Strongyloidiasis – A rare cause for recurrent urinary tract infection in a cirrhotic

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ABSTRACT

Strongyloides stercoralis is an intestinal nematode that is commonly found in the tropical regions. *Strongyloid larvae* are very rarely seen in urine, especially in non-renal transplant patients. We report the case of a 60-year-old lady with cirrhosis, hypothyroidism, diabetes, hypertension, and history of methotrexate therapy for psoriasis presenting with decompensation of cirrhosis due to recurrent episodes of urinary tract infection. Urine microscopy showed the presence of *S. larvae*. The patient was treated with ivermectin after which her general condition improved.

Key words: *Cirrhosis, Recurrent urinary tract infection, Strongyloides stercoralis*

Strongyloides stercoralis is an intestinal nematode which is endemic in tropical countries. Disseminated infection is the term used to refer migration of the larvae to the organs beyond the range of the normal life cycle (skin, gastrointestinal tract, and lungs) [1]. Larvae have been reported to disseminate to the skin, mesenteric lymph nodes, gallbladder, liver, diaphragm, heart, pancreas, skeletal muscle, kidneys, ovaries, and brain. The presence of larvae in the urine has been reported in post-renal transplant patients on immunosuppressants [2]. However, the detection of larvae in urine in non-renal transplant patients is very rare [3]. We report the case of a 60-year-old diabetic lady with cirrhosis and disseminated strongyloidiasis presenting as recurrent urinary infection with the presence of *S. stercoralis* in the urine.

CASE REPORT

A 60-year-old diabetic female presented with complaints of fever, vomiting, and dysuria followed by progressive distension of the abdomen and altered sensorium of 1-week duration. She had consumed methotrexate (cumulative dose of 1.8 g) for psoriasis and was recently diagnosed with cirrhosis. She has a history of two similar episodes of urinary infection in the last month.

On examination, she was icteric with bilateral pitting pedal edema, facial hyperpigmentation, and extensive psoriatic skin lesions. There was no generalized lymphadenopathy or mucocutaneous bleeds. She was febrile (100.6°F). Her pulse rate was 92/min, blood pressure was 106/70 mmHg, and respiratory rate was 22/min. Abdominal examination showed mild hypogastric tenderness, splenomegaly, and ascites with shifting dullness. There was no asterixis. Examination of other systems was within normal limits.

Investigations revealed mild neutrophilic leukocytosis and mild eosinophilia (9%), thrombocytopenia, hyperbilirubinemia (5.8 gm/dL), and prolonged prothrombin time with international normalized ratio of 1.49. Her serum creatinine was 1.4 mg/dL and blood urea nitrogen was 22 mg/dL. Ultrasound of the abdomen was consistent with cirrhosis, portal hypertension, and moderate ascites. Bilateral kidneys were normal in size with normal corticomedullary differentiation. The bladder showed mild thickening and echogenic particles in urine which was considered as possible cystitis. Urine examination showed 6–8 pus cells/hpf and multiple rhabditiform larvae of *S. stercoralis* (Figs. 1 and 2). Urine culture for bacteria was sterile. Repeated microscopic examination of the stool and duodenal aspirate did not show *S. stercoralis*. Gastroduodenoscopy revealed mild portal hypertensive gastropathy and duodenal erosions. The duodenal biopsy did not reveal *Strongyloid larvae*.

The patient was diagnosed with decompensated cirrhosis with disseminated strongyloidiasis. She was treated with albendazole and ivermectin (days 1, 2, 14, and 15). Differential diagnoses of bacterial urinary tract infection (UTI) and bladder malignancy were also considered. However, her fever and dysuria subsided and ascites improved after initiation of ivermectin. She did not have any further episodes of urinary infection over the next 3 months, thereby confirming our diagnosis.

DISCUSSION

Disseminated strongyloidiasis although often neglected is a serious condition, occurring mostly in the immunosuppressed.

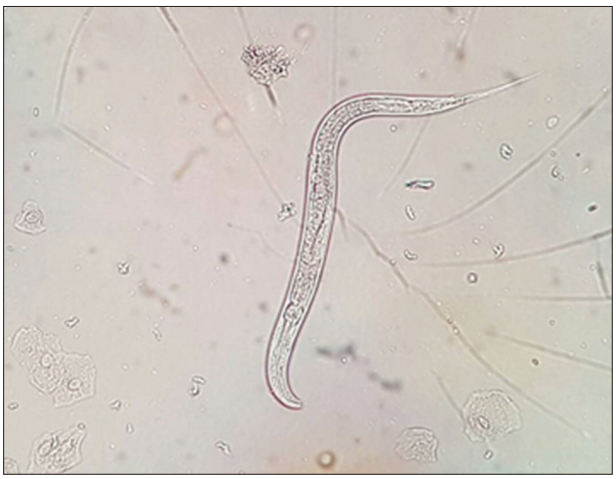

Figure 1: Urinary microscopy showing *Strongyloid larva*

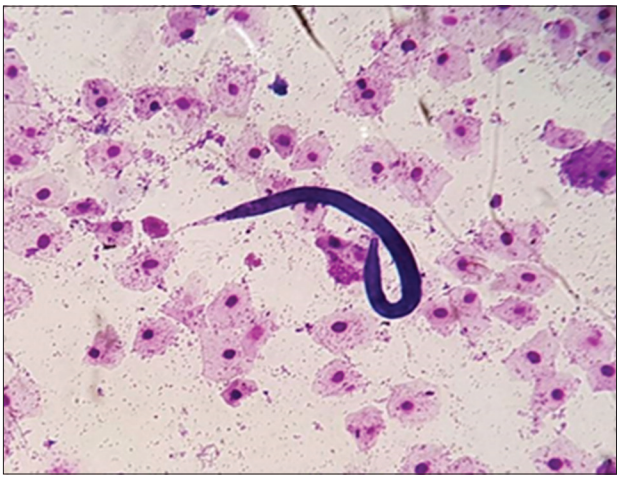

Figure 2: Leishman staining showing rhabditiform larva of *Strongyloides stercoralis*

It is prevalent in the tropics and subtropical regions. *S. stercoralis* has the unique ability to complete its lifecycle within the host through an autoinfection cycle which can become amplified into a fatal hyperinfection in the immunocompromised. The presence of larvae in urine has been detected in renal transplant recipients [4]. Lodh *et al.* showed that detection of strongyloid DNA in urine by polymerase chain reaction has better sensitivity than stool examination for larvae to diagnose infection [5]. Among the two cases of *Strongyloides* hyperinfection reported by Lemos *et al.* [6], *S. larvae* were detected in the urine and colonic biopsy specimens of the patient who was on steroids for autoimmune hemolytic anemia for 8 years.

However, a clinical presentation as isolated stongyloiduria causing urinary infection in cirrhosis is extremely rare. In a meta-analysis of 106 cases of strongyloidiasis reported in China by Wang *et al.* [7], only one patient had features suggestive of urinary infection alone. However, larva was isolated from urine in 8% of the patients by microscopic evaluation. A similar case was reported by Rifaat *et al.* [8], where the patient presented with repeated UTIs for 1 year and was found to have rhabditiform larvae in the urine.

The patients who are immunocompromised are more prone to strongyloid hyperinfection. Our patient had multiple risk factors (cirrhosis, diabetes, and methotrexate therapy). In a study conducted by Mathurin *et al.* [9], including 211 admissions of 132 consecutive patients, 28.1% of the 32 stool examinations done demonstrated the presence of *S. stercoralis* larvae. Chronic liver disease or cirrhosis was a risk factor in 8 of the 27 patients with strongyloidiasis in a retrospective analysis done in South Taiwan [10]. Thus, strongyloidiasis has been reported to have a close association with cirrhosis. However, to the best of our knowledge, this is the first case report of a cirrhotic patient presenting with recurrent UTI due to strongyloidiasis. Demonstration of larvae in the urine, clinical improvement and the stabilization of general condition following therapy, confirms the diagnosis.

CONCLUSION

Cirrhosis due to the associated immunosuppression should be considered as a risk factor for strongyloidiasis. Although rare, strongyloidiasis is a rare cause of recurrent UTI, it should be considered in patients with poor response to antibiotics. A high index of suspicion for this potentially life-threatening condition in the immunocompromised population and prompt initiation of treatment can be lifesaving.

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