Thoracic Empyema

28

Francesco Molinaro and François Becmeur

28.1 Introduction

Empyema thoracis is an accumulation of pus within the pleural cavity. The correct definition of empyema is the presence of pus in the pleural space. There are various criteria to define and classify parapneumonic pleural effusions [1].

It may result from a direct extension of infective agents from the lung parenchyma or bronchial tree, ruptured intrathoracic abscesses, hematologic spread or contamination resulting from surgery or trauma and mediastinal sources (esophageal perforation).

Approximately 0.6% of pneumonia episodes in children are complicated by empyema. The incidence of parapneumonic empyema in children is 0.4–6 per 1,000 hospital admissions [2]. Moreover, the incidence of empyema thoracis in children has increased significantly in recent years in the western world. The incidence of empyema is around 3.3 cases per 100,000 children [1].

The microbiology of childhood empyema dictates appropriate antibiotic selection. *Haemophilus influenzae*, *Staphylococcus aureus*, and *Streptococcus pneumoniae* remain the most common pathogens cultured in empyema [3].

Empyema can been divided into three stages. In stage 1 (exudative stage), there is a simple parapneumonic effusion with normal levels of glucose and a correct pH in pleural fluid. In stage 2 (fibrinopurulent stage), there is an increase in fibrin levels and the number of polymorphonuclear leukocytes. At this stage, the fluid in the pleural space begins to loculate. There also are associated changes in the chemistry of pleural fluid: glucose levels and the pH decreases and lactate dehydrogenase (LDH) levels increase. In stage 3 (organizing stage), fibroblasts grow from both pleural surfaces, forming an inelastic pleural "peel" or "rind". This results in entrapment of the involved lung [4].

Physical examination and laboratory analyses of pleural fluid aid the diagnosis. CT of the chest frequently provides others information. Recently, the use of ultrasonography has become increasingly helpful, but remains operator-dependent. Radiological images can define the pleural rind and locate its cavities.

The morbidity and mortality of this condition have undoubtedly improved over recent years, the nature and timing of surgical intervention is controversial.

In the past, empyema was treated with antibiotics, prolonged drainage of the chest

F. Becmeur (✉)
Pediatric Surgery
Hôpital de Hautepierre
University of Strasbourg, France
e-mail: Francois.Becmeur@chru-strasbourg.fr

M. Lima (ed.), *Pediatric Thoracic Surgery*,
DOI: 10.1007/978-88-470-5202-4_28, © Springer-Verlag Italia 2013

and, if this failed, open thoracotomy for debridement. This was associated with long hospitalizations and significant morbidity due to the delayed referral to a surgeon and to the surgical procedure. With the expanded use of endoscopic techniques, thoracoscopy has been increasingly used in the treatment of empyema and much earlier in the course of the disease [5].

The role of intrapleural fibrinolytic agents for the treatment of empyema is not well defined because this type of therapy could fail with more advanced disease. Some authors, because fibrinolytic therapy can be used to dissolve fibrin-produced loculations, suggest attempting this therapy before video-assisted thoracoscopic surgery (VATS). Flexible bronchoscopic evaluation with bronchoalveolar lavage fluid (BALF) can be undertaken before the surgical procedure to evaluate associated endobronchial lesions [6]. VATS enables the removal of pus, elimination of loculations, and insertion of a chest drain. The pleural effusion can be used to test for bacteria (aerobic and anaerobic) and fungal cultures.

Air leaks and residual pneumonia are the most common complications. The prevalence of mortality is 0–13%. Aggressive postoperative pulmonary therapy and increased activity benefit patients. The long-term outcomes of patients treated by VATS are encouraging [5].

28.2 Etiology

Usually, empyemas follow acute bacterial lobar pneumonia. Underlying conditions with associated immune suppression could predispose a child to empyema. The most common causative organism is *Streptococcus pneumoniae*; less common agents are Gram-negative bacteria and fungi [6].

Empyema can follow an infection from a traumatic hemothorax, lung contusion, a penetrating injury to the chest, or after infection in the pleural space following thoracotomy. A secondary empyema in children could follow intrathoracic rupture of the esophagus as a result of a leaking anastomosis or rupture after dilatation of an esophageal stricture. Peritonitis caused by acute appendicitis could lead to empyema as a result of reduced host resistance, postoperative pneumonia, or local spread of infection through the diaphragm. Sometimes, tuberculous effusions present with secondary bacterial infection. Similarly, hydatid cysts may rupture into the pleural space or become infected due to a secondary infection [7].

28.3 Pathogenesis

The variation in presentations of pleural empyema is dependent upon the virulence of the organisms, resistance of the host, and use of appropriate antibiotics and drainage procedures.

28.3.1 Stages

Exudative stage: The inflammation caused by pneumonia results in increased capillary permeability [8]. Accumulation of fluid and cells into the pleural space from the visceral pleura leads to the exudative phase of the empyema. The fibrinolytic system is activated by the products of inflammation, and capillary permeability is increased further [9].

Fibropurulent stage: The coagulation cascade is activated as inflammation increases with suppression of fibrinolysis, which favors fibrin deposition in the pleural cavity. A certain membrane called the "pyogenic membrane" proliferates due to the accumulation of fibroblasts, phagocytes, bacteria, and fibrin. 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 [10].

Organization phase: Fibroblast proliferation leads to the formation of fibrous tissue. This process localizes the infection. The fibroblasts deposit layers of fibrous tissue (rinds) on the visceral and parietal pleura and

within the empyema cavity. The fibrous rind encases the collapsed lung and prevents it from re-expanding. As the empyema continues to organize, the fibrous rind thickens with further lung collapse and restriction of chest-wall movement [11].

28.3.2 Extrapulmonary Complications

Convulsions, osteomyelitis, toxic shock syndrome, disseminated intravascular coagulation, gastric hemorrhage, thrombosis, and multiple organ failure are extrapulmonary complications of staphylococcal and streptococcal pneumonia with overwhelming sepsis [12].

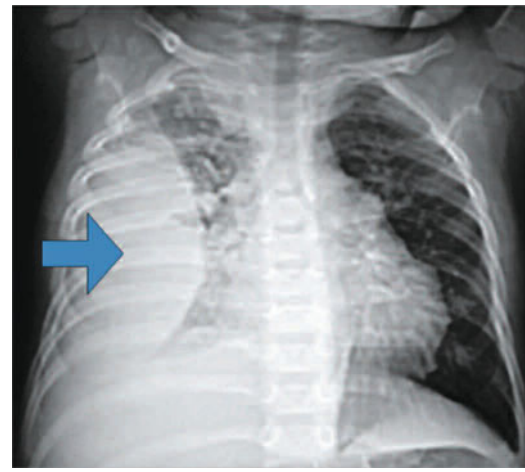

Fig. 28.1 Chest radiograph showing a right large pleural effusion (*arrow*)

28.4 Clinical Features

In children, symptoms related to pneumonia such as fever, cough, malaise, and loss of appetite, precede symptoms related to pleural empyema. Tachypnea, fever and lethargy can be the main early symptoms in younger children. Some children also complain of pleuritic chest pain or abdominal pain. Possible contact with subjects infected with tuberculosis should be examined. The possibility of an inhaled foreign body should not be forgotten [13]. Some children will be cyanosed and for this reason measurement of oxygen saturation (SaO_2) by pulse oximetry is particularly important.

The thorax can appear decreased in chest expansion, with stony dullness to percussion, and reduced (or absent) breath sounds. A mediastinal shift may be detectable by tracheal deviation and displacement of the apex beat to the opposite side. Consolidation of the underlying lung causes bronchial breathing and reduced air entry that is apparent on auscultation [14].

28.5 Diagnosis

Blood tests and radiological parameters are very important for the diagnosis. Neutrophilia is frequently present and is associated with normochromic normocytic anemia. Levels of C-reactive protein (CRP) and other inflammatory markers such as procalcitonin are also elevated. The serum albumin is invariably low. Coagulation abnormalities may be present and must be recognized prior to surgery.

A plain radiograph of the chest is usually the first imaging investigation. Pneumonic consolidation will be seen (Fig. 28.1). A mediastinal shift with large effusions can be present. Lung abscesses, pneumatoceles, and pyopneumothorax are all visible on a chest radiograph [15].

Ultrasonography is particularly valuable: ultrasound can be used to identify fluid in the pleural space. Ultrasound is most useful to ascertain if an effusion is loculated (Figs. 28.2 and 28.3). This examination is also useful for guiding insertion of a chest drain or carrying out thoracocentesis (Fig. 28.4) [16].

Contrast-enhanced CT complements ultrasonography for imaging children with complicated pneumonia. CT gives more important information about anatomical extension of the disease, clearly showing the lung parenchyma and pleural wall, a mediastinal shift, and bronchopleural fistulae (Figs. 28.5–28.7) [14].

Cultures of sputum, blood and, if possible, pleural fluid, can be carried out routinely.

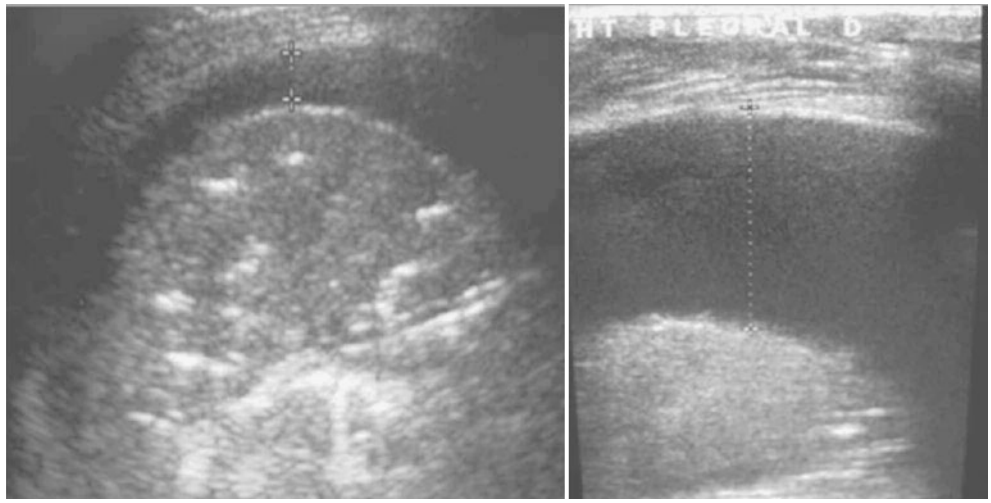

Fig. 28.2 Ultrasound evaluation showing an echogenic focus that has resulted in a pleural effusion

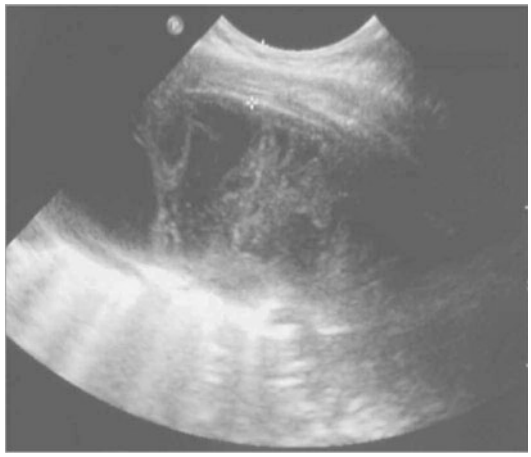

Fig. 28.3 Ultrasound evaluation showing a central zone which represents pus

Microbial cultures should always include tests for bacteria and tuberculosis. A Mantoux test should be carried out if tuberculosis is suspected [13].

28.6 Management

Early recognition and treatment of lobar pneumonia in children reduces the incidence of empyema. Appropriate antibiotic therapy is essential as a first step of the management of empyema in children. Response to antibiotics is dependant upon the pathogen involved, empyema stage, and immune status. In the early exudative stage, high concentrations of antibiotics alone may be effective treatment. However, antibiotics are unlikely to be effective in more advanced disease without surgical intervention [17].

Recommendations for antibiotic therapy for childhood empyema are lacking. A first-line treatment of a generic antibiotic is usually adopted (such as cephalosporin given intravenously). Therapy should then be altered depending on the sensitivity of the pathogen isolated [18].

Along with antibiotic therapy, medical support with humidified oxygen, fluids, analgesia and physiotherapy is required. Antipyretics should be given regularly. Children who fail to improve clinically and upon imaging findings should be referred to a center with expertise in the drainage procedures for empyema [17].

28.6.1 Surgical Management

28.6.1.1 General Principles

Thoracoscopic debridement has significantly changed the treatment of empyema in chil-

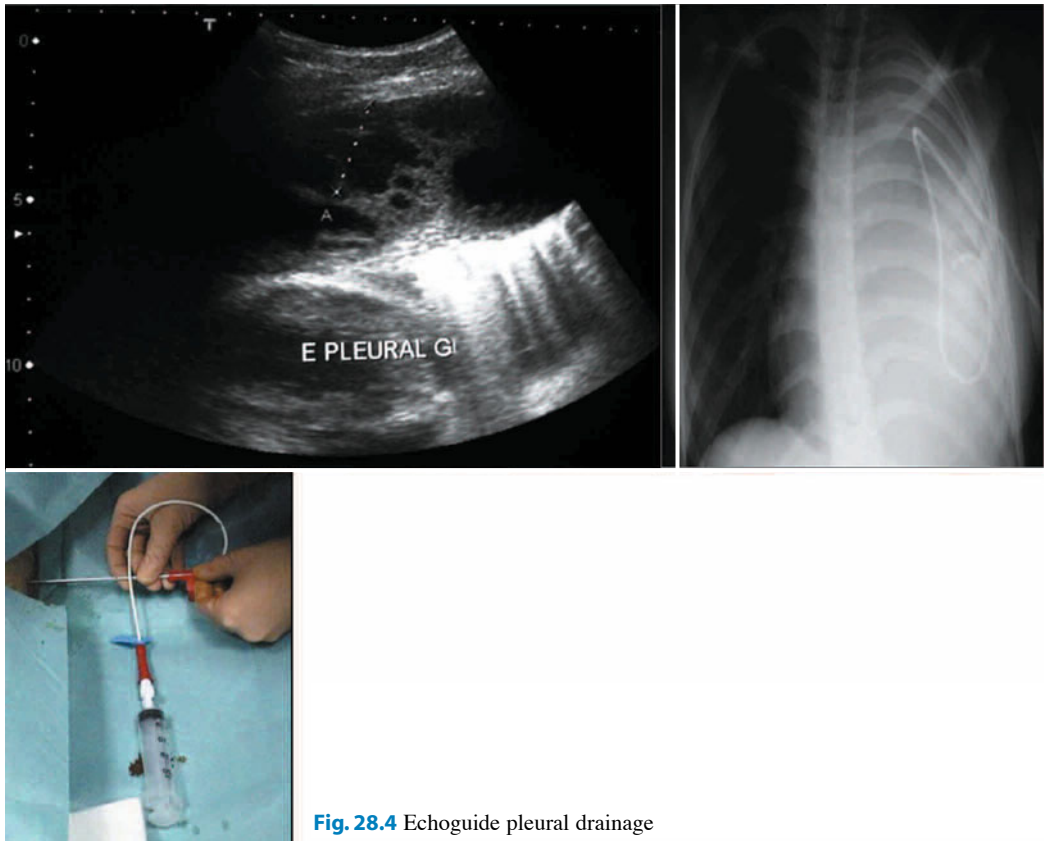

Fig. 28.4 Echoguide pleural drainage

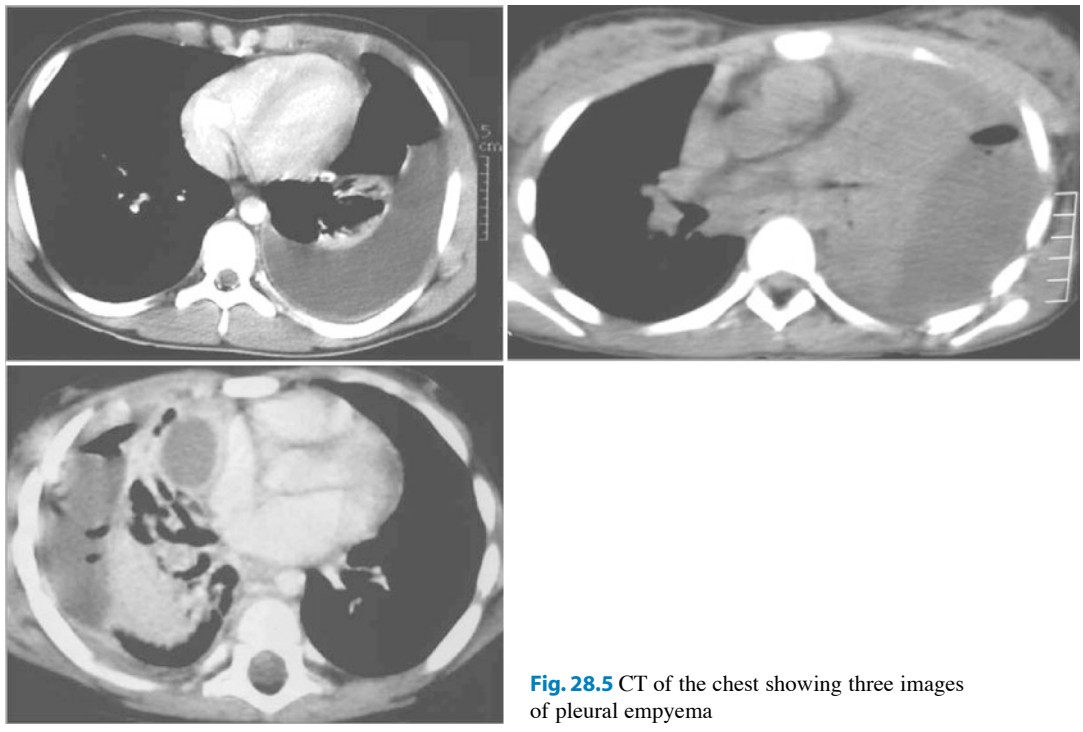

Fig. 28.5 CT of the chest showing three images of pleural empyema

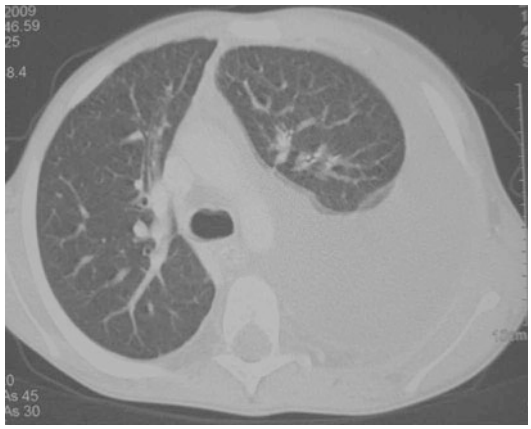

Fig. 28.6 CT of the chest showing a large mediastinal shift

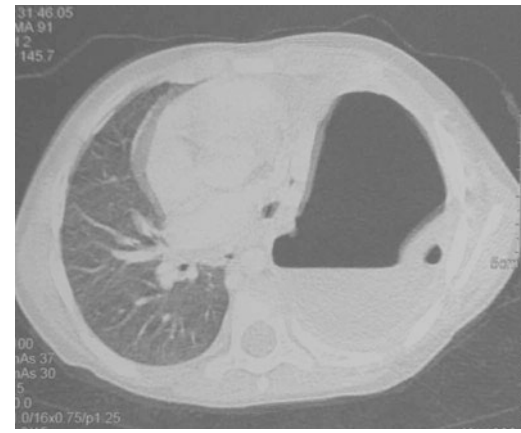

Fig. 28.7 CT of the chest showing a bronchopleural fistula

dren. The ability to quickly clean out infected fluid and debris through minimal incisions with minimal morbidity has lowered the threshold for intervention. In general, any child thought to need a chest tube for drainage of empyema is taken to the operating room for thoracoscopic debridement. Earlier intervention has resulted in quicker recovery with minimal complications [18]. The goal of surgery is to achieve full expansion of the lung and resolution of empyema. An added advantage of the procedure is that the lung parenchyma may itself be readily evaluated.

The time the child is brought to the surgeon is very important for the timing of surgery. In the fibrinopurulent phase, even drainage of the pus with lung expansion is imperative to reduce progression of the disease to the organization phase [19]. If surgical consultation is delayed for a prolonged period of time, thoracoscopic drainage and debridement may not be possible and open decortication may be the only option. This can, however, be assessed by thoracoscopy.

For these reasons, 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 thoracotomy [19, 20].

However, one of the major criticisms of an all-inclusive surgical approach is that children who may otherwise respond to less-invasive therapies (i.e., those with stage-I and -II empyema) are subjected to general anesthesia and surgery. Because of their active lung infections, these patients are an increased anesthesia risk (American Society of Anesthesiologists (ASA) class II and III). In addition, these children are catabolic because of stress, making recovery from surgery slower [18].

Successful treatment of empyema has also been described for some children receiving tube drainage alone or with adjuvant fibrinolytic therapy. Therefore, if some patients are treated successfully with these less-invasive methods and have comparable outcomes, then VATS can be reserved for patients truly needing this procedure.

Ultrasound can be used to characterize pleural disease. With higher sonographic grades of empyema, the chance of successful treatment with tube drainage and fibrinolytic agents decreases. As suggested by Gates et al., ultrasound characterization can help guide therapy (tube drainage *versus* surgical consultation). If the likelihood of resolution of pleural disease is low based on the characterization of solid peel, tube drainage should not be attempted [16]. However, if the pleural disease does not appear to be predominately organized, tube drainage can be done. There are conflicting studies concerning the use of intrapleural fibrinolytics. Fibrinolytic agents are

adjuncts used to improve drainage, and their use should not change the underlying principles of empyema management [19].

Chen et al. found that >70% of children with late-presenting empyema required surgery, and that more than half of the children received initial chest tube drainage. Delay in surgery was associated with more procedures, more radiographs, and an increased length of stay in hospital. Despite later intervention, patients undergoing surgery as an initial approach had the shortest length of stay in hospital. Early surgical intervention is indicated for most children referred with established empyema [20].

28.6.1.2 VATS

The use of VATS as an initial intervention in children with empyema has obvious diagnostic and therapeutic advantages. However, there remains strong opposition to this indication because of the lack of randomized trials. A multicenter prospective study is required to define the most effective method of assessment of pediatric parapneumonic empyema and the most successful treatment at each stage of the disease process.

Early VATS can give good lung re-expansion and improve drainage of the empyema. Loculi can be separated, allowing thick pus to drain effectively. Several series have shown reduced postoperative pain, shorter stay in hospital, and better cosmetic results when VATS is compared with conventional thoracotomy [21]. Although most studies have been retrospective with small cohorts of patients, VATS has proven effective, with minimal complications, and is well tolerated. Conversion to open thoracotomy is necessary if access to the pleural cavity cannot be achieved because of a thick pyogenic rind or excessive bleeding. Mini-thoracotomy and debridement or decortication in these instances is safer and curative [22].

Debridement using VATS: Patients are brought to the operating room. General anesthesia with simple or double-lumen endotracheal intubation is initiated, and patients placed in a full or semilateral position according to empyema location (Fig. 28.8).

Selective endobronchial intubation (or the use of bronchial blocker in young children if double-lumen tubes are not available) may be useful in selected cases to avoid contamination of the contralateral lung. If the patient already has a chest tube *in situ*, this can be removed prior to positioning and preparation of the patient. Also, the site may be used for placement of one of the ports. A suction trap should be prepared to obtain a sample of pleural fluid for cell counting, Gram stain, and culture if this has not been done previously [20].

The surgeon may stand on either side of the patient. The surgical assistant is usually positioned on the other side of the table to hold the camera. The scrub nurse is toward the patient's feet, on either side.

The thoracic area is prepared and draped for possible open thoracotomy. The first 5-mm port is placed on the estimated location of fluid. A sample is collected for biochemical and cytologic examination, as well as bacterial, mycobacterial, and fungal smear and culture. This port is usually inserted in the fifth or sixth intercostal space in the posterior axillary line. Creation of a working space is essential (Fig. 28.9). A 0° telescope is usually employed for visualization and insufflation with CO_2 at a pressure of 5–8 mmHg. The VATS approach to empyema uses one scope port with or without an additional port to three working port(s) or utility incision. The thoracoscope with a working channel or angled end-viewing thoracoscope is used to facilitate initial dissection or examination. A second port for instrumentation is placed under thoracoscopic visualization. Two ports are usually sufficient if debridement is done early. Figures 28.10–28.12 show the three stages of empyema using VATS.

Laparoscopic instruments are used for the lysis of adhesions from the chest wall and breakage of fibrous septa. Sometimes, a thick membrane is seen encasing the lung and inhibiting expansion; a peanut sponge is used to gently peel the membrane off the visceral

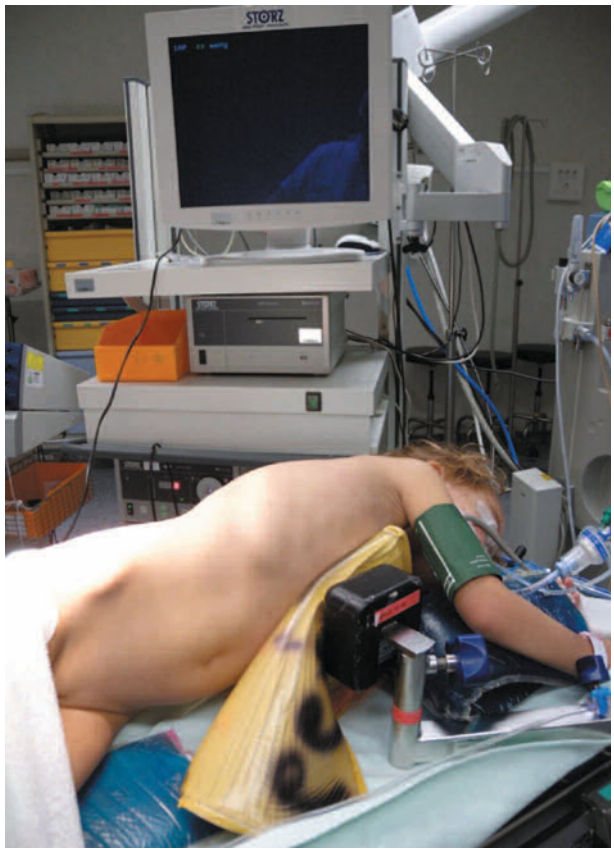

Fig. 28.8 VATS: patient positioning

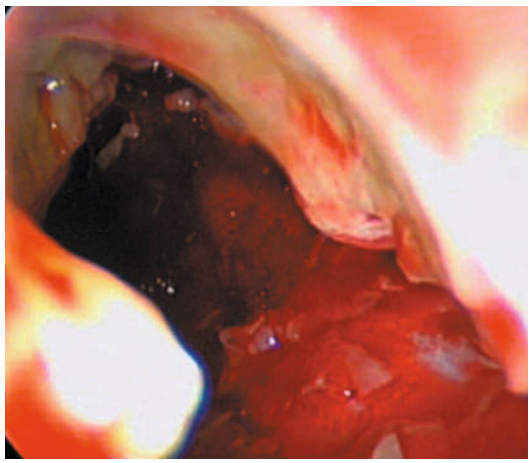

Fig. 28.9 VATS. Intraoperative image: creation of working space

pleura. A curved large-bore suction tube with or without connection to a 32-F chest tube is introduced into the chest cavity for suction. Peels on the parietal or visceral pleura are removed for re-expansion of trapped lung and for debridement (decortication) (Fig. 28.13). Pleural or lung biopsy is done if the underlying cause is in doubt [20, 21].

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

The chest drain should remain *in situ* until the losses reduce to <30 mL/day and become clear. The child is kept in hospital on intravenous antibiotics until afebrile for ≥24 h. Chest radiography should be carried out after drain removal [20].

Conversion to open thoracotomy is indicated for failure of separation of the fibrous rind from the visceral pleura after thoracoscopic

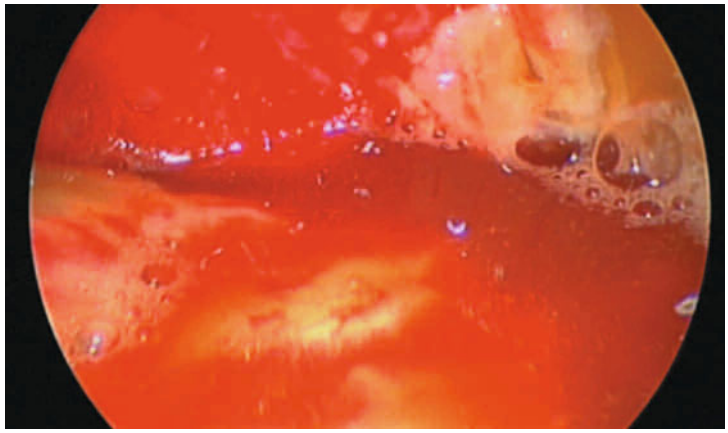

Fig. 28.10 VATS showing stage 1 of empyema: exudative stage

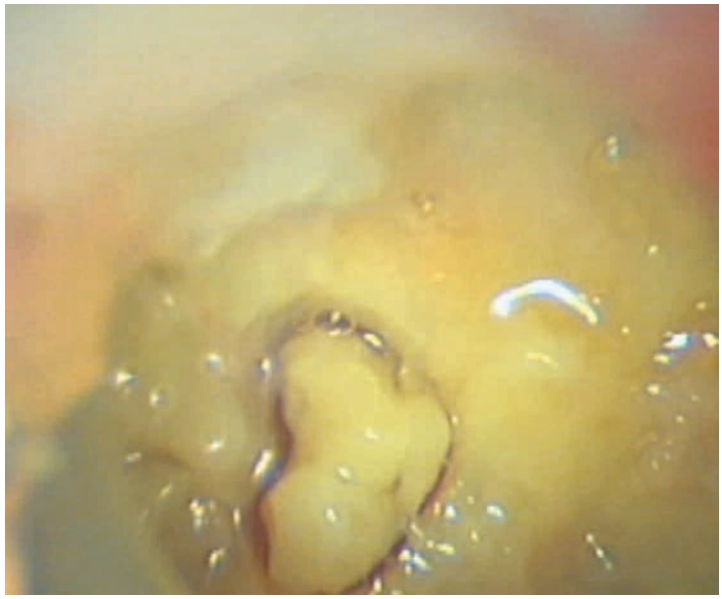

Fig. 28.11 VATS showing stage 2 of empyema: fibropurulent stage

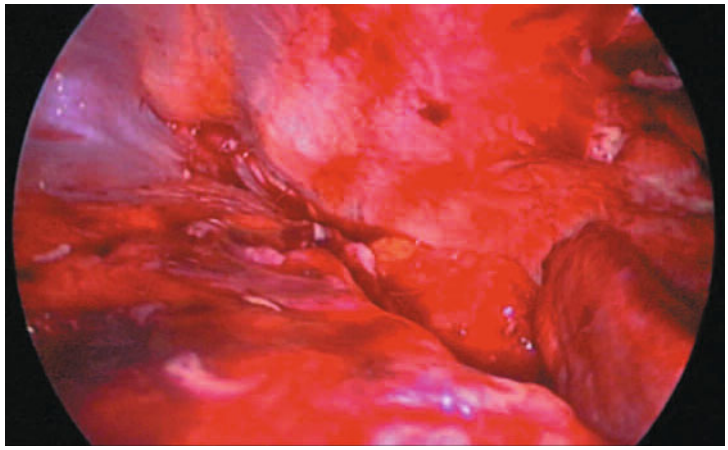

Fig. 28.12 VATS showing stage 3 of empyema: organization stage

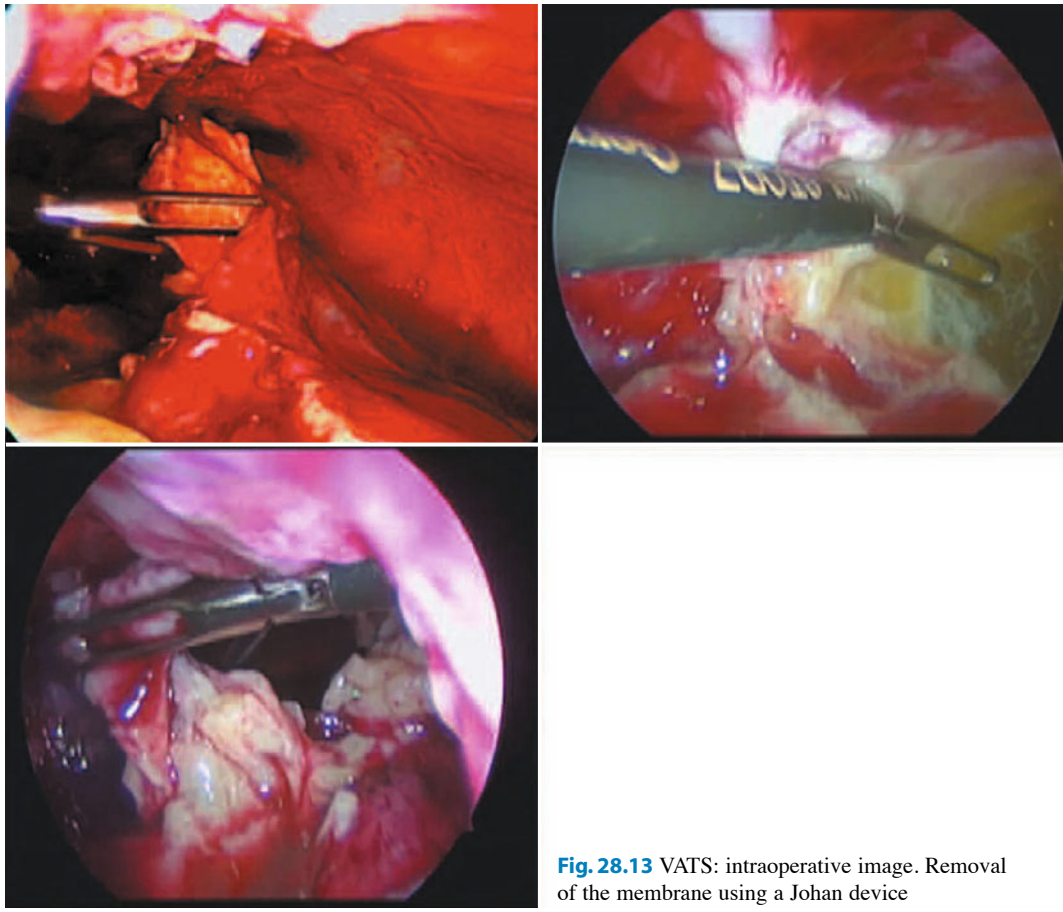

Fig. 28.13 VATS: intraoperative image. Removal of the membrane using a Johan device

maneuvers, excessive bleeding, inadequate visualization, and failure of lung re-expansion [23].

28.6.2 Management of Complex Empyemas

28.6.2.1 Bronchopleural Fistulae

A spontaneous bronchopleural fistula occasionally complicates necrotizing pneumonia (Fig. 28.14). Management of a bronchopleural fistula poses a major challenge and is associated with high morbidity. An air leak into the pleural cavity and persistent contamination of the pleural space hinder lung re-expansion. Aspiration of infectious material may contaminate the opposite lung. Preoperative contrast-enhanced CT is essential to determine lung necrosis and lung abscesses. Conservative management of the fistula has been advocated in the past, along with various surgical approaches, but all are associated with prolonged hospitalization and morbidity. 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. Sometimes, it is possible to suture the lung parenchyma to close the fistula (Fig. 28.15). Excision of the parietal pleura encourages the expanded lung to adhere to the chest wall and aids healing of the fistula [17].

28.6.2.2 Bilateral Empyema

Bilateral parapneumonic empyema is encountered occasionally in infants and immunocom-

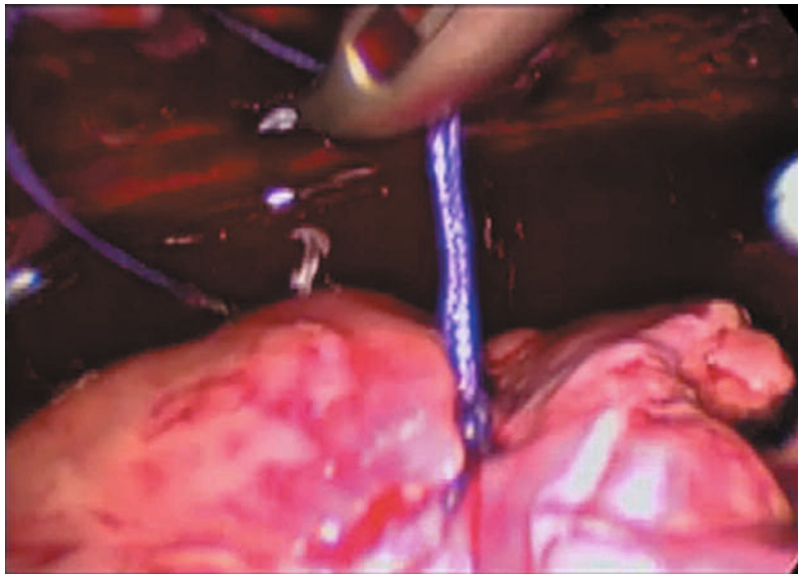

Fig. 28.15 VATS: intraoperative image. Closing of a bronchopleural fistula with a stitch

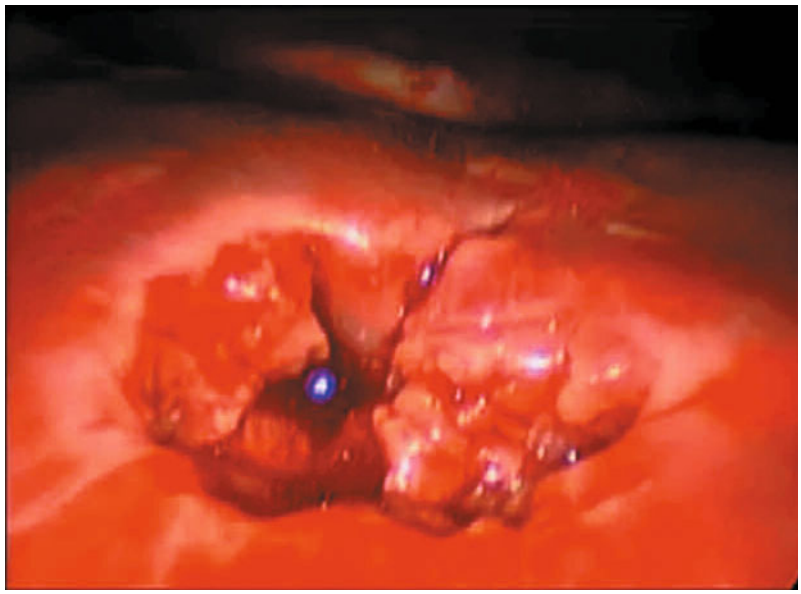

Fig. 28.14 VATS: intraoperative image. Bronchopleural fistula

promised children. However, the most common cause of bilateral empyema in children is esophageal injury, either a leaking anastomosis or perforation after dilatation of a stricture. Caustic strictures are particularly prone to perforation during dilatation.

Bilateral parapneumonic empyema is not common. The infective organism is usually staphylococcus, pneumococcus or, rarely, *Pseudomonas aerugenosa*. Staphylococcal infection is more common in malnourished children. Bilateral empyema is seen as a common complication of necrotizing mediastinitis. Necrotizing mediastinitis occurs as a result of descending infection from the retropharyngeal plane of the neck. Bilateral empyema has

been managed successfully with VATS debridement [24].

28.7 Follow-up

Children with empyema should be seen 4–6 weeks after hospital discharge, with subsequent follow-up depending upon improvement based on evaluation of clinical and findings. Attention to nutrition is essential. Iron supplements may be necessary if the child is anemic. Antibiotics (p.o.) should be continued for 4 weeks after discharge from hospital [18]. The long-term prognosis after treatment for empyema is excellent in most subjects despite the different methods of management. Complex empyema, however, carries a significantly higher morbidity and longer stay in hospital, and some of these children require prophylactic antibiotics during the winter months to prevent further respiratory infections [20].

28.8 Conclusion

The rapid identification of patients at risk of complicated parapneumonic effusions or empyema can improve outcome by allowing early drainage of the pleural space. The findings of chest radiography and CT as well as the characteristics of pleural fluid suggest that a parapneumonic effusion has a high likelihood of requiring drainage.

Use of VATS as the initial intervention in children with empyema has obvious diagnostic and therapeutic advantages. Adequate drainage of the abscess (empyema) within the pleural space and re-expansion of the collapsed lung are the fundamental principles of empyema management. However, there remains strong opposition because of the lack of randomized trials showing efficacy, which makes physicians reluctant to relinquish their patients to surgeons. A multicenter prospective study is required to define the most effective method of assessment of pediatric parapneumonic empyema as well as the most successful treatment at each stage of the disease process.

VATS is a safe and effective procedure for the treatment of complicated parapneumonic effusions or empyema. Earlier intervention using VATS to treat complicated parapneumonic effusions or empyema can elicit better results than the other methods. Additional prospective, randomized, or controlled studies should be carried out to identify the optimal timing and conditions of VATS for complicated parapneumonic effusions or empyema.

References

1. Byington CL, Spencer LY, Johnson TA et al (2002) An epidemiological investigation of a sustained high rate of pediatric parapneumonic empyema: Risk factors and microbiological associations. Clin Infect Dis 34:434–440
2. Lewis KT, Bukstein DA (1992) Parapneumonic empyema in children: diagnosis and management. Am Fam Physician 46:1443–1455
3. Hoth JJ, Burch PT, Bullock TK, Cheadle WG, Richardson JD (2003) Pathogenesis of post traumatic empyema: the impact of pneumonia on pleural space infections. Surg Infect 4:29–35
4. Mandal AK (1997) Post-traumatic empyema thoracis: a 24 year experience at a major trauma centre. J Trauma 43:764–761
5. Avanoglu A, Ergun O, Mutaf O (1998) Management instrumental perforations of the esophagus occurring during treatment of corrosive strictures. J Pediatr Surg 33:1393–1395
6. Kroegel C, Anthony VB (1997) Immunobiology of pleural inflammation: potential implications for pathogenesis, diagnosis and therapy. Eur Resp J 10:2411–2418
7. Quadri A, Thomson AH (2002) Pleural fluids associated with chest infection. Paediatr Resp Rev 3:349–355
8. Joosten KFM, Hazelzet JA, Tiddens Harm AWM et al (1995). Staphylococcal pneumonia in childhood: will early surgical intervention lower mortality? Pediatr Pulmonol 20:83–88
9. Wong KS, Chiu CH, Yeow KM, Huang YC, Liu HP, Lin TY (2000) Necrotising pneumonitis in children. Eur J Pediatr 159:684–688
10. Buckingham SC, King MD, Miller ML (2003) Incidence and etiologies of complicated parapneumonic effusions in children, 1996 to 2001. Paediatr Infect Dis J 22:499–504
11. Tiryaki T, Abbasoglu L, Bulut M (1995) Management of thoracic empyema in childhood. A study of 160 cases. Pediatr Surg Int 10:534–536
12. Bai KJ, Wu IH, Yu MC (1998) Tuberculous empye-

ma. Respirology 3:261–266
13. Kearney SE, Davies CWH, Davies RJO, Gleeson FV (2000) Computed tomography and ultrasound in parapneumonic effusions and empyema. Clin Radiol 55:542–547
14. Akhan O, Demirkazik FB, Ozmen MN (1992) Tuberculous pleural effusions: Ultrasonic diagnosis. J Clin Ultrasound 20:461–465
15. Kilic N, Celebi S, Gurpinar A et al (2002) Management of thoracic empyema in children. Pediatr Surg Int 18:21–23
16. Gates RL, Hogan M, Weinstein S, Arca MJ (2004) Drainage, fibrinolytics, or surgery: a comparison of treatment options in pediatric empyema. J Ped Surg 39: 1639–1642
17. Angelillo-Mackinlay T, Lyons GA, Chimondeguy DJ et al (1996) VATS debridment versus thoracotomy in the treatment of loculated post pneumonia empyema. Ann Thorac Surg 61:1626–1630
18. Merry CM, Bufo AJ, Shah RS, Schropp KP, Lobe TE (1999) Early intervention by thoracoscopy in pediatric empyema. J Ped Surg 34:178–181
19. Chen LE, Langer JC, Dillon PA et al (2002) Management of late-stage parapneumonic empyema. J Ped Surg 37:371–374
20. Luh SP, Chou MC, Wang LS, Chen JY, Tsai TP (2005) Video-assisted thoracoscopic surgery in the treatment of complicated parapneumonic effusions or empyemas: outcome of 234 patients. Chest 127:1427–1432
21. Kern JA, Rodgers BM (1993) Thoracoscopy in the management of empyema in children. J Pediatr Surg 289:1128–1132
22. Stovroff M, Teague G, Heiss KF, Parker P, Ricketts RR (1995) Thoracoscopy in the management of pediatric empyema. J Pediatr Surg 30:1211–1215
23. Eerola S, Virkkula L, Varstela E (1988) Treatment of postpneumonectomy empyema and associated bronchopleural fistula. Experience with 100 consecutive pneumonectomy patients. Scan J Thorac Cardiovasc Surg 22:235–239
24. Hankins JR, Miller JE, Attar S, Satterfield JR, McLaughlin JS (1978) Bronchopleural fistula. Thirteen year experience with 77 cases. J Thorac Cardiovasc Surg 76:755–762