

Case Report

Hospital-Acquired Pneumonia: A Case Report

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Abstract

Hospital-acquired pneumonia (HAP) is one of the most common and serious healthcare-associated infections, contributing substantially to patient morbidity, mortality, prolonged hospitalization, and increased healthcare expenditures. HAP is defined as pneumonia that develops 48 hours or more after hospital admission and was neither present nor incubating at the time of admission. Surgical patients, particularly older adults and those with underlying comorbid conditions, are at an elevated risk of developing this infection. Prompt diagnosis and initiation of appropriate antimicrobial therapy are essential for improving clinical outcomes and reducing complications. This case report describes a 68-year-old male who developed HAP following elective abdominal surgery. The report outlines the patient's clinical presentation, diagnostic workup, microbiological findings, treatment approach, and preventive measures undertaken during management. The case underscores the importance of antimicrobial stewardship, strict infection prevention and control practices, and a multidisciplinary approach in reducing the burden and adverse outcomes associated with hospital-acquired pneumonia.

Keywords: Hospital-acquired pneumonia (HAP), Nosocomial pneumonia, Postoperative pneumonia, Healthcare-associated infections, *Pseudomonas aeruginosa*, Gram-negative bacterial infections, Antimicrobial stewardship

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Introduction

Hospital-acquired pneumonia (HAP) is among the most frequent and serious healthcare-associated infections, ranking second only to urinary tract infections in hospitalized patients. It is defined as pneumonia that develops 48 hours or more after hospital admission and was neither present nor incubating at the time of admission. HAP is associated with prolonged hospitalization, increased healthcare costs, and significant mortality, particularly among critically ill patients. Mortality rates range from 20% to 50%, especially in individuals requiring intensive care support or mechanical ventilation.

The pathogenesis of HAP involves colonization of the lower respiratory tract by pathogenic microorganisms acquired within the hospital environment. Several factors predispose patients to HAP, including advanced age, chronic medical conditions, immunosuppression, prolonged hospitalization, prior antibiotic exposure, and invasive procedures. Common causative pathogens include Gram-negative organisms such as *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Escherichia coli*, as well as Gram-positive organisms, particularly *Staphylococcus aureus*, including methicillin-resistant strains (MRSA). The increasing prevalence of multidrug-resistant organisms continues to complicate management and adversely affect patient

outcomes.

Prompt diagnosis and timely initiation of appropriate antimicrobial therapy are essential for improving prognosis. This case report describes the development of HAP in a postoperative patient and highlights the clinical presentation, diagnostic evaluation, therapeutic management, and preventive strategies associated with this important nosocomial infection.

Case presentation

Patient information

A 68-year-old man was admitted for an elective colectomy for symptomatic diverticulosis. His medical history was significant for type 2 diabetes mellitus, hypertension, and mild chronic kidney disease. He had no history of smoking, chronic respiratory disease, or previous episodes of pneumonia. Preoperative investigations, including routine laboratory tests and chest radiography, were within normal limits.

Clinical course

The patient underwent an uncomplicated colectomy and demonstrated satisfactory postoperative recovery. By postoperative day 3, he was ambulating independently and tolerating oral intake. However, on postoperative day 5, he developed fever, productive cough, and progressive respiratory distress.

On examination, his temperature was 38.5°C, respiratory rate was 28 breaths per minute, oxygen saturation was 88% on room air, and blood pressure was 95/60 mmHg. Auscultation revealed coarse crackles over the right lower lung zone, accompanied by dullness to percussion. No wheeze or pleural friction rub was present.

Diagnostic evaluation

Laboratory investigations demonstrated leukocytosis with a white blood cell count of 15,500/μL and neutrophil predominance of 82%. Inflammatory markers were elevated, with a C-reactive protein level of 120 mg/L and procalcitonin concentration of 2.5 ng/mL. Serum creatinine increased slightly to 1.4 mg/dL from a baseline value of 1.2 mg/dL. Arterial blood gas analysis revealed hypoxemia with a PaO₂ of 68 mmHg and PaCO₂ of 35 mmHg while breathing room air.

Parameter	Result	Interpretation
Temperature	38.5°C	Fever
Respiratory Rate	28/min	Tachypnea
Oxygen Saturation	88%	Hypoxemia
Blood Pressure	95/60 mmHg	Mild hypotension
WBC Count	15,500/μL	Leukocytosis
Neutrophils	82%	Neutrophilia
CRP	120 mg/L	Elevated
Procalcitonin	2.5 ng/mL	Elevated
Chest X-ray	Right lower lobe consolidation	Pneumonia
CT Chest	Right lower lobe consolidation	Confirmed HAP

Table 1: Clinical, laboratory, and radiological findings.

Chest radiography demonstrated a new right lower lobe consolidation with air bronchograms. Computed tomography of the thorax confirmed right lower lobe consolidation without evidence of pleural effusion or pulmonary abscess [Table 1].

Microbiological evaluation revealed Gram-negative bacilli on sputum Gram stain. Sputum culture subsequently grew *Pseudomonas aeruginosa*, which was susceptible to piperacillin-tazobactam, ceftazidime, and meropenem. Blood cultures remained sterile, and viral respiratory testing by nasopharyngeal swab was negative.

Investigation	Result
Sputum Gram Stain	Gram-negative bacilli
Sputum Culture	<i>Pseudomonas aeruginosa</i>
Blood Culture	No growth
Viral Respiratory Panel	Negative
Piperacillin–Tazobactam	Susceptible
Ceftazidime	Susceptible
Meropenem	Susceptible

Table 2: Microbiological Findings and Antimicrobial Susceptibility.

Based on the clinical findings, radiological evidence, and microbiological confirmation, a diagnosis of hospital-acquired pneumonia was established [Table 2].

Management

Empirical treatment

Considering the patient's postoperative status and risk factors for healthcare-associated pathogens, empirical intravenous piperacillin-tazobactam (4.5 g every 8 hours) was initiated. Supplemental oxygen was administered via nasal cannula to maintain oxygen saturation above 92%.

Additional supportive measures included intravenous fluid therapy, optimization of glycemic control, pulmonary physiotherapy, and close clinical monitoring.

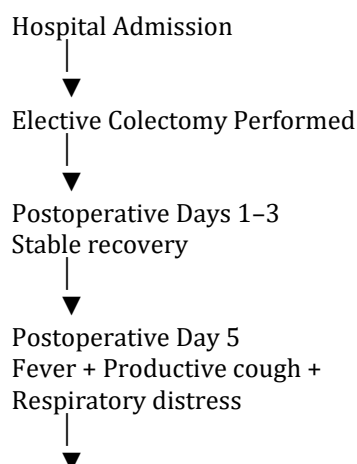
Targeted therapy

Following availability of culture and susceptibility results, piperacillin-tazobactam therapy was continued as definitive treatment because the isolated organism demonstrated full susceptibility. A treatment duration of 10–14 days was planned according to clinical and radiological response.

Supportive care

Comprehensive supportive management included incentive spirometry, pulmonary hygiene measures, and early mobilization to reduce the risk of atelectasis and further pulmonary complications. Standard infection control precautions and strict hand hygiene practices were maintained throughout hospitalization.

Outcome and follow-up



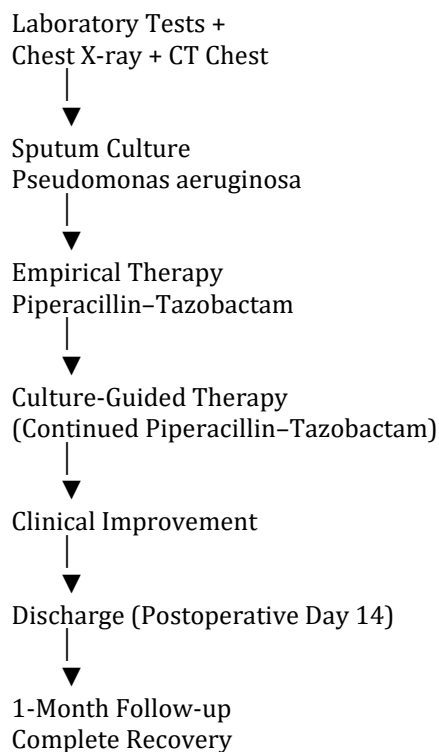


Figure 1: Clinical timeline of hospital-acquired pneumonia.

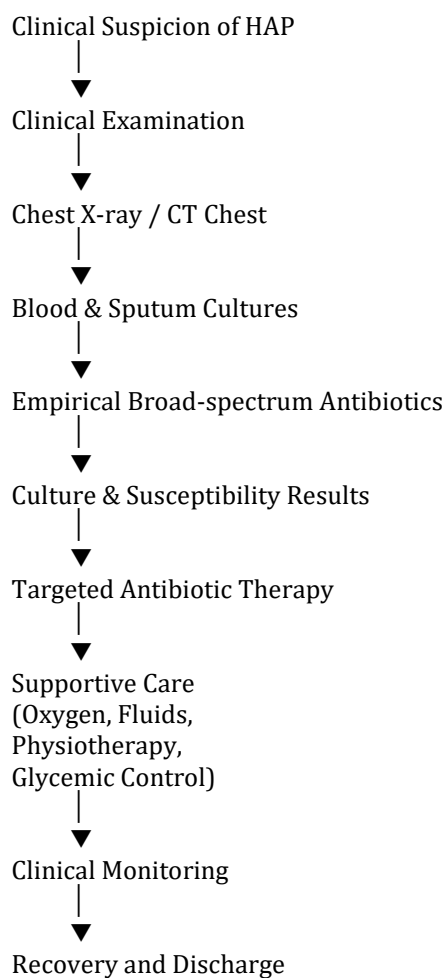


Figure 2: Management algorithm for hospital-acquired pneumonia.

The patient's condition improved steadily over the subsequent five days. Fever resolved, respiratory symptoms diminished, and oxygen requirements progressively decreased. Laboratory parameters also improved, with the white blood cell count declining to 8,500/ μ L, CRP decreasing to 20 mg/L, and procalcitonin falling to 0.5 ng/mL.

A repeat chest radiograph demonstrated near-complete resolution of the right lower lobe infiltrate. The patient was discharged on postoperative day 14 in stable condition with instructions for outpatient follow-up [Figure 1].

At one-month review, he remained asymptomatic and exhibited complete clinical and radiographic recovery without evidence of recurrent infection [Figure 2].

Discussion

Hospital-acquired pneumonia develops when pathogenic microorganisms bypass normal host defense mechanisms and establish infection within the lower respiratory tract. Common mechanisms include aspiration of colonized oropharyngeal secretions, microbial colonization of the upper airway, and direct inoculation through medical devices. In the present case, advanced age, diabetes mellitus, recent abdominal surgery, and hospitalization represented significant predisposing factors.

Pseudomonas aeruginosa is a well-recognized cause of HAP and is particularly concerning because of its intrinsic resistance mechanisms and capacity to acquire additional antimicrobial resistance determinants. Consequently, early microbiological identification and susceptibility testing are essential for optimizing therapy and limiting adverse outcomes.

Diagnosis of HAP requires integration of clinical, laboratory, microbiological, and radiological findings. Typical manifestations include fever, leukocytosis, purulent respiratory secretions, hypoxemia, and new pulmonary infiltrates on imaging. Microbiological confirmation through sputum culture or lower respiratory tract sampling facilitates targeted antimicrobial therapy and supports antimicrobial stewardship efforts.

Effective management involves prompt initiation of empirical broad-spectrum antimicrobial therapy, followed by de-escalation based on culture results. Supportive measures such as oxygen supplementation, fluid management, pulmonary rehabilitation, and glycemic control are equally important. Infection prevention strategies, including hand hygiene, environmental cleaning, and adherence to isolation protocols, remain fundamental components of care.

Prevention of HAP requires a multifaceted approach. Key preventive measures include strict compliance with infection control practices, minimization of invasive devices, early patient mobilization, pulmonary physiotherapy, vaccination of high-risk populations against influenza and pneumococcal disease, and implementation of antimicrobial stewardship programs.

Despite advances in diagnosis and treatment, HAP remains a major clinical challenge due to the growing prevalence of multidrug-resistant organisms, delays in pathogen identification, and limited availability of novel antimicrobial agents. Emerging technologies, including rapid molecular diagnostics and artificial intelligence-based predictive tools, may improve early detection and guide more precise antimicrobial selection in the future.

Conclusion

Hospital-acquired pneumonia remains a significant cause of postoperative morbidity and mortality, particularly among elderly patients with underlying comorbidities. Early recognition, prompt initiation of empirical broad-spectrum antibiotics, subsequent culture-guided therapy, and rigorous infection control practices are essential for achieving favorable outcomes. This case demonstrates the effectiveness of timely diagnosis and targeted antimicrobial treatment in managing HAP caused by *Pseudomonas aeruginosa*. Continued emphasis on multidisciplinary care, antimicrobial stewardship, and preventive strategies is vital for reducing the burden of HAP and improving patient outcomes in healthcare settings.

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