural involvement with amyloid, only two of which had been diagnosed by percutaneous needle pleural biopsy. We wish to report the cases of three patients (Table 1), two previously communicated,\(^2\) with systemic amyloidosis, in whom there were similar findings in specimens obtained by percutaneous pleural biopsy.

**Case 1:** A 60-year-old man was referred for evaluation of an eight-month history of dyspnea in exertion, orthopnea, pedal edema, chest oppression, and right pleural effusion. Physical examination revealed findings consistent with biventricular congestive heart failure. An electrocardiogram showed low voltage of the QRS complex. Results of both serum and urine protein electrophoresis were normal. Right thoracentesis and a Cope needle biopsy of the pleura were performed. A hemodynamic study showed biventricular congestive failure, and examination of an endomyocardial biopsy specimen revealed deposits of amyloid. Specimens with Congo red showed similar deposits in the pleural biopsy.

**Case 2:** A 65-year-old woman was referred for evaluation of nephrotic syndrome and renal failure, with dyspnea on exertion, generalized edema, and bilateral pleural effusion. She had a 20-year history of seronegative rheumatoid arthritis. A two-dimensional echocardiogram revealed a moderate amount of pericardial fluid. A renal biopsy specimen was positive for amyloid. Rectal biopsy was negative. A Cope needle biopsy specimen of the pleura demonstrated amyloid deposition by Congo red stain.

**Case 3:** A 64-year-old woman was referred for evaluation of a six-week history of dyspnea on exertion, ankle edema, and bilateral pleural effusion. A two-dimensional echocardiogram revealed a mildly dilated noncompliant left ventricle. Results of serum and urine immunoelectrophoresis did not reveal any monoclonal bands. Examination of a bone marrow aspiration sample showed plasmacytosis of 20 percent. The patient was diagnosed as having amyloidosis on the basis of findings in a biopsy specimen from the kidney. Seven months later, right thoracentesis and a Cope needle biopsy of the pleura were performed. Congo red stain showed amyloid deposits and green birefringence with polarization.

Although the actual incidence of amyloidosis among cases of pleural effusion of undetermined cause is not known, we agree with Kavuru et al\(^1\) that amyloid deposition in the pleura in systemic amyloidosis is probably very common. In all, eight of eight patients with systemic amyloidosis and pleural effusions had marked deposition of amyloid in appropriately stained specimens of pleura obtained by closed Cope needle biopsy. However, the diagnostic role of pleural biopsy in the two studies was different. In three of their five patients percutaneous pleural biopsy was the initial diagnostic procedure to establish the presence of systemic amyloidosis, while we used special stains for amyloid only in cases in which amyloidosis had been previously diagnosed on the basis of biopsy findings in other organs.

In view of the frequent association between congestive heart failure or nephrotic syndrome and biopsy-proved pleural amyloidosis, no one can conclude with certainty that the pleural effusions originated from the pleural amyloid deposits. Kavuru et al\(^1\) stated that the amyloid deposited in the pleura is likely an incidental finding and probably does not play a role in the pathogenesis of the pleural effusions. However, seven of 13 patients so far reported had effusions with characteristics of exudate. The possibility that previous diuretic treatment may have changed the characteristics of the exudate or the limitations of the present series for separation of transudates and exudates may be involved.\(^3\) The incidental nature of pleural deposits of amyloid can be demonstrated only when such deposits are found in patients without pleural effusions. As far as we know, this has not yet been described.

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To the Editor:

We read with great interest the letter from Dr. Romero Candeira and his co-workers. They report the cases of three patients with pleural effusion and amyloidosis who were incidentally found to have pleural amyloid involvement. Even though closed pleural biopsy was not the sole diagnostic test in any of their cases, their report supports our concept that closed pleural biopsy should be the initial diagnostic procedure of choice in patients with pleural effusion when amyloidosis is suspected.

We have encountered yet another patient, a 73-year-old woman with multiple myeloma, who presented with pleural effusion that was considered idiopathic on the basis of conventional studies but in whom a closed pleural biopsy specimen was diagnostic of amyloidosis. Pathogenesis of pleural effusion in patients with amyloidosis is most likely multifactorial. However, in patients with

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Table 1—Clinical Features of Three Patients with Amyloidosis and Pleural Disease

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<thead>
<tr>
<th>Case</th>
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<tbody>
<tr>
<td>Age, yr/Sex</td>
<td>60/M</td>
<td>65/F</td>
<td>64/F</td>
</tr>
<tr>
<td>Effusion</td>
<td>Right</td>
<td>Bilateral</td>
<td>Bilateral</td>
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<tr>
<td>Protein ratio*</td>
<td>0.46</td>
<td>0.56</td>
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<tr>
<td>Lactate dehydrogenase ratio*</td>
<td>1.1</td>
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<tr>
<td>Lactate dehydrogenase, IU/L</td>
<td>149</td>
<td>255</td>
<td>72</td>
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<tr>
<td>Nature of effusion</td>
<td>Exudate</td>
<td>Exudate</td>
<td>Transudate</td>
</tr>
<tr>
<td>Other biopsies</td>
<td>Myocardial</td>
<td>Renal</td>
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<tr>
<td>Cause of amyloid</td>
<td>Primary</td>
<td>Rheumatoid</td>
<td>Primary with arthritis</td>
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<tr>
<td>Plasma cell dyscrasia</td>
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*Ratio of pleural fluid concentration to serum concentrations.
amyloidosis who otherwise do not have a clear cause for pleural effusion (eg, heart failure or nephrotic syndrome), amyloid itself may be implicated if demonstrated in pleural effusion.

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Pleuropulmonary Amebiasis

To the Editor:

Amebic liver abscess caused by Entamoeba histolytica can present with primary pulmonary symptoms.1 This disease process may be easily confused with more common pulmonary disorders and is almost always associated with a right-sided lesion seen on chest radiograph. The correct diagnosis is suggested by a history of potential exposure along with appropriate abdominal findings. We report the unusual case of a previously healthy woman who presented with hemoptysis associated with a left-sided radiographic abnormality who was ultimately found to have amebiasis.

A 24-year-old white woman from the Central Valley of California, was admitted with frank hemoptysis of one day’s duration. She denied recurrent pulmonary infections, bronchitis, or recent travel. There was one pack/day tobacco history. She was mildly febrile, with clear lungs and a benign abdomen. Laboratory findings included a hematocrit of 35 percent, white blood cell count of 14.7 × 10^9/L, and a serum alkaline phosphatase concentration of 196 IU (normal, <108 IU/L). The chest radiograph was notable for a small, wedge-shaped left lower lobe infiltrate (Fig 1). Fiberoptic bronchoscopy showed only mild erythema and edema of the left main-stem and lower lobe bronchi. Washings and transbronchial biopsies were negative for acid-fast bacilli, fungi, bacteria, and malignant cells. A left lower lobe bronchogram was similarly unremarkable. Ultrasonography of the abdomen disclosed an echogenic mass in the right upper quadrant suggestive of a liver abscess; serologic study confirmed the diagnosis of hepatic amebiasis. The patient was treated with intravenous metronidazole (3,000 mg/d) and oral di-iodohydroxyquin (1,950 mg/d), with resolution of the hemoptysis and clearing of the chest radiograph at eight days.

Patients with amebic liver abscesses classically present with abdominal pain, fever, anorexia, and weight loss.2 Thoracic complications occur in approximately 8 percent of cases (reported range, 4 to 14 percent) and may involve the pleura, lung parenchyma, or pericardium. The right hemidiaphragm may be elevated owing to upward displacement by the liver, and a sympathtic pleural effusion is often seen.3 Invasion into the chest occurs by direct extension through the diaphragm and is right-sided in the vast majority of cases. Hemoptysis may occur with rupture of the abscess through a bronchial wall.

Pleuropulmonary complications of amebic liver abscesses can be easily confused with tuberculosis, pneumonia, bacterial abscess, or carcinoma. This case was unusual in that the patient denied risk factors for amebiasis, did not manifest signs of amebic liver abscess, and demonstrated a left-sided radiographic lesion. This case also underscores the difficulty in establishing the diagnosis of amebiasis in the absence of abdominal findings in patients not considered to be at risk for the disease. Pleuropulmonary amebiasis should be considered in cases of atypical or refractory hemoptysis, regardless of whether pulmonary abnormalities are right- or left-sided.

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Henoch-Schönlein Vasculitis and Tuberculosis

To the Editor:

I read with interest the case report by Chan and Pang,1 which appeared in the August 1990 issue of Chest. I was surprised, however, by their last sentence, “This type of vasculitis associated with tuberculosis has not been reported earlier” (referring to a patient with disseminated vasculitis associated with tuberculous lymphadenitis).

In 1987 we reported the case of a 33-year-old man with active pulmonary tuberculosis that had not been treated with drugs, who developed a disease compatible with leukocytoclastic vasculitis of the Henoch-Schönlein type, without immunologic evidence of collagen-vascular disease. The patient had lymphadenitis and vasculitic lesions of the gut and skin, with palpable purpura in both legs. Biopsies of the vasculitic lesions revealed deposition in the walls of the small vessels of IgA, IgM, C3, and C1Q. The serum IgA concentration was 600 mg/dl (normal, 90 to 350 mg/dl). These clinical abnormalities abated in six weeks with corticosteroid and antituberculosis treatment. It is possible that the immune complex deposition in the vessel wall was the cause of the vasculitis, as demonstrated by Parish and Rhodes2 with the finding of mycobacterial antigen in four cases of vasculitis and tuberculosis. This case and the case of Chan et al strengthen the likelihood of the presence


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