selected reports

Severe Acute Respiratory Syndrome Complicated by Spontaneous Pneumothorax*

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Severe acute respiratory syndrome (SARS) presents an unprecedented diagnostic and therapeutic challenge to clinicians. Despite recent progress in identifying and analyzing the coronavirus that is responsible for it, few reports have addressed the clinical complications of SARS. The present study was a two-center retrospective cohort study. All patients in the study had SARS, were managed in the two major Hong Kong hospitals (ie, Prince of Wales Hospital and United Christian Hospital), and had developed spontaneous pneumothorax during their hospitalization between March 10, 2003, and April 28, 2003. Spontaneous pneumothorax was reported in 6 of 356 SARS patients who were treated at the two hospitals during the period. This represents an incidence of 1.7%. None of the six patients had a history of smoking or pulmonary disease. The rate of admission to the ICU was 66.7% and the crude mortality rate was 33.3% in this group of patients. There was a trend for the mean neutrophil count in these patients to be higher than in previously reported cohorts of comparable SARS patients (14.5×10^9 vs 4.6×10^9 neutrophils per liter, respectively). Conservative measures like tube thoracostomy or observation alone offered satisfactory initial symptomatic management in five of six patients. Spontaneous pneumothorax is a specific and potentially life-threatening complication in SARS patients. Patients with extensive lung injury, as indicated by severe clinical courses, and in particular high neutrophil counts, appear to be most at risk. The benefits of surgical management must be balanced against the potential risks to health-care workers. *(CHEST 2004; 125:2345–2351)*

Key words: complication; pneumothorax; severe acute respiratory syndrome

Abbreviations: HRCT = high-resolution CT; LDH = lactate dehydrogenase; PWH = Prince of Wales Hospital, SARS = severe acute respiratory syndrome; UCH = United Christian Hospital

S evere acute respiratory syndrome (SARS) is a form of atypical pneumonia that is caused by a mutant coronavirus,[1] and was first identified as a new disease in February 2003.[2,3] SARS has created a major challenge to clinicians, but that challenge has been met with a historical worldwide effort. In the space of months, a considerable

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For editorial comment see page 1982

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body of information has been generated regarding this virus, in particular regarding its genome and molecular biology.[4–6] Although definitive algorithms for its diagnosis and management are now beginning to emerge,[7–9] there have been few reports on its complications. SARS appears primarily to cause severe respiratory distress from diffuse pulmonary inflammation.[10] Additional pulmonary complications such as pneumothorax, with its impact on the already compromised lung function, are therefore of great concern to the patients and their clinicians.

The Prince of Wales Hospital (PWH) in Hong Kong, a university teaching hospital with nearly 1,500 beds, was the first epicenter of the SARS outbreak in Hong Kong, one of the areas in the world most heavily hit with the disease.[8] Between March 10, 2003, and April 28, 2003, a total of 214 SARS patients were treated at PWH. The United Christian Hospital (UCH) is a major district general hospital with 1,265 beds, and it received 142 SARS patients during the same period. Most of the SARS admissions to UCH represented cases from the Amoy Gardens housing complex, which was the site of the largest community-based outbreak in Hong Kong, accounting for > 300 cases.[8] In this study, we report our combined experience in managing six SARS patients who developed spontaneous pneumothorax during their hospitalization at these two institutions.

Materials and Methods

All patients in whom SARS was diagnosed according to the World Health Organization case definition[11] at either PWH or UCH during the period from March 10, 2003, to April 28, 2003, and who experienced a spontaneous pneumothorax to one or both pleural spaces during their hospitalization were eligible for inclusion in the study. The World Health Organization case definition is as follows: history of high fever (ie, temperature, > 38°C); one or more respiratory symptoms, including cough, shortness of breath, and difficulty breathing; and close contact within 10 days before the onset of symptoms with a person in whom SARS was diagnosed, history of travel within 10 days before the onset of symptoms to an area with reported foci of SARS transmission, or both.[11] Patients whose pneumothorax could be attributed to a preexisting pulmonary disease or to an iatrogenic cause (eg, barotrauma or invasive procedures to the chest or neck) were excluded from the study. At PWH, we were aware of four patients who developed pneumothorax secondary to mechanical ventilation (two patients) or central venous cannulation (two patients). As the causes of pneumothorax in these cases were clearly iatrogenic and not spontaneous, these patients were not included in our series.

Six patients met the inclusion criteria for our study. Patients in cases 1 to 3 were admitted to PWH. Patients in cases 4 to 6 were all residents of Amoy Gardens and were admitted to UCH. The demographic, clinical, and laboratory data for these patients are summarized in Table 1.

The medical treatment protocol for SARS patients at both of our institutes is the same and has been previously reported.[7–9] All patients in this study received therapy with IV ribavirin (Rebetol; Schering-Plough; Kenilworth, NJ) and steroids (typically, methylprednisolone) according to this protocol.

Case Reports

Case 1

A 47-year-old man who was a nonsmoker with no significant medical history presented with a 2-day history of acute onset of fever, chills, and severe myalgia. He was a radiographer at PWH who had been in close contact with SARS patients for the 3 weeks since the initial outbreak. His chest radiograph showed diffuse bilateral pulmonary infiltrates at the time of hospital admission. Treatment with broad-spectrum antibiotics, ribavirin, methylprednisolone, and convalescent serum was given with initial clinical improvement. On the 32nd day after hospital admission, he was noted to have a sudden increase in dyspnea. Chest radiography confirmed a left pneumothorax with parts of the left lung adherent to the parietal pleura as a result of postinflammatory adhesions (Fig 1), creating several loculations of air and fluid within the left pleural space. The pneumothorax was managed with a chest tube. However, despite suction of 10 cm H_2O from the chest drain, the chest radiograph showed that the left lung had only partially reexpanded, with several large loculations of air still remaining. A CT scan of the thorax confirmed the presence of loculated air pockets, which were not adequately drained, and showed diffuse, patchy inflammatory changes in both lungs, with a "honeycomb" appearance that was similar to that of mild bronchiectasis (Fig 2). A pigtail catheter was inserted into the largest loculation under ultrasound guidance. The pigtail catheter then was replaced with another chest tube for better drainage, and both drains were set to suction at 10 cm H_2O. Symptomatic relief of the dyspnea was achieved, with improved lung reexpansion seen on chest radiography. An air leak persisted from both drains but eventually resolved with conservative management after 21 days, after which both the drains were successfully removed.

Case 2

A frail 82-year-old woman presented with a fever (ie, temperature, 38°C), chills, dizziness, and rhinorrhea. She was a non-smoker with a history of hypertension but no pulmonary disease. Her son, a health-care worker with prior contact with SARS patients, had recently received a diagnosis of SARS but had been living with this patient for over a week prior to his own hospital admission. Her chest radiograph on presentation showed mild bilateral focal haziness, and a CT scan of the thorax revealed consolidations in the upper and lower lobes of her right lung. She

Table 1—Demographic and Clinical Details of SARS Patients Who Developed Spontaneous Pneumothorax

Variables	Cases					
	1	2	3	4	5	6
Age, yr	47	82	47	32	48	32
Sex	Male	Female	Male	Female	Male	Male
Smoking history	No	No	No	No	No	No
Underlying lung disease	No	No	No	No	No	No
Chest radiograph findings on hospital admission	Bilateral lower zone infiltrates	Bilateral lower zone infiltrates	Right upper lobe consolidation	Right lower zone consolidation	Right lower zone infiltrates	Left lower lobe consolidation (on HRCT)
Peak LDH, U/L	301	573	801	854	691	713
Neutrophils × 10^9 cells/L	14.4	11.1	7.5	21.2	19.1	13.5
Total leukocyte count, %	93	90	90	96	97	96
Appearance of pneumothorax from hospital admission, d	32	37	14	25	17	21
Side of pneumothorax	Left	Right	Right	Bilateral	Bilateral	Bilateral
Cumulative dose of methylprednisolone at the time of pneumothorax, g	5.0	5.0	5.5	5.0	5.0	4.5
Mechanical ventilation subsequent to pneumothorax	No	No	No	Yes	Yes	No
Chest drainage duration, d	21	No drain used	14	28 (left)/22 (right)	31	No drain used
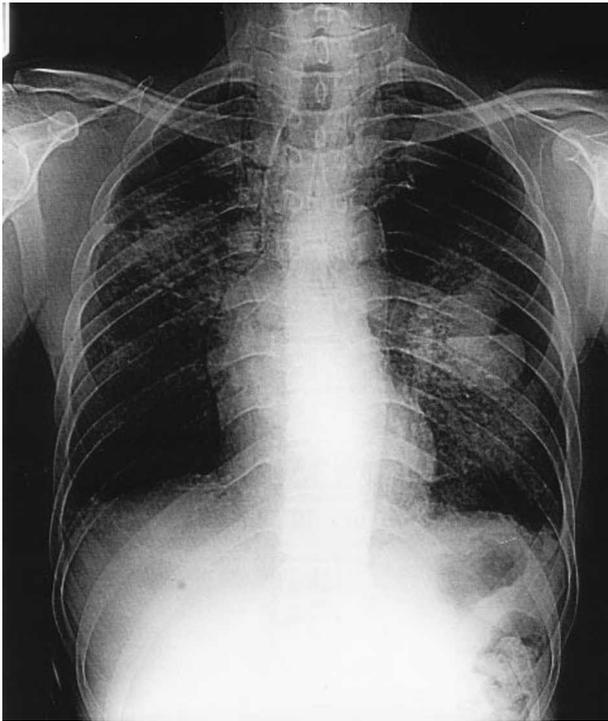
FIGURE 1. Chest radiograph of the patient in case 1 on the occurrence of a left pneumothorax with loculations. Note the fluid levels in the left middle zone and the mild pneumomediastinum. There are patchy pulmonary infiltrates and consolidations, which are typical of SARS, apparent in both lungs.

initially showed a good response to treatment with ribavirin and methylprednisolone, with settling of her fever, but on the 37th day after hospital admission, she had an acute increase in dyspnea. A chest radiograph confirmed a 20% right-sided pneumothorax. Despite her dyspnea, she remained fully conscious and was able to communicate verbally. Immediate chest drainage was offered to the patient, but she strongly refused. In view of the patient's wishes and the family's acceptance of her poor prognosis, it was agreed that invasive management of the pneumothorax would be withheld. Serial chest radiographs showed no further increase in the size of the pneumothorax. The patient gradually developed multiple organ failure from the SARS and died 4 days after the occurrence of the pneumothorax.

Case 3

A 47-year-old man who was a nonsmoker with no previous pulmonary history presented with a 5-day history of acute fever (*ie*, temperature, > 38°C) and dry cough. His initial chest radiograph showed right upper lobe consolidation only, but the radiographic changes progressed rapidly, with the development of pulmonary infiltrates in both lower zones after only 2 days. The clinical condition correspondingly worsened, and the patient required transfer to the ICU 4 days after the initial hospital admission, although he did not require mechanical ventilatory support. On the 14th day after hospital admission, his shortness of breath acutely worsened, and the chest radiograph demonstrated a 20% spontaneous right pneumothorax (Fig 3). Chest tube insertion was performed, with rapid improvement in his dyspnea. Even without suction applied to the chest drain, full reexpansion of the lung was demonstrated on chest radiography. However, an air leak persisted after chest tube insertion and took 14 days to resolve spontaneously without further intervention.

Case 4

A previously healthy 32-year-old woman who was a nonsmoker presented with a 1-day history of acute onset of fever, myalgia, and mild nonproductive cough. Her chest radiograph on presentation showed right lower zone consolidation. After initial improvement in response to treatment with ribavirin and steroids, she showed progressive clinical deterioration from day 15 onward. A high-resolution CT (HRCT) of the thorax showed widespread ground-glass opacifications in both lungs. On the 25th day after hospital admission, she developed sudden respiratory distress requiring urgent transfer to the ICU. The chest radiograph taken immediately confirmed a 50% left pneumothorax and a very small right apical pneumothorax. A left chest tube was inserted immediately with good symptomatic relief of her dyspnea. Suction at 10 cm H_2O was applied, and subsequent chest radiographs showed complete reexpansion of the left lung and spontaneous resolution of the tiny right apical pneumothorax. She suddenly became more dyspneic again on the 31st day, and a chest radiograph showed a 50% right pneumothorax had recurred. A right chest drain was inserted, and 10 cm H_2O of suction was applied. Although the chest radiograph showed good reexpansion of the right lung, she developed progressively severe respiratory failure with ground-glass lung infiltrations seen on her subsequent chest radiographs. She was intubated 2 days later and received mechanical ventilation. This appeared to exacerbate the air leakage from the right lung, with chest radiography showing a recurrent right pneumothorax. A second right chest drain was inserted, and both were set on suction of 20 cm H_2O. Chest radiography confirmed complete right lung reexpansion, but her lung infiltrates progressed and she developed refractory hypoxemia. She died with intractable respiratory failure 4 weeks after ICU admission.

Case 5

A 48-year-old man presented with a 1-day history of acute onset of fever (temperature, 39°C) and chills. He had neither smoked nor experienced any significant medical illness. His chest radiograph on hospital admission showed mild right lower zone infiltration. Therapy with ribavirin and steroid yielded a good initial clinical response, but he deteriorated in the second week with a recurrence of high fever, increasing dyspnea, and diffuse bilateral patchy infiltrates seen on chest radiographs. On the 17th day after hospital admission, increased dyspnea and mild subcutaneous emphysema in the neck were noted, and the chest radiograph (Fig 4) showed a pneumomediastinum and thin rims (< 1 cm) of pneumothoraces bilaterally. He was transferred to the ICU, and a right chest drain was inserted. However, his dyspnea persisted, and he promptly required endotracheal intubation and mechanical ventilatory support. A left chest drain insertion was subsequently performed, and 10 cm H_2O suction was applied to both chest drains. A chest radiograph showed the full expansion of both lungs. However, after a few days of positive-pressure mechanical ventilation, increasing subcutaneous emphysema was noted, which is indicative of persistent air leakage. Therefore, one additional chest drain was inserted on each side. The air leaks gradually resolved after 1 month, and all chest drains were removed immediately prior to his weaning from mechanical ventilation.

Case 6

A 32-year-old man who was a nonsmoker presented with a 2-day history of fever (temperature, up to 40°C), chills, rigors, and myalgia. The findings of his chest radiograph taken on hospital admission were unremarkable, but the HRCT scan of his thorax demonstrated consolidation in the medial basal segment of the left lower lobe, which is consistent with SARS. Therapy with

FIGURE 2. CT scan of the thorax of the patient in case 1 showing inflammatory changes and honeycombing, which is typical of SARS, in both lungs. There is a loculated left pneumothorax, and a left chest drain is *in situ*.

ribavirin and methylprednisolone was administered, with a good initial clinical response. However, his fever recurred on day 5, followed by both clinical and radiologic deterioration. Seventeen days after hospital admission, his condition acutely deteriorated further with worsening dyspnea. Chest radiography showed bilateral lower zone ground-glass infiltrates with patchy consolidations. His HRCT scan demonstrated extensive ground-glass opacities and consolidation, plus an extensive pneumomediastinum with air tracking up to the roots of the neck, creating subcutaneous emphysema. This was managed conservatively, but on the 21st day after hospital admission, he experienced oxygen desaturation to an arterial oxygen saturation of 70%, despite receiving maximal oxygen therapy via a rebreathing mask. He was transferred to the ICU, where a chest radiograph confirmed small (ie, < 10%) bilateral apical pneumothoraces. He was able to maintain borderline oxygenation while receiving maximal oxygen therapy. Chest drain insertion and mechanical ventilation were refused by the patient. Serial chest radiographs showed a gradual reduction in the size of the pneumothoraces. An HRCT scan performed on day 31 after hospital admission showed only a small residual pneumothorax on the right side and a slowly resolving pneumomediastinum. On day 41, an HRCT scan demonstrated complete resolution of the pneumothoraces on both sides.

RESULTS

During the 7-week period between March 10, 2003, and April 28, 2003, a total of 214 patients in whom SARS had been diagnosed were treated at PWH. A further 142 SARS patients were treated at UCH. From a total of 356 patients, the 6 patients reported on here represent an incidence of spontaneous pneumothorax complicating SARS of 1.7%.

The clinical details of the six SARS patients developing spontaneous pneumothoraces are summarized in Table 1. These patients included four men and two women, with a mean age of 48 years (age range, 32 to 82 years). None of the patients were smokers or had a history of pulmonary disease. Prior to the development of pneumothorax, none of the patients received any invasive procedures to the neck or chest, and none had received positive-pressure ventilatory support.

In each of the six patients, the pneumothorax was first discovered on a chest radiograph that had been taken as a result of an acute increase in dyspnea. The mean time between hospital admission and the appearance of a pneumothorax was 24.3 days (range, 14 to 37 days). At the time of the pneumothorax, all patients were clinically dyspneic, and five required maximal oxygen therapy via masks. They had each received a mean dose of 5.0 g IV methylprednisolone (dose range, 4.5 to 5.5 g). The mean peak lactate dehydrogenase (LDH) level for the six patients was 655.5 U/L (range, 301 to 854 U/L), and the mean neutrophil count was 14.5×10^9 neutrophils per liter (range, 7.5×10^9 to 21.2×10^9 neutrophils per liter).

Three patients developed bilateral pneumothoraces, two had a right pneumothorax only, and one had a left pneumothorax only. Four patients received chest tube insertions for treatment of the pneumothorax, with three patients (cases 1, 3, and 4) reporting good initial relief of dyspnea.

In all four patients who received chest tube insertion, persistent air leakage was noted. Surgical management was considered for these patients but ultimately was not
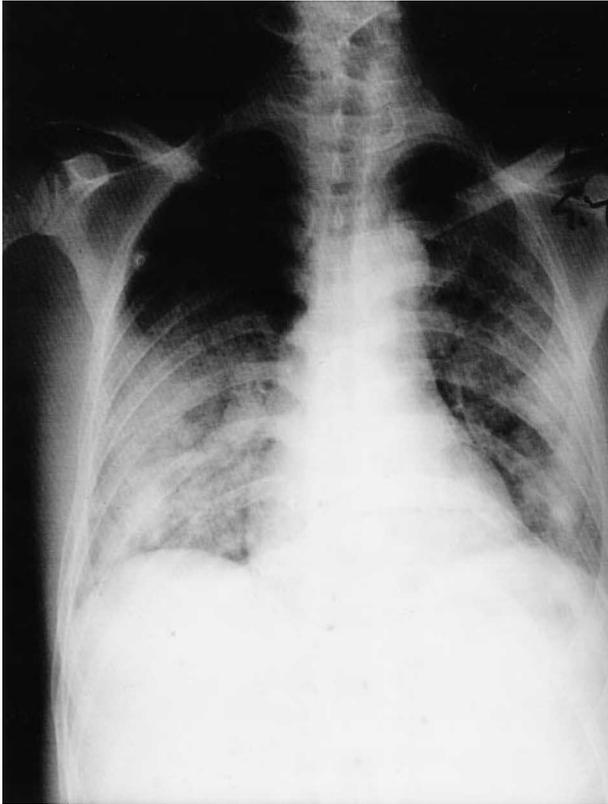
FIGURE 3. Chest radiograph of the patient in case 3 on the occurrence of a right pneumothorax. There are extensive bilateral lower zone pulmonary infiltrates seen, which is consistent with SARS.

undertaken. The factors taken into consideration included the severe compromise of each patient's lung function, which contributed to high anesthetic risk, and, most importantly, the ongoing SARS in each patient, which posed a potential infection risk to all operating room staff.[12] It was envisaged that endotracheal intubation, positive-pressure ventilation, lung manipulation during surgery, and the process of postoperative extubation might increase the risk of releasing virus-laden droplets into the operating theater environment. In the presence of a new infectious disease, the means of transmission of which had not been determined at the time, this represented a serious consideration. In case 1, the abundance of pleural adhesions noted on chest radiograph and CT scan also would give rise to a difficult operation with the possibility of intraoperative bleeding and significant postoperative air leakage. This added consideration mitigated against surgery for that patient. Using treatment with chest drainage alone, the air leaks in the four patients spontaneously resolved after a mean duration of 23.5 days (range, 14 to 31 days). In all six cases, pleurodesis, either chemical or surgical, was not used.

Three patients were admitted to the ICU because of exacerbations of dyspnea resulting from pneumothoraces. Two patients (cases 4 and 5) required mechanical ventilatory support for the respiratory distress resulting from

their bilateral pneumothoraces. In both cases, the effect of positive-pressure ventilation was to increase air leakage, which was manifested by an increasing pneumothorax despite drainage with a single chest tube in case 4 and surgical emphysema in case 5.

No patient in this study died as a direct result of their pneumothoraces. The patient in case 2 died of multiple organ failure related to generalized systemic inflammation. In case 4, the patient's bilateral pneumothoraces had been adequately drained, and she died from diffuse inflammation of the lungs rather than from mechanical respiratory compromise as a result of the pneumothoraces.

DISCUSSION

We have shown that spontaneous pneumothorax is a complication in 1.7% of SARS patients. To put this incidence into perspective, a parallel can be drawn between spontaneous pneumothoraces complicating SARS patients and secondary spontaneous pneumothoraces in patients with underlying pneumonia. Such cases are relatively uncommon and have been most prominently reported in immunocompromised patients with AIDS. The reported incidence of secondary spontaneous pneumothoraces in AIDS patients with *Pneumocystis carinii* pneumonia is approximately 2 to 6%.[13] In such patients, *P carinii* pneumonia may cause peripheral tissue necrosis in the lung, predisposing the patient to pneumothorax.

Several observations support the view that a similar pathogenetic process may occur in SARS patients. The onset of the pneumothorax in these six patients occurred at 14 to 37 days after the initial diagnosis of SARS, suggesting that a sustained period of lung inflammation is first required. All six patients were clinically very dyspneic, with high oxygen requirements and extensive lung radiographic changes at the time of their pneumothorax. All six patients also had high peak LDH levels and neutrophil counts, which are suggestive of a greater extent of pulmonary injury.[2] These observations suggest that extensive pulmonary injury may be a predisposing factor for spontaneous pneumothorax in SARS patients.

When compared to previously published data on SARS patients from PWH,[2] the cases of the six patients presented here show evidence of particularly severe lung injury. The ICU admission rate (66.7%) and the crude mortality rate (33.3%) were higher in these six patients than those in the 138 patients in the earlier study from PWH (23.2% and 13.8%, respectively).[2] That previous study from our institute also had identified absolute neutrophil counts and high peak LDH levels as independent predictors of adverse outcome in SARS patients.[2,9] The mean neutrophil counts and mean peak LDH levels in the 32 patients with poorer clinical courses (those who were either admitted to the ICU or died because of SARS) were significantly higher than those in the other 106 patients not requiring ICU support. In our six patients, the mean neutrophil count was 14.5×10^9 neutrophils per liter, and the mean peak LDH level was 655.5 U/L. In comparison, the mean figures for the 32 patients with poorer clinical courses in the earlier study were 4.6×10^9 neutrophils per liter and 629.7 U/L, respectively. Patients
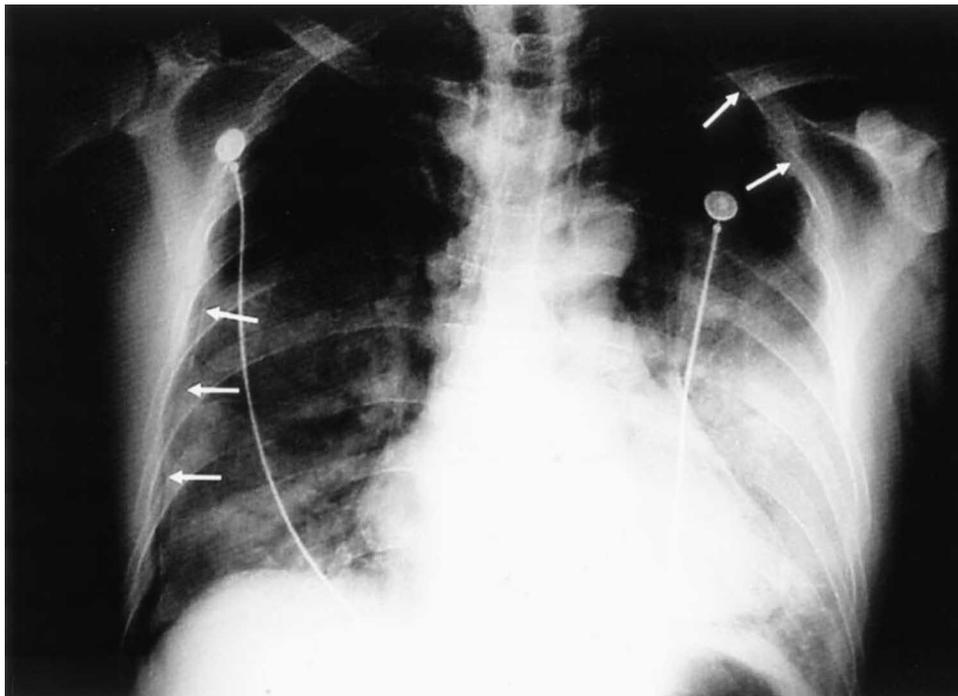
FIGURE 4. Chest radiograph of the patient in case 5 on day 17 after hospital admission. There are very thin rims of pneumothoraces on both sides. There is extensive inflammatory infiltration in both lungs from SARS.

with particularly severe clinical courses, and those with high peak LDH levels and neutrophil counts therefore appear to be the most at risk for developing spontaneous pneumothoraces. In particular, the trend for a higher neutrophil count in our six patients suggests that this may be an indicator for SARS patients who are at risk for developing spontaneous pneumothorax.

Histologic findings appear to support the hypothesis of severe pulmonary injury predisposing the patient to spontaneous pneumothorax. In postmortem analyses of other SARS patients, including patients from both PWH and UCH, evidence of histologic damage to the lung tissue by the disease previously has been reported.[2,9,14] The pulmonary pathology has been reported to be dominated by diffuse alveolar damage with pulmonary edema and hyaline membrane formation. The diffuse alveolar damage may give rise to dilated, cystic airspaces in severe cases and a gross appearance of honeycombing. Such destructive changes may predispose the lung to air leakage from the rupture of the cystic lesions, with the consequent development of a pneumothorax. The frequent diffuse involvement of both lungs by these changes also could account for the occurrence of the bilateral pneumothoraces seen in three of our six patients in this series.

A previous study[15] of SARS patients in UCH has identified the fact that spontaneous pneumomediastinum can occur in 12% of SARS patients. The occurrence of pneumomediastinum appeared to be unrelated to intubation and mechanical ventilation. A similar mechanism to that postulated earlier has been speculated to be responsible, with rupturing cysts causing the leakage of air into the mediastinum.

There has been debate among clinicians regarding the use of large doses of steroids in patients with severe sepsis or respiratory disease. Although the experience in Hong Kong has shown steroids to be of value in controlling the rapid and damaging host inflammatory response,[8,9] we appreciate the concerns of others[16] regarding the potential for steroid-related complications (including an increased risk of infection and impaired wound healing). It can be speculated as to whether aggressive steroid therapy may have a role to play in the pathogenesis of spontaneous pneumothorax in SARS patients. Indeed, the use of steroids to dampen the harmful inflammatory response has become one of the mainstays in the treatment of patients severely afflicted with SARS in Hong Kong.[7,8] We note that the doses of methylprednisolone received by the six patients reported on are no higher than those given to other SARS patients at PWH and UCH with similar clinical presentations. However, therapy with steroids may delay wound healing and perpetuate air leakage. The high doses of steroids given to the patients in cases 1, 3, 4, and 5 may be partly responsible for the persistent air leaks observed in these patients. In our six patients, no incidence of superinfection secondary to steroid use was noted.

Regarding the management of a spontaneous pneumothorax in SARS patients, conservative measures appeared to offer adequate initial symptomatic management in five of the six patients. Chest tube drainage alone brought about adequate short-term control of symptoms in cases 1, 3, and 4. The patients in cases 2 and 6 refused chest tube insertion yet showed no severe deterioration in their respiratory function. Only in the patient in case 5, who

experienced especially severe bilateral pulmonary inflammatory infiltrates, did the initial chest drain insertion not prevent the progression of his respiratory distress leading to intubation and ventilatory support.

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Enhanced Alveolar Clearance With Chest Percussion Therapy and Positional Changes During Whole-Lung Lavage for Alveolar Proteinosis*

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Pulmonary alveolar proteinosis has traditionally been treated with whole-lung lavage (WLL). The literature describes a variety of techniques used in performing the WLL, including mechanical vs manual chest percussion, use of prone positioning, and variances in lavage volume. We have quantified and compared the effective alveolar clearance for each component of the lavage by measuring the dry weight of material in the lavage effluent. We measured this in five patients who underwent six consecutive WLLs at the University of Pittsburgh Medical Center. We performed the lavage in the following three stages: stage I, passive drainage; stage II, assisted clearance; and stage III, positional clearance. Aliquots of lavage effluent were centrifuged to determine the dry weight of material present in sequentially recorded bottles within each stage. At the initiation of each augmentation, there was a statistically significant improvement in the clearance of material (stage II, p = 0.009; stage III, p = 0.012). Furthermore, we show that lipoproteinaceous material is present in the lavage effluent in all stages of latter portions of the lavage. The effective removal of material would be expected to have an impact on the physiologic and clinical response to WLL. This finding emphasizes the importance of performing an adequate and standardized lavage.
(CHEST 2004; 125:2351–2356)

Pulmonary alveolar proteinosis (PAP) is a disease characterized by the accumulation of lipoproteinaceous material within the alveolar spaces.[1–3] The most common therapeutic intervention for this disorder has been whole-lung lavage (WLL).[2,4] We have performed WLL on > 100 patients with PAP since 1968. WLL in patients with PAP has been associated with a reduction in dyspnea and improved physiologic parameters.[4–6] The clinical and physiologic response to WLL has been attributed to the removal of lipoproteinaceous material from the alveolar space.[3] To maximize the effectiveness of alveolar clearance, we have modified our previously published technique.[6–8] Our technique now focuses on large-volume WLL (ie, 40 to 70 L per lung), positional clearance with prone positioning for the last 10 to 15 L fluid, and assisted