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CASE REPORT

Community-Acquired Severe Pseudomonas Aeruginosa pneumonia: A Case Report and Review of the Literature

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Abstract: *Pseudomonas aeruginosa* (*P. aeruginosa*), traditionally regarded as a nosocomial pathogen, has emerged as an increasingly recognized etiologic agent in community-acquired pneumonia (CAP). Cavitary lung lesions—a severe complication of CAP characterized by parenchymal necrosis and cavity formation—are exceptionally rare in *P. aeruginosa*-associated CAP. We report a 64-year-old male with CAP complicated by *P. aeruginosa* infection (*OXA*-positive genotype carrying the virulence factors *exoU* and *lasA*), which rapidly progressed to cavitary lesions in the right upper lobe. This case highlights the aggressive clinical trajectory and antimicrobial resistance challenges inherent to community-acquired *P. aeruginosa* infections. Furthermore, it underscores the imperative for serial radiographic monitoring to detect cavitary evolution and the critical role of comprehensive antimicrobial susceptibility testing in guiding precision therapy.

Keywords: community-acquired pneumonia, pseudomonas aeruginosa, cavitary lung lesions, multidrug resistance, personalized treatment

Introduction

The emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) PA strains due to inappropriate antimicrobial use poses significant therapeutic challenges in clinical practice.[1] Notably, community-acquired Pseudomonas aeruginosa pneumonia (PA-CAP) demonstrates rapid progression and high mortality rates.[2] This article synthesizes current domestic and international research advancements, focusing on three critical aspects: the acquired resistance mechanisms of *P. aeruginosa*, characteristic imaging findings of PA-CAP, and contemporary pathogen detection methodologies. Timely diagnosis and targeted therapeutic interventions for this severe respiratory infection are systematically discussed based on these key considerations.

Case Report

A 64-year-old male with a decade-long history of chronic obstructive pulmonary disease (COPD), non-compliant with inhaled ICS-LABA therapy (mMRC score 0), presented to our fever clinic on September 5, 2024, with a 5-day history of productive cough, yellowish sputum, and acute-onset high-grade fever (peak 39.0°C). This systemic inflammatory response manifested as progressive dyspnea, chest tightness, and severely diminished activity tolerance, prompting urgent clinical evaluation based on the CURB-65 criteria (Confusion, Urea >7 mmol/L, Respiratory rate ≥30 breaths/min, Blood pressure <90/60 mmHg, and 65 years of age) proposed by the American Thoracic Society, with a total score of 2 indicating intermediate risk (30-day mortality rate 9.2%), necessitating short-term hospitalization or closely monitored outpatient care. Notably, the patient denied comorbidities (hypertension, diabetes, coronary artery disease) or drug

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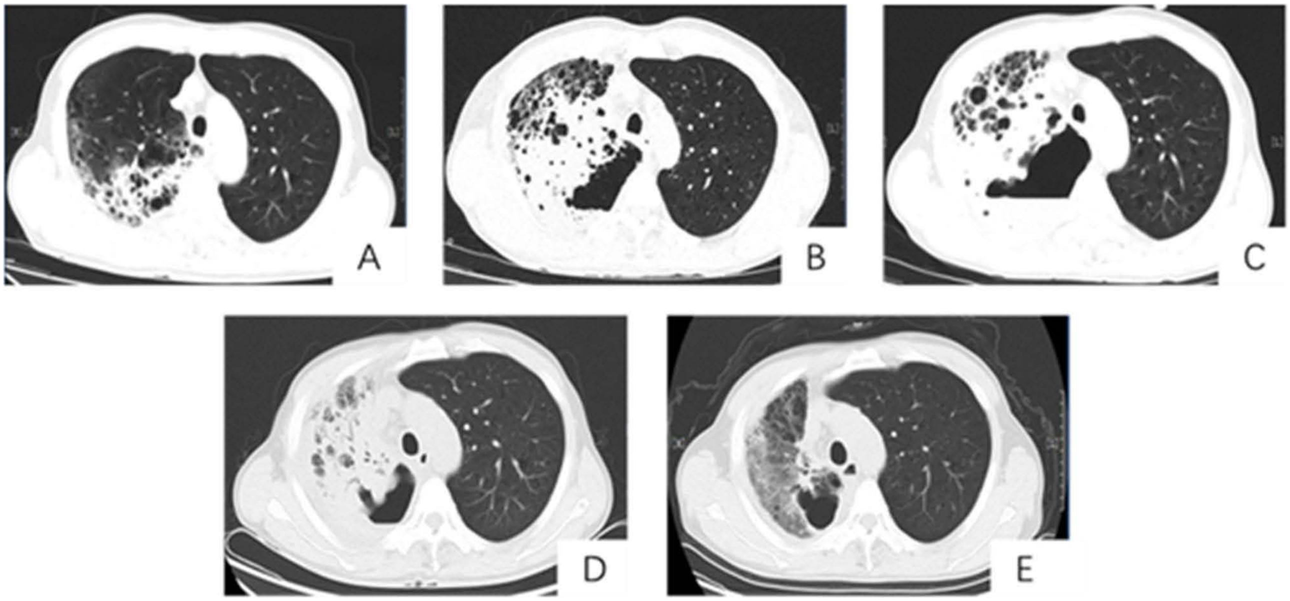

Figure 1 Imaging changes at various time intervals. (**A**) September 5th chest CT demonstrates consolidative infiltrates in the right upper and lower lobes. (**B**) September 9th follow-up CT reveals a thick-walled cavity in the right lung, demonstrating significant progression of the infectious consolidation compared to September 5th. (**C**) September 17th CT identifies a cavitary lesion with an air-fluid level in the right lung, suggesting a developing lung abscess, accompanied by a small right-sided pleural effusion. (**D**) October 1st imaging shows persistent right upper lobe cavitation with surrounding inflammatory changes and partial parenchymal destruction, alongside emphysematous changes with bullous formation in the left lung. (**E**) The November 30th chest CT scan demonstrates residual cavitary remodeling in the right lung, with significant resolution of surrounding inflammatory infiltrates and pleural effusion compared to the October 1st imaging.

allergies. The day's chest computerized tomography (CT) revealed infectious lesions in the upper and lower lobes of the right lung (Figure 1A), and the patient was admitted for further diagnosis and treatment.

The patient maintained hemodynamic compensation (blood pressure 104/74 mmHg) but demonstrated severe hypoxemia (SpO_2 84% on nasal oxygen at 2 L/min), marked sinus tachycardia (heart rate 129 bpm), and tachypnea (respiratory rate 30 breaths/min) despite being afebrile (37.2°C). Neurological assessment revealed alert consciousness with restlessness and agitation. Pulmonary auscultation identified coarse breath sounds with diffuse inspiratory crackles bilaterally, cardiovascular examination confirmed sinus rhythm without murmurs or pericardial friction rubs. Abdominal evaluation showed a soft, non-tender abdomen with no hepatosplenomegaly. Extremities exhibited no signs of fluid retention (absence of pitting edema).

Initial hematological tests demonstrated leukocytosis (18.95×10^9/L) with neutrophilia (17.87×10^9/L), lymphopenia (0.5×10^9/L), and markedly elevated inflammatory markers (C-reactive protein: 213.8 mg/L; procalcitonin: 41.3 ng/mL) (Table 1). Arterial blood gas analysis revealed compensated respiratory alkalosis (pH 7.45, PaO_2 74.7 mmHg, $PaCO_2$

Table 1 Changes in Inflammatory Indicators at Different Time Intervals

	WBC ($\times10^9$/L)	N ($\times10^9$/L)	L ($\times10^9$/L)	CRP (mg/L)	IL-6 (pg/L)	PCT (ng/mL)
2024/09/05	18.95	17.89	0.50	309.15		41.30
2024/09/07	11.20	10.08	0.50	212.31		
2024/09/08	15.51		0.60	167.28	497.37	23.21
2024/09/10		18.75	0.50	203.67	53.63	
2024/09/13	22.59	20.28	1.51	84.48	149.75	
2024/09/16	21.22	18.80	1.27	140.93		
2024/09/21	15.15	12.31	1.38	78.31	45.38	
2024/09/26	15.96	10.76	3.54	39.88	23.51	
2024/10/01	11.99	7.09	3.68	17.07		
2024/10/07	11.15	6.29	3.83	11.10		

Abbreviations: RBC, Red Blood Cells; N, Neutrophil; L, Lymphocyte; CRP, C-Reactive Protein; PCT, Procalcitonin.

26.5 mmHg) superimposed by lactic acidosis (lactate: 4.0 mmol/L; base excess: −5.7 mmol/L). Metabolic derangements included systemic inflammation-induced hypoalbuminemia (29.00 g/L), hyponatremia (130.32 mmol/L), and hypochloremia (94.52 mmol/L). Renal function showed mild impairment (creatinine: 96.00 μmol/L; blood urea nitrogen: 9.55 mmol/L). Cardiac evaluation identified elevated NT-proBNP (2,916.30 pg/mL) and supraventricular tachycardia on electrocardiogram. The patient was diagnosed with COPD exacerbation complicated by severe community-acquired pneumonia (CAP) and type I respiratory failure. Laboratory findings supported bacterial sepsis (procalcitonin >20 ng/mL) with secondary multi-organ dysfunction. Hypoxemia-driven atrial arrhythmia and inflammation-mediated hypoalbuminemia were established as critical complications requiring intensive monitoring.

The patient received initial antimicrobial therapy with ceftazidime/avibactam (3.0 g every 8 hours) alongside bronchodilators and mucolytics. Sputum culture on September 8 identified *P. aeruginosa* susceptible to ceftazidime/avibactam, imipenem, cefotaxime/avibactam, ciprofloxacin, and gentamicin, but resistant to ampicillin, ampicillin/sulbactam, cefotetan, ceftriaxone, cefazolin, meropenem, and cotrimoxazole. Fungal and blood cultures remained negative. Despite therapy, clinical deterioration ensued, characterized by persistent fever (peak 39.0°C), worsening dyspnea, hemoptysis, and hemodynamic instability (blood pressure 88/57 mmHg; respiratory rate 31 breaths/min). Oxygenation indices declined to PaO_2/FiO_2 137.5 mmHg on 6 L/min mask oxygen. Repeat hematology on September 8 demonstrated leukocytosis (15.51×10^9/L) with neutrophilia (14.29×10^9/L), lymphopenia (0.6×10^9/L), and elevated inflammatory markers (CRP 213.8 mg/L; PCT 23.31 ng/mL; IL-6497.37 pg/mL) (Table 1). Arterial blood gas revealed compensated respiratory alkalosis (pH 7.47, PaO_2 60.5 mmHg, $PaCO_2$ 27.7 mmHg) with residual lactic acidosis (lactate 2.10 mmol/L; BE −5.7 mmol/L). September 9 chest CT revealed progressive cavitary lesions in the right lung (Figure 1B), prompting antimicrobial escalation to meropenem 1.0 g every 8 hours (for carbapenem coverage) and adjunctive esmolol 0.2 g every 8 hours (for tachycardia control). Bronchoscopic lavage metagenomic next-generation sequencing (mNGS) confirmed *P. aeruginosa* infection (52,090 sequences) harboring *OXA* resistance genes and virulence factors *ExoU/LasA*. Persistent fever (38.3°C) and emerging ertapenem/imipenem resistance necessitated regimen adjustment to cefoperazone/sulbactam 3.0 g every 8 hours plus azithromycin 0.5 g daily on September 14. Subsequent clinical improvement was evidenced by normalized temperature, resolving dyspnea, and declining inflammatory markers (September 17: WBC 15.96×10^9/L; CRP 39.88 mg/L) (Table 1).

Serial chest CT imaging demonstrated progression from consolidative lesions (September 5, Figure 1A) to cavitary lesions (September 9, Figure 1B), progressing to abscess formation (September 17, Figure 1C) and subsequent cavitary changes with inflammation and partial destruction of the right upper lobe (October 2, Figure 1D; 2-month follow-up, Figure 1E). Disease control was achieved with intravenous cefoperazone/sulbactam combined with ciprofloxacin and aminoglycoside nebulization. The inflammatory indicators have shown a significant decrease compared to baseline levels at admission, indicating effective control of the infection (Table 1). The patient was discharged on October 8 and maintained on oral ciprofloxacin (0.1 g once daily, qd) and tiotropium inhalation therapy.

Discussion

Epidemiological studies demonstrate that *P. aeruginosa* exhibits low isolation rates in CAP, ranging from 0.9% to 1.9% in the United States and 1.0% in Chinese populations. However, its prevalence rises markedly in severe CAP requiring intensive care unit (ICU) admission, reaching 1.8%–8.3%.[3] Notably, high-risk patients with prior *P. aeruginosa* infection/colonization and at least one chronic pulmonary comorbidity (post-tracheostomy status, bronchiectasis, or Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage IV COPD) show a dramatic increase in *P. aeruginosa* -associated CAP incidence (67%).[4] Radiologically, PA-CAP typically presents with single or multiple thick-walled cavities accompanied by air-fluid levels, irregular walls, and peri-cavitary inflammatory infiltration.[5] In this case, rapid progression of cavitary lesions in the right upper lobe (RUL) on CT imaging aligned with *P. aeruginosa's* aggressive pathological profile. Previous studies report RUL involvement in two-thirds of *P. aeruginosa* infection cases,[6] which may be attributed to the lobe's elevated ventilation-perfusion ratio and oxygen partial pressure, creating a favorable micro-environment for *P. aeruginosa* proliferation. Furthermore, impaired lymphatic drainage in upper pulmonary regions promotes the accumulation of inflammatory mediators and necrotic debris, accelerating cavitation.[7]

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Cavitation may result from diverse pathological mechanisms, including suppurative necrosis, ischemic necrosis, caseous necrosis (eg, tuberculosis), or coagulative necrosis. Compared to other pathogens, *Mycobacterium tuberculosis, Klebsiella pneumoniae*, and *Staphylococcus aureus* demonstrate higher propensity for cavitation.[8] Distinct radiological patterns emerge among these pathogens: *Tuberculous cavities* predominantly involve apical/posterior upper lobe segments and lower lobe superior segments, presenting as thick-walled cavities within consolidations, which correlate with heightened transmissibility.[9]

Staphylococcal pneumonia typically manifests as thin-walled cavities with frequent abscess formation.[10] *Klebsiella pneumonia* exhibits lobar consolidations containing multiple small liquefied foci ("bubbly lucencies"), accompanied by downward displacement of interlobar fissures and peripheral ground-glass opacities. Characteristic features include small hypodense areas or microcavities within lesions.[11] Serial CT imaging in this case demonstrated rapidly expanding cavitary lesions with air-fluid level formation. Coupled with positive *P. aeruginosa* cultures from bronchoalveolar lavage fluid, these findings corroborate the pathogen-specific diagnosis of *P. aeruginosa* -associated pulmonary infection.

P. aeruginosa a critical nosocomial pathogen exhibits intrinsic resistance to multiple antimicrobial agents. Its MDR arises from synergistic mechanisms including: (1) production of inactivating enzymes (eg, AmpC β-lactamases and metallo-β-lactamases), (2) reduced outer membrane permeability (eg, porin deficiency), (3) overexpression of efflux pumps, (4) target site modifications (eg, PBP mutations), and (5) biofilm formation.[12] Globally, Multidrug-Resistant *P. aeruginosa* accounts for 23.7–44.6% of clinical isolates.[13] Genetic Determinants in This Case: The isolate harbored the *OXA* carbapenemase gene and virulence factor*s exoU/lasA*. *exoU*, a phospholipase cytotoxin, enhances *P. aeruginosa* pathogenicity by accelerating host cell necrosis and indirectly promoting resistance through co-expression of resistance genes and disruption of antibiotic penetration.[14] *lasA* facilitates resistance via biofilm potentiation and quorum sensing (QS)-mediated regulation of efflux pumps and membrane proteins.[15] Therapeutic Implications: The *OXA* genotype aligns with Chinese *P. aeruginosa* resistance epidemiology, conferring resistance to imipenem, meropenem, and ceftazidime.[16] Guided by bronchoalveolar lavage (BAL) and serial antimicrobial susceptibility testing (AST), the therapy was escalated to ceftazidime-avibactam. Subsequent CT imaging demonstrated radiographic improvement, paralleled by defervescence and significant alleviation of dyspnea and systemic symptoms, confirming therapeutic efficacy.

Discrepancies may arise between conventional microbiological assays and metagenomic next-generation sequencing (mNGS) in antimicrobial resistance (AMR) gene detection. The pan-pathogen screening capacity of mNGS introduces susceptibility to background microbial interference, while incomplete reference databases may compromise accurate resistance gene classification.[17] Critical limitations of mNGS-based AMR profiling include. False positives from non-functional resistance genes (eg, silent or truncated sequences) False negatives due to insufficient sequencing depth (<50× coverage). Genotype–phenotype discordance caused by low-level gene expression or non-genetic resistance mechanisms. Ambiguous gene origin (pathogen vs colonizer vs environmental contaminant).[18] Thus, phenotypic confirmation through antimicrobial susceptibility testing (AST) or targeted validation (eg, PCR/Sanger sequencing) remains imperative to verify functional resistance. The integration of conventional diagnostics and mNGS proves pivotal in resolving diagnostic impasses for severe/complex infections. We propose a dual-pathogenomics strategy focusing on: Systematic identification of Pseudomonas aeruginosa virulence determinants driving cavitary pneumonia progression. Mechanistic dissection of molecular pathways underlying rapid clinical deterioration in severe infections Tailored antimicrobial regimens based on validated genotype–phenotype correlations.

Conclusion

High-risk factors for P. aeruginosa necrotizing pneumonia include underlying structural lung disease, immunodeficiency, and environmental exposure.[19] In recent years, the incidence of PA-CAP has been increasing in China (accounting for 11.3% of all CAP cases[20]). Given this trend, clinicians should maintain a high index of suspicion for PA-CAP in patients presenting with upper-lung cavitary lesions. Key clinical management strategies include: prompt risk assessment, inclusion in the differential diagnosis, dynamic monitoring of lesion progression through serial imaging, and immediate initiation of antimicrobial therapy guided by antimicrobial susceptibility testing (AST). This strategy, which combines early radiographic surveillance with targeted treatment, is critical for improving patient prognosis and reducing mortality.

Ethical Statement

The manuscript has been approved by the institution (The Third Affiliated Hospital of Anhui Medical University) and can be published. The approval code is 2025-052.

Informed Consent

The patient's signed informed consent explicitly authorizes the publication of case specifics and related imaging data.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Breidenstein EBM, De La Fuente-Núñez C, Hancock REW. Pseudomonas aeruginosa: all roads lead to resistance. *Trends Microbiol*. 2011;19 (8):419–426. doi:10.1016/j.tim.2011.04.005
2. Cotran-Lenrow A, Tefera LS, Douglas-Vail M, Ayebare A, Kpokpah LN, Davis BP. Community-acquired pseudomonas aeruginosa meningitis in a pediatric patient. *Cureus*. 2023. doi:10.7759/cureus.42376
3. Chinese Thoracic Society, Infection Group of Respiratory Branch, Chinese Medical Association. Expert consensus on diagnosis and treatment of Pseudomonas aeruginosa lower respiratory tract infections in China (2022 update). *Zhonghua Jie He He Hu Xi Za Zhi*. 2022;45(8):739–752. doi:10.3760/cma.j.cn112147-20220407-00290
4. Pelegrin AC, Palmieri M, Mirande C, et al. *Pseudomonas aeruginosa*: a clinical and genomics update. *FEMS Microbiol Rev*. 2021;45(6):fuab026. doi:10.1093/femsre/fuab026
5. Riviere P, Patin D, Delaporte E, et al. Septic shock secondary to an acute necrotizing community-acquired pneumonia with bacteremia due to Pseudomonas aeruginosa. *IDCases*. 2019:17:e00563. doi:10.1016/j.idcr.2019.e00563
6. Hatchette TF, Gupta R, Marrie TJ. Pseudomonas aeruginosa community-acquired pneumonia in previously healthy adults: case report and review of the literature. *Clin Infect Dis*. 2000;31(6). doi:10.1086/317486
7. Nemec SF, Bankier AA, Eisenberg RL. Upper lobe-predominant diseases of the lung. *AJR Am J Roentgenol*. 2013;200(3). doi:10.2214/AJR.12.8961
8. Canan A, Batra K, Saboo SS, Landay M, Kandathil A. Radiological approach to cavitary lung lesions. *Postgrad Med J*. 2021;97(1150):521–531. doi:10.1136/postgradmedj-2020-138694
9. Lovey A, Verma S, Kaipilyawar V, et al. Early alveolar macrophage response and IL-1R-dependent T cell priming determine transmissibility of mycobacterium tuberculosis strains. *Nat Commun*. 2022;13(1):884. doi:10.1038/s41467-022-28506-2
10. Ahmad-Mansour N, Loubet P, Pouget C, et al. Staphylococcus aureus toxins: an update on their pathogenic properties and potential treatments. *Toxins*. 2021;13(10). doi:10.3390/toxins13100677
11. Fernández Vecilla D, Unzaga Barañano MJ, García De Andoin Sojo C, Díaz De Tuesta Del Arco JL. cavitary pneumonia and sepsis caused by ST23 hypervirulent klebsiella pneumoniae. *Enferm Infecc Microbiol Clin*. 2023;41(2):129–131. doi:10.1016/j.eimce.2022.11.017
12. Altan G, Rakici E, Özgümüş OB. Molecular analysis of antibiotic resistance in carbapenemresistant pseudomonas aeruginosa isolates isolated from clinical samples. *Mikrobiyol Bul*. 2024;58(2):148–170. doi:10.5578/mb.202498200
13. Castanheira M, Kimbrough JH, Lindley J, Doyle TB, Ewald JM, Sader HS. *In vitro* development of resistance against antipseudomonal agents: comparison of novel β-lactam/β-lactamase inhibitor combinations and other β-lactam agents. *Antimicrob Agents Chemother*. 2024;68(5):e01363–23. doi:10.1128/aac.01363-23
14. Foulkes DM, McLean K, Haneef AS, et al. Pseudomonas aeruginosa toxin ExoU as a therapeutic target in the treatment of bacterial infections. *Microorganisms*. 2019;7(12). doi:10.3390/microorganisms7120707
15. Duan X, Boo ZZ, Chua SL, et al. A bacterial quorum sensing regulated protease inhibits host immune responses by cleaving death domains of innate immune adaptors. *Adv Sci*. 2023;10. 10.1002/advs.202304891
16. Han MJ, Tang JH, Pu JK, et al. Correlation of T3SS virulence factor carriage, expression, and pathogenicity with drug resistance and molecular epidemiology of carbapenem-resistant *Pseudomonas aeruginosa* in a hospital. *Zhongguo Yi Yuan Yao Xue Za Zhi*. 2025;45(4):402–409. doi:10.13286/j.1001-5213.2025.04.06
17. Jenks JD, White PL, Kidd SE, et al. An update on current and novel molecular diagnostics for the diagnosis of invasive fungal infections. *Expert Rev Mol Diagn*. 2023;23(12):1135–1152. doi:10.1080/14737159.2023.2267977
18. Liang WY, Shi Y, Sun WK. Research progress on metagenomic next-generation sequencing in detecting drug-resistant phenotypes of gram-negative bacteria. *Zhonghua Jie He He Hu Xi Za Zhi*. 2022;45(2):209–213. doi:10.3760/cma.j.cn112147-20210705-00468
19. Kunimasa K, Ishida T, Kimura S, et al. Successful treatment of fulminant community-acquired pseudomonas aeruginosa necrotizing pneumonia in a previously healthy young man. *Intern Med*. 2012;51(17):2473–2478. doi:10.2169/internalmedicine.51.7596
20. Restrepo MI, Babu BL, Reyes LF, et al. Burden and risk factors for Pseudomonas aeruginosa community-acquired pneumonia: a multinational point prevalence study of hospitalised patients. *Eur Respir J*. 2018;52(2):1701190. doi:10.1183/13993003.01190-2017