Management of Parapneumonic Effusions

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Despite the advent of potent antibiotics, bacterial pneumonia still results in morbidity and mortality in the American population. It is estimated that the annual incidence of bacterial pneumonia exceeds 1.2 million, with a mortality that exceeds 70,000 each year. Bacterial pneumonias have an associated pleural effusion (parapneumonic effusion) approximately 40% of the time. Most of these parapneumonic effusions resolve without operative intervention, but about 10% of these parapneumonic effusions require a tube thoracostomy for their resolution, and these are designated as complicated parapneumonic effusions. The morbidity and mortality of these patients with parapneumonic effusions, particularly when the effusions are complicated, are greater than those of patients without pleural effusions. The purpose of this article is to review the natural history and rational management of parapneumonic effusions in patients, with the goal of minimizing morbidity and mortality.

**NATURAL HISTORY OF PARAPNEUMONIC EFFUSIONS**

The evolution of a parapneumonic effusion can be divided into three stages. The first stage is the exudative stage, which is characterized by the rapid outpouring of sterile pleural fluid into the pleural space in response to inflammation of the pleura. The associated pneumonic process is usually contiguous with the visceral pleura, resulting in increased permeability of the capillaries of the visceral pleura. The pleural fluid in this stage is characterized by a relatively low WBC count and lactic dehydrogenase (LDH) level, and a normal glucose level and pH.

The second stage is the fibropurulent stage, which is characterized by the invasion of the pleural fluid by bacteria. In this stage, the pleural fluid begins to look like pus since it contains fibrin, cellular debris, and great numbers of polymorphonuclear leukocytes. During this stage, ever-increasing amounts of fibrin are deposited as continuous sheets that cover both the visceral and parietal pleura. As this stage evolves, there is a progressive tendency toward loculation of the fluid and the formation of limiting membranes. This loculation prevents extension of the pleural infection, but it makes drainage of the pleural space difficult.

The last stage is the organization stage in which fibroblasts grow into the exudate from both the visceral and parietal pleural surfaces and produce an inelastic membrane called the "pleural peel." The inelastic pleural peel encases the lung and renders it virtually functionless. The exudate is thick, and if the patient has remained untreated, the fluid may drain spontaneously through the chest wall (empyema necessitatis) or into the lung and produce a bronchopleural fistula.

In view of the natural history of parapneumonic effusions, one would like to start tube drainage of the pleural space as early as possible in those patients who will require this procedure, because drainage becomes progressively more difficult the longer it is delayed. On the other hand, the great majority of patients with parapneumonic effusions will not require a tube thoracostomy. Therefore, the goal of the management of parapneumonic effusions in patients is to diagnose, as early as possible, the conditions of those patients who will require a tube thoracostomy. In general, the conditions of patients in stage 1 can be managed with antibiotics alone, while patients in stage 2 or 3 will require a tube thoracostomy.

**INITIAL MANAGEMENT OF PARAPNEUMONIC EFFUSIONS**

When a patient with acute bacterial pneumonia is initially examined, a physician should determine whether or not a parapneumonic effusion is present. This is most easily done from the lateral chest x-ray film. If the posterior costophrenic angles are not blunted, a physician can assume that there is not a clinically significant pleural effusion. If the posterior costophrenic angles are blunted or if the diaphragm is obscured by the infiltrate, then a lateral decubitus chest roentgenogram should be obtained, with the side in question in a downward position. The amount of pleural fluid can be semiquantitated on the decubitus film by measuring the distance between the inside of the chest wall and the bottom of the lung. If the amount of fluid measures less than 10 mm, one can assume that the effusion is not clinically significant; therefore, a thoracentesis is not indicated. In a recent series, 53 patients had such small parapneumonic effusions; in all of these patients, the pneumonia and pleural effusion cleared...
with only antibiotics, and no residual remained. If the thickness of the fluid is greater than 10 mm on the decubitus x-ray film, a diagnostic thoracentesis should be performed since it is impossible without thoracentesis to separate those effusions that are complicated from those that are not complicated. At times when the patient is initially examined, the pleural fluid will already be loculated and will not layer on the decubitus roentgenogram. In such instances, loculated pleural fluid is usually suggested by the chest roentgenogram. The diagnosis of loculated pleural fluid can be facilitated by ultrasonic techniques. Not only can the presence or absence of fluid be demonstrated by ultrasound, but the location of the fluid can be pinpointed for the subsequent diagnostic thoracentesis.

With the diagnostic thoracentesis, 30 to 50 mL of pleural fluid is withdrawn into a syringe that contains heparin sodium. The fluid is examined grossly for color, turbidity, and odor, and aliquots are sent for determination of the pleural fluid glucose, LDH, and protein levels, pH, amylase level, and differential and total WBC counts. Samples of pleural fluid are also sent for bacterial cultures (both aerobic and anaerobic) and for Gram’s stain of the pleural fluid, as well as for cytologic studies and mycobacterial and fungal smears and cultures, if clinically indicated.

The purpose of this initial pleural fluid examination is to determine whether or not a tube thoracostomy should be started immediately. As much pleural fluid as possible would like to start a tube thoracostomy as early as possible in those patients who will eventually require it; however, since a tube thoracostomy is painful and results in at least some morbidity, it is preferable to start it in only those patients who will require a tube thoracostomy. In general, parapneumonic effusions that have reached stage 2 require a tube thoracostomy, i.e., these are complicated parapneumonic effusions. The administration of appropriate antibiotics to patients whose conditions are classified in stage 1 will halt the evolution of the parapneumonic effusion and will obviate the necessity of tube drainage of the pleural space. As a parapneumonic effusion evolves, the level of the pleural fluid LDH becomes higher and higher. Eventually, the pleural fluid pH begins to fall; later, the level of the pleural fluid glucose begins to fall, and Gram’s stain of the pleural fluid becomes more and more positive. At the present time, I recommend the immediate insertion of chest tubes if any one of the following four conditions are met: (1) gross pus is obtained with the thoracentesis; (2) Gram’s stain of the pleural fluid is positive for organisms; (3) the pleural fluid glucose level is less than 40 mg/dL; and (4) the pleural fluid pH is below 7.00. Gram’s stain will be a more sensitive measurement if an aliquot of pleural fluid is subjected to centrifugation, and the sediment is stained. To use the pleural fluid pH measurement, the pleural fluid must be collected anaerobically and placed on ice during its transfer to the laboratory. The pH must be determined with a blood gas analyzer; paper pH strips are not sufficiently accurate. The use of the pleural fluid pH and glucose level as guides to the placement of chest tubes only pertains to parapneumonic effusions. Pleural effusions with other causes, e.g., malignancy, tuberculosis, or rheumatoid disease, may have a low pleural fluid pH and/or glucose level, but the placement of chest tubes in these conditions should not be dictated by the pleural fluid pH or glucose level.

If, on the initial evaluation of the pleural fluid, the pH is above 7.20, the LDH level is below 1,000 IU/L, and the glucose level is above 60 mg/dL, and Gram’s stain is negative, the parapneumonic effusion is classified in stage 1, and no further diagnostic or therapeutic measure needs to be directed toward the effusion. In particular, serial therapeutic thoracenteses are not necessary.

Patients who have an initial pleural fluid pH between 7.00 and 7.20 or an LDH level above 1,000 IU/L, and whose conditions do not meet any of the previously mentioned criteria for the placement of chest tubes, present a special problem. Since some of these patients will need chest tubes, while other patients will not, each patient’s condition should be considered individually. If the patient has a large effusion and the pH is close to 7.00, the patient probably should have chest tubes inserted. Alternatively, if the effusion is small and the pH is close to 7.20, the patient probably will not require a tube thoracostomy. In borderline cases, serial thoracenteses at 12- to 24-hour intervals are useful. If the pleural fluid pH and glucose level tend to increase and the pleural fluid LDH level tends to fall with serial thoracenteses, the patient is able to handle the parapneumonic effusion, and a tube thoracostomy probably will not be necessary. In contrast, if the pleural fluid pH and glucose level fall and the LDH level increases, tube thoracostomy drainage of the pleural space should be started.

The pleural fluid WBC count is not used in the decision process to start a tube thoracostomy. Several series have now demonstrated that, in patients with parapneumonic effusions, there is virtually no relationship between the pleural fluid WBC count and the necessity for tube drainage of the pleural space. Indeed, I have seen several patients in whom the pleural fluid WBC counts exceeded 100,000/cu mm; however, in these patients, the parapneumonic effusions resolved completely with only the administration of antibiotics. The pleural fluid differential WBC count should always be noted since a preponderance of mononuclear cells suggests a different diagnosis, eg, pulmonary embolus with effusion, tuberculous pleuritis, or malignant pleural effusion.

The initial antibiotic therapy for patients with parapneumonic effusions should be based on Gram’s stain of the sputum, as it is with any patient with bacterial pneumonia. Since the antibiotic levels in the pleural fluid are comparable with those in the serum, the dose of antibiotics is not increased in patients with parapneumonic effusions. If a pleural infection is present, it should be treated with a tube thoracostomy rather than by increased doses of antibiotics.

**TUBES IN THORACOSTOMY FOR COMPLICATED PARAPNEUMONIC EFFUSIONS**

The goal of a tube thoracostomy in patients with complicated parapneumonic effusions is to completely drain the pleural space and to allow the underlying lung to reexpand. Since the pleural fluid is frequently viscous and, at times, has a tendency to clot, a thoracostomy tube, of as large a diameter as possible, should be inserted. The positioning of the tube is important, because to have complete drainage, it must be in the most dependent area of fluid accumulation, which most commonly is the posterior costophrenic sulcus. Of course, if the fluid is loculated, the chest tube should be placed in the most dependent portion of the loculus. The tube should be attached to underwater-seal drainage. To my knowledge, there is no evidence that the application of subatmospheric pressure is beneficial, and since its application may lead to reexpansion pulmonary edema, underwater-seal drainage is preferred.

The adequacy of pleural drainage should be assessed daily by examination of the patient’s clinical status, chest roentgenograms, and measurements of the chest tube drainage. Adequate drainage is characterized by defervesc-
cence and general clinical improvement in conjunction with radiologic improvement. If the pleural fluid is loculated, more than one thoracostomy tube may be necessary. If the drainage with the chest tubes is unsatisfactory, some investigators advocate the intrapleural injection of streptokinase. The theory behind this maneuver is that the streptokinase will convert the inactive plasminogen in the pleural space to the active fibrinolytic enzyme, plasmin, which digests the fibrin and minimizes loculation. The usual dose of streptokinase is 250,000 units intrapleurally, repeated daily for up to five days. Although, to my knowledge, there have been no controlled studies that demonstrate the efficacy of this maneuver, in my experience, it has been an impressive adjunct to tube thoracostomy in some patients.

The chest tubes should be left in place until the purulence of the drainage disappears, and the amount of drainage decreases to less than 50 mL/day. At that time, the chest tubes should be removed, and the patient should be observed for recurrence of toxic effects. Chest tubes that cease to function should be removed expeditiously since these are serving no useful purpose and can serve as conduits for pleural infection.

In some patients, closed drainage is unsuccessful because the lung does not reexpand to fill the pleural cavity and/or locules of infected pleural fluid remain. Failure of closed drainage is usually caused by a delay in starting initial drainage. With the delay, the pleural fluid becomes loculated. In addition, a thick peel forms over the visceral pleura, which prevents the lung from reexpanding. If the lung has not expanded after seven to 14 days of closed drainage, either an open thoracotomy drainage procedure or a decortication should be considered.

With open thoracotomy drainage, a short segment of the rib is removed at the most dependent part of the cavity, and a large drainage tube is inserted into the pocket. Alternatively, the tube may be omitted, and a pleural-cutaneous fistula may be maintained by a flap of skin called an Eloesser flap. A colostomy bag can be placed over the opening in the chest to capture the drainage. The pleural cavity gradually heals from within, via the formation of granulation tissue. If an Eloesser flap is used, the cavity is packed with gauze. The advantages of open drainage over closed drainage are that the drainage is more complete with the larger opening and the patient is freed from the chest tube bottles. With open thoracotomy drainage, the cavity is exposed to atmospheric pressure, and it is important to ascertain before the procedure that the lung will not collapse. This can be done by exposing the chest tube to atmospheric pressure for several hours and by obtaining a repeated chest roentgenogram. If the lung collapses, the open procedure should be delayed until the lung no longer collapses.

Decortication is the removal of the inflammatory peel that covers the visceral pleura of the affected lung. This procedure allows for the underlying lung to reexpand and obliterate the pleural space. During the early weeks of a pleural infection, the visceral peel is adherent to the underlying pleura, and it is difficult to remove. For this reason, many surgeons perform decortication only after six weeks or longer. However, it has been shown that decortication can successfully be performed much earlier. In contrast with the relatively minor open-drainage procedure, decortication is major surgery and should never be performed on a seriously ill patient. However, in relatively healthy patients, decortication is the treatment of choice when closed drainage fails, since it allows the patient to return to a normal life much sooner than does open drainage.

Some thoracic surgeons recommend decortication in all cases in which a thick pleural peel remains after either closed or open drainage of a pleural infection. Since the peel frequently improves spontaneously in the months after the drainage, it is recommended that decortication be delayed for at least six months if the infection has been controlled and the lung has reexpanded. After this time, decortication should be performed only if patients are limited in their exercise capacity and if close examination of their pulmonary status suggests that the procedure will improve their pulmonary function.

Nonproprietary Name and Trademark of Drug

Streptokinase—Streptase.

References