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ABOUT COVER

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CASE REPORT

Fatal community-acquired bloodstream infection caused by *Klebsiella variicola*: A case report

Da-Li Long, Yu-Hui Wang, Jin-Long Wang, Si-Jie Mu, Li Chen, Xian-Qing Shi, Jian-Quan Li

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Abstract

BACKGROUND

Klebsiella pneumoniae (*K. pneumoniae*) is an infective microorganism of worldwide concern because of its varied manifestations and life-threatening potential. Genetic analyses have revealed that subspecies of *K. pneumoniae* exhibit higher virulence and mortality. However, infections with *Klebsiella* subspecies are often misdiagnosed and underestimated in the clinic because of difficulties in distinguishing *K. pneumoniae* from its subspecies using routine tests. This case study reports the rapid and fatal effects of *K. pneumoniae* subspecies.

CASE SUMMARY

A 52-year-old male patient was febrile and admitted to hospital. Examinations excluded viral and fungal causes along with mycoplasma/chlamydia and parasitic infections. Bacterial cultures revealed blood-borne *K. pneumoniae* sensitive to carbapenem antibiotics, although corresponding treatment failed to improve the patient's symptoms. His condition worsened and death occurred within 72 h of symptom onset from sepsis shock. Application of the PMseq-DNA Pro high throughput gene detection assay was implemented with results obtained after death showing a mixed infection of *K. pneumoniae* and *Klebsiella variicola* (*K. variicola*). Clinical evidence suggested that *K. variicola* rather than *K. pneumoniae* contributed to the patient's poor prognosis.

CONCLUSION

This is the first case report to show patient death from *Klebsiella* subspecies infection within a short period of time. This case provides a timely reminder of the clinical hazards posed by *Klebsiella* subspecies and highlights the limitations of classical laboratory methods in guiding anti-infective therapies for complex cases. Moreover, this report serves as reference for physicians diagnosing similar

diseases and provides a recommendation to employ early genetic detection to aid patient diagnosis and management.

Key Words: Community-acquired bloodstream infection; Mixed infection; *Klebsiella variicola*; *Klebsiella pneumoniae*; High throughput gene detection; Case report

Core Tip: *Klebsiella pneumoniae* infection leads to worldwide concerns with its high mortality and varied manifestation. However, it is difficult to distinguish *Klebsiella pneumoniae* from its subspecies using classic clinical examinations. We here report a case who died with *Klebsiella* subspecies infection within 72 h. This case was diagnosed by genetic detection rather than classic laboratory methods. This case suggests that we should be alert to the clinical hazards and fatal effect of *Klebsiella* subspecies, classic method is limited in guiding the anti-infection therapy for complex cases, and early genetic detection should be performed in the diagnosis and management of complex infection.

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INTRODUCTION

Klebsiella pneumoniae (*K. pneumoniae*) infections are known to be associated with high incidence and mortality. This microorganism causes outbreaks of nosocomial infections and even drug resistance, and can lead to infection in the community among health-care patients or people with underlying immunodeficiency[1]. Based on genetic analysis, *K. pneumoniae* is divided into three phylogroups: *K. pneumoniae* (KpI), *K. quasipneumoniae* (KpII), and *K. variicola* (KpIII)[2]. KpI is the most frequent group encountered in the clinic, followed by KpIII and KpII[1,3]. KpI is usually defined as classic *Klebsiella* (Ck) or hypervirulent *Klebsiella* (Hvkp) according to their invasiveness or virulence, while subspecies of *Klebsiella* (KpII and KpIII) usually present with higher virulence[1]. Currently, approximately 20% of the human isolates assumed to be *K. pneumoniae* are in fact *K. variicola* or *K. quasipneumoniae*[1]. However, since classical laboratory examinations cannot readily distinguish KpI from the KpII and KpIII phylogroups[4], the clinical hazards and importance of KpII and KpIII are often overlooked.

CASE PRESENTATION

Chief complaints

A 52-year-old man presented with unexplained high fever, abdominal pain, and headache for 1 d.

History of present illness

The patient was subsequently admitted to the intensive care unit with diarrhea and confusion.

History of past illness

The patient reported a 5-year history of type 2 diabetes mellitus (T2DM) and 7 years of suffering gout but had no prior medical history related to the current symptoms.

Personal and family history

The patient had no particular individual or family history.

Physical examination

Physical assessment revealed a body temperature of 40 °C but without other obvious abnormal signs.

Laboratory examinations

Laboratory examinations revealed slightly deteriorated hepatorenal function and clotting function and increases in inflammatory parameters (Tables 1-3). Other laboratory biochemical tests proved negative for signs of viral and mycobacterial infections along with mycoplasma/chlamydia and biomarkers of

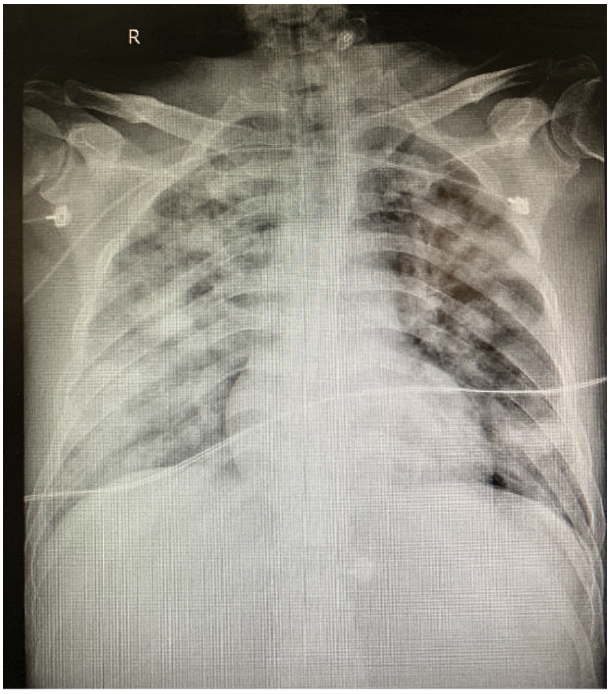

Figure 1 Chest radiograph.

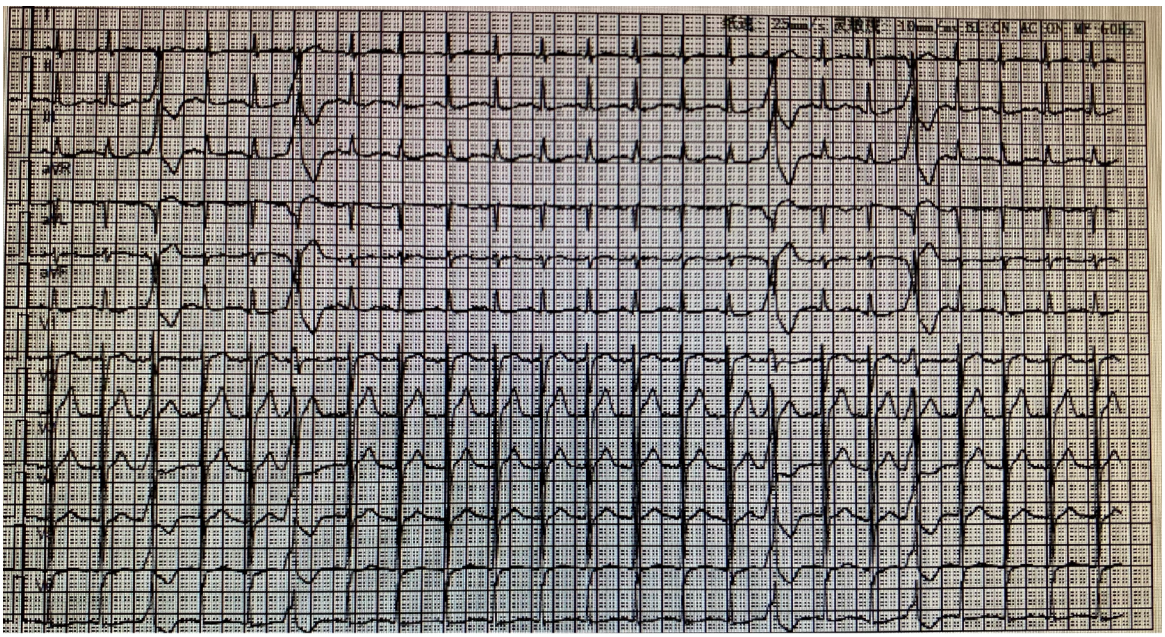

Figure 2 Electrocardiogram.

autoimmune diseases (Table 4). Traditional bacterial culture of the patients' blood sample showed bacterial infection with *K. pneumoniae* (Table 5).

Imaging examinations

Chest radiography showed the manifestations of an inflammatory response, while other imaging results showed no obvious abnormalities (Figures 1-3).

Table 1 Liver and renal function results

Item	Result (1st)	Reference range
TBIL (μmol/L)	19.4	3.6-20.5
TP (μmol/L)	49.8	65-85
ALB (g/L)	31.4	40-55
ALT (U/L)	156	9-50
AST (U/L)	182	15-40
sCrea (μmol/L)	160	57-97
Urea (μmol/L)	12.40	3.1-8
GLU (mmol/L)	13.89	3.9-6.1
K^+ (mmol/L)	3.22	3.5-5.5
UA (μmol/L)	591	210-420

Blood samples were collected on admission and liver and renal function parameters examined. Results were acquired about 2 h after sample collection. TBIL: Total bilirubin; ALB: Serum albumin; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GLU: Glutamate; sCrea: Serum creatinine; UA: Uric acid.

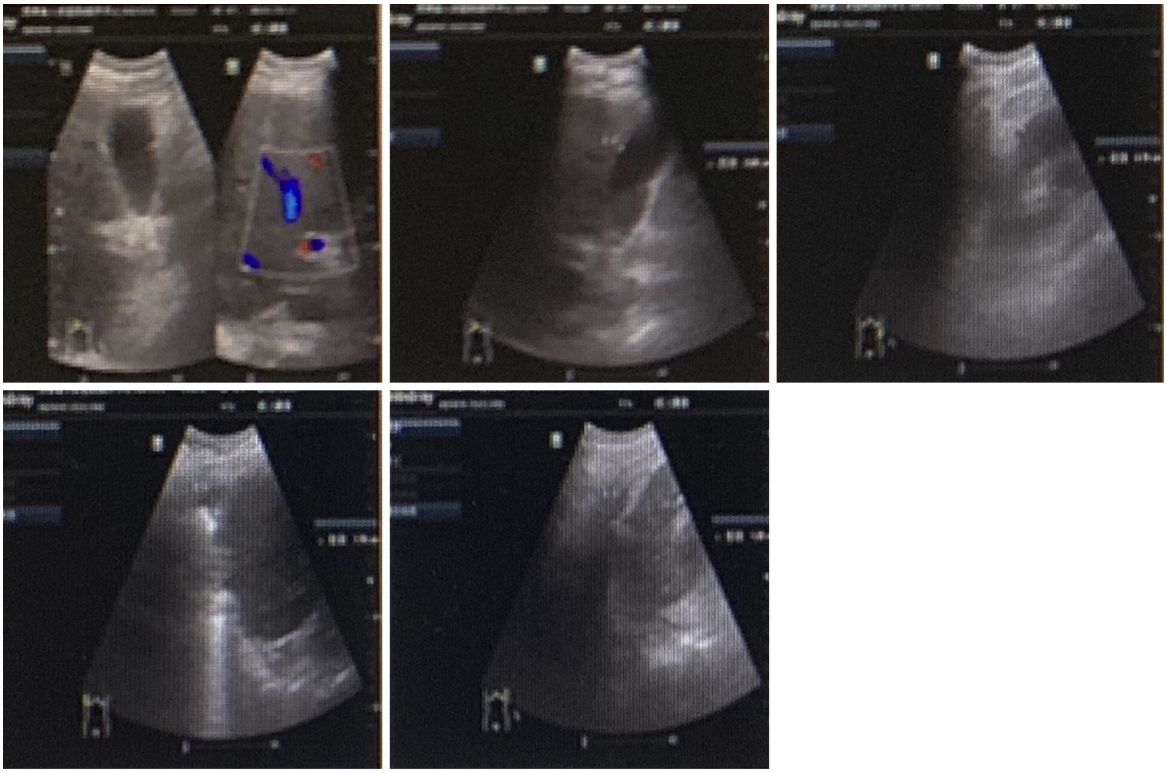

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Figure 3 Images of abdominal ultrasonography.

FINAL DIAGNOSIS

Based on the patient's symptoms and laboratory data, sepsis and septic shock were diagnosed according to the diagnostic criteria.

TREATMENT

The initial treatment prescribed after the availability of the laboratory results involved a broad-spectrum

Table 2 Clotting function parameters tested after referral

Item	Result	Reference range
3P	Negative	Negative
D-D (μg/mL)	60.75↑	0-1
FDP (μg/mL)	128.5↑	0-5
PT (s)	18.3↑	9.2-12.2
INR	1.59↑	0.8-1.2
APTT (s)	67.6↑	21.1-36.5
TT (s)	23.4↑	14-21
FBG (g/L)	22.58	1.8-3.5

On admission, blood samples were sent for clotting assessment. Results were acquired 1 h after sample collection. FDP: Flexor digitorum profundus; PT: Prothrombin time; INR: International normalized ratio; APTT: Activated partial thromboplastin time; TT: Thromboplastin time; FBG: Fibrinogen.

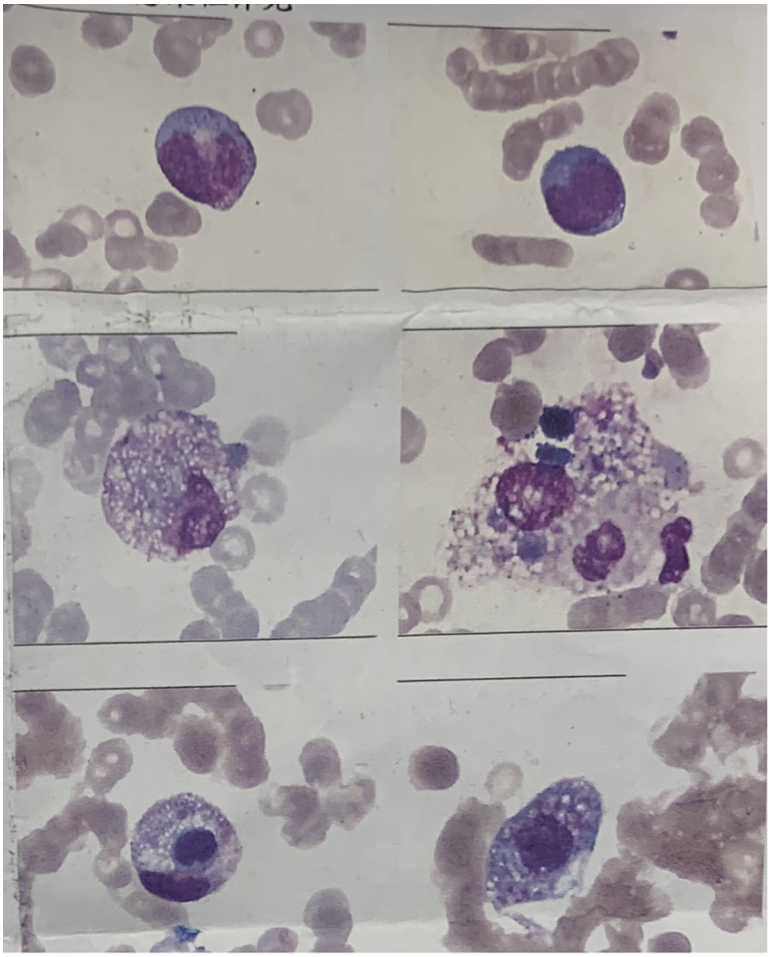

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Figure 4 Bone marrow biopsy result.

regimen for bacterial and fungal infections (intravenous meropenem 1 g per 6 h + intravenous caspofungin 70 mg/initial dose).

OUTCOME AND FOLLOW-UP

The patient's condition rapidly deteriorated within hours of admission with decreased blood pressure

Table 3 Inflammatory parameters

Item	Result	Reference range
WBC ($\times 10^9$)	1.2	3.5-10
NEUT ($\times 10^9$)	0.63	1.8-6.3
%NEUT	52.5	40-75
MONO ($\times 10^9$)	0.05	0.1-0.6
%MONO	4.2	3-10
LYMBP ($\times 10^9$)	0.39	1.1-3.2
%LYMBP	32.5	20-50
RBC ($\times 10^{12}$)	1.83	3.5-5.5
HGB (g/L)	52.0	114-163
%HCT	15.6	35-50
PLT ($\times 10^9$)	10	125-350
CRP (mg/L)	230.33	0-5
PCT	> 100	0-0.046

On admission, blood samples were sent for routine examination including C-reactive protein and procalcitonin. Results were acquired 1 h after sample collection. WBC: White blood cells; NEUT: Neutrophils; MONO: Monocytes; LYMBP: Lymphocytes; RBC: Red blood cells; HGB: Hemoglobin; HCT: Hemocrit; PLT: Platelets; CRP: C-reactive protein; PCT: Procalcitonin.

and reduced oxygen saturation. Life support therapies including mechanical ventilation and vasoactive drugs did not improve the patient's condition. He suffered cardiac arrest on the second day of admission and was declared clinically dead after several rescue efforts. The PMseq-DNA Pro high throughput gene detection assay was initiated on the second day after admission with the results acquired 2 d later. This analysis revealed infection with *K. pneumoniae* and *K. variicola* (Table 6). A bone marrow biopsy supported the findings of severe bacterial infection (Figure 4).

DISCUSSION

Klebsiella is a genus of Gram-negative bacterium within the Enterobacteriaceae family. It usually causes opportunistic nosocomial infections among hospitalized patients or outbreaks of community-acquired infections. *Klebsiella* mainly colonizes human gut but it has also been isolated from the skin surfaces such as hands and face, and can be isolated from various environmental sources including water, plants, and soil[1,5]. The genus contains several subspecies that manifest varied clinical outcomes, even death, leading to significant concerns about the accurate and timely identification of the *Klebsiella* subspecies involved together with a better understanding of the patient risk factors involved to reduce mortality risks[6].

Recent research has revealed that diabetes is a significant risk factor for hypervirulent *K. pneumoniae* infection and for causing serious complications[7-9]. In our patient's case, a medical history of T2DM could have contributed to an underlying immunodeficiency that was responsible for the fatal systemic infection. Although both *K. pneumoniae* and *K. variicola* were detected in the patients' blood sample, *K. variicola* may have played a more decisive role in the resulting outcome since treatment to target *K. pneumoniae* failed to improve the patient's condition. Moreover, this is consistent with the notion that *K. variicola* is a frequent cause of bloodstream infections and higher mortality[3,4].

It is difficult to distinguish *K. pneumoniae* and its subspecies by classic bacterial culture methods. This may lead to misdiagnosis or delayed diagnosis and incorrect treatment[4]. As shown in this case, *K. pneumoniae* was found in blood culture and although the clinical isolate was shown to be sensitive to the carbapenem class of antibiotics, the patient did not respond to treatment with meropenem. Similar to *K. pneumoniae*, drug-resistant plasmids in the bacterial structure of *K. variicola* contribute to its virulence and resistance, but the *K. variicola* has the higher-risk antibiotic resistance-related genes sequences, thus giving it higher virulence and resistance[10,11]. Recent clinical observations have shown that tigecycline and polymyxin display higher rates of treatment success in hypervirulent *Klebsiella* infection than other antibacterial drugs such as carbapenem[12]. Moreover, a combination of treatments is preferred to monotherapy in cases of severe infections[13,14]. Unfortunately, treatments to target *K. variicola* infection were not prescribed here because the patients' illness rapidly progressed before genotyping

Table 4 Results related to virus, mycobacteria, mycoplasma/chlamydia, and autoimmune disease

Item	Result	Reference
RSV-IGM	Negative	Negative
ADV-IGM	Negative	Negative
IFZA-IGM	Negative	Negative
IFZB-IGM	Negative	Negative
HPIVs-IGM	Negative	Negative
MP-IGM	Negative	Negative
CP-IGM	Negative	Negative
CBV-IGM	Negative	Negative
CAV-IGM	Negative	Negative
ECHO-IGM	Negative	Negative
LP-IGM	Negative	Negative
2019-nCoV	Negative	Negative
EB-DNA (copies/mL)	< 5E + 2	< 5E + 2
EB-DNA	Negative	Negative
CMVDNA DL (copies/mL)	< 5E + 2	< 5E + 2
CMV DNA DX	Negative	Negative
t1	Test method: Blotting	
A-PR3	Negative	Negative
A-MP0	Negative	Negative
A-GBM	Negative	Negative
t2	Test method: Fluorescence	
cANCA	Negative	Negative
pANCA	Negative	Negative

After admission, blood samples were sent for assessment of infection by virus, mycobacteria, and mycoplasma/chlamydia along with changes in autoimmune disease markers.

results were available.

As well illustrated by our case, *K. pneumoniae* subspecies can be rapidly fatal although their presence may often be overlooked due to the limitations of routine clinical examinations. This case should raise awareness among clinicians to consider *Klebsiella* subspecies infections, especially in cases of unexplained fever or other suspicious clinical presentations that may indicate this condition. Moreover, this case highlights the need to introduce genetic techniques into current clinical practices, especially for the early diagnosis of severe infections.

CONCLUSION

In summary, we have reported a patient dead with fatal infection caused by *K. variicola*. This fatal infection was identified by PMseq-DNA Pro high throughput gene detection assay. This case calls attention to *Klebsiella* subspecies infections and the need for early introduction of genetic technology in critically ill patients.

Table 5 Results of bacterial culture and drug sensitivity

Specimen	Blood		
Equipment	Phoenix100		
Items	Bacterial culture + antimicrobial susceptibility		
Results	*Klebsiella pneumoniae*		
Antibiotics	MIC	Result interpretation	Cutoff
Cefotaxime		S	S ≤ 1; R ≥ 4
Cotrimoxazole	≤ 20	S	S ≤ 2/38; R ≥ 4/76
Tigecycline	≤ 0.5	S	
Levofloxacin	≤ 0.12	S	S ≤ 0.5; R ≥ 2
Amikacin	≤ 2	S	S ≤ 16; R ≥ 64
Imipenem	≤ 0.25	S	S ≤ 1; R ≥ 4
Er ertapenem	≤ 0.12	S	S ≤ 0.5; R ≥ 2
Cefepime	≤ 0.12	S	S ≤ 2; R ≥ 16
Ce foperazone/sulbactam	≤ 8	S	S ≤ 16; R≥ 64
Ceftriaxone	≤ 0.25	S	S ≤ 1; R ≥ 4
Ceftazidime	≤ 0.12	S	S ≤ 4; R ≥ 16
Cefoxitin	≤ 4	S	S ≤ 8; R ≥ 32
Cefuroxime axetil	4	S	
Cefuroxime	4	S	S ≤ 4; R ≥ 32
Piperacillin/tatabatam	≤ 4	S	S ≤ 16/4; R ≥ 128/4
Amoxicillin/clavulanate	≤ 2	S	S ≤ 8/4; R ≥ 32/16
ESBL	Neg	-	

A blood sample was collected and examined following the standards of bacterial culture. The results of bacterial growth and antimicrobial susceptibility were acquired 24 h later.

Table 6 PMseq-DNA Pro high throughput gene detection of blood sample

Type	Genus (number of sequences)		Species (number of sequences)	
G	*Klebsiella*	68405	*Klebsiella pneumoniae*	243747
			Klebsiella variicola	543

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FOOTNOTES

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