Case Report
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A case report of Pseudomembranous Colitis

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Abstract

Background: Pseudomembranous colitis is an infectious, inflammatory condition of the colon due to *Clostridiodes difficile* infection (previously called *Clostridium difficile* infection) which occurs due to changes in gut microbiota brought about by broad-spectrum antibiotic use. Complications are related to toxin-mediated injury. Diagnosis in resource-limited countries like Ethiopia is a challenge particularly where the laboratory is not advanced enough to do enzyme immunoassay tests or toxin assays. If there is a high index of suspicion, a limited colonoscopy with a cautious biopsy from the distal colon can help in the diagnosis. Oral vancomycin is the treatment of choice both for mild and severe cases.

Presentation of Case: We present a case of a 40-year-old female who presented with diarrhea of 4 weeks duration after taking oral ciprofloxacin for a urinary tract infection at a local health center. She was evaluated with a colonoscopy, showing multiple yellowish-white plaques at the sigmoid colon. Histology later suggested features of *Clostridium difficile* infection.

Conclusion: *Clostridiodes difficile* infection should be considered a possible cause of diarrhea and fever after recent exposure to antibiotics. Limited colonoscopy with biopsy may help diagnose when there is a high degree of suspicion. Effective antibiotic stewardship must be applied in our practice.

Keywords: *Clostridiodes difficile*, Microbiome, Pseudomembranous colitis, Toxin, Ethiopia

Introduction

C. difficile is a spore-forming, anaerobic, Gram-positive bacillus. Proliferating *C. difficile* produces toxins that cause several complications, including PMC, toxic megacolon, colon perforations, and sepsis (1). It is a common complication after antibiotic therapy. The infection is classically associated with clindamycin, though it is also associated with penicillins, cephalosporins, and fluoroquinolones. The incidence of *C. difficile* infection has increased due to the increased use of antibiotics (2).

C. difficile infection is the major identifiable cause of antibiotic-associated diarrhea. It is responsible for 15–25 percent of all cases, with a marked increase in the incidence of CDI since the turn of the 21st century (3). A systematic review of CDI in low- and middle-human development index countries showed a 15.8% CDI incidence rate amongst symptomatic patients. It appears that the frequency of CDI is lower in these regions than in more developed countries. In a 2013 publication, Groote Schuur Hospital, Cape Town, reported 8.7 cases per 10,000 hospitalizations (hospital-acquired CDI [HA-CDI]). Hospital-acquired CDI comprised 68% of CDI cases at this institution, with the remainder being community-acquired CDI (CA-CDI) (4).

Significant patient-related risk factors for CDI are antibiotic exposure, older age, and hospitalization. Nearly every antibiotic has been associated with the development of CDI, including the drugs used for the treatment of CDI: metronidazole and vancomycin. Age>65 years is a significant risk factor not only for CDI itself but also for poor clinical outcomes including severity and mortality. Other well-defined risk factors for CDI include inflammatory bowel disease, gastrointestinal surgeries, immunological incompetence caused by malignant neoplasms, transplantations, chronic kidney diseases, or immunosuppressant use (5).

When providing care for patients with *C. difficile*-associated diarrhea, contact precautions, including the routine use of gloves, can help decrease the risk of iatrogenic spread. The recommendation to stop the inciting antibiotic(s) once CDI is established is almost universal (6). Clinical practice guidelines have historically recommended oral vancomycin and/or metronidazole for most patients. However, following the Food and Drug Administration (FDA) approval of fidaxomicin in 2011, many clinical practice guidelines have considered fidaxomicin equivalent, if not preferred, to oral vancomycin in most scenarios (7).

Case presentation

This is a 40-year-old female patient from Awi Zone, Ethiopia who was diagnosed with a urinary tract infection at a local health center for which she was taking oral ciprofloxacin. Later she developed blood-mixed diarrhea (6th day after starting ciprofloxacin) with a frequency of 3-4 times per day for 4 weeks duration before being referred to our Hospital. She had associated nausea and crampy diffuse abdominal pain since the onset of the illness. She reported fever and reddish discoloration of urine with a recent decrement in urine amount for 1 week before being referred. There was a history of recurrent vomiting of ingested matter after admission but no hematemesis or melena. There was no history of diagnosed “PUD”. There was no similar presentation with diarrhea previously. She had regular menses with no menorrhagia. She was diagnosed with “acute kidney injury” and “severe malaria” at the referring health center (The blood film done identified Plasmodium falciparum malaria).

On examination, the patient’s vital signs were BP=100/52mmHg, PR=92 beats per minute, RR=22 breaths per minute, and temperature of 36.4°C. On HEENT evaluation she had pale conjunctiva and non-icteric sclera. There was no tenderness on abdominal examination, and percussion over the hepatic area was dull, which is normal. There was no mass, prolapsed vessel, or tenderness on per-rectal examination. No blood was seen on the examining finger. The anal tone was intact. A genitourinary evaluation was unremarkable. There was pitting pedal edema. Her mental status was normal.

On laboratory evaluation complete blood count showed white blood cell count of 8.9 x103 /µL, hemoglobin of 8.8gm/dL with MCV of 86.6fL, and platelet count of 26, 000/µL. The anemia and thrombocytopenia were explained with severe malaria. Repeat blood film was negative for malaria parasites. Peripheral morphology showed normochromic, normocytic RBCs. Creatinine was 10.42mg/dL and urea was 340mg/dL. Liver enzymes were normal. Serum electrolytes showed Na of 127mmol/L(135-145mmoL/l), ionized calcium level is 1.08mmol/L(1.2-1.4mmol/L), and serum potassium and chlorine were normal. Urinalysis evaluation showed +2 blood with many WBCs and RBCs. Stool examination as well as culture were unremarkable. Abdominal ultrasound was suggestive of bilateral renal parenchymal disease with minimal ascites. She was started on intravenous ceftriaxone, omeprazole, and metronidazole after

considering infectious colitis. Prednisolone 40mg po/d was started for presumed RPGN as a cause for AKI. The patient's symptoms worsened with an increase in the frequency of diarrhea to five times daily, elevated WBC count, and serum Cr after 3 days of admission and a colonoscopy was done considering the possibility of PMC. Limited colonoscopy revealed multiple raised yellowish-white plaques over the rectum and sigmoid colon (Figure 1). A biopsy was taken. The histology result showed mushroom-like eruption of fibrin, mucin, and inflammatory cells mainly neutrophils which was suggestive of clostridiodes difficile infection. Follow-up serum creatinine after 1 month was normal.

Ceftriaxone, omeprazole, furosemide, and prednisolone were discontinued. Intravenous metronidazole was continued, and oral vancomycin 125mg PO QID was started, significantly improving the diarrhea. The patient was placed in an isolation room, and infection prevention strategies were implemented. Renal side evaluation was performed after which septic acute tubular necrosis was considered as the cause for the acute kidney injury in addition to the insult due to volume loss and severe malaria. The patient began hemodialysis on the second day of admission considering uremic gastrointestinal manifestations (nausea, vomiting). Laboratory investigations were monitored progressively (Table 1). Finally, diarrhea and fever completely resolved after completing treatment for 10 days, and is currently under follow-up in an outpatient department.

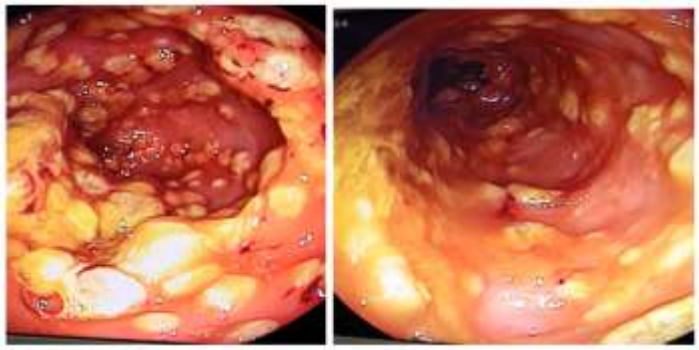

Figure 1: Multiple yellowish-white plaques with friable and edematous mucosa at the sigmoid colon.

Table 1: Laboratory results

		At admission	After 3 days	After 6 days	After 1 month
CBC	WBC (cells/µL)	8900	18500	13300	8700
	HB (gm/dL)	8.8	8.2	8.3	9.1
	PLT (x10³cells/µL)	26	676	485	331
RFT	Cr (mg/dL)	10.42	15.4	3.23	0.73
	Urea (mg/dL)	340.2	146.8	55	19
Electrolytes	K (mmoL/L)	4.08		3.6	
	Na (mmoL/L)	127		126	
	Cl (mmoL/L)	88		98	
	Ica (mmoL/L)	1.08		1.23	

Discussion

Clostridium difficile is an anaerobic gram-positive, spore-forming, toxin-producing bacillus that is transmitted among humans through the fecal-oral route. The relationship between the bacillus and humans was once thought to be commensal, but *C. difficile* has emerged as a major enteric pathogen with worldwide distribution (8). *Clostridioides difficile* infection is a common nosocomial and community-acquired cause of diarrhea and one of the most prevalent healthcare-associated infections (9).

Factors such as gastric acid suppression, advanced age, severe illness, enteral feeding, obesity, chemotherapy, gastroenteral surgery, and hematopoietic stem cell transplant are recognized as predisposing elements for C. difficile infection (10). Antibiotic exposure is the most important modifiable risk factor for CDI. Virtually every antibiotic has been associated with CDI, even following short antibiotic courses. Antibiotic classes that confer the highest risk of CDI include third- and fourth-generation cephalosporins, fluoroquinolones, carbapenems, and clindamycin (11). In our patient, the identified risk factors were antibiotic use (ciprofloxacin and ceftriaxone), the use of acid-suppressant drugs, and kidney disease.

The pivotal role of gastrointestinal microbiome in preventing CDI has increasingly becoming clear in recent studies. One such study highlighted how the loss of microbiome can create an environment that promotes growth of C. difficile and stimulates the production of toxins. A possible mechanism in which the microbiome can prevent infection is the conversion of primary bile salts to their secondary forms which inhibits toxin-producing vegetative cells of C. difficile and prevent their multiplication. A loss of microbiome leads to an accumulation of primary bile salts which can stimulate germination of C. difficile spores. When the microbiome is diminished by antimicrobials, C. difficile spore formation, growth of the organism, and toxin production is enhanced in colonized persons (12).

Most patients present with watery diarrhea, abdominal pain, and malaise; however, bloody diarrhea is rarely seen. In our case the patient had bloody diarrhea, which is unusual in terms of presentation.

Culturing viable organisms from stool followed by the confirmation of toxin production is considered the "gold standard" for diagnosing CDI (13).

Severe CDI is characterized by one of the following factors at presentation: fever, i.e. core body temperature >38.5°C, marked

leucocytosis, i.e. leucocyte count >15 X 10 9 /L, and rise in serum creatinine, i.e. >50% above the baseline (14). In our patient, severe CDI was considered as the WBC count at the time of consideration fulfilled the criteria.

Infection by *Clostridioides difficile (CDI)*, may result in severe sepsis and fulminant disease. Severe sepsis might be accompanied by acute kidney injury (AKI). The mechanisms include acute tubular necrosis due to systemic hypotension followed by hypoperfusion and/or hypoxemia, systemic hypotension, direct renal vasoconstriction, release of cytokines, and activation of neutrophils by endotoxins (15). In our patient, the acute kidney injury is explained by continued volume loss due to diarrhea, sepsis-related to CDI, and the concomitant infection with malaria.

Treatment strategies for CDI have evolved significantly. In the past, metronidazole has been the main therapy choice for a first mild CDI episode. Oral vancomycin gradually replaced metronidazole, due to multiple evidence supporting its superiority in the treatment of both severe and mild/moderate CDI (16).

Existing recommendations suggest washing hands with soap and water before and following contact with patients infected with C. difficile. Immediate isolation of patients displaying infectious diarrhea is a recognized infection control measure. In addition, patients should be placed in a private room with a toilet or a dedicated commode as soon as CDI is anticipated (17). Effective antimicrobial stewardship enhances infection control measures and environmental strategies to create a holistic approach to the prevention and management of CDI outbreaks (18).

The pitfalls in our patient's diagnosis and management included failure to consider CDI as a possible cause of diarrhea following antibiotic use, delayed referral to diagnostic facilities, failure to avoid acid-suppressive drugs and repetitive courses of antibiotics, and the use of steroids without proper consideration of such infection. The learning lessons for future clinical practice include the importance of considering CDI as a potential cause of diarrhea following antibiotic use, ensuring prompt referral to diagnostic facilities to avoid delays in diagnosis, practicing cautious antibiotic stewardship to prevent unnecessary use, and using steroids with care, especially in patients at risk of infections like CDI. By addressing these issues, healthcare providers can improve patient outcomes and reduce the risk of complications.

Conclusion

Clinicians should maintain a high index of clinical suspicion for diagnosing Clostridiodes difficile infection in any patient with diarrhea following antibiotic treatment. Furthermore, practicing cautious antibiotic stewardship to prevent unnecessary use and using steroids with care, especially in patients suspected of having CDI, is also vital.

Abbreviations

AKI: Acute kidney injury; CA-CDI: Community-acquired clostridiodes difficile infection; CDI: Clostridiodes difficile infection; HA-CDI: Hospital-acquired clostridiodes difficile infection; PMC: Pseudomembranous colitis; RPGN: Rapidly progressive glomerulonephritis

Declarations

Informed consent

Prior to data collection, written informed consent was acquired from the patient after the studies had been well explained.

Data Sharing

Supporting data for the current case report is available from the corresponding author upon reasonable request.

Competing interests

The authors report no conflicts of interest in this work.

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