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Case Report

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CRYPTOSPORIDIUM PARVUM COLITIS IN A PEDIATRIC LIVER TRANSPLANT RECIPIENT: A CASE REPORT

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Abstract

Cryptosporidiosis is an opportunistic infection caused by Cryptosporidium species. It leads to severe and prolonged diarrhoea in immunosuppressed patients such as paediatric solid organ transplant recipients. We present the case of a 17-year-old male with a history of Crigler–Najjar syndrome type I who underwent living-donor liver transplantation. He was admitted with a 3-week history of vomiting and abundant watery diarrhoea. The patient was maintained on triple immunosuppressive therapy with tacrolimus, methylprednisolone, and mycophenolate mofetil. On admission, stool cultures and antigen assays for parasitic and viral pathogens, including *Cryptosporidium parvum*, were negative. Despite supportive treatment, his symptoms persisted, and a colonoscopic biopsy was performed. Although the mucosa appeared normal, histopathological examination revealed the presence of *C. parvum*. Azithromycin 500 mg was administered daily for 10 days, resulting in the rapid resolution of gastrointestinal symptoms. Early recognition and timely therapy may prevent serious complications, including rejection, biliary disease, and septic shock. This case underscores the limitations of stool antigen assays in immunosuppressed paediatric patients and highlights the diagnostic value of endoscopic biopsy in persistent diarrhoea.

Keywords *Cryptosporidium parvum* · Liver transplantation · Immunocompromised host · Diarrhoea · Colitis · Colonoscopy

INTRODUCTION

Diarrhoea is a common complication after solid organ transplantation, occurring in approximately 10%–40% of recipients (1). Among transplant patients presenting with persistent diarrhoea, infectious aetiologies are frequently identified, and *Cryptosporidium* species have been reported in up to 10% of cases. Although the overall incidence of *Cryptosporidium* infection after paediatric liver transplantation is relatively low, its clinical impact can be significant (2).

Cryptosporidiosis is a worldwide infectious disease caused by the *Cryptosporidium* parasite. It affects various vertebrate hosts, including humans. Cryptosporidium infection is characterised by abdominal pain, diarrhoea and acute gastroenteritis. The

primary route of transmission is faecal–oral, such as through the ingestion of water and food contaminated with viable oocysts shed in faeces. While waterborne outbreaks of *Cryptosporidium* are well documented, the precise natural reservoirs and exact transmission pathways remain incompletely understood (3).

Cryptosporidium sp. is commonly recognised to have significant medical implications for both immunocompetent and immunocompromised patients alike. Immunocompromised individuals are more susceptible to contracting *Cryptosporidium sp.* infections than immunocompetent individuals. *C. parvum* causes life-threatening diarrhoea in immunosuppressed hosts, including paediatric solid organ transplant recipients. In this population, the infection may present with persistent watery diarrhoea, vomiting, and malnutrition and may even lead to graft dysfunction or rejection (2).

Diagnosis of cryptosporidiosis remains challenging. Routine stool examinations and antigen-based tests are often used initially; however, their sensitivity is low due to intermittent oocyst shedding in the stool and low parasite load in some patients. Repeated stool examinations, molecular techniques, or endoscopic biopsies may be required to establish a definitive diagnosis (4).

Treatment consists of intravenous fluid hydration, decreasing immunosuppressive therapy, and antiparasitic medications, such as nitazoxanide (possibly along with another agent, such as azithromycin or paromomycin), followed by surveillance. In immunocompetent children, a short course (3 days) of nitazoxanide can effectively clear the infection; however, in transplant patients and other immunosuppressed children, longer and more intensive therapy is usually required (2, 5).

Here, we present the case of an adolescent liver transplant recipient who developed persistent watery diarrhoea and vomiting. Despite a negative initial stool antigen test for *Cryptosporidium*, colonoscopic biopsy confirmed the presence of *C. parvum*. This case highlights the limitations of stool antigen tests and underscores the importance of invasive diagnostic tests in the context of unexplained, persistent diarrhoea in transplant patients.

CASE REPORT

A 17-year-old male with a history of Crigler–Najjar syndrome type I underwent living-donor liver transplantation from his mother at the age of one year. His post-transplant course was complicated by an acute rejection episode at fifth year and de novo autoimmune hepatitis at eight year. He has been maintained on triple immunosuppressive therapy with tacrolimus, methylprednisolone, and mycophenolate mofetil. He presented with a 3-week history of abundant watery diarrhoea and recurrent vomiting. The stools were non-bloody and did not contain mucus. On admission, vital signs were within normal limits. Physical examination revealed reduced skin turgor and hyperactive bowel sounds.

Laboratory evaluation revealed: urea 7 mg/dL, creatinine 0.7 mg/dL, AST 37 U/L, ALT 31 U/L, GGT 24 U/L, total/direct bilirubin 0.8/0.3 mg/dL, serum albumin 4.6 g/dL, C-reactive protein 0.9 mg/L, haemoglobin 13 g/dL, haematocrit 42%, WBC 5,290/µL, and platelets 184,000/µL. Serum electrolyte levels were within normal limits.

Stool cultures were negative, and stool antigen tests for adenovirus, rotavirus, *Clostridioides difficile*, and *Cryptosporidium* were negative. PCR-based testing for *Cryptosporidium* was not available in our institution.

Intravenous hydration and probiotic supplementation were administered. However, diarrhoea and vomiting persisted. Endoscopic evaluation showed normal upper gastrointestinal endoscopy and colonoscopy; however, multiple biopsies were obtained. Histopathological examination of the colonic biopsies revealed numerous small round-to-oval basophilic organisms attached to the apical surface of enterocytes, consistent with *Cryptosporidium parvum*. The organisms were identified using haematoxylin–eosin staining and confirmed using Giemsa and periodic acid–Schiff (PAS) staining (Figure 1-4).

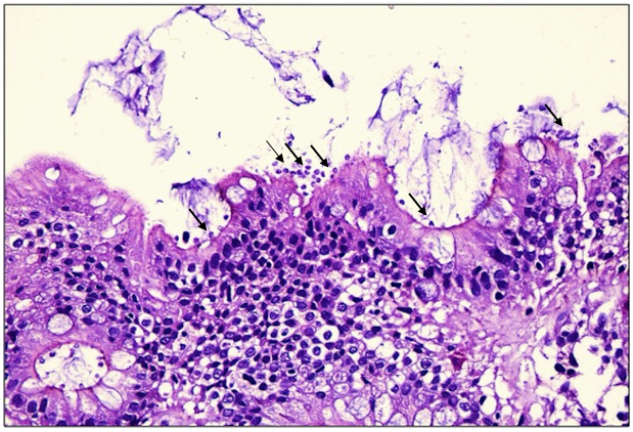


Figure 1. Hematoxylin–eosin staining shows small ovoid-to-round basophilic *Cryptosporidium* organisms attached to the surface of enterocytes (arrows).

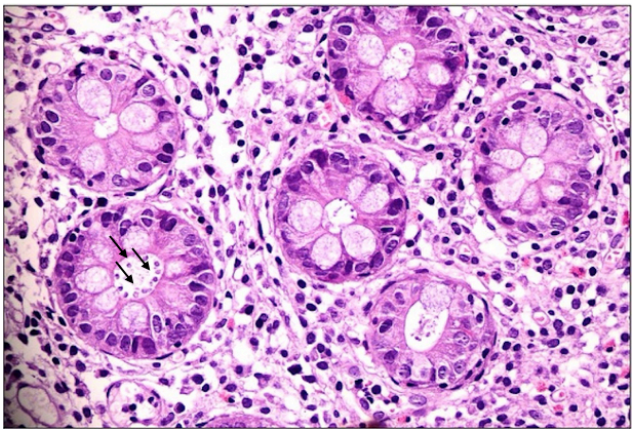


Figure 2. *Cryptosporidium* organisms are visible along the apical surface of enterocytes within the crypts (arrows).

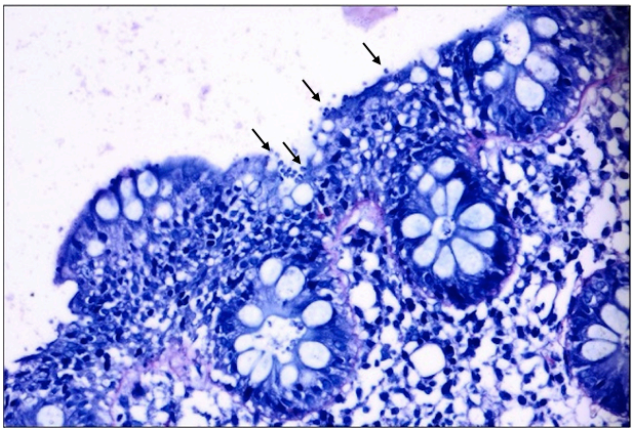


Figure 3. The *Cryptosporidium* oocysts are highlighted by Giemsa staining (arrows).

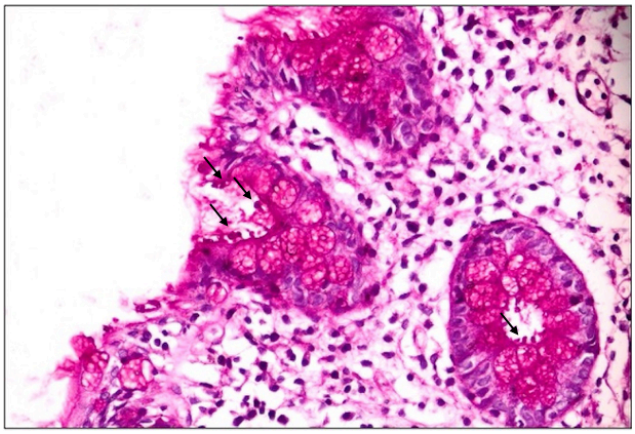


Figure 4. The *Cryptosporidium* oocysts are highlighted by PAS staining (arrows).

Antimicrobial therapy with azithromycin (500 mg daily for 10 days) was initiated because the patient was immunosuppressed. By the third day of treatment, stool consistency had normalised, and gastrointestinal symptoms had resolved.

DISCUSSION

This report contributes to the limited literature on cryptosporidiosis in paediatric liver transplant recipients presenting with persistent diarrhoea. Fischer et al. reported six paediatric liver transplant patients with *Cryptosporidium* enteritis; four developed acute rejection and three had steroid-refractory episodes, while half showed bile duct injury resembling sclerosing cholangitis. These findings indicate that *Cryptosporidium* can cause marked immune dysregulation and biliary complications in transplant recipients (2). In comparison, our 17-year-old liver transplant patient had a mild course without rejection or cholangitis. This favourable outcome may relate to early diagnosis, timely treatment, and the long post-transplant interval. Moreover, a case of septic shock secondary to *Cryptosporidium* infection has also been described in a liver transplant recipient (6). Our case underscores the importance of timely therapy: prompt eradication of the parasite may mitigate the inflammatory cascade that can jeopardise the graft.

Diagnosing *Cryptosporidium* in immunosuppressed paediatric patients is challenging. Noninvasive stool tests have limitations: microscopy may miss oocysts when shedding is low or intermittent. Enzyme immunoassays (EIA) and ELISA show higher sensitivity (94–100%) and specificity (93–100%) than acid-fast staining and can be used without specialised training. However, their sensitivity decreases with low oocyst counts, and some assays (e.g., ProSpect) may yield false positives (4). This was observed in our patient: despite persistent watery diarrhoea, the initial stool antigen was negative. Such false negatives may occur as oocyst shedding is intermittent and a single sample is often insufficient.

In the case by Tie et al., a post-liver transplant patient had persistent diarrhoea, with initial stool tests and antigen assays failing to detect *Cryptosporidium*. Diagnosis was confirmed only after colonic biopsy, with additional analysis through faecal oocyst staining and blood next-generation sequencing, highlighting the limitations of noninvasive tests in immunosuppressed patients (6). Similarly, in our patient, stool antigen tests were negative, and *C. parvum* was diagnosed only after colonic biopsy, highlighting the need for invasive diagnostics when suspicion persists.

Treatment options remain limited. The efficacy of nitazoxanide in immunosuppressed patients is uncertain. In contrast, macrolides such as azithromycin have increasingly been reported to be effective either as monotherapy or in combination regimens, with a particular benefit observed in severe cases (5). The rapid improvement of symptoms after 10 days of azithromycin highlighted the importance of early diagnosis and the efficacy of macrolide therapy.

CONCLUSION

In liver transplant recipients with unexplained persistent diarrhoea, *Cryptosporidium* infection should be considered even when stool antigen tests are negative. When non-invasive methods fail, colonic biopsy obtained during colonoscopy may help establish a definitive diagnosis. Early recognition and targeted therapy are essential for rapid recovery and preventing serious complications.

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Informed Consent	Informed consent was obtained from the patient who participated in this study.
Conflict of Interest	The authors have no conflict of interest to declare.
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