9
Empyema Thoracis

Dakshesh H. Parikh

Introduction

Empyema thoracis is defined as an accumulation of pus within the pleural cavity. While the morbidity and mortality of this condition have undoubtedly improved over recent years debate continues regarding the nature and timing of surgical intervention. The management planning and the selection of the most appropriate treatment option require good understanding of the empyema disease process. Moreover, the incidence of empyema thoracis in children has increased significantly in recent years in the western world.[1,2] The incidence of parapneumonic effusion and empyema is approximately 3.3 cases per 100,000 children.[3] It has been estimated that 1 in every 150 children hospitalized with pneumonia will develop an empyema.[4]

Historical Perspective

The natural history of empyema thoracis was described by Hippocrates who realized that drainage could result in cure.[5] He observed that death was likely "if pus that flows after opening was mixed with blood, muddy and foul smelling."[5] He also remarked that if the empyema was drained "with knife or cautery" and the pus was "pale and white" the patient would survive.[6,7] The brilliant French surgeon Pare described evacuation of infected blood from the pleural cavity in the sixteenth century. These early physicians understood the importance of early diagnosis and drainage to avoid mortality in this disease. Browditch[8] described thoracocentesis while Wyman performed the first therapeutic pleural aspiration, describing the method in a letter to Sir William Osler.[9] Commenting on pleural space infections, Osler later wrote "It is sad to think of number of lives which are sacrificed annually by the failure to recognise that empyema should be treated as an ordinary abscess by free incision."[10] Sir William Osler himself underwent a rib resection and drainage of parapneumonic empyema in his home in 1819.

Playfair modified the technique of thoracocentesis to closed tube drainage in 1875.[11] Schede introduced thoracoplasty in 1890.[12] Fowler reported the first decortication in 1893 and soon became apparent that release of the trapped lung was a better procedure in chronic nontuberculous empyema than thoracoplasty.[12–15] A survey carried out by the Surgeon General of the United States of America in 1918 concluded that the high mortality (30%) following rib resection in acute empyema was related to the open pneumothorax.[16] This US commission, headed by Graham, recommended closed drainage for the management of acute empyema. This single act reduced the mortality from empyema dramatically to 5–10%.[17] In addition, it was realized that prevention of chronicity by complete obliteration, sterilization of the empyema cavity, and careful attention to the nutrition was responsible for better results.[18]

Pathogenesis and Basic Science

Etiology

The majority of empyemas in children follow acute bacterial lobar pneumonia.[19] During recovery from viral infections such as chicken pox and measles children are more susceptible to lower respiratory tract infections and therefore empyema. Underlying conditions such as chronic pulmonary diseases, diabetes mellitus, long-term steroid therapy, organ transplantation with associated immune suppression, and recurrent aspiration could predispose the child to empyema.

Empyema may follow secondary infection of a traumatic hemothorax or lung contusion.[20,21] Occasionally a secondary empyema follows a penetrating injury of the chest or after infection in the pleural space following thoracotomy. More commonly secondary empyema in children invariably follows intrathoracic rupture of the esophagus either as a result of a leaking anastomosis or rupture following dilatation of an esophageal stricture.[22] Secondary infection of a sympathetic effusion has been reported after acute pancreatitis and a subphrenic abscess. Pneumonia and empyema have been reported in the postoperative period in children with acute appendicitis. In these cases the empyema probably occurs as a result

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of reduced host resistance, postoperative pneumonia, or local spread of infection through the diaphragm.

Infection in congenital lung abnormalities is a rare cause of empyema in children. In tropics rupture of an amebic liver or lung abscess may cause an infection of the pleural space.[23,24] Similarly, hydatid cysts may rupture into the pleural space or get infected with secondary infection (Fig. 9.1a, b). Approximately 10% of tuberculous effusions present with secondary bacterial infection.

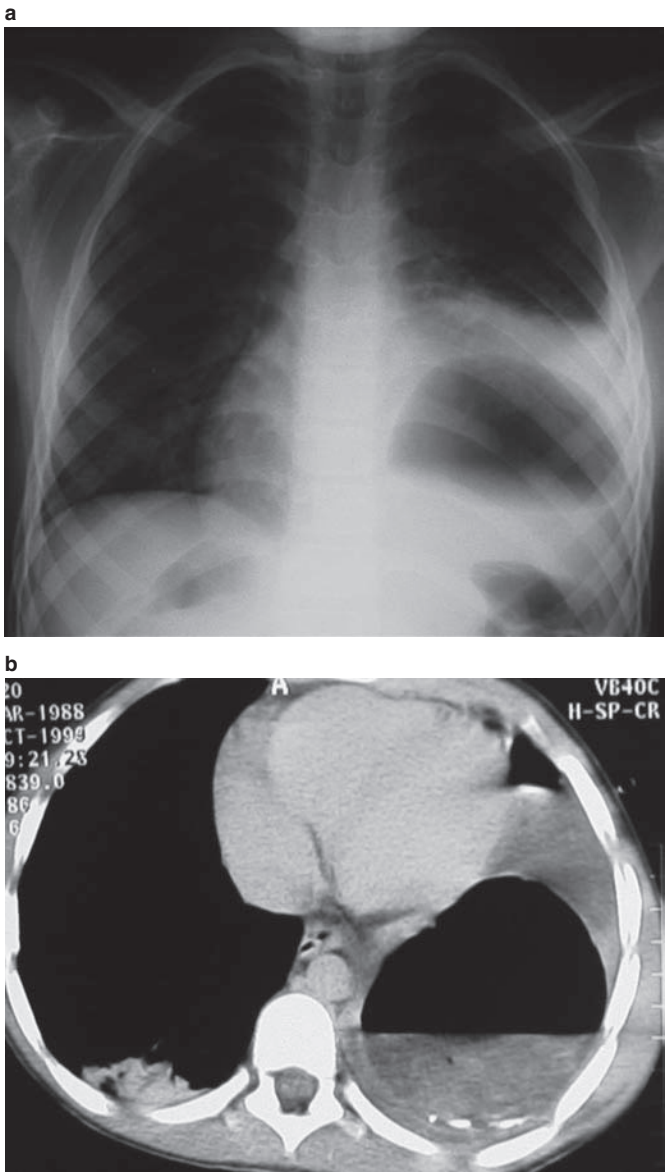


FIG. 9.1. Infected hydatid cyst with empyema: Chest X-ray (**a**) and CT scan with IV contrast (**b**) showing a large cyst with fluid level in the left lower lobe and empyema as well as another cyst in the liver suggesting a diagnosis of hydatid disease. The diagnosis was confirmed preoperatively by serology. Appropriate intravenous antihelmintics were administered along with antibiotics before thoracotomy and resection of the complicated hydatid disease

Pathogenesis

Depending on the extent of the lobar consolidation and the rapidity with which pus accumulates within the pleural space, respiratory compromise may or may not occur. The variability of clinical presentations of postpneumonic empyema also depends on virulence of the organisms, resistance offered by the host, and the use of appropriate antibiotics and appropriate drainage procedures. The consistency of pus in empyema may vary with the type of bacterial infection and the host immune response.

Rarely a large untreated empyema will drain to the surface spontaneously, usually alongside a perforating vessel in the second intercostal space anteriorly. This is called "empyema necessitans" and, if arising from the left pleural space, the swelling may transmit the pulsation of the heart and great vessels – a "pulsating empyema." An empyema may rupture into the airway or the pericardium. Pleural space infection has been known to spread to the brain or bone in children. These complications from an untreated empyema are all rare in western world although still seen regularly in underdeveloped countries.

The pathological progression in empyema can be divided broadly into three stages:

Stage I exudative phase
Stage II fibropurulent phase
Stage III organization phase

The empyema disease process is a continuum. The progression of the empyema disease process continues if not halted or inappropriately managed.

Stage I: Exudative Phase

Bacterial infection of the lung (pneumonia) causes intense inflammation resulting in increased capillary permeability. The proteinaceous exudate – fluid and cells – from the inflamed visceral pleura escape into the pleural space initially resulting in a clear parapneumonic effusion. Bacterial infection of the parapneumonic effusion leads to the exudative phase of the empyema. Chemical mediators including vasoactive amines, vasoactive polypeptides, and products of cascade enzyme systems activate the fibrinolytic system, which further increase capillary permeability. Activation of the coagulation cascade and complement system occurs.[25] Lysosomal enzymes from polymorphs, cytokines released from mesothelial cells lining the pleural space, and toxins from bacteria all drive this inflammatory process.[26] Suppurative products containing inflammatory exudates, living and dead polymorphs and bacteria, and fragments of cell and other particulate matter accumulate in the pleural space. The progression of disease from exudative stage to the next stage is multifactorial; phagocytosis of bacteria by active polymorphonuclear neutrophils, the bacterial virulence, host's immunity status, and administration of intravenous antibiotics in adequate doses all play part.

Stage II: Fibropurulent Phase

As the inflammatory process advances the coagulation cascade is activated and fibrinolysis suppressed, favoring fibrin deposition. Fibrin strands are deposited in the pleural cavity. Proliferating fibroblasts intermingled with phagocytes, bacteria, and fibrin form a pyogenic membrane. This membrane initially covers the parities of the thorax but subsequently fibrin strands septate the empyema cavity and loculations form. This defines the fibrinopurulent phase of the disease. In practice fibrinopurulent empyema is the commonest pathological form encountered in children. Antibiotics penetrate the empyema cavity poorly because of the loculations and are of little use without adequate drainage and debridement.

Stage III: Organization Phase

As fibroblasts proliferate, fibrous tissue gradually replaces fibrin. This process localizes the infection. The fibroblasts deposit layers of fibrous tissue (rinds) on both the visceral and parietal pleura and within the empyema cavity. The fibrous rind encases the collapsed lung and prevents it from reexpanding. Septic foci within the organized empyema contribute to the child's chronic ill health, failure to thrive, and poor respiratory reserve. As the empyema continues to organize, the fibrous rind thickens with further lung collapse and restriction of chest wall movement.

The consolidated lung acts as a source of chronic infection if allowed to remain collapsed. Delayed intervention in the organization phase will result in permanent loss of lung function and bronchiectasis. The fibrous sheet encasing lung derives its blood supply from surrounding tissues, which explains the often significant blood loss and air leak from the underlying collapsed lung during decortication.

Pneumatocele and Necrotizing Pneumonia

Occasionally, during the acute phase of staphylococcal, pneumococcal pneumonia and rarely with hemophilus pneumonia, necrosis and liquefaction of the lung parenchyma lead to pneumatocele formation.[27–29] *Staphylococcus aureus* pneumonia is associated with a high incidence of pneumatoceles – 39% of cases in one series.[27] Joosten reported a high incidence of staphylococcal empyema in children less than 1 year of age and also a high incidence of pyopneumothorax.[27] For unknown reasons the incidence of necrotizing pneumonia is increasing.

Pneumatocele formation is the result of necrosis of the lung parenchyma with cavitation in the consolidated lung (Fig. 9.2). The combination of peribronchiolar inflammation causing blockage of small airways and coughing probably also contribute. However, occasionally a pneumatocele will enlarge rapidly like a balloon. A tension pneumatocele can cause significant respiratory compromise. Pneumatoceles are known to rupture into the pleural space causing a tension pneumothorax and/or a pyopneumothorax. Most pneumatoceles, however, gradually decrease in size and resolve after 6–8 weeks following treatment of the pneumonia. Rarely pneumatoceles persist for prolonged periods and occasionally resection is necessary if there is diagnostic uncertainty. The author has resected persistent "pneumatoceles" in three children with recurrent infections only to find on subsequent pathological examination that the lesions were infected cystic adenomatoid malformations (Fig. 9.3).

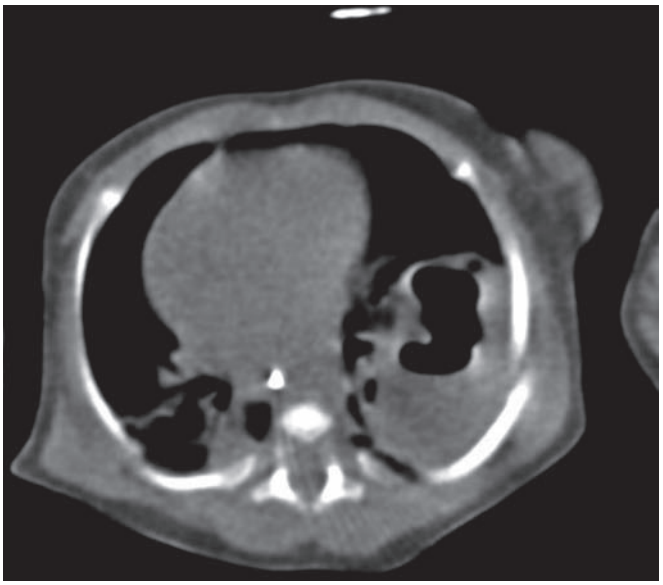


FIG. 9.2. Pneumatocele left lower lobe with empyema: CT scan with IV contrast showing a cavitating lesion in the left lower lobe and empyema. This lesion was missed on ultrasound. The pneumatocele resolved on follow-up after three months

Extrapulmonary Complications

Extrapulmonary complications have been reported in staphylococcal[27] and streptococcal pneumonia with overwhelming sepsis, especially in young and immunologically compromised children. Convulsions, osteomyelitis, toxic shock syndrome, disseminated intravascular coagulation, gastric hemorrhage, thrombosis, and multiple organ failure are all known to occur. These complications must be managed aggressively to avoid mortality. Hyponatremia related to septicemia, inappropriate ADH secretion and, more commonly, inappropriate fluid therapy is a well-recognized complication.

Microbiology

The success with which the underlying organism causing an empyema can be identified varies from 8 to 76%.[19,30,31] The use of preoperative antibiotics invariably renders the pleural fluid sterile explaining the variability of the positive culture. *Streptococcus pneumoniae* is the commonest organism responsible for childhood empyema in recent publications from both the UK and USA.[3,31–33] Newer molecular techniques can identify pneumococcus in almost 75% pleural fluid samples.[34,35] In children under 6 months of age and posttraumatic empyema the com-

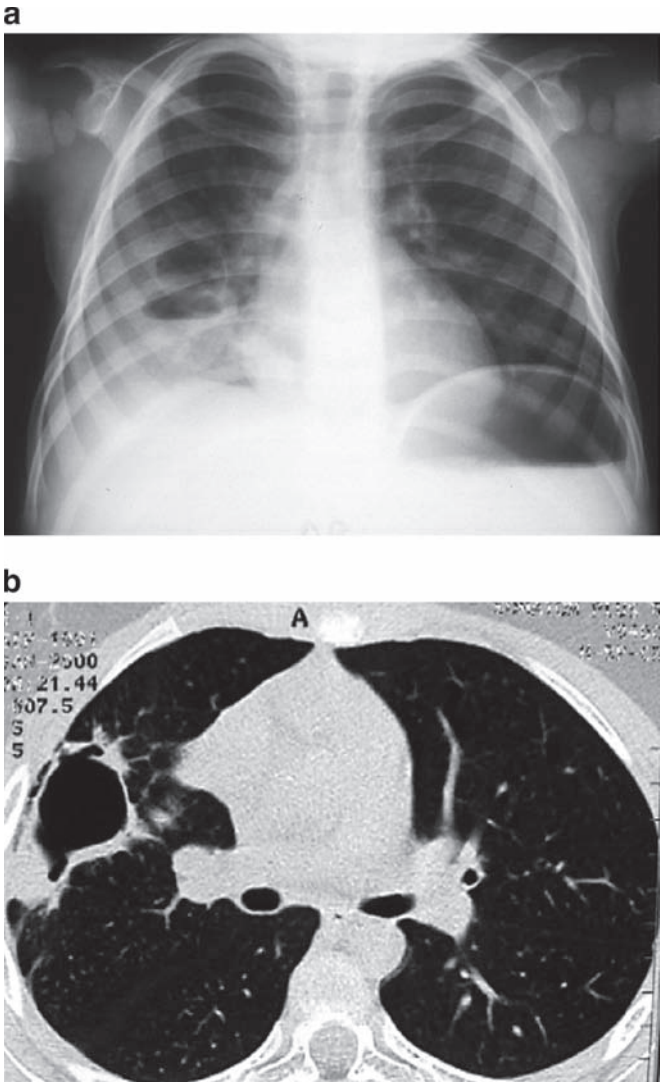


FIG. 9.3. Congenital lung malformation presenting with recurrent pneumonia: **(a)** Chest X-ray showing consolidation with pneumatoceles in the right lower lobe **(b)** convalescent CT scan compatible with a cystic adenomatoid malformation, confirmed after a right lower lobectomy

monest causative organism is *Staphylococcus aureus*.[20,21,36] *Haemophilus influenzae, Streptococcus pyogenes* and, less often, *Klebsiella pneumoniae, Bacteroides*, and other anaerobes are isolated from childhood empyemas. There are sporadic reports of empyemas due to *Pseudomonas aeruginosa*, rare species of *Streptococci, Proteus, Salmonella*, and *Yersinia*.[37]

The bacterial profile in developing countries differs with *S. aureus* being the predominant pathogen, especially during the hot and humid months when staphylococcal skin infections are prevalent.[38] There has been a decline in culture-positive *Streptococcus pneumoniae*, probably because of prior antibiotic use.[38] Various Gram-negative organisms (e.g., Enterobacteriaceae such as *Klebsiella* spp. and *Pseudomonas aeruginosa*) are also more common than in the UK. This may be related to protein energy malnutrition.[36,38–40] Tuberculous empyema may result from progressive pulmonary tuberculosis. It has been reported to account for up to 6% of all empyema cases[41] worldwide although it is seldom seen in the UK.[26]

Fungal empyemas are rare in immunocompetent children.[42,43] *Histoplasma* infection may be related to environmental exposure.[44] *Mycoplasma* is rarely associated with empyema. Mycoplasma serology confirms the association.[45] *Legionella pneumophila*, adenovirus, and influenza[46–48] may be primary agents causing pneumonia associated with a pleural effusion but the contribution of these agents to a subsequent empyema is probably small.

Immunodeficiency predisposes children to infection and overwhelming sepsis. In this context consideration has to be given to opportunistic pathogens as well as organisms recognized to cause empyema. Nocardia and other rare infections have been reported. Conversely if an unusual causative organism is encountered in an empyema, the child should be investigated for an immunodeficiency. These children are usually malnourished and have a history of recurrent infection.

Clinical Features

Clinical History

Symptoms of pneumonia in children such as, cough, breathlessness, fever, malaise, fetor, and loss of appetite precede symptoms related to the pleural collection/empyema. Younger children tend to present early with tachypnea and fever, lethargy, and cough. Some children also complain of pleuritic chest pain. Infection in the lower lobes may present with abdominal pain. In children already on treatment for pneumonia, a spiking fever and lack of improvement after 48 h of antibiotics may signal the presence of an effusion. A comprehensive history should be taken including recent antibiotic therapy, previous medical history, and contact with tuberculosis. The possibility of an inhaled foreign body should not be forgotten in children.

Physical Examination

Almost all children with an empyema will have an intermittent pyrexia, tachycardia, and an increased respiratory rate. Some children will be cyanosed, although this may be difficult to detect in the presence of anemia. Measurement of oxygen saturation (SaO_2) by pulse oximetry is particularly important, with levels below 92% in room air indicating severe disease.[49] Clinical examination must include assessment of the child's state of hydration and a full physical examination.

Clinical signs in the thorax include decreased chest expansion, stony dullness to percussion, reduced or absent breath sounds, and scoliosis. If a large effusion is present mediastinal shift may be detectable by tracheal deviation and displacement of the apex beat to the opposite side although this is relatively uncommon because the effect of the effusion is counterbalanced by collapse of the infected lung. Consolidation of the underlying lung causes bronchial breathing and reduced air

entry apparent on auscultation. Regular physical examinations and keeping good clinical records can help a clinician monitor child's condition and pneumonia effectively, and help reduce reliance on blood and radiological parameters.

Diagnosis

Investigations

Acute Phase Reactants

The white blood cell count is invariably raised in a child with an empyema, with a neutrophilia and frequently associated with anemia. C-reactive protein (CRP) and other inflammatory markers such as procalcitonin are also elevated. These have been used as markers to assess response to treatment.

Although it is commonly believed that elevated levels of acute phase reactants such as white cell count, total neutrophil count, CRP, ESR, and procalcitonin will distinguish bacterial infections from viral infections of the respiratory tract several recent prospective studies have shown this to be incorrect.[50–54]

There is no correlation between levels of acute phase reactants in acute bacterial pneumonia and empyema formation. In addition, there is no evidence in the literature supporting the view that trends in acute phase reactants correlate with clinical progress, although clinical practice has shown that serial measurements of CRP and the white cell count can be helpful in monitoring response to treatment.

Biochemistry, Hematology, and Coagulation Studies

Routine measurement of urea and electrolytes forms part of the assessment of the state of hydration. Hyponatremia is relatively common in children with severe sepsis, and this must be corrected preoperatively. Hyponatremia may be due to inappropriate ADH (SIADH) and/or hypotonic intravenous fluid infusions (4% dextrose 0.18% saline). Correction involves modest fluid restriction and, if intravenous fluids are necessary, 5% dextrose with 0.45% saline or 0.9% saline should be used. The serum albumin is invariably low.

Virtually all children with an empyema will be anemic. Usually this is a normochromic normocytic anemia but occasionally hemolysis occurs, particularly in association with pneumococcal infections. The direct Coomb's test will be positive in these children, and specific advice from a hematologist should be sought. Occasionally abnormalities in coagulation are seen in children with empyemas and it is important to recognize this prior to surgery.

Microbiology

Blood, pleural fluid, and sputum should all be sent for culture as a matter of routine in a child with an empyema. In an ideal world these samples should be taken prior to the administration of antibiotics.[49] Microbial culture should always include bacterial and tuberculosis culture. A Mantoux test should be performed if there is suspicion of tuberculosis.

A recent large retrospective study of 540 children with community-acquired pneumonia, 153 of whom subsequently developed an empyema, confirmed the value of these investigations.[55] Blood cultures were positive in 15/153 (10%) of children who developed an empyema and in 25/387 (6.4%) of those with pneumonia alone. In a series of 76 children with complicated parapneumonic effusions blood cultures were positive in 22%, compared with pleural fluid that was positive in 33% cases.[33] Hardie et al. reported a series of 56 children with pneumococcal effusions.[3] Blood cultures were positive in 10/56 cases (18%), and in 7/10 children with positive blood cultures the pleural fluid was sterile.

Diagnostic Pleural Aspiration

Thoracocentesis is not mandatory but is recommended prior to starting antibiotics in a child with a suspected empyema. Pleural fluid should be sent for urgent microscopy, Gram stain, culture, and differential cell count. Cytological examination of the pleural fluid in an empyema classically shows a predominance of polymorphonuclear leukocytes. A lymphocytic preponderance should raise the possibility of either tuberculosis or malignancy[56] although it is important to note that in at least 10% of tuberculous effusions there is a neutrophil preponderance. Most malignant effusions in children are blood stained although cytological examination of the pleural fluid may not show malignant cells.[57]

On many occasions pleural fluid is sterile because of prior antibiotic administration.[58] A diagnostic tap will be unsuccessful if the parietal peel is very thick or the pus particularly viscid. In the event of a failed diagnostic tap, it is mandatory to evaluate all clinical and radiological evidence of empyema thoroughly before undertaking definitive management.

Sophisticated laboratory analysis may reveal causative organisms when conventional culture fails. Latex agglutination tests are available for detection of pneumococcal antigens.[35] Polymerase chain reaction techniques are available for the detection of pneumococcal DNA[34,35] and mycobacterial DNA.[59]

Routine biochemical analysis of the pleural fluid is unnecessary and does not contribute to the management of empyema in childhood. Pleural fluid taken from children with an empyema shows a glucose concentration < 2.2 mMol/L, pH < 7.2, LDH > 1,000 I/dL, protein > 25 g/L, and a specific gravity > 1.018. Pleural fluid from a child with a sterile clear or sympathetic effusion will show a pH > 7.2 and a glucose concentration > 2.2 mMol/L.

Radiology

First radiological investigation is usually a plain chest X-ray. Pneumonic consolidation will be seen. The appearance of a pleural effusion is signaled by a fluid meniscus. As the effusion enlarges mediastinal shift is rare (Fig. 9.4a). A lung abscess, pneumatoceles, and a pyopneumothorax (Figs. 9.5 and 9.6)

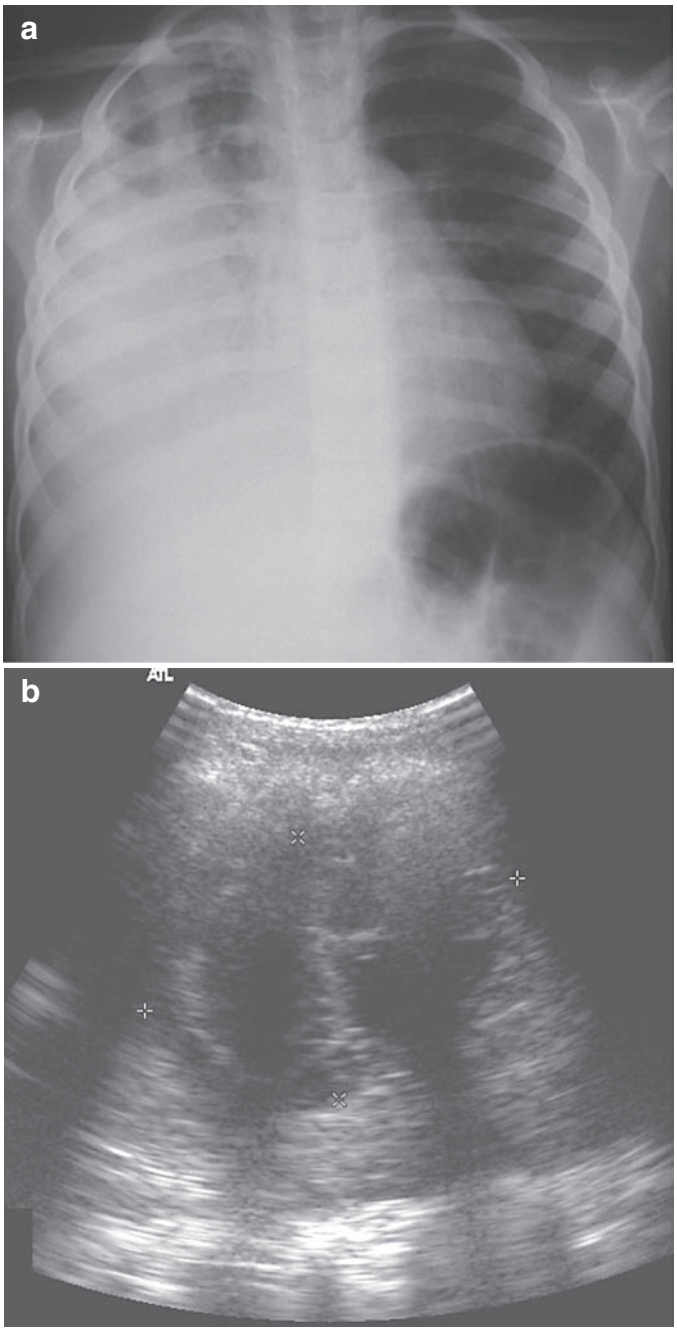


Fig. 9.4. Empyema: (**a**) Plain chest X-ray showing meniscus sign and consolidation with little or no mediastinal shift, which would suggest consolidation rather than collapse related to pleural effusion. (**b**) Ultrasound showing loculations and septa with the pleural effusion containing debris suggestive of empyema

are all visible on a chest X-ray. The plain X-ray cannot differentiate an empyema from a tumor. A pulmonary blastoma is commonly mistaken for an empyema as the clinical presentation and some of the radiological features are similar.[60,61] Therefore, the plain chest radiograph especially in the fibrinopurulent and organization phases of an empyema is suggestive but not diagnostic of empyema.

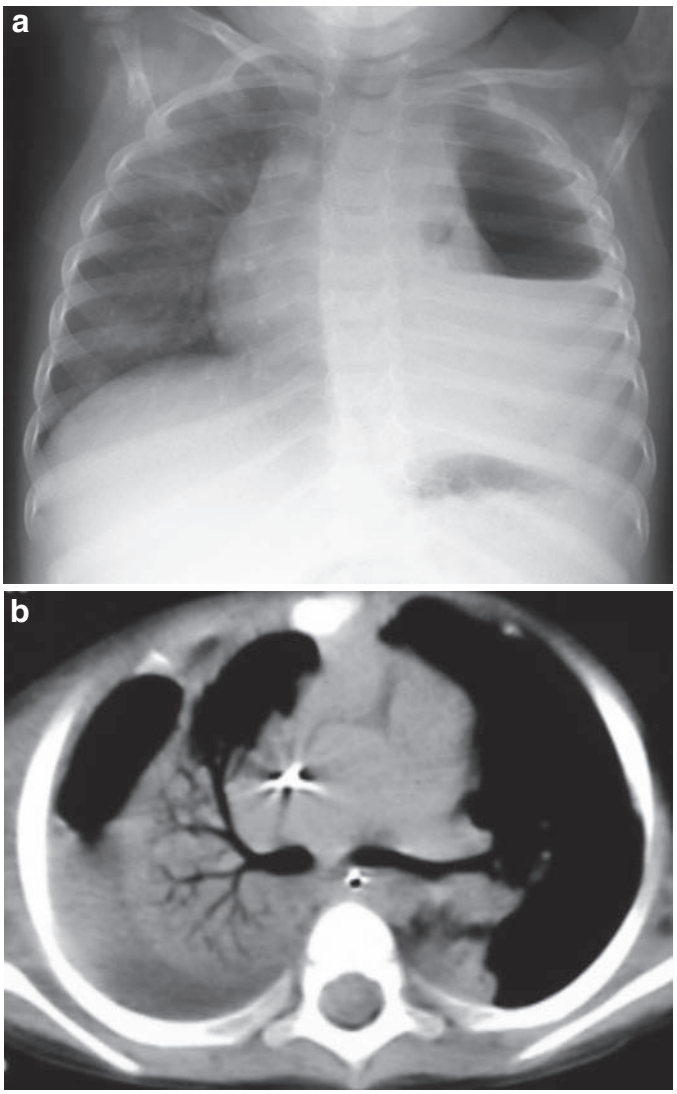


Fig. 9.5. Pyopneumothorax: (**a**) Chest X-ray showing a fluid level in the pleural collection. (**b**) CT scan with IV contrast showing a rupture of a pneumatocele as a cause of a bronchopleural fistula

Ultrasonography is particularly valuable for imaging the child with a suspected empyema. Ultrasound is portable and thereby available for immediate bedside examination of critically ill children. Fluid in the pleural space is easy to identify. Ultrasound is most useful to determine whether an effusion is loculated (Fig. 9.4b).[62] Although an impression of the density and echogenicity of the pleural fluid can be gained from ultrasound this does not correlate well with the pathological stage of the empyema.[63] Akhan et al. reported a characteristic appearance of tuberculous effusions on ultrasound, noting the presence of diffuse small nodules on the pleural surface.[64]

Ultrasound may be used to guide chest drain insertion or thoracocentesis. The optimum site for drainage or aspiration can be marked on the skin by the radiologist.[65,66] The key limiting factors of sonographic evaluation of the chest are based

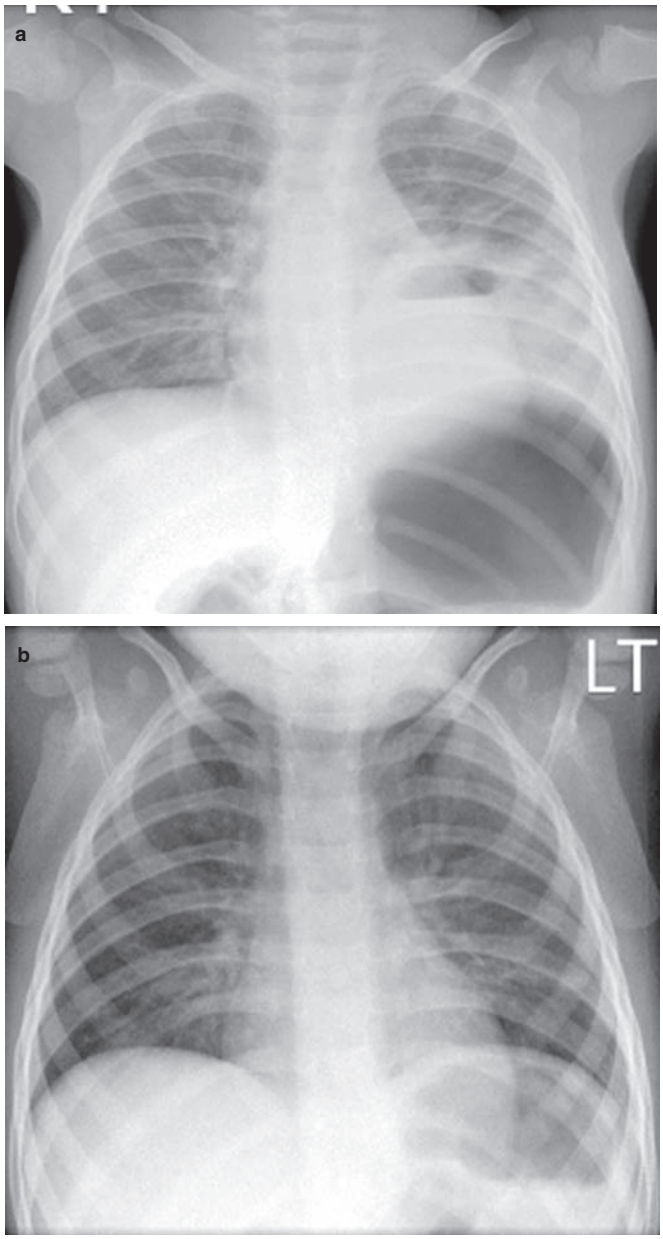


FIG. 9.6. (**a**) Chest X-ray showing consolidated lung, a cavity containing fluid level (an abscess) and empyema. (**b**) Follow-up chest X-ray showing complete resolution of the cavity. The management consisted of empyema debridement and drainage without resection of the cavity

on the physical limitations of the ultrasound beam. As the intercostal spaces narrow, the value of ultrasound becomes limited.[67] Occasionally, homogenous solid lesions are mistaken for fluid collections on sonography.

Computed tomography (CT) with intravenous (IV) contrast complements ultrasonography for imaging children with complicated pneumonia.[67,68] Parenchymal lung pathology can be identified clearly. CT with IV contrast distinguishes the rare lung tumors presenting as empyema in childhood as well as identifying mediastinal pathology.[61] Thickened pleural rind, consolidated lung, and associated lung pathology, such as a lung abscess, are all clearly visible on CT[63,69,70] (Fig. 9.5b). An empyema associated with lung necrosis, pneumatoceles, or a lung abscess invariably takes a more protracted postoperative course than a simple empyema. Furthermore, the presence of underlying lung pathology will require prolonged follow-up and monitoring. While unnecessary for most cases of pediatric empyema, CT undoubtedly has a role in complicated cases (including initial failure to aspirate pleural fluid and failing medical management) and in immunocompromised children.

Bronchoscopy

Routine bronchoscopy is unnecessary unless a foreign body is suspected. Bronchoalveolar lavage may be useful in identifying the infective organism but it is considered unnecessary when pleural fluid is available for culture.

Differential Diagnosis

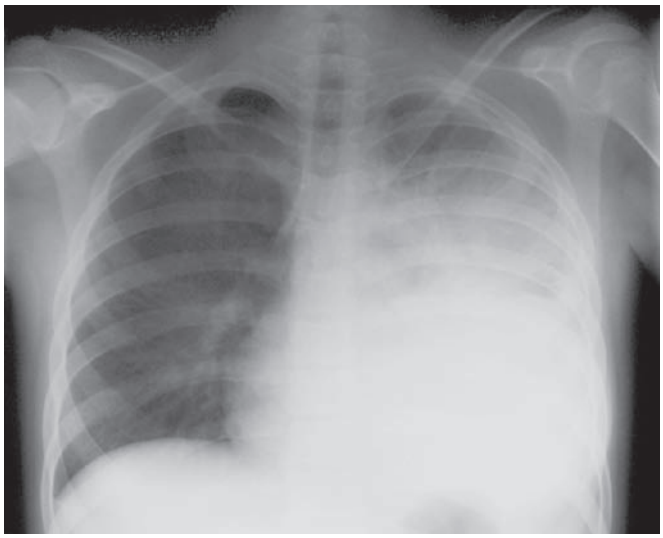
Intrathoracic Tumors Presenting as Empyema

Thoracic tumors presenting as an empyema appear in literature as case reports.[60,71–75] Initial clinical, biochemical, and radiological findings in all these cases were consistent with a diagnosis of empyema. Although diagnostic pleural aspiration and cytology is not routinely performed in children, if a high lymphocyte count is found underlying malignancy should be suspected and investigated. Most malignant effusions in children are blood stained although it is uncommon for malignant cells to be identified on cytology.

The author reported a series of eight children with intrathoracic tumors mimicking empyema.[62] Ultrasound was performed in four children but failed to identify the presence of intrathoracic tumor in all. Preoperative CT scans without IV contrast were misleading and failed to identify the underlying pathology. Four children underwent thoracotomy for debridement of the empyema, with substantial blood loss in two cases. A third child, with a pleuropulmonary blastoma, developed a tumor implantation nodule at the site of thoracotomy incision necessitating excision of the chest wall with residual tumor in the left lower lobe. In the remaining four cases CT scans with IV contrast correctly identified the intrathoracic tumors preoperatively.

It is possible that in spite of careful preoperative evaluation intrathoracic tumors will occasionally be diagnosed only at the time of emergency thoracotomy. In this situation the surgeon should take a biopsy and then achieve hemostasis before closure. Although incidence of tumors presenting as empyema is thought to be low, the eight children we reported represented 5.3% of all empyemas presenting to us during that period.

In the pediatric population pleuropulmonary blastoma, benign cystic teratoma (Fig. 9.7), primitive neuroectodermal tumors and lymphomas have all been mistaken for empyemas.[61,75]



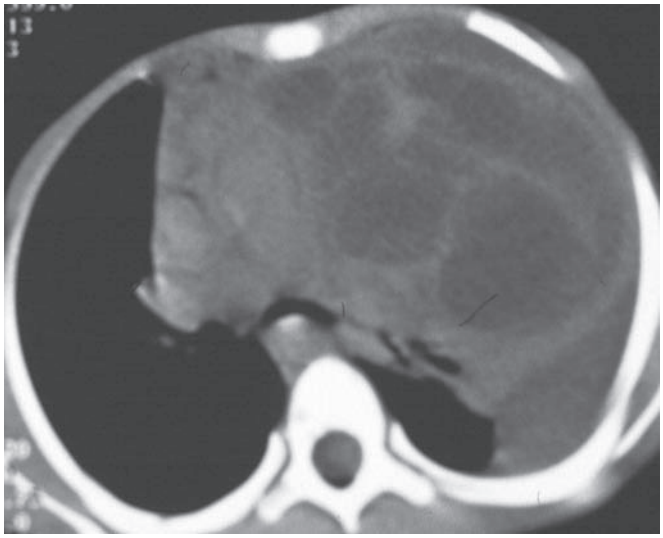


FIG. 9.7. Infected cystic mediastinal teratoma with effusion: **(a)** Chest X-ray showing signs of pleural collection, ultrasound confirmed pleural collection but failed to identify mediastinal pathology **(b)** CT scan with IV contrast showing the mediastinal pathology which was resected and the empyema was drained. The CT scan prompted an open thoracotomy approach rather than VATS debridement of the empyema alone.

Clinical vigilance and careful preoperative evaluation are necessary. A nonresolving empyema, blood-stained pleural effusion, and pleural fluid with a raised lymphocyte count and a very large pleural effusion are all potentially suspicious where a preoperative CT scan with IV contrast is recommended.

Management

The cardinal principles of empyema management are administration of appropriate intravenous antibiotics in combination with adequate drainage to achieve full expansion of the lung.

Medical Management

- Optimum perioperative medical management should be an integral component of a successful management strategy of childhood empyema.

Early recognition and treatment of lobar pneumonia in children reduces the incidence of empyema. Lack of clinical improvement after 48 h suggests that either the choice of antibiotic is inappropriate or that there is an associated empyema. The chest radiograph should be repeated, and if there is any suspicion of an effusion an ultrasound examination is requested.

Small sympathetic effusions are commonly associated with acute lobar pneumonia, and these usually resolve with antibiotics alone.

Supportive medical therapy is essential. This includes oxygen, fluids/nutrition, analgesia, and respiratory physiotherapy. Humidified oxygen should be administered to maintain oxygen saturations above 95%. Isotonic intravenous fluids should be given to correct dehydration and electrolyte imbalances. Antipyretics and analgesics should be given regularly. Children who fail to improve clinically and radiologically should be referred to a center with expertise in surgery of empyema drainage procedures.

Antibiotics

Response to antibiotics is dependant upon the pathogen involved, the stage of empyema, and the immune status of the child. In the early exudative stage high concentrations of antibiotics alone may be effective treatment whereas antibiotics are unlikely to be effective in more advanced disease without surgical intervention.[76] Unless a pathogen with known sensitivity to antibiotics has been isolated first-line treatment with high doses of a cephalosporin is recommended. In the authors' institution first-line treatment comprises IV cefuroxime at 50 mg/kg doses three times daily. In the event of impaired renal function reduced doses are recommended. Recommendations for antibiotic therapy for childhood empyema are not evidence based but based on local patterns of bacterial resistance. Other antibiotics such as flucloxacillin, amoxycillin, gentamicin, and meropenem may be necessary depending on the sensitivity of organisms isolated from blood or pleural aspirate culture.

Occasionally despite adequate drainage sepsis continues. This is usually related to necrotizing pneumonia or occasionally to a distant septic focus. In these circumstances, clindamycin or rifampicin may be useful to manage infection with Gram-positive organisms. In these complex empyemas it is usually necessary to give combination therapy with more than one antibiotic. Advice from microbiology is essential.

Pleural Aspiration

Parapneumonic effusions can be drained with either by repeated thoracocentesis or by closed tube thoracostomy. Repeated aspiration is not a satisfactory option in children as

it is painful and requires considerable cooperation from the patient. Closed chest drainage is the preferred option.

Traditionally this involves insertion of a 16–20 F chest drain through the fifth intercostal space between the anterior and mid-axillary lines under either local or general anesthesia. Practice is changing in many centers in favor of small-bore tubes or pigtail catheters placed under ultrasound guidance.

Surgical Management

- The goal of surgery is to achieve full expansion of the lung and resolution of the empyema.
- Early surgical intervention in childhood empyema reduces morbidity.
- Treatment failure should be recognized early to avoid disease progression.

Surgical practice for the management of childhood empyema varies around the world. The principal problem, however, is establishing the pathological stage of the empyema preoperatively. By the time the child arrives in the hands of a surgeon it is safe to assume that the empyema is in the fibrinopurulent phase even if the pus aspirated is not thick. Effective drainage with lung expansion in this phase is imperative to reduce morbidity and progression of the disease to the organization phase.

A large meta-analysis of literature search from 1984 to 2004 by Avinsino et al. concluded that primary operative therapy is associated with reductions in mortality, reintervention rate, length of hospital stay, time with tube thoracostomy, and duration of antibiotic therapy, compared with nonoperative treatment.[77]

Intercostal Tube Drainage and Fibrinolytics

Closed intercostal tube insertion with administration of IV antibiotics may be successful in early stage empyemas and in some later stage cases if loculation is minitmal. Some authors advocate intrapleural fibrinolytic therapy[58] to improve drainage.

It is important to recognize when conservative therapy is failing to reduce the morbidity associated with delayed referral for surgery. Persistent sepsis with a pleural collection after a maximum of 7 days conservative management is an absolute indication for surgical referral.[56] Seven days is an arbitrary period chosen to acknowledge different rates of disease progression yet provide a clear limit on the duration of medical therapy.

The author recommends pediatricians managing empyemas in peripheral hospitals to seek surgical advice after 3 days of intrapleural fibrinolytics if there is continuing pyrexia and a persisting pleural collection.

There are conflicting studies in the literature concerning the use of intrapleural fibrinolytics. It must be emphasized that the use of fibrinolytics is an adjunct to improve drainage, which should not change the underlying principles of empyema management.

Technique: Intercostal Chest Tube Drainage

Intercostal chest tubes in young children are best inserted under general anesthesia by a pediatric surgeon using an open technique. In some centers pigtail catheters or small-bore catheters are inserted using the Seldinger technique by respiratory physicians or interventional radiologists using intravenous sedation and local anesthesia.

Informed consent must be obtained. The procedure should be explained to the child. The operating surgeon must ensure that the chest radiographs are available to confirm the diagnosis and the side of the empyema.

If conscious IV sedation and local anesthesia is to be used then it is helpful to apply topical anesthetic (EMLA® or Ametop®) to the skin 1–2 h prior to the procedure. The safety of the child is paramount and this necessitates that the person administering the sedation should be competent in airway management and resuscitation of children. General anesthesia is safer than intravenous sedation in a child with respiratory compromise and certainly this is the preferred option for noncooperative children.

Chest drains should be inserted using an aseptic technique. Whether conscious sedation or general anesthesia is used, local anesthetic should be instilled to minimize postoperative discomfort. Plain bupivacaine 0.25% to a maximum dose of 2 mg/kg (0.8 ml/kg) can be infiltrated into the skin and deeper layers or, alternatively, used for intercostal nerve blocks.

In some centers ultrasound is used to mark the optimum site (location of the pleural fluid) for chest drain insertion. If this technique is used it is important to document patient position when the spot is being marked.[78–80] Catheter placement using CT guidance has been recommended for difficult cases.[80–85]

The safest site for an open method of intercostal chest drain is the fifth intercostal space between the anterior and mid-axillary lines. This region is termed the "safe triangle."[86] The safe triangle is outlined by the anterior border of the latissimus dorsi muscle, the lateral border of the pectoralis major muscle, and a horizontal line at the level of the nipple. This region minimizes the risk to underlying structures, avoids damage to muscle, breast tissue, and an unsightly scar. A more posterior position is uncomfortable and risks the drain kinking. A more anterior position risks inadvertent laceration of an intercostal artery.

The skin should be incised and a track dissected down to the pleura on the upper border of the rib using an artery forceps. This track should be developed to allow direct insertion of the chest drain without force. Forceful insertion of a chest tube or use of an introducer risks damaging intrathoracic organs with potentially fatal consequences and should be avoided. The drain must be well secured after insertion to prevent inadvertent displacement. This involves a heavy-gauge nonabsorbable suture placed through the skin and tied tightly repeatedly around the drain. An airtight occlusive dressing should be applied. A purse-string suture is unnecessary. Special dressings and fixation devices are available to hold small pigtail catheters in place instead of a conventional suture (e.g., Drain-Fix®, Maersk Medical Ltd, Stonehouse UK).

The pleural effusion should start to drain immediately after successful insertion of a chest tube. The chest drain should be connected to an underwater seal of approximately 1–2 cm in a bottle, which must be kept below the level of the patient's chest at all times. The air/water meniscus will swing with respiration. Continuous bubbling of air indicates either a bronchopleural fistula or that one of the chest tube holes has slipped out of the patient and is open to the atmosphere. A chest radiograph should be taken after insertion of a chest drain to confirm the tube position and to ensure that an inadvertent pneumothorax has not developed.

There is controversy about the size of chest drains used to treat childhood empyema. Many pediatricians now prefer small-bore catheters, as adult studies have shown that they as effective as larger catheters.[87] Smaller catheters are generally better tolerated.[88] Surgeons generally continue to use large-bore drains out of concern that smaller tubes will block with thick pus.[89–91] Pierrepoint has shown that ultrasonographically guided small pigtail catheters can be effective for treatment of early loculated empyemas.[81] Thomson et al. also used small-bore catheters successfully in a multicenter trial with fibrinolytics.[58]

Low-pressure suction (5–10 mm of water) is often recommended especially if small bore catheters are used. Suction seems to be helpful when used in combination with small-bore chest tubes and fibrinolytic agents. However, there is no incontrovertible evidence that suction confers any benefit over simple closed drainage as most studies are observational only.[86] A bubbling chest drain implies a bronchopleural fistula. If this is associated with a residual pneumothorax then low-pressure suction should be applied because once the lung reexpands fully the leak may seal. Under no circumstances should a bubbling chest drain be clamped as the continued air leak will lead to a potentially fatal tension pneumothorax within a short while.

Ward Instructions for Management of Chest Drains

1. Specially trained nursing staff should manage patients with chest drains.
2. Underwater seal bottle should be kept upright and below the level of the chest at all times. The drain bottle should contain adequate water to cover the end of the tube by at least 1–2 cm below water.
3. Daily records must be kept of the volume of fluid drained along with temperature, pulse, and fluid balance charts. The presence of respiratory swing in the chest drain should be noted along with any bubbling on coughing.
4. Medical personnel giving instructions for chest drain clamping should record this in writing in case notes. Note:
   - Bubbling chest drains should never be clamped.
   - If chest pain or breathlessness develops the chest drain should be unclamped immediately.
5. Parents and patients should be encouraged to look after the chest tube and underwater seal bottles so that all connections and tapes attaching the drain to the patient are secure at all times.
6. Blockage or kinking of the tubing should be suspected if the drain suddenly stops draining. Obstruction from fibrin or clots can be cleared by carefully flushing the drain with 10–15 ml of normal saline and a bladder syringe. In presence of persisting pleural collection a blocked drain that cannot be rescued should be removed or replaced.
7. The chest tube should be removed using one of the following techniques. Older children can be asked to perform a Valsalva's maneuver while the drain is removed. In younger children the drain should be removed as rapidly as possible during expiration. A sterile occlusive dressing should be applied to the drain wound and left undisturbed for 48 h.
8. A chest radiograph should be taken shortly after drain removal to ensure that a pneumothorax has not developed.

If a very large pleural effusion is to be drained the drain should be clamped for 1 h after approximately 10 ml/kg of fluid is removed to avoid reexpansion pulmonary edema.[92,93]

A chest drain should be removed once the lung is fully expanded and drainage is less than 30 ml per day of clear fluid. Improvement in the general condition of the child, resolution of fever, and decreasing volumes of serous drainage from the chest tube are generally good signs encouraging removal of the chest drain. It is not necessary to wait for complete cessation of drainage. Cessation of drainage associated with continuing fever and malaise indicates continuing sepsis and an inadequately treated empyema.

Intrapleural Fibrinolytic Agents

Insertion of an ultrasound guided pigtail catheter combined with instillation of fibrinolytic agents such as urokinase into the pleural space has shown to dissolve fibrinous material and achieve effective drainage of an empyema in some studies.[37,58] Tillett and Sherry were the first to suggest enzyme instillation for the management of empyema in 1949. Intrapleural fibrinolytics have been used in an attempt to reduce the need for surgery in empyema and as an adjunct to thoracoscopic drainage.

Three agents are commonly used for intrapleural instillation – streptokinase, urokinase, and Alteplase (tissue-type plasminogen activator). Although there is no evidence to suggest which of these agents is most effective, only urokinase and Alteplase have been the subject of a randomized trial in children.[58,94] Studies comparing urokinase and streptokinase in adults suggested that both were equally effective.[95,96] Streptokinase is now rarely used for intrapleural fibrinolysis because of the risk of severe allergic reactions. Urokinase is both nonpyrogenic and nonallergenic and has become the enzyme of the choice for fibrinolysis.

Urokinase Treatment Schedule (UK Pediatric study[65])

- Forty-thousand units in 40 ml of normal saline instilled twice daily for 3 days for children >10-kg weight
- Ten-thousand units in 10 ml of saline instilled twice daily for 3 days for children <10-kg weight
- Intercostal tube clamped for 4 h after instillation
- Response to treatment is assessed after six doses (3 days) using clinical, CRP, and radiological parameters

Higher doses of urokinase 25,000–100,000 (mean 3,100 u/kg/day) were used once daily with a 1-h dwell time by Wells.[94] Alteplase was used in a dose of 0.1 mg/kg once daily with a 1-h dwell time.[94] These regimens appear effective and safe. Minor side effects reported in the two largest pediatric studies include discomfort during intrapleural injection and transient blood staining of the drainage fluid.[58,94] Intrapleural bupivacaine can be given with the urokinase if the instillation is uncomfortable. Significant bleeding has been mentioned in case reports after the use of urokinase.[97,98]

Seven pediatric studies exist reporting a total of 136 cases treated with streptokinase, urokinase, or Alteplase.[94,99–104] These studies report an overall 90% success rate in avoiding the need for surgery. Thomson in a multicenter randomized placebo-controlled trial reported 60 children with a median age 3.3 years (range 5 months to 15 years) who were randomized to receive either urokinase or saline.[58] The primary outcome measure was length of hospital stay, and this was significantly reduced in the group treated with urokinase (7.39 vs. 9.49 days; ratio of geometric means – 1.28, CI – 1.16–1.41, $p = 0.027$). Although evidence from this trial and other small sample trials has favored the use of urokinase in childhood empyema and has shown benefits in terms of hospital stay, time to defervescence, improvement in chest radiograph, and requirement for surgery, these trials have been criticized because the improved results of fibrinolytics were not consistent across the studies.

These studies also have low statistical power and were unable to quantify the need for surgery after fibrinolytic therapy. In a previous review of the Cochrane database by Coote (2002) it was suggested that primary video-assisted thoracoscopic surgical (VATS) drainage had a significantly higher success rate with shorter duration of hospital stay compared with patients managed with chest tube drainage with streptokinase. A recently published large double-blind randomized trial of intrapleural streptokinase in adults concluded that fibrinolysis did not improve mortality, the need for surgical drainage, the length of the hospital stay, or outcome in terms of lung function.[105] This study concluded that intrapleural fibrinolytics conferred no advantage over conventional drainage in the management of empyema. The accompanying editorial to this study commented that neither ultrasound nor CT studies were assessed at the time of enrollment, nor was there any uniformity in antibiotic therapy, raising the possibility that the study population was heterogeneous, obscuring the possibility that fibrinolytics might benefit for early stage empyemas.[106] Moreover, the results of clinical studies involving adults may not be directly comparable with children because comorbidity is considerably less common and preillness lung function is generally normal.

Early Surgery

Early definitive surgical intervention is highly effective in childhood empyema.

Unfortunately it is extremely difficult to predict which children will respond to simpler measures and avoid the need for a thoracotomy. Adult guidelines recommend surgical treatment if there is persistent sepsis associated with a pleural collection despite IV antibiotics, chest tube drainage, and fibrinolytics for 7 days.[56] Case studies and retrospective reviews comparing different treatment strategies inevitably make comparisons with historical controls.[98] The conclusions from these studies depend largely on surgical expertise and local referral practices.

A retrospective study by Hillard et al. compared length of stay according to therapy in 48 children with empyema over 3-year period. Surgery was necessary in 3/8 children treated with chest drainage alone, and 2/14 in the group receiving fibrinolytics. Overall hospital stay was shorter for children undergoing surgery as the initial treatment.[107] Doski et al. reviewed 139 children with empyema comparing primary chest tube drainage with fibrinolytics and delayed thoracotomy/VATS for unresolved cases with primary early VATS debridement.[108] This study suggested that early definitive surgery was associated with a shorter hospital stay. Other studies have compared historic data with recent changes of practice to prove the point that early surgery was beneficial. Early thoracotomy and debridement produces good postoperative lung expansion in almost all cases.[109,110]

Minithoracotomy and Debridement

Open drainage by minithoracotomy to break down and debride fibrinous septa is highly effective, achieving full lung expansion more reliably than any other method of treatment.[111] Muscle sparing confers additional benefits in terms of reduced pain and shorter recovery. Thoracotomy and evacuation of the fibrinous coagulum and pyogenic material after breaking down loculations has been described incorrectly in the literature as early decortication.[35,112] This actually describes debridement of an empyema. Decortication involves sharp dissection to excise the thickened parietal and visceral pleura, which surround and encase the lung.

Minithoracotomy has effectively drained empyema purulent material under vision leading to a rapid expansion of lung.[113] In earlier years thoracotomy was performed in cases where chest tube drainage had failed to expand the lung and the child had persistent intrapleural collection and lack of clinical improvement.[114] Nowadays, VATS or thoracotomy is used as a therapeutic option for the failed cases of the chest tube drainage or

urokinase therapy.[111] Open thoracotomy results in significant postoperative pain and inevitably leaves behind a scar causing rib crowding and subsequent chest deformity and scoliosis.

Technique of Minithoracotomy and Debridement

The anesthetised child is placed in a lateral decubitus position. An incision is made along the fifth or sixth intercostal space from posterior axillary line to anterior axillary line. A muscle cutting approach involves cutting through both latissimus dorsi and serratus anterior muscles in the line of the incision. A muscle-sparing incision involves mobilization of the latissimus dorsi muscle from its anterior margin, retracting it posteriorly, and then either splitting the serratus anterior between digitations or reflection of the lower digitations after detaching them from the chest wall, which is preferred. Muscle-sparing incisions are believed to be less painful. The thorax can then be opened through an intercostal space or after subperiosteal resection of a segment of rib. Debridement is carried out and all the purulent material is evacuated under direct vision. The collapsed lung is seen to expand before the thoracotomy is closed. One of two chest drains are placed before closure and connected to an underwater seal.

Video-Assisted Thoracic Surgery (VATS) Debridement

With the advent of video-assisted techniques, many surgeons are challenging the traditional approaches to empyema management in children. Kehr and Rodgerson were the first to describe thoracoscopic drainage in children. VATS techniques offer advantages in terms of accurate disease staging and debridement of the fibrinous pleural disease, separating the loculi under vision.[115] The technique is less invasive than thoracotomy yet in experienced hands as effective and safe. There have been no comparative trials in children to suggest that early surgery is superior to drainage and fibrinolytics. A small study included in the Cochrane review, randomized 20 patients with empyema to receive intrapleural streptokinase for 3 days via a chest drain with immediate VATS. The surgical group had a high treatment success rate 10/11. Five of nine patients who failed to respond to streptokinase therapy were salvaged by VATS. The VATS patients required chest drainage for shorter periods and had shorter hospital stays.[116] However, this study can be criticized based on small sample size and an unusually high failure rate following fibrinolysis.

Proponents of early endoscopic surgery claim that if general anesthesia is used for insertion of a chest drain then the procedure should be combined with VATS.[117] Early VATS has been shown to benefit lung reexpansion and improve the drainage of the empyema.[117–119] Loculi can be separated allowing thick pus to drain effectively.[108,115,120–122] Many series have shown reduced postoperative pain, shorter hospital stay, and better cosmetic results when VATS is compared with conventional thoracotomy.[108,120,123,124] Unfortunately VATS is difficult in children when first-line management with fibrinolytics has failed,[124] and it is not suitable for advanced empyemas.[108,118] Several studies comparing conventional thoracotomy and VATS debridement[115,125,126] have favored VATS approach claiming reduced duration of hospital stay, reduced blood loss, less discomfort, and reduced chest drainage. However, Goldschlager's study showed no difference in length of postoperative chest drainage or hospital stay. Although most studies are retrospective containing small number, VATS has proven effective, with minimal complications, and was well tolerated.

Thoracoscopy requires expertise and a well-trained operating team familiar with the equipment. In the presence of a thick pyogenic membrane covering the visceral and parietal pleura, the likelihood of damaging the inflamed lung while removing the pyogenic membrane is high. Conversion to open thoracotomy is necessary when access to the pleural cavity proves impossible because of a thick pyogenic rind or if there is excessive bleeding. Minithoracotomy and debridement or decortication in these instances is safer and curative.[110,127,128]

Technique of VATS Debridement

VATS is performed under general anesthesia with a single lumen endotracheal tube in majority of cases. Selective endobronchial intubation, or the use of bronchial blocker in young children where double lumen tubes are not available, may be useful in selected cases to avoid contamination of the contralateral lung. The child is placed in a lateral decubitus position with the involved side upward. The best site for trocar placement is determined by needle aspiration of pleural fluid. Firstly a 5-mm port is inserted under direct vision in order to avoid damage to the inflamed lung. This port is usually inserted in the fifth or sixth intercostal space in the posterior axillary line. Creation of a working space is essential. A 0° or 30° telescope is used for visualization. Liquid pus is aspirated before insufflation with CO_2 at 5–8 mmHg pressure. Carbon dioxide insufflation is not mandatory throughout the length of the procedure because the lung will often collapse sufficiently to allow visualization of the empyema cavity. A second port for instrumentation is placed under thoracoscopic visualization. Two ports are usually sufficient if debridement is performed early.

The empyema cavity is irrigated with warm saline, and pyogenic material is gently removed either with the suction aspirator or with the help of Yohan's or equivalent atraumatic forceps. Pleural debris should be sent for culture. The entire pleural cavity can be debrided effectively under vision. In majority of cases the lung starts to reexpand at this stage of operation. All surfaces of the lung, including the fissures, should be inspected, and a chest drain is left in place through one of the port sites. Local anesthetic is infiltrated around the port sites or used for intercostal nerve blocks.

Most children can be managed safely on a pediatric surgical ward postoperatively with nurse- or patient-controlled analgesia. The chest X-ray is repeated the following day to confirm lung expansion and assess the extent of lung con-

solidation. Fibrinolytics may be helpful in the postoperative period after thoracoscopic debridement if lung expansion is incomplete. This is especially useful when the surgeon is in the learning phase of thoracoscopic debridement or if debridement is known to be incomplete. The chest drain should remain in place until the losses reduce to less than 30 ml/day and become clear. The child is kept in hospital on intravenous antibiotics until afebrile for at least 24 h. A further chest X-ray should be taken following drain removal.

Thick membrane is seen on occasions encasing the lung not allowing expansion; a peanut sponge is used gently to peel the membrane off the visceral pleura. Conversion to open thoracotomy is indicated in cases of failure of separation of the fibrous rind from the visceral pleura after the thoracoscopic maneuvers, excessive bleeding, inadequate visualization, and failure of lung reexpansion.

Thoracotomy and Decortication

Decortication of an organized empyema carries a significant morbidity from bleeding and air leaks.[27,129–131] Decortication requires removal of the thickened fibrous parietal peel and sharp dissection to remove the visceral peel encasing the lung. This is best performed through a conventional posterolateral thoracotomy. The aim of decortication is to free the encased lung to allow reexpansion and to free the chest wall to allow proper respiratory excursion. Excision of the pleural rind may result in substantial bleeding and an air leak and occasionally nerve damage. Blood must be available. Early recognition of the fibrous stage and prompt intervention minimize these risks. Provided decortication is complete, the prognosis for lung function is excellent in most cases. However, inadequate management places the child at risk of chronic ill health with continuing sepsis, restrictive lung disease, failure to thrive, and anemia. The child becomes a respiratory cripple. The chest X-ray shows persistent lung collapse, loss of volume, and a scoliosis attributable to progressive fibrosis.[130]

Technique of Decortication

Decortication is carried out using a conventional posterolateral thoracotomy.

The chest is usually entered through sixth intercostal space although the rib resection may be necessary to achieve sufficient exposure. The empyema cavity is entered through the organizing parietal peel, which is usually thick and very dense. The cavity is evacuated with samples taken for bacterial culture. The parietal peel is stripped from the inside of the chest wall by digital dissection. This can be difficult but as the chest wall is freed the thoracotomy will open progressively. The peel should not be disturbed over the mediastinal surface because of the risk of damaging vital structures.

Separating the peel from the diaphragm is generally unsatisfactory because it is often impossible to find a plane of cleavage.

The encased lung is then freed. This involves gentle finger dissection around the edge of the lung and between the fissures. The thick visceral rind is carefully incised with knife down to the plane immediately beneath the cicatricial coat and the visceral pleura. The visceral pleura if possible should be left intact if possible. Development of this plane invariably results in significant bleeding and often multiple air leaks. Once the correct plane is entered blunt dissection is generally adequate to liberate the collapsed lung.

After decortication is complete the anesthetist expands the lung gradually to identify any air leaks and to allow excision of any remaining restrictive peel. Injury to the lung parenchyma should be repaired if possible. At least one large-bore chest drain should be placed and sometimes two are necessary. The thoracotomy is then closed using standard techniques. Application of low-pressure suction to the drain helps lung expansion in presence of an air leak.

Major complications from decortication are related to persistent air leak, bleeding, and sepsis. Air leaks up to 10–14 days after surgery are not uncommon in advanced cases. Residual blood in the pleural space may become secondarily infected defeating the purpose of the operation because of ensuing fibrosis. While the mortality following decortication in experienced hands should be low, some series have reported mortalities between 1.3 and 10%.[27,129,131,132] Death is fortunately rare but invariably due to hemorrhage or septic complications.

A chronic empyema causes significant derangement of lung function. Both ventilation and perfusion of the collapsed lung are markedly reduced, particularly perfusion. Physiological improvement following successful decortication depends largely on the nature and extent of any residual parenchymal lung disease, which, in turn, depends on the infecting organism and the duration of the chronic empyema. Functional improvement continues for many months after decortication.

Thoracoplasty

This is rarely necessary in childhood empyema and is to be avoided if at all possible. There are two indications for thoracoplasty: First, if the underlying lung is so badly damaged that it is incapable of reexpansion despite decortication (this leaves a large residual intrapleural space, which will become reinfected unless the chest wall is collapsed), and second, if there is a bronchopleural fistula that fails to close after decortication. In this situation vascularized tissue must be brought into the thorax to cover the damaged lung surface. A pedicled muscle flap thoracoplasty is usually the best solution.

Management of Complex Empyemas

Empyema with Lung Abscess and Necrotizing Pneumonia

The outcome of this type of empyema depends on adequate postoperative lung expansion and appropriate antibiotic therapy. Pneumatoceles will generally resolve after debridement or decortication of the empyema.[109,133] Similarly most lung

abscesses can be managed along the same lines without the need for drainage. The morbidity involved in excision of a lung abscess in the presence of infection is significant.

Necrotizing pneumonia can only be identified accurately using CT with IV contrast. The presence of lung necrosis complicating an empyema is a poor prognostic factor. Prolonged antibiotic treatment is necessary. Bronchopleural fistulae are common in these children.[134] If lung necrosis is extensive ventilatory support may be necessary in the pre- and postoperative period, and this frequently results in a bronchopleural fistula.

Bilateral lung changes and empyemas occur occasionally in infants and young children. Mortality is significant in these children because of the extensive lung disease. Mechanical ventilation, high-frequency oscillation, and even extracorporeal membrane oxygenation (ECMO) may be necessary.

There are reports advocating resectional surgery of the damaged lung in this situation including pneumonectomy in children.[134,135] The associated mortality after the resection surgery is significant if undertaken during empyema drainage and only advisable in selected cases. In the western world thise situation is rare, and the management of such cases could be optimized by intensive care prior to surgery. Good medical management including resection of the affected lung in such cases in the underdeveloped country is life saving.[135]

Bronchopleural Fistula and Pyopneumothorax

A spontaneous bronchopleural fistula with resultant pyopneumothorax occasionally complicates a necrotizing pneumonia. Management of the bronchopleural fistula poses a major challenge with a high morbidity. The presence of an air leak into the pleural cavity and persistent pleural space contamination hinder lung reexpansion. Aspiration of infectious material may contaminate the opposite lung. Preoperative contrast CT scanning with IV contrast is essential to determine the presence of lung necrosis and a lung abscess. Conservative management of the fistula has been advocated in the past, along with various surgical approaches, all associated with prolonged hospitalization and morbidity.[136] The optimum management of a spontaneous bronchopleural fistula is drainage of pus from the pleural cavity with liberation of the lung to achieve complete expansion. Excision of the parietal pleura encourages the expanded lung to adhere to the chest wall and aids the healing of the fistula. Primary muscle-flap thoracoplasty can be helpful to bring a new blood supply to the necrotic area and promote healing.

Various muscle flap techniques have been described in adults to manage bronchopleural fistulae complicating pneumonectomy or lobectomy.[137–139] Chest wall and abdominal wall-based muscle flaps have been reported including trapezius,[140] intercostal muscle,[141] diaphragm,[141] pedicled diaphragm,[142] latissimus dorsi,[143] and rectus abdominis.[144] Other locally available tissues such as thickened pleura,[145] omentum, and jejunum have been reported to manage bronchopleural fistula.[146,147,148]

In children large muscle thoracoplasty is rarely feasible technically. Moreover, these would carry an unacceptable postoperative morbidity, as the grafts occupy a significant intrathoracic volume with permanent reduction in vital capacity. Apart from this major concern, flaps used in adults for myoplastic thoracoplasty require detailed anatomical knowledge and a separate incision to place them into the chest.

Flaps that occupy less intrathoracic volume suitable for use in childhood include pericardium, intercostal muscles, greater omentum, and thickened pleura. Although traditionally, a staged approach in the management of the bronchopleural fistula has been used in children, repeated thoracotomy and chronic infection lead to fibrosis and further increase the morbidity. Consequently, the author adopts aggressive one-stage approach for empyema-associated bronchopleural fistulae.[149]

An intercostal muscle flap can be raised through the thoracotomy incision at the time of decortication. However, this may complicate wound closure and lead to rib crowding with subsequent scoliosis. Flaps based on thickened pleura are poorly vascularized and difficult to position. The author's preference is a myoplastic flap based on serratus anterior. This muscle comprises a series of digitations with an axial blood supply and lends itself well to use as a myoplastic flap. Detailed anatomical knowledge is not required, and the flap can be raised easily through the thoracotomy incision.

The author has used this flap successfully over the years with good results and minimal morbidity.[149] However, depending on the underlying damage related to necrotizing pneumonia postoperative recovery varies.

These children require long-term monitoring for immunological deficiencies, bronchiectesis, and growth and development. Recurrent infections should be aggressively managed and if required should be immunized against specific bacteria such as pneumococci and *Haemophilus influenzae* as well as treating them with prophylactic antibiotics during winter months.

Serratus Anterior Myoplastic Flap Technique

The chest is opened though a conventional posterolateral thoracotomy, using either a muscle-cutting- or a muscle-sparing technique. A digitation of serratus anterior is then raised from its insertion from either the fifth or sixth rib. The muscle flap can be inserted into the thoracic cavity either through the thoracotomy wound itself or through a separate intercostal space incision, depending on the site of the fistula. Length on the muscle flap is easy to obtain by separating the digitations from the remainder of the serratus anterior muscle, leaving the posterior attachment intact. The muscle flap is loosely attached to the surrounding lung tissue to occlude the bronchopleural fistula. Both the blood and nerve supply to serratus anterior are segmental so that separation of one or more digitations in this fashion does not impair function of the remainder of the muscle. No special precautions are necessary closing of the thoracotomy.

Bilateral Empyema

Bilateral parapneumonic empyema is occasionally encountered in infants and immunocompromised children. However, the commonest cause of bilateral empyema in children is esophageal injury, either a leaking anastomosis or perforation following dilatation of a stricture. Caustic strictures are particularly prone to perforation during dilatation. In a large study of 1,249 caustic strictures reported by Avnoglue, 52 dilatations resulted in perforation. Seventeen unilateral and two bilateral empyemas occurred as a consequence.[22] The mortality from sepsis in this series was 23%. In another smaller series of 98 caustic esophageal strictures 14 patients required repeated balloon dilatations. Three perforations occurred with one bilateral empyema.[150]

Bilateral parapneumonic empyema is uncommon.[107,151,152] The infective organism is usually staphylococcus, pneumococcus or, rarely, *Pseudomonas aerugenosa*.[153] Staphylococcal infection is more common in malnourished children.[38] The author has managed three children with bilateral empyemas, including a bulimic adolescent girl with recurrent aspiration and multiple bilateral abscesses, lung abscess,[154] and a two-year-old girl with bilateral pneumonia and septic shock, who needed ECMO after conventional ventilation, which resulted in a tension pneumothorax.

Bilateral empyema is seen as a common complication of necrotizing mediastinitis.[155,156] Necrotizing mediastinitis occurs as a result of descending infection from the retropharyngeal plane of the neck. The condition has a 30–40% mortality, and aggressive surgical management is necessary.[155–157] Bilateral empyema has been successfully managed with VATS debridement.[158]

Secondary Empyema

The commonest cause of secondary empyema in children is infection of a posttraumatic lung contusion.[20,21] Postlobectomy empyema occasionally occurs after resection of a bronchiectatic lobe or after lung resection in immunologically compromised children. Penetrating injuries of the chest may result in an empyema. Infection of sympathetic effusions following a subphrenic abscess or acute hemorrhagic pancreatitis have been reported. Amebic empyemas may follow rupture of a liver abscess.[23,24]

Management of secondary empyemas has to be tailored to the underlying cause. Most sympathetic effusions that become infected will resolve with intercostal drainage and antibiotics, and treatment of the underlying cause, occasionally requiring VATS or thoracotomy drainage, is required.[159] Posttraumatic empyemas invariably require surgical intervention with early thoracotomy or VATS.[21] Esophageal rupture inevitably results in mediastinitis and empyema. Immediate management comprises antibiotics and closed chest drainage but definitive treatment depends on the state of the esophagus, which is discussed elsewhere in this book.

Outpatient Follow-Up After Empyema

Children with empyema should be seen at 4–6 weeks after discharge, with subsequent follow-up depending upon clinical and radiological improvement.

Attention to nutrition is essential. Iron supplements may be necessary if the child is anemic. Oral antibiotics should be continued for a period of 4 weeks after discharge from hospital. Children should be evaluated for immune deficiencies at follow-up as previously healthy children may have undiagnosed abnormalities, including cystic fibrosis.[107] Antibody status for pneumococcal and *H. influenza* should be assessed and vaccinations provided if necessary.

The long-term prognosis following treatment of empyema is excellent in the majority, despite the heterogeneity of management.[160,161] Complex empyema, however, carries a significantly higher morbidity and longer hospital stay, and some of these children require prophylactic antibiotics during winter months to prevent further respiratory infections. Although the vast majority of children recover without any long-term respiratory consequences some studies reported mild obstructive defects associated with poor exercise tolerance.[162,163] In another study most children were found to have normal respiratory function by 1 year although 19% had mild restrictive defects and 16% mild obstructive defects. None of these children had symptoms of respiratory insufficiency.[164]

Conclusion and Future Perspective

Adequate drainage of the abscess (empyema) within the pleural space and reexpansion of collapsed lung are the fundamental principles of empyema management. Despite the fact that this has been known since the time of Hippocrates, the management of empyema thoracis in children remains controversial. In part, this is because of reluctance on the part of physicians to relinquish their patients to surgeons but also because of the lack of randomized trials in this field. The number of studies trying to reinvent the wheel rather than learning form past experience is depressing.

Minimally invasive surgery (VATS) is slowly changing the management of empyema in the developed world in favor of early intervention. The use of small-bore catheters and fibrinolytics probably has a role in the early stages of empyema but these children must be monitored closely and referred promptly for surgery if the response is less than perfect. The incidence of childhood empyema is rising in the western world, and there is urgent need for randomized controlled clinical trials to guide treatment.

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