

## Case Report

**Chronic diarrhoea due to severe strongyloidiasis in a Chronic Kidney Disease patient: A case report**A. Karunatilaka<sup>1\*</sup>, T. Hewageegana<sup>1</sup>, N. Y. Perera<sup>1</sup>, C. Kumarasinghe<sup>1</sup>, D. Sinharachchi<sup>1</sup><sup>1</sup>Teaching Hospital, Anuradhapura, Sri Lanka.**Abstract**

*Strongyloides stercoralis* is a soil-transmitted nematode endemic throughout tropical and subtropical regions. Although most of the healthy infected individuals are asymptomatic, it can be life-threatening for immunocompromised patients, and is responsible for a wide range of complications including chronic diarrhoea. Hence, it is vital to have a high level of suspicion and routine screening in immunosuppressed patients from endemic regions. We report a case of chronic diarrhoea due to *Strongyloides stercoralis* infection in a 55-year-old man who was on dialysis for end-stage Chronic Kidney Disease (CKD). The patient was initially managed as culture-negative continuous ambulatory peritoneal dialysis (CAPD) peritonitis. Later a thorough examination of his stools revealed *Strongyloides stercoralis* rhabditiform larvae, after which he was treated with albendazole. However, he eventually died of CKD. We emphasize the need for a thorough examination of stools with a high degree of suspicion in chronic diarrhoeas, particularly in immunocompromised patients.

**Keywords:** Chronic diarrhoea, Chronic Kidney Disease, *Strongyloides stercoralis*, examination of stool**Copyright:** © 2021 Karunatilaka A *et al.*  This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.**Funding:** None**Competing interest:** None**Received:** 12.03.2021**Accepted revised version:** 19.07.2021**Published:** 31.12.2021**\*✉ Correspondence:** : [anjanakaru1990@gmail.com](mailto:anjanakaru1990@gmail.com)  <https://orcid.org/0000-0003-4881-1534>

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**Introduction**

*Strongyloides stercoralis* is a soil-transmitted helminth that is endemic in tropical and subtropical regions. *S. stercoralis* is considered to have a complicated life-cycle on its ability to reproduce through asexual autoinfection and the capability of remaining dormant inside the host for decades [1]. The adult worms live in the small intestine while the larvae penetrate other vital organs such as the liver, brain, lungs, and kidneys, resulting in strongyloidiasis. Therefore, the clinical spectrum of strongyloidiasis varies largely, from asymptomatic to fatal disseminated infection [1]. Chronic kidney disease (CKD) is affiliated with the development of severe strongyloidiasis, owing to the associated immune

system dysfunction [2]. This is a report of a patient with end-stage kidney disease who was infected with *S. stercoralis*. We aim to highlight the importance of high clinical suspicion and timely diagnosis of *S. stercoralis* in high-risk populations from endemic regions.

**Case report**

A 55-year-old man from the north-central province of Sri Lanka, with end-stage kidney disease, presented with a two-month history of diarrhoea while on peritoneal dialysis. He also had a history of long-term diabetes mellitus (DM), hypertension, treatment complete tuberculosis, and chronic eczema. He was initially investigated for loose stools,

vomiting, and abdominal pain two months back. Eventually, the patient was managed as culture-negative continuous ambulatory peritoneal dialysis (CAPD) associated peritonitis. However, the symptoms worsened with an increased frequency (more than ten times per day) of loose motions with mucoid and watery stools. In addition, he complained of generalized body aches, loss of appetite, and several episodes of new-onset generalized tonic-clonic seizures over the last two months. The patient used to be a mason until five years back, and he has continued to be unemployed since then, with minimal social support.

On admission, the patient was found to be hypotensive with a blood pressure of 80/50 mmHg, dry scaly skin, anal excoriation, mild leg oedema, and a few fine lung crepitations. His abdomen was distended with generalized tenderness. His full blood count revealed mild eosinophilia, 6.4% (1%-6%), with a normal white cell count of 7.91 (4.0–10.0) × 10<sup>3</sup> cells/μL, and an increased C-reactive protein (CRP) level of 104 (<5) mg/L. The sigmoidoscopy disclosed haemorrhoids. The results of blood tests on day 2 of the last admission are presented in Table 1.

Repeated stool specimens were examined, all of which displayed only occasional pus cells, until the second date of current admission. The presence of *S. stercoralis* was established as rhabditiform larvae were identified together with 5-7 pus cells and 15-20 red blood cells (Figure 1). Grounded on these findings and the clinical background, the patient was diagnosed with severe *S. stercoralis* infection, resulting in worsening of existing CKD.



Figure 1. The larvae found on the patient’s stool sample

Albendazole (400 mg/day) was started immediately from day 2 of admission while a multi-disciplinary team management was established. Parasitology referral, as well as gastroenterology referral were arranged, after which the addition of ivermectin was suggested to prevent hyperinfection syndrome. Nonetheless, ivermectin could not be administered due to its unavailability in the country. A

venereology referral was also done to evaluate the status of sexually transmitted diseases and immunosuppressive infections, along with human immunodeficiency virus (HIV) screening.

Table 1. Investigation results on day 2 of admission

Test	Result	Reference range
<b>Full Blood Count</b>		
WBC (10 <sup>9</sup> cells/L)	7.91	4-10
Neutrophil (10 <sup>9</sup> cells/L)	5.57(70%)	2-7
Lymphocyte (10 <sup>9</sup> cells/L)	1.03(13%)	1-3
Eosinophil (10 <sup>9</sup> cells/L)	0.51(6.4%)	0.02-0.5
Basophil (10 <sup>9</sup> cells/L)	0.08(1%)	0.02-0.1
Monocyte (10 <sup>9</sup> cells/L)	0.72(9%)	0.2-0.5
Hb (g/dL)	9.8	11-17
Platelet (10 <sup>9</sup> cells/L)	302	150-400
<b>Serum</b>		
Total Protein (g/L)	55.4	60-85
Albumin (g/L)	20.5	35-45
Globulin (g/L)	34.9	25-35
Creatinine (μmol/L)	2494	60-120
Total Calcium (mmol/L)	1.8	2.2-2.7
Inorganic phosphate (mmol/L)	3.3	0.81-1.58
Na+ (mmol/L)	134	135-148
K+ (mmol/L)	2.3	3.6-5
CRP (mg/L)	104.7	0-5
<b>Stool Full Report</b>		
Pus cells (cells/HPF)	5 - 7	
RBC (cells/HPF)	15 - 20	
Amoebae, ova, or cyst	<i>S. stercoralis</i> larvae present	

Nevertheless, the patient remained in a life-threatening condition with no significant improvement. After one week of extensive treatment and support, he eventually expired of CKD complicated with *S. stercoralis* infection.

Discussion

The global burden of *Strongyloides stercoralis* infection - originated by the limited data available - estimates a prevalence of 30 to 100 million people affected worldwide [3]. The data availability is minimal, possibly due to underdiagnosis as well as underreporting. The prevalence is generally high among immunocompromised patients: HIV or human T-cell lymphotropic virus (HTLV) infected, haematological malignancies, kidney or bone marrow transplanted, malnourished, and patients on immunosuppressive drugs [1,3]. The fact that our patient had repeated institutionalizations, along with low

socioeconomic status and long-term DM, might have played a part in elevating his risk of developing complicated strongyloidiasis. Hence, we recognize the importance of early suspicion of *S. stercoralis* infection, in a given clinical background, in order to implement early treatment.

CKD is a syndrome that demonstrates simultaneous involvement of multiple organs with systemic inflammation and acquired immune deficiency. Although no specific mechanism behind the cause-effect relationship between CKD and immune suppression has been elucidated, several studies have attempted to provide mechanistic insights. It has been suggested that an imbalance between the natural microflora of a patient, especially that of the gut, is possible secondary to uraemia, metabolic acidosis, dietary modifications, volume overload with intestinal wall congestion, and frequent use of antibiotics and oral iron tablets. This process of imbalance in flora persistently activates innate immunity, which in turn induces immunoregulatory mediators to suppress the innate and adaptive immunity: a concept called “endotoxin tolerance.” [4]. Hence, a CKD patient is prone to develop rare infections.

Strongyloidiasis among patients with isolated CKD is rarely published. On review of literature, many articles were

reported on strongyloidiasis either among kidney transplanted patients or among patients on immunosuppressive or steroid therapy. In most cases, the primary complaint was evaluated in view of various differential diagnoses other than strongyloidiasis and, therefore, laboratory analysis of parasites in stool was delayed [5,6]. In our case, the patient was initially treated as CAPD peritonitis, pertaining to his primary abdominal symptoms. The delay in diagnosis, which might have reduced the efficacy of treatment leading to treatment failure, and the possible deterring of enteral absorption of antihelminthics due to bowel obstruction can be considered the main reasons for the fatal outcome in this patient. Moreover, the first-line drug, ivermectin, is considered superior in terms of efficacy to albendazole, with a low profile of adverse effects [7]. Thus, the plausibility of having a better outcome provided ivermectin was available locally, also needs to be considered.

In conclusion, strongyloidiasis should be routinely investigated in patients with CKD who have concomitant risk factors including high geographical prevalence. A high level of clinical suspicion is required to make the diagnosis of strongyloidiasis in at-risk patients so that lethal consequences can be prevented by early diagnosis and treatment.

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