

Empyema and Its Management– A Brief Review of Literature

Girish L Dandagi*

Department of TB and chest diseases, Belgaum Institute of Medical Sciences, Belgaum, Karnataka, India.

Review Article

Received: 18/02/2013

Revised: 13/03/2012

Accepted: 22/03/2013

***For Correspondence**Plot no 49, sector # 9, Malmaruti Extn,
Belgaum, Karnataka, India 590016**Keywords:** Empyema, Thoracoplasty,
streptokinase, thoracentesis.**ABSTRACT**

Empyema has been derived from the Greek word which means pus in the body. The first description of this pleural infection has been described by Egyptian physician Imhotep around 3000B.C, but it was famously described by Hippocrates in 500B.C. Till the early twentieth century, the only treatment option available was open surgical drainage with associated high morbidity and mortality. The other modalities of management are thoracentesis, thoracoplasty and use of fibrinolytic agents. Over the last several years; additional studies were done to explore the efficacy and safety of management modalities which are discussed in detail in the below article.

INTRODUCTION

Empyema occurs more commonly during childhood and in the elderly^[1,2]. Pleural infection is most commonly a complication of bacterial pneumonia; hence, patients at risk for pneumonia are also at risk of pleural infection. Independent risk factors for empyema include diabetes mellitus, alcohol abuse, gastroesophageal reflux disease, and intravenous drug abuse. Poor dental hygiene and aspiration predispose to infection with anaerobic organisms^[3]. Up to one third of cases occur without as yet identified risk factors^[4]. The remaining cases of pleural infection unrelated to pneumonia are largely iatrogenic, including thoracic (20%) and esophageal surgery, esophageal perforation, and trauma (5%). Infection may be introduced into a pleural effusion of any cause by thoracentesis (2% of cases of empyema) or during intervention for primary spontaneous pneumothorax (2%), underlining the importance of appropriate use of invasive pleural procedures for these conditions. The following article gives an idea about various techniques which are followed to treat empyema.

DISCUSSION

There are two basic principles for the successful management of thoracic empyema: (i) control of infection with appropriate antimicrobial therapy and (ii) adequate drainage of pus. Adherence to these principles should return the pleural cavity to its former sterile state and allow full re-expansion of the underlying lung architecture^[5].

Antimicrobial Therapy

The choice of antibiotic is usually determined from the results of microbiological culture and sensitivity testing.

Closed Drainage

Closed drainage may be either 'intermittent', in which repeated aspiration (thoracentesis) is carried out, or 'continuous', in which an intercostal tube is connected to an underwater seal (closed-tube thoracostomy). Closed drainage is preferred provided that the pus is accessible and not too viscid to permit adequate removal by these methods. These conditions are more likely to appertain in the exudative stage but can be continued into the fibrino purulent stage in some cases^[5].

Thoracentesis

The site of the pus can often be confirmed with the local anaesthetic needle. Aspiration is thereafter best carried out using a needle or cannula of sufficient bore to allow the pus to flow without too much exertion on the part of the operator. Sharp needles are best avoided for fear of damaging underlying lung^[5]. The use of an Abrams punch biopsy needle is useful initially, as it is of

sufficiently wide calibre to allow easy aspiration and also permits diagnostic biopsy of the parietal pleura/empyema cortex for histological examination and culture. The frequency with which thoracentesis is repeated depends upon the rate at which pus reaccumulates, which in turn is judged by the clinical and radiographic findings. Aspiration may be required daily or two or three times per week at first, diminishing as infection responds to antibiotics, and may sometimes extend over a prolonged period, during which time the patient is usually ambulant and visits the hospital as an outpatient. Such medical treatment with antibiotics and repeated thoracentesis is appropriate for many individuals with pleural empyema and these patients may have a shorter and less complicated stay than those treated by tube drainage [5].

Closed-Tube Thoracostomy

Closed-tube thoracostomy is preferred by some clinicians from the start once the diagnosis of empyema has been confirmed⁵. It has the advantages that drainage is continuous and that it is more likely to be successful when the infected material is too viscid to be removed by manual aspiration, but with the disadvantage of greater discomfort and immobility for the patient; furthermore introduction of new infection at the drainage site is a possibility and the tube itself may become blocked by fibrin [5]. Malécot-type self-retaining soft rubber catheters have advantages over the modern disposable rigid plastic cannulae in widespread use, in that underlying structures are less likely to be impaled during introduction, they can be made to fit the cutaneous incision snugly with less chance of leakage around the tube and they are less likely to fall out [6]. Regrettably, however, they are no longer available in most centres. Under local anaesthesia the tube is placed in the most dependent part of the empyema cavity, a site that may be determined by ultrasound examination, CT or the injection of a little radio-opaque contrast material. Ultrasound may also be used to place a small catheter (such as an 8 French gauge pigtail nephrostomy tube with 10 side-holes) in larger loculations and this may be connected to a plastic drainage bag attached to the chest wall. Patency may be maintained using streptokinase.

When underwater seal drainage through a conventional large-bore intercostal tube becomes slight, the tube may be cut off, transfixed with a safety pin (to prevent it falling into the empyema cavity) and covered with a gauze dressing to allow the patient greater mobility until it is removed. Sonography may be carried out by injecting contrast down the tube in order to assess the adequacy of drainage [7]. When the residual sinus is small and drainage minimal (e.g. <150mL daily for two consecutive days, including 6-hourly 20-mL saline flushes), the tube is removed.

A technique somewhere between thoracentesis and closed-chest drainage has been described in which a 20–28 French gauge plastic cannula is passed into the empyema cavity under local anaesthesia, fibrinous septa being broken down and pus rapidly removed by strong negative pressure (–13 kPa), after which the cannula is removed and the skin resutured [7].

Closed drainage, whether by thoracentesis or intercostal tube, is likely to be successful if the empyema is small and if treatment of the empyema is started in the acute exudative or early fibrinopurulent stages of infection, in which case the wall of the empyema cavity gradually becomes reabsorbed allowing re-expansion of underlying lung and obliteration of the pleural space [7]. This process can be expected to take several weeks in adult practice. Bronchoscopy is recommended following the successful conclusion of closed drainage in order to exclude any possible endobronchial causes of obstruction, such as tumour or foreign body. High resolution CT may be used if underlying bronchiectasis is thought likely. The techniques outlined above may fail to cure an empyema if the pus is too thick to drain by thoracentesis or tube, if a bronchopleural fistula has developed or if pockets of pus become loculated and inaccessible. When closed drainage has failed to enable the lung to re expand fully, a more invasive surgical procedure has to be employed. Such procedures include thoracoscopy, open drainage with rib resection, thoracotomy and decortication and, rarely, thoracoplasty [7].

Video-Assisted Thoracoscopic Surgery

If closed drainage does not result in prompt re-expansion of the lung and especially if loculi have been identified ultrasonically, a decision to intervene relatively early using video-assisted thoracoscopic surgery (VATS) with débridement and drainage is sometimes made. A small prospective randomized controlled trial in patients with fibrinopurulent empyema has compared treatment by VATS with fibrinolytic therapy using chest-tube pleural drainage and streptokinase; this showed that VATS was more effective, with faster resolution and a shorter hospital stay than that achieved with fibrinolytic therapy [8] Provided that the empyema is in the fibrinopurulent stage, with the thickened rind caused by fibrin rather than the mature scar tissue that forms when an empyema has become chronic, VATS enables the operator to achieve adequate débridement, breaking down loculi, evacuating pus and debris and freeing the lung. This may then result in prompt re-expansion of the lung and obliteration of the pleural cavity, avoiding the need for repeated or prolonged attempts at drainage [9,10].

Open Drainage

Open surgical drainage is used if an empyema persists both clinically and radiographically in a patient in whom closed drainage has proved unsuccessful. It may be avoided if the empyema is suitable for drainage by VATS [11, 12].

When VATS is unavailable, unsuccessful or considered inappropriate, open drainage may be carried out in patients whose general condition is such that they are judged to be too debilitated to undergo the more invasive procedure of thoracotomy and

decortication. Open drainage, which has been carried out in a sitting position under local anaesthetic to avoid spread of sepsis to the unaffected lung, often involves resection of the lowest rib above and below which pus can be aspirated ^[11, 12]. The surgeon makes an incision large enough to allow adequate drainage, cleans the inside of the empyema cavity and places a wide-bore tube surrounded by gauze in the unsutured wound. Daily redressing under surgical supervision with sterilization and replacement of the tube is recommended. Successful open drainage results in gradual obliteration of the empyema space, which allows ultimate removal of the tube and cure. Thoracotomy and decortication may still be recommended if, during open drainage, the patient's general condition improves sufficiently. When drainage is protracted and the patient remains too ill or is otherwise unsuitable for thoracotomy, then a more permanent fenestration or open-window thoracostomy (sometimes referred to as an Eloesser flap) may be performed. Such procedures involve the removal of sections of two or more ribs in order to fashion a larger stoma, which is kept open by suturing the skin to the parietal pleura/cortex thereby creating a pleurocutaneous fistula. The stoma may be closed if the underlying lung re-expands or may occasionally be left permanently open with daily dressing changes. Fenestration procedures are sometimes used in empyemas that complicate pneumonectomy. This form of empyema is often associated with a bronchopleural fistula through the bronchial stump. These fistulae, if small, may close spontaneously but if large do not close without a surgical procedure, such as resuturing with or without the transposition of pediculated intercostal muscle in order to cover the bronchus and also to help obliterate the empyema space. A persistent bronchopleural fistula in this situation prevents successful closure of the fenestration ^[11, 12].

Decortication

This is an elective surgical procedure, unsuitable for patients who are ill and toxic, in which the fibrous wall of the empyema cavity, variously referred to as the cortex, rind or peel, is exposed at thoracotomy and stripped off the adjacent visceral and parietal pleura, which may be left intact. A bronchoscopy is carried out first in non traumatic cases to exclude an underlying tumour or foreign body⁶. Decortication is carried out in patients in whom closed drainage and/or thoracoscopic methods have been unsuccessful, provided that they are fit enough to undergo this major procedure. It may also be used in the patient whose condition has stabilized following open drainage but who has entered a chronic phase in which the underlying lung does not expand because of failure of the cortex or rind to become reabsorbed. There is no consensus about the optimal time at which to perform a decortication, some surgeons arguing for early intervention and others adopting a more conservative approach ^[13,14,15].

In the early stage of organization the empyema cortex is poorly demarcated and friable, making decortication difficult. In intermediate stages of formation it is rather vascular so that decortication may be best delayed 2 or 3 bronchopleural fistula, pleuro pneumonectomy may be required.

Intrapleural Fibrinolysins

The hypotheses that fibrin strands within organizing pleural exudates initiate intrapleural loculation and that clearance of intrapleural fibrin by intrapleural administration of fibrinolysins prevents intrapleural organization and loculation are not novel. Beginning in the 1940s, Drs Tillett and Sherry ^[16] and Tillett et al ^[17] tested the possibility that fibrinolytic agents could be used on this setting. They demonstrated that preparations of streptokinase or streptodornase could be used to resolve pleural loculations attributable to parapneumonic effusions or hemothoraxes ^[18]. Surgical findings that intrapleural adhesions appeared to be fibrinous were used as predicates for administration of intrapleural fibrinolytic agents. These reports antedate our current understanding of the scope of effects exerted by disordered fibrin turnover as may occur in pleural injury or organizing hemothorax. However, the success of the original approach provided a strong rationale for the continued utilization of fibrinolytic therapy to address extensive intrapleural loculation and lung entrapment.

Over the last several years, additional studies were done to explore the efficacy and safety of intrapleurally administered fibrinolysins.

Currently Available Fibrinolysins

Streptokinase has been associated with febrile reactions but has been generally well tolerated, as reported in a number of studies. Intrapleural streptokinase, as commonly used for intrapleural administration, does not induce systemic fibrinolysis and is relatively free of hemorrhagic risk ^[19, 20].

Intrapleural urokinase has likewise been reported to be well-tolerated. The cost of intrapleurally administered agents has favored streptokinase as reported in prior studies, but urokinase has been reported to be cost-effective ^[21] The true test of cost-effectiveness should ideally take into account reductions of hospitalization or avoidance of surgical remediation of intrapleural organization, considerations that are not comprehensively evaluated in the literature. Larger interventional trials may more clearly address these issues. The cost of intrapleural fibrinolysins varies considerably. Streptokinase is available in a 750,000 IU dose and 1.5 lakh IU dose Urokinase is now available in 250,000 IU / 5,000,000 I.U. / 7,50,000 I.U. / 10,00,000 I.U. dose.

tPA is available in 50-mg vials ^[22] Cost comparisons are affected by the relative efficacy of these agents, the number of daily treatments required, and relative effects on total hospital stay or mortality. These are all issues that remain to be definitively

addressed. Therefore, the relative cost-effectiveness of intrapleural fibrinolysis has yet to be comprehensively evaluated, as has the use of fibrinolytic therapy as an adjunct to or in comparison with video-assisted or surgical decortication.

Fibrinolytic Agents Used for Intrapleural Applications

Streptokinase

Single-chain glycoprotein of molecular weight 40,000–50,000 kDa. Not an enzyme. Generates plasmin through complex formation with and catalysis of plasminogen. Rapid half-life; cleared within minutes. Can induce antistreptokinase antibodies. Least expensive fibrinolytic [23].

Urokinase Plasminogen Activator (uPA)

Low-molecular-weight form predominates in commercial preparations; molecular weight 33,000 kDa. Rapid plasma half-life; cleared within minutes. Directly activates plasminogen to form plasmin. Endogenous plasminogen activator that can be detected in pleural fluids and plasma [23].

Tissue Plasminogen Activator (tPA)

Glycosylated protein of molecular weight 68,000 kDa. Rapid plasma half-life; cleared within minutes. Recombinant material is used therapeutically. Endogenous tPA detectable in pleural fluid and plasma [23].

REFERENCES

1. Davies CW, Kearney SE, Gleeson FV, Davies RJO. Predictors of outcome and long-term survival in patients with pleural infection. *Am J Respir Crit Care Med*. 1999;160(5 Part 1):1682–7.
2. Givan DC, Eigen H. Common pleural effusions in children. *Clin Chest Med*. 1998;19(2):363–71.
3. Brook I, Frazier EH. Aerobic and anaerobic microbiology of empyema. A retrospective review in two military hospitals. *Chest*. 1993;103(5):1502–7.
4. Ferguson AD, Prescott RJ, Selkon JB, Watson D and Swinburn CR. The clinical course and management of thoracic empyema. *QJM*. 1996;89(4):285–9.
5. Moores DWO. Management of acute empyema. *Chest* 1992; 102: 1316–1317
6. Storm HKR, Krasnik M, Bang K, Frimodt MN. Treatment of pleural empyema secondary to pneumonia: thoracentesis regimen versus tube drainage. *Thorax*. 1992; 47: 821–824.
7. Le Roux BT, Mohlala ML, Odell JA, Whitton ID. Suppurative diseases of the lung and pleural space. Part 1. Empyema thoracis and lung abscess. *Curr Probl Surg*. 1986 Jan;23(1):1–89.
8. Antoni LA, Conlan AA. Pleural sonography in the management of thoracic empyema. A study of 52 cases. *S Afr Med J*. 1985; 67: 216–221.
9. Cummin ARC, Wright NW, Joseph AEA. Suction drainage: a new approach to the treatment of empyema. *Thorax*. 1991; 46: 259–260
10. Wait MA, Sharma S, Hohn J, Dal Nogare A. A randomized trial of empyema therapy. *Chest*. 1997; 111: 1548–1551.
11. Lawrence DR, Ohri SK, Moxon RE, Townsend ER, Fountain SW. Thoracoscopic debridement of empyema thoracis. *Ann Thorac Surg*. 1997; 64: 1448–50
12. Shamji FM, Ginsberg RJ, Cooper JD, Spratt EH, Goldberg M, Waters PF, et al. Open window thoracostomy in the management of postpneumonectomy empyema with or without bronchopleural fistula. *J Thorac Cardiovasc Surg*. 1983; 86: 818–822
13. Wong PS, Goldstraw P. Post-pneumonectomy empyema. *Eur J Cardiothorac Surg*. 1994; 8: 345–349.
14. Hoover EL, Hsu HK, Ross MJ. Reappraisal of empyema thoracis. Surgical intervention when the duration of illness is unknown. *Chest*. 1986; 90: 511–515.
15. Mayo P. Early thoracotomy and decortication for nontuberculous empyema in adults with and without underlying disease. A twenty-five year review. *Am Surg*. 1985; 51:230.
16. Tillett WS, Sherry S: The effect in patients with streptococcal fibrinolysis (streptokinase) and streptococcal desoxyribonuclease on fibrinous, purulent, and sanguinous pleural exudations. *J Clin Invest*. 1949, 28:173–190.
17. Tillett WS, Sherry S, Read CT. The use of streptokinase–streptodornase in the treatment of postpneumonic empyema. *J Thorac Surg* 1951;21:275–297.
18. Bergh NP, Ekroth R, Larsson S, Nagy P: Intrapleural streptokinase in the treatment of haemothorax and empyema. *Scand J Cardiovasc Surg*. 1977, 11:265–268
19. Dionne AS, Edmund JR, Scott AS, Jeffrey EA, Parker LA., and Preston B R. Intrapleural tissue plasminogen activator for complicated pleural effusions. *J Trauma*. 2004;57:1178–1183.
20. Cameron R. Intra-pleural fibrinolytic therapy vs. conservative management in the treatment of parapneumonic effusions and empyema. *Cochrane Database Syst Rev*. 2000;(3):CD002312.
21. Sahn SA. Use of fibrinolytic agents in the management of complicated parapneumonic effusions and empyemas. *Thorax*. 1998;53(suppl 2): S65–S72.

22. Walker CA, Shirk MB, Tschampel MM . Intrapleural alteplase in a patient with complicated pleural effusion. *Ann Pharmacother.* 2003;37:376-379.
23. Bell WR. Present day thrombolytic therapeutic agents:pharmacotherapeutics and pharmacodynamics. *Rev Cardiovasc Med.* 2002;2(suppl 2): S24-44.