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Case Report

Study of community-acquired carbapenemase-producing *Klebsiella pneumoniae* infection

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ABSTRACT

Community-acquired carbapenemase-producing *Klebsiella pneumoniae* (CPKP) infection is a rare condition and presents diagnostic and therapeutic challenges. We report two such cases describing their clinical status, microbiological findings, therapeutic interventions, and outcomes. We report two cases with community-acquired CPKP infection without hospital-related risk factors. Both patients presented with fever with chills, productive cough, generalized weakness, and raised inflammatory markers. Bronchoalveolar lavage detected CPKP carrying resistance genes such as NDM, CTX-M, IMP, OXA-48-Like, and VIM. One patient developed multiple complications, including sepsis, acute respiratory distress syndrome (ARDS), and heart failure with preserved ejection fraction, ultimately causing multiple organ dysfunction syndrome. While the other patient developed ARDS with type 1 respiratory failure (T1RF). Both received adjusted antibiotic therapy and supportive care based on their clinical conditions and have shown positive recovery. These cases emphasize the occurrence of community-acquired CPKP pneumonia posing severe complications and with timely interventional therapy, favourable outcomes are achieved.

Keywords: Carbapenemase, *Klebsiella pneumoniae*, Bronchoalveolar lavage, Acute respiratory distress syndrome, Drug resistant genes, Antibiotic therapy

INTRODUCTION

Community-acquired pneumonia (CAP) is among the most prevalent infectious diseases globally and remains a significant contributor to morbidity, mortality, and healthcare burden.[1] Patients often present with acute respiratory symptoms such as cough, fever, dyspnea, and pleuritic chest pain, although clinical manifestations vary depending on the pathogen. CAP is diagnosed based on compatible clinical features and evidence of a new pulmonary infiltrate on chest imaging.[2] Timely diagnosis and appropriate antimicrobial therapy are crucial for improving clinical outcomes and reducing disease-related complications.[3] Since the causative pathogen is often unidentified at admission, broad-spectrum antibiotic therapy is initiated based on clinical severity, comorbidities, and the likelihood of drug-resistant organisms.[4]

Gram-negative bacteria, including *Klebsiella pneumoniae*, are recognized as major causative pathogens, particularly in patients with risk factors. Culture and sensitivity testing using microbiological procedures such as bronchoalveolar lavage (BAL) is essential for identifying the causative organism and guiding appropriate antimicrobial therapy, especially in patients unresponsive to empirical treatment.[5]

The rise of carbapenemase-producing Enterobacteriaceae (CPE) has become a major global public health issue because of broad antimicrobial resistance and limited treatment alternatives. Carbapenemases produced by CPE hydrolyze carbapenem antibiotics, compromising their effectiveness.[6] CPE infections are primarily associated

with healthcare settings and frequently linked to prior hospitalization, intensive care unit (ICU) admission, invasive procedures, and previous empirical antibiotic exposure.[7] Moreover, intestinal colonization by carbapenemase-producing *Klebsiella pneumoniae* can serve as a reservoir for future invasive infections, emphasizing the need for prompt microbiological diagnosis and antimicrobial stewardship.[8]

This study reports two cases initially diagnosed with CAP and were treated with empirical antibiotics. Following BAL culture identified carbapenemase-producing *Klebsiella pneumoniae*, revealing it to be a community-acquired CPE infection. Modification in the antimicrobial therapy emphasized the importance of culture-guided treatment in managing multi drug-resistant respiratory infections.

CASE REPORT

Case 1

A 35-year-old male presented with fever with chills, productive cough, shortness of breath, and generalized weakness. On admission initial investigations revealed leucocytosis (20,070 cells/cumm), markedly elevated C-reactive protein (CRP) level of 22.40 mg/dl and procalcitonin (4.12 ng/ml) indicating an active infectious and inflammatory process. Chest radiography showed cardiomegaly with bilateral prominent bronchovascular markings, while chest CT revealed areas of consolidation with air bronchograms in bilateral lower lobes, lingula, and right middle lobe, along with mild bilateral pleural effusions (Figure 1).

Liver function tests demonstrated hyperbilirubinemia (3.70 mg/dl) and elevated liver enzymes indicating sepsis-related hepatic dysfunction. 2D echocardiogram revealed a left ventricular ejection fraction (LVEF) of 58% and an increase in NT-proBNP level of 713 pg/ml, indicating heart failure with preserved ejection fraction (HFpEF). For managing respiratory distress associated with acute respiratory distress syndrome (ARDS), the patient was placed in an awake proning position and supported with high-flow nasal canula (HFNC) under non invesive respiratory therapy. The concurrent dysfunctions in pulmonary, hepatic, and cardiovascular systems demonstrated the presence of multiple organ dysfunction syndrome (MODS).

The patient was initially administered empirical IV antibiotics, including meropenem, clarithromycin, along with the appropriate supportive care (HFNC). The patient was initially stabilized during the course of treatment. However, despite early improvement, the patient experienced a deteriorated condition by developing a new onset of fever accompanied by worsening hypoxia. Microbiological investigations, including blood culture, pneumonia panel and BAL, were performed. BAL demonstrated the presence of *Klebsiella pneumoniae* group with antimicrobial resistance genes CTX-M, NDM, IMP, and OXA-48-Like. These findings indicated that the identified resistance genes were making the initial therapy ineffective, especially carbapenems, leading to the subsequent deterioration of the patient's condition.

Hence, the treatment approach replaced the initial antibiotics with a targeted antimicrobial therapy including ceftazidime-avibactam and aztreonam. During the clinical course, the patient demonstrated clinical improvement with stable vital signs and resolved hypoxia. Follow-up investigations have shown positive responses towards the adjusted treatment regimen, including the reductions in total leucocyte count to 12,690 cells/cumm and CRP to 4.30 mg/dl, while repeated chest radiography also showed improvement. As the patient's respiratory status improved, ventilation was also withdrawn. During the time of discharge, the patient maintained stable hemodynamics. No recurrence was noted in follow-up.

Case 2

A 62-year-old male presented with fever with chills, productive cough, shortness of breath, generalized weakness, and decreased appetite. On primary evaluation the patient was toxic and dehydrated. Initial laboratory investigations have shown leucocytosis (24,210 cells/cumm), an elevated c-reactive protein (17.20 mg/dl) and procalcitonin (1.34 ng/ml) suggesting the presence of a bacterial infection and inflammatory response.

Chest radiography demonstrated patchy inhomogeneous opacities in the right upper mid-zone, and chest CT further revealed multifocal tree-in-bud centrilobular nodular opacities and mild ground-glassing in both lungs, along with minimal left pleural effusion (Figure 1).

The patient was initially diagnosed with a lower respiratory tract infection (LRTI) complicated by ARDS with type 1 respiratory failure (T1RF) and empirically treated with intravenous meropenem and clarithromycin, along with antipyretics, analgesics and other supportive care.

BAL culture, susceptibility test, and pneumonia panel have detected *Klebsiella pneumoniae* group carrying antimicrobial resistant genes such as CTX-M, NDM, OXA-48-Like, and VIM. These findings confirmed the final diagnosis of carbapenemase-producing *Klebsiella pneumoniae* (CPKP) infection. Thus, the initial therapy was discontinued and switched to intravenous ceftazidime-avibactam, aztreonam and minocycline as targeted antimicrobial therapy.

During the hospital course, the patient improved symptomatically and showed gradual decline in total leucocyte count to 11,150 cells/cumm and CRP to 6.22 mg/dl and the repeated BAL has shown no bacterial growth on culture after 72 hours of incubation. Patient responded well to the tailored intravenous antibiotic

regimen and other supportive care. The patient recovered and was discharged in a hemodynamically stable condition. No recurrence was noted in follow-up.

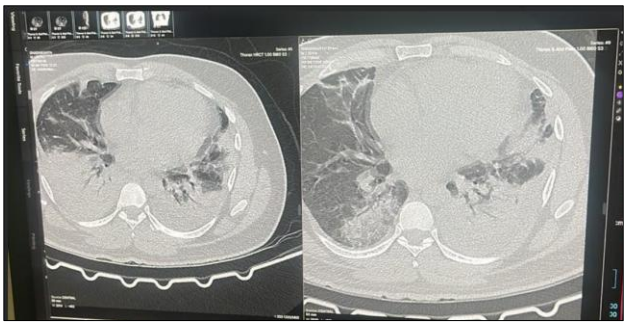


Figure 1: Chest CT findings in community-acquired *Klebsiella pneumoniae* infection.

DISCUSSION

CPE have become a significant worldwide public health concern due to their swift spread, presence of carbapenemase genes such as NDM, OXA-48-like, and VIM, broad antimicrobial resistance, few treatment choices, and link to heightened morbidity and mortality.[9,10] Prompt initiation of suitable treatment is necessary to enhance clinical recovery in CPE-infected patients.[11]

In the current study, two patients exhibited fever, productive cough, dyspnea, and overall weakness, which are clinical characteristics of bacterial community-acquired pneumonia.[12] Both cases exhibited significant leukocytosis alongside increased levels of C-reactive protein and procalcitonin, suggesting an ongoing bacterial infection with a notable systemic inflammatory response.[13]

Radiological examinations were crucial in identifying lung infections and assessing the extent of the disease. In case 1, imaging studies showed cardiomegaly with bilaterally prominent bronchovascular markings, bilateral consolidations with air bronchograms, and pleural effusions. In case 2, findings included patchy inhomogeneous opacities, multifocal tree-in-bud nodules, ground-glass opacities, and pleural effusion. These imaging results are typical characteristics of bacterial lung infections and illustrate the supportive functions of chest X-rays and computed tomography in diagnosing pneumonia.[14]

Microbiological analyses played a crucial role in determining the etiological diagnosis. Blood cultures continue to be the reference standard for identifying bloodstream pathogens, whereas BAL cultures and molecular pneumonia panels enhance pathogen detection from lower respiratory tract samples and support targeted antimicrobial treatments.[15]

In case 1, BAL culture detected *Klebsiella pneumoniae* harboring CTX-M, NDM, IMP, and OXA-48-like resistance genes. In case 2, BAL culture and pneumonia panel identified *Klebsiella pneumoniae* carrying CTX-M, NDM, OXA-48-like, and VIM carbapenemase genes, confirming CPKP infection.

In case 1, the dynamic development of severe pneumonia was first complicated by bacteremia and hepatic dysfunction. Despite empirical antimicrobial treatment leading to temporary clinical improvement, the recurrence of fever and respiratory distress necessitated further microbiological testing, which revealed multidrug-resistant *Klebsiella pneumoniae*. This clinical course highlights the significance of re-evaluating patients who show unexpected deterioration despite receiving broad-spectrum empirical treatment, especially when resistant Gram-negative infections are suspected.

A key characteristic in case 1 was the onset of MODS affecting the respiratory, hepatic, and cardiovascular systems. Sepsis serves as a significant factor in MODS, where organ dysfunction often surpasses the initial infection site and affects extrapulmonary organs like the liver and cardiovascular system.[16] Hepatic dysfunction is a well-known complication of sepsis and correlates with heightened disease severity and negative clinical results.[17]

The patient needed awake prone positioning and HFNC for acute hypoxemic respiratory failure related to ARDS. HFNC is a useful non invesive respiratory therapy method that enhances oxygen levels and may decrease the need for invasive methods in suitably chosen patients .[18]

In our study, both patients were initially administered broad-spectrum antibiotics while waiting for microbiological confirmation, in line with current guidelines for severe pneumonia.[19] Identification of NDM along with OXA-48-like, IMP, or VIM carbapenemases clarified the diminished effectiveness of carbapenems and the clinical decline seen in case 1.[10] The existence of several carbapenemase genes indicates the widespread antimicrobial resistance frequently seen in CPE isolates, which further limits effective treatment choices.[20]

After microbiological confirmation, antimicrobial therapy was suitably adjusted to include ceftazidime-avibactam alongside aztreonam in both patients, while minocycline was also given to the second patient based on susceptibility results. European society of clinical microbiology and infectious diseases (ESCMID) guidelines suggest that therapy should be chosen based on the type of carbapenemase present, as the effectiveness of treatment varies depending on the resistance mechanism. The pairing of ceftazidime-avibactam and aztreonam is especially potent against Enterobacterales that produce metallo-β-lactamases, as aztreonam is stable in the presence of these enzymes, while avibactam safeguards it from co-produced serine β-lactamases, thus maintaining its antibacterial efficacy.[11]

Recent clinical findings have shown positive results with ceftazidime plus avibactam and aztreonam synergistic action in CPKP infection, while a systematic review has additionally reinforced the use of combination antimicrobial therapy for severe infection caused by CPKP.[21,22] Timely initiation of microbiologically suitable treatment is a key predictor of survival in bloodstream infections caused by carbapenem-resistant *Klebsiella pneumoniae*.[23] Similarly, the cessation of ineffective empirical antibiotics after microbiological confirmation in both patients demonstrates the antimicrobial stewardship practices and suitable antimicrobial de-escalation.[24]

Both patients showed significant clinical improvement towards targeted antibiotic treatment. The first patient improved on inflammatory parameters, imaging, and respiratory function along with successful discontinuation of ventilation, while the second patient improved on decreasing inflammatory parameters along with culture-proven sterile bronchoalveolar lavage.

CONCLUSION

These positive results further highlight the significance of combining repeated microbiological studies, early identification of resistance mechanisms, targeted antibiotic therapy, and overall supportive treatment in the management of severe CPE-associated pneumonia.

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