Case Report:

Disseminated strongyloidiasis with acute kidney injury

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ABSTRACT

Strongyloides stercoralis, an intestinal parasitic nematode, infects more than 100 million people worldwide. Strongyloides are unique in their ability to exist as a free-living and autoinfective cycle. Strongyloidiasis infection usually remains asymptomatic, but in immunocompromised hosts hyperinfection and dissemination can occur, which has a high mortality. We report a 30-year-old male patient with membranous nephropathy who was receiving oral corticosteroids and cyclophosphamide who presented with disseminated strongyloidiasis. Larvae of *S. stercoralis* were isolated from the stool and the sputum specimens. Seven days later despite anti-helminthic therapy and intensive care support the patient died. Early detection and diagnosis of this condition is based on a high index of clinical suspicion, especially in immunocompromised hosts. Screening for Strongyloidiasis infection before the initiation of immunosuppressive therapy can be considered.

Key words: *Strongyloides, Immunocompromised hosts, Dissemination*

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INTRODUCTION

Strongyloides stercoralis is one of the most common globally distributed human pathogens of clinical importance that infects around 30-100 million people worldwide.[1] *Strongyloides stercoralis* a nematode, unique in its ability, persists in the human host for decades through auto infection. The adult female lives in small intestine. In the majority of the patients, the parasite load is low and they are free of symptoms. However, corticosteroid therapy, malnutrition, malignancy are known to change the host parasite relationship and result in hyperinfection and dissemination.[2]

CASE REPORT

A 30-year-old male, a patient with membranous nephropathy was receiving oral corticosteroids and cyclophosphamide, prescribed from a different facility, presented with history of loose motions and vomiting of 3 days duration. At presentation, he was conscious, afebrile, severely dehydrated, oliguric (urine output 400 ml/24 hours), hypotensive (blood pressure 80/60 mm Hg), Central venous pressure (CVP) was 6 cm water. Evaluation revealed anaemia (haemoglobin 9 g/dL), low normal leucocyte count (4300/mm^3) with a differential count: neutrophils 69%, lymphocytes 27%, monocytes 3%, eosinophils 1%. Urinalysis was normal. Urine sodium was low (14.2 mmol/L) and renal failure index was < 1%. Serum biochemistry revealed a progressive increase in serum creatinine from 0.93 mg/dL to 1.5 mg/dL over a period of few days. Serum sodium and potassium were low (119 mEq/L and 2.9

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mEq/L respectively) at presentation which improved (135 mmol/L and 4.5 mmol/L respectively) subsequently. Abdominal utrasonography was normal including kidneys. There were plenty of strongyloides larvae (Figure 1) on stool examination. With intravenous crystalloid fluid replacement, vasopressors support and ivermectin for strongyloidiasis, his blood pressure improved to 110/70 mm Hg and urinary output improved to more than 1000 mL/24 hours). Keeping these findings in view, the possibility of intestinal strongyloidiasis with pre-renal type of acute kidney injury (AKI) was considered. However, on the third day, patient developed cough with expectoration and sputum examination also revealed the larvae of Strongyloides (Figure 2) which suggested disseminated strongyloidiasis. Patient was continued on ivermectin and antibiotics. On the seventh day of hospital stay, he developed respiratory distress and succumbed despite ventilator supports.

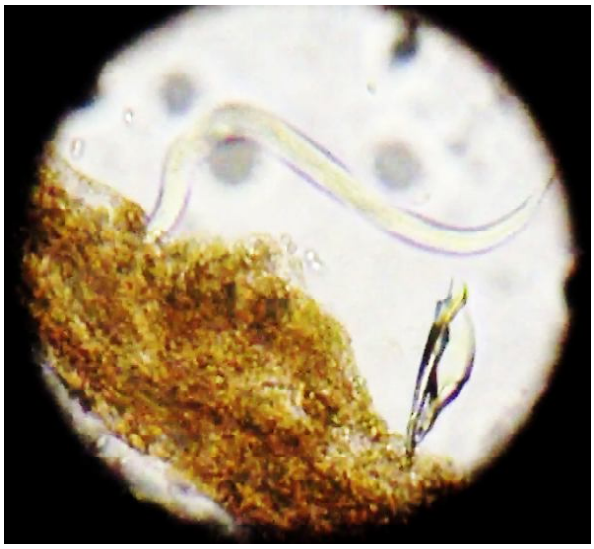

Figure 1: Photomicrograph of stool showing the larvae of Strongyloides (Wet mount, × 400)

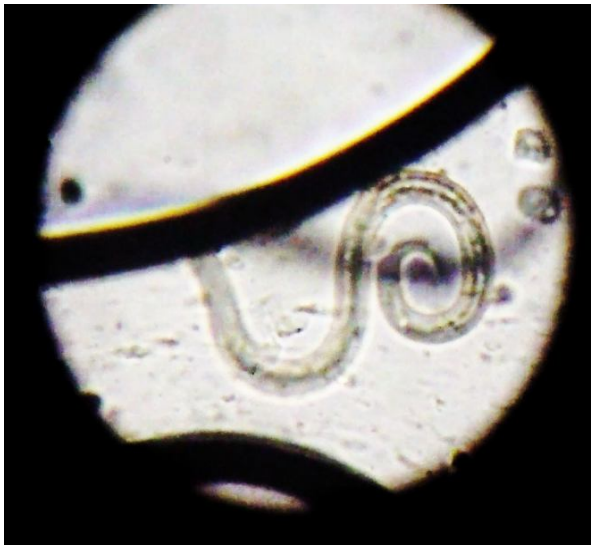

Figure 2: Photomicrograph sputum specimen showing the larvae of strongyloides (wet mount, × 400)

DISCUSSION

The hallmark of strongyloidiasis hyperinfection syndrome is an increase in number of larvae in the stool and/or sputum along with manifestations confined to respiratory and gastro intestinal symptoms with peritoneum involvement.[3] The strongyloidiasis hyper-infection syndrome happens from the enormous multiplication and migration of infective larvae especially in an immunosuppressed state.[4,5] The intestinal manifestations include severe cramping, abdominal pain, watery diarrhoea, weight loss, nausea, vomiting and occasionally gastrointestinal bleeding. The extra-intestinal manifestations include mainly bronchial asthma like symptoms, pneumonia and rarely pulmonary haemorrhage. Corticosteroids affect the immunity by increasing the apoptosis of Th2 cells, reducing the eosinophil count and inhibiting the mast cell response thereby leading to hyperinfection.[4,5] They also increase steroid like substances in the intestinal wall which give moulting signals that lead to increased production of auto-infective filariform larvae leading to hyperinfection. Dissemination and hyper infection occur in about 1.5%-2.5% of patients with strongyloidiasis and carries increased morbidity and mortality, which is reported to be up to 87%. The high mortality is frequently due to secondary bacterial infection. Ivermectin is the treatment of choice for strongyloidiasis. It is given for 2 days in asymptomatic states and for 5-7 days or until the parasites are eradicated in disseminated states.[6]

Our patient with disseminated strongyloidiasis presented with pre-renal type of AKI due to intravascular volume contraction and respiratory distress resulting in a fatal outcome despite specific management. This presentation is to reiterate the following. Fatal disseminated strongyloidiasis is a rare entity. Strongyloidosis must be considered in sick immuno-compromised patients with multi-organ involvement and excluding the asymptomatic infection with strongyloides in nephrotic syndrome patients before considering for immunosuppressive therapy can be useful.

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