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(CASE REPORT)

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FATAL SEPTIC SHOCK IN HYPERMUCOVISCOUS *KLEBSIELLA PNEUMONIAE* PNEUMONIA DESPITE IN VITRO ANTIBIOTIC SUSCEPTIBILITY: A CASE REPORT

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ABSTRACT

Background: *Klebsiella pneumoniae* can cause rapidly progressive severe pneumonia, particularly in older adults with chronic illnesses. A positive string test demonstrates hypermucoviscosity but does not, in isolation, confirm the hypervirulent pathotype.

Case presentation: An 80-year-old woman with type 2 diabetes, hypertension and atrial fibrillation presented after four days of respiratory symptoms and a rapid decline. She had profound hypoxemia (oxygen saturation 80% on room air), persistent hypotension, lactate 5.5 mmol/L and procalcitonin 92 ng/mL. Chest radiography showed bilateral confluent air-space opacities. A tracheal aspirate tested with the BioFire FilmArray Pneumonia Plus panel detected *K. pneumoniae* group in its highest semiquantitative reporting category (≥10^7 copies/mL), together with human rhinovirus/enterovirus. Culture yielded *K. pneumoniae* with markedly mucoid colonies and a positive string test (12 mm). The isolate was reportedly susceptible to cefotaxime and multiple other antibacterial agents. Despite cefotaxime and gentamicin, invasive ventilation and vasopressor support, the patient died 36 hours after admission.

Conclusion: Severe *K. pneumoniae* pneumonia may have a fatal outcome despite in vitro antimicrobial susceptibility. The positive string test documents hypermucoviscosity; additional specific testing is necessary to establish hypervirulence. Molecular detection categories must be interpreted alongside culture and clinical findings.

Keywords: *Klebsiella pneumoniae*; Hypermuco-viscosity; Septic Shock; Pneumonia; Multiplex PCR; String Test

1 INTRODUCTION

Klebsiella pneumoniae is an important cause of severe lower respiratory tract infection, particularly in older adults and patients with chronic illnesses. Hypervirulent *K. pneumoniae* (hvKp) has emerged as a clinically relevant pathotype, often associated with invasive, community-onset infection and additional virulence determinants. However, the hypermucoviscous phenotype demonstrated by a positive string test is neither sensitive nor specific for hvKp; phenotypic appearance cannot replace genetic characterization [1,2].

Syndromic multiplex polymerase chain reaction (PCR) assays may rapidly identify bacterial and viral targets in respiratory samples and provide semiquantitative bacterial results before conventional culture is finalized [3]. The highest reporting category indicates a substantial amount of detected bacterial target DNA in the submitted specimen, but it is not an exact measure of viable bacterial burden or systemic infection severity. We describe a fatal episode of *K. pneumoniae* pneumonia associated with septic shock, in which early syndromic molecular detection, a hypermucoviscous isolate, and in vitro antibiotic susceptibility were documented.

2 CASE PRESENTATION

An 80-year-old woman presented to the emergency department with acute respiratory distress and altered mental status. Her medical history included type 2 diabetes mellitus for 15 years treated with oral glucose-lowering medications, essential hypertension, and atrial fibrillation treated with oral anticoagulation. According to family members, she had experienced four days of dry cough, rhinorrhea, and low-grade fever. Marked worsening of dyspnea and general condition had occurred during the 12 hours before admission.

On arrival, her blood pressure was 90/50 mmHg and remained low after initial fluid resuscitation, reported as 30 mL/kg of crystalloids. The heart rate was 118 beats/min, respiratory rate 30 breaths/min, and oxygen saturation 80% while breathing ambient air. She received immediate high-flow oxygen support. Mottling around the knees and cold extremities suggested impaired peripheral perfusion. Chest auscultation revealed bilateral crackles, predominantly in the middle and lower lung fields.

At presentation, laboratory tests demonstrated marked systemic inflammation, neutrophilic leukocytosis, thrombocytopenia, elevated creatinine and hyperlactatemia (Table 1). Other arterial blood gas values could not be verified from the available source documentation. A portable chest radiograph was described as showing bilateral, asymmetric confluent air-space opacities, especially in the right upper and left middle lung zones, without an obvious large pleural effusion. These findings were consistent with severe multilobar pneumonia. No computed tomography or other imaging confirming pulmonary parenchymal necrosis was available.

Table 1: Available laboratory findings at hospital admission.

Parameter	Reported result
Leukocytes	18,500 cells/mm^3 (neutrophils 90%)
Platelets	95,000/mm^3
C-reactive protein	285 mg/L
Procalcitonin	92 ng/mL
Arterial lactate	5.5 mmol/L
Urea	1.1 g/L
Creatinine	22 mg/L
Blood glucose	1.79 g/L

A tracheal aspirate was analyzed with the BioFire FilmArray Pneumonia Plus multiplex PCR panel. The assay detected the *K. pneumoniae* group in its highest semiquantitative bin (≥10^7 genomic copies/mL) and human rhinovirus/enterovirus. None of the documented bacterial resistance markers targeted by the panel (CTX-M, KPC, NDM, VIM, IMP, and OXA-48-like) was detected. The source report indicates a turnaround time of approximately 60 minutes after testing; the exact collection-to-result interval was not recorded. The viral target was reported as a combined rhinovirus/enterovirus result and did not distinguish the two.

Following approximately 24 hours of incubation, respiratory culture yielded *K. pneumoniae* as a pure isolate. The colonies were described as markedly mucoid. A string test performed with an inoculating loop produced a 12-mm filament, documenting a hypermucoviscous phenotype. The reported antimicrobial susceptibility profile included susceptibility to amoxicillin–clavulanate, piperacillin–tazobactam, cefotaxime, ceftazidime, cefepime, ertapenem, imipenem, meropenem, gentamicin, amikacin and ciprofloxacin, with intrinsic resistance to amoxicillin. The methods, minimum inhibitory concentrations, and interpretive breakpoint standards were not supplied in the source report.

The patient required intensive care, invasive mechanical ventilation and norepinephrine for refractory circulatory shock. Intravenous cefotaxime (2 g every eight hours) and gentamicin (5 mg/kg as a single dose) were administered

early in her admission; the exact first-dose time was unavailable. Her condition deteriorated with persistent shock, lactic acidosis and anuria. Death occurred 36 hours after hospital admission in the setting of multiorgan failure.

Table 2: Clinical, microbiological and treatment timeline (times relative to admission).

Time relative to admission	Clinical event / investigation	Treatment or result
About 4 days before	Dry cough, rhinorrhea and low-grade fever began	No prehospital treatment documented
About 12 h before	Rapid worsening of dyspnea and general condition	Emergency assessment subsequently required
Admission (0 h)	Hypoxemia, hypotension and altered consciousness; chest radiograph and blood tests	Fluid resuscitation, supplemental oxygen and ICU-level support
Early hospitalization; exact hour not recorded	Tracheal aspirate sent for FilmArray Pneumonia Plus	*K. pneumoniae* group $\geq 10^7$ copies/mL; rhinovirus/enterovirus detected; reported assay time ~60 min
During ICU stay; exact time not recorded	Refractory shock and respiratory failure	Intubation, invasive ventilation, norepinephrine, IV cefotaxime and one dose of gentamicin
After ~24 h of culture incubation	Pure culture of *K. pneumoniae*; mucoid colonies and string test	Positive string test: 12-mm filament; susceptibility testing reported, exact release time unknown
36 h after admission	Progressive shock, anuria and multiorgan failure	Death

3 DISCUSSION

The principal feature of this case is rapid deterioration from a community-onset respiratory illness to fatal pneumonia and septic shock, despite a *K. pneumoniae* isolate reported susceptible to the antibiotics administered. Septic shock is characterized by circulatory and metabolic abnormalities accompanying infection, including vasopressor dependence and hyperlactatemia after fluid resuscitation [4]. In this patient, hypoxemia, persistent hypotension, lactate elevation, and organ dysfunction indicate extreme clinical severity on presentation. A susceptible laboratory result cannot reverse established severe host organ injury and should not be interpreted as a prognostic guarantee.

The visually mucoid isolate and 12-mm positive string test demonstrate hypermucoviscosity but do not establish the hvKp pathotype. Although hvKp has been linked to accessory virulence determinants such as aerobactin biosynthesis genes and regulators of mucoidy, classical *K. pneumoniae* isolates may also display hypermucoviscosity, and some hvKp isolates are not string-test positive. Genomic or targeted molecular evaluation of markers such as iucA, iroB, peg-344, and plasmid-associated rmpA/rmpA2 would strengthen pathotype assessment [1,2]. These markers were not tested in the present record, and the organism should therefore be described as "hypermucoviscous *K. pneumoniae*" rather than genetically confirmed hvKp.

The FilmArray result adds important microbiological context. For *K. pneumoniae* group, the assay provides approximate bacterial DNA abundance in reporting bins rather than an exact quantitative culture; the highest category is $\geq 10^7$ genomic copies/mL [3]. In this case, the high-category result was reinforced by recovery of *K. pneumoniae* in pure culture from the respiratory sample and an accompanying clinical picture of severe pneumonia. Nevertheless, detection of a high target concentration alone cannot establish an inoculum-mediated antibiotic effect, prove bloodstream invasion, or quantify endotoxin exposure. Similarly, a negative targeted resistance-gene result cannot exclude other genetic or phenotypic resistance mechanisms; conventional susceptibility testing remains necessary [3].

Human rhinovirus/enterovirus nucleic acid was also detected. A preceding viral respiratory illness may have contributed to susceptibility to bacterial pneumonia, but the panel does not distinguish rhinovirus from enterovirus in this output, and detection cannot establish the relative contribution or the temporal sequence of two pathogens. Advanced age and diabetes were additional clinically relevant risk factors for a severe infection. Procalcitonin was strikingly elevated (92 ng/mL), consistent with marked systemic inflammation in the setting of a serious bacterial infection; however, a single procalcitonin measurement cannot establish the mechanism of shock or provide a validated patient-specific mortality prediction [5].

The combination of a positive string test and a severe clinical phenotype is clinically noteworthy, especially in a patient with diabetes. However, a causal link between hypermucoviscosity and the fatal outcome cannot be established from a single observation. In particular, the absence of targeted virulence-gene testing, strain sequencing, and documented bloodstream infection precludes classification of this isolate as genetically confirmed hypervirulent *K. pneumoniae*. The course of illness may reflect a combination of host vulnerability, high disease severity at presentation, delayed physiological reversibility and pathogen-related factors, none of which can be quantified here [1,2,4].

Several limitations merit emphasis. Blood culture findings, prior antimicrobial exposure, first-dose timing, details of the susceptibility method and breakpoint interpretation, serial gas measurements, and longitudinal organ-support data were unavailable in the submitted source material. The available gas analysis parameters could not be verified against an original laboratory printout and were not presented. Although the radiograph was described, the original radiographic image was not available for inclusion, and no CT evidence of cavitation or pulmonary necrosis was provided. Consequently, neither "necrotizing pneumonia" nor an independently verified hvKp designation is established by the available observations. These limitations should be resolved, where possible, by checking the original clinical and microbiological records.

4 CONCLUSION

This case illustrates rapid progression of severe *K. pneumoniae* pneumonia to fatal septic shock despite reported in vitro susceptibility to administered antibacterial agents. The documented 12-mm string test identifies a hypermucoviscous isolate but cannot alone confirm the hypervirulent pathotype. Rapid multiplex respiratory testing can support early microbiological recognition, while quantitative categories and resistance-gene findings require careful interpretation alongside culture, phenotypic susceptibility results and the patient's clinical condition.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

Ali Bouddounit, Youssef El Kamouni, Said Zouhair and Lamiae Arsalane declare no conflicts of interest in connection with this report

Statement of ethical approval

The case report uses de-identified information obtained during routine clinical care. The report was prepared in accordance with the applicable institutional ethical and confidentiality requirements.

Statement of informed consent

Consent for publication of the anonymized clinical information was obtained in accordance with the applicable institutional and legal requirements.

Authors' contributions.

All four authors contributed to the preparation of the case report, reviewed the manuscript and approved the final version for submission

Data availability

The patient-level clinical records are not publicly available in order to safeguard confidentiality. Relevant de-identified findings are presented in the article.

REFERENCES

[1] Russo TA, Marr CM. Hypervirulent *Klebsiella pneumoniae*. Clin Microbiol Rev. 2019;32(3):e00001-19. doi:10.1128/CMR.00001-19.

[2] Russo TA, Olson R, Fang CT, et al. Identification of biomarkers for differentiation of hypervirulent *Klebsiella pneumoniae* from classical *K. pneumoniae*. J Clin Microbiol. 2018;56(9):e00776-18. doi:10.1128/JCM.00776-18.

[3] Murphy CN, Fowler R, Balada-Llasat JM, et al. Multicenter evaluation of the BioFire FilmArray Pneumonia/Pneumonia Plus Panel for detection and quantification of agents of lower respiratory tract infection. J Clin Microbiol. 2020;58(7):e00128-20. doi:10.1128/JCM.00128-20.

[4] Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287.

[5] Leli C, Ferranti M, Moretti A, et al. Procalcitonin levels in Gram-positive, Gram-negative, and fungal bloodstream infections. Dis Markers. 2015;2015:701480. doi:10.1155/2015/701480.