Pneumatocele Formation in Adult Pneumonia*

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Pneumatocele formation is unusual in adult pneumonia, particularly in pneumococcal pneumonia. We report three cases of pneumatocele formation in adults, including one with probable pneumococcal pneumonia. All three patients were severely ill and two expired. Although they are usually asymptomatic, pneumatoceles may enlarge and compress the adjacent lung and mediastinum. This occurred in two patients causing respiratory insufficiency and cardiovascular compromise. The placement of a chest tube into the enlarging pneumatocele resulted in successful decompression.

Pneumatoceles are thin-walled cystic lesions in the lungs. They are differentiated from lung abscesses by their tendency to change in appearance, size and location rapidly.† Commonly seen as sequelae of staphylococcal pneumonia in infants and young children, pneumatoceles are infrequent in adults with pneumonia. They usually resolve spontaneously and completely over several weeks or months. Pneumatoceles, however, may enlarge to cause displacement and compression of the adjacent lung, resulting in cardiorespiratory insufficiency which requires urgent treatment. Spontaneous pneumothorax may also occur.

We describe three adult patients with pneumonia and extensive pneumatocele formation. Two were intravenous drug abusers with staphylococcal endocarditis and one had probable pneumococcal pneumonia complicated by the adult respiratory distress syndrome (ARDS). The patients were treated by intracavitary suction of the enlarging pneumatoceles with clinical improvement.

Case Reports

Case 1

A 38-year-old previously healthy Hispanic woman presented with fever, chills, and dyspnea for three days. She denied alcoholism or drug abuse. There was no significant past medical history. On admission, she was in moderate respiratory distress with temperature of 38.3°C; pulse rate, 110/min; respiratory rate, 36/min; and blood pressure 100/60 mm Hg. Chest examination revealed dullness and bronchial breath sounds in the right axilla and right lower anterior chest. There was no cardiac murmur. The white blood cell count was 13,700/cu mm. Arterial pH was 7.42; \(PCO_2\), 27 mm Hg; and \(PO_2\), 152 mm Hg on 4 L 100 percent oxygen via a nasal cannula. Chest roentgenogram showed a right middle lobe infiltrate. Repeated sputum examinations revealed Gram-positive diplococci and many neutrophils. Multiple blood, sputum and urine cultures failed to grow pathogens.

Shortly after admission, her blood pressure dropped and respiratory distress worsened. She was intubated, placed on a mechanical ventilator, and given broad spectrum antibiotics. The next day, she developed severe hypoxemia (\(PO_2\), 50 mm Hg on 65 percent oxygen) and bilateral diffuse homogenous infiltrates on chest roentgenogram, compatible with the adult respiratory distress syndrome. On the fourth day, she was found to have a right tension pneumothorax, possibly related to the PEEP of 15 cmH₂O. A chest tube was placed. She showed gradual but continuous improvement. The pneumothorax was completely resolved. She was extubated and all antibiotics were discontinued on the 24th day.

One week later, she was found again in severe respiratory distress. Spontaneous pneumothorax on the left side was diagnosed and a chest tube placed. At the same time, a single large pneumatocele was noted at the right lower lung field (Fig 1). Despite complete

![Figure 1. Case 1. Large pneumatocele at right base.](Image)

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recovery from the spontaneous pneumothorax, she was continuously in respiratory distress and had severe hypoxemia. The pneumatocele in the right lung had enlarged markedly in size. A chest tube was inserted into the enlarging pneumatocele for suctioning. She required intubation and assisted mechanical ventilation. Follow-up chest roentgenogram showed resolution of the pneumatocele, and she was relieved from all respiratory and cardiac symptoms. One week later, she was extubated and the chest tube was removed.

She remained asymptomatic for three days, but then developed renewed dyspnea and hypoxemia. Chest roentgenogram showed multiple large pneumatoceles on the right side causing a shift of the mediastinum (Fig 2). This necessitated chest tube placement into a large right pneumatocele with immediate return of the mediastinum to the midline (Fig 3). From this point on, she gradually improved clinically. She was treated with antibiotics for left lower lobe pneumonia. The chest tube was kept for six days. Follow-up chest roentgenograms showed resolution of the pneumatocele and pneumonia. She was discharged and has remained well with no apparent abnormality on chest examination.

Case 2

A 32-year-old Hispanic woman, an intravenous drug abuser, presented with a two-week history of fever, chills and dry cough. She was in moderate respiratory distress. Temperature was 40.0°C and respiration rate 40 per minute. Chest examination revealed rales over the lower two thirds of the lung bilaterally. There was a grade 3/6 holosystolic murmur at the left sternal border. Arterial pH was 7.52; Pco2 22 mm Hg; and Po2, 61 mm Hg breathing room air. The white blood cell count was 17,300/cu mm.

Chest roentgenogram showed multiple nodular patchy infiltrates on both sides. She was treated with intravenous penicillin, nafcillin and gentamicin. On the third hospital day, she defervesced. Blood cultures grew Staphylococcus aureus, and tricuspid vegetations were seen on echocardiogram. Follow-up chest film two days later showed rapid progression and cavitation of the infiltrates. Hypoxemia became progressively worse. She was intubated and placed on a mechanical ventilator. Subsequent chest film showed multiple bilateral pneumatoceles, with a shift of the mediastinum to the left side. She suddenly became cyanotic and pulseless, with a shift of the trachea to the right. The rapidly enlarging pneumatocele was treated by needle aspiration with transient relief. Subsequently, a chest tube was placed into the pneumatocele, resulting in reduction in size and return of the mediastinum to the midline. Three days later a large pneumatocele was found on the contralateral side and another chest tube was placed (Fig 4). Although the pneumatoceles were successfully decompressed, the patient remained septic, and expired one month later.

Case 3

A 30-year-old Hispanic woman, with a history of intravenous heroin abuse and alcoholism, presented with dyspnea, chest pain and fever. She was in respiratory distress. Diffuse rales were heard throughout both lungs and a grade 2/6 holosystolic murmur was heard at the lower left and right sternal border. The white blood cell count was 11,500/cu mm. Chest roentgenogram showed right pneumothorax and diffuse bilateral infiltrates. A chest tube was placed for drainage of air. She was initially treated with intravenous nafcillin, penicillin and gentamicin, and maintained on nafcillin when blood
cultures grew Staphylococcus aureus sensitive to methicillin. On the sixth hospital day she developed acute respiratory distress and hypotension following spontaneous left pneumothorax. The lung was fully expanded after chest tube placement. The subsequent chest film showed multiple bilateral pneumatoceles. She remained critically ill, and went into marked respiratory failure. She was intubated and placed on mechanical ventilation. Chest roentgenogram showed rapidly progressing diffuse alveolar infiltrates, and the pneumatoceles became more numerous and individually larger. Echocardiography showed tricuspid vegetations. Her clinical course continued to deteriorate and she expired on the 17th hospital day.

**DISCUSSION**

Pneumatoceles have been described most often as a complication of aerogenous staphylococcal pneumonia in infants and children. In contrast, our patients were young adults. Two had acute infective endocarditis with septic embolization. Although pneumatocele formation secondary to extrapulmonary sources of staphylococcal sepsis has been reported, pneumatoceles have not been described in infective endocarditis. Pneumatoceles rarely have been reported in pneumococcal pneumonia, with only four reports describing a total of eight patients.

Our patients with staphylococcal endocarditis were gravely ill and remained febrile for long periods despite appropriate antibiotics. Both expired after prolonged, unremitting sepsis, though in neither case was death directly due to the pneumatoceles. Both patients were intravenous heroin abusers and may have been immunocompromised. Neither patient had an opportunistic infection nor absolute lymphopenia. Diagnosis of the acquired immune deficiency syndrome could not be ascertained because testing for the human immunodeficiency virus was not available when these patients were seen. Immunocompromise, if present, may have contributed to the poor outcome.

Staphylococcus aureus infection often causes necrotizing pneumonia, and it is thought this results in pneumatoceles when there is necrosis at the bronchial and bronchiolar walls leading to rupture and interstitial emphysema. Large pneumatoceles can develop if the site of rupture acts as a check-valve. An alternative mechanism is leakage of air from the inflamed respiratory tree into the subpleural space. Rarely, necrotizing pneumococcal pneumonia can behave similarly, as exemplified by our first case. Other reported causes include Klebsiella pneumonae, Hemophilus influenzae, Escherichia coli, group A streptococci, Mycobacterium tuberculosis, Pneumocystis carinii, and hydrocarbon-induced pneumonia.

The relationship between positive pressure ventilation and pneumatocele formation is uncertain. All three patients required mechanical ventilation with endotracheal intubation. Pneumatoceles developed after mechanical ventilation in cases 1 and 2, but they were present prior to mechanical ventilation in case 3. The pneumatoceles increased in size during positive pressure ventilation. The proposed check-valve mechanism of pneumatocele formation suggests that positive pressure would lead to distention of these spaces, and we advocate minimizing inspiratory positive pressure and positive expiratory pressure.

Spontaneous pneumothorax occurred four times, (two each in cases 1 and 3). Two of the pneumothoraces occurred prior to radiographic evidence of pneumatoceles and two were accompanied by pneumatoceles on chest roentgenogram. Three episodes occurred during positive pressure ventilation. The one pneumothorax that occurred prior to intubation and ventilation also preceded radiologic evidence of pneumatoceles. From these findings it is conceivable that pneumothoraces could result from rupture of pneumatoceles, especially in the course of positive pressure ventilation.

The majority of pneumatoceles do not cause any apparent symptoms and resolve spontaneously over weeks to months without clinical or radiographic sequelae, although persistence up to three years has been described. However, pneumatoceles can enlarge compressing the adjacent lung and the mediastinum. This may cause sudden respiratory or cardiovascular compromise and possibly fatal outcome as was demonstrated in two of our patients. In such a case, rapid relief of the compression is required. Two methods of achieving this have been described: intracavitary suction, or thoracotomy and surgical excision. Those who advocate excision consider that it is more direct and precise, and cite the danger of persistent bronchopleural fistula developing with intracavitary suction. However, chest tube placement directly into the pneumatocele is a relatively simple bedside procedure which we and others have performed successfully, and can avoid the need for a more complicated surgical procedure in acutely ill patients. In an emergency situation, this procedure may be lifesaving, as seen in case 1.

In summary, we describe three adult patients who developed pneumatoceles in the course of bacterial pneumonia. Intracavitary suction was applied when enlarging pneumatoceles caused mediastinal shift and cardiopulmonary compromise. The pneumatoceles were successfully decompressed. In the critically ill patient who is too ill to undergo surgery and requires rapid relief of an enlarging pneumatocele, intracavitary suction appears to be a useful means of treatment.

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**REFERENCES**

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