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HOSPITAL-ACQUIRED KLUYVERA GEORGIANA BACTEREMIA IN A PATIENT WITH DECOMPENSATED CIRRHOSIS: A CASE REPORT FROM VIETNAM AND A LITERATURE REVIEW

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ABSTRACT

Bacteraemia caused by *Kluyvera georgiana* is an exceedingly rare clinical entity. As of 2024, only one case had been reported in the world literature: a 67-year-old female patient with recurrent pancreatic cancer undergoing chemotherapy in Japan, in whom the portal of entry for the pathogen was the biliary tract following choledochojejunotomy. We report the second case worldwide and the first from Vietnam, occurring in a 34-year-old male with decompensated cryptogenic cirrhosis. On hospital day 10, the patient developed high-grade fever (39.5°C) with rigors. Two independent blood culture sets drawn from two separate peripheral venous sites both yielded *K. georgiana*, identified by matrix-assisted laser desorption/ionization time of flight mass spectrometry. Notably, the patient exhibited leukopenia (1.710 cells/µl) with only minimally elevated procalcitonin (0.08 ng/ml), reflecting a blunted inflammatory response characteristic of cirrhosis-associated immune dysfunction. Antimicrobial susceptibility testing demonstrated resistance to ampicillin-sulbactam, ciprofloxacin, cefotaxime, ceftazidime, and trimethoprim-sulfamethoxazole, a substantially broader resistance phenotype than reported in the first case. The patient was treated with intravenous imipenem/cilastatin for 14 days and achieved complete clinical recovery. This case expands the recognized at-risk population for *K. georgiana* bacteraemia to include patients with decompensated cirrhosis, underscores the critical diagnostic value of early blood cultures when inflammatory markers are unreliable, and highlights the necessity of comprehensive antimicrobial susceptibility testing given the potentially variable resistance phenotypes among *K. georgiana* strains.

KEYWORDS

Bacteraemia, liver cirrhosis, cirrhosis-associated immune dysfunction (CAID), MALDI-TOF MS, hospital-acquired infection.

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LEARNING POINTS

- *Kluyvera georgiana* bacteraemia can occur in patients with decompensated cirrhosis via gut bacterial translocation, expanding the recognized at-risk population beyond oncologic or chemotherapy-immunosuppressed hosts.
- Conventional sepsis biomarkers are unreliable in cirrhosis-associated immune dysfunction attenuates both leukocyte and procalcitonin responses. Blood cultures must be drawn whenever clinical suspicion arises, regardless of inflammatory marker values.
- Antimicrobial susceptibility of *K. georgiana* is unpredictable. Resistance profiles may vary substantially between strains, mandating comprehensive susceptibility testing and precluding empirical de-escalation based on historical data.

INTRODUCTION

Kluyvera spp. are Gram-negative, rod-shaped, facultatively anaerobic bacilli belonging to the family Enterobacteriaceae. These organisms are widely distributed in environmental reservoirs, including water, soil, sewage, hospital sinks, and animal habitats, and they regularly colonize the human gastrointestinal tract as commensals without causing disease[1,2]. The genus currently comprises four recognized species: *K. ascorbata*, *K. cryocrescens*, *K. georgiana*, and *K. cochleae*. Human infections are primarily caused by *K. ascorbata* and *K. cryocrescens*, with diverse clinical manifestations including urinary tract infections, cholangitis, soft tissue infections, peritonitis, and bacteraemia. A cumulative literature review through 2024 identified 24 adult cases of *Kluyvera spp.* infection, the majority attributed to *K. ascorbata* in patients with underlying malignancy, diabetes mellitus, or other immunocompromising conditions[3].

K. georgiana was formally reclassified within the genus Kluyvera in 2005 based on molecular taxonomic analysis[2]. Of particular relevance to antimicrobial resistance, CTX-M extended-spectrum β-lactamases (ESBLs) - the most prevalent β-lactamase group among Enterobacteriaceae globally - have been demonstrated to have evolved from the chromosomal blaKLU β-lactamase genes of *Kluyvera spp.* through mutation and horizontal gene transfer[4]. This evolutionary trait raise significant concerns regarding the potential for multidrug resistance in this genus.

In a literature review by Endo et al. published in 2024, only a single case of *K. georgiana* bacteraemia had been reported, in a 67-year-old female with recurrent pancreatic cancer chemotherapy undergoing in Japan, with the infection occurring in the context of acute ascending cholangitis following choledochojejunotomy[3]. We describe the second reported case worldwide, occurring in a patient with decompensated cirrhosis in Vietnam, and notable for a considerably broader antimicrobial resistance profile.

CASE DESCRIPTION

A 34-year-old male was admitted for an episode of hepatic decompensation. He had a 12-year history of cryptogenic cirrhosis that had been established after systematic exclusion of hepatitis B virus and hepatitis C virus infection, autoimmune hepatitis, Wilson's disease, and hereditary hemochromatosis; he had no history of alcohol consumption. Prior to admission, he was receiving propranolol 40 mg/day for prophylaxis of variceal secondary bleeding to portal hypertension. He presented with progressive fatigue, jaundice, and ascites. Vital signs were stable at presentation. Abdominal ultrasonography demonstrated ascites and splenomegaly. Esophagogastroduodenoscopy revealed grade II oesophageal varices and gastritis. Admission laboratory investigations showed a white blood cell (WBC) count of 2.6 $\times 10^9$/l, consistent with hypersplenism and secondary bone marrow suppression in the context of cirrhosis. Liver function tests showed total bilirubin 80.6 µmol/l, serum albumin 29 g/l, and prothrombin activity 32%.

On hospital day 10, during treatment for hepatic decompensation, the patient developed an acute high-grade fever (39.5°C) with rigors. Vital signs at that time were: heart rate 78 bpm, blood pressure 110/70 mmHg, and oxygen saturation (SpO_2) 96% on room air. Systematic physical examination did not identify a focal source of infection. The chest radiograph was unremarkable. The rapid influenza antigen test and the rapid dengue NS1 antigen test were both negative. Urinalysis revealed pyuria (70 leukocytes/µl) with positive ketones, raising the possibility of a urinary source; however, urine culture returned negative.

Immediately upon onset of symptoms, two independent sets of blood cultures were collected from two separate peripheral venous sites. Laboratory investigations at the time of fever documented the following: WBC count 1.710/µl (neutrophils 73%), procalcitonin 0.08 ng/ml, aspartate aminotransferase 50 U/l, and alanine aminotransferase 29 U/l. Both blood culture sets yielded *K. georgiana*, identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) using the Bruker Biotyper system (identification score ≥2.0). Concordant positivity of two independent blood culture sets confirmed true bacteraemia, effectively excluding contamination.

Antimicrobial susceptibility testing demonstrated that the *K. georgiana* isolate was susceptible to amikacin, gentamicin, cefepime, imipenem/cilastatin, meropenem, and levofloxacin; resistant to ampicillin-sulbactam, ciprofloxacin, cefotaxime, ceftazidime, and trimethoprim-sulfamethoxazole (TMP-SMX); and intermediate to

ceftriaxone. The patient was promptly initiated on intravenous imipenem/cilastatin 0.5 g every 6 hours (2 g/day total), commenced immediately after blood culture collection, consistent with current principles for early empirical treatment of hospital-acquired bacteraemia in the severely immunocompromised host. Subsequent susceptibility results confirm the appropriateness of this therapeutic selection.

Following antibiotic initiation, no further febrile episodes were recorded. A follow-up blood culture obtained on day 7 of antibiotic therapy (hospital day 17) was negative. The WBC count improved progressively from 1.71 to 2.96 × 10^9/l over 7 days, though remaining below the normal reference range due to the underlying cirrhosis. Upon completion of a 14-day course of imipenem/cilastatin, the patient was discharged in stable condition on hospital day 26. The clinical course, laboratory data, and treatment timeline are summarized in *Table 1*.

DISCUSSION

To the best of our knowledge, this is the second reported case of *K. georgiana* bacteraemia in the world literature and the first documented case in Vietnam[3]. A detailed comparison of the two reported cases is presented in *Table 2*. The first reported case of *K. georgiana* bacteremia, published by Endo et al. in 2024, occurred in a patient with pancreatic malignancy on cytotoxic chemotherapy, with the presumed portal of entry being the biliary tract following choledochojejunotomy[3]. Our case differs in that the predominant predisposing condition was decompensated cirrhosis with its associated immune. In patients with advanced cirrhosis, gut microbiota dysbiosis combined with impaired intestinal mucosal barrier integrity and cirrhosis-associated immune dysfunction (CAID) - a syndrome encompassing reduced complement activity, impaired neutrophil function, diminished reticuloendothelial phagocytic capacity, and attenuated cytokine-mediated responses, significantly increases the risk of spontaneous bacterial translocation from the intestinal lumen into the systemic circulation[5,6]. This mechanism is the most probable route of infection in our patient, although a urinary portal of entry, suggested by pyuria, cannot be excluded.

A particularly salient feature of this case was the markedly blunted inflammatory response at the time of bacteremia: leukopenia (1.710/µl) and only minimally elevated procalcitonin (0.08 ng/ml). This biochemical profile is atypical of the systemic inflammatory response ordinarily expected in Gram-negative bacteremia and directly reflects the immune dysregulation inherent to CAID, in which impaired neutrophil mobilization and reduced pro-inflammatory cytokine production attenuate the classical sepsis phenotype[5,6]. This observation carries important clinical implications: bacteremia should never be excluded in cirrhotic patients solely based on a near-normal WBC count or procalcitonin, as these traditional sepsis biomarkers are unreliable in this population. Prompt and protocol-driven blood culture collection at any clinical sign of suspected infection - even when fever is the sole manifestation - is essential in these patients.

<table>
<tr><th></th><th colspan="14">Hospital day</th></tr>
<tr><th></th><th colspan="6">Admission and cirrhosis management</th><th colspan="3">Bloodstream infection</th><th colspan="5">Antimicrobial therapy</th></tr>
<tr><th></th><th>0</th><th>1</th><th>2</th><th>3</th><th>5</th><th>7</th><th>10</th><th>12</th><th>14</th><th>17</th><th>19</th><th>21</th><th>24</th><th>26</th></tr>
<tr><td>Clinical events</td><td colspan="5">Admission and cirrhosis management</td><td></td><td colspan="3">Fever 39.5°C with chills
Imipenem/cilastatin initiated</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>Blood cultures</td><td colspan="7">Admission
Decompensated cirrhosis
Ascites, esophageal varices</td><td colspan="3">K. georgiana (+)
2 independent sets
MALDI-TOF MS</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td>Antimicrobial therapy</td><td colspan="7">No antibacterial therapy</td><td colspan="7">Imipenem/cilastatin 0.5 g q6h IV (2 g/day)
14-day course</td></tr>
<tr><td>WBC count (×10⁹/l)</td><td>2.6</td><td>Leukopenia</td><td></td><td></td><td></td><td></td><td>1.71 ↓</td><td></td><td></td><td>2.96</td><td></td><td></td><td></td><td>3.0</td></tr>
<tr><td>Procalcitonin (ng/ml)</td><td></td><td></td><td></td><td></td><td></td><td></td><td>0.08</td><td colspan="4">Only minimally elevated despite confirmed bacteremia (CAID)</td><td></td><td>0.1</td></tr>
<tr><td>Temperature (°C)</td><td>37</td><td></td><td></td><td></td><td></td><td></td><td>39.5°C</td><td></td><td></td><td>36.8</td><td></td><td></td><td>37.5</td><td>36.8</td></tr>
<tr><td>Hospital day</td><td colspan="7">Cirrhosis management (HD 0–9)</td><td colspan="7">Imipenem/cilastatin for 14 days (HD 10–24) → Discharge on HD 26</td></tr>
</table>

Table 1. *Clinical course, laboratory findings, and antimicrobial treatment of a 34-year-old male with decompensated cryptogenic cirrhosis and hospital-acquired Kluyvera georgiana bacteremia.*

Abbreviations: WBC, white blood cell; MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry; CAID, cirrhosis-associated immune dysfunction; IV, intravenous; HD, hospital day.

Antimicrobial susceptibility profile:

Susceptible: amikacin, gentamicin, cefepime, imipenem/cilastatin, meropenem, levofloxacin.

Resistant: ampicillin-sulbactam, ciprofloxacin, cefotaxime, ceftazidime, trimethoprim-sulfamethoxazole. Intermediate: ceftriaxone.

Characteristic	Endo et al., 2024[3]	Present case (2026)
Country	Japan	Vietnam
Age / Sex	67 years, female	34 years, male
Underlying condition	Chronic hepatitis; recurrent pancreatic cancer	Decompensated cryptogenic cirrhosis
Mechanism of immunosuppression	Cytotoxic chemotherapy (gemcitabine + nab-paclitaxel)	Cirrhosis - CAID
Clinical context of infection	Acute ascending cholangitis post-choledochojejunotomy	Hospital-acquired bacteremia; no identifiable primary focus
WBC / CRP / PCT at fever onset	WBC 4,200/μl; CRP 2.01 mg/dl	WBC 1,710/μl; PCT 0.08 ng/ml
Resistance (R)	Ampicillin; cefazolin	Ampicillin-sulbactam; ciprofloxacin; cefotaxime; ceftazidime; TMP-SMX (Intermediate: ceftriaxone)
Treatment regimen	Piperacillin/tazobactam IV → amoxicillin/ clavulanate oral	Imipenem/cilastatin IV (0.5 g q6h, 2 g/day)
Treatment duration	14 days	14 days
Outcome	Full recovery	Full recovery

Table 2. *Comparison of the two reported cases of Kluyvera georgiana bacteremia in the world literature.*
Abbreviations: CAID, cirrhosis-associated immune dysfunction; TMP-SMX, trimethoprim-sulfamethoxazole; IV, intravenous; WBC, white blood cell; CRP, C-reactive protein; PCT, procalcitonin.

Regarding antimicrobial resistance, the isolate in the first reported case demonstrated resistance only to ampicillin and cefazolin, with preserved susceptibility to piperacillin/tazobactam[3]. In contrast, the isolate in our case demonstrated considerably broader resistance, including third-generation cephalosporins (cefotaxime, ceftazidime), a fluoroquinolone (ciprofloxacin), and TMP-SMX, with intermediate susceptibility to ceftriaxone. This resistance pattern is consistent with the presence of an ESBL - producing mechanism or other complex resistance determinants, in keeping with the established evolutionary origin of CTX-M enzymes from the chromosomal β-lactamase genes of *Kluyvera spp.*[4,7]. The marked divergence in resistance phenotypes between the two reported cases underscores the need for comprehensive antimicrobial susceptibility testing for every *K. georgiana* isolate, as relying on historical susceptibility patterns for empirical de-escalation is not justifiable. In immunocompromised patients with hospital-acquired infection - those particularly with cirrhosis and CAID, early initiation of broad-spectrum empirical antibiotics, including carbapenems, represents a rational and potentially life-saving strategy, followed by targeted de-escalation once susceptibility data become available[5,6].

Although no definitive primary infectious focus was established, pyuria suggested the urinary tract as a potential portal of entry, consistent with the wider *Kluyvera spp.* literature in which urinary tract infection represents the most frequently reported clinical syndrome[3]. Additionally, spontaneous bacteremia secondary to gut bacterial translocation in the absence of an identifiable primary focus is a well-recognized and prevalent mechanism in cirrhotic patients. It may have been operative in this case. A limitation of this report is that diagnostic paracentesis for ascitic fluid analysis and culture was not performed, precluding complete exclusion of spontaneous bacterial peritonitis as a concurrent or primary infectious focus.

MALDI- TOF MS played a pivotal diagnostic role in achieving accurate species-level identification of this rare pathogen, which is phenotypically indistinguishable from other Kluyvera species by conventional biochemical methods. An identification score ≥2.0 by the Bruker Biotyper system is accepted as reliable species-level confirmation[8]. The true incidence of K. georgiana infection is likely underestimated globally: many historical reports of Kluyvera spp. infection due to not document species-level identification, and limited MALDI-TOF MS availability in resource-constrained settings may result in misidentification or failure to recognize this pathogen.

CONCLUSION

We report the second case of *Kluyvera georgiana* bacteremia in the world literature and the first from Vietnam, successfully treated with a 14-day course of intravenous imipenem/cilastatin. This case expands the recognized spectrum of at-risk populations for *K. georgiana* infection to include patients with decompensated cirrhosis and CAID, highlights the critical diagnostic value of early blood cultures when conventional infection biomarkers are attenuated, and highlights the indispensability of comprehensive antimicrobial susceptibility testing given the potentially significant variability in resistance phenotypes across *K. georgiana* strains.

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