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MANAGEMENT OF PARAPNEUMONIC EFFUSIONS

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The annual incidence of bacterial pneumonia in the United States is estimated at 4 million and approximately 20% of those cases require hospitalization.[32] The incidence of parapneumonic effusion in patients hospitalized with pneumonia is about 40%.[25] The morbidity and mortality rates in patients with pneumonia and pleural effusions are greater than in patients with pneumonia alone. In one recent study,[16] the relative risk of mortality in patients with community-acquired pneumonia was 7.0 times higher for patients with bilateral pleural effusions and 3.4 times higher for patients with unilateral-pleural effusion of moderate or greater size compared with other patients with community-acquired pneumonia. Delay in instituting proper therapy for effusions is responsible for much of the increased morbidity and mortality.

DEFINITIONS

Any pleural effusion associated with bacterial pneumonia, lung abscess, or bronchiectasis is a parapneumonic effusion.[26] An empyema, by definition, is pus in the pleural space. Pus is thick, purulent-appearing pleural fluid. A complicated-parapneumonic effusion is a parapneumonic effusion for which tube thoracostomy is necessary for resolution. A loculated parapneumonic effusion is a parapneumonic effusion that is not free-flowing. A multiloculated parapneumonic effusion is a loculated-parapneumonic effusion with more than one compartment.

HISTORY

Writings on the treatment of empyema date back to around 500 BC, when Hippocrates recommended treating empyema with open drainage.[1] At that early time, Hippocrates recognized that the prognosis of the patient depended on the characteristics of the fluid. He wrote the following: "Those cases of empyema are treated by incision or the cautery, if the water flows rapidly all at once certainly prove fatal. When empyema is treated, either by the incision or the cautery, if pure and white pus flows slowly from the wound, the patients recover."

The treatment of empyema remained essentially unchanged from the time of Hippocrates until the middle of the nineteenth century. At that time, Trousseau[51] in France and Bowditch[8] in the United States popularized the use of thoracentesis and demonstrated that open drainage was not necessary in many cases. The next advance was provided by Hewitt,[17] who described a method of closed drainage of the chest in which a rubber tube was placed into the empyema cavity through a cannula. He was the first to use the water-seal for chest tubes.

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When dealing with an empyema, the management of the empyema cavity poses a significant problem. In the 1890s, articles by Estlander[11] and Schede[38] described thoracoplasty as a means of obliterating the empyema cavity. Thoracoplasty involves the resection of the ribs, intercostal muscles, and parietal pleural peel over the cavity. The remaining defect is covered by the few remaining muscles, the scapula, and the subcutaneous tissue and skin. At approximately the same time, Fowler[13] and Beck[5] initially described decortication. With decortication, all pus is removed from the pleural space and all the fibrous tissue is removed from the visceral and parietal pleura.

Hippocrates had recognized before the birth of Christ that open drainage procedures were dangerous if the empyema fluid was not thick.[1] Paget[34] again emphasized in 1896 that open drainage should not be instituted for empyema before at least the fifteenth day of the illness. Yet, by World War I, open drainage had become the accepted treatment for all cases of parapneumonic effusions. During World War I, there was a high incidence of parapneumonic effusions in American soldiers and the treatment of all such patients with open drainage had disastrous results. The United States Surgeon General, in 1919, found that the average mortality of individuals with pleural infections was 30.2% and, in some hospitals, the mortality rate was as high as 70%.[15] The explanation for the high mortality rate was that the open drainage procedure was performed too early, resulting in collapse of the underlying lung. *Streptococcus hemolyticus* was responsible for many of those cases and that organism characteristically produces a large nonloculated pleural effusion.[33]

An empyema commission headed by Evarts Graham was formed to address the high mortality rates. Graham[14] demonstrated that when dogs with experimental empyema were treated by the early insertion of open chest tubes, the mortality rate was higher and deaths occurred earlier than in dogs that were not treated with chest tubes. The empyema commission made the following recommendations: (a) The pleural fluid should be drained, but one must avoid an open pneumothorax in the acute-pneumonic phase; (b) care should be taken to avoid a chronic empyema by rapid sterilization and obliteration of the infected cavity, and (c) careful attention should be paid to the nutrition of the patient.[10] When those guidelines were followed, the mortality from streptococcal empyema secondary to influenza fell to 4.3%.[44] Those guidelines provide the basis for the treatment of parapneumonic effusions today.

NATURAL HISTORY OF PARAPNEUMONIC EFFUSIONS

The evolution of a parapneumonic pleural effusion can be divided into three stages—the exudative, the fibropurulent, and the organization stage.[3] The three stages are not sharply defined, but rather form a continuous spectrum. The first stage is the exudative stage, characterized by the rapid outpouring of fluid into the pleural space. The pleural fluid probably originates in the interstitial spaces of the lung.[52] In the exudative stage, the pleural fluid is not loculated and is characterized by a low white blood cell count and lactic acid dehydrogenase (LDH) level and a normal glucose level and pH.[23] If appropriate antibiotic therapy is instituted at this time, the pleural effusion does not progress to the fibropurulent stage and tube thoracostomy is not required.[23]

The second stage is the fibropurulent stage, characterized by the accumulation of large amounts of pleural fluid with many polymorphonuclear leukocytes, bacteria, and cellular debris. As the stage progresses, the pleural fluid pH and glucose levels become progressively lower and the LDH level progressively higher. The pleural fluid white blood cell count is frequently lower than one would anticipate from looking at the thickness of the fluid; fibrin and cellular debris rather than intact white cells account for the thickness and opacity of the fluid. Fibrin is deposited in a continuous sheet, covering the visceral and parietal pleura. As the stage progresses, there is a tendency toward loculation and the formation of limiting membranes. The loculations contain the infected pleural fluid within compartments, but make drainage of the pleural space increasingly difficult.

The third stage is the organization stage, characterized by the ingrowth of fibroblasts into the thick exudate from the visceral and parietal pleural surfaces to produce an inelastic membrane called *the pleural peel.* The peel prevents the underlying lung from expanding; decortication is required to cure the patient at this stage. In the third stage, the fluid is usually multiloculated and thick. If the patient is not treated, the fluid may drain

spontaneously through the chest wall *(empyema necessitatis)* or into the lung, producing a bronchopleural fistula. If a bronchopleural fistula develops in this situation, immediate drainage of the pus from the pleural space is imperative. Otherwise, the pus from the pleural space will enter the tracheobronchial tree and produce an overwhelming pneumonia.[23]

CLASSIFICATION OF PARAPNEUMONIC EFFUSIONS

When a patient with pneumonia is first evaluated, one should attempt to determine whether or not a pleural effusion is present. A lateral radiograph should be obtained to screen for the presence of a pleural effusion. If both diaphragms cannot be seen throughout their entirety on the lateral chest radiograph, decubitus chest radiographs should be obtained. The amount of free-pleural fluid can be semiquantitated by measuring the distance between the inside of the chest wall and the outside of the lung. If that distance (the thickness of the pleural fluid) is greater than 10 mm, thoracentesis should be performed. The only way to determine whether invasive measures are necessary for the treatment of a parapneumonic effusion is to examine the pleural fluid.[25] One should perform the thoracentesis as soon as the pleural effusion is recognized because a free-flowing, easily treated parapneumonic effusion can progress to a multiloculated parapneumonic effusion within hours.[35]

It is important to realize that not all parapneumonic effusions and not all complicated parapneumonic effusions are the same. The classification outlined in Table 1 was developed to assist the practicing physician in the initial care of patients with parapneumonic effusions. It is based on the amount of fluid, the gross and biochemical characteristics of the pleural fluid, and whether or not the pleural fluid is loculated. As one moves down on Table 1, the treatment of the parapneumonic effusion becomes more difficult and increasingly invasive procedures are indicated.[23]

When a patient with a parapneumonic effusion more than 10 mm in thickness on the decubitus radiograph is evaluated, one must perform thoracentesis to place the patient in one of the six lower categories. Because it is not known whether an aggressive approach to the pleural fluid collection is indicated until after the pleural fluid has been analyzed, the question is whether a diagnostic or therapeutic thoracentesis originally should be performed.

Table 1. A CLASSIFICATION AND TREATMENT SCHEME FOR PARAPNEUMONIC EFFUSIONS AND EMPYEMA

Adapted from Light RW: Pleural Diseases, ed 3, Baltimore, MD, Williams and Wilkins, 1995; with permission.

I recommend that a therapeutic thoracentesis be performed in all patients whose pleural fluid is more than 10 mm thick on the decubitus view. If there is no reaccumulation of fluid, one need not worry about the parapneumonic effusion. Performance of the therapeutic thoracentesis will also delineate whether the pleural fluid is loculated.

The pleural fluid should be sent for Gram's stain and bacterial culture, white blood cell count and differential, and for determination of its level of glucose, LDH, and pH. If the pH is determined, it must be done with the same care as is an arterial pH level. The fluid should be collected in a heparinized syringe and placed in ice for its transfer to the blood gas laboratory. The pH cannot be determined accurately with a pH meter or tape. If the pH is low (< 7.2) and the glucose is close to 100 mg/dL and there is no large increase in pleu-

ral fluid LDH, one should measure the arterial blood pH before a tube thoracostomy is performed; if systemic acidosis is present, the pleural fluid pH will be acidotic.[26] The differential cell count is important because, if polymorphonuclear leukocytes do not predominate, an alternate diagnosis must be sought.

Class 1—Nonsignificant Parapneumonic Effusion

Patients with class 1 parapneumonic effusions have free-flowing fluid that is less than 10 mm thick on the decubitus chest radiograph. Individuals with class 1 effusions should not be subjected to thoracentesis because treatment with appropriate antibiotics almost always resolves the effusion.[25] In addition, a thoracentesis is more difficult in patients with a small amount of pleural fluid. If a patient with a Class 1 effusion subsequently develops a larger pleural effusion, a diagnostic thoracentesis should be performed.

Class 2—Typical Parapneumonic Effusion

Patients with a typical parapneumonic effusion have pleural fluid that is free-flowing, with a thickness of greater than 10 mm on the decubitus radiograph. In addition the pleural fluid glucose level is greater than 40 mg/dL, the pleural fluid pH is higher than 7.2, the pleural fluid LDH level is below 1000 U/L, and the bacterial smears and cultures are negative. As a parapneumonic effusion evolves from the exudative stage to the fibropurulent stage, the LDH level becomes progressively higher, whereas the pH and glucose level become progressively lower. When glucose and pH are greater than 40 mg/dL and 7.2, respectively, and the LDH is less than 1000 U/L (or three times the upper normal limit for serum), the effusion is early in the exudative stage and no invasive procedure is necessary.[25] If the effusion rapidly recurs after the initial therapeutic thoracentesis or if the patient remains toxic with significant pleural fluid, then a repeat therapeutic thoracentesis should be performed.

Class 3—Borderline Complicated Parapneumonic Effusion

Patients with class 3 parapneumonic effusions have negative bacterial smears and cultures and a glucose level above 40 mg/dL, but the pH is between 7 and 7.2, the LDH level is above 1000 U/L, or the pleural fluid is loculated. The relatively low pH, relatively high LDH, and the loculation all indicate a high level of inflammation in the pleural space. Some class 3 pleural effusions resolve with no invasive procedure but others do not. Our philosophy has always been that we would rather insert a few too many chest tubes than insert some chest tubes too late.

If the patient has an earlier therapeutic thoracentesis that demonstrated the pleural fluid was loculated and the size of the loculated effusion did not decrease with antibiotic treatment or the patient remained toxic, then a small chest tube should be inserted. Thrombolytics can be injected through the small chest tube in an effort to break down the loculations.

If the fluid is not loculated, a repeat therapeutic thoracentesis is indicated if the fluid reaccumulates. If the pleural fluid pH and glucose show a tendency to decrease and the pleural fluid LDH shows a tendency to increase, then a small chest tube should be inserted. If the pleural fluid pH increases above 7.2 and the pleural fluid glucose increases above 40 mg/dL, then resolution is expected and additional therapeutic thoracenteses are not indicated.

Class 4—Simple-Complicated Parapneumonic Effusion

Patients with class 4 parapneumonic effusions have a pleural fluid pH less than 7, a pleural fluid glucose less than 40 mg/dL, and a positive Gram's stain or culture. The pleural fluid does not look like pus and it is not loculated. Patients with class 4 parapneumonic effusions should be treated with some form of invasive therapy because many will not resolve with antibiotic therapy alone.

If the patient's initial thoracentesis was therapeutic, the patient can be managed with either a repeat therapeutic thoracentesis or the placement of a chest tube, should the effusion recur. A reasonable approach is to perform a repeat serial therapeutic thoracentesis if the pH, LDH, and glucose levels in the pleural fluid are improving. Alternatively, if the pH and the glucose levels in the pleural fluid are not improving, then tube thoracostomy should be performed.

If only a diagnostic thoracentesis was per-

formed originally, then the patient should be treated with either a therapeutic thoracentesis or a tube thoracostomy. It appears that such effusions can be managed with relatively small chest tubes (8.3–16F) inserted percutaneously.[21, 42] The advantage of the smaller tube over a larger one is that its insertion is easier and less painful and its presence is less uncomfortable to the patient.

Class 5—Complex Complicated Parapneumonic Effusion

Patients with class 5 parapneumonic effusion meet the criteria for class 4 parapneumonic effusions but the fluid is also loculated. Such patients should be treated with a small chest tube plus intrapleural-thrombolytic agents. Without the thrombolytic therapy, the pleural space cannot be drained completely. If complete drainage of the pleural space is not accomplished after one or two doses of thrombolytic therapy, then more aggressive therapy, such as the breakdown of the loculations with thoracoscopy or thoracotomy with decortication, should be performed.

Class 6—Simple Empyema

Patients with class 6 parapneumonic effusions have pleural fluid that is frank pus and the pus is either free-flowing or confined to a single loculus. Such patients should be treated with a relatively large (~28—36F) chest tube because the thick pus is likely to obstruct a smaller one. Patients who have class 6 parapneumonic effusions frequently have a thick peel over the visceral pleura that prevents the underlying lung from expanding. If a sizable empyema cavity remains after several days of chest tube drainage, consideration should be given to performing a decortication to eradicate the empyema cavity.

Class 7—Complex Empyema

Patients with class 7 parapneumonic effusions have frank pus in their pleural space that is multiloculated. Although such patients initially should be treated with large chest tubes and intrapleural thrombolytic agents, more invasive measures such as thoracoscopy with the breakdown of adhesions or thoracotomy with decortication are necessary in the majority.[43] If the drainage of the pleural space is unsatisfactory or a large empyema cavity remains after several days, either thoracoscopy or thoracotomy should be considered.

TREATMENT MODALITIES FOR PARAPNEUMONIC EFFUSIONS

Antibiotic Therapy

Patients with pneumonia and pleural effusion should be treated with antibiotic agents. The initial antibiotic selection is based on whether the pneumonia is community- or hospital-acquired and on the severity of illness. The initial antibiotic selection and the dose are not influenced by the presence or absence of a pleural effusion. Most antibiotic agents are present in pleural fluid at levels that are comparable with those in serum.[23] Aminoglycosides, however, appear to penetrate poorly into purulent pleural fluid.[50]

Hospitalized patients with community-acquired pneumonias that are not severe should be treated with second- or third-generation cephalosporins or a β-lactam/β-lactamase inhibitor. If infection with *Legionella* spp is likely, a macrolide antibiotic agent should be added. Patients with severe community-acquired pneumonia should be treated with a macrolide plus a third-generation cephalosporin such as ceftazidime or cefoperazone. Patients with hospital-acquired pneumonia should be treated with a third-generation cephalosporin with anti-*Pseudomonas* activity. If *Staphylococcus aureus* infection is suspected, either nafcillin or vancomycin should be administered.[32]

Therapeutic Thoracentesis

Therapeutic thoracentesis was first proposed as treatment for parapneumonic effusions in the middle of the nineteenth century.[8, 51] In 1962, the American Thoracic Society recommended repeated thoracenteses for nontuberculous empyemas in the early exudative phase.[3] Recently, however, therapeutic thoracentesis has received relatively little attention as a treatment for parapneumonic effusions.

Therapeutic thoracentesis is the least invasive of the invasive treatment modalities for parapneumonic effusions. As discussed ear-

lier, a logical approach to patients with parapneumonic effusions is to do a therapeutic thoracentesis when the effusion is first recognized. If the fluid subsequently recurs, a second therapeutic thoracentesis should be performed. If the pleural fluid again recurs and the pleural fluid biochemical parameters were worsening at the time of the second thoracentesis, a chest tube should be inserted.

A few recent studies suggested there is a role for therapeutic thoracentesis in the management of patients with parapneumonic effusions. Sasse and coworkers[36] reported that therapeutic thoracentesis was at least as good as chest tube placement in the treatment of early pleural infections in a rabbit model of empyema. Storm and coworkers[44] reported that 48 of 51 patients (94%) with empyema (purulent pleural fluid or positive microbiologic studies on the pleural fluid) were treated successfully with daily thoracentesis. Mandal and associates[28] reported that 28 of 111 patients (25%) with bacterial empyema (purulent exudate or positive culture) were treated successfully with serial therapeutic thoracentesis and antibiotic agents. Ferguson et al[12] reported that 19 of 46 patients (41%) with empyema (opaque fluid in the pleural space with the cloudiness attributable to neutrophils or organisms) were treated successfully with repeated thoracentesis.

There have been no controlled studies comparing therapeutic thoracentesis with small tube thoracostomy in the treatment of patients with class 3 or class 4 parapneumonic effusion.

Tube Thoracostomy

When a chest tube is used to treat a parapneumonic effusion, it should be placed in a dependent part of the effusion. Failure of tube thoracostomy in the treatment of parapneumonic effusions is frequently attributable to placing the tube in the wrong position.[21] Initially, the chest tube should be connected to an underwater-seal drainage system with suction. If the visceral pleura is covered with a fibrinous peel, the application of negative pressure may facilitate the expansion of the underlying lung and hasten obliteration of the empyema cavity.

In the past, it was recommended that relatively large (26–36F) chest tubes be used in the treatment of parapneumonic effusions based on the belief that smaller tubes would become obstructed with the fluid. It appears, however, that class 3, 4, and 5 parapneumonic effusions can be managed with smaller tubes. Forty-one of 53 patients (77%) in two recent series were managed successfully with smaller chest tubes (8.3–16F).[21, 42] Those results are at least as good as those reported in recent series in which larger tubes were used.[2, 4] The small chest tubes in the two series[21, 42] were inserted percutaneously by an interventional radiologist. It is likely that the excellent results are related to accurate placement of the catheter. If the pleural fluid is frank pus, we still prefer to use a large chest tube.

Successful closed-tube drainage of complicated parapneumonic effusions is evidenced by improvement in the clinical and radiologic status within 24 hours. If the status of the effusion is not clear from the posteroanterior and lateral radiograph, chest CT scan can provide valuable information. If the patient is not improving, either the drainage is inadequate or the patient is receiving the wrong antibiotic agent. Inadequate drainage can be caused by poor positioning of the chest tube, obstruction of the chest tube, or loculated pleural fluid. If the drainage is incomplete because of loculations, thrombolytic agents can be administered intrapleurally or the patient can be subjected to thoracoscopy, with the breakdown of the loculations.

If the patient responds satisfactorily, how long should the chest tube be left in place? Although there has been surprisingly little written on the subject, we recommend that the chest tubes should be left in place until the volume of the pleural drainage is under 50 mL/24 hours and until the draining fluid becomes clear yellow.[23] If the chest tube ceases to function (no fluctuation with respiratory efforts), it should be removed because it serves no useful purpose and can be a conduit for pleural suprainfection. On occasion, purulent drainage continues from the chest tube despite improvement clinically and radiologically. In that situation, one must decide whether a more aggressive approach—e.g., decortication—is indicated. That decision can be aided by radiographs obtained after the injection of contrast material into the pleural space through the chest tube.[40] If only a tube tract is demonstrated, the chest tube can be withdrawn over several days while the tract is allowed to fill in with granulation tissue. When a larger (>100 mL) cavity is demonstrated, consideration should be given

to performing an empyemectomy. If one elects to continue with chest tube drainage, progress of the empyema cavity can be assessed by repeated contrast studies through the tube at weekly intervals.

Intrapleural Thrombolytic Therapy

The role of thrombolytic agents in the management of loculated-parapneumonic effusions remains to be determined. The theory behind their use is that loculations in the pleural space are produced by fibrin membranes; if a thrombolytic agent is injected into the pleural space, then it may dissolve the fibrin membranes and facilitate drainage of the pleural space.

In the past 3 years, there have been at least five *uncontrolled* studies,[7, 20, 22, 30, 48] each with more than 20 patients, that have concluded that thrombolytic agents are useful in the management of patients with loculated pleural effusions. Both streptokinase [7, 20, 22, 48] and urokinase[7, 30, 48] have been used in that situation. In several reports, the basis for the endorsement of the intrapleural thrombolytics was the increase in pleural fluid drainage after their administration.

One report compared the efficacy of the two thrombolytic agents and concluded that both were useful adjuncts in the management of complicated parapneumonic effusions, but that urokinase was the thrombolytic agent of choice because there is a greater chance of dangerous allergic reactions to streptokinase.[7]

Each agent can be given daily in a total volume of 50 to 100 mL as long as they appear to be facilitating pleural drainage. The usual dose of urokinase is 100,000 U, that for streptokinase is 250,000 U.

Two placebo-controlled studies evaluated thrombolytic agents in the management of patients with complicated parapneumonic effusion and they reached different conclusions. In the first study, streptokinase, 250,000 U daily, was compared with no thrombolytic agent in the management of 52 patients with loculated parapneumonic effusion.[9] The study[9] showed that there was more pleural fluid drainage in patients who received streptokinase, but the duration of hospitalization, the need for more invasive surgery, and the mortality rate in the two groups did not differ significantly.[9] In the second study, as yet reported only in abstract form, 128 patients were randomized to receive 50,000 U urokinase, 250,000 U streptokinase, or saline lavage daily. The groups that received the thrombolytic agents had significantly greater radiologic improvement and significantly less need for decortication.[6]

Animal studies of thrombolytic agents have not been definitive. Strange and coworkers[46] showed in one model of experimental empyema that the intrapleural administration of streptokinase increases the amount of pleural fluid but does not decrease the number of adhesions. In a recent study, we demonstrated that, when empyema fluid is incubated with either streptokinase or urokinase, the fluid does not become more liquid. When the empyema fluid is incubated with Varidase, a combination of streptokinase and streptodornase (streptococcal DNAase), however, it becomes completely liquified.[37]

Given the aforementioned conflicting sets of data, what should be the role of thrombolytics in the management of loculated parapneumonic effusions? When one wishes to drain a loculated parapneumonic effusion, a trial of thrombolytic therapy should be tried if there are no contraindications to their use. If the patient improves clinically and radiologically (not just an increase in the amount of pleural-fluid drainage), so much the better. If there is no such improvement after one or two doses, however, the therapy should be abandoned and more invasive measures taken.

Thoracoscopy

Thoracoscopy, with the breakdown of adhesion, is the procedure of choice when tube thoracostomy plus intrapleural thrombolytic therapy fails. With the advent of video-assisted thoracic surgery, video-thoracoscopy is being used increasingly in the situation under discussion. Several papers (reporting uncontrolled studies) in the past few years demonstrated the use of thoracoscopy in the management of complicated parapneumonic effusions.[19, 27, 39, 47] One advantage of the procedure is that the chest tube can be positioned in the most dependent part of the empyema cavity.

All patients who have incomplete drainage of their pleural space with chest tubes plus thrombolytic therapy should be considered to be candidates for thoracoscopy. If thorough debridement and complete lung expansion are achieved with thoracoscopy, chest tubes

usually can be removed in 3 to 5 days.[41] It should be noted, however, that decortication is best done with a full thoracotomy rather than thoracoscopy. It has been recommended that a chest CT scan be obtained prior to thoracoscopy.[41] That examination provides anatomic information about the size and extent of the empyema cavity that guides the planned procedure. A thickened visceral-pleural peel without septations suggests that the empyema may be chronic and probably will not be amenable to thoracoscopic debridement alone.[41]

Thoracotomy with Decortication

With this procedure, which involves a full thoracotomy, all the fibrous tissue is removed from the visceral pleura and all pus is evacuated from the pleural space. Decortication eliminates the pleural sepsis and allows the underlying lung to expand. It is a major thoracic operation requiring a full thoracotomy incision and therefore should not be performed on patients who are markedly debilitated.

Decortication is the procedure of choice in a patient whose pleural sepsis is not controlled by the less invasive measures of tube thoracostomy, intrapleural-thrombolytic agents, and thoracoscopy with the breakdown of adhesions. Decortication should be done as soon as it is recognized that the less invasive therapies are ineffective.[18, 29, 49] In the acute states of a parapneumonic effusion, decortication should be performed only for the control of pleural sepsis, not for pleural thickening. The pleural thickening usually resolves spontaneously over a period of several months.[31] If after 6 months, the pleura remains thickened and the patient's pulmonary function is reduced sufficiently to limit his activities, decortication should be considered.

Open Drainage Procedures

An open drainage procedure is an alternative to decortication in patients who are too debilitated to undergo decortication. Segments of one to three ribs overlying the lower part of the empyema are resected and one or more short, large-bore tubes are inserted into the empyema cavity. The empyema space must be irrigated daily with a mildly antiseptic solution and the drainage can be collected in a colostomy bag. The open-drainage procedure must not be performed unless there is fusion of the visceral and parietal pleura throughout most of the pleural space. If such fusion is not present, the lung will collapse. Performance of the open-drainage procedure too early led to the high mortality from parapneumonic effusions experienced during World War I (discussed earlier).

The advantages of open drainage over closed-tube drainage is that the drainage is more complete and the patient is freed from attachment to a closed-drainage system. When the procedure is undertaken, however, one must realize that the median time for complete healing of the wound after an open-drainage procedure is generally lengthy—about 6 months.

SUMMARY

When a patient with a parapneumonic pleural effusion is first evaluated, a therapeutic thoracentesis should be performed if more than a minimal amount of pleural fluid is present. Fluid obtained at the therapeutic thoracentesis should be gram-stained and cultured and analyzed for glucose, pH, LDH, white blood cells, and differential cell count. If the fluid cannot be drained because of loculations, a chest tube should be inserted and thrombolytic agents administered. If the pleural fluid recurs after the initial therapeutic thoracentesis but the patient is doing well clinically and the initial pleural fluid glucose was greater than 60 mg/dL; the pH, greater than 7.2; the LDH, less than three times the upper normal limit for serum and the cultures are negative; he or she can be observed. If one or more of the aforementioned criteria are not met, a second therapeutic thoracentesis should be performed, with repeat diagnostic evaluations of the pleural fluid. If the fluid recurs a second time, a small chest tube should be placed if the pleural fluid glucose and pH were lower and the LDH higher on the second thoracentesis than on the first thoracentesis.

Patients with loculated-parapneumonic effusions should be treated with tube thoracostomy and thrombolytic agents. If drainage is incomplete, thoracoscopy, with breakdown of adhesions and debridement of the pleural space, is indicated. If thoracoscopy is unsuccessful, then thoracotomy, with decortication,

is indicated unless the patient is too debilitated.

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