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Severe Community-Acquired Pneumonia Due to Coinfection with Streptococcus pneumoniae and β-Lactamase-Negative Ampicillin-Resistant Haemophilus influenzae in an Elderly Patient

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1. Abstract
An 80-year-old man was admitted with fever, dyspnea, and productive cough. Imaging revealed right lower lobe pneumonia, and urinary antigen testing confirmed Streptococcus pneumoniae. Empiric treatment with meropenem led to rapid improvement, prompting de-escalation to high-dose ampicillin. Shortly afterward, the patient developed acute respiratory failure requiring mechanical ventilation due to ARDS. Sputum cultures later identified coinfection with penicillin-sensitive S. pneumoniae and β-lactamase-negative ampicillin-resistant (BLNAR) Haemophilus influenzae. Reinstating meropenem resulted in clinical recovery. This case emphasizes the clinical risk of BLNAR H. influenzae and the potential dangers of premature antibiotic narrowing in severe pneumonia.

2. Keywords
Community-acquired pneumonia, Streptococcus pneumoniae, Haemophilus influenzae, BLNAR, antibiotic de-escalation, ARDS

3. Introduction
Community-acquired pneumonia (CAP) remains a major infectious cause of hospitalization and death in older adults. Among bacterial pathogens, Streptococcus pneumoniae is the most frequently isolated organism, while Haemophilus influenzae also contributes significantly, particularly in elderly or vulnerable patients.

Although β-lactam antibiotics remain first-line therapy, emerging resistance—especially penicillin-resistant S. pneumoniae and β-lactamase-negative ampicillin-resistant (BLNAR) H. influenzae—complicates treatment decisions. BLNAR strains are particularly challenging because they do not produce β-lactamase but exhibit altered penicillin-binding proteins, leading to reduced susceptibility to ampicillin.

We report a severe case of CAP caused by dual infection with these organisms, complicated by respiratory failure following antibiotic de-escalation.

4. Case Presentation
An 80-year-old male presented to the emergency department with a 2-day history of high-grade fever, increasing shortness of breath, and brownish sputum production. He had experienced a mild upper respiratory illness one week earlier. His medical history included hypertension and chronic gastritis, and he had no history of smoking or chronic lung disease.

On admission, his temperature was 39.2°C and oxygen saturation was 92% on room air. Auscultation revealed coarse crackles over the right lower lung field. Laboratory tests showed leukocytosis (12,000/mm^3) with predominant neutrophils and elevated CRP (2.84 mg/dL). HbA1c was 7.2%, suggesting previously undiagnosed diabetes mellitus.

Chest radiography and CT imaging demonstrated consolidation in the right lower lobe consistent with bacterial pneumonia. Urine antigen testing was positive for S. pneumoniae, and sputum Gram stain showed Gram-positive diplococci with evidence of phagocytosis.

The patient was started on intravenous meropenem, resulting in marked clinical improvement within 72 hours. Fever subsided, inflammatory markers decreased, and respiratory symptoms improved significantly.

Given this improvement, therapy was de-escalated to high-dose intravenous ampicillin based on presumed susceptibility of S. pneumoniae.

5. Clinical Deterioration
Within 24 hours of switching antibiotics, the patient developed recurrent fever, worsening hypoxemia, and increased respiratory distress. His oxygen requirement escalated rapidly, and arterial blood gas analysis showed severe hypoxemia (PaO_2 65.3 Torr) with respiratory alkalosis.

By the fifth hospital day, chest imaging revealed diffuse bilateral infiltrates consistent with acute respiratory distress syndrome (ARDS). The patient required intubation and mechanical

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ventilation due to progressive respiratory failure.

6. Microbiological Results

Sputum cultures obtained at admission subsequently revealed mixed infection with:

- Penicillin-sensitive Streptococcus pneumoniae
- β-lactamase-negative ampicillin-resistant (BLNAR) Haemophilus influenzae

The H. influenzae isolate was resistant to ampicillin but susceptible to third-generation cephalosporins and carbapenems, including meropenem.

The clinical deterioration was attributed to inadequate coverage of BLNAR H. influenzae after antibiotic de-escalation.

7. Outcome

Meropenem therapy was restarted immediately after identification of the resistant organism. The patient showed rapid clinical improvement, with resolution of fever, stabilization of oxygenation, and gradual recovery of pulmonary function. He was successfully weaned off mechanical ventilation and later discharged in stable condition.

8. Discussion

This case highlights an important diagnostic and therapeutic challenge in severe CAP: mixed bacterial infection with differing antibiotic susceptibilities. While S. pneumoniae was initially identified and appropriately treated, concurrent infection with BLNAR H. influenzae was not recognized early, leading to treatment failure after narrowing antibiotic coverage.

BLNAR H. influenzae is increasingly reported in certain regions and is characterized by altered penicillin-binding proteins rather than β-lactamase production. As a result, it may appear susceptible in some settings but is clinically resistant to ampicillin.

The case underscores several key clinical lessons:

1. CAP may involve multiple pathogens simultaneously, especially in elderly patients.
2. Early clinical improvement does not guarantee full microbial coverage.
3. BLNAR H. influenzae should be considered even when pneumococcal infection is confirmed.
4. Premature de-escalation of antibiotics can lead to severe and life-threatening relapse.

Meropenem provided effective broad-spectrum coverage and was essential in reversing respiratory failure in this patient.

9. Conclusion

Severe CAP in elderly patients may involve coinfection with resistant organisms such as BLNAR H. influenzae. Clinicians should exercise caution when narrowing antibiotic therapy, even in the presence of early clinical improvement. Definitive microbiological results should guide therapy to avoid treatment failure and severe complications such as ARDS.

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