Pneumothorax Complicating BiPAP Therapy for Pneumocystis carinii Pneumonia

To the Editor:

Pneumothorax is increasingly recognized as a complication of Pneumocystis carinii pneumonia (PCP) in patients with AIDS. Of 20 patients with AIDS and spontaneous pneumothorax treated at the Sloan-Kettering Cancer Center in New York in a decade, 19 had concurrent PCP, and 18 had received inhaled pentamidine prophylaxis. We report the case of a patient who developed bilateral pneumothoraces as a complication of PCP and AIDS while receiving ventilatory support with a bilevel positive airway pressure (BiPAP) mask. There was no prior history of inhaled pentamidine use.

A 32-year-old previously well homosexual man presented with a 3-day history of progressive dyspnea, nonproductive cough, and fever. He was found to have bilateral interstitial infiltrates and was treated with intravenous co-trimoxazole (trimethoprim-sulfamethoxazole), erythromycin, and methylprednisolone. Bronchoalveolar lavage revealed 44 P carinii organisms on Giemsas stain. Over the next 2 days he developed progressively worsening hypoxemia and dyspnea and was transferred to the ICU.

In the ICU, furosemide was administered intravenously, and a BiPAP mask was applied with an inspiratory pressure of 10 cm H2O, an expiratory pressure of 5 cm H2O, and an oxygen flow rate of 12 L/min. The patient had a spontaneous respiratory rate of 24 breaths per minute. The patient became less dyspneic and had adequate oxygenation (Po2, 11.2 mm Hg; Po2, 5.72 mm Hg; and [H+] 4.52 mm Hg), but after 12 h he suddenly developed increased dyspnea and fatigue. A chest radiograph showed bilateral pneumothoraces, which had not been evident 12 h previously. He was endotracheally intubated. Bilateral thoracostomy tubes were placed, and tension pneumothoraces were drained. Over the next 7 days he remained intubated and ventilated. His temperature normalized, and his gas exchange and infiltrates improved. He was weaned from the ventilator and discharged to the ward and was subsequently sent home 10 days later. His corticosteroid dosage was tapered, and he completed 20 days of intravenous co-trimoxazole and an additional week of oral co-trimoxazole as an outpatient. An enzyme-linked immunosorbent assay was positive for antibody to HIV.

We believe this to be the first report of pneumothorax associated with noninvasive intermittent positive-pressure ventilation by face mask in the treatment of acute respiratory failure due to PCP in an AIDS patient. It was tried in this patient as a temporizing measure to avoid endotracheal intubation. It is likely that pneumothorax would have arisen had any other form of positive-pressure ventilation, such as conventional assist mode, been used. Pneumothorax is an important adverse effect of BiPAP and/or PCP that can produce rapid deterioration and can occur without prior prophylactic inhaled pentamidine therapy.

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Giant T-Wave Inversion in Patients With Acute Coronary Insufficiency

To the Editor:

In the April 1992 issue of Chest, Fisher and colleagues reported the cases of an interesting series of patients with giant T-wave inversion and acute coronary insufficiency. In view of our recent report of global T-wave inversion1 and their Figure 1, which is an example of global T-wave inversion fulfilling our criteria,1 I should like to ask the authors to clarify some points in their report.

First, the methodology includes the ECG criterion "T-wave inversion greater than or equal to 10 mm in at least two contiguous precordial leads." The leads are not specified by case in their table, and it would be important to know how many leads were involved in each case. Was Figure 1, for example, typical? We found that same ubiquitous T-wave inversion pattern to occur in coronary disease, but to be nonspecific; a minority of our patients had coronary disease (and few had giant inversions). If, however, two- or three-lead involvements were more typical then Figure 1 suggests, coronary disease in the authors' series is better understood.

Finally, the authors' Methods section excluded patients whose ECGs showed left ventricular hypertrophy (LVH). Table 1, however, lists six of nine patients as having LVH, and the authors conclude that a possible cause of the patterns they report may be ischemia "and some degree of left ventricular hypertrophy." Clarification would be most helpful; for example, was the LVH in six of nine patients determined by echocardiography or other imaging?

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REFERENCES

To the Editor:

We thank Dr. Spodick for his interest in our article and offer the following additional data on our patients.

The precordial leads in which the criteria for giant T-wave inversion were met in our nine patients are as follows: patient 5, V3 and V4; patients 2, 6, and 7, V2 and V3; patient 8, V3 through V6; patient 4, V3 through V5; and patients 1, 3, and 9, V2, V3, and V4. It should be noted that while these were the leads with giant T-wave inversion, patients 2 through 9 had some T-wave inversion in V3 through V5, and patient 1 had T-wave inversion in V1 through V6. It is important to recognize that our patient population was probably different from that screened by Dr. Spodick. We limited our screening to patients admitted to our coronary care unit and, as would be expected, found mainly patients with coronary disease.

Our diagnosis of LVH in six of the nine patients was determined by echocardiogram. We did exclude patients with ECG evidence of LVH but were not surprised to find echocardiographic evidence of LVH since echocardiography is known to be far more sensitive.

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Communications to the Editor