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Giardiasis mimicking acute abdomen in a renal transplant patient; a clinical and diagnostic challenge

Vaibhav Tiwari^{1*}, Anurag Gupta¹, Pallav Gupta², Devinder Singh Rana¹

¹Department of Nephrology, Sir Ganga Ram Hospital, New Delhi, India

²Department of Pathology, Sir Ganga Ram Hospital, New Delhi, India

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ABSTRACT

Background: Intestinal infection in the post-transplant period is a common source of morbidity and mortality. Diarrhea with or without abdominal pain, bloating, nausea and vomiting constitute the most common symptomology which guides the clinician towards appropriate treatment, but what happens when the clues are few?

Case Presentation: We report a case of recent renal allograft recipient who presented with severe abdominal pain without diarrhea or fever. After undergoing various tests, the diagnosis was finally clinched by jejunal biopsy since, it is supported by stool polymerase chain reaction.

Conclusions: This case highlights a common organism like giardia can have a varied presentation. This case mimicking almost as an acute abdomen while can present with a diagnostic challenge.

Implication for health policy/practice/research/medical education:

In an immunosuppressed individual, a common pathogen can have a varied presentation which may pose a diagnostic dilemma. Giardiasis is a common parasitic infection in the post-transplant period. Mostly symptoms include diarrhea with bloating and abdominal pain. We report a case of giardiasis which solely presented as severe abdominal pain without diarrhea. After undergoing several investigations, the diagnosis was finally made by jejunal biopsy. The patient was successfully treated with oral metronidazole. Atypical presentations of common organisms in the endemic region should always be kept in the differential diagnosis in challenging cases in immunocompromised patients.

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Background

Transplantation is the treatment of choice in chronic kidney disease (CKD) (1). However, there is an increased risk of typical as well as atypical infections in transplant recipients. Among the etiologies of all infections, gastrointestinal (GI) infections are common sources of morbidity and mortality (2).

Most infections are caused by bacteria and viruses. True incidence of parasitic infection is understated as most infections are asymptomatic or most patients receive empirical treatment (3).

Intestinal parasitemia is common after renal transplantation. Giardiasis is one of the most common intestinal parasites seen in transplant recipients (3). Prevalence is more common in developing countries with the infection rate as high as 20% (4). Transmission

generally occurs from contaminated food and water, person to person and zoonotic exposure. Giardiasis commonly presents as diarrhea, abdominal pain, malabsorption, and malaise. Stool examination is the investigation of choice although it is mostly unreliable as trophozoites are secreted at an irregular interval. The absence of the usual features may result in a low clinical index of suspicion for diagnosis.

Case Presentation

A 34-year-old male presented with two days of severe abdominal pain. The patient underwent live related renal transplant 2 months back, induction with anti-thymocyte globulin (ATG), on triple immunosuppressive regimen including tacrolimus, mycophenolate mofetil, prednisolone. He had a stable graft function with serum

*Corresponding author: Vaibhav Tiwari, Email: drvt87@gmail.com

creatinine of 0.9 mg/dL.

The onset of pain was rapid, diffuse with cramping character occurring intermittently with severe intensity. It was associated with nausea and vomiting but not with food intake or diarrhea. There was no history of fever, dysuria or hematuria. On examination, the patient was in discomfort, along with diffuse tenderness and guarding. Erect abdomen X-Ray showed mildly dilated bowel loops with fecal loading without features of obstruction or perforation of the viscus. Serum amylase (45 U/L) and lipase (12 U/L) were normal (Table 1). The patient was started on antiemetic, H2 blocker, antispasmodic and analgesic without much relief. An ultrasonography (USG) abdomen showed small bowel wall thickening without any evidence of appendicitis or cholecystitis. A suspicion of abdominal tuberculosis arose but the erythrocyte sedimentation rate (21 mm/h) was normal and Mantoux test (<5 mm) was negative. Accordingly, a possibility of post-transplant lymphoproliferative disorder was also suggested. CT abdomen with oral contrast showed jejunal thickening without any lymphadenopathy. Upper gastroscopy and colonoscopy were normal. Since lesion was suspected in jejunum, a double balloon enteroscopy was conducted. On inspection, the mucosa was visibly normal without any enteritis or ulcers. A biopsy showed pear shaped flagellated organisms suggestive of giardiasis lamblia as shown in Figure 1. Subsequently, stool analysis was conducted which did not reveal any ova or cyst. Accordingly, stool polymerase chain reaction (PCR) was positive for giardiasis lamblia. In view of severity of symptoms, combination therapy of metronidazole with nitazoxanide was initiated. In the next 4-5 days, our

patient improved drastically and discharged in stable condition. He continued metronidazole for 14 days. Our last follow up showed stable serum creatinine (0.87mg/dL). Repeat USG abdomen showed disappearance of bowel edema with negative stool PCR for giardiasis

Discussion

We report a case in the early transplant period presented with the sole manifestation of severe pain abdomen. Imaging studies were suggestive of bowel edema. Stool PCR and jejunal biopsy were consistent with giardiasis.

Diarrhea is a common complication after transplant. Although it is usually attributed to immunosuppressive drugs (5), the most common is infectious pathology.

Giardiasis infection is well established in the post-transplant period. Its incidence dramatically increases after transplantation likely due to immunocompromised state when compared to the general population (7.4% versus 1.8%) (6). Diarrhea is the most common symptom of giardiasis, followed by bloating and abdominal pain.

Giardia lamblia has two morphological forms; cysts and trophozoites. Cysts are the infectious forms of the parasite. They are excreted in stool and can survive in moist environments for prolonged periods. Following cyst ingestion, excystation occurs in the proximal small bowel with the release of trophozoites. Trophozoites are pear-shaped, binucleate, multi-flagellated parasite forms capable of division by binary fission; they localize principally to the proximal small bowel. An adhesive disk on the ventral surface of the trophozoite facilitates trophozoite attachment to the mucosal surface of the duodenum and jejunum, although the trophozoite usually does not invade the mucosal epithelium. Trophozoites that do not adhere to the small bowel move forward to the large intestine, where they revert to the infectious cyst

Table 1. Parameters at the time of admission

Parameters	Baseline	Normal Range
Hemoglobin (g/dL)	13.1	12-17
Total leukocyte count / μ L	5.3	4-10
Platelets / μ L	312	15-350
Creatinine (mg/dL)	0.9	0.6-1.3
Blood Urea Nitrogen (mg/dL)	12	7-20
Sodium (meq/L)	143	135-145
Potassium (meq/L)	3.9	3.5-5.0
Calcium (mg/dL)	8.8	8.8-10.2
Erythrocyte sedimentation rate (mm/h)	21	0-20
C-Reactive Protein (mg/dL)	0.8	<1
Amylase (U/L)	45	28-100
Lipase (U/L)	12	<60
Aspartate aminotransferase (U/L)	12	0-18
Alanine aminotransferase (U/L)	17	0-23

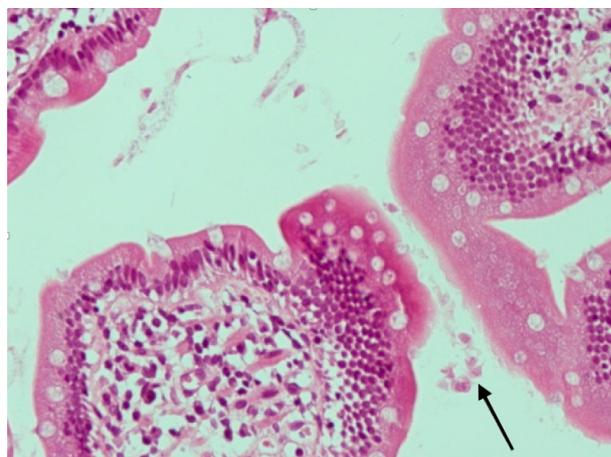


Figure 1. Jejunal biopsy of the patient showing pear shaped flagellated organisms (arrow) characteristic of giardiasis lamblia over the epithelium (H & E X200),

form and conjugated bile salts appear to foster encystation. Cysts are passed back into the environment in excreted stool in the setting of diarrhea. Trophozoites can also be found in the stool.

Giardia lamblia is a non-invasive parasite and rarely has been described with extra-intestinal complication. *Giardia* can spread from the duodenum to the biliary and pancreatic ducts, leading to cholecystitis, cholangitis, or granulomatous hepatitis. Impaired exocrine pancreatic function with the diminished secretion of trypsin and lipase has also been described. Hypersensitivity phenomena such as rash, urticaria, aphthous ulceration, and reactive arthritis or synovitis have been described in the setting of giardiasis, although these manifestations are rare (4,7,8). Abdominal pain as a sole manifestation has rarely been described in the literature. In a case report by Choi et al described a case of *Giardia* induced colitis presenting as abdominal discomfort for 2 weeks without diarrhea. The diagnosis was made on colonoscopy and biopsy (9).

Nitroimidazoles and nitazoxanide are most preferred agents for the treatment of *Giardia lamblia* (10,11). Antimicrobial resistance has been observed in up to 20 percent of *Giardia* isolates (12,13). Antimicrobial resistance testing is not routinely available in most clinical settings. Duration of therapy varies from single dose to up to 5-7 days depending upon the response to treatment. Metronidazole was continued for 14 days in this case because of severe giardiasis. In general, the presence of persistent or recurrent symptoms should prompt suspicion for antimicrobial resistance.

In patients with giardiasis, abdominal cramps may be part of altered intestinal motility due to disruption of the intestinal tight junction with increased permeability leading to the increased amount of fluid in the lumen resulting in diarrhea and cramps (14). Abdominal pain without diarrhea as in our case may not be explained completely with this mechanism. One postulate is that the heavy parasitic load may have induced a state of severe inflammation which could have given rise to severe pain as severe as almost equivalent to peritonitis. In an experiment on mice, Tzu-ling Cheng et al demonstrated that giardiasis leads to persistent damage to gut epithelia leading to further bacterial influx and subsequent inflammation (15).

Conclusion

This case throws light on the diagnostic odyssey in the identification of a common intestinal pathogen even in the endemic region when the clinical suspicion is low. Atypical manifestations of common pathogens should be kept in mind while formulation of differential diagnosis in resistant cases. Immunocompromised status provides

a canvas on which even a common organism may paint a picture which would have been seldom seen in the literature, therefore a high clinical suspicion and over-investigating is required in difficult cases.

Conflicts of interest

The authors declare no conflict of interest.

Authors' contribution

VT has drafted the initial manuscript which was further modified by AG and DSR. PG provided the light microscopy figures. All authors have reviewed and agreed on the final version of this manuscript prior to submission.

Ethical consideration

Ethical issues including plagiarism, double publication, and redundancy have been completely observed by the author. The patient has provided informed consent to publish as a case report.

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