Review Article

Post-resection complications: abscesses, empyemas, bronchopleural fistulas

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Abstract: The role of thoracic surgeons in the management of pulmonary infection has evolved over time as the medical treatments have improved. We herein review historical and current management for surgically-treated pulmonary infections—lung abscesses, empyemas, and bronchopleural fistulas. In particular, we review when the surgeons need to be involved for infectious cases, our algorithm/approach to empyemas, and summary of post-operative bronchopleural fistula in tuberculosis cases.

Keywords: Empyema; bronchopleural fistula (BPF); pulmonary abscess

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Introduction

Upper respiratory and pulmonary infections are common in the United States, and thoracic surgeons may be involved for both diagnostic and management purposes. Pneumonia is an acute pulmonary infection, and may be commonly caused by viruses and bacteria, and less commonly by fungi, mycoplasma, and parasites (*Table 1*). Centers for Disease Control (CDC) data from 2014 identify influenza and pneumonia as the primary cause of death for over 55,000 or 2.9% of all deaths during that period, making it the 8th leading cause of death in the United States (1). Mortality rates increase based on infection contraction location (hospital *vs*. outpatient) and associated comorbidities, and can approach 40% in critically ill patients (2). While these infections are highly morbid in themselves, the acute inflammation and immune response predisposes patients to other infections that may require thoracic surgical intervention.

The majority of pulmonary infections are from inhalation sources though direct spread from adjacent tissue versus hematogenous spread is not uncommon (3). Pulmonary infections may remain confined to the lung and cleared by the immune system, or may become isolated and localized within lung parenchyma, causing a smoldering infection or abscess, or may spread beyond the lung parenchyma into the pleural space and cause an empyema. Abscesses are localized collections of purulent material contained within necrotic lung parenchyma, most frequently caused by aspiration of oral secretions (4). These historically required operative management to achieve source control; however, with modern antibiotic regimens, operation is usually now reserved for recalcitrant infections failing to improve with medical management (5). Effusions are collections of fluid within the pleural space, most often triggered by local inflammation from infection causing disruption in the lymphatic absorptive capacity of the lung, or post-operatively versus post-traumatic due to direct trauma to the visceral pleura (6). These can become infected, causing an empyema, or pleural space infection. These frequently require surgical management (7).

We herein review the historical and current guidelines for management of lung abscesses, empyemas, and bronchopleural fistulas (BPFs).

Table 1 Mycobacterial and mycotic infections

Organism	Major risk factors	Diagnosis	Treatment	Role for surgery
Aspergillus	Immunosuppression, diabetes	Radiographic (fungus ball, Monod's sign), serology, culture, Gomori methenamine silver, birefringent calcium oxalate crystals with polarizing microscopy, galactomannan test	Resect if symptomatic (hemoptysis most common symptom, initially treat with embolization), Antifungal for invasive pulmonary aspergillosis	Resect if symptomatic
Blastomyces	Immunosuppression, travel to endemic area (southeastern and central United States)	Microscopy (wide-based budding with double refractile walls), DNA-based (culture time-consuming, serology not useful)	Antifungal	Rule out malignancy
Coccidioides	Immunosuppression, travel to endemic area (Mexico, Central America, southwest United states)	Serology and culture	Antifungal for persistent symptoms	Complications (effusion, empyema, pneumothorax, bronchopleural fistula), rule out malignancy
Cryptococcus	Immunosuppression, exposure to pigeons	Microscopy (organism with capsule and narrow budding), culture	Check CSF if found in lung, amphotericin if meningitis	Rarely needed. To rule out malignancy
Entamoeba histolytica	Travel to endemic areas, poor sanitation	Fecal sample, aspiration of abscess	Antibiotics, occasional surgical drainage	Drainage of abscess if risk of rupture
Histoplasma	Immunosuppression, exposure to caves, endemic area (Mississippi Valley)	Isolation in culture, blood cultures, serologic tests (remote history), urine antigen levels	Amphotericin in disseminated histoplasmosis	Fibrosing mediastinitis, Alleviation of compression of adjacent structures. Rarely needed for pulmonary parenchymal lesions. To rule out malignancy
Mycobacterium tuberculosis	Immunosuppression, birth in endemic area, diabetes	PPD test (*M. tuberculosis*), sputum cultures, plain films, high level of adenosine deaminase in pleural/ pericardial fluid	Isoniazid and rifampin +/– pyrazinamide and ethambutol	Massive hemoptysis, bronchopleural fistula, bronchial stenosis, entrapped parenchyma, failure of medical treatment, persistent cavitary disease, destroyed lung, rule out malignancy, diagnosis (biopsy of pleura/ pericardium)
Mycobacterium, non-tuberculosis	Diseased lung, women, Caucasian	Sputum culture	More resistant to drug therapy	Consider surgery earlier
Mucorales	Diabetic ketoacidosis, steroids, neutropenia	Microscopy (broad aseptate hyphae with right-angled, finger-like projections)	Treat diabetic ketoacidosis, reverse immunosuppression, GM-CSF if neutropenic, amphotericin	Rapid and aggressive surgical resection
Pneumocystis jiroveci	Immunosuppression	Sputum sample, bronchoalveolar lavage, tissue biopsy	Bactrim	Not well established

CSF, cerebrospinal fluid; GM-CSF, granulocyte-macrophage colony-stimulating factor; PPD, purified protein derivative.

Table 2 Risk factors for lung abscesses (4,12-16)

Systemic
Age >65 years
Corticosteroid usage
Immunosuppression
Sepsis
Malnutrition
Diabetes
Alcoholism
Altered mental status/coma
Recumbent positioning
Cystic fibrosis
Focal
Seizure/neuromuscular disorders
Oropharyngeal dysfunction
Mechanical ventilation (endotracheal intubation)
Gastroesophageal reflux disease
Gingival/periodontal infections
Bronchial obstruction
Pneumonia
Underlying lung disease
Tube feeding (with overdistention)

Abscess

Background

Lung abscesses are cavities within lung parenchyma containing debris and fluid as products of an infection. Historically, abscesses were a surgically-managed disease but now infrequently require operative control. Mortality rates prior to described surgical intervention were approximately 75% for untreated abscesses (8). Surgical management in the 1920's and 1930's was predicated on drainage and debridement of affected tissue—rib resection, aspiration of purulence to identify the abscess, then performing cutdown and debridement of vitalized tissue—and this decreased mortality to approximately 20–35% (8). However, with development of antibiotics in the 1940's, and with interval improvements in therapy, surgical control is now only employed in about 10–15% of cases refractory to non-operative management (5,9-11).

Abscesses may be described as acute or chronic and have multiple risk factors (*Table 2*). Acute abscesses have symptoms of less than one month while chronic abscesses have symptom duration of over one month. Abscesses may also be described as primary or secondary. Primary abscesses, from direct inoculation of bacteria such as during aspiration events, represent 80% of abscesses (17) while secondary abscesses from underlying lung or systemic process, such as bronchial obstruction from cancer, septic emboli, and underlying lung problems like bronchiectasis, represent 20%. Organisms are often oral flora, and thus anaerobic and polymicrobial infections are common, each representing about 40% of all abscesses (12). Purely aerobic abscesses are less common and represent 10–15% of abscesses (10,12). Mortality remains elevated and highly variable, ranging from 5–75% in select patient series, especially with frequently nosocomial organisms such as *Staphylococcus aureus*, *Klebsiella pneumoniae*, and *Pseudomonas Aeruginosa* though other factors predicting death include larger abscess size, underlying lung disease, neoplasms, altered mental status, immunocompromise, airway obstruction, and hemoglobin levels under 10 g/dL (13-16).

Diagnosis

The most typical presenting symptoms are cough, fever, and purulent sputum (18,19). Night sweats, weight loss, pleuritic chest pain, and hemoptysis are also commonly described symptoms (10,18,19). These symptoms may be difficult to distinguish from non-cavitary pulmonary infections. Cough with purulent sputum demonstrates communication of the abscess cavity with the bronchial tree, but is not specific to abscess formation (19). Depending on the organism and underlying health of the patient, the usual course is indolent and resembles pneumonia though some bacteria, most notably Staphylococcus aureus, are known to drive necrotizing pneumonia, which has an explosive course and may frequently require surgical source control if sepsis develops (20,21). Empyema is associated in one third of cases, typically from direct extension of the causative organism across the visceral pleura into the pleural space (22). BPF may be present in these cases of empyema (22). Other organisms, such as mycobacterial and mycotic infections, may also form abscesses though these are far less common.

The diagnosis is reinforced by imaging. The differential diagnosis of cavitary lung lesions includes solid pulmonary lesions or underlying malignancy (primary lung, lymphoma, Kaposi's sarcoma), non-malignant cystic lesions (pulmonary

Table 3 Common treatment regimens for important bacterial pathogens (32-35,37)

Organism types	Suggested initial regimen
Oral flora	Beta lactam/beta lactamase inhibitor, carbapenem
Single pathogen (non-oral flora, non-putrid sputum)	Specific to organism sensitivity pattern
Single pathogen (non-oral flora, putrid sputum)	Beta lactam/beta lactamase inhibitor, carbapenem
Gram Negatives (*K. pneumoniae, P. aeruginosa*)	Specific to organism sensitivity (broad resistance)
Methicillin-sensitive *S. aureus*	Cefazolin, nafcillin, oxacillin
Methicillin-resistant *S. aureus*	Vancomycin, linezolid

sequestration, bronchial cysts), localized empyema, and granulomatous disease such as sarcoidosis and Wegener's granulomatosis (15). Atypical causes include bronchiolitis obliterans organizing pneumonia and lung necrosis after pulmonary embolism (15). Computed tomography (CT) is the imaging modality of choice due to ability to demonstrate anatomy that cannot be seen in plain films such as small abscesses or solid lesions, differentiate abscess from empyema, and identify underlying causes for abscess (22,23). Chest radiographs may show a thick-walled cavity, occasionally with an air-fluid level, and are usually sufficient to initiate treatment (23). Specificity of chest radiographs to detect abscesses is, in some select patient series, similar to CT; however, sensitivity of CT is superior (24).

Sputum cultures and gram stain should be obtained prior to starting empiric therapy; however, these results may be easily misinterpreted due to oral flora contamination and cultures selectively favoring aerobic causes (25,26). Trans-tracheal aspirate, transthoracic biopsy, bronchoscopic aspiration, and bronchoalveolar lavage are other methods employed to obtain culture diagnosis (26,27) but are either not frequently used today nor not well validated. Pleural cultures and blood cultures are useful adjuncts in specific cases but unlikely to identify anaerobes after empiric treatment is started. Regular bronchoscopy has limited use for diagnosis; however, has use if there is concern for anatomic alterations or mass lesions. In addition, it can help identify causal factors for patients that do not respond appropriately to antibiotic therapy (28).

Medical treatment

Historically, surgical control was necessary; however, with advancement of antibiotic regimens, the majority of patients with bacterial and mycobacterial causes do not require surgical intervention. Multiple successful courses are described in the literature (25,29-37). Intravenous antibiotics with beta lactam/beta lactamase inhibitors or carbapenems, which have good lung penetration and broad activity against anaerobes, are preferred for most organisms (*Table 3*) (10,33-37). Therapy type and duration is frequently dictated by likely causal organisms, speciation and sensitivity data, and clinical response. Most patients will respond clinically within 7–10 days and can be transitioned to oral therapy until radiographic improvement is demonstrated (35).

Treatment for non-bacterial causes vary widely based on the organism. Fungi, amoeba, and parasite causes have varying susceptibility patterns. Mycobacterium tuberculosis (TB) is historically treated with quadruple therapy of rifampin, isoniazid, ethambutol, and pyrazinamide, though newer regimens are more common with emerging worldwide resistance (36).

Surgical treatment

Surgical control of infection is not as commonly employed anymore due to the success of antibiotic regimens. However, 10 to 15 percent of patients do not clear their infection using antibiotic therapy or recur after therapy completion, develop pulmonary hemorrhage, or have underlying suspected neoplasms, and for these patients' surgical control is recommended (38,39). Other predictors for eventual need for operative source control include large abscesses at diagnosis (>6 cm), underlying lung disease such as severe chronic obstructive pulmonary disease (COPD), bronchiectasis, or obstructing lesions, particularly virulent pathogens causing necrotizing pneumonia, progressive hemoptysis, pulmonary sepsis, empyema with or without BPF (40,41). Studies have shown that concurrent empyemas increase intensive care unit admission rate, 30-day mortality, and overall mortality compared to patients with abscess alone (42). Resection is

typically lobectomy or segmentectomy with the goal being to remove the abscess and surrounding necrotic tissue that remains a nidus for further infection. Pneumonectomy is rarely needed, with rare exceptions largely being necrotizing infections with entire lung involvement (8,41,43). Few reports exist since 1980 in these mortality remains in the 10–25% range (41,43-46).

There is significant disagreement on the best time to perform lung resection—many of these patients are debilitated from increased catabolic activity from the acute infection, and with delays caused by attempted medical management, patients lose additional reserve and ability to tolerate major thoracic surgery. Some studies have touted open drainage to achieve source control as superior to resection though with open drainage, often a second stage of resection is subsequently needed (44,47). Others have demonstrated no survival disadvantage from single-stage procedure (43,45).

Much of the recent work on techniques for lung resection focus on resections for bronchiectasis and mycobacterial infections, with these series containing some patients having developed abscesses (47-51). While the original standards of resection involved open thoracotomy, video-assisted thoracoscopic surgery (VATS) has proven safe for resection in prospective studies (41,50).

Percutaneous and endoscopic drainage is well described in the literature, and is an effective means of control, especially in patients who may not tolerate a lung resection, or have other significant medical comorbidities that increase risk of operative control (52). Decision to proceed with surgical resection *vs* drainage, however, lacks well-validated direct comparison, and requires assessment based on individual patient factors, hospital resources, and operator skill. Multiple limited studies have demonstrated utility, with an average success rate of 84%, complication rate of 16%, and mortality rate of 5% (5). Risks of percutaneous drainage include pneumothorax, hemothorax, hemoptysis. Current overall mortality rate appears to be under 4–13% when surgery is performed by experienced surgeons (5,53). The feared complication is creation of a BPF by the drain tract, by which bacteria can enter the pleural space, causing an empyema.

Empyema

Background

Empyemas are infected pleural effusions. Infection occurs when microorganisms, often bacteria, cross the pleural membrane in the context of an effusion, commonly from heart failure, cirrhosis, nephrotic syndrome, bacterial or viral pneumonia, post-operative, or post-traumatic causes (7). Risk factors share overlap with abscesses, and include diabetes mellitus, alcohol abuse, gastro-esophageal reflux, and immunosuppression (54). They are a major source of morbidity because, unlike abscesses which are predominantly managed with antibiotics as primary therapy, empyemas usually require surgical drainage in addition to antibiotics (55,56). Even with operative intervention, empyemas carry significant risk of mortality; in one study, even with drainage by tube thoracostomy, 10% of patients still died (55).

Empyemas progress along a well stereotyped pathway in three phases: exudative, fibrinopurulent, and organizing (57). Exudative empyemas represent the earliest stage of development, and are characterized by either serous or progressively more purulent fluid. Exudative empyemas may have an acidotic pH (<7.2), diminished glucose (<40 mg/dL), elevated lactate dehydrogenase (>1,000 IU/dL), elevated protein (>2.5 g/dL), elevated white blood cell count (>500/μL), and elevated specific gravity (>1.018) (57). Bacteria may be seen on culture, but this is typically associated with later stages of empyema development. All of these features define an effusion with decreased function of the pleural lining or bacterial presence. Fibrinopurulent empyemas represent the progression of exudative empyema, where the infection has progressed with subsequent inflammatory response and neutrophil recruitment. Thick purulent fluid may be encountered, and developing fibrin deposition from coagulation cascade activation occurs as the body attempts to wall-off and localize the infectious process. This causes the pleural space to septate, a feature which may be identified on imaging. Organizing empyema represents the final progression of this immune response, as the fibrin deposition forms a rind covering the visceral pleura. With remodeling of this rind, a thick capsule is formed and the lung becomes trapped (58,59).

Diagnosis

Unlike abscesses, empyemas are less frequently polymicrobial, and tend to be composed predominantly of aerobic bacterial causes (60-62). Obtaining pleural fluid samples is important to identify the characteristics and determine the nature of the fluid, so simultaneous gram

stain and culture is feasible method for identifying the underlying organisms responsible. Aerobic and anaerobic cultures should be performed (60). Plain films may demonstrate a pleural effusion, but plural ultrasound is preferred as it allows for localization of collections prior to attempted aspiration (63). CT helps differentiate empyema from other lung disease and may be helpful from drainage but is not needed for initial evaluation given the utility of plain films and ultrasound.

Treatment

Empyemas, unlike abscesses, frequently require a surgical approach to control since the rapid progression and spread along the pleural space with fibrin deposition quickly causes a situation where antibiotic penetration is limited. Presence of frank pus or cloudy fluid on initial aspirate suggests bacterial infection and necessitates tube thoracostomy placement for source control (64). Antibiotics are used in combination with tube thoracostomy for initial management and source control and should be started as soon as infection is identified. Beta lactam/beta lactamase inhibitor therapy is recommended given the usual organism profile of an empyema, plus good penetration into the pleural space (63,64). In the setting of penicillin allergy, clindamycin may also be used (64).

Depending on the stage of the empyema, aspiration and/or chest tube placement alone may allow sufficient drainage for adequate antibiotic penetration and clearance of the infection. Open tube thoracostomy or image-directed placement of catheters are both well-accepted measures for treatment. For organized empyemas (fibrinopurulent or organizing), other factors such as loculations preventing adequate source control, persistent sepsis despite source control, and rind formation causing lung entrapment, are considerations for thoracoscopic versus open surgical management (65). Studies also show that administration of intrapleural tissue plasminogen activator (tPA) and DNase twice daily over 3 days via tube thoracostomy improves drainage and reduces need for surgical intervention, compared to chest tube and tPA or DNase monotherapy (65). In a study in which 210 patients with pleural infection were randomized one of 3 arms: (I) double placebo; (II) intrapleural tPA and DNase, and (III) tPA and placebo or DNA and placebo—frequency of surgical referral at 3 months was lower in the tPA-DNase group (2/48 patients, 4%) compared to the placebo group (8/51, 16%; P=0.003). Furthermore, tPA-DNase therapy was associated with a reduction in hospital stay compared to placebo (difference of 6.7 days, P=0.006). Chest tubes should be kept in place until clinical improvement of septic symptoms, and after confirmation of successful evacuation of the collection. For patients requiring operative intervention, VATS or open surgery may be performed for evacuation of all infected fluid plus excision of pleural rind and adhesions—VATS is usually better tolerated due to smaller incision size, and achieves similar results in terms of infection control and lung re-expansion (66). For patients with extensive involvement, failure to clear infection and/or chronic empyema, poor candidacy for major thoracic procedures such as decortication, creation of an Eloesser flap or Clagett window is an invasive but established option for control, and creates a large path for drainage, plus direct treatment of the involved pleural surfaces (67,68). A proposed algorithm for diagnosis and evaluation of empyema, along with lung abscesses, is proposed in *Figure 1*.

Broncho-pleural fistula

TB and atypical mycobacterial diseases

Due to an overall increase in the global incidence of TB, we have seen a resurgence of surgery in the management of TB. In addition, this is also due to an increase in human immunodeficiency virus incidence, improved survival of immuno-compromised patients, and the emergence of multi-drug-resistant TB (MDR-TB) since the mid-1980s.

A BPF is a serious and most feared complication after TB surgery. BPF in the post-operative setting indicates a breakdown of the bronchial stump. TB patients are at particular risk for BPF given their often poor nutritional status at the time of surgery and the usually already inflamed/infected bronchi. In reviewing large series (>20 patients) since 2000, BPF rate after lung resection for TB is in the range of 0–6.6% (*Table 4*) (69-82). There are no factors which have consistently been associated with BPF; however, some observations can be made. Bronchial stump reinforcement has been shown to decrease BPF in only one study (82). Despite the lack of clear evidence, most thoracic surgeons reinforce the bronchial stump with vascularized autografts, especially for pneumonectomy. The flaps can be fashioned from the pericardial fat pad, pleura, pedicled muscles such as intercostal, latissimus, or serratus, or omentum. The

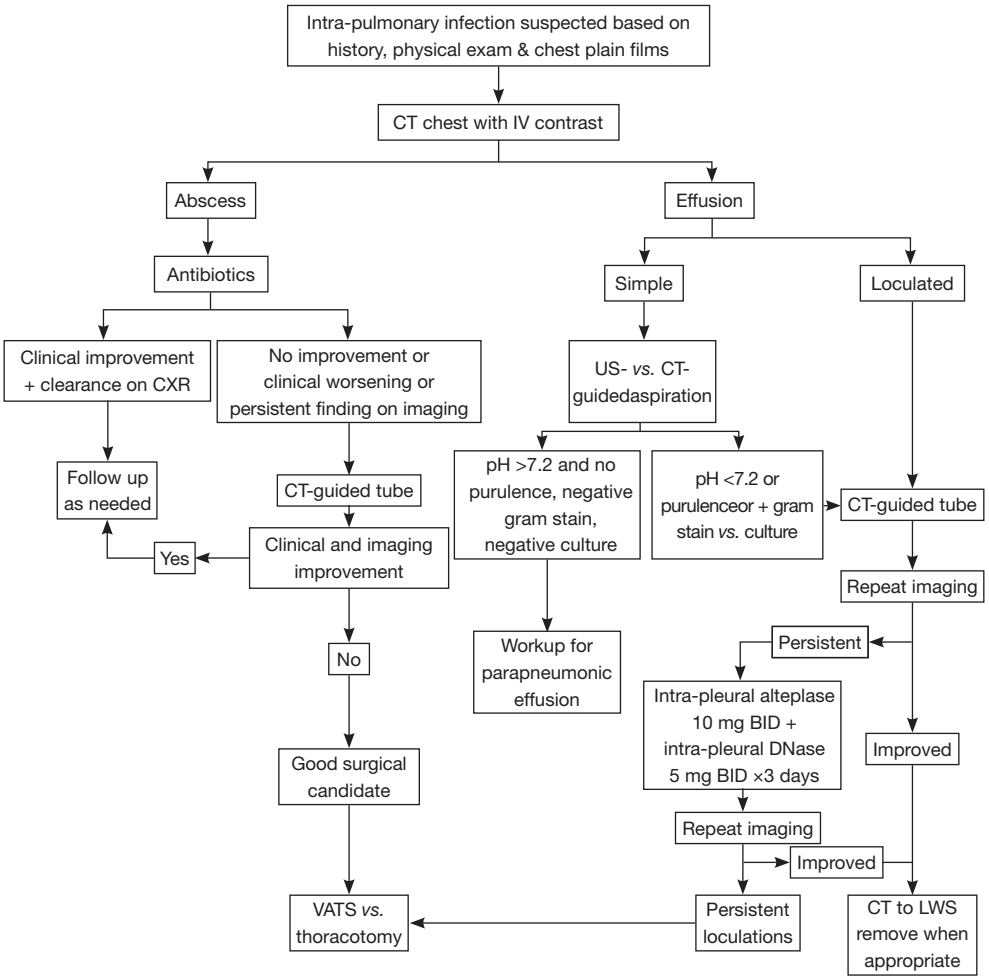

Figure 1 Diagnostic and management algorithm for lung abscess/empyema. CT, computed tomography; IV, intravenous; US, ultrasound; CXR, chest X-ray; LWS, low wall suction; VATS, video-assisted thoracoscopic surgery; BID, twice per day.

need for flaps highlights the importance of preoperative nutrition status. The type of closure (sutures versus stapler) also does not appear to be a risk factor. While peri-operative sputum positivity was found to be associated with increased BPF in one study (82), this also has not been found to be true in other studies since. In two studies, BPF was strictly seen in late post-operative period in the setting of disease relapse (70,79). This calls for strict control of disease peri- and post-operatively, especially in MDR-TB. In addition, in one study, endobronchial TB has been identified as a risk factor for BPF (72). For this reason, some surgeons perform frozen section to rule endobronchial TB at the time of resection.

Conclusions

Here we present a review of historical and current management for surgically-treated pulmonary infections; lung abscesses, empyemas, and BPFs. As treatments continue to evolve and improve, the role of the surgeon for the management of pulmonary infection will also change. The skills needed to operate on these infections are the accepted technical standard for thoracic surgeons. Thus, in this evolving landscape of infection management, current knowledge of accepted and changing treatment practices is necessary for the thoracic surgeon to provide optimal care for patients with these significant, often life-threatening infections.

Table 4 Summary of surgical series for pulmonary tuberculosis and bronchopleural fistula findings since 2000 (69-82)

Author/Journal	# patients [resections]	Resection type	Stump coverage	BPF rate	Factors associated with BPF
Kang *et al*., Ann Thorac Surg 2010 (69)	72 [73]	3 CP (4%), 20 P (28%), 25 L (35%), 12 L + S (17%), 2 L + W (3%), 10 S (14%), 1 W (1%)	12 (17%)	0	–
Shiraishi *et al*., J Thorac Cardiovasc Surg 2009 (70)	56 [61]	3 CP (5%), 19 P (31%), 25 L (41%), 8 L + S (13%), 6 S (10%)	54 (89%)	3 (4.9%)	Disease relapse
Orki *et al*., Thorac Cardiovasc Surg 2009 (71)	55 [56]	1 CP (2%), 17 P (31%), 37 L (67%), 1 L + S (2%)	18 (33%; for all pneumonectomy)	2 (3.6%)	–
Wang *et al*., Ann Thorac Surg 2008 (72)	56 [56]	25 P (45%), 31 L (55%)	42 (75%; 30 parietal pleura, 12 pericardium)	9 (16.1%)	Endobronchial disease, no bronchial reinforcement
Mohsen *et al*., J Thorac Cardiovasc Surg 2007 (73)	23 [23]	11 P (48%), 12 L (52%)	All (100%)	1 (4.3%)	–
Naidoo, Asian Cardiovasc Thorac Ann 2007 (74)	106 [106]	59 P (56%), 47 L (44%)	0	1 (0.9%)	No relation to sputum status
Kir *et al*., J Thorac Cardiovasc Surg 2006 (75)	79 [81]	4 CP (5%), 39 P (48%), 30 L (37%), 7 L + S (9%), 1 S (1%)	4 pneumonectomy (4.9%)	4 (4.9%)	No relation to sputum status
Kim *et al*., Eur Respir J 2006 (76)	79 [79]	17 P (19%), 55 L (63%), 7 W (8%)	NR	4 (5.1%)	–
Somocurcio *et al*., Thorax 2007 (77)	117	27 P (22%), 76 L (63%), 11 L + S (9%), 3 S/W (2%)	NR	8 (6.6%)	–
Takeda *et al*., Ann Thorac Surg 2005 (78)	35	7 P (20%), 22 L (63%), 5 L + S (14%), 1 S (3%)	5 (14%; pericardial fat pad)	0	–
Shiraishi *et al*., J Thorac Cardiovasc Surg 2004 (79)	30 [33]	12 P (36%), 17 L (52%), 4 S (12%)	30 (91%; 29 latissimus dorsi, 1 pericardial fat)	2 (6.1%)	Disease relapse
Park *et al*., Int J Tuberc Lung Dis 2002 (80)	49 [47]	12 P (24%), 28 L (57%), 7 L + S/L + W (14%)	0	0	No relation to sputum status
Chiang *et al*., Int J Tuberc Lung Dis 2001 (81)	27 [27]	10 P (37%), 13 L (48%), 4 S/W (15%)	NR	1 (3.7%)	–
Pomerantz *et al*., J Thorac Cardiovasc Surg 2001 (82)	172 [180]	16 CP (9%), 66 P (37%), 93 L (52%), 5 S (3%)	92 (51%; 91 muscle, 1 omentum)	5 (2.7%)	Positive sputum

CP, completion pneumonectomy; P, pneumonectomy; L, lobectomy; L + S, lobectomy/segmentectomy; L + W, lobectomy/wedge; S, segmentectomy; W, wedge; BPF, bronchopleural fistula; NR, not reported.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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