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BACTERAEMIA DUE TO COMAMONAS KERSTERSII OF UNKNOWN ORIGIN IN A PATIENT WITH DECOMPENSATED LIVER CIRRHOSIS AND TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Comamonas kerstersii is a Gram-negative, non-fermenting bacterium previously considered non-pathogenic. Recent reports demonstrate its pathogenicity in humans, particularly in intra-abdominal infections and bacteraemia among at-risk populations.

Case description: A 44-year-old male with decompensated liver cirrhosis due to alcohol abuse and type 2 diabetes mellitus presented with fever, altered consciousness and jaundice. Blood cultures grew *C. kerstersii*, confirmed by matrix-assisted laser desorption/ionisation-time of flight mass spectrometry. The ascitic fluid culture was negative and transudative, excluding intra-abdominal infection as the source of bacteraemia. The patient was treated with ceftazidime and made a full recovery.

Conclusion: This case highlights the emerging pathogenic potential of *C. kerstersii* in immunocompromised patients, particularly those with liver cirrhosis and diabetes mellitus. Unlike previously reported cases, which were mainly associated with intra-abdominal infections, our case represents bacteraemia of unknown origin, suggesting bacterial translocation across the intestinal barrier and causing a systemic infection. Accurate identification of *C. kerstersii* by matrix-assisted laser desorption/ionisation-time of flight mass spectrometry is crucial for appropriate antibiotic therapy.

KEYWORDS

Comamonas kerstersii, bacteraemia, matrix-assisted laser desorption/ionisation-time of flight mass spectrometry, 16S rRNA gene sequencing

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LEARNING POINTS

- *Comamonas kerstersii* is an emerging opportunistic pathogen in immunocompromised patients, particularly those with liver cirrhosis or diabetes, who present with bacteraemia of unknown origin after the exclusion of common pathogens.
- Matrix-assisted laser desorption/ionisation-time of flight mass spectrometry enables precise identification of *C. kerstersii*. Conventional automated systems may misidentify it as *C. testosteroni*, potentially leading to inappropriate therapy and missed diagnoses.
- In decompensated liver cirrhosis, *C. kerstersii* bacteraemia may result from bacterial translocation across the impaired intestinal barrier, rather than typical intra-abdominal sources, highlighting a novel pathogenic mechanism.

INTRODUCTION

Comamonas kerstersii is a Gram-negative, non-fermenting, oxidase-positive, catalase-positive bacterium belonging to the β-proteobacteria family *Comamonadaceae*. The species was named in honour of Belgian microbiologist Karel Kersters for his contributions to bacterial taxonomy[1].

Prior to 2013, *C. kerstersii* was considered non-pathogenic to humans. That year, Almuzara et al. reported the first four cases of human intra-abdominal infections caused by this microbe[2]. In 2014, the first case of *C. kerstersii* bacteraemia was reported in a 65-year-old patient with colonic diverticulosis, with matrix-assisted laser desorption/ionisation-time of flight mass spectrometry (MALDI-TOF MS), which is a mass spectrometry technique that can rapidly and accurately identify the protein 'fingerprints' of microbes from a database – facilitating rapid and accurate bacterial identification[3]. Since then, an increasing number of human infections attributed to *C. kerstersii* have been documented. Most clinical isolates have been obtained from peritoneal fluid, with bacteraemia remaining rare. The majority of cases have been associated with intra-abdominal infections such as appendicitis, diverticulitis and peritonitis[4-6].

However, more recent reports have demonstrated that *C. kerstersii* can also cause extra- abdominal infections including pneumonia, urinary tract infections and psoas muscle abscesses. Notably, some strains have exhibited resistance to third- and fourth-generation cephalosporins.

Here, we report a case of *C. kerstersii* bacteraemia of unknown origin in a patient with liver cirrhosis and diabetes mellitus.

CASE DESCRIPTION

A 44-year-old male with a history of decompensated liver cirrhosis secondary to chronic alcohol abuse, type 2 diabetes mellitus (on insulin therapy for five years), and multiple episodes of oesophageal variceal bleeding (most recently one month prior to admission) presented to the emergency department with a one-week history of progressive fatigue, poor oral intake and abdominal distension.

The patient initially developed a high-grade fever with a maximum temperature of 39°C. In the evening of day 1 (19 August) he became lethargic with an altered mental status and was brought to our emergency department by family members.

The physical examination on admission (day 1) showed the following vital signs: pulse 90 bpm, blood pressure 91/60 mmHg, temperature 39.3°C, Glasgow Coma Scale score 14–15. The patient had icteric skin and sclera, no palpable hepatomegaly, but splenomegaly was present with a soft abdomen showing moderate ascites and diminished breath sounds accompanied by bilateral basal crackles. Oxygen saturation (SpO_2) was 94% on 2 l/min oxygen via a nasal cannula. No focal neurological deficits were observed. Initial blood tests revealed normal white blood cell count, C-reactive protein and procalcitonin *(Table 1)*.

Blood cultures obtained on day 1 yielded Gram-negative rods after 24 hours of incubation *(Fig. 1)*. The organism was identified as *C. kerstersii* by MALDI-TOF MS. Antimicrobial susceptibility testing showed sensitivity to ceftazidime, cefepime, amikacin, gentamicin and ciprofloxacin, with ciprofloxacin resistance noted. Repeat blood cultures performed on day 4 and day 7 were negative.

Ascitic fluid analysis (day 1) showed the fluid as yellow and clear, with a white blood cell count of 200/µl (30% neutrophils, 60% lymphocytes, 10% epithelial cells), protein

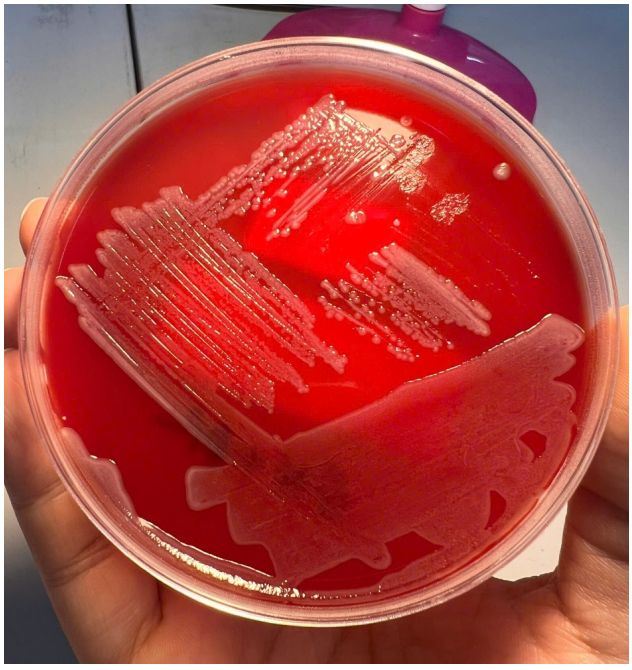


Figure 1. C. kerstersii colonies are circular, moist and white, growing on blood agar (the colonies appear smooth, with a glistening surface and well-defined edges, typically measuring 1–2 mm in diameter after 24 hours of incubation at 37°C).

Peripheral blood laboratory		
Parameter	Result	Normal range
White blood cells (×10⁹/l)	4.6	4–10
Neutrophils (%)	76.5	50–70
Haemoglobin (g/l)	112	120–160
Platelets (×10⁹/l)	49	150–450
Prothrombin time (%)	50	70–140
International normalised ratio	1.6	0.8–1.2
Total bilirubin (µmol/l)	202	≤21
Direct bilirubin (µmol/l)	132.4	0–8
Aspartate aminotransferase (U/l)	242	8–33
Alanine transaminase (U/l)	86	4–36
Albumin (g/l)	29.1	35–54
Creatinine (µmol/l)	65	44–120
Ammonia (µmol/l)	78.8	18–72
Glucose (mmol/l)	30.8	4.1–5.9
CRP (mg/l)	17.7	<5
Procalcitonin (mg/l)	1.33	<0.25
Microbiology		
Type	Result	Notes
Blood culture D1	Comamonas kerstersii	Gram-negative, growth after 24 hours
Antibiotic sensitivity	Sensitive to ceftazidime, cefepime, amikacin, ciprofloxacin, gentamicin	Resistant to ciprofloxacin
Blood culture D4, D7	Negative	
Imaging		
Type	Result	
Chest X-ray	Bilateral pneumonia	
Abdominal ultrasound	Moderate ascites, liver cirrhosis, portal hypertension	
Treatment course and follow-up		
Time	Treatment	Clinical course
D1	Ceftazidime 2 g every 12 hours Oxygen therapy Rifaximin 550 mg Aminoleban® Insulin	Fever, altered consciousness
D2	Continue treatment Procalcitonin 2.34 ng/ml	Fever resolved
D6	Continue treatment	Mental status improved (GCS 15)
D9	Continue treatment	Room air breathing, SpO_2 93%
D12	Continue treatment Procalcitonin: 1.68 ng/ml	Stable
D14	Continue treatment	Antibiotic discontinued
D15-22	Symptomatic treatment for cirrhosis	
D23	Discharge	Stable
Treatment summary		
Criteria	Result	Criteria
Treatment duration	14 days of ceftazidime	Treatment duration
Hospital stay	23 days	Hospital stay
Treatment response	Good – fever resolved within 48 hours	Treatment response
Discharge condition	Stable, inflammatory markers normalised	Discharge condition
Remaining issues	Elevated liver enzymes, high bilirubin, low albumin (due to decompensated cirrhosis)	Remaining issues
Follow-up care	Management of liver disease and diabetes	Follow-up care

Table 1. Peripheral blood laboratory, microbiology, imaging characteristics and treatment course, follow-up.

6.1 g/l, a negative Rivalta test and sterile bacterial culture.

A chest X-ray demonstrated bilateral pneumonia; abdominal ultrasound revealed moderate ascites with features of cirrhosis and portal hypertension.

The patient was initiated on empirical antibiotic therapy with ceftazidime 2 g every 12 hours. Supportive management included oxygen supplementation, rifaximin 550 mg twice daily, Aminoleban® for hepatic encephalopathy and insulin for glycaemic control.

Within 48 hours of treatment, the patient showed marked clinical improvement. Fever resolved by day 2 of treatment, mental status returned to normal (Glasgow Coma Scale 15), and oxygen requirements decreased to 1 l/min. By day 9, he was breathing room air with SpO_2 of 93%.

The patient completed a 14-day course of ceftazidime and was discharged on day 23 in a stable condition. At discharge, inflammatory markers were within normal limits; however, liver enzymes, bilirubin and serum albumin remained abnormal, consistent with decompensated alcoholic cirrhosis. He continues to receive follow-up care for liver disease and diabetes management.

DISCUSSION

In 2014, Opota et al. reported the first case of *C. kerstersii* bacteraemia[3]. Since then, most published studies on *C. kerstersii* infections have documented intra-abdominal involvement including appendicitis, diverticulitis, perforated appendicitis and colonic perforation, with bacteraemia being reported only rarely. When present, bacteraemia typically occurs secondary to peritonitis.

Through a systematic literature review, we identified seven previously reported cases of *C. kerstersii* bacteraemia. Most cases occurred in patients with acute abdominal conditions, including appendicitis and perforated appendicitis (four cases), diverticular disease (one case), colonic perforation (one case), intrauterine infection following caesarean section (one case), and one case of unknown origin *(Table 2)*. Our patient represents the eighth reported case and the second case of *C. kerstersii* bacteraemia of unknown origin, with the previous case occurring in an 82-year-old male with diabetes mellitus[7].

Beyond intra-abdominal infections, several recent reports have described *C. kerstersii* causing pneumonia, urinary tract infections and psoas muscle abscesses, demonstrating its expanding pathogenic potential. These cases suggest a trend from predominantly intra-abdominal towards more diverse systemic manifestations. In 2022, Liu et al. compiled 26 cases of *C. kerstersii* infection, of which 19 (73%) originated from Argentina. The most common sites of isolation were peritoneal fluid (80.8%), blood (11.5%), urine (3.8%) and psoas muscle abscess (3.8%). Predisposing conditions included perforated appendicitis (30.8%), appendicitis (34.6%), colonic perforation (15.4%) and peritonitis (50%). All isolates were identified by MALDI-TOF MS[5]. More recently, Wang et al. reported cases of *C. kerstersii* pneumonia in a patient with traumatic brain injury and a secondary urinary tract infection in a patient with benign prostatic hyperplasia, further expanding the spectrum of extra-abdominal infections[8]. Our patient had risk factors of decompensated liver cirrhosis and type 2 diabetes mellitus, both representing immunocompromised conditions that predispose to bacteraemia even with uncommon opportunistic pathogens. This differs from most previously reported cases of *C. kerstersii* infection, which have predominantly involved patients with inflammatory bowel lesions, intestinal perforation or peritonitis.

In this case, ascitic fluid aspiration and bacterial culture were performed, yielding negative results and thereby excluding intra-abdominal infection, including both primary and secondary peritonitis. Moreover, the ascitic fluid was transudative, with an albumin concentration of 29.1 g/l and a cell count of 200/ml, findings consistent with decompensated cirrhosis. These results suggest that bacteraemia may have arisen via direct bacterial translocation across the intestinal barrier. In cirrhosis, portal hypertension and immune dysfunction facilitate the passage of bacteria through the intestinal mucosa into the bloodstream. Diabetes comorbidity further amplifies this risk, as hyperglycaemia impairs neutrophil function and cellular immunity.

Multiple reports emphasise that automated bacterial identification systems such as Vitek and API may misidentify *C. kerstersii* as *C. testosteroni*, potentially leading to underrecognition of *C. kerstersii* infections in the past. Accurate identification of *C. kerstersii* requires advanced techniques such as MALDI-TOF MS or 16S rRNA gene sequencing. In our case, MALDI-TOF MS enabled rapid and reliable species-level identification, thereby guiding appropriate antimicrobial therapy. By analysing ribosomal protein mass spectra, MALDI-TOF MS allows precise identification of species within the Comamonas genus, which is critical given the potential for misclassification.

In our patient, *C. kerstersii* was susceptible to carbapenems and cephalosporins. Treatment with ceftazidime led to clinical improvement and recovery. Previous studies have similarly reported broad susceptibility of *C. kerstersii* to carbapenems, third- and fourth-generation cephalosporins, certain fluoroquinolones and aminoglycosides. However, emerging resistance has been described in association with novel class A β-lactamase genes, highlighting the potential for increasing antimicrobial resistance. Given the growing recognition of *C. kerstersii* as an opportunistic pathogen, ongoing surveillance of resistance patterns and rational antibiotic use are essential to prevent further resistance development.

The optimal duration of antibiotic therapy for *C. kerstersii* infections remains undefined due to the limited number of reported cases. In our patient, a 14-day course of ceftazidime resulted in favourable outcomes, with marked clinical improvement observed within the first 48 hours. Consistent with previous reports, most *C. kerstersii* infection cases have shown a favourable prognosis when treated promptly and appropriately.

Table 2. Case reports of bacteraemia caused by C. kerstersii worldwide.

Age/ sex	Blood culture	Other cultures	Comorbidity	Predisposing conditions	Clinical presentation	Identification method	Antibiotic sensitivities	Main antibiotic treatment	Outcome	Reference
65/M	*C. kerstersii and Bacteroides fragilis*	None reported	Diabetic	Diverticulosis	Sudden onset of fever, chills, vomiting, diarrhoea	MALDI-TOF MS	R to SMZ/TMP and CIP	IMP	Recovered	Opota et al.[3]
31/M	*C. kerstersii*	None reported	None	Acute peritonitis and perforated appendix with abdominal abscess	Right lower abdominal pain, temperature of 39°C	MALDI-TOF MS	R to CIP, T/S and LVX	CXM, MNZ	Recovered	Zhou et al.[4]
62/M	*C. kerstersii*	None reported	None	Intra-abdominal infection due to perforated colon	Abdominal pain	MALDI-TOF MS	R to CAZ and COL	TZP	Recovered	Liu et al.[5]
16/M	*C. kerstersii*	None reported	None	Appendicitis	Abdominal pain, fever, nausea, vomiting	MALDI-TOF MS	I to AMK	SAM, MNZ, TZP	Recovered	Palacio et al.[6]
82/M	*C. kerstersii*	None reported	Type 2 diabetes, diabetic neuropathy, dyslipidaemia, colon tubular adenoma, tubulovillous adenoma	bacteraemia of unknown origin	Chills, rigours, constipation, fever	MALDI-TOF MS	R to CIP	TZP, CRO, AMC	Recovered	Rong et al.[7]
29/F	*C. kerstersii*	Posterior fornix puncture fluid: C. kerstersii	None	Intrauterine infection following caesarean section	Chills, shortness of breath, intermittent tingling on both sides of the lower abdomen, and abdominal distension temperature of 40°C	MALDI-TOF MS and metagenomic next-generation sequencing (mNGS)	R to CIP and SMZ/TMP	MEM	Recovered	Qu et al.[9]
28/M	*C. kerstersii*	None reported	None	Localised peritonitis perforated appendix	Abdominal pain and distension	MALDI-TOF MS and 16S rRNA gene sequencing	R to CIP and LVX	LVX, CRO	Recovered	Zhang et al.[10]
65/M	*C. kerstersii*	Ascites: negative	Cirrhosis, diabetes	Bacteraemia of unknown origin	Fever and fatigue	MALDI-TOF MS	R to FEP, AMK, CIP and GEN	CAZ	Recovered	Present case

Abbreviations: R, resistant; I, intermediate; SMZ/TMP, trimethoprim/sulfamethoxazole; CIP, ciprofloxacin; LVX, levofloxacin; CAZ, ceftazidim; CRO, ceftriaxone; COL, colistin; FEP, cefepime; AMK, amikacin; GEN, gentamicin; AMC, amoxicillin/clavulanic acid; MNZ, metronidazole; IMP, imipenem/cilastatin; MEM, meropenem; CXM, cefuroxime; TZP, piperacillin/tazobactam; SAM, ampicillin/sulbactam.

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The present case expands existing understanding of *C. kerstersii* as a potential cause of bacteraemia in immunocompromised patients. Clinically, in patients with cirrhosis presenting with bacteraemia of unknown origin, *C. kerstersii* should be considered in the differential diagnosis, particularly when more common pathogens have been excluded. Accurate identification using MALDI-TOF MS is crucial, as conventional automated systems may misidentify this microbe as *C. testosteroni*.

This report has some limitations. The source of bacteraemia could not be determined despite the comprehensive and systematic diagnostic evaluation. Long-term follow-up of this patient was limited, leaving the risk of recurrent infection in this immunocompromised patient uncertain. Furthermore, due to practical constraints, we could not perform 16S rRNA gene sequencing or genome analysis to characterise specific virulence factors of this isolate.

CONCLUSION

This case expands the clinical spectrum of *C. kerstersii* infections and highlights the importance of considering this emerging pathogen in immunocompromised patients presenting with bacteraemia of unknown origin. MALDI-TOF MS plays a critical role in the accurate identification of *C. kerstersii*, enabling distinction from other species within the Comamonas genus. This precise identification is essential to guide appropriate antibiotic therapy and achieve favourable clinical outcomes. Further research is warranted to elucidate the pathogenic mechanisms, define the optimal duration of antimicrobial therapy and assess the long-term prognosis of infections caused by this emerging pathogen.

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