Spontaneous Pneumothorax Associated with Pneumocystis carinii Pneumonia

Successful Treatment with Talc Pleurodesis

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Spontaneous pneumothoraces occur in patients with acquired immunodeficiency syndrome and Pneumocystis carinii pneumonia. However, treatment with tube insertion and tetracycline sclerosis often fails to prevent recurrence. We present a single case of such a patient successfully treated with talc sclerosis. (Chest 1992; 101:1177-78)

AIDS = acquired immunodeficiency syndrome; PCP = Pneumocystis carinii pneumonia

Recurrent spontaneous pneumothoraces have been described in patients suffering from Pneumocystis carinii pneumonia (PCP) treated with aerosolized pentamidine, and could be related to inhaled pentamidine or to a focal presentation of PCP. However, treatment with chest tube insertion and tetracycline sclerosis has failed to avoid PN recurrence, and reported cases were associated with a high mortality rate.

We present a single case successfully treated with talc pleurodesis.

CASE REPORT

A 43-year-old male homosexual presented in December 1989 with acquired immunodeficiency syndrome (AIDS)-related PCP. Symptomatic and radiographic resolution followed standard therapy. He then received prophylactic treatment with aerosolized pentamidine (100 mg once a week via a Respigrad nebulizer) and zidovudine (1,200 mg a day).

On July 27, 1990, the patient was admitted with dyspnea, no productive cough, and bilateral chest pain. Physical examination revealed an elevated temperature of 38°C, generalized lymphadenopathy, and rales in the right lung field. The chest roentgenogram showed an apical reticulonodular infiltrate in the right lung. Fiberoptic bronchoscopic examination was performed, and bronchoalveolar lavage fluid revealed P carinii. A therapeutic regimen including trimethoprim-sulfamethoxazole was given, and the patient clinically improved with no further complaints of dyspnea or chest pain.

Seven days later, a chest roentgenogram showed a 50 percent pneumothorax on the right (Fig 1), necessitating chest tube placement. Three days after lung reexpansion, the chest tube was removed, but four days later a complete pneumothorax on the right recurred. After a second chest tube insertion, the pneumothorax resolved, but a computed tomographic (CT) examination showed two cysts in the apex of the right lung with subpleural necrosis allowing the development of bronchopleural fistula (Fig 2). However, the chest tube was removed eight days later, after no further air leak was noted.

On August 30, 1990, the patient was admitted again, complaining

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FIGURE 1. Chest roentgenogram obtained on the seventh hospital day shows a large pneumothorax on the right.

of dyspnea and right chest pain. Physical examination demonstrated diminished breath sounds on the right side, and a chest roentgenogram revealed recurrent pneumothorax on the right. A pleuroscopic examination showed a violated pleural surface. Talc pleurodesis was attempted, a chest tube was inserted, and the right lung reexpanded. Six days after lung reexpansion, the chest tube was removed. The patient remained well without any recurrence of pneumothorax after talc pleurodesis.

On October 20, 1990, the patient was readmitted to a toxic septicemic state with status epilepticus. A chest roentgenogram showed bilateral interstitial infiltrates but did not reveal any pneumothorax. Despite palliative care, the patient died in the following hours.

DISCUSSION

Pneumothoraces frequently occur in AIDS patients with

FIGURE 2. Chest CT scan obtained after administration of contrast material. Two cysts are depicted in the apex of the right lung. Central cavitation is seen in the subpleural one.
PCP. These may be related to treatment with aerosolized pentamidine or to focal presentation of PCP. It is noteworthy that pneumothoraces are recurrent in most of the reported cases. In addition, tetracycline sclerotherapy via chest tube failed to maintain lung reexpansion. We treated spontaneous pneumothorax associated with PCP in one AIDS patient by means of talc pleurodesis.

As previously described, pneumothorax occurred in association with a focal recurrence of PCP in a patient who had received aerosolized pentamidine. Although no air leak was noted after the first chest tube placement, recurrence of pneumothorax suggested that a bronchopleural fistula had developed. The CT scans clearly showed that one of the two cysts in the right lung apex had central necrosis with a rupture into the pleural space (Fig 2). This finding, as well as pathologic demonstration of subpleural infected cysts associated with pleural violations, strongly argues that pneumothoraces in AIDS patients with PCP are related to peripheral sequestered infections, perhaps fostered by the inhomogeneity of aerosolized pentamidine distribution. As has been recently shown, aerosol distribution could be improved by supine positioning of the patient during the treatment. We thus can speculate that pneumothoraces related to focal PCP recurrence could be avoided.

Since conflicting reports have been reported regarding the effect of tetracycline sclerosis in such cases, we used talc pleurodesis for the treatment of recurrent pneumothorax. Pleural poudrage with talc has already been used for the production of pleural adhesions and the control of malignant pleural effusions. However, it is not used extensively for the treatment of recurrent pneumothorax; surgical pleurodesis is often preferred. In patients with AIDS and PCP surgery may be difficult, and pleurodesis can be achieved by means of talc poudrage during a pleuroscopic examination. In addition, talc pleurodesis has been shown to be more efficient than tetracycline sclerotherapy for the control of pleural effusion; moreover, good pleural adhesion can be achieved since talc is not resorbed. Although in our case talc pleurodesis was successful, one should keep in mind that other treatments, such as bleomycin pleurodesis, can also be used. Further studies comparing bleomycin/tetracycline sclerotherapy with talc pleurodesis are required before a management strategy can be definitively recommended.

Focal presentation of PCP with cavitation could explain the recurrence of spontaneous pneumothoraces in AIDS patients. Talc pleurodesis could be an alternative therapeutic possibility when chest tube insertion fails to maintain lung reexpansion.

REFERENCES


Idiopathic Hypereosinophilic Syndrome-Related Pulmonary Involvement Diagnosed by Bronchoalveolar Lavage*

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Patients with HES and pulmonary infiltrates may pose certain diagnostic problems as the infiltrates may be attributed to infection, infarction, congestive heart failure, or HES itself. We report an 87-year-old woman with idiopathic HES presenting with bibasal alveolar infiltrates. Differential cell count in BAL fluid yielded a very high percentage (73 percent) of eosinophils. Other authors previously mentioned the absence of eosinophils in the lavage fluid despite an important peripheral eosinophilia in a patient with the idiopathic HES but without HES-related pulmonary involvement. Thus, BAL fluid eosinophilia may suggest HES-related pulmonary involvement. Therefore, BAL might be an important diagnostic tool in the management of pulmonary infiltrates in idiopathic HES.

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HES = hypereosinophilic syndrome

The value of BAL in the management of a large variety of pulmonary diseases has become increasingly apparent since the use of flexible fiberoptic bronchoscopy in the 1970s.

Modifications of differential cell count in lavage fluid have been observed in many disorders. For instance, sarcoidosis and hypersensitivity pneumonitis both show T-lymphocyte predominant inflammation, in contrast to idiopathic pulmonary fibrosis mainly showing increased neutrophils.

Many pulmonary disorders may be associated with moderately increased eosinophils in BAL fluid including asthma, histiocytosis X, sarcoidosis, idiopathic lung fibrosis, and

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