Drug Treatment of Chronic Tuberculous Empyema

To the Editor:

We read with great interest the article by Iseman and Madsen,1 which appeared in the July 1981 issue of Chest. The message that impaired drug penetration into infected tuberculous empyema can result in subtherapeutic concentrations, leading to acquired drug resistance and treatment failure, deserves attention. Adjunctive surgical intervention, as put forward by Iseman and Madsen, should be advocated. However, when one is limited by the compromised cardiopulmonary status of the patient or refusal of surgical treatment, intensive chemotherapy with multiple drugs has to be considered.

Ofloxacin, a fluoroquinolone, has been shown to be active against Mycobacterium tuberculosis in vitro.2,3 It has also been demonstrated to exhibit clinical efficacy in patients with severe cavitary pulmonary tuberculosis who harbor multiply resistant organisms.4 In addition, we have found lately that the drug penetrates tuberculous effusion exceedingly well.5 The sputum/serum and pleural fluid/serum drug ratios have been found to be about 0.8 and 0.9, respectively, in the two aforementioned studies. Although, as Iseman and Madsen1 pointed out, the behavior of tuberculous empyema might be different from that of effusion, we still feel, subject to further verification studies on tuberculous empyema, that ofloxacin might have a place in combination chemotherapy in the former clinical setting.

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1 Iseman MD, Madsen LA. Chronic tuberculous empyema with bronchopleural fistula resulting in treatment failure and progressive drug resistance. Chest 1991; 100:124-27

To the Editor:

We appreciate very much the interest and comments about our article on the management of chronic tuberculous empyema. Our group has also been studying the role of fluoroquinolone drugs (particularly ciprofloxacin and ofloxacin) in the treatment of patients with tuberculosis resistant to multiple drugs. In fact, four of the five patients in our article, all four of whom failed therapy, received one of the agents.

Although we do not have drug levels directly from the empyema spaces to document poor penetration, we do have serum drug levels to confirm that we achieved high, therapeutic concentrations of the fluoroquinolones and other medications in the blood of these patients. Hence, in view of the poor response, we infer that there

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3 Adamo JP, Mehta AC, Stelmach K, Meeker D, Rice T, Stoller JK. The Cleveland Clinic's initial experience with transtracheal oxygen therapy. Respir Care 1990; 35:153-60

To the Editor:

The communications of van der Werf et al and Hoffman et al reinforce our concern about the potentially serious complication of mucus ball airway obstruction complicating TTQT. In our case, catheter stripping was performed seven days after SCOOP I catheter placement, as recommended by the Institute of Transtracheal Oxygen Therapy. No expectoration of mucus balls was observed following this procedure.

The fatal outcome described in our case report1 has resulted in a heightened clinical index of suspicion for the presence of this complication, but has not prevented our team's use of this valuable therapy adjunct in appropriately selected patients. Vigorous mucolytic therapy in such patients is clearly important, as it is in all patients with chronic obstructive pulmonary disease who demonstrate airway secretion clearance impairments.2

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Inflammatory pseudotumors originating from major airways have been polypoid or sessile masses in all of the reported cases. In contrast, the tumor we encountered did not form a mass but was seen to be invading beneath the bronchial epithelium. We believe that this type of inflammatory pseudotumor, which invades the subepithelium, has not been previously reported.

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Spontaneous Pneumomediastinum

To the Editor:

I read with interest the article by Abolnik et al.1 on spontaneous pneumomediastinum (SPM), which appeared in the July 1991 issue of Chest. The observations and conclusions in this study are very similar to those published by us a few years ago,2 although the annual incidence in our study was more than twofold higher. Abolnik et al included 11 patients who were initially admitted to other hospitals. It was not clear whether the original roentgenograms and records were examined. In addition, they also included three patients with concurrent pneumothorax, a subgroup we excluded from our report. There is no way to determine whether spontaneous pneumothorax preceded SPM or vice versa. We excluded pediatric patients with acute asthma, a group in which the rate of SPM may exceed 5 percent.3

The benign nature of SPM has been proved by us4 and others.4 We have shown that esophageal contrast studies and bronchoscopic procedures are not required unless there is reason to suspect a perforation of the aerodigestive system. Yet such studies were performed in 25 percent of the patients of Abolnik et al. The mean hospitalization of 6.3 days seems to be extremely long. We now observe most cases for 24 to 48 h, and about 25 percent are presently not admitted at all and are justified in being released in our outpatient clinic.

It was initially believed that SPM and spontaneous pneumothorax are two presentations of the same condition—rupture of subpleural blebs. This is supported by the similar age and sex distribution for both entities. The rarity of recurrence of SPM and the lack of a distinguishing body habitus are consistent with different processes. We are presently evaluating 40 patients with SPM and comparing them radiologically and functionally with patients with spontaneous pneumothorax. Although not completed, the study reveals surprising similarities between the two conditions.

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