

CASE REPORT

Severe Clostridium Difficile Infection Presenting with Acute Kidney Injury and Prolonged Diarrhoea: A Case Report and Clinical ReviewSmriti Arora¹, Mohit Yadav²**HOW TO CITE THIS ARTICLE:**

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ABSTRACT

Clostridium difficile infection (CDI) is a significant cause of healthcare associated diarrhoea¹ and may lead to severe complications, including acute kidney injury (AKI). We report the case of a 54-year-old male who presented with prolonged diarrhoea and AKI following recent antibiotic use. The patient exhibited hypotension, tachycardia, and marked dehydration. Laboratory investigations revealed elevated serum creatinine, urea, leukocytosis, and a positive stool assay for C. difficile toxins A/B. Aggressive fluid resuscitation, electrolyte correction, and oral vancomycin therapy led to gradual clinical improvement. This case highlights the importance of early recognition and comprehensive management of CDI complicated by AKI in emergency settings. Preventive strategies, including antimicrobial stewardship and infection control measures, remain critical in reducing disease burden and improving outcomes.

KEYWORDS

- Clostridium difficile • Acute renal failure • Acute kidney injury

INTRODUCTION

Clostridium difficile infection (CDI) is an increasingly common and severe² gastrointestinal condition, particularly among hospitalized or immunocompromised individuals. This article presents a rare and complex case of CDI complicated by acute kidney injury (AKI) and

prolonged diarrhoea, highlighting diagnostic and therapeutic challenges encountered in emergency medicine.

Case Report

A 54-year-old male presented with 10 days of profuse watery diarrhoea, generalized weakness, and decreased urine output. The

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patient had recently completed a course of amoxicillin-clavulanate for lower respiratory infection.

Examination Findings: BP 90/60 mmHg

HR: 110 bpm Severe dehydration

Serum creatinine: 4.1 mg/dL Urea: 156 mg/dL

TLC: 22,000 cells/mm³

Stool assay: Positive for *C. difficile* toxins A/B

He was admitted to the ICU and diagnosed with severe CDI and dehydration-induced AKI.

Background on Clostridium Difficile Infection (CDI)

CDI is caused by the *Clostridium difficile* bacterium, an anaerobic, spore-forming organism that produces toxins A and B leading to colitis, mucosal damage, and severe systemic complications.

Common Presentations Include:

- Watery, foul-smelling diarrhoea
- Abdominal pain and cramping
- Fever
- Nausea and anorexia
- Leukocytosis
- In severe cases: pseudomembranous colitis, sepsis, perforation, and multiorgan failure

Risk Factors and Epidemiology CDI primarily affects:

- Elderly patients
- Recent antibiotic users (especially cephalosporins, fluoroquinolones, and clindamycin)
- Hospitalized individuals
- Immunosuppressed patients
- Patients with gastrointestinal surgeries or feeding tubes

According to recent studies, the incidence of severe CDI has been steadily increasing, especially in low and middle-income countries (LMICs)³ due to increased antimicrobial use and inadequate infection control.

Case Presentation Summary

A 54-year-old male presented to the emergency department with 10-day history of profuse watery diarrhoea, generalized weakness, and decreased urine output. He had recently completed a 7-day course of amoxicillin-

clavulanate for a lower respiratory tract infection.

Key Findings:

- Hypotension (BP: 90/60 mmHg)
- Tachycardia (HR: 110 bpm)
- Severe dehydration
- AKI: Creatinine 4.1 mg/dL, Urea 156 mg/dL
- Leukocytosis: TLC 22,000 cells/mm³
- Positive stool toxin A/B assay for *Clostridium difficile*

This patient was diagnosed with severe CDI with dehydration-induced AKI and admitted to the ICU for supportive care.

Diagnostic Workup Essential Investigations:

- Stool tool test for CDI:
- *C. difficile* GDH antigen
- Toxin A/B ELISA⁴
- PCR for toxin genes (if available)
- Diagnostic criteria for AKI:
- CBC (leukocytosis with left shift)
- Renal function tests (elevated creatinine/urea)
- Serum electrolytes (hypokalemia, hyponatremia)
- Imaging:
- Abdominal X-ray/CT may show colonic wall thickening, ileus, or megacolon

CDI and Acute Kidney Injury (AKI): The Link AKI in the context of CDI may arise from:

- Profound fluid loss from diarrhoea
- Systemic inflammation due to toxin-mediated injury
- Sepsis-induced hypoperfusion
- Drug toxicity from previous antimicrobials or contrast exposure

This combination significantly increases mortality. Literature suggests that severe CDI with AKI has a 2-3x higher risk of ICU admission and prolonged hospital stay.

Differential Diagnoses Considered

- Infective diarrhoea (e.g., *Salmonella*, *Shigella*, *E. coli*)
- Inflammatory Bowel Disease (IBD) flare
- Ischemic colitis

- Antibiotic-associated diarrhoea (non-CDI)
- Tropical infections (e.g., amoebiasis, giardiasis)
- COVID-19-associated colitis

Stool microscopy, cultures, colonoscopy (if indicated), and serological workup helped rule out these causes.

Management Approach Initial Management:

- Aggressive IV hydration
- Correction of electrolytes
- Supportive renal care (strict input-output, nephrology referral) Specific Treatment for CDI:
- First-line⁵: Oral vancomycin 125 mg QID × 10 days
- Alternative/add-on: Fidaxomicin or metronidazole (if vancomycin is unavailable)
- In severe/complicated cases: IV metronidazole + oral vancomycin ± rectal vancomycin
- Faecal Microbiota Transplant (FMT): For refractory/recurrent CDI Monitoring:
- Daily labs for renal function and leukocytosis
- Strict isolation to prevent nosocomial spread
- Nutritional support (NG feeds or IV nutrition in prolonged cases)

Complications and Outcomes

If not promptly treated, severe CDI can lead to:

- Toxic megacolon
- Bowel perforation
- Sepsis and multiorgan dysfunction
- ICU admission
- High risk of mortality (20–30% in severe AKI cases)

In our case, the patient improved after 72 hours of vancomycin initiation, with gradual normalization of renal function over 10 days.

Prevention Strategies Hospital Protocols:

- Antimicrobial stewardship programs
- Isolation of CDI-positive patients
- Hand hygiene using soap and water (not alcohol rubs)

- Disinfection with sporicidal agents

Patient-Centered Measures:

- Avoid unnecessary antibiotics
- Educate high-risk patients about CDI symptoms
- Probiotics may be considered in some patients (evidence still mixed)

Clinical Pearls and Learning Points

- Always consider CDI in any hospitalized patient with diarrhoea, especially with recent antibiotic use.
- Renal involvement is not uncommon in severe cases and must be promptly addressed.
- Stool toxin assays and PCR are crucial for diagnosis don't rely on cultures alone.
- A combination of supportive care and early antibiotic therapy is key.
- Prevention and education remain the strongest weapons against hospital-acquired CDI.

Future Perspectives

- Novel agents like bezlotoxumab (anti-toxin B monoclonal antibody) show promise in reducing recurrence.
- AI-driven diagnostics may enhance early identification.
- Wider availability of FMT and probiotics could reduce reliance on antibiotics.
- Global health policies must address infection control in LMICs.

DISCUSSION

Clostridium difficile infection (CDI) has emerged as a major cause of healthcare-associated diarrhoea, with increasing incidence of severe forms involving complications like acute kidney injury (AKI), especially in resource-limited settings. In this case, the patient developed AKI likely due to hypovolemia and systemic inflammation associated with severe CDI. Literature supports that dehydration, sepsis, and antibiotic-induced nephrotoxicity contribute significantly to renal impairment in CDI patients⁶.

The importance of timely diagnosis using enzyme immunoassays for toxins A/B and PCR-based testing is emphasized, given the

limitations of stool cultures in urgent clinical settings.⁷ Moreover, empirical treatment with oral vancomycin remains the standard for severe CDI, although fidaxomicin and bezlotoxumab are gaining popularity for refractory or recurrent cases. In the present case, early recognition and targeted therapy led to full clinical recovery, highlighting the role of multidisciplinary management.

The role of antimicrobial stewardship is crucial, as inappropriate antibiotic use remains the foremost modifiable risk factor for CDI. Infection control measures especially hand hygiene and environmental decontamination using sporicidal agents are vital to prevent transmission.⁸ Future approaches such as fecal microbiota transplant (FMT) and monoclonal antibody therapy show promise but remain underutilized in low and middle-income countries (LMICs).

CONCLUSION

This case of severe Clostridium difficile infection presenting with acute kidney injury and prolonged diarrhoea highlights the importance of early recognition, aggressive supportive care, and timely antibiotic administration. Emergency physicians, intensivists, and internists must maintain a high index of suspicion in patients with high-risk profiles to reduce morbidity and mortality. Prevention through antibiotic stewardship and infection control remains crucial.

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