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Early Detection and Management of Pseudomonas Bacteremia in a Patient With Complex Cystic Fibrosis: A Case Report

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Abstract

Cystic fibrosis (CF) is an autosomal recessive genetic disorder caused by mutations in the cystic fibrosis transmembrane conductance regulator (*CFTR*) gene. Affected individuals have defective chloride channels, resulting in abnormal fluid transport across epithelial surfaces. These abnormalities can lead to systemic diseases and complications, with the most prominent being recurrent pulmonary infections. This report describes the case of a 28-year-old male patient who was diagnosed with CF at birth and had a past medical history positive for recurrent small-bowel obstructions, multiple pulmonary embolisms, bronchiectasis, and recurrent pneumonias with polymicrobial growth found on bronchoscopy. He also had chronic respiratory failure, requiring 2 L of nasal cannula oxygen therapy at home for 24 hours. Initial antibiotic therapy included IV piperacillin-tazobactam and vancomycin, then cefepime and vancomycin, which only partially treated the systemic bacteremia. After susceptibility testing, IV cefepime was discontinued, and IV meropenem and amikacin were added to the regimen. This antimicrobial therapy resulted in normalization of the white blood cell count and clearance of bacteremia prior to discharge. The patient was prescribed oral ciprofloxacin based on susceptibility testing at discharge to complete an extra 14-day course of antibiotics to eliminate the possible recurrence of systemic bacteremia. This case highlights the importance of early detection of antimicrobial susceptibility testing when initial empiric antibiotic therapy is less effective at treating bacteremia. Furthermore, it emphasizes the importance of prompt blood culture testing to quickly identify the causative organism and determine susceptibilities to effectively treat infections in patients with comorbidities who are at risk of being infected with multidrug-resistant organisms, while starting immediate treatment with a broad-coverage antibiotic regimen.

Categories: Genetics, Internal Medicine, Infectious Disease
Keywords: antimicrobial susceptibility testing, antimicrobial therapy, bacteremia, cftr , cystic fibrosis, pseudomonas aeruginosa

Introduction

Cystic fibrosis (CF) is an autosomal recessive disorder that impacts various epithelial tissues throughout the body. It arises from a mutation in the *CFTR* (cystic fibrosis transmembrane conductance regulator) gene, leading to a defective chloride channel on the apical plasma membranes of epithelial cells. This defect predisposes patients to a range of complications, including recurrent respiratory infections, malabsorption, pancreatitis, liver disease, small bowel obstruction, and diabetes [1].

A major concern for individuals with CF is respiratory compromise, primarily caused by thick, adhesive secretions that obstruct airflow. These secretions can further compromise respiration by making it difficult to clear complex bacterial flora, including *Pseudomonas aeruginosa, Staphylococcus aureus, and Haemophilus influenzae* [2]. The frequent recurrence of respiratory infections in individuals with CF leads to long-term pulmonary deterioration and poses challenges for effective management and treatment. While the median survival age for CF patients has improved over the past decade, chronic pulmonary infections and airway diseases continue to be significant contributors to respiratory failure, responsible for 80-95% of deaths [3]. The warning signs usually include a falling forced expiratory volume in one second (FEV_1) below 80% and failure to recover to baseline following a course of antibiotics, new growth of *P. aeruginosa*, weight loss, and increasing frequency of pulmonary exacerbations [4,5].

During an exacerbation requiring intravenous therapy, an antipseudomonal beta-lactam antibiotic such as ceftazidime, cefepime, piperacillin-tazobactam, or meropenem is used [6]. These medications are conventionally paired with an agent from a different class, most often once-daily tobramycin with serum level monitoring [6]. The Cystic Fibrosis Foundation found insufficient evidence to consider single-agent therapy equivalent to multi-class therapy for *Pseudomonas* during an exacerbation, and recommends once-daily over thrice-daily aminoglycoside dosing [6]. In a patient with recurrent polymicrobial pneumonia, empiric coverage must extend beyond *P. aeruginosa* to co-pathogens documented on prior blood cultures. For this purpose, vancomycin or linezolid should be added to address methicillin-resistant *S. aureus* (MRSA),

and trimethoprim-sulfamethoxazole can be considered for *Stenotrophomonas maltophilia* [6].

This report describes the case of a patient with several predisposing factors, including a history of recurrent pneumonia in which bronchoscopic cultures yielded multiple organisms, including MRSA, *P. aeruginosa, S. maltophilia, and Mycobacterium avium* complex (MAC). The history included recurrent small bowel obstruction (SBO), multiple pulmonary emboli, and gastrojejunostomy (GJ) tube placement. The combination of chronic polymicrobial airway infection and repeated SBOs leading to immune modulation, and long-term enteral access provides more than one plausible portal for positive blood culture results confirming *Pseudomonas* bacteremia [4-6]. It is important to note the rarity of *Pseudomonas* bacteremia in CF patients because their intact immune defenses and biofilm-bound airway organisms typically prevent the bacteria from spreading into the bloodstream [4,7]. The case presented here examines the early detection and treatment of *Pseudomonas* bacteremia in a medically complex patient with CF.

Case Presentation

A 28-year-old Caucasian male patient with a history of CF presented to the emergency room with chest pain, hemoptysis, and severe upper abdominal pain rated 10/10 that started one day prior. The patient experienced constant pain without vomiting or constipation and reported no prior interventions. The following day, the patient developed a productive cough with brown and bloody sputum, along with shortness of breath, but denied palpitations, light-headedness, or syncope. The patient described the chest pain as diffuse, pleuritic, and worsening with cough and deep breaths.

The patient stated he was diagnosed with CF at birth; however, diagnostic values of sweat chloride, *CFTR* genetic analysis, immunoreactive trypsinogen (IRT), nasal potential difference (NPD), and intestinal current measurement (ICM) were not available during this visit. The patient had a significant past medical history of multiple pulmonary embolisms, recurrent small bowel obstructions, bronchiectasis, recurrent pneumonias, mycobacterial abscesses, and chronic respiratory failure on 2L nasal cannula every 24 hours on home oxygen therapy. Polymicrobial growth was found in past bronchoscopies, including MRSA*, Pseudomonas group A, Stenotrophomonas*, and MAC. He had two past pulmonary embolisms, for which he was on oral anticoagulants (Apixaban) for six months, but had not taken any anticoagulants for the last two years. The patient denied inferior vena cava filter (IVCF) placement and was on a regular diet. He had a GJ tube placed primarily for occasional laxative administration due to recurrent bowel obstructions. His surgical history also included partial colectomy, small bowel resection, and ventral hernia repair. Medications being taken included ipratropium-albuterol as required for shortness of breath and polyethylene glycol to treat gastrointestinal complications. He did not use tobacco, alcohol, or recreational drugs and was allergic to several medications.

On arrival at the emergency department, his vital signs were abnormal as shown in Table *1*. He was tachycardic with a pulse of 120 beats/minute and tachypneic with a respiratory rate of 22 breaths/minute. Blood pressure was elevated at 142/94 mmHg, pulse oximetry was 94% on supplemental oxygen, and temperature was 99.3°F. The physical examination was unremarkable, including atraumatic head and scalp, extraocular movements intact bilaterally, breath sounds equal bilaterally, and regular rhythm noted. The patient presented with a non-infected GJ tube, extremities unremarkable on inspection, and no focal motor or sensory deficits noted. Complete blood count (CBC) results on admission showed leukocytosis with a white blood cell count of 15.1 X 10^3/μL, with neutrophils at 86.6% and lymphocytes at 3.4%. Mild anemia was noted, with a red blood cell count of 3.76 X 10^6/μL and hemoglobin of 10.0 g/dL. Platelets, D-dimer, troponin, and lipase were unremarkable, as seen in Table *2*. A heart rate over 90 beats/minute and a respiratory rate over 20 breaths/minute, together with the leukocytosis, met three of the four Systemic Inflammatory Response Syndrome (SIRS) criteria.

Parameters	Initial Patient Values (Day 0)	Final Patient Values (Day 10)	Reference Range (with units)
Temperature	99.3 °F	98.1 °F	97.8°F - 99.1°F
Pulse	120 bpm	84 bpm	60-100 beats per min (bpm)
Respirations	22 bpm	18 bpm	12-18 breaths per min
Blood Pressure	142/94 mmHg	135/85 mmHg	<120/80 mmHg
Pulse Oximetry	94%	98%	95% -100% on room air

TABLE 1: Significant presenting vital signs

Parameters	Day 0 Patient Values	Day 5 Patient Values	Day 10 (final) Patient Values	Reference Range
White Blood Cells	15.1 X 10^3/µL	3.4 X 10^3/µL	5.6 X 10^3/µL	4.8–10.8 X 10^3/µL
Red Blood Cells	3.76 X 10^6/µL	3.39 X 10^6/µL	3.52 X 10^6/µL	4.7-6.1 X 10^6/µL (male)
Hemoglobin	10.0 g/dL	9.0 g/dL	9.3 g/dL	13.5 – 17.5 g/dL (male)
Platelets	341,000/mm^3	86,000/mm^3	165,000/mm^3	150,000 – 450,000/mm^3
Neutrophil %	86.6 %	65.60 %	69.0 %	40.0 - 74.0 %
Lymph %	3.4 %	21.4 %	19.9 %	19.0 - 48.0 %
D-dimer	0.42 mg/L	Not repeated	Not repeated	0 - 0.49 mg/L
Troponin	2.70 ng/L	Not repeated	Not repeated	0.00 - 60.00 ng/L
Lipase	<4 U/L	Not repeated	Not repeated	10 - 60 U/L
Procalcitonin	TNP	Not ordered	21.54 ng/mL	<0.10 ng/mL

TABLE 2: Significant laboratory values

Chest X-rays (Figures *1*, *2*), taken on the day of admission, showed mild reticulonodular opacities without consolidation, effusion, or pneumothorax. Due to a history of anaphylactic reaction to iodinated contrast, a CT of the chest without contrast was taken on day 2 of admission to assess lung and pleural morphology. It showed scattered bronchial wall thickening and bronchiectasis within the right upper lobe. A small quantity of peribronchial consolidation was seen within the lingula, as well as diffuse centrilobular nodules (Figures *3*-*5*).

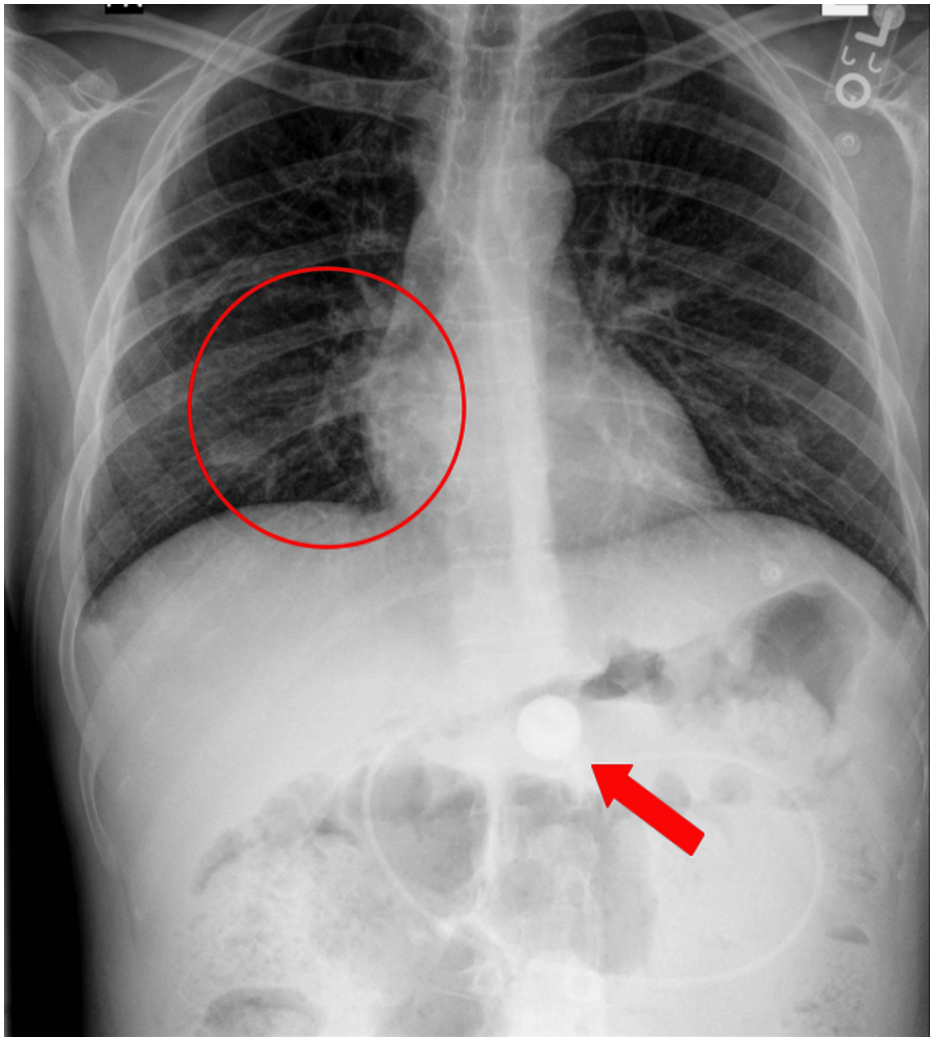


FIGURE 1: Chest X-ray (anteroposterior view) showing mild reticulonodular opacities (circle).

Gastrojejunostomy tubing is visible on the midline (arrow).

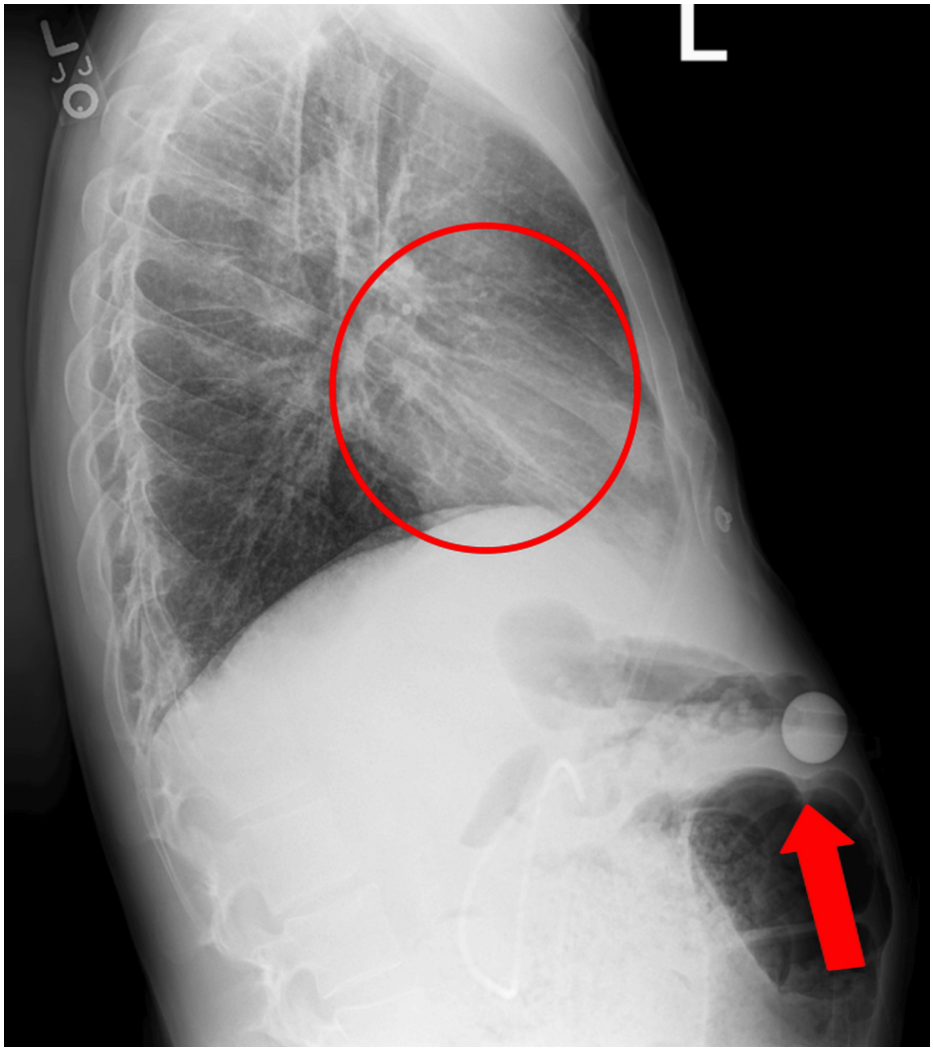


FIGURE 2: Chest X-ray (lateral view) showing mild reticulonodular opacities (circle).

Gastrojejunostomy tubing is partially visualized (arrow).

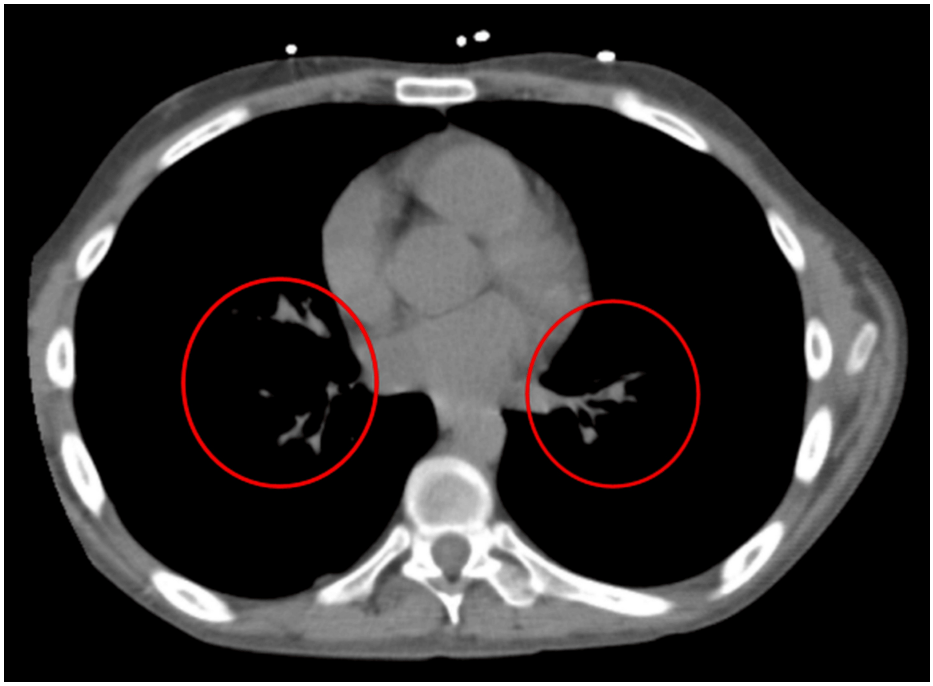


FIGURE 3: Chest CT (axial view) at the level of the aortic root and left atrium.

Consolidations are visible in the lung parenchyma bilaterally (circles).

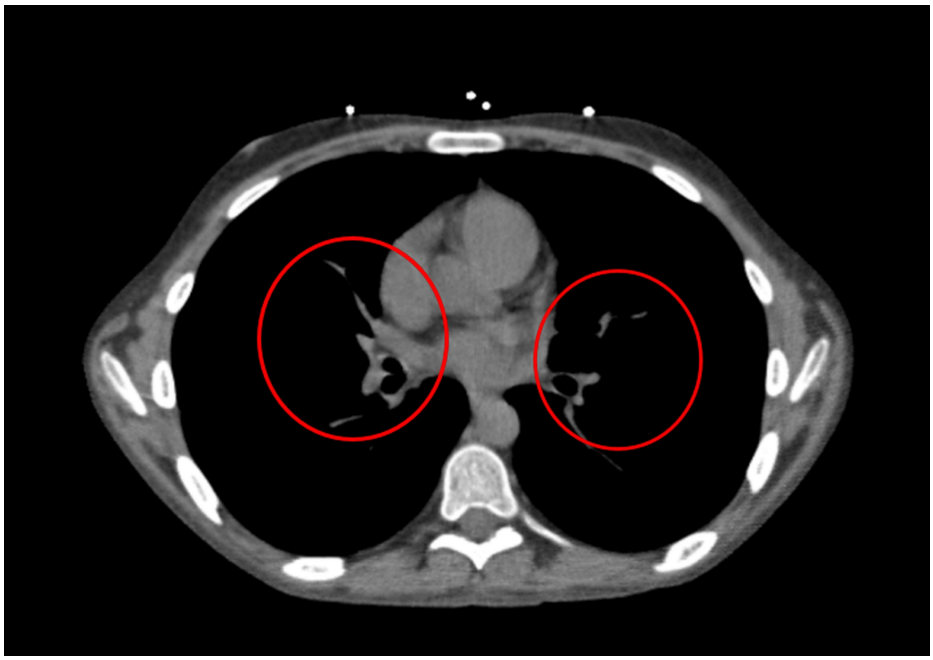


FIGURE 4: Chest CT (axial view) at the level of the pulmonary trunk.

Scattered bronchial wall thickening and bronchiectasis within the right upper lobe (circles) is seen.

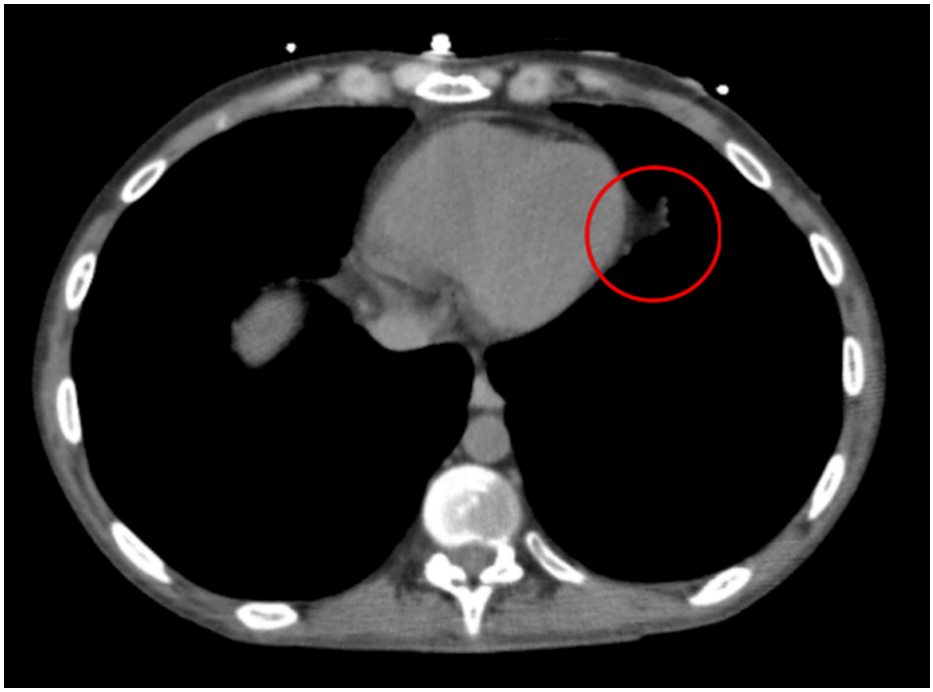


FIGURE 5: Chest CT (axial view) shows a small quantity of peribronchial consolidation within the lingula (circle).

The patient was admitted and treated for pulmonary exacerbation, under the care of the hospitalist, with consults from pulmonology/critical care medicine for maintenance therapy of his respiratory exacerbation and infectious disease consultation for the systemic bacteremia, for which he was treated with ipratropium/albuterol nebulized inhalers. The patient was also placed on enoxaparin 40 mg subcutaneously daily as part of hospital protocol for deep vein thrombosis (DVT) prophylaxis by the hospitalist.

Prior to the infectious disease consult, the patient was initially treated with piperacillin/tazobactam 3.375 g intravenously in the emergency room due to suspicion of community-acquired pneumonia. Upon infectious disease consultation, he was switched to vancomycin 1 g intravenously every 12 hours and cefepime 1 g intravenously every eight hours. Ultimately, the IV cefepime was discontinued due to intermediate susceptibility, as shown by the minimum inhibitory concentration (MIC) of 16 µg/ml, as seen in Table *3*, and the IV vancomycin was discontinued due to lack of Gram-positive organisms on the blood culture. The patient was subsequently started on meropenem 1 g intravenously every eight hours, minocycline 100 mg orally every 12 hours, and amikacin 500 mg intravenously once on day 3 and again on day 6 of admission, which were added following aerobic blood cultures that were positive for *P. aeruginosa*. Furthermore, the patient showed an elevated procalcitonin level of 21.54 ng/mL, as shown in Table *2*. According to the interpretation guidelines of the Association of Diagnostics and Laboratory Medicine 2023 Guidelines [8], this indicated systemic bacterial infection, further confirming the bacteremia. The course of meropenem was completed, and the patient was switched to oral ciprofloxacin 750 mg every 12 hours for 14 days upon discharge. Final laboratory values and vital signs (Tables *1* and 2) showed clinical stabilization.

Timepoint	Culture result	Susceptibility	Antibiotic tested	MIC (µg/mL)	Antibiotics in use
Admission (pre-treatment)	POSITIVE: *Pseudomonas aeruginosa*	Intermediate	Cefepime	16	Piperacillin/tazobactam, Vancomycin, Cefepime
		Susceptible	Amikacin	≤16	
		Susceptible	Ceftazidime	8	
		Susceptible	Ciprofloxacin	0.5	
		Susceptible	Imipenem	≤1	
		Susceptible	Levofloxacin	2	
		Susceptible	Meropenem	≤1	
		Susceptible	Piperacillin/tazobactam	16	
		Susceptible	Tobramycin	≤2	
Breakthrough	POSITIVE: *Pseudomonas aeruginosa*	Intermediate	Cefepime	16	Meropenem, Vancomycin, Minocycline, Amikacin
		Susceptible	Amikacin	<16	
		Susceptible	Ceftazidime	8	
		Susceptible	Ciprofloxacin	0.5	
		Susceptible	Imipenem	≤1	
		Susceptible	Levofloxacin	2	
		Susceptible	Meropenem	≤1	
		Susceptible	Piperacillin/tazobactam	16	
		Susceptible	Tobramycin	≤2	
Discharge planning	NO GROWTH	After 5 days	—	—	Ciprofloxacin 750 mg PO, q12h × 14 days

TABLE 3: Blood culture results, susceptibility profile, and antimicrobial therapy across the hospital course

PO, by mouth; q12h, every 12 hours; MIC, minimum inhibitory concentration

On day 5 of treatment, worsening thrombocytopenia was noted as the platelet count dropped from 341,000/mm³ to 86,000/mm³ (Table *2*). Thus, hematology/oncology were consulted, and the patient was consequently diagnosed with suspected heparin-induced thrombocytopenia (HIT). An intermediate 4T score for HIT, a PF4 antibody assay, serotonin-release assay, and peripheral smear were sent for further evaluation. The patient was started on anticoagulation with fondaparinux 5 mg subcutaneous injection every 24 hours. The patient's platelet count decreased substantially and was found to be 165,000/mm³ on the discharge date (Table *2*). The patient was discharged on apixaban 5 mg orally twice daily, which is to be continued for three to six months, with follow-up with his hematologist.

The patient was discharged in stable condition and indicated that he plans to return to his primary medical team, including his primary care physician and pulmonologist. Figure *6* shows a complete chronological timeline summarizing the patient's hospital course. This includes all relevant vitals, laboratory data, physiological changes, and changes in antimicrobial therapy.

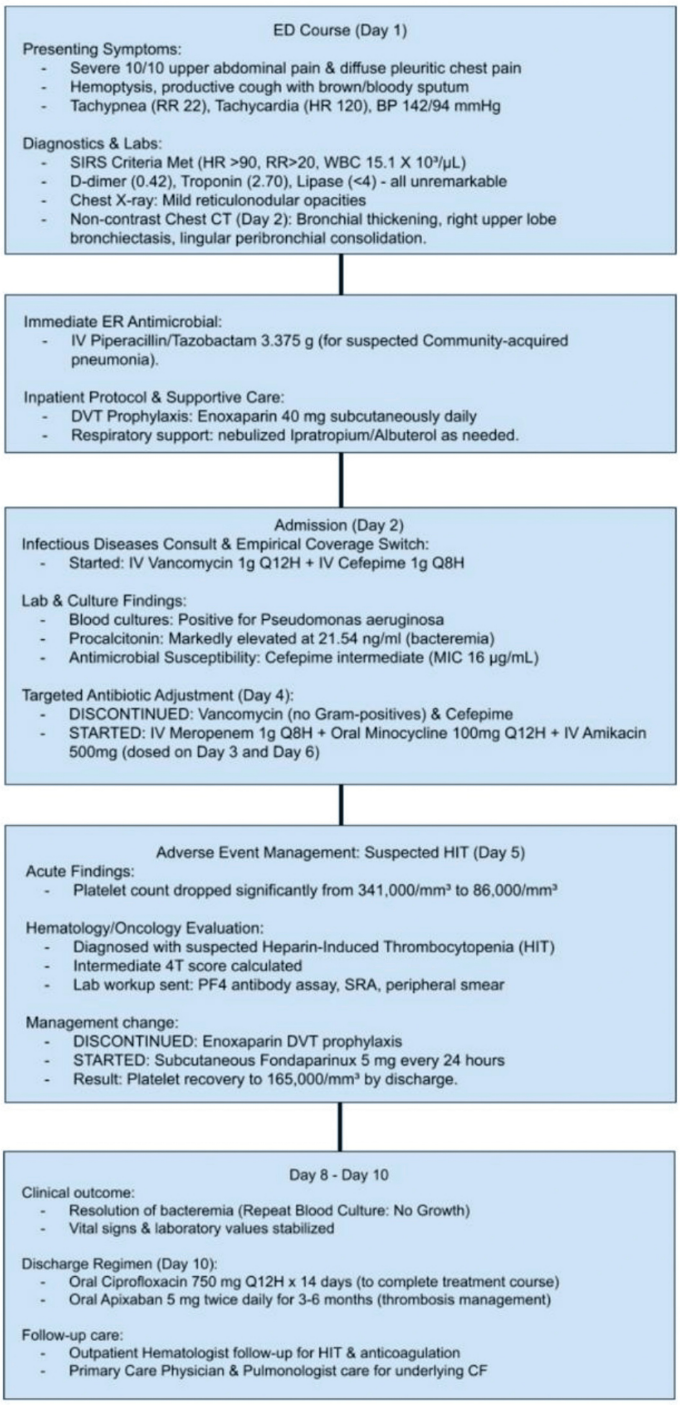


FIGURE 6: Clinical course and management timeline from hospitalization day 1 to day 10

RR, respiration rate; HR, heart rate; BP, blood pressure; SIRS, systemic inflammatory response syndrome; DVT, deep vein thrombosis; Q12H, *quaque 12 hora* (every 12 hours); Q8H, *quaque 8 hora* (every 8 hours); HIt, heparin-induced thrombocytopenia; SRA, serotonin-release assay; CF, cystic fibrosis

Discussion

CF is a multisystem disorder characterized by chronic airway infection and progressive pulmonary disease, arising from *CFTR* gene mutations that impair chloride channel function, deplete airway surface liquid, and disrupt mucociliary clearance. This, in turn, leads to significant mucus accumulation because of decreased mucociliary clearance, which can manifest as persistent airway inflammation of the bronchioles and bronchi, and structural lung damage primarily by targeting the airway epithelial lining, bronchial walls, and surrounding extracellular matrix [9]. The overall result of this is recurrent lower respiratory infections and

pulmonary exacerbations and eventually progressive loss of lung function [10]. The microbiology of CF pulmonary disease has proved to evolve over the course of the disease and increase in severity and complexity. Common respiratory pathogens include *S. aureus* and *H. influenzae*, while *P. aeruginosa* has predominantly been the leading source of infection in adults with CF [11,12]. *P. aeruginosa* can adapt to and persist in the airway, allowing persistent infection despite aggressive antibiotic therapy. Other notable organisms include, but are not limited to, *S. maltophilia* and nontuberculous mycobacteria [13,14].

Although *P. aeruginosa* is a well-established respiratory pathogen in patients with CF, a bloodstream infection is an uncommon complication. In a prospective observational study of patients with CF experiencing febrile pulmonary exacerbations, positive blood cultures were identified in only a small proportion of exacerbations, thus supporting the relative rareness of bacteremia in this patient population [15]. The same study also suggests that pathogens causing bacteremia likely disseminate from the airways or from central venous catheters, if present in the patient, and enter the bloodstream [15,16].

This case is notable not only because of the occurrence of *P. aeruginosa* bacteremia, but also because bacteremia persisted despite initial antibiotic therapy that is known to be effective against *P. aeruginosa*. Initial CT imaging demonstrated bronchial wall thickening with bronchiectasis within the right upper lobe, in addition to a small quantity of peribronchial consolidation seen within the lingula, and diffuse centrilobular nodules. Given the established structural lung disease in this patient, the identification of *P. aeruginosa* in the blood raised concern for an invasive infection associated with the pulmonary process. The persistence of bacteremia, despite initial therapy, further complicated management and required reassessment of antimicrobial coverage.

Management of *P. aeruginosa* infection is complicated and requires consideration of both antimicrobial susceptibility and the clinical severity of the infection [4]. In this case, as shown in Table *3*, the patient was initially treated with intravenous piperacillin-tazobactam 3.375 g every six hours for a presumed pulmonary exacerbation, though the patient was subsequently transitioned to IV cefepime 1 g every eight hours and IV vancomycin 1 g every 12 hours. Despite antipseudomonal coverage, repeat blood cultures on day 4 remained positive for *P. aeruginosa*. Notably, the isolate demonstrated intermediate susceptibility to cefepime with a MIC of 16 µg/mL, while remaining susceptible to several other antipseudomonal agents. Given the persistently positive blood cultures and intermediate susceptibility to cefepime, cefepime was discontinued in favor of a more aggressive antipseudomonal regimen consisting of meropenem and amikacin. Minocycline was also added to provide additional coverage given the patient's history of MRSA infection. Vancomycin was subsequently discontinued once it was found that gram-positive coverage was no longer indicated. Following clinical improvement and repeated blood cultures indicating no growth, the patient was transitioned to a two-week, twice daily oral ciprofloxacin 750 mg antibiotic course.

The persistence of positive blood cultures in the setting of intermediate cefepime susceptibility emphasizes the challenge of selecting effective antimicrobial coverage for *P. aeruginosa* in patients with CF. The ability of *P. aeruginosa* to adapt within the airways of patients with CF can result in antimicrobial resistance and persistence of infection despite treatment [13,14]. This case illustrates the importance of interpreting antimicrobial susceptibility results alongside the patient’s clinical course and response to antimicrobial regimen. In particular, persistence of bacteremia, despite adequate antimicrobial coverage, should prompt reassessment of treatment efficacy, with the support of susceptibility testing and clinical judgment to decide on alternative agents.

This case highlights bacteremia, specifically from *P. aeruginosa*, as an uncommon but serious complication in patients with CF. In CF patients, the presence of febrile pulmonary exacerbations or other signs of systemic infection should be evaluated for bloodstream infection and adjustments of antimicrobial therapy as needed. The persistence of said bacteremia despite antimicrobial therapy warrants the importance of measuring susceptibility of certain medications against obtained isolates, in conjunction with the patient response to treatment.

Conclusions

This case highlights the diagnostic and therapeutic challenges associated with P. aeruginosa bacteremia in an adult patient with CF and multiple comorbidities. In patients with CF, pulmonary exacerbations and recurrent infections are major contributing factors that may lead to respiratory compromise, particularly when clinical signs of systemic failure are not promptly detected. Careful monitoring for poor prognostic indicators, timely reassessment of antimicrobial therapy, and adjustment of treatment when clinically indicated are important. Culture-directed antimicrobial therapy and susceptibility testing facilitate appropriate treatment and may help limit the progression of infection while preserving pulmonary function. The patient's initial lack of response to empiric antimicrobial therapy consisting of IV piperacillin-tazobactam, vancomycin, and cefepime emphasizes the importance of maintaining clinical suspicion for pathogenic organisms that may not be immediately considered. Continued diagnostic reassessment and positive identification of pathogenic organisms with their antibiotic susceptibility ultimately led to the finalized treatment modalities.

Although this case presents an individualized antibiotic treatment regimen for a patient with a *P. aeruginosa*

systemic infection with multiple inducing risk factors, it is important to individualize treatment plans based on antibiotic sensitivities and readily available treatment options. Early recognition and detection of clinical deterioration, as well as prompt microbiologic evaluation and adjustments in antimicrobial therapy on an individual case-by-case basis, are essential to optimizing clinical management and reducing the potential for severe long- and short-term complications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Jincy A. Abraham, Malak S. Hamed, Anoushka Mukherjee, Tyson Tetoff, Carlos H. Zambrano

Supervision: Carlos H. Zambrano

Disclosures

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