Chronic Diarrhea Due to Duodenal Candidiasis in a Patient With a History of Kidney Transplantation

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Candida infection in the small intestine is uncommon. We report an unusual case of duodenal candidiasis that presented as chronic diarrhea in a patient who had previously undergone kidney transplantation. A 60-year-old man presented with profuse watery diarrhea that had lasted 6 months 13 years after kidney transplantation. Upper gastrointestinal endoscopy results indicated candidiasis within the esophagus and duodenum. Biopsy results revealed active duodenitis with hyphal and yeast forms of Candida overlying the duodenal epithelium in periodic acid Schiff staining. The patient was successfully treated with fluconazole. After 6 months of follow-up, the patient had no complaint of diarrhea. Duodenal candidiasis may be the result of chronic diarrhea in patients with a history of kidney transplantation.

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INTRODUCTION

In humans, Candida is considered a normal flora within the gastrointestinal tract; however, Candida species may grow abnormally and may result in mucosal plaques on the oropharynx or esophagus in healthy or immunocompromised patients.1,2 Candida infection within the small intestine is uncommon, although a few cases of Candida-associated duodenal plaques and ulceration have been described.3,4 Duodenal candidiasis-associated diarrhea is very rare in cases of organ transplantation.5 Here, we report a case of chronic diarrhea due to duodenal candidiasis in a patient with a history of kidney transplantation.

CASE REPORT

A 60-year-old man with a history of chronic hypertension-induced end-stage renal disease who had undergone kidney transplantation 13 years earlier was admitted because of diarrhea that had lasted for 6 months. The watery diarrhea was of a large volume and did not contain blood, mucus, or fat. The diarrhea was not controlled by fasting or changes in the patient’s nutritional regimen. The patient had no history of nausea, vomiting, abdominal pain, tenesmus, or fever. Since the diarrhea initiation, he had lost approximately 25 kg; in addition, he had lost his appetite. The diarrhea persisted even after the patient discontinued the treatment with mycophenolate mofetil.

On admission, he was under the immunosuppressive drug therapy involving prednisolone, 5 mg/d, and cyclosporine, 100 mg/d. The patient was also taking pantoprazole, 20 mg/d. Physical examination revealed bitemporal atrophy and pale conjunctivae. Laboratory test results included the following: serum bicarbonate level, 5 mEq/L; creatinine level, 5.6 mg/dL; leukocyte count, 4.3 \( \times 10^9 \)/L; hemoglobin level, 10.6 g/dL; platelet count, 192 \( \times 10^9 \)/L; erythrocyte sedimentation rate, 10 mm/h; and C-reactive protein, negative. The patient’s serum was negative for hepatitis
B surface antigens, hepatitis C virus antibodies, human immunodeficiency virus antibodies, and cytomegalovirus immunoglobulin M antibodies. No leukocytes, erythrocytes, or parasites were observed in his stool sample.

Upper gastrointestinal endoscopy showed multiple patchy white plaques on the esophageal and duodenal mucosa. No ulcers or other abnormalities were noted (Figure 1). Hematoxylin-eosin and periodic acid Schiff staining of the duodenal biopsy specimens indicated hyphal and yeast forms of *Candida* overlying the duodenal epithelium as well as moderate mixed inflammatory reaction and diffuse infiltration of polymorph nuclear and lymphoplasmacyte cells in lamina propria compatible with active duodenitis (Figures 2 and 3).

Small bowel transition study and colonoscopy findings were normal.

The patient was given fluconazole, 200 mg/d. Subsequently after 2 days, he experienced relief from diarrhea. At discharge, the patient’s serum bicarbonate level was 10.3 mEq/L, and his serum creatinine level was 3 mg/dL. During the 6-month follow-up, serum creatinine level was around 3 mg/dL and during this time, the patient had no complaint of diarrhea.

**DISCUSSION**

Our diagnosis of duodenal candidiasis-associated diarrhea was based on the clinicopathological evidence and the patient’s response to fluconazole. Mycophenolate mofetil may be associated with gastrointestinal adverse effects, including diarrhea; however, despite discontinuation of this medication, the patient continued to experience diarrhea.

Possible causes of diarrhea in kidney transplant patients consist of infection and medications, especially mycophenolate mofetil. Some infectious causes are *Clostridium difficile*, *Campylobacter jejuni*, cytomegalovirus, *Microsporidia*, *Strongyloides rcoralis*, and *Cryptosporidium parvum*. Duodenal candidiasis is uncommon. Rajablou and coworkers reported a patient with chronic lymphocytic leukemia who had multiple ulcerous masses in the stomach and duodenum. A duodenal ulcer due to candidiasis was also described in a patient with immunoglobulin A deficiency and a T-cell defect. In the literature, we found only 1 case of a duodenal ulcer and jejunal candidiasis in a patient who had undergone kidney transplantation. In contrast to this study,
our patient presented with diarrhea only. Diarrhea due to duodenal candidacies is very rare. Gupta and colleagues described 10 patients hospitalized because of severe diarrhea associated with abnormal Candida growth in the intestine.

Our patient had a history of taking pantoprazole. Acid-reducing therapy is known to result in Candida overgrowth. Therefore, in our patient, both immunosuppressive therapy and the long-term use of the proton pump inhibitor may have caused duodenal candidiasis. In summary, duodenal candidiasis-associated diarrhea may occur in patients with a history of kidney transplantation.

CONFLICT OF INTEREST
None declared.

REFERENCES

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