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Clinical Microbiology

CYCLOSPORIASIS IN A CASE OF RENAL TRANSPLANT.

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ABSTRACT Opportunistic infections (OIs) are encountered in immunosuppressed individuals, often observed in HIV patients or post-transplant individuals. Cyclosporiasis is one such OI which is rarely encountered. Precise identification of rarely encountered pathogens is a critical factor as these infections present with vague symptoms which may not improve by empirical treatment. Accurate diagnosis especially of opportunistic pathogens catalyzes targeted antimicrobial therapy and may bring about remarkable improvement in the patient's outcome. The present report is a case of cyclosporiasis in a 30-year-old female. Patient is a known case of renal transplant which suffered an allograft failure. She suffered with gastrointestinal symptoms which were unresponsive to empirical therapy. Investigation of stool revealed cyclospora infection in the patient. This finding paved the way for targeted therapy which led to resolution of symptoms. This case highlights the necessity of precise diagnosis and targeted treatment.

KEYWORDS : Cyclospora, renal transplant, opportunistic infection

INTRODUCTION

Cyclosporiasis is caused by an intestinal coccidian parasite of the genus *Cyclospora.* It was discovered in 1881 but it's pathogenicity was established in 1979. *Cyclospora cayetanensis* is the only known pathogenic species of this genus.[1] *C. cayetanensis* is not encountered frequently. Hence there is limited data about the parasite.

It is transmitted by feco-oral route. People acquire infection by the consumption of contaminated water and farm fresh food such as raspberries, lettuce etc. No reservoirs of the parasite have been encountered. It completes the whole life cycle in one host, which is humans. Cyclosporiasis is the infection of small intestine. Patients usually present with diarrhea or alternating constipation, nausea, weight loss, fatigue, etc. Although majority of cyclospora infections manifest as gastrointestinal symptoms, rare cases of cholelithiasis due to the infection have been encountered.[2]

The parasite is an acid-fast organism which can be demonstrated in feces of patients suffering from the infection by Modified Z-N staining. Microscopic examination shows presence of unsporulated oocyst of *Cyclospora* containing two sporocysts, each containing two sporozoites. Thus the oocysts contain some granules in it or give a bubbly appearance. Some oocysts may not stain and give a clear wrinkled or ghost like appearance.

Other methods for diagnosis include demonstration of autofluorescence under UV epifluorescence microscope. PCR assay of stool specimen is also available to detect the parasite. In spite of these advanced methods for the diagnosis, microscopic examination is still the most cost-effective method.

Trimethoprim-Sulfamethoxazole is the drug of choice for cyclosporiasis. It is given orally, twice a day for 7 – 10 days but for longer duration in case of HIV infected individuals. Relapse is often seen with this infection. Infection could be prevented by following food safety guidelines and avoid consumption of contaminated food and water.

The parasite generally affects immunocompromised patients. These conditions include HIV infection, transplant recipients, malignancies such as Hodgkin's lymphoma, etc.

Here, we report a case of intestinal cyclosporiasis in transplant recipient which responded to the standard treatment.

Case History:

A 30-year-old female presented to the outpatient department with complaints of multiple episodes of watery diarrhea & vomiting since one month. Associated symptoms included significant unintentional weight loss of 10 kilograms in last two months. There were no other gastrointestinal symptoms.

The general examination showed pallor without icterus. There was no evidence of any cyanosis, clubbing, lymphadenopathy, edema or swelling too.

The findings of respiratory and nervous system were normal. Tachycardia (120/minute) was present. Per-abdominal findings were also normal. At the time of admission, complete blood count showed 2.74 x 10^3/µl of white blood cells, 2.60x10^6/µl of red blood cells, 7.9 g/dL of hemoglobin and 23.1 % of hematocrit. The analysis showed 151 x 10^3/µl of platelets. Differential leukocyte count was 76% neutrophils, 16.8% lymphocytes, 5.5% monocytes, 1.1% eosinophils and 0.4% basophils. Serum electrolytes level for Na^+ was 129 mmol/L, K^+ was 2.80 mmol/L and Cl^- was 89.7 mmol/L. Liver function tests showed that SGOT level was 25 U/L and SGPT was 9 U/L. Serum creatinine level was 9.43 mg/dL. Urine analysis demonstrated turbid acidic urine with 35-40 pus cells/high power field, 12-15 epithelial cells/high power field, protein and bacteria without red blood cells, casts and sugar. Iron studies showed that serum iron level was 40.05 µg/dl, unsaturated iron binding capacity was 105.85 µg/dl, total iron binding capacity was 145 µg/dl and transferrin saturation was 27.45%. The patient has a known history of bilateral small kidneys for which she underwent a renal transplant seven years ago. Post-transplant immunosuppression treatment included Mycophenolate Mofetil, Tacrolimus, and Prednisolone for seven years till the current episode, followed by maintenance therapy.

Over the past two years, she had chronically elevated serum creatinine levels, ranging between 2.5 and 4.5 mg/dL. Concurrently, she has been persistently anemic, with hemoglobin levels between 7.1 and 9 g/dL. During this period, she also experienced intermittent episodes of diarrhea, which were empirically managed with Nitazoxanide.

Two months prior to the current presentation, she developed an episode of allograft dysfunction complicated by uremic encephalopathy, requiring initiation of hemodialysis. Azathioprine was added to her immunosuppressive regimen at that time. Following this episode, she developed persistent diarrhea that was unresponsive to Nitazoxanide. In view of her immunocompromised status and lack of response to empirical therapy, stool microscopy for opportunistic parasites was performed on three consecutive days.

Modified acid-fast staining of stool samples from days 2 and 3 revealed

oocysts of *Cyclospora cayetanensis* as shown in image 1. The patient was initiated on Trimethoprim-Sulfamethoxazole.

At follow-up after 15 days, the patient reported complete resolution of symptoms. Repeat stool microscopy was negative for *Cyclospora*. At present, the patient has been completely weaned off with immunosuppressives and clinically improved.

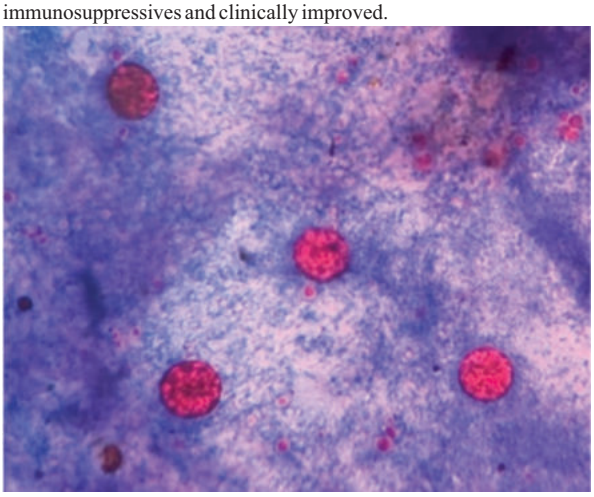

Image 1. Modified acid fast stain of stool specimen of the patient demonstrating oocysts of *Cyclospora cayetanensis*. (x1000)

DISCUSSION:

Cyclosporiasis can be seen worldwide. The infection is endemic in some tropical and subtropical countries. Global prevalence of cyclosporiasis is around 3.4% to 3.55%.[3] In India, the prevalence of *C. cayetanensis* is around 2.4% but there are some studies showing high prevalence of 22.2%.[4-5] A study from North India conducted by Ghoshal et al showed prevalence of 1.1%.[6] Gupta et al from Bihar found a prevalence of 12.90% of Cyclospora in stool specimens of HIV infected patients.[7] However metanalysis done by Chen et al revealed a prevalence of 4.8% from lower-middle-income countries which is an average of the numbers reported from India. In India, there are few studies on cyclosporiasis. But there are sporadic reports. Despite low number of documented cases in Mumbai, the actual prevalence in the city is likely higher.

Cyclosporiasis is reported mainly from immunocompromised patients. However infection may occur in immunocompetent individuals too. The most common immunocompromised condition is HIV infection. Other cases include patients on renal transplants, Hepatitis B infection, Hodgkins lymphoma, etc. Zope et al indicates a relation between HIV patients and cyclospora infection which might have decreased now due to ART to all patients irrespective of the CD4 count.[8]

The first published case report from eastern India exhibits Hodgkin's lymphoma as a predisposing factor for immunodeficiency.[9] The patient in the present study was given prolonged immunosuppressive therapy which is the probable cause of cyclosporiasis. Although Nitazoxanide was given to the patient for a duration of 2 weeks, it could not eliminate cyclosporiasis. Nitazoxanide may be used for cyclosporiasis but it is not the drug of choice. A major Indian study also reported five cases of cyclosporiasis in patients with renal transplant. Hussain et al has reported a similar case finding in a renal allograft recipient but their case had a very acute onset diarrhea on fourth day of admission.[10] While the patient in picture of our report had episodes lasting over a month.

Burden of the disease exists despite limited documentation. Clinicians attending the patient should be more cognizant that cyclosporiasis may affect both immunocompetent as wells as immunocompromised individuals, leading to notable gastrointestinal symptoms. Diagnosis of cyclospora infections could be easily established by simple and cost-effective measures such as modified acid-fast staining. Furthermore, it is necessary that the stool specimens are accompanied by a relevant clinical history for detection of opportunistic parasites. Correct diagnosis would facilitate targeted therapy, as empirical treatment using broad-spectrum antiparasitic agents would not be effective for Cyclospora infection.

Key Messages:

Accurate diagnosis bears great significance as it facilitates targeted therapy which can transform patient outcomes.

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