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Presumed Bergeyella zoohelcum Bacteremia of Uncertain Source in a Patient With Cirrhosis and Concurrent Lower-Extremity Cellulitis

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Abstract

We report presumed *Bergeyella zoohelcum* bacteremia in a man in his early 70s with primary sclerosing cholangitis (PSC)-associated cirrhosis, prior transjugular intrahepatic portosystemic shunt (TIPS) placement, ulcerative colitis, and Parkinson disease. He presented after a ground-level fall and was concurrently found to have left lower-extremity cellulitis. Gram-negative rods were recovered from the aerobic bottles of two independently collected peripheral blood-culture sets and were reported by the clinical microbiology laboratory as consistent with *B. zoohelcum*. The specific species-identification method could not be retrieved, and repeat blood cultures showed no growth. The patient had seven household cats but reported no recent animal bite or scratch. Although the cellulitis was considered a possible site of entry, the wound culture yielded mixed organisms without establishing *B. zoohelcum* at the skin site, and the source of bacteremia remained unconfirmed. He completed a 10-day course of ceftriaxone 2 g intravenously once daily with clinical improvement and persistently negative repeat blood cultures. This case highlights the importance of matching diagnostic certainty to the available microbiologic evidence and distinguishing epidemiologic exposure from a microbiologically confirmed source of infection.

Categories: Internal Medicine, Infectious Disease, Dermatology
Keywords: bacteremia, bergeyella zoohelcum, cellulitis, cirrhosis, gram-negative bacteremia, rare bacterial infection, zoonotic infection

Introduction

Bergeyella zoohelcum, formerly known as *Weeksella zoohelcum* and historically designated Centers for Disease Control and Prevention (CDC) group IIj, is a non-motile, aerobic, Gram-negative rod that was reassigned to the genus *Bergeyella* in 1994 [1]. The organism has been identified in the oral and upper respiratory flora of dogs and cats [2,3]. Human infection is rare, with published literature consisting predominantly of isolated case reports and small case series [3,4].

Animal exposure has been reported in several human infections. Direct inoculation following dog bites has been described, whereas other cases have occurred after close animal contact without a recognized bite [5-7]. A foodborne infection following ingestion of a dish prepared with goat blood has also been reported [8]. Reported clinical manifestations include cellulitis, abscess formation, tenosynovitis, bacteremia, pneumonia, meningitis, and infective endocarditis [3,5,6,9]. Although animal exposure is epidemiologically relevant, its presence alone does not establish the route or source of infection in an individual patient.

Identification of *B. zoohelcum* may be challenging because uncommon Gram-negative organisms can be misidentified by conventional methods. In published reports, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and 16S ribosomal RNA gene sequencing have been used to improve species-level identification [3]. Invasive infection has been described in patients with underlying conditions including cirrhosis, malignancy receiving chemotherapy, acquired immunodeficiency syndrome (AIDS), and end-stage renal disease (ESRD) [3,6,7]; however, the limited number of reported cases does not establish an organism-specific risk profile. Cirrhosis is independently associated with clinically important immune dysfunction and increased susceptibility to bacterial infection [10,11].

We report bacteremia due to a Gram-negative organism reported by the clinical microbiology laboratory as consistent with *B. zoohelcum* in a man in his early 70s with primary sclerosing cholangitis (PSC)-associated cirrhosis and concurrent left lower-extremity cellulitis. He had seven household cats but reported no recent animal bite or scratch. The cellulitis was considered a possible, but unproven, site of entry; the wound culture yielded mixed organisms, and the source of bacteremia remained unconfirmed.

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Case Presentation

A man in his early 70s with PSC-associated cirrhosis and portal hypertension, with prior transjugular intrahepatic portosystemic shunt (TIPS) placement, ulcerative colitis treated with sulfasalazine, and Parkinson's disease presented to the emergency department after a ground-level fall with head strike. He reported progressive difficulty with ambulation before the fall and denied loss of consciousness. On presentation, he was disoriented to place and time. Physical examination was notable for Parkinsonian tremor and rigidity and erythema, warmth, tenderness, and edema of the left lower extremity (LLE) consistent with cellulitis (Figure *1*). The admission examination documented no discrete ulcer, laceration, draining lesion, interdigital fissure, or other disruption of the skin barrier.



FIGURE 1: Left lower-extremity erythema and edema at presentation

He was afebrile at 36.8°C, with a heart rate of 114 beats/min, blood pressure of 136/73 mmHg, respiratory rate of 16 breaths/min, and oxygen saturation of 97% on room air. Initial laboratory evaluation showed a white blood cell count of 13.3 × 10^3/µL (reference range: 3.1-8.5 × 10^3/µL) and thrombocytopenia with a platelet count of 79 × 10^3/µL (reference range: 140-440 × 10^3/µL). Serum lactate was 1.2 mmol/L (reference range: 0.5-2.0 mmol/L), creatinine was 1.0 mg/dL (reference range: 0.7-1.3 mg/dL), and the estimated glomerular filtration rate was 80 mL/min/1.73 m^2 (reference: >60 mL/min/1.73 m^2).

Given his PSC-associated cirrhosis, the admission hepatic biochemical profile was also reviewed. Serum albumin was 2.4 g/dL (reference range: 3.5-5.0 g/dL), total bilirubin was 1.1 mg/dL (reference range: 0.2-1.2 mg/dL), alkaline phosphatase was 127 U/L (reference range: 44-147 U/L), aspartate aminotransferase was 41 U/L (reference range: 10-40 U/L), alanine aminotransferase was <7 U/L (reference range: 7-56 U/L), and the international normalized ratio was 1.6 (range: 0.8-1.2 in patients not receiving anticoagulation). No clinical diagnosis of acute cholangitis was documented during the admission.

Urinalysis showed cloudy, dark-yellow urine with trace leukocyte esterase (reference: negative), 0-5 white blood cells per high-power field (reference range: 0-5/high-power field), 6-10 red blood cells per high-power field (reference range: 0-2/high-power field), negative nitrites (reference: negative), and no bacteria (reference: none detected). The urine culture yielded mixed organisms that were interpreted by the laboratory as collection contaminants.

Noncontrast CT of the head demonstrated a small left parietal intraparenchymal hematoma measuring 1.0 × 0.5 × 1.1 cm with minimal local mass effect and no midline shift. Repeat imaging showed no significant

progression, and the hemorrhage was managed conservatively after neurosurgical evaluation.

CT of the abdomen and pelvis demonstrated cirrhotic hepatic morphology with TIPS-related changes and prominent intrahepatic bile ducts that were grossly stable compared with prior imaging. The gallbladder was contracted but otherwise unremarkable. Mild radiographic proctitis and a diffusely enlarged heterogeneous prostate were also noted. There was no hydronephrosis, obstructing urinary calculus, free intraperitoneal fluid, or free air. These findings were considered during evaluation for potential alternative sources of bacteremia but did not establish a hepatobiliary, gastrointestinal, urinary, or other intra-abdominal source.

Two peripheral blood-culture sets were collected, one from the left arm at 21:46 and the other from the right arm at 21:50. Gram-negative rods were recovered from the aerobic bottle of each set. A multiplex blood-culture identification panel performed on the right-arm aerobic bottle did not detect any of its included bacterial or fungal targets; *B. zoohelcum* was not included among the organisms tested by this panel. The accompanying laboratory narrative stated that subculture was performed for species identification and susceptibility testing. The final blood-culture reports subsequently described the isolates as "consistent with *B. zoohelcum*."

The specific method used for species identification after subculture could not be retrieved from the available record. Therefore, it could not be determined whether matrix-assisted laser desorption/ionization time-of-flight mass spectrometry, biochemical identification, sequencing, or another method had been used. The antimicrobial susceptibility testing method, minimum inhibitory concentrations, and interpretive criteria were likewise unavailable. Accordingly, the species-level identification was considered presumptive rather than independently confirmed.

Two repeat peripheral blood cultures obtained approximately 37 hours after the initial cultures, from the right antecubital region and right hand, remained negative through 120 hours of incubation. A specimen documented as a wound culture yielded mixed organisms and did not establish *B. zoohelcum* at the skin site. The exact site of collection, specimen type, and whether the specimen represented a superficial swab or deeper sample could not be determined from the retrieved record.

The exposure history included seven household cats and frequent gardening. The patient denied any recent animal bite or scratch, and no licking of compromised skin was documented. Neither the household animals nor environmental samples were microbiologically tested. Household cat exposure, therefore, represented an epidemiologic association rather than evidence of confirmed zoonotic transmission. The concurrent left lower-extremity cellulitis was considered a possible, but unproven, site of entry.

No transthoracic or transesophageal echocardiogram was identified in the available record. Repeat blood cultures cleared, no recurrent fever was documented, and subsequent cardiovascular examination documented no cardiac murmur. Infective endocarditis was not documented as a clinical diagnosis.

Empiric IV cefazolin was initiated for the cellulitis and was subsequently changed to ceftriaxone after the blood-culture results became available. The exact duration and number of cefazolin doses could not be reconstructed from the available record. The patient subsequently received ceftriaxone 2 g intravenously once daily for a total of 10 days. No infectious-diseases consultation guiding antimicrobial selection or duration was identified in the retrieved record. Because isolate-specific susceptibility data were unavailable, the clinical response to ceftriaxone was not interpreted as evidence of documented in vitro susceptibility.

The leukocytosis resolved, repeat blood cultures remained negative, and no recurrent signs of systemic infection were documented. The patient remained hemodynamically stable and was transferred to inpatient rehabilitation, where he completed ceftriaxone therapy. During rehabilitation, he remained afebrile and denied abdominal pain or urinary frequency or urgency. At subsequent follow-up, his ulcerative colitis was clinically well controlled, with formed stools and no abdominal pain, urgency, diarrhea, or rectal bleeding.

Discussion

Microbiology and identification

B. zoohelcum is a fastidious Gram-negative rod that may be difficult to identify using conventional microbiological methods. Published isolates have been described as slow-growing and as producing mucoid or tan-pigmented colonies on blood or chocolate agar, with oxidase, catalase, indole, and urease positivity [3]. Because uncommon Gram-negative organisms may be misidentified by conventional phenotypic methods, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and 16S ribosomal RNA gene sequencing have been used in published reports to improve species-level identification [3].

In the present case, Gram-negative rods were recovered from the aerobic bottles of two independently collected peripheral blood-culture sets, and the final laboratory report for each described the isolate as "consistent with *B. zoohelcum*." A multiplex blood-culture identification panel performed on the right-arm aerobic bottle did not detect any of its included targets; *B. zoohelcum* was not included among the organisms

tested by that panel. The laboratory documentation stated that subculture was performed for species identification and susceptibility testing, but the specific post-subculture identification method could not be retrieved. The concordant findings from two independently collected cultures support true bacteremia; however, they do not establish independently verified species-level identification. Accordingly, the organism is regarded as presumptively identified as *B. zoohelcum* in this report.

Epidemiology and Clinical Spectrum

Human infection attributed to *B. zoohelcum* is rare, and the published literature consists predominantly of isolated case reports and small case series [3,4]. Reported clinical manifestations include cellulitis, abscess formation, tenosynovitis, bacteremia, pneumonia, meningitis, and infective endocarditis [3-9,12,13]. Selected illustrative reports are summarized in Table *1*.

Author (Year)	Age/Sex	Exposure	Key Comorbidities	Presentation	Treatment	Outcome
Montejo et al. (2001) [5]	33/M	Dog bite	None	Cellulitis, bacteremia	Amoxicillin-clavulanate	Recovered
Yi et al. (2016) [12]	22-month-old/M	Dog bite	None	Cellulitis, abscess	Amoxicillin → ceftriaxone + clindamycin → amoxicillin-clavulanate; 10 days total	Recovered
Lin et al. (2007) [6]	73/M	Dog contact, no bite	Liver cirrhosis, HCV, bladder carcinoma	LLE cellulitis, bacteremia	Cefazolin + gentamicin for 14 days	Recovered
Kivinen et al. (2003) [13]	77/F	Cat contact	Diabetes mellitus, chronic corticosteroid therapy, multiple comorbidities	Cellulitis, bacteremia	Cefuroxime → cephalexin	Recovered
Beltran et al. (2006) [8]	44/F	Ingestion of a dish prepared with goat blood	None	Diarrhea, bacteremia	Ciprofloxacin, initially IV then oral	Recovered
Chen et al. (2017) [9]	27/F	No mammalian exposure documented	None reported	Bacteremia, infective endocarditis	Cefuroxime for 6 weeks + valve surgery	Recovered
Sharma et al. (2019) [7]	43/transgender woman	Therapy dog contact	AIDS, ESRD, heart failure	Bacteremia	Meropenem + levofloxacin → oral levofloxacin; 10 days total	Recovered
Grams et al. (2023) [3]	69/M	Household cats, no bite	Metastatic prostate cancer on chemotherapy, bilateral lower-extremity lymphedema	Cellulitis, bacteremia, septic shock	Ampicillin-sulbactam → ciprofloxacin	Recovered
Present case	Early 70s/M	Seven household cats; no reported bite or scratch	PSC-associated cirrhosis	LLE cellulitis, presumed B. zoohelcum bacteremia; source unconfirmed	Cefazolin → ceftriaxone 2 g IV once daily for 10 days	Clinical improvement; repeat blood cultures negative

TABLE 1: Selected illustrative published cases of human infection attributed to B. zoohelcum

AIDS: Acquired immunodeficiency syndrome; ESRD: End-stage renal disease; F: Female; HCV: Hepatitis C virus; LLE: Left lower extremity; M: Male; PSC: Primary sclerosing cholangitis

These cases were selected from publications cited in this manuscript to demonstrate the range of reported exposures, host characteristics, clinical presentations, and antimicrobial treatments; the table is not intended to represent a systematic or exhaustive review.

Animal exposure is a recurring epidemiologic feature, although the nature of that exposure varies. Direct inoculation after dog bites has been reported by Montejo et al. and Yi et al. [5,12]. Lin et al. described cellulitis and bacteremia in a patient with cirrhosis who had dog exposure without a recognized bite [6]. Kivinen et al. described septicemia associated with severe skin infection in a patient with cat exposure [13], whereas Sharma et al. reported bacteremia following close contact with a therapy dog [7]. Grams et al. described bacteremia, cellulitis, and septic shock in an immunocompromised patient with household cat

exposure and no reported bite [3]. However, animal exposure has not been documented in all reported cases. Chen et al. described *B. zoohelcum* bacteremia associated with infective endocarditis without documented mammalian exposure, while Beltran et al. reported bacteremia following ingestion of a dish prepared with goat blood [8,9]. These reports demonstrate heterogeneity in the epidemiologic settings associated with infection and caution against assigning a route of acquisition solely based on animal contact.

Assessment of the Possible Site of Entry

Determining the source of bacteremia was the principal diagnostic challenge in this case. The concurrent left lower-extremity cellulitis was considered a possible, but unproven, site of entry. The admission examination documented no discrete ulcer, laceration, draining lesion, interdigital fissure, or other disruption of the skin barrier. Although the record subsequently referenced a wound culture, the exact collection site, specimen type, and whether the specimen represented a superficial swab or deeper sample could not be determined. The culture yielded mixed organisms and did not establish *B. zoohelcum* at the skin site.

The patient had seven household cats but reported no recent bite or scratch, and no licking of compromised skin was documented. Neither the animals nor environmental samples were microbiologically tested. Household cat exposure, therefore, represents an epidemiologic association rather than evidence of confirmed zoonotic transmission. Similarly, although the patient reported frequent gardening, no environmental cultures or other microbiological evidence linked gardening or soil exposure to the bloodstream isolate. Gardening could therefore not be established as a route of acquisition.

Alternative potential sources were also evaluated. The urine culture yielded mixed organisms that were interpreted by the laboratory as collection contaminants, providing no microbiologically established urinary source. Abdominal imaging demonstrated mild proctitis, but no clinical or microbiological evidence established a gastrointestinal source. The patient also had PSC-associated cirrhosis with prominent intrahepatic bile ducts; however, these findings were grossly stable compared with prior imaging, the gallbladder was otherwise unremarkable, total bilirubin was 1.1 mg/dL, and no clinical diagnosis of acute cholangitis was documented. These findings do not exclude all possible urinary, gastrointestinal, hepatobiliary, or intra-abdominal sources but support classifying the source of bacteremia as unconfirmed.

Host Susceptibility

Cirrhosis is associated with clinically important immune dysfunction and increased susceptibility to bacterial infection. Cirrhosis-associated immune dysfunction includes abnormalities in innate immune responses, impaired phagocytic function, altered complement activity, reduced hepatic bacterial clearance, and disruption of intestinal barrier integrity [10,11].

Lin et al. also reported *B. zoohelcum* bacteremia with lower-extremity cellulitis in a patient with cirrhosis [6]. Nevertheless, the limited number of published infections does not establish cirrhosis as an organism-specific risk factor for *B. zoohelcum*. In the present patient, cirrhosis is therefore best regarded as a biologically plausible contributor to general susceptibility to bacterial infection rather than a demonstrated predisposing factor for this particular organism.

The patient's prior TIPS placement, ulcerative colitis, and Parkinson disease were relevant components of his medical history, but none has been established as an organism-specific risk factor for *B. zoohelcum* infection. Available evidence does not demonstrate that TIPS placement specifically increases susceptibility to this organism. Similarly, ulcerative colitis treated with sulfasalazine without systemic immunosuppressive therapy and Parkinson disease should not be characterized as established immunocompromising conditions in this case. These comorbidities therefore were not used to infer a specific mechanism of acquisition.

Antimicrobial Therapy

Dedicated species-specific antimicrobial susceptibility breakpoints for *B. zoohelcum* have not been established. More broadly, available guidance for members of the order Flavobacteriales groups susceptibility interpretation with that of other less commonly encountered Gram-negative organisms, and the clinical implications of applying such broader interpretive frameworks to individual uncommon species remain uncertain [14]. The available evidence for *B. zoohelcum* is limited primarily to case reports and small numbers of clinical isolates.

Susceptibility to several β-lactams and fluoroquinolones has been reported among published *B. zoohelcum* isolates, whereas resistance to colistin and polymyxin B has also been described [3,14]. Given the small and heterogeneous evidence base, these observations should not be interpreted as establishing a uniform susceptibility profile for the species. No standardized antimicrobial regimen or treatment duration has been established.

Previously reported treatment regimens have included amoxicillin-clavulanate, ampicillin-sulbactam, cefazolin, cefuroxime, ceftriaxone, ciprofloxacin, and other agents, with treatment duration determined by the clinical syndrome [3,5-9,12,13]. In the present case, empiric cefazolin was changed to ceftriaxone after the blood-culture results became available. After transition to ceftriaxone 2 g intravenously once daily, the patient completed 10 days of ceftriaxone therapy with clinical improvement, resolution of leukocytosis, and persistently negative repeat blood cultures. The exact antimicrobial susceptibility testing method, minimum inhibitory concentrations, and interpretive criteria for this isolate could not be retrieved. Isolate-specific susceptibility to ceftriaxone therefore could not be independently verified, and the favorable clinical course represents an observed treatment outcome rather than documented in vitro susceptibility.

Clinical Significance and Limitations

This case describes bacteremia due to an organism reported by the clinical microbiology laboratory as consistent with *B. zoohelcum* in two independently collected peripheral blood-culture sets in a patient with cirrhosis and concurrent lower-extremity cellulitis. The concordant culture findings support true bacteremia, but the unavailable species-identification method limits the certainty of species-level attribution.

Several additional limitations should be acknowledged. The exact post-subculture identification method and antimicrobial susceptibility testing methodology could not be retrieved. The precise source and type of the wound-culture specimen were unavailable, and the culture did not establish *B. zoohelcum* at the skin site. The household animals and environment were not tested. Exact blood-culture time to positivity and the temporal relationship between blood-culture collection and the first cefazolin dose could not be established from the available record. No transthoracic or transesophageal echocardiogram was identified, although repeat blood cultures cleared, recurrent fever was not documented, and subsequent cardiovascular examination documented no murmur. Consequently, this case does not establish definitive species-level confirmation, a microbiologically proven skin portal of entry, non-bite zoonotic transmission, or a causal relationship between prior TIPS placement and infection.

Conclusions

An organism reported by the clinical microbiology laboratory as consistent with *B. zoohelcum* was recovered from two independently collected peripheral blood-culture sets in the setting of concurrent left lower-extremity cellulitis. Although household cat exposure and cellulitis were considered possible clues to the source of infection, the patient reported no recent bite or scratch, and the wound culture yielded mixed organisms without establishing *B. zoohelcum* at the skin site. The portal of entry, therefore, remained unconfirmed. The patient completed a 10-day course of IV ceftriaxone with clinical improvement and persistently negative repeat blood cultures. This case emphasizes the importance of matching diagnostic certainty to the available microbiologic evidence and distinguishing epidemiologic exposure from a microbiologically confirmed source of infection.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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