# Empyema and parapneumonic effusions in children: an update

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Childhood empyema is an important complication of bacterial pneumonia. The incidence of empyema is increasing worldwide. *Streptococcus pneumoniae* and *Staphylococcus aureus* are the most common aetiologies in high- and low-income countries, respectively. The diagnosis is based on clinical, radiographic and pleural fluid examination. Tuberculosis (TB) is an important cause of a pleural effusion in high TB prevalence areas. There is controversy about the optimal treatment for empyema in children. Sepsis should be controlled with antibiotics and drainage of the pleural cavity. Intrapleural fibrinolysis and video-assisted thoracoscopic surgery (VATS) are modern interventions widely used in high-income countries, but mostly unavailable in the developing world. There are however few properly conducted studies that would support one therapeutic approach over the other. Despite this, the clinical outcome of paediatric empyema is usually good regardless of therapeutic approach. This review summarises aetiology, pathogenesis and clinical presentation of childhood empyema and discusses the various treatment modalities with an emphasis on clinical practice in developing countries.

Lower respiratory tract infections (LRTIs) are a leading cause of morbidity and mortality in children throughout the world. In developing countries poverty, HIV infection and lack of universal access to new vaccines contribute to the high incidence of severe and complicated pneumonia. Parapneumonic effusion (PPE) and empyema most frequently occur as a complication of bacterial pneumonia. The lack of consensus on the optimal management of PPE and empyema has generated renewed interest in and research on the subject, especially with the use of modern interventions such as fibrinolysis and video-assisted thoracoscopic surgery (VATS). Research and experience with these interventions are however limited to resource-rich countries. This article reviews the aetiology, pathophysiology and management of empyema in children, with an emphasis on clinical practice pertinent to resource-limited settings such as South Africa.

## Aetiology

Empyema is defined as the presence of pus in the pleural space. A sterile pleural effusion associated with pneumonia with few or no inflammatory cells is termed PPE. Empyema develops as a complication of bacterial pneumonia in 0.6 - 3% of hospital admissions but small pleural effusions may be present in up to 40% of bacterial pneumonias.[1-3] Data from developing countries are limited, but studies from Brazil and South Africa have reported pleural effusions and PPEs in 15% and 8%, respectively, of children hospitalised with pneumonia.[4,5] The aetiology of empyema thus closely correlates with that of community-acquired pneumonia. Although *Streptococcus pneumoniae* is the predominant cause of bacterial pneumonia throughout the world, it is most commonly implicated as the cause of PPE and empyema in developed countries and certain middle-income countries such as Brazil.[2,5] The incidence of empyema caused by *S. pneumoniae* is reported to be increasing – for poorly understood reasons. Reduced antibiotic use at primary care level, earlier tertiary care referral, and increased use of the polyvalent pneumococcal vaccine are thought to play a role.[1,6] Furthermore, *S. pneumoniae* serotype-1 (a conjugate vaccine serotype) and community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) have shown a proportional increase as the cause of PPE and empyema.[7-12] Prior antibiotic use will frequently result in sterile pleural fluid, but more sensitive diagnostic methods such as polymerase chain reaction (PCR) techniques can detect *S. pneumoniae* in culture-negative pleural fluid and may be a factor in the apparent increase of pneumococcal empyema.[1,13] Empyema occurs more commonly in the winter months and in boys.[1]

*S. aureus* is the leading cause of empyema in developing countries in Africa, the Middle East and Asia. Studies in these regions document *S. aureus* as the aetiological agent in 20 - 77% of cases and MRSA is rarely reported. It occurs more frequently in young infants and in the summer months when skin infections are said to be more prevalent.[14-17] A South African study of 100 cases of primary *S. aureus* pneumonia reported that 93% were complicated by PPE, empyema or pyopneumothorax. Pneumatoceles occurred in 37 patients and the case fatality rate was 7%.[18] The enterobacteriaceae *Escherichia coli, Klebsiella* and *Pseudomonas* species are also reported more frequently in developing countries and may be associated with an increased prevalence of malnutrition.[1] *Haemophilus influenzae* type b (Hib) is now an unusual cause of empyema in countries (including South Africa) where the Hib vaccine has been introduced into the routine immunisation schedule.[19] Anaerobic infections are rare (e.g. *Bacteriodes* species) but may occur in children with neurological disorders at risk of aspiration pneumonia and periodontal disease.[1] *Mycoplasma pneumoniae* is reported to be a common cause (19%) of PPE but rarely of empyema in Asia, where it is a common cause of childhood pneumonia[20, 21] Rare causes of empyema include *Mycobacterium tuberculosis* (not a rare cause of pleural effusion), *Entamoeba histolytica*, and complicated pulmonary hydatid cysts.[1,22] Features distinguishing an empyema from a tuberculous pleural effusion are shown in Table I.



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TABLE I . COMPARISON OF THE CLINICAL FEATURES OF EMPYEMA AND TB PLEURAL EFFUSION

| | Empyema and PPE | TB pleural effusion |
|---|---|---|
| Age | More common in young children and infants | More common in older children |
| Symptom duration | Short history of cough dyspnoea, fever | Chronic symptoms of cough, weight loss; TB contact |
| Clinical | Very unwell, high fever | Less severe illness, unless large effusion |
| Underlying pneumonia | Common | Uncommon |
| CXR | Pneumothorax, pneumatoceles and cavitatory pneumonia not uncommon. Lymphadenopathy uncommon | Air leaks or lung cavitation is rare; lymphadenopathy frequent |
| Fluid characteristics | Turbid or pus with neutrophil predominance; low ADA | Straw coloured and clear with lymphocytes predominance; high ADA |
| Culture | Bacterial cultures often positive | Bacterial cultures negative |
| TST | Negative unless co-infected | Usually positive |

CXR = chest X-ray; ADA = adenosine deaminase; TST = tuberculin skin test.

## Pathogenesis

The pleural membranes are permeable to fluid and gas and are lined by a thin layer of mesothelial cells that contain lymphatic stomas. Normally, pleural fluid is alkaline (pH 7.60) and contains a small number of cells (mostly mesothelial cells and macrophages) and protein. Equilibrium between filtration and reabsorption of fluid ensures that a small amount of sterile clear fluid (0.1 - 0.2 ml/kg) remains within the pleural space and prevents friction between the visceral and parietal pleura. Normally, the venous circulation reabsorbs 90% of filtered pleural fluid with the remaining 10% reabsorbed through lymphatic channels. Any disruption of this equilibrium through increased fluid production or leakage, impaired pleural membrane integrity, or decreased capillary and lymphatic reabsorption will result in an accumulation of pleural fluid.[23]

The inflammatory process of a pulmonary infection extends to the visceral pleura and results in increased permeability of local tissue and capillaries. Cytokines (IL-1, IL-6, IL-8 and TNFα) released by mesothelial cells cause a regional migration of inflammatory cells and leakage of fluid into the pleural space. Pleural effusions associated with infection may progress to empyema in the following three classically described stages:[24]

### Exudative stage (uncomplicated PPE)

Clear, sterile pleural fluid accumulates in the pleural space. There is usually a low white cell count, the pH is normal and lactate dehydrogenase (LDH) activity is <1 000 IU.

### Fibropurulent stage (complicated PPE or empyema)

This stage is characterised by deposition of fibrin in the pleural space leading to septation and loculations. It is usually accompanied by bacterial invasion and may evolve rapidly (within hours) without antibiotics. The fluid is usually turbid or frank pus, and microscopy shows neutrophils and degenerated cells. In the absence of prior antibiotic therapy, Gram stain and bacterial cultures are often positive. The pH is <7.2 and LDH often >1 000 IU, reflecting high metabolic and cytological activities in the pleural fluid.

### Organisational stage

Fibroblasts infiltrate the pleural cavity and transform the thin fibrin membranes into thick non-elastic pleural peels. This may result in 'trapped lung' and functionally impair gas exchange. Complications such as chronic empyema, bronchopleural fistula, lung abscess or spontaneous perforation through the chest wall (empyema necessitatis) may complicate this stage.

## Impact of HIV infection on childhood empyema

The HIV epidemic has led to an increase in the incidence and severity of childhood pneumonia and therefore a similar increase in empyema may be expected. However, this effect is poorly reported in the literature. A South African study found a 41.7-fold increase in invasive pneumococcal disease and a higher incidence of antibiotic-resistant isolates in HIV-infected children compared with uninfected children.[25] Although the use of antiretroviral therapy (ART) has reduced the burden of invasive pneumococcal disease among HIV-infected populations, it remains more common than in HIV-uninfected populations.[26] Pneumoccocal conjugate and Hib vaccines are also less efficacious in HIV-infected children than in uninfected children.[13] In addition, *S. aureus* pneumonia occurs more commonly in HIV-infected children and there is a higher rate of methicillin resistance than in HIV-uninfected children.[4,27] A study from Zambia of 85 HIV-infected patients (including children) with empyema in the pre-ART era reported culture-confirmed *M. tuberculosis* as the cause in 47% of cases, followed by bacterial infections (35%), peritonitis, chest trauma and septicaemia. Empyema necessitatis was seen in 7 children with bacterial empyema and in-hospital mortality for all patients was 30%.[28] Similar high rates of *M. tuberculosis* as a cause of



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empyema have however not been reported from other high TB and HIV prevalence areas. The increased incidence of TB infection and disease severity in HIV-infected children might lead to increased pleural TB complications if diagnosis and treatment is delayed.[29] Kaposi's sarcoma, non-Hodgkin's lymphoma and Gram-negative infections (*Salmonella* species, *Pseudomonas*) have been reported to cause pleural effusion with an increased incidence in HIV-infected adults.[30-32]

**Practice points**

- Empyema is the presence of pus in the pleural space and occurs as a complication of bacterial pneumonia in most cases.
- *S. pneumoniae, S. aureus,* and *H. influenzae* are frequent causes of empyema in developing countries.
- Gram-negative organisms and MRSA are important pathogens in HIV-associated empyema.

# Diagnosis

## Clinical features

Most children with empyema will present with symptoms of acute pneumonia. Empyema should be suspected in a child with pneumonia who remains persistently febrile despite adequate antibiotic treatment. Pleuritic chest pain or referred abdominal pain may be reported in older children. High fever, tachypnoea and chest-wall recession accompanied by dull percussion (often 'stony dullness') and reduced or absent breath sounds are suggestive of empyema. Large pleural effusions that cause mediastinal shift will displace the trachea and cardiac apex away from the affected side.

Tuberculous pleural effusions complicate 2 - 38% of cases of childhood TB and must be differentiated from PPE or empyema (Table I). A history of persisting cough, weight loss and a household TB contact is suggestive of childhood TB. In primary TB, a unilateral pleural effusion develops 6 - 12 weeks after infection. It represents a hypersensitivity reaction and is associated with a positive tuberculin skin test (TST) in over 90% of well-nourished immunocompetent children. Parenchymal pulmonary TB with pleural effusion represents reactivation disease or progressive primary disease with parenchymal spread and may be indistinguishable from PPE.[33] TB alone is a rare cause of empyema and bacterial co-infection is a more likely scenario.

Further history and examination should note any evidence of systemic illnesses that may cause pleural effusions and include congestive heart failure, oedema and hypoalbuminaemia (e.g. nephrotic syndrome), malignancy, post-streptococcal glomerulonephritis and connective tissue disorders. Questioning and examining for signs of HIV disease are equally important.

## Imaging

An anteroposterior (AP) chest X-ray (CXR) should be done in all children with suspected empyema. Obliteration of the costophrenic angle indicates a fluid collection. Complete 'white-out' of the affected hemithorax with mediastinal shift to the contralateral side is seen in large fluid collections, but is occasionally seen with extensive, dense lung consolidation and/or collapse (Fig. 1). A rim of fluid ascending along the lateral chest wall is known as the meniscus signs.[1] Radiological evidence of pneumonia is usually found and the presence of pneumothorax, pneumatoceles or lung cavities suggests *S. aureus* as the aetiology – or rarely cavitary TB or a complicated hydatid cyst (Fig. 2). The lateral CXR is useful to detect the presence of hilar lymphadenopathy when TB is suspected. In uncomplicated PPE, fluid in the lateral decubitus view will migrate and settle in dependent areas. This technique is useful to differentiate loculated from unloculated empyema, especially when ultrasound is not readily available. Scoliosis towards the affected side is common and reported in 71% of children with PPE or empyema.[1,34]

Ultrasound is useful to differentiate consolidated lung from pleural fluid, especially when there is white-out on the CXR and clinical signs do not clearly distinguish between the two.

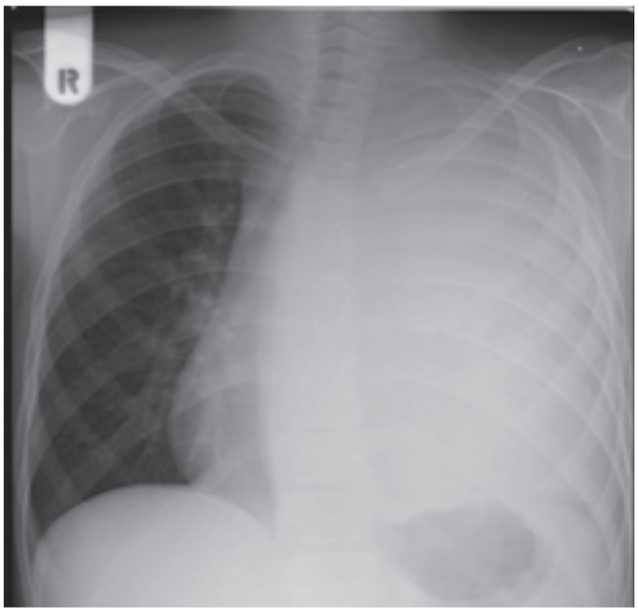


***Fig. 1. Chest radiograph of an 8-year-old girl presenting with chronic cough, weight loss, percussion dullness and absent breath sounds over the left lung. Complete 'white-out' of the left hemithorax and poorly defined left main bronchus is demonstrated.***

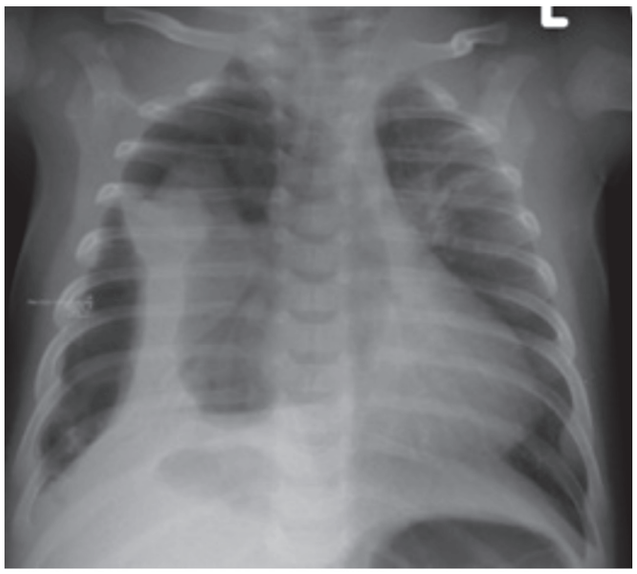

***Fig. 2.* Staphylococcus aureus *pneumonia and right-sided pyopneumothorax.***



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Ultrasound is also helpful in identifying pleural thickening and loculations (Fig. 3) and to guide chest drain insertion or thoracocentesis.[1, 23]

A computed tomography (CT) scan is usually not recommended but may be useful in delineating parenchymal lung abnormalities, endobronchial lesions, lung abscess or mediastinal pathology that may be associated with pleural fluid collections[1] (Fig. 4).

## Fluid examination

A diagnostic tap should only be done where a diagnosis other than empyema is considered (TB or malignancy), as chest drain insertion must be performed if empyema is suspected. Frank pus or turbid fluid indicates empyema and is associated with a neutrophil-predominant cellular infiltrate. Fluid should be sent for Gram stain and bacterial culture. The yield for an aetiological pathogen by conventional culture varies widely (8 - 76%) and depends largely on prior antibiotic use. New molecular techniques such as PCR and pneumococcal latex agglutination tests are useful for identifying an aetiological pathogen in cases of sterile empyema.[1] These investigations, however, are generally not available for routine use in most public health facilities in South Africa. Straw-coloured clear fluid is typical of TB or rarely of malignancy (frequently blood stained) and usually has lymphocyte predominance; however, 10% of TB effusions may be neutrophil predominant.[1] Pleural fluid in TB is rarely acid-fast bacilli (AFB) positive and cultures are positive in only 20 - 40% of cases.[33] Sputum or gastric lavage samples should therefore be sent for TB culture. Fluid should be sent for cytology if malignancy is suspected. Milky fluid that fails to separate after centrifugation is characteristic of chyle and is lymphocyte predominant. Special stains for chylomicrons and the presence of triglycerides will confirm the diagnosis.

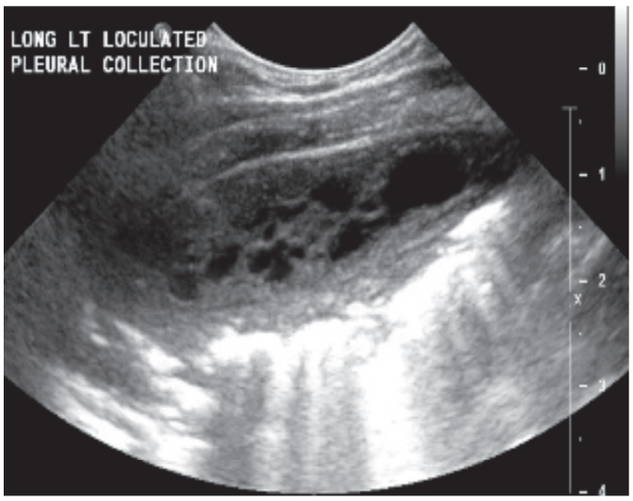


*Fig. 3. Longitudinal chest ultrasound image of a loculated empyema adjacent to aerated lung.*

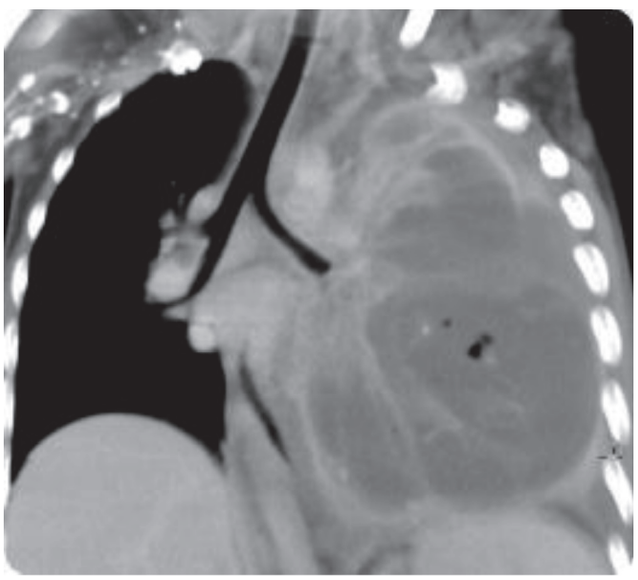

*Fig. 4. Coronal view CT scan of the same patient as in Fig. 1 shows that the entire left hemithorax is filled with thick multi-loculated fluid. The normal lung and pleural anatomy are poorly defined. The abrupt cut-off of the left main bronchus makes the diagnosis of a chronic retained foreign body likely.*

In adult practice, biochemical analysis of pleural fluid is traditionally used to differentiate transudates from exudates according to Light's criteria (Table II).[24,35] Application of these criteria to children has not been validated. Routine biochemical analysis of pleural fluid is of less importance in paediatric practice as the vast majority of pleural effusions are transudates. The clinical presentation, fluid macroscopic appearance, cell count and culture remain the most reliable criteria in determining aetiology and need for drainage of any pleural fluid collection. Further investigations are guided by clinical circumstances and are aimed at identifying the aetiological pathogen and any underlying disorder or complication such as HIV infection or air leaks.

## Other investigations

Further investigations are aimed at identifying an aetiological agent. Blood culture is the most important as it may be positive in 10 - 22% of empyema cases.[1, 2] Lower respiratory tract secretions (sputum, induced sputum, tracheal aspirate or bronchoalveolar lavage) should be sent for routine bacterial culture whenever possible. In cases where TB is suspected, microscopy and culture of sputum or gastric lavage and a TST are indicated. Acute-phase reactants including white cell count, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR) and procalcitonin are unhelpful at differentiating bacterial from viral infections but may have a role in monitoring clinical progress and response to treatment.[1]

**TABLE II. FEATURES DIFFERENTIATING TRANSUDATES FROM EXUDATES**

| Transudates | Exudates |
|---|---|
| Clear/straw coloured | Turbid/ frank pus/ bloody |
| May be bilateral | Unilateral |
| Non-viscous* | Viscous* |
| Protein <30 g/dl* | Protein >30 g/dl* |
| Protein fluid:serum ratio <0.5* | Protein fluid:serum ratio >0.5* |
| LDH fluid:serum ratio <0.6* | LDH fluid:serum ratio >0.6* |

*Quadri and Thomson.[35]
LDH = lactate dehydrogenase.



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**Practice points**

- Empyema is diagnosed clinically and radiologically.
- Tuberculous pleural effusion is an important differential diagnosis.
- The aetiological agent is determined by pleural fluid culture, blood culture and sputum culture. Cultures are often negative with prior antibiotic use.
- Ultrasound of the chest is useful in demonstrating a loculated empyema and differentiating pleural fluid from lung consolidation.

## Management

Assessment of the child's general condition and needs is important throughout the course of the illness. Ensuring adequate oxygenation (oxygen saturation >90 - 92%), analgesia, nutrition and fluid hydration are important aspects of care and require regular monitoring. The principal aim of empyema treatment is to limit sepsis by evacuating and sterilising the pleural cavity, thereby restoring pleural fluid circulation and function. Treatment approaches differ according to severity of the illness, available resources and clinical expertise. Despite these differences the outcome of paediatric empyema is ultimately good, irrespective of the treatment received.[1,2,6,36] Most available evidence for best practice in the management of empyema is derived from high-income countries where clinical disease patterns, aetiologies, available resources and expertise differ considerably from those in developing countries.[1,6,36]

### Antibiotics

The choice of empirical antibiotic therapy depends on various factors. These include age, HIV status, nutritional state and local antimicrobial susceptibility patterns. Empirical antibiotic therapy should include treatment for the likely pathogen(s) of pneumonia and empyema, and in addition be effective against *S. pneumoniae, S. aureus,* and *H. influenzae* – the common causes of empyema. Recommendations adapted from the South African Thoracic Society Guidelines' treatment for community-acquired pneumonia (CAP) for empirical antibiotic therapy in children with PPE and empyema are given in Table III.[37] Initial antibiotic therapy should be administered intravenously and changed to a suitable oral agent when the child is symptomatically improved, afebrile and able to drink without difficulty. Some guidelines recommend continuation of intravenous therapy for 5 - 7 days after fever or drainage has abated but no evidence exists for the optimal timing or duration of antibiotic therapy.[2]

### Drainage

A chest drain should always be inserted in any child with suspected empyema on clinical, radiological or fluid characteristics. Occasionally, antibiotics alone can be considered in very small PPEs. Repeated thoracocentesis is not advisable in children as it causes significant discomfort to the patient and results in less effective drainage.[1,6,36] Conservative treatment with antibiotics and chest tube drainage will be effective in 60 - 80% of cases, but is associated with an increased rate of treatment failure needing surgical intervention and longer hospitalisation (14 - 24 days) compared with other interventions.[36] Insertion of the drain should be performed with adequate analgesia and sedation to minimise the child's pain and anxiety. Good analgesia must always be administered at regular intervals for as long as the drain is *in situ*. Practical guidelines for chest drain insertion and management are provided in Appendix A. The British Thoracic Society (BTS) guidelines recommend the use of small (8 -12 FG) percutaneously inserted drains as there is little evidence to suggest the superiority of large-bore drains.[1] The use of small percutaneously inserted drains (e.g. pigtail

**TABLE III. ANTIBIOTIC RECOMMENDATIONS FOR EMPIRICAL TREATMENT OF COMMUNITY-ACQUIRED EMPYEMA IN CHILDREN[37]**

| Category/age | First-line IVI | Dose | Alternative IVI | Dose | Oral equivalent | Dose |
|---|---|---|---|---|---|---|
| <2months of age<br>*or*<br>HIV infection<br>*or*<br>malnutrition | Ampicillin<br>*or*<br>penicillin G<br>*and*<br>gentamycin<br>*and*<br>cloxacillin | 50 mg/kg qid<br>50 mg/kg qid<br>7.5 mg/kg then<br>5 mg/kg od<br>50 mg/kg | Cefotaxime<br>*or*<br>ceftriaxone<br>*and*<br>cloxacillin | 50 mg/kg tds<br>50 mg/kg od<br>50 mg/kg qid qid | Co-amoxiclav<br>*and*<br>flucloxacillin<br>*or*<br>clindamycin | 30 mg/kg amoxicillin equiv. tds<br>25 mg/kg qid<br>6 mg/kg qid |
| >2 months of age | Ampicillin<br>*or*<br>penicillin G<br>*and*<br>cloxacillin | 50 mg/kg qid<br>50 mg/kg qid<br>50 mg/kg qid | Cefotaxime<br>*or*<br>ceftriaxone<br>*or*<br>cefuroxime<br>*and*<br>cloxacillin | 50 mg/kg tds<br>50 mg/kg od<br>50 mg/kg tds<br>50 mg/kg qid | Amoxicillin<br>*or*<br>cefuroxime<br>*and*<br>flucloxacillin<br>*or*<br>clindamycin | 30 mg/ kg tds<br>15 mg/kg tds<br>25 mg/kg qid<br>6 mg/kg qid |

1. Adequate Gram-negative cover should be provided for children with HIV infection or malnutrition.
2. Consider adding metronidazole, piptazobactam or substituting with clindamycin if anaerobic infection is suspected.
3. Total duration of antibiotic treatment should be 2 - 6 weeks, depending on severity and symptoms.
4. Antibiotic treatment should be modified according to drug-susceptibility patterns of bacterial cultures.



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catheters) is associated with improved patient comfort but their efficacy in late-stage empyema with thick pus, without frequent fibrinolytic or saline flushing, needs to be established. Chest drains should be removed once the pleural fluid has been effectively drained and no residual air or fluid collection exists.

## Intrapleural fibrinolytic therapy

Fibrinolytic therapy has become standard practice in many developed countries and is recommended by the BTS as first-line therapy for any complicated PPE or empyema.[1] Instillation of fibrinolytic agents via a chest drain into the pleural cavity aims to lyse fibrinous strands and clear lymphatic pores, thus facilitating better drainage. Most studies have been conducted with urokinase, but streptokinase and tissue plasminogen activator (PTA) are alternative agents. The recommended dose of urokinase is 40 000 U in 40 ml 0.9% saline (>1 year) or 10 000 U in 10 ml 0.9% saline (<1 year) instilled twice daily for 3 days. The drain is clamped for 4 hours, during which time the child alternates between prone and supine positions, when it is unclamped and allowed to drain freely.[1] Streptokinase and PTA are available in South Africa but are very expensive. The recommended dose for streptokinase is 15 000 U/ kg in 20 - 50 ml normal saline per installation once daily for 3 days.[38] A recent review of the published evidence for fibrinolysis in prospectively designed randomised studies found only 4 small paediatric studies: 3 from developed countries and 1 from India.[36] The studies compared fibrinolysis versus 0.9% saline or VATS. However, none compared conservative treatment (antibiotics and chest tube) and fibrinolysis. The authors concluded that fibrinolysis should be the initial treatment of choice for all PPE and empyema as its main benefit is a reduction of hospitalisation time (6 - 13 days v. 14 - 24 days). The study from India, however, showed no short-term benefit of intrapleural streptokinase versus 0.9% saline.[38] A large meta-analysis comparing primary operative and non-operative therapy (including salvage fibrinolysis) demonstrated an overall benefit of fibrinolysis compared with conservative management but showed an increased rate of complications.[39] This analysis is however limited as most of the studies included were either observational or retrospective reviews.[36,39] Similarly, a Cochrane review of randomised controlled trials in adults showed significant benefits of fibrinolysis compared with saline, but did not recommend routine use of fibrinolysis in adult empyema as the trial numbers were too small.[40] The role of routine intrapleural fibrinolysis in the setting of developing countries needs to be studied and cannot currently be recommended as routine therapy owing to its prohibitive cost and limited evidence of efficacy.

## Surgery

Surgical treatment should be considered when antibiotics, chest tube drainage and fibrinolysis (where available) have failed to achieve adequate drainage of the pleural collection in the face of persisting sepsis. Other indications for surgery include complicated pyopneumothorax, where the lung fails to re-expand, and bronchopleural fistula.[1] Surgical options are mini-thoracotomy and debridement; open decortication involving removal of the thickened pleural peel and pleural irrigation; and VATS (thoracoscopic decortication and irrigation). The indications and choice of surgical intervention are largely dependent on local surgical experience, preference, extent of the illness and expertise. There is currently little consensus with regard to the optimal surgical management of childhood empyema. A literature review of randomised studies found only a few small studies indicating that primary surgical therapy (VATS or thoracotomy) may be superior to conservative therapy with or without fibrinolysis, but this was not a consistent finding.[36] A large meta-analysis comparing primary operative and non-operative therapy demonstrated an overall benefit of primary operative therapy compared with conservative management, but this analysis is also limited as most of the studies included were either case series or retrospective reviews.[36,39] There are no studies directly comparing primary VATS with open decortication. However, it is clear that ultimately the outcome of childhood empyema is similar regardless of the surgical approach adopted.[1,2,6,41] This is of relevance to resource-poor settings where access to cardiothoracic surgical services is frequently limited.

**Practice points**

- Empirical antibiotics should cover *S. pneumoniae*, *S. aureus* and *H. influenzae*. Further antibiotic treatment should be guided by microbiology results.
- Chest tube drainage is always indicated if empyema is suspected. Small PPEs may be managed with antibiotics alone.
- Adequate analgesia and anxiolysis are important at all times when managing children with chest drains.
- Intrapleural fibrinolysis shortens hospital stay and should be considered as a primary treatment in cases of loculated empyema.
- Surgery is indicated in children with persistent pleural sepsis manifested by persistent fever and pleural fluid collections.

## Conclusion

Childhood pneumonia and empyema continue to be an important problem in developing countries. The impact of HIV on childhood empyema has been poorly studied. Management of empyema in many resource-poor countries remains conservative, with antibiotics and chest tube drainage the primary therapy. The roles of fibrinolysis, VATS or early open surgery need to be studied further. Although there are few data for different interventions on the long-term outcome in HIV-infected children or children in developing countries, the available evidence suggests that the outcome is good regardless of the therapeutic approach.

We would like to thank Dr Jenny Thomas for providing advice and guidance on pain management in children with chest drains.

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## APPENDIX A. PRACTICAL GUIDELINES FOR THE MANAGEMENT OF CHEST DRAINS IN EMPYEMA [1, 42,43]

### 1. Inserting a chest drain

- Ultrasound-guided insertion of chest drains is recommended in complicated empyema.
- Administer adequate analgesia with anxiolytics if required:
  - Paracetamol (20 mg/kg) with codeine (1 mg/kg) and midazolam (0.2 - 0.5 mg/kg po) administered 30 - 45 minutes before the planned procedure *or*
  - Valoron 1 mg/kg (1 drop = 2.5 mg) *or*
  - Ketamine (1 - 2 mg/kg IVI) and midazolam (0.1 - 0.2 mg/kg IVI). Ensure that adequate monitoring and resuscitation equipment is available at all times during and after the procedure.
- Site of drain insertion: 4th or 5th intercostal space mid-axillary line.
- Infiltrate the site of drain insertion with local anaesthetic (lignocaine up to 3 mg/kg). Where available, topical anaesthetic cream (e.g. Emla) should be applied 1 hour before the procedure.
- Make a small incision in the skin. With an artery forceps, gently dissect to the parietal pleura and enter the pleural space. Insert the drainage tube approximately one-third of the diameter of the chest. Aim the tube anteriorly if air needs to be drained, or posteriorly if only fluid needs to be drained.
- Trochar-guided chest tubes are *contraindicated.*
- Secure the drain with a suture (not necessary in small infants) and adhesive plaster to the chest wall. Stabilise the chest drain to the lateral abdominal wall ('omental tape') with adhesive plaster to prevent dislodgement.
- Repeat a CXR to confirm the position of the drain.

### 2. Chest tube drainage

- Use an underwater drain system, ensuring the tube is no more than 1 cm below the water surface and below the patient's chest at all times.
- A chest drain that continues to bubble indicates a bronchopleural fistula (BPF).
- Low pressure continuous suction (5 - 10 cm water) may be useful in pyopneumothorax to prevent chronic empyema and to facilitate drainage with fibrinolytics. Uncomplicated empyema does not require continuous suction.
- Never clamp a bubbling drain as it may result in tension pneumothorax.
- Ensure adequate analgesia while the drain is *in situ:* paracetamol (20 - 30 mg/kg/dose po) *and* ibuprofen (6 - 10 mg/kg/dose po) *or* clonidine 1 - 5 µg/kg/dose po) *and* Valoron (1 mg/kg) *or* morphine infusion (20 - 80 µg/kg/h) *or* morphine (0.2 - 0.4 mg/kg/dose po).
- Physiotherapy is not indicated. Early mobilisation should be encouraged.

### 3. Removing a chest drain

- Remove the chest drain if it has stopped draining or becomes blocked. A blocked drain in a symptomatic patient should be replaced if there is significant residual pleural fluid.
- Timing of chest drain removal is a clinical decision. CXR and/or ultrasound are useful to detect significant residual pleural fluid.
- Provide adequate analgesia and anxiolytics (if required). See 1 (above) for doses.



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