
To the Editor:

The communication from Dr. Dens and colleagues focuses attention on another potential etiology for life-threatening right heart thrombus. Our own approach is to avoid the use of indwelling central venous lines whenever possible. In particular, Swan-Ganz catheter use has markedly declined in our coronary care unit. Rather, the steadily increasing trend is toward image and Doppler echocardiography of left and right ventricular systolic performance and calculation of pulmonary arterial pressure for the accurate, non-invasive reflection of cardiac function in the acute care setting.

The increased recognition of right heart thrombus in its transient passage from peripheral locations to the pulmonary vascular bed has, in part, occurred because of this change in intensive care practice. In the last several months we have diagnosed two further cases of in vivo right heart thrombus in the medical intensive care unit of the University of Alberta Hospitals. In both cases, diagnosis was made by echocardiography and cardiac surgery was successfully performed with survival of the patients.

The sum of our more recent experience and that presented previously has further increased our awareness that massive pulmonary embolism is a not-infrequent cause of death in severely or chronically ill patients with acute compromise of their cardiovascular status. Hopefully, future developments in noninvasive testing will allow the earlier detection of substrate pathology before life-threatening clinical status is reached.

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REFERENCE


Aerosolized Pentamidine and Spontaneous Pneumothoraces in AIDS Patients

To the Editor:

We were quite interested in the six cases of spontaneous pneumothoraces reported by Martinez et al. We have not experienced any episodes of spontaneous pneumothorax in our AIDS patients in either of our two secondary prophylaxis aerosolized pentamidine studies.

From June 1, 1988 to December 31, 1988, there have been 55 patients involved in our double-blind randomized, placebo-controlled trial comparing treatment with pentamidine vs placebo for secondary prophylaxis of Pneumocystis carinii pneumonia (PCP). Since the study is still in progress, we do not know the exact number of individuals on pentamidine therapy. However, after eight months of prophylactic treatment, no spontaneous pneumothorax has been detected. These patients were clinically assessed on a monthly basis, had complete pulmonary function tests on a bi-monthly basis, and had chest radiographs (CXR) at baseline and at six months.

In another study (an open-labelled trial which was initiated in October, 1988) we have 14 patients on aerosolized pentamidine therapy for secondary prophylaxis of PCP. During the first two months of this study, none of the 14 individuals developed spontaneous pneumothorax.

Aside from our formal trials, we have one patient who developed three episodes of spontaneous pneumothorax and one episode of pneumomediastinum while he was on secondary prophylactic therapy with aerosolized pentamidine. The patient obtained his own supply of pentamidine and the dosage was 60 mg every two weeks. The nebulizer used was obtained through a US source and was a FISONeb. However, it should be pointed out that all four episodes occurred in the setting of early relapse of PCP. Therefore, it is difficult to attribute the cause of pneumothorax/pneumomediastinum to aerosolized treatment because they may be due to concurrent PCP.

The reason for the "alarming number of pneumothoraces" experienced by Martinez et al is unclear. However, the authors might help the readers by providing the following information: 1) How many patients were treated (ie, the denominator for calculation of the occurrence rate of spontaneous pneumothorax)? 2) Were all the episodes of pneumothorax clinically relevant or were they detected by a routine surveillance protocol? 3) What was the dose of pentamidine and the type of nebulizer, and how often was the drug administered? 4) What was the frequency of cough and bronchospasm with aerosolized treatment?

The dosage of pentamidine in both our studies is 60 mg per treatment. We administer five loading doses over a two-week interval, followed by a maintenance dose at bi-weekly intervals. The nebulizer used in these studies is a FISONeb ultrasonic nebulizer (Fisons Corp, Bedford, MA). In our open trial, the frequency of cough/bronchospasm as detected by spirometry was about 14 percent. These episodes were generally mild and responded well to inhaled bronchodilator.

We suspect that the ultrasonic nebulizer used may be the principal cause for the development of spontaneous pneumothorax. However, if that were true then one would expect a direct correlation between the occurrence of spontaneous pneumothorax and the frequency of cough/bronchospasm.

We agree with Martinez et al that continued pulmonary surveillance is both necessary and important in patients on prophylactic aerosolized pentamidine treatment.

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2 Joe L, Gordin F, Parker R. Spontaneous pneumothorax with Pneumocystis carinii infection. Arch Intern Med 1986; 146:1816-17

To the Editor:

The correspondants provide important clinical experience in the prophylactic use of aerosolized pentamidine.

Since we only observed that we were and are seeing an alarming
number of spontaneous pneumothoraces at our hospital, we cannot comment on true occurrence or frequency of cough and bronchospasm.

The patients we reported all required chest tube insertion, either because of respiratory compromise or the size of the pneumothoraces (large and potentially life-threatening).

Since our letter, we have had 18 more cases of pneumothoraces, for a total of 24 cases of pneumothoraces; 96 percent were among homosexual men, and 4 percent intravenous drug abusers. Only one case had no previous history of Pneumocystis carinii pneumonia; all were pharyngally treated with aerosolized pentamidine.

The nebulizers used have included Aerotech II, Respigard II and DeVilbiss ultrasonic nebulizer; the majority used the Respigard II. Dr. Chan has only used FISONeb, which brings up the point of particle size and efficacy of deposits as variables in the evaluation of causology. Smaldone et al. have shown that there are important differences in the size of the particles produced by Aerotech II, Respigard II and FISONeb, with FISONeb being least efficient of the three regarding deposition. Whether the differences in efficiency of deposits correlate with differences in success of prophylaxis will only be born out in careful studies.

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REFERENCE


Proteus vulgaris Empyema and Increased Pleural Fluid pH

To the Editor:

The decision on the need for thorocostomy and drainage of a parapneumonic effusion can be difficult. A pleural fluid pH of less than 7.00 is often used to identify an empyema needing immediate drainage.1,2 Infection with urea-splitting organisms may, however, cause an empyema with an elevated pH. We report a case of Proteus vulgaris empyema demonstrating that a high pleural fluid pH does not preclude the need for thorocostomy.

A 55-year-old man was admitted with foul-smelling sputum, fever and an abscess cavity in the right lower lobe. Sputum culture showed multiple organisms and bronchoscopy demonstrated no obstructing lesion or malignancy. The patient was treated with intravenous penicillin and, subsequently, clindamycin.

He returned two weeks later with increasing right-sided chest pain, cough and fever. He had a temperature of 37.8°C on admission and an increasing air-fluid level on chest roentgenogram. Intravenous clindamycin was started but the patient remained febrile to 39°C. Sputum culture grew Proteus vulgaris. Five days after admission, a new right pleural effusion developed. Thoracentesis yielded hazy yellow fluid with a protein content of 3.1 gm/dl, LDH 208 IU and a leukocyte count of 3500/mm² with 97 percent polymorphonuclear cells and 3 percent lymphocytes. Serum values were as follows: protein 6.4 gm/dl and LDH 184 IU. Pleural fluid gram stain was negative and pleural fluid pH was 7.61. Culture of pleural fluid grew Enterococcus and two strains of Proteus vulgaris. Antibiotics were changed to gentamicin, penicillin and cefoxitin. A chest tube was placed and the patient improved.

Pine and Hollman reported three cases of Proteus mirabilis empyema with elevated pleural fluid pH.3 Ammonia levels were measured where patients. Proteus vulgaris as well as P mirabilis has a urease enzyme that splits urea to produce ammonia, resulting in an increased pH. Our patient provides further evidence that Proteus infections of the pleural space may be associated with an elevated as well as depressed pleural fluid pH. It further emphasizes that empyema cannot be excluded on pleural fluid pH alone and routine bacterial cultures are still needed.

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1 Light RW, MacGregor MI, Ball WC, Luchsinger PC. Diagnostic significance of pleural fluid pH and Pco₂. Chest 1973; 64:591-96

Drug-resistant Tuberculosis in AIDS

To the Editor:

Tuberculosis has been recognized as a manifestation of the acquired immunodeficiency syndrome (AIDS)1 and its treatment has become an important and controversial issue.2 Many questions regarding adequate number of drugs and length of treatment have not been answered and await long-term, controlled clinical trials. We have recently reviewed the prevalence of drug-resistant Mycobacterium tuberculosis in our hospital3 and decided to evaluate it among the growing population of AIDS.

We retrospectively searched our mycobacteriology register for cases of M tuberculosis infection from 1982 to early 1988. Of 137 isolates, 13 had a clinical diagnosis of AIDS or AIDS-related complex, and their clinical records were reviewed.

Of the 13 patients, 11 were men and two women, with a mean age of 38 years; six were Hispanic, three black, three white and one Asian; eight were admitted to non-private services and five acknowledged a prior history of tuberculosis. Regarding risk factors and diagnosis criteria: seven were homosexual or bisexual, four were intravenous drug abusers, one was married to an AIDS patient (also with tuberculosis) and one did not have any recognized risk. Two were cases of AIDS-related complex, five had Kaposi's sarcoma (one in the lung simultaneous with tuberculosis), four had P carinii pneumonia (two concomitant with tuberculosis) and the remaining had other opportunistic infections. Four had a diagnosis of AIDS first; tuberculosis preceded it in three of our 13 cases. At admission, diagnosis of tuberculosis was made in four cases and only considered in four more. Patients had an average of six symptomatic weeks.

Eleven of the 13 cases had infection in the lung, two in the blood, two in bone marrow, one in CSF and one in a lymph node. Chest x-ray examinations showed diffuse interstitial infiltrates in 6 patients and apical infiltrates, with or without cavitation, in only four. Most of the patients were started on therapy with three drugs and did well clinically and radiologically after four weeks of treatment, except for two who died from other complications of AIDS. Three cases (0.23% vs 0.13% in our total tuberculosis population) were resistant to either INH, RMP, EMB or SM, and two of these to both INH and RMP (both patients were homeless).

Ten percent of our tuberculosis cases are AIDS-related and most of them have been seen in the last three years. Of the three cases with mycobacteremia, two had AIDS. In only five of our 13 patients was tuberculosis only and alone in the lungs, presenting in half of the cases with diffuse infiltrates on chest x-ray film. Drug resistance was seen in three cases, and in two it was multiple (these patients

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