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Campylobacter jejuni Bacteremia Complicating Gastroenteritis in an Immunocompetent Patient Without Classic Risk Factors

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Abstract

Campylobacter jejuni (*C. jejuni*) is an uncommon manifestation of *Campylobacter* infection and may occur in patients without recognized immunocompromising conditions.

A 35-year-old woman with a history of gastroesophageal reflux disease (GERD) on chronic proton pump inhibitor (PPI) therapy and prior bariatric surgery (Roux-en-Y gastric bypass) presented with acute watery diarrhea, diffuse abdominal pain, vomiting, and fever. Imaging was consistent with enterocolitis, and she was treated supportively with empiric ceftriaxone and metronidazole before clinical improvement and discharge. She was recalled to the hospital one day later after blood cultures from the initial admission grew gram-negative, curved, aerophilic rods, subsequently confirmed as *C. jejuni*. She was treated empirically with meropenem and transitioned to levofloxacin. HIV testing, hepatitis panel, and stool studies for alternative pathogens were unrevealing. She improved clinically, with the normalization of inflammatory markers, and was discharged to complete a 14-day antibiotic course as an outpatient.

This case illustrates that *C. jejuni* bacteremia can occur in young, immunocompetent patients without traditional predisposing risk factors, challenging the paradigm that invasive *Campylobacter* disease is largely confined to the immunocompromised and elderly.

Categories: Internal Medicine, Infectious Disease, Gastroenterology
Keywords: bacteremia, bloodstream infection, campylobacter jejuni, gastroenteritis, immunocompetent host

Introduction

Campylobacter species (spp.) remain one of the leading causes of bacterial gastroenteritis worldwide, with an estimated incidence of 96 million cases annually [1]. Infection typically presents with watery diarrhea, abdominal pain, and fever [2]. Among *Campylobacter* species, *Campylobacter jejuni* (*C. jejuni*) is the most common pathogen, and infection is generally confined to the gastrointestinal tract [3]. *Campylobacter* bacteremia is a rare complication, occurring in less than 1% of cases [3]. *Campylobacter fetus* has a greater propensity to cause bacteremia, whereas *Campylobacter jejuni* and *Campylobacter coli* are most commonly isolated from stool cultures. The mechanisms by which *Campylobacter* causes bacteremia and the reasons why certain species are more likely than others to result in bloodstream infection remain poorly understood [4].

Although these mechanisms remain unclear, several risk factors have been associated with *Campylobacter* bacteremia; these include immunosuppression, hematologic malignancies, solid neoplasms, diabetes mellitus, liver disease, hypogammaglobulinemia, and advanced HIV infection with low CD4 count. It may also be associated with newly available immunosuppressive treatments, including biologic agents and targeted immunomodulatory therapies such as tumor necrosis factor-alpha (TNF-α) inhibitors, rituximab, and Janus kinase (JAK) inhibitors [5,6].

However, some studies have reported *Campylobacter* bacteremia in patients with no significant underlying comorbidities, suggesting that specific bacterial virulence factors may facilitate blood invasion independent of host immunodeficiency [5].

We report a case of *C. jejuni* bacteremia complicating acute gastroenteritis in a patient without classic immunocompromising risk factors. The patient initially presented with nausea, vomiting, diarrhea, and imaging findings consistent with enterocolitis. She was treated with supportive care and discharged but was subsequently contacted for readmission after blood cultures returned positive for *C. jejuni*. She was admitted for IV antibiotics, initially receiving broad-spectrum antimicrobial treatment that was subsequently narrowed. Inpatient evaluation, including HIV testing, was negative, and there was no known history or clinical evidence of a major immunocompromising condition. Notably, she had two features increasingly recognized as contributors to invasive *Campylobacter* infection: prior gastrointestinal surgery and chronic

proton pump inhibitor (PPI) use [7,8]. This case adds to the growing evidence that *C. jejuni* bacteremia can occur in immunocompetent individuals and highlights underrecognized, acid-suppression and surgery-related risk factors for invasive disease.

Case Presentation

We present the case of a 35-year-old woman with gastroesophageal reflux disease (GERD) on chronic proton pump inhibitor therapy and a Roux-en-Y gastric bypass two years prior, complicated by small bowel obstruction requiring exploratory laparotomy. She presented with a one-day history of watery, non-bloody diarrhea (>10 episodes); diffuse abdominal pain radiating to the back; one episode of non-bloody, non-bilious vomiting; and fever/chills. She denied recent travel or sick contacts; animal exposure, including contact with pets; or the consumption of high-risk foods. She had presented with similar symptoms on two occasions in the preceding month, at which time evaluations for cholecystitis, including abdominal ultrasound (US) and magnetic resonance cholangiopancreatography (MRCP), were negative.

On admission, she was febrile (maximum temperature {Tmax}: 101.1°F/38.4°C) and tachycardic (heart rate {HR}: 103-118 beats per minute {bpm}), with diffuse abdominal tenderness, most pronounced in the right upper quadrant. Laboratory studies were notable for leukocytosis, mild transaminitis, and mildly elevated inflammatory markers, with stool studies negative for alternative pathogens. Stool electrolyte and osmolality testing demonstrated a calculated stool osmotic gap of 24 mOsm/kg, consistent with a secretory pattern of diarrhea. Stool microbiologic testing was otherwise negative for the tested alternative pathogens (Table *1*).

Category	Test	Result	Reference Range
Hematology	WBC	12.5 × 10^3/μL	4.5-11.0 × 10^3/μL
Liver enzymes	AST	65 U/L	10-40 U/L
	ALT	82 U/L	7-56 U/L
	Alkaline phosphatase	129 U/L	44-147 U/L
	Lipase	60 U/L	10-140 U/L
Liver function	Total bilirubin	0.65 mg/dL	0.2-1.2 mg/dL
	Albumin	3.5 g/dL	3.5-5.0 g/dL
	Total protein	6.5 g/dL	6.0-8.3 g/dL
	PT	13.2 seconds	11-13.5 seconds
	INR	1.0	0.8-1.2
	aPTT	26 seconds	25-35 seconds
Metabolic panel	BUN	9 mg/dL	7-20 mg/dL
	Creatinine	0.67 mg/dL	0.6-1.1 mg/dL
	Sodium	134 mmol/L	135-145 mmol/L
	Potassium	5.3 mmol/L	3.5-5.0 mmol/L
	Chloride	104 mmol/L	98-107 mmol/L
	CO_2	24.4 mmol/L	23-29 mmol/L
	Magnesium	1.8 mg/dL	1.7-2.2 mg/dL
	Phosphorus	2.2 mg/dL	2.5-4.5 mg/dL
	Glucose	97 mg/dL	70-100 mg/dL
	Ethanol	<10 mg/dL	Undetectable
Inflammatory markers	CRP	3.9 mg/dL	<0.5 mg/dL
	Procalcitonin	0.69 ng/mL	<0.5 ng/mL
	Lactate (initial)	2.35 mmol/L	0.5-2.2 mmol/L
Infectious serologies	Hepatitis A IgM	Negative	

	Hepatitis B surface antigen (Ag)	Negative	
	Hepatitis B core IgM	Negative	
	Hepatitis C antibody (Ab)	Negative	
	HIV 1/2 Ag/Ab	Negative	
Stool studies	Bacterial culture	Negative	
	Clostridioides difficile toxin	Negative	
	Helicobacter pylori antigen	Negative	
	Ova and parasites	Negative	
	Rotavirus antigen	Negative	
	Stool sodium	116 mmol/L	Specific reference range is not established
	Stool potassium	17 mmol/L	Specific reference range is not established
	Stool osmotic gap	24 mOsm/kg	Secretory
Others	Urinalysis	Unremarkable	

TABLE 1: Laboratory and diagnostic findings on initial presentation.

WBC, white blood cell; AST, aspartate aminotransferase; ALT, alanine aminotransferase; PT, prothrombin time; INR, international normalized ratio; aPTT, activated partial thromboplastin time; BUN, blood urea nitrogen

CT of the abdomen and pelvis showed mildly distended, fluid-filled small and large bowel consistent with enterocolitis/ileus, without evidence of obstruction (Figure *1*). Abdominal ultrasound showed a distended gallbladder (Figure *2*) with a gallstone and a mildly dilated common bile duct (8 mm) (Figure *3*), without wall thickening or pericholecystic fluid.

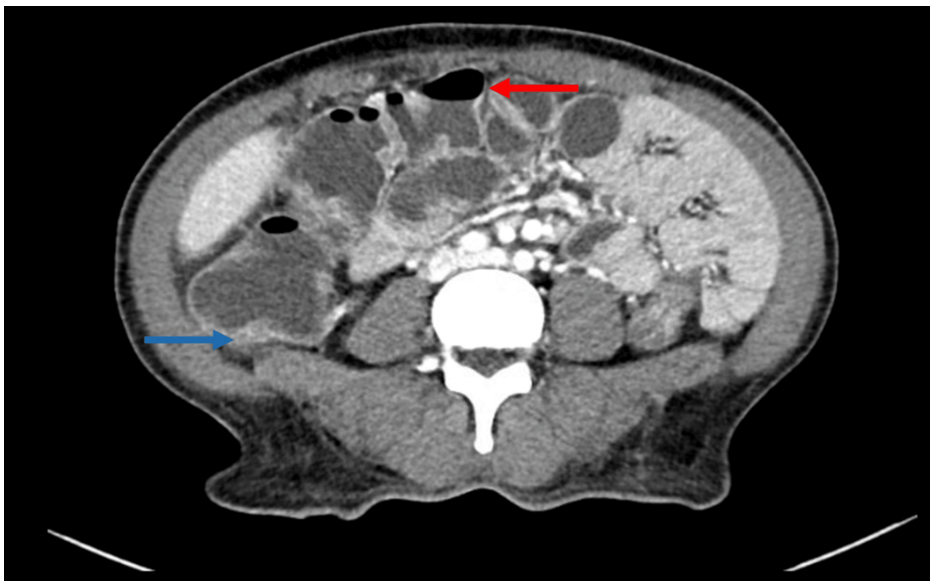


FIGURE 1: Contrast-enhanced CT of the abdomen and pelvis demonstrating mildly distended, fluid-filled small and large bowel (red arrow) with wall enhancement (blue arrow), consistent with enterocolitis, without evidence of intestinal obstruction.

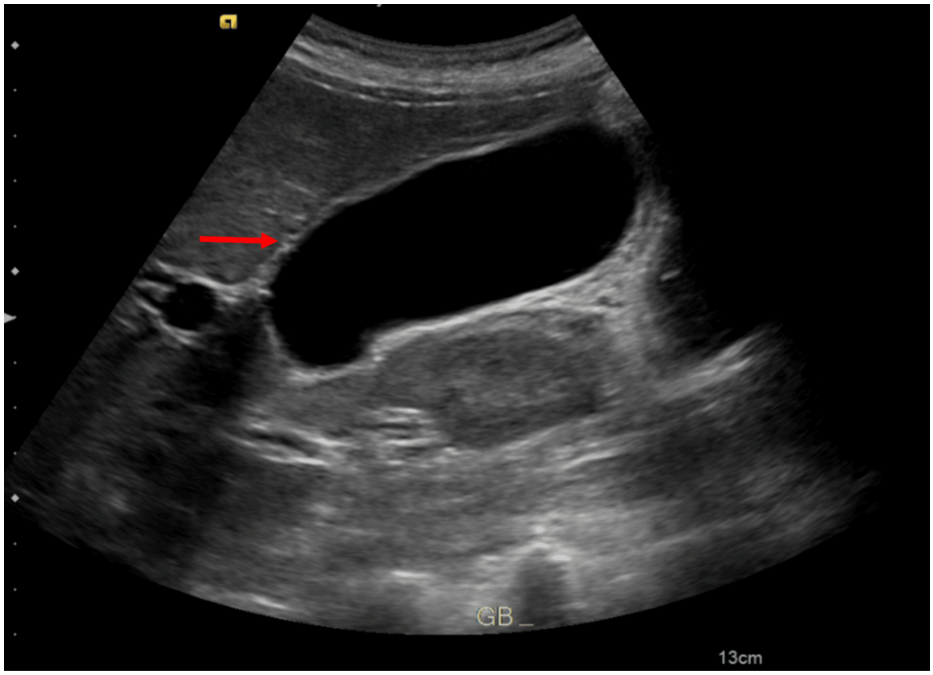


FIGURE 2: Abdominal US image demonstrating gallbladder distension (red arrow).

US: ultrasound

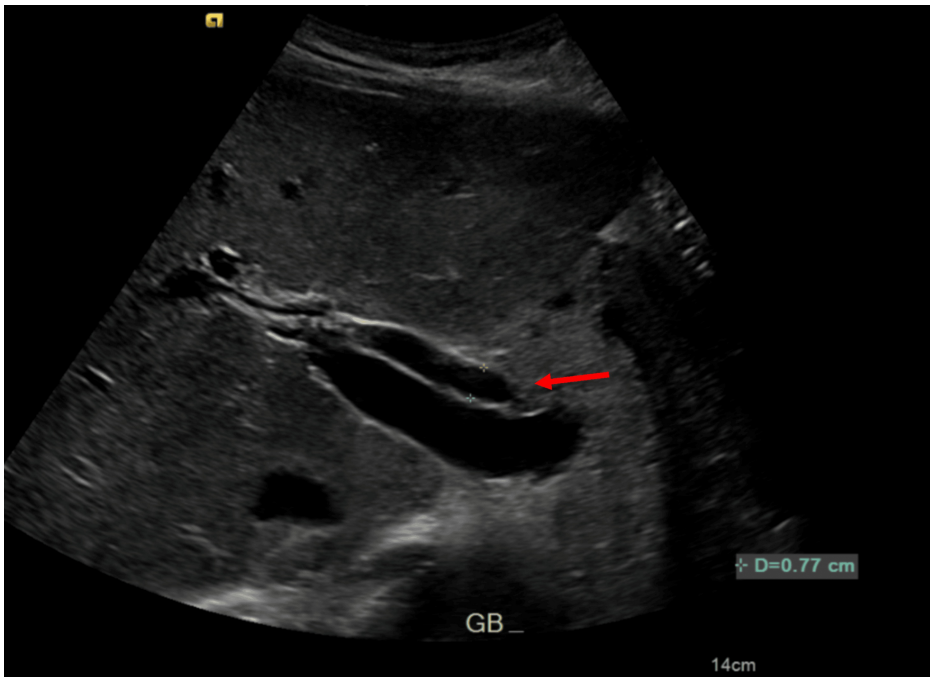


FIGURE 3: Abdominal US image demonstrating common bile duct dilation (red arrow).

US: ultrasound

She was treated empirically for acute enteritis with IV fluids, ceftriaxone and metronidazole for two days, pain control, and antiemetics. Surgery was consulted and did not recommend inpatient intervention for her gallstones. She improved clinically and was discharged two days later, with outpatient follow-up arranged with general surgery and primary care for the management of her gallstones.

She was recalled to the hospital three days after her initial presentation (one day after discharge) after two sets of aerobic blood cultures obtained during the initial admission grew gram-negative, curved, aerophilic rods. Based on the preliminary Gram stain and morphology, *Campylobacter* and *Vibrio* species were initially considered. On re-presentation, she reported improving diarrhea and persistent mild abdominal pain and remained afebrile. Infectious disease was consulted, and she was started empirically on meropenem, while definitive organism identification and antimicrobial susceptibility testing were pending, given the preliminary identification of a gram-negative curved rod and uncertainty regarding the organism and its antimicrobial susceptibility. Blood cultures were subsequently confirmed to grow *Campylobacter jejuni*, and after three days of meropenem, she was transitioned to levofloxacin 750 mg daily. She defervesced, her white blood cell count normalized ($4.9 \times 10^3/\mu L$), and her diarrhea and abdominal pain progressively improved. She was discharged home four days later, after the resolution of her symptoms, to complete a 14-day course of oral levofloxacin as an outpatient. Close outpatient infectious disease follow-up was planned; however, she did not attend the scheduled follow-up appointment. Antimicrobial susceptibility testing had been sent to an outside laboratory; however, the results could not be retrieved despite multiple attempts because of an issue with the outside laboratory.

Discussion

Most published series describe *Campylobacter* bacteremia as a disease of older, immunocompromised hosts, though this pattern is not universal.

The largest multicenter study to date was conducted in France on 592 patients across 37 hospitals. It showed that the median age of patients diagnosed with campylobacteremia was 68 years, and most patients had underlying conditions, such as immunosuppression (43.4%), hematologic malignancies (25.9%), solid neoplasms (23%), and diabetes (22.3%) [4].

Another study conducted in Spain identified liver disease (32.8%), HIV infection (23.4%), and hypogammaglobulinemia (15.6%) as the most common predisposing conditions [1]. Similarly, an Indian case series published in 2024 identified liver disease as a predisposing factor, with 75% of patients in the cohort having underlying liver disease classified as Child-Pugh class B or C [6]. In contrast, a nationwide Finnish study of 76 patients conducted over a 10-year period reported a younger median age of 46 years, with 70% of patients having no underlying comorbidities [9]. A Japanese study also demonstrated a bimodal age distribution, with a peak occurring among patients in their 20s [5].

These differences may reflect different population demographics, blood culture practices, or true epidemiologic variation.

Our patient, a 35-year-old woman without classic immunocompromising conditions such as malignancy, HIV infection, or hematologic disease, aligns with these latter cohorts and reinforces that *C. jejuni* bacteremia is not exclusively a disease of the immunocompromised or the elderly.

Although the exact mechanism through which *Campylobacter* species cause bloodstream infection is not fully understood, several theories exist. *Campylobacter* spp. have virulence factors that may explain bacteremia in the absence of host immunocompromise.

Campylobacter jejuni uses fibronectin-binding proteins (CafF and FlpA) for adhesion to the basolateral surface of intestinal epithelial cells and secretes invasion antigens (CiaB and CiaC), triggering membrane ruffling and therefore facilitating cellular entry. The cytolethal distending toxin (CDT) induces host cell apoptosis and disrupts epithelial barrier integrity [9,10].

Serum resistance, a prerequisite for survival in the bloodstream, has been linked to specific capsular polysaccharide phase variation, and certain clonal lineages are overrepresented among blood isolates [11].

Although molecular typing was not performed on our patient's isolate, these findings suggest that specific bacterial genotypes may possess an intrinsic capacity for bloodstream invasion regardless of host immune status, which may explain the development of bacteremia even in immunocompetent, otherwise healthy patients. Whether specific bacterial virulence factors contributed to bacteremia in our patient cannot be determined from the available data.

Two features of our patient's history warrant specific consideration as potential contributors to invasive disease despite the absence of classic immunocompromising conditions. First, she was on chronic proton pump inhibitor therapy for GERD. PPI-induced hypochlorhydria impairs the stomach's acid-mediated bactericidal barrier; a case-control study by Neal et al. [7] identified omeprazole use as an independent risk factor for *Campylobacter* gastroenteritis, and a subsequent meta-analysis found that PPI users have approximately fivefold increased odds of *Campylobacter* infection compared to nonusers [8]. Second, she had a history of prior Roux-en-Y gastric bypass surgery, which similarly reduces gastric acid exposure and alters gut microbial architecture; a population-based Danish study of *Campylobacter* bacteremia identified previous gastrointestinal surgery among the comorbidities more common in bacteremic patients compared

with those with uncomplicated enteritis [4]. Taken together, these two factors may have lowered the inoculum required for mucosal invasion and facilitated bloodstream translocation, independent of any defect in systemic immune function. However, a causal relationship between chronic proton pump inhibitor use, prior Roux-en-Y gastric bypass, and bacteremia cannot be established from this case.

The clinical phenotype of *C. jejuni* is also important to consider. *Campylobacter* bacteremia has two distinct clinical presentations: a gastroenteritis-predominant type presenting with fever and diarrhea, which is almost exclusively caused by *C. jejuni*, and a fever-predominant type without gastrointestinal symptoms, usually due to either *C. fetus* or *C. coli* [5]. This pattern is consistent with our patient's presentation, as she initially developed gastrointestinal symptoms and was subsequently found to have *C. jejuni* bacteremia.

Another phenomenon to discuss is the increasing detection of *Campylobacter* bacteremia. A study conducted by Tau et al. reported a 10-fold increase in *Campylobacter* bloodstream infection detection following the introduction of new blood culture bottles at their center, suggesting the possibility of historical underdetection, which may have contributed to the perceived rarity of this entity in immunocompetent individuals [12]. At our institution, however, this does not appear to be the case, as no new blood culture bottles have been introduced recently, yet *Campylobacter* bacteremia was identified in our patient. Nevertheless, this observation is interesting to note and suggests that factors other than changes in blood culture technology may contribute to the detection of *Campylobacter* bacteremia.

Concerning treatment, the Infectious Diseases Society of America (IDSA) recommends that *Campylobacter* bacteremia be treated for at least 14 days with a fluoroquinolone if the isolate is susceptible, with the consideration of adding a second agent such as an aminoglycoside to limit the emergence of resistance. Azithromycin, although first-line for uncomplicated enteritis, is not recommended for bacteremia [13]. Third-generation cephalosporins are not reliably active against *Campylobacter*, and their use was independently associated with mortality (OR: 10.2) in one French cohort [14]. Appropriate antimicrobial therapy was independently associated with improved 30-day survival (OR: 0.47) in the French campylobacteremia study [3].

Antimicrobial resistance is another major issue: fluoroquinolone resistance has been reported in approximately 29% of *C. jejuni* isolates in the United States, highlighting the importance of initiating empiric broad-spectrum antimicrobial therapy and later tailoring treatment based on susceptibility results [13]. In our patient, empiric meropenem was initiated while organism identification and susceptibility testing were pending. Following the identification of *C. jejuni*, therapy was de-escalated to oral levofloxacin as a narrower outpatient regimen, with close clinical and laboratory follow-up planned. However, susceptibility testing submitted to an outside laboratory could not be retrieved despite multiple attempts, and therefore, the isolate's final susceptibility profile was unavailable.

The prognosis of *C. jejuni* bacteremia in immunocompetent patients appears favorable. A Finnish study found that antimicrobial therapy had limited impact on hospitalization duration and mortality in immunocompetent hosts, suggesting that *C. jejuni* bacteremia in healthy individuals may follow a more benign, self-limiting course [15]. This finding is consistent with the observation of Tee and Mijch, who reported that campylobacteremia is typically a self-limiting illness in immunocompetent patients, in contrast to the severe, debilitating disease observed in patients with advanced HIV disease with low CD4 counts (33% mortality versus rare death in immunocompetent hosts) [16]. Our patient's favorable outcome is consistent with these observations and supports the notion that host immune competence is a key determinant of prognosis.

Complications of *Campylobacter* bacteremia should not be overlooked. Secondary localizations can occur, including vascular infections, endocarditis, and osteoarticular infections [3]. Post-infectious sequelae, including Guillain-Barré syndrome and reactive arthritis, have been reported as well [17]. No secondary localization or post-infectious complications were documented in our patient during the available period of observation; however, longer-term assessment was limited because she did not attend scheduled outpatient follow-up.

This case has several limitations inherent to case reports, including the inability to establish causality or generalize findings. The molecular typing of the isolate was not performed, which would have allowed the assessment of whether the strain belonged to a lineage with known invasive potential. In addition, antimicrobial susceptibility testing was submitted to an outside laboratory, but the final results could not be retrieved despite multiple attempts, limiting the characterization of the isolate's antimicrobial susceptibility profile. Finally, longer-term clinical follow-up was limited because the patient did not attend the scheduled outpatient infectious disease appointment.

Conclusions

This case demonstrates that *C. jejuni* bacteremia can occur in a relatively young patient without classic immunocompromising conditions, challenging the traditional association of this entity with immunosuppressed and elderly populations. Our patient's chronic proton pump inhibitor use and prior

Roux-en-Y gastric bypass surgery represent potential predisposing factors for *Campylobacter* infection and may warrant further investigation in relation to invasive disease. The favorable outcome observed in our patient is consistent with the literature suggesting generally positive outcomes among immunocompetent patients; however, this single case cannot establish the expected clinical course of *C. jejuni* bacteremia. Clinicians should remain vigilant for potential complications of invasive *Campylobacter* infection, including secondary vascular infection and post-infectious sequelae such as Guillain-Barré syndrome, even in patients without recognized immunocompromising conditions.

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: Camile Akkari, Ismail Althunibat, Jihad Slim

Drafting of the manuscript: Camile Akkari

Critical review of the manuscript for important intellectual content: Ismail Althunibat, Jihad Slim

Supervision: Jihad Slim

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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