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Campylobacter jejuni Bacteremia Following Acute Enteritis in an Older Adult With Diabetes Mellitus: A Case Report

Sayako Tanaka [1], Kanata Tonosaki [1], Eno Tanaka [1], Takumi Chiba [1], Ayumu Suzuki [1], Ryuichi Ohta [2], Mori Nakai [1]

1. Department of General Medicine, Towada City Hospital, Towada, JPN 2. Community Care, Unnan City Hospital, Unnan, JPN

Corresponding author: Ryuichi Ohta, ryuichiohta0120@gmail.com

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Abstract

Campylobacter jejuni is a common cause of bacterial gastroenteritis, whereas bloodstream infection is uncommon and occurs predominantly in patients of advanced age or with underlying medical conditions. We report a 71-year-old man with type 2 diabetes mellitus who developed fever, altered mental status, vomiting, profuse watery diarrhea, and hypoxemic respiratory failure after consuming inadequately cooked chicken gizzards. Computed tomography demonstrated edema of the ascending colon without evidence of pneumonia or pulmonary embolism. *C. jejuni* was subsequently isolated from both stool and blood cultures, establishing the diagnosis of *Campylobacter* enteritis complicated by bacteremia. The patient initially received empiric meropenem because of severe systemic illness and was subsequently managed according to microbiological identification and antimicrobial susceptibility results, with eventual transition to oral azithromycin. His clinical condition gradually improved, and he was discharged without recurrence.

This case illustrates that *C. jejuni* enteritis, although usually self-limited, can progress to invasive bloodstream infection and clinically significant organ dysfunction in older adults with comorbidities. Blood cultures and careful assessment for invasive infection should be considered when patients with suspected *Campylobacter* enteritis present with severe systemic manifestations.

Categories: Family/General Practice, Geriatrics, Infectious Disease
Keywords: bacteremia, bacterial gastroenteritis, campylobacter enteritis diabetes mellitus, campylobacter jejuni, general medicine, rural, sepsis

Introduction

Campylobacter species are among the leading causes of bacterial gastroenteritis worldwide, with *Campylobacter jejuni* being the most frequently identified species [1]. Infection is commonly associated with the consumption of contaminated or inadequately cooked poultry and typically presents with diarrhea, abdominal pain, fever, nausea, or vomiting. Although most cases are self-limited, antimicrobial treatment may be necessary in patients with severe or prolonged illness [1].

Bloodstream infection caused by *Campylobacter* species is uncommon but can result in serious systemic complications, particularly in older adults and patients with underlying medical conditions or immunocompromising conditions [2-4]. Although *Campylobacter fetus* is recognized for its association with invasive infection, *C. jejuni* can also cause bacteremia [3-5]. The progression from enteritis to bloodstream infection may involve intestinal mucosal barrier disruption and impaired host defenses, although the precise mechanisms remain incompletely understood.

We report a case of *C. jejuni* bacteremia associated with acute enteritis in an older adult with multiple comorbidities who developed severe systemic manifestations, including altered mental status and acute hypoxemic respiratory failure. Although *C. jejuni* bacteremia has been previously documented, this case highlights the importance of recognizing invasive infection and assessing organ dysfunction in patients presenting with gastrointestinal symptoms. It also illustrates the challenges of interpreting severe respiratory manifestations and individualizing antimicrobial management when the optimal treatment strategy for *Campylobacter* bacteremia remains uncertain.

Case Presentation

A 71-year-old man presented to the Emergency Department with fever, altered mental status, and vomiting. His medical history included type 2 diabetes mellitus, hypertension, and hypokalemia, for which he was receiving regular outpatient care. He had undergone surgery for colon cancer nine years earlier. He was a former smoker and consumed approximately three glasses of whiskey daily. He was independent in activities of daily living before admission and had no history of overseas travel. A few days before presentation, he had consumed inadequately cooked chicken gizzards that had previously been frozen. His regular

medications included liraglutide, potassium L-aspartate, irbesartan/amlodipine, omeprazole, dapagliflozin, mitiglinide, and metformin. On the day of presentation, he developed urinary incontinence and vomiting, and his wife found him confused, prompting an emergency medical services call.

On arrival, his body temperature was 38.8°C, heart rate was 110 beats/min, blood pressure was 154/82 mmHg, and peripheral oxygen saturation (SpO_2) was 85% despite supplemental oxygen at 5 L/min via a face mask. He had frequent vomiting and profuse watery diarrhea without abdominal pain. Computed tomography showed no pulmonary infiltrates or pulmonary embolism but revealed edema of the ascending colon, suggesting acute enteritis (Figure *1*).

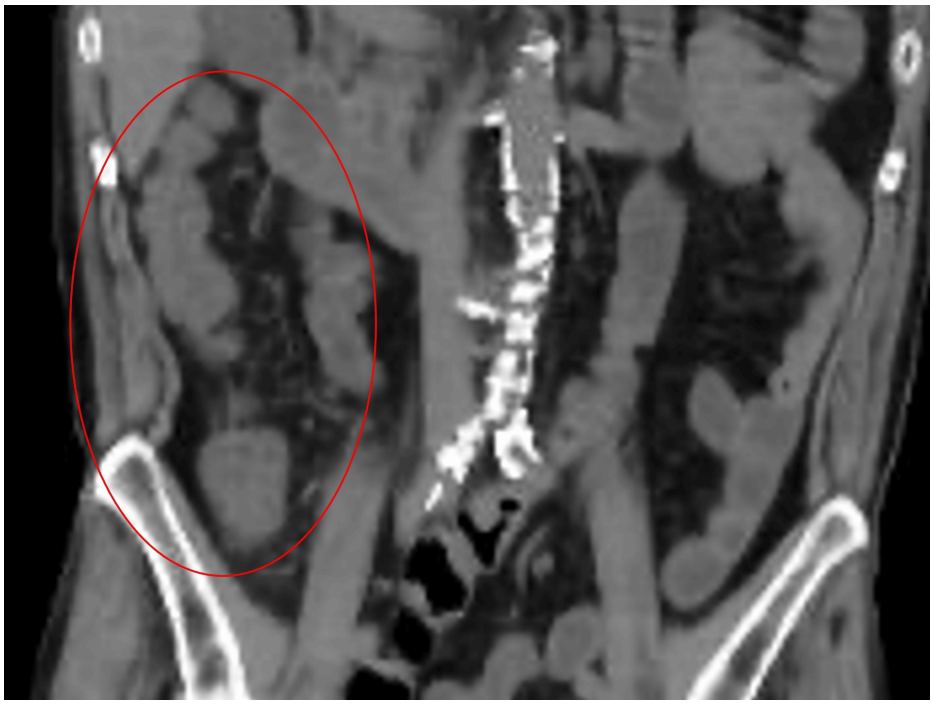


FIGURE 1: Computed tomography findings at admission.

Chest computed tomography showed no apparent pulmonary infiltrates or pulmonary embolism. Abdominal computed tomography demonstrated edema of the ascending colon (red circle).

Despite the severity of hypoxemia, initial chest computed tomography revealed no apparent pulmonary infiltrates or pulmonary embolism. Given the preceding vomiting and altered mental status, aspiration-related lung injury was considered a possible cause of respiratory failure. Sepsis-associated pulmonary dysfunction was also considered because of the concurrent systemic infection and acute organ dysfunction. However, the absence of radiographic pulmonary abnormalities made the underlying mechanism difficult to establish. The available clinical findings did not identify a definitive cause of the respiratory failure. Therefore, the etiology of the acute hypoxemia remained uncertain, although a transient respiratory complication associated with severe systemic infection was considered possible.

Laboratory investigations revealed an elevated C-reactive protein level of 4.41 mg/dL, a mildly elevated lactate level of 2.4 mmol/L, and a white blood cell count of 7,800/μL. Arterial blood gas analysis demonstrated a pH of 7.510, $PaCO_2$ of 25.0 mmHg, and PaO_2 of 81.0 mmHg during supplemental oxygen administration, indicating acute hypoxemic respiratory failure with respiratory alkalemia. The patient's altered mental status (Glasgow Coma Scale score of 13), together with substantial hypoxemia in the setting of suspected infection, raised concern for sepsis-associated organ dysfunction. The reported Sequential Organ Failure Assessment (SOFA) score was 4, supporting the clinical diagnosis of sepsis. Although the serum lactate level was mildly elevated, the patient remained hemodynamically stable without hypotension, and there was no evidence of septic shock. Detailed laboratory findings are presented in Table *1*.

Parameter	Level	Reference
White blood cells	7.8	3.5-9.1 × 10^3/μL
Neutrophils	72.7	44.0-72.0%

Lymphocytes	22.3	18.0-59.0%
Monocytes	4.6	0.0-12.0%
Eosinophils	0.3	0.0-10.0%
Basophils	0.1	0.0-3.0%
Red blood cells	4.90	3.76-5.50 × 10^6/μL
Hemoglobin	15.9	11.3-15.2 g/dL
Hematocrit	45.3	33.4-44.9%
Platelets	19.6	13.0-36.9 × 10^4/μL
Total protein	7.4	6.5-8.3 g/dL
Albumin	4.2	3.8-5.3 g/dL
Total bilirubin	0.3	0.2-1.2 mg/dL
Aspartate aminotransferase	27	8-38 IU/L
Alanine aminotransferase	35	4-43 IU/L
Alkaline phosphatase	138	106-322 U/L
Lactate dehydrogenase	218	121-245 U/L
Blood urea nitrogen	13.4	8-20 mg/dL
Creatinine	1.13	0.40-1.10 mg/dL
eGFR	50.1	>60 mL/min/1.73 m²
Serum Na	136	135-150 mEq/L
Serum K	3.4	3.5-5.3 mEq/L
Serum Cl	98	98-110 mEq/L
HbA1c	7.5	6.2%
CK	81	<56-244 U/L
CRP	4.41	<0.30 mg/dL
PT	10.5	10.5-13.5 seconds
APTT	30.3	23.0-40.0 seconds
Arterial blood gas		
pH	7.510	-
pCO2	25.0	35.0-45.0 mmHg
pO2	81.0	85.0-105.0 mmHg
Lactate	2.4	0.4-2.2 mmol/L
HCO3	23.9	22.0-28.0 mmol/L
Urine test		
Leukocyte	Negative	Negative
Nitrite	Negative	Negative
Protein	3+	Negative
Glucose	4+	Negative
Urobilinogen	±	Negative
Bilirubin	±	Negative

Ketone	1+	Negative
Blood	1+	Negative
pH	5.5	6-8
Specific gravity	1.026	1.010-1.030

TABLE 1: Laboratory findings on admission.

WBC, white blood cell count; RBC, red blood cell count; eGFR, estimated glomerular filtration rate; CK, creatine kinase; CRP, C-reactive protein; PT, prothrombin time; APTT, activated partial thromboplastin time; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; HCO_3^-, bicarbonate

Given the severity of hypoxemia and systemic illness, high-flow nasal cannula (HFNC) oxygen therapy was initiated with an initial FiO_2 of 1.0 and a flow rate of 60 L/min. Empirical intravenous meropenem (1 g every eight hours) was administered after blood and urine cultures were obtained. Stool specimens were collected for microbiological examination on hospital day 2.

On hospital day 3, *Campylobacter* species were detected in both blood and stool cultures. Blood cultures were processed using an automated blood culture system (BACTEC™; Becton, Dickinson and Company, Franklin Lakes, NJ, USA), whereas stool specimens were cultured on *Campylobacter*-selective agar under microaerophilic conditions at 42°C for 48 hours. Bacterial identification was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). The bloodstream isolate was subsequently identified as *C. jejuni* on hospital day 8.

Antimicrobial susceptibility testing was outsourced to an external clinical microbiology laboratory. According to information provided by the referring laboratory, susceptibility testing was performed using the disk diffusion method, with interpretations reportedly based on Clinical and Laboratory Standards Institute (CLSI) criteria. However, the specific CLSI document and version, inhibition zone diameters, and antimicrobial-specific interpretive criteria could not be verified. Minimum inhibitory concentrations (MICs) were not available.

The laboratory reports categorized both blood and stool isolates as susceptible to ampicillin, gentamicin, erythromycin, clarithromycin, chloramphenicol, fosfomycin, tobramycin, ofloxacin, and minocycline. Meropenem was reported as susceptible only for the stool isolate, whereas no corresponding result was available for the bloodstream isolate. Azithromycin was not directly tested. Because the original quantitative measurements and interpretive criteria were unavailable, the reported susceptibility categories could not be independently validated. These findings are summarized in Table *2*.

Antimicrobial agent	Stool isolate	Blood isolate
Ampicillin	S	S
Gentamicin	S	S
Erythromycin	S	S
Clarithromycin	S	S
Chloramphenicol	S	S
Fosfomycin	S	S
Tobramycin	S	S
Ofloxacin	S	S
Minocycline	S	S
Meropenem	S	NR

TABLE 2: Laboratory-reported antimicrobial susceptibility results of Campylobacter isolates from stool and blood cultures.

Reported S, categorized as susceptible in the original laboratory report; NR, not reported; NT, not tested.

Antimicrobial susceptibility testing was outsourced to an external laboratory and was reportedly performed using the disk diffusion method with Clinical and Laboratory Standards Institute (CLSI)-based interpretation. Minimum inhibitory concentration (MIC) values, inhibition zone diameters, and the specific interpretive breakpoints were unavailable. Therefore, the reported susceptibility classifications could not be independently verified. These results represent the original laboratory reports rather than independently validated susceptibility determinations.

Following the detection of *Campylobacter* species in both blood and stool cultures on hospital day 3, minocycline (200 mg/day) was temporarily added while definitive species identification and antimicrobial susceptibility results were pending. Intravenous meropenem was continued because of the severity of the initial systemic illness, including bacteremia, sepsis, and substantial hypoxemic respiratory failure. With antimicrobial therapy and supportive respiratory management, the patient's clinical condition and oxygenation gradually improved, allowing discontinuation of HFNC oxygen therapy on hospital day 5.

On hospital day 8, the bloodstream isolate was identified as *C. jejuni*, and minocycline was discontinued while meropenem was maintained. Although the stool isolate was reported as susceptible to meropenem, susceptibility testing results for meropenem were not available for the bloodstream isolate. Therefore, the continuation of meropenem reflected the individualized management of severe invasive infection rather than documented susceptibility of the bloodstream isolate to this agent. The patient's respiratory and gastrointestinal symptoms continued to improve, inflammatory markers decreased, and oral intake was resumed.

Repeat blood cultures were not obtained because the patient demonstrated progressive clinical improvement following antimicrobial therapy, including improvement in respiratory function and systemic manifestations. In accordance with the general principle that routine follow-up blood cultures are not required in clinically improving patients with uncomplicated Gram-negative bloodstream infections, microbiological confirmation of bloodstream clearance was not pursued. Nevertheless, the patient remained clinically stable without recurrence of fever or gastrointestinal symptoms during hospitalization.

On hospital day 12, meropenem was discontinued, and antimicrobial therapy was switched to oral azithromycin (500 mg/day) following sustained clinical improvement and recovery of oral intake. This transition was consistent with the documented susceptibility of both isolates to erythromycin and clarithromycin, although azithromycin susceptibility was not directly tested. Azithromycin was completed on hospital day 14, resulting in an approximately 14-day total antimicrobial treatment course from admission. The duration was an individualized treatment decision in the setting of bloodstream infection with initial organ dysfunction and subsequent clinical recovery. The patient remained clinically stable without recurrence of fever or gastrointestinal symptoms and was discharged on hospital day 24 (Figure *2*).

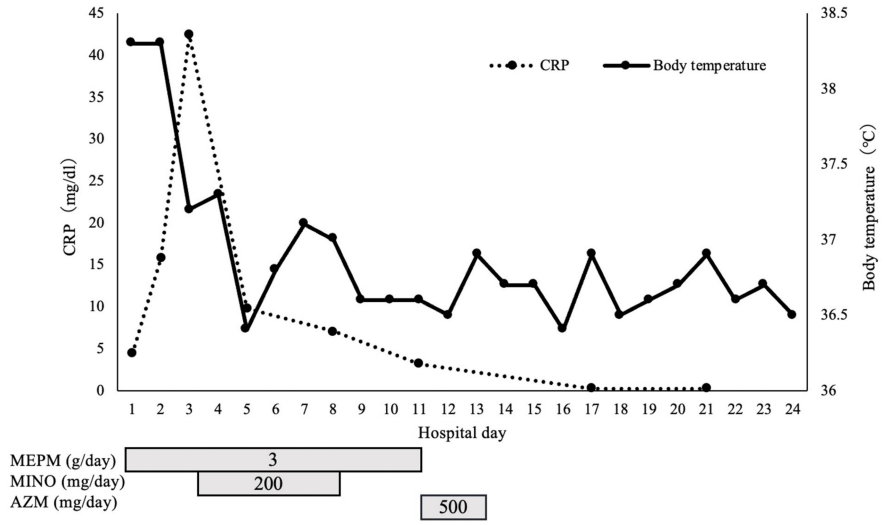


FIGURE 2: Clinical course and antimicrobial treatment during hospitalization.

The figure illustrates changes in body temperature and C-reactive protein levels, antimicrobial administration, and the patient's clinical recovery during hospitalization.

CRP, C-reactive protein; MEPM, meropenem; MINO, minocycline; AZM, azithromycin; HFNC, high-flow nasal cannula

Discussion

The present case demonstrates that *C. jejuni* enteritis can progress to bloodstream infection and sepsis in an older patient with underlying medical conditions. Although campylobacteriosis is generally a self-limited gastrointestinal infection, invasive disease is increasingly recognized as an important clinical manifestation, particularly in vulnerable hosts. A recent systematic review and meta-analysis of 32 studies involving 2,138 patients with *Campylobacter* bloodstream infection found that approximately 59% of affected patients had some form of immunocompromising condition and that *C. jejuni* was the most frequently identified species, accounting for 43.4% of cases [6]. Another recent meta-analysis including 2,480 patients reported that *C. jejuni* accounted for approximately 60% of bloodstream isolates and that the pooled case-fatality risk associated with *Campylobacter* bacteremia was approximately 5% [7]. These findings indicate that *C. jejuni*, although primarily recognized as an enteric pathogen, is also an important cause of clinically significant bloodstream infection.

In this case, foodborne transmission was strongly suspected because gastrointestinal symptoms developed after consuming inadequately cooked chicken gizzards. Poultry and poultry products are well-established reservoirs of *Campylobacter*, particularly *C. jejuni*, and consuming contaminated or insufficiently cooked poultry is an important route of human infection [8]. The patient developed profuse watery diarrhea and vomiting without prominent abdominal pain, followed by systemic manifestations and organ dysfunction. Importantly, we isolated *C. jejuni* from both stool and blood cultures. The simultaneous identification of the same organism at the presumed primary site of infection and in the bloodstream strongly supports progression from enteric infection to invasive disease.

Several host-related factors may have influenced the clinical course in this patient. He was 71 years old and had type 2 diabetes mellitus, hypertension, and a remote history of surgically treated colon cancer. Previous studies have reported that *Campylobacter* bloodstream infections occur frequently in patients with advanced age, comorbidities, or immunocompromising conditions [2-4,6,7]. However, diabetes mellitus alone should not be interpreted as an established independent cause of susceptibility to invasive *C. jejuni* infection. Although diabetes may affect host immune responses, the available evidence does not establish that it directly contributed to bacteremia in this patient. Furthermore, his history of colon cancer surgery nine years earlier did not necessarily indicate current malignancy or immunosuppression. The patient's severe presentation may instead be understood in the context of advanced age, multimorbidity, and potentially reduced physiological reserve, together with the systemic consequences of acute infection. Nevertheless, the relative contribution of individual host factors cannot be determined from a single case. This interpretation avoids attributing the development of invasive infection to diabetes mellitus alone and emphasizes the multifactorial nature of disease severity.

The clinical significance of the present case should be considered in the context of previously reported

Campylobacter bloodstream infections. Recent systematic reviews have demonstrated that *C. jejuni* is an established cause of *Campylobacter* bacteremia, particularly among patients with advanced age, underlying medical conditions, or immunocompromising conditions [6,7]. Therefore, the occurrence of *C. jejuni* bacteremia itself does not represent a novel clinical phenomenon. Rather, the distinctive feature of this case was the severity of systemic manifestations accompanying acute enteritis, including altered mental status and substantial hypoxemic respiratory failure requiring HFNC oxygen therapy, despite the absence of apparent pulmonary infiltrates or pulmonary embolism on initial computed tomography. Although the precise mechanism of respiratory failure remained uncertain, this presentation illustrates the importance of recognizing organ dysfunction and considering invasive infection in patients presenting with gastrointestinal symptoms.

Furthermore, the simultaneous recovery of *Campylobacter* species from stool and blood cultures supported an enteric source of the bloodstream infection. The case therefore provides a practical example of how a common foodborne gastrointestinal infection may be accompanied by clinically significant systemic illness. Its principal educational value lies not in demonstrating a previously unrecognized association but in emphasizing the need for early recognition of severe infection, appropriate microbiological evaluation, and individualized management in vulnerable patients.

Antimicrobial treatment of *Campylobacter* infection depends on disease severity, host factors, and antimicrobial susceptibility. Most uncomplicated gastrointestinal infections resolve without antimicrobial therapy, whereas treatment should be considered in patients with severe or prolonged illness and in those at increased risk of complications [9]. Azithromycin is generally a preferred agent for *Campylobacter* enteritis, but fluoroquinolone resistance has become increasingly common worldwide [9,10]. Macrolide resistance in *C. jejuni* remains relatively uncommon compared with fluoroquinolone resistance, although resistance patterns vary geographically and continued surveillance is necessary [11]. Antimicrobial resistance in *Campylobacter* involves multiple mechanisms, including mutations in antimicrobial targets and multidrug efflux systems, further supporting susceptibility-guided treatment whenever invasive infection is identified [10].

The antimicrobial management of *Campylobacter* bacteremia remains challenging because standardized treatment regimens and optimal treatment durations have not been established. Recent systematic reviews have demonstrated substantial heterogeneity in the antimicrobial agents and treatment durations used for *Campylobacter* bloodstream infections, reflecting differences in disease severity, host characteristics, and microbiological findings [6,7]. In the present case, antimicrobial treatment was adjusted sequentially according to the patient's clinical severity, microbiological findings, and response to therapy.

Initially, intravenous meropenem was selected empirically because the patient presented with sepsis complicated by acute hypoxemic respiratory failure before the causative pathogen had been identified. Following the detection of *Campylobacter* species in blood and stool cultures on hospital day 3, minocycline was temporarily added while species-level identification and susceptibility results were pending. Although macrolides are generally preferred for *Campylobacter* enteritis, the presence of bloodstream infection and substantial organ dysfunction complicated the selection of definitive therapy [9]. Meropenem was therefore continued during the early phase of clinical recovery, and minocycline was discontinued after identification of *C. jejuni* on hospital day 8. Importantly, meropenem susceptibility was documented for the stool isolate but was not reported for the bloodstream isolate, limiting the extent to which continued carbapenem treatment can be described as susceptibility-guided.

As the patient's respiratory condition and systemic manifestations improved, intravenous meropenem was discontinued on hospital day 12, and treatment was transitioned to oral azithromycin. This step-down approach was supported by sustained clinical improvement, recovery of oral intake, and reported susceptibility to erythromycin and clarithromycin, although azithromycin susceptibility was not directly assessed. The total antimicrobial treatment duration was approximately 14 days, reflecting an individualized approach to invasive infection associated with initial organ dysfunction. Although shorter treatment courses may be sufficient in selected patients, the optimal duration for *C. jejuni* bacteremia has not been established, and the present case cannot determine the necessity of a 14-day regimen. The absence of recurrent symptoms following treatment completion was reassuring but does not establish the superiority of this treatment strategy.

An important feature of this case was the development of substantial hypoxemic respiratory failure despite the absence of apparent pulmonary infiltrates or pulmonary embolism on initial chest computed tomography. The precise mechanism of respiratory failure could not be established from the available clinical findings. Aspiration-related lung injury was a possible explanation because the patient experienced repeated vomiting and altered consciousness before admission. Alternatively, systemic inflammation associated with sepsis may have contributed to pulmonary dysfunction and impaired gas exchange. However, the absence of characteristic bilateral pulmonary opacities did not support a definitive diagnosis of acute respiratory distress syndrome. Cardiogenic pulmonary edema and other cardiopulmonary causes could not be fully evaluated from the available information. The patient's respiratory condition improved with antimicrobial treatment and supportive oxygen therapy, allowing discontinuation of HFNC oxygen therapy on hospital day 5. Although this clinical course was compatible with a transient respiratory complication

accompanying severe systemic infection, the exact etiology remained uncertain. This diagnostic uncertainty should be acknowledged when interpreting the relationship between *Campylobacter* bacteremia and respiratory failure.

Another important consideration is the role of follow-up blood cultures in *Campylobacter* bacteremia. Although repeat blood cultures are essential in selected bloodstream infections, routine cultures to document clearance are generally not recommended for uncomplicated Gram-negative bacteremia when patients demonstrate an appropriate clinical response to antimicrobial therapy. A consensus guidance on uncomplicated Gram-negative bloodstream infections recommends reserving follow-up cultures for patients with inadequate clinical improvement, suspected endovascular infection, or insufficient source control [12]. In the present case, repeat blood cultures were not performed because the patient's respiratory and systemic conditions progressively improved with treatment. Nevertheless, given the severity of the initial presentation and the limited evidence specifically addressing *Campylobacter* bacteremia, the absence of microbiological confirmation of clearance should be acknowledged. Clinical recovery was considered reassuring, although it could not definitively exclude persistent bacteremia or an occult endovascular focus.

Several limitations should be acknowledged. First, this is a single case and cannot establish a causal relationship between individual host factors, such as diabetes mellitus, and the development of bloodstream infection. Second, although the temporal association with consumption of inadequately cooked chicken gizzards strongly suggests foodborne transmission, the suspected food source was not microbiologically examined; therefore, the route of transmission cannot be definitively proven. Third, evidence on the optimal antimicrobial regimen and duration for *C. jejuni* bloodstream infection remains limited, and current knowledge comes predominantly from retrospective observational studies and case reports rather than randomized controlled trials [6,7]. Nevertheless, the isolation of *C. jejuni* from both stool and blood cultures provides compelling evidence that enteric infection was associated with the subsequent bloodstream infection in this patient. Finally, the underlying mechanism of acute hypoxemic respiratory failure could not be definitively established. Although aspiration-related injury and sepsis-associated pulmonary dysfunction were considered plausible explanations, the available clinical and imaging findings were insufficient to distinguish between these mechanisms or fully exclude other cardiopulmonary causes.

An important limitation of this case concerns the interpretation of antimicrobial susceptibility testing. The testing was outsourced to an external laboratory, and the original report provided categorical susceptibility results without quantitative measurements. Although disk diffusion testing with CLSI-based interpretation was reportedly performed, the inhibition zone diameters, specific interpretive breakpoints, and CLSI document version could not be verified. MIC values were also unavailable. Furthermore, validated *Campylobacter*-specific breakpoints are not established for all antimicrobial agents included in the report, particularly meropenem, minocycline, and ofloxacin. Therefore, antimicrobial management in this patient should not be characterized as strictly susceptibility-guided. Initial meropenem treatment was based on the severity of systemic infection and associated organ dysfunction. Minocycline was temporarily added following microbiological detection of *Campylobacter* species, and subsequent treatment decisions were informed by species identification, available laboratory-reported findings, and the patient's clinical improvement. The transition to oral azithromycin was supported by clinical recovery and its established role in treating *Campylobacter* infection, although direct azithromycin susceptibility testing was not performed. These findings emphasize the importance of distinguishing laboratory-reported susceptibility categories from validated antimicrobial susceptibility results when interpreting the management of invasive *Campylobacter* infection.

Conclusions

Although *C. jejuni* bacteremia has been previously reported, this case highlights the importance of recognizing severe systemic manifestations, including altered mental status and substantial hypoxemic respiratory failure, in patients presenting with acute enteritis. The occurrence of bacteremia in this older patient with multiple comorbidities should not be attributed to diabetes mellitus alone, as the contributions of individual host factors cannot be established from a single case. Unlike uncomplicated *Campylobacter* enteritis, this presentation required intensive respiratory support and management of systemic infection, despite the absence of apparent pulmonary abnormalities on initial imaging. Clinicians should consider bloodstream infection and carefully assess organ dysfunction when patients with suspected *Campylobacter* enteritis develop severe systemic symptoms. The clinical significance of this case lies in reinforcing the importance of early recognition, appropriate microbiological investigation, and individualized management of invasive infection rather than identifying a previously unrecognized association.

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.

Concept and design: Ryuichi Ohta, Mori Nakai, Ayumu Suzuki, Kanata Tonosaki, Takumi Chiba, Eno

Tanaka, Sayako Tanaka

Acquisition, analysis, or interpretation of data: Ryuichi Ohta, Mori Nakai, Ayumu Suzuki, Kanata Tonosaki, Takumi Chiba, Eno Tanaka, Sayako Tanaka

Drafting of the manuscript: Ryuichi Ohta, Mori Nakai, Ayumu Suzuki, Kanata Tonosaki, Takumi Chiba, Eno Tanaka, Sayako Tanaka

Critical review of the manuscript for important intellectual content: Ryuichi Ohta, Mori Nakai, Ayumu Suzuki, Kanata Tonosaki, Takumi Chiba, Eno Tanaka, Sayako Tanaka

Supervision: Ryuichi Ohta, Mori Nakai, Kanata Tonosaki

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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