



Case Report



# Challenging Management of Postoperative Empyema: A Case Report with Literature **Review**

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Introduction: Pleural empyema is the collection of pus within the pleural cavity, typically arising as a complication of pneumonia, chest trauma, thoracic surgery, or bacterial invasion of the pleural space. This report presents a case of post-surgical pleural empyema caused by Pseudomonas aeruginosa, successfully managed with a targeted combination of fosfomycin and colistin, with intrapleural lavage.

Case Presentation: A 37-year-old male developed epigastric pain 12 days after a laparoscopic near-total gastrectomy. A chest computed tomography scan revealed a right-sided pleural empyema. Ultrasound-guided drainage was performed, followed by the intrapleural instillation of alteplase to facilitate breakdown of the loculated empyema. Pseudomonas aeruginosa was identified as the causative agent. Based on antimicrobial susceptibility, the patient received intravenous fosfomycin and colistin, along with daily pleural lavage using colistin. Inflammatory markers declined, and the patient showed notable clinical improvement.

Literature Review: A review of five cases of Pseudomonas aeruginosa pleural empyema was conducted, including two carbapenem-resistant and one extensively drug-resistant case. The mean patient age was 53.8 years, and 60% (3/5) were female. Four of the five cases (80%) were confirmed using computed tomography, and all patients received antimicrobial therapy, most frequently ceftolozane/tazobactam (60%), ciprofloxacin (60%), and colistin (40%). Surgical management was required in 60% of cases, whereas bacteriophage therapy was utilized in 20%. During follow-up, 60% of patients remained stable, 20% experienced repeated hospital admissions during which antibacterial therapy was withheld, and 20% died due to infectious disease.

Conclusion: Pleural lavage combined with antibiotics such as fosfomycin and colistin may provide an effective treatment for postoperative pleural empyema, with early intervention being critical to prevent clinical deterioration.

Keywords: Pleural empyema, Pseudomonas aeruginosa, intrapleural lavage, colistin, antibiotic, antibiotic resistance

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## 1. Introduction

Pleural empyema is the accumulation of pus within the pleural cavity. Although its pathogenesis is not entirely clear, it typically arises from pneumonia, chest trauma, postoperative complications following thoracic surgery, or bacterial invasion from adjacent infection [1]. Epidemiological data from France report an incidence of approximately 0.78 cases per 10,000 people annually [2]. Mortality is not uncommon, with overall rates around 17%, and most patients require hospitalization [2]. Approximately 25–50% of patients with pneumonia develop a parapneumonic effusion, which may progress to empyema in some cases [3]. The factors contributing to the progression of pneumonia to empyema remain poorly understood [4]. However, pleuritic pain, higher severity scores for community-acquired pneumonia, multilobar involvement on imaging, and elevated inflammatory markers have been associated with an increased risk of progression. In contrast, administration of systemic corticosteroids at the time of admission has been linked to a reduced risk, possibly by decreasing pleural inflammation [5]. The use of corticosteroids in community-acquired pneumonia remains debated: the Infectious Diseases Society of America recommends them only for septic shock unresponsive to vasopressors, whereas European guidelines suggest their use more broadly in cases of shock [5].

Pseudomonas aeruginosa is a widely distributed Gram-negative bacterium that can be found in various environmental settings and hosts [6]. It can cause various infections in humans, especially affecting the respiratory tract of patients with cystic fibrosis [7]. Its clinical impact is amplified by intrinsic antibiotic resistance, mediated through biofilm formation, antibiotic-modifying enzymes, multiple efflux pumps, and low cell permeability, making infections particularly challenging in immunocompromised or chronically ill patients [6].

This report presents a case of pleural empyema due to *Pseudomonas aeruginosa* approximately two weeks after a laparoscopic near-total gastrectomy, successfully managed with a combination of fosfomycin and colistin alongside intrapleural lavage. This report was prepared in accordance with CaReL guidelines, and all references cited have been carefully assessed for credibility [8, 9].

## 2. Case Presentation

#### 2.1. Patient Information

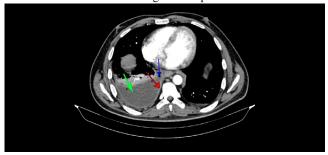
A 37-year-old male smoker presented with epigastric pain, low-grade fever, anorexia, and generalized weakness following 12 days after laparoscopic near-total gastrectomy with spleen-preserving D2 lymph node dissection for gastric cancer. The patient had a recent influenza infection three weeks prior to surgery. Other than the gastrectomy, no significant medical or surgical history was reported.

## 2.2. Clinical Findings

Upon examination, he appeared agitated and fatigued, with a generally ill appearance. Vital signs showed tachycardia (heart rate 105 bpm) and tachypnea (25 breaths per minute), while oxygen saturation and blood pressure remained within normal limits. Chest auscultation revealed diminished breath sounds over the right lower lung.

## 2.3. Diagnostic Approach

Laboratory investigations revealed markedly elevated inflammatory markers, with a C-reactive protein (CRP) level of 180 mg/L (Normal range: < 3 mg/L) and an erythrocyte sedimentation rate (ESR) of 95 mm/hr (Normal range: < 15 mm/hr). Chest computed tomography (CT) confirmed the presence of a right-sided pleural empyema (Figure 1), while abdominal CT ruled out an anastomotic leak. These findings suggested the empyema likely resulted from postoperative pneumonia progressing to a complicated pleural infection rather than a direct surgical complication.



**Figure 1.** CT demonstrates mild right side pleural effusion (green arrow), small air bubbles inside (blue arrow), thickening & enhancement of parietal & visceral pleura (red arrow), and adjacent right lung atelectatic changes noted.

#### 2.4. Therapeutic Intervention

Ultrasound-guided drainage was performed, and a chest tube was inserted, initially draining 100 cc of pus. The following day, 50 mg of alteplase diluted in 100 cc of normal saline was administered through the tube. After a 4-hour dwell time, drainage increased to 500 cc, indicating effective fibrinolysis and breakdown of loculated empyema. Empirical broad-spectrum antibiotic therapy with meropenem (1 g every 8 hours) was initiated pending culture results, and the patient maintained a regular oral diet.

#### 2.5. Culture Results and Targeted Antimicrobial Therapy

Microbiological analysis of the drained pus, performed using the advanced VITEK-II Compact (bioMérieux) diagnostic system, identified *Pseudomonas aeruginosa* as the causative agent. Antimicrobial susceptibility testing showed that the strain was multidrug-resistant (MDR), with sensitivity limited to colistin, aztreonam, and fosfomycin (Table 1). The patient received intravenous fosfomycin (4 g every 8 hours for 7 days) and colistin (initially 3 million units every 8 hours for 7 days, followed by 1 million units every 8 hours for 12 days). Pleural lavage with colistin was performed every 12 hours for 5 days, once daily for 7 days, and then on alternate days for an additional 7 days to enhance local antibiotic concentrations.

### 2.6. Monitoring and Outcome

Daily clinical assessments and serial inflammatory marker measurements were performed. Vital signs normalized, CRP returned to normal, and ESR decreased to 40 mm/hr. Follow-up cultures from the chest drain were negative. Given significant clinical improvement, the chest tube was removed, and the patient was discharged in stable condition. He was subsequently referred to an oncology for ongoing management of gastric cancer. At 10 months post-treatment, there was no evidence of recurrence. The patient has completed adjuvant chemotherapy and continues to undergo regular follow-up with upper gastrointestinal endoscopy and chest and abdominal CT scans.

## 3. Discussion

Empyema has increasingly been recognized as a reservoir for resistant bacteria, largely because many antibiotics penetrate the pleural space at subtherapeutic concentrations, thereby promoting resistance development [10]. Clinically, the disease progresses through three distinct stages—uncomplicated parapneumonic effusion, complicated parapneumonic effusion, and established empyema—which highlight its dynamic pathophysiology and the need for timely intervention [1]. In parallel with this progression, the incidence of MDR and XDR Pseudomonas aeruginosa has risen substantially. This trend is driven by the organism's ability to cause severe healthcare-associated infections, its exceptional capacity to accumulate and spread resistance determinants within the host, and the global dissemination of high-risk clones. As a result, infections caused by these strains pose significant clinical challenges and are associated with increased morbidity and mortality due to limited therapeutic options [11]. The severity of empyema and its outcomes are reflected in epidemiologic data. Bobbio et al. studied adults hospitalized with pleural infections in France between 2013 and 2017, identifying 25,512 empyema cases with an annual incidence of 7.15–7.75 per 100,000 inhabitants. Mortality rates varied substantially depending on underlying conditions, reaching 30% in cancer patients, 18% after lung resection, and 11% among patients without these comorbidities [2]. This variability underscores the importance of understanding pathogen behavior in the context of patient-specific risk factors.

<b>Table 1.</b> Antimicrobial susceptibility testing result	Table 1.	Antimicrobial	susceptibility	testing results
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Antimicrobials	Inhibition zone (mm)	Interpretation	Antimicrobials	Inhibition zone (mm)	Interpretation
Cefixime	0	Resistant	Moxifloxacin	0	Resistant
Cefuroxime	0	Resistant	Levofloxacin	0	Resistant
Cefotaxime	0	Resistant	Doxycycline	0	Resistant
Ertapenem	0	Resistant	Minocycline	0	Resistant
Cefpodoxime-clavulanate	0	Resistant	Tigecycline	0	Resistant
Aztreonam	20	Sensitive	Nitrofurantion	0	Resistant
Rifampin	0	Resistant	Streptomycin	0	Resistant
Colistin	19	Sensitive	Azithromycin	0	Resistant
Fosfomycin	24	Sensitive	Clarithromycin	0	Resistant

Among the common pathogens, *Pseudomonas aeruginosa* remains a notable cause of hospital-acquired pneumonia and is frequently detected in airway colonization, especially in previously treated individuals. The development of MDR phenotypes not only complicates therapy but may also alter the organism's virulence characteristics [12]. Its adaptability is further demonstrated by its ability to colonize diverse hospital environments, including soap solutions and saline, facilitating persistent contamination and transmission. Consequently, treatment strategies must be tailored to the organism's susceptibility pattern and the clinical scenario, often relying on agents such as colistin, ceftazidime, meropenem, and amikacin. However, therapeutic success is hindered by multiple resistance mechanisms, ranging from chromosomal mutations to horizontally acquired β-lactamases [13].

A review of five similar cases of empyema illustrates this complexity. The cases involved patients aged 22-77 years, with Pseudomonas aeruginosa identified in all isolates. While two isolates were carbapenem-resistant and one was XDR, resistance profiles were not reported for the remaining two. Imaging modalities, most commonly CT, confirmed empyema or associated cavitary lesions. All patients received antimicrobial therapy, with ceftolozane/tazobactam, colistin, and ciprofloxacin being most frequently used. Surgical interventions such as thoracotomy or video-assisted thoracoscopic surgery were required in 60% of patients, while adjunctive measures like bacteriophage therapy were rarely used (20%). Only one patient died, emphasizing both the severity of these infections and the potential for successful outcomes when appropriate therapy is provided (Table 2) [10, 14-17]. In the present case, treatment with intravenous fosfomycin in combination with colistin was guided by the antimicrobial susceptibility profile and resulted in a favorable clinical response. Evidence supports the synergy of this combination, in a study of 87 carbapenem-resistant Pseudomonas aeruginosa isolates, the fosfomycin and colistin regimen exhibited synergistic or partially synergistic effects in nearly half of the isolates and significantly reduced colistin MIC values compared with monotherapy [18]. These findings suggest that fosfomycin may enhance colistin's efficacy and therapeutic window. Conversely, when resistance to fosfomycin does occur, it is typically mediated through FosA overexpression or inactivation of the glycerol-3-phosphate transporter responsible for drug uptake [19].

The relevance of this combination is supported by a comparable case involving a 64-year-old patient with chronic post-pleuropneumonectomy empyema caused by carbapenem-resistant *Pseudomonas aeruginosa* [16]. In that case, surgical debridement followed by cefiderocol, fosfomycin, and colistin—along with pleural lavage using colistin—resulted in clinical improvement, highlighting the potential role of both systemic and local therapy. Monitoring inflammatory markers, especially C-reactive protein, further assists in assessing response, as a rapid decline typically correlates with resolution of infection [20], consistent with the trajectory observed in the present case.

Given the rise of resistance, interest has grown in adjunctive and alternative therapeutic approaches. Bacteriophage therapy is particularly notable, as combining phages with antibiotics can produce synergistic antimicrobial effects, a finding supported by multiple in vitro studies [13]. Although limited by narrow host ranges, phages may offer options for otherwise untreatable infections [21]. This was exemplified by Maddocks et al., who treated a 77-year-old patient with ventilator-associated pneumonia and empyema due to *Pseudomonas aeruginosa* using the AB-PA01 phage cocktail after resistance emerged during antibiotic therapy. Intravenous and nebulized administration led to gradual clinical improvement, demonstrating the potential of phage—antibiotic combinations in refractory cases [17].

Further emerging strategies include vaccines and monoclonal antibodies targeting virulence factors such as the type III secretion system. Candidates like IC43, KB001-A, and KBPA-101 are being investigated for preventive and therapeutic roles in high-risk populations [11]. In the current case, such alternatives were not required, as the selected antibiotic regimen successfully controlled the infection without the need for surgical intervention.

A limitation of this study is the unavailability of certain clinical data, including abdominal CT images obtained to exclude an anastomotic leak and specific laboratory values..

## 4. Conclusion

Pleural lavage combined with antibiotics such as fosfomycin and colistin may provide an effective treatment for postoperative pleural empyema, with early intervention being critical to prevent clinical deterioration.

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**Table 2.** Review of five cases of empyema caused by *Pseudomonas aeruginosa*.

Author, year of publication	Age (years)	Sex	Clinical finding	Diagnosis	Imaging	Antimicrobial susceptibility	Treatment	Outcome
Sousou <i>et al.</i> 2024 [ <u>14</u> ]	61	M	Left-sided chest pain and shortness of breath, worsening dyspnea on exertion.	Influenza A initially, later empyema from <i>P. aeruginosa</i> .	CT: large left pleural effusion. US: extensively multiloculated, dense pleural fluid.	Sensitivity: cefepime & cipro- floxacin	Video-assisted thoracoscopic surgery, & left thoracotomy with complete decortication. First given cefepime then ciprofloxacin.	Stable
Parra <i>et al</i> . 2022 [ <u>15</u> ]	22	F	respiratory failure with necrotizing pneumonia	Pleural empyema due to carbapenem-resistant <i>P. aerugino</i> sa.	Radiologically compatible with necrotizing pneumonia.	Resistance: ceftolozane/tazobact- am & ceftazidime-avibactam.	Intravenous ceftazidime later ceftolozane/ tazobactam, then changed to ciprofloxacin & nebulized colistin.	Deceased
Borghesi <i>et al.</i> 2021 [ <u>16</u> ]	64	M	Increased discharge from the pleural drainage.	Chronic left pleural empyema caused by a carbapenem-resistant <i>P. aeruginosa</i> .	A CT scan of the thorax was sugges- tive of interstitial pneumonia.	Resistance: all β-lactams & quinolones. Sensitivity: ceftolozane/tazobactam, gentamicin, amikacin & colistin.	ceftolozane/tazobactam & gentamicin. Surgical debridement procedure, cefiderocol, colistin & Fosfomycin.	Stable
Kufel <i>et al.</i> 2020 [ <u>10</u> ]	45	F	Leukocytosis, respiratory failure, fever, & increased tracheal secretions.	Empyema caused by XDR <i>P. aeru-ginosa</i> .	CT: loculated left & right pleural effusions.	Sensitivity: cefiderocol, but later 2 cefiderocol-resistant morphologies of XDR <i>P. aeruginosa</i> were found.	Ceftazidime-avibactam, polymyxin B, & fluco- nazole, later fluconazole & cefiderocol. Esoph- ageal stent replacement, video-assisted thora- coscopic surgery with left lung thoracotomy & decortication, & esophageal rupture repair with muscle flap placement.	Repeated hospital admissions; antibacteri- al therapy withheld
Maddocks <i>et al.</i> 2019 [ <u>17</u> ]	77	F	Pleuritic chest pain, respiratory distress, drowsiness & fever, parenchymal cavi- tation.	Ventilator-associated pneumonia and empyema caused by <i>P. aeruginosa</i> .	CT: cavitation, consolidation, empyema, & subcutaneous emphysema. X-ray: right lung field consolidation, & later pneumothorax.	Sensitivity: piperacillin tazobactam, ciprofloxacin, & meropenem. But later resistant to meropenem, imipenem, piperacillin-tazobactam in vitro, & fluoroquinolone.	First meropenem, then gentamicin, & ciproflox- acin later ceftolozane/tazobactam. Bacteriophage therapy: two Myoviridae & two Podoviridae.	Stable

P. aeruginosa: Pseudomonas aeruginosa, M: Male, F: Female, CT: Computed tomography, US: Ultrasonography, XDR: Extensively drug-resistant.

#### **Declarations**

**Conflicts of interest:** The authors have no conflicts of interest to disclose.

Ethical approval: Not applicable.

Consent for participation: Not applicable.

Consent for publication: Written informed consent for publication was obtained from the patient.

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**Use of AI:** ChatGPT-4.5 was used to assist with language refinement and improve the overall clarity of the manuscript. All content was thoroughly reviewed and approved by the authors, who bear full responsibility for the final version.

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