


CASE REPORT

Open Access



Strongyloidiasis infection in a borderline lepromatous leprosy patient with adrenocortical insufficiency undergoing corticosteroid treatment: a case report

Rumala Morel^{1*} , Kusala Maddumabandara², Nisansala Amarasinghe³, Sujeewa Amarangani³, Anjalie Amarasinghe¹, Mihiri Gunathilaka¹, Gayani Wathsala¹, Lakmalee Bandara¹, Sunil Wijesundara¹, Nilupuli Gunaratne³, Roshitha Waduge³ and Arjuna Medagama²

Abstract

Background: Strongyloidiasis is a soil-transmitted helminthiasis mainly caused by *Strongyloides stercoralis*. It is endemic to the tropics and subtropics. Sri Lanka has a 0–1.6% prevalence rate. *S. stercoralis* infection was identified in a 33-year-old Sri Lankan male patient treated with corticosteroids for borderline lepromatous leprosy with adrenocortical dysfunction.

Case presentation: In March 2020, a 33-year-old Sri Lankan (Sinhalese) male patient presented with watery diarrhea, lower abdominal pain, and post-prandial abdominal fullness. Previously, he was diagnosed with borderline lepromatous leprosy and was treated with rifampicin, clofazimine, and prednisolone 60 mg daily since July 2019. After developing gastrointestinal symptoms, he had defaulted leprosy treatment including the prednisolone for 3 months. Duodenal biopsy revealed numerous intraepithelial nematodes within the lumina of glands in the duodenum whose appearance favored *Strongyloides*. Fecal wet smear revealed numerous *Strongyloides stercoralis* L1 rhabditiform larvae. Larval tracks were seen in the agar plate culture. L3 filariform larvae of *Strongyloides stercoralis* were seen in the Harada–Mori culture.

In addition, the short synacthen test revealed adrenocortical insufficiency, and oral hydrocortisone and fludrocortisone were started with albendazole treatment against strongyloidiasis. Fecal wet smear and culture repeated after treatment with albendazole were negative for *Strongyloides stercoralis*. The patient was discharged in July 2020 on oral hydrocortisone. One month later his condition was reviewed and the repeated fecal wet smear and agar plate culture was normal. He is being followed up every 3 months.

Conclusion: This is the first case of strongyloidiasis diagnosed in a patient with borderline lepromatous leprosy from Sri Lanka. The patient manifested symptoms of strongyloidiasis while on high-dose steroid therapy for his lepromatous reaction. Subsequently, the patient not only discontinued his steroid therapy, but also developed adrenocortical insufficiency as a complication of leprosy. Therefore, although diagnosis of strongyloidiasis was delayed, his

*Correspondence: rumala.morel@gmail.com

¹ Department of Parasitology, Faculty of Medicine, University of Peradeniya, Peradeniya, Kandy, Sri Lanka
Full list of author information is available at the end of the article



© The Author(s) 2022. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

subsequent low steroid levels probably protected him from disseminated disease. This is an interesting case where symptomatic strongyloidiasis was diagnosed in a patient who was initially treated with high-dose steroids but subsequently developed adrenocortical insufficiency. We emphasize the need to screen all patients prior to the commencement of immunosuppressive therapy.

Keywords: *Strongyloidis stercoralis*, Leprosy, Corticosteroid therapy, Adrenocortical insufficiency, Sri Lanka

Introduction

Strongyloidiasis is a soil-transmitted helminthiasis mainly caused by *Strongyloidis stercoralis* and, on rare occasions, by *Strongyloides fuelleborni fuelleborni* and *Strongyloidis fuelleborni kelleyi* [1]. Soil-dwelling filariform larvae can penetrate through the human skin when the host walks barefoot on contaminated soil. Filariform larvae migrate to the lungs, then into the small intestine and become adults. The female adult worms release eggs into the gastrointestinal tract [2, 3]. Strongyloidiasis is endemic to tropical and subtropical countries [1]. Sri Lanka which is a tropical country, has a 0–1.6% prevalence rate [4]. Previous cases report strongyloidiasis in transplant patients and immunocompromised patients in Sri Lanka [5–7]. Corticosteroids have a particularly strong and specific association with the development of hyperinfection syndrome and dissemination [8].

We report a case of strongyloidiasis in a patient with borderline lepromatous leprosy who was given corticosteroids to treat his lepromatous reaction. He later developed severe adrenocorticoid insufficiency, which necessitated placing him on lifelong steroids. This is an interesting case where strongyloidiasis was diagnosed in a patient who was previously on high dose steroids but then developed adrenocorticoid insufficiency and therefore had a low steroid level. To our knowledge this is also the first case report of strongyloidiasis in a patient with leprosy in Sri Lanka. Together with a literature review, this case reports *S. stercoralis* infection that was identified in a 33-year-old male patient who was on corticosteroids while being given anti-leprosy drugs.

Case presentation

In March 2020, a 33-year-old Sri Lankan (Sinhalese) male patient presented with watery diarrhea, lower abdominal pain, and post-prandial abdominal fullness to a rural hospital in the North Central Province of Sri Lanka. He was a member of the Civil Defense Force. Although the patient was transferred to a District General Hospital from the rural hospital, they were unable to diagnose the cause of his severe gastrointestinal symptoms. After 3 months he was transferred to a tertiary hospital in the Central Province, which was 170 km away. The patient had 3–4 episodes of loose stools per day, mainly of food particles without any blood or mucus. Previously, he was

diagnosed with borderline lepromatous leprosy and was treated since July 2019 with rifampicin, clofazimine, and 60 mg prednisolone daily. While he was on those drugs, he developed severe gastrointestinal symptoms and therefore defaulted leprosy treatment for 3 months.

The patient had severe loss of appetite and lost 15 kg of body weight during these 3 months. He did not have vomiting or nausea but had lower abdominal pain with post-prandial abdominal fullness. He denied cough, fever, shortness of breath, night sweats, urinary symptoms, dysuria, and joint pains. On examination, the patient was cachectic and pale. His body weight was 32 kg and he had no skin rashes. There was mild left iliac fossa tenderness with no hepatosplenomegaly.

His white blood cell count was high ($14.54 \times 10^3/\mu\text{L}$) with neutrophil predominance ($10.2 \times 10^3/\mu\text{L}$). The eosinophil count was normal. Hemoglobin level was marginally low ($10.20/\mu\text{L}$) and the platelet counts were normal ($436 \times 10^3/\mu\text{L}$). C-reactive protein was normal (2.8 mg/L), sodium (Na^+) level was low (119 mmol/L), and potassium (K^+) was in the lower margin of the normal range (4.20 mmol/L). Urinary Na^+ (47 mEq/L) and urinary K^+ (23.5 mEq/L) were high. His serum creatinine was normal (50.4 $\mu\text{mol/L}$). Total protein was low (38.4 g/L) with a very low albumin level (17.5 g/L). Aspartate aminotransferase test (AST), alanine aminotransferase test (ALT), and international normalized ratio (INR) were normal. The blood picture was compatible with anemia of chronic disease.

His Mantoux test was negative, and Hepatitis B and C, as well as human immunodeficiency viruses (HIV) tests were also negative. His CD4, CD8, and CD19 cell counts were normal. The short Synacthen test was positive (9.00 am—24 nmol/L; 9.30 am—12 nmol/L) and adrenocortical insufficiency was diagnosed. A short Synacthen test is used for diagnosis of adrenocortical insufficiency on the basis of the measurement of serum cortisol before and after injection of synthetic adrenocorticotrophic hormone (ACTH). A cortisol level of more than 420 nmol/L at 30 min post-Synacthen indicates an adequate adrenal response. Computed tomography (CT) scan of the chest and abdomen was normal except for bilateral small adrenal glands, which did not show any calcifications.

The patient was started on oral steroids (oral hydrocortisone 35 mg daily and fludrocortisone 100 μg daily) in

June 2020. Complement factors C3 was normal (71 mg/dL), C4 was marginally low (19 mg/dL), immunoglobulin G (IgG) was normal (966 mg/dL), immunoglobulin M (IgM) was low (39 mg/dL), and immunoglobulin A (IgA) was normal (209 mg/dL).

Owing to persisting diarrhea, upper gastrointestinal endoscopy (UGIE), duodenal biopsy, colonoscopy, and biopsy were performed. A severely inflamed oedematous duodenum was noted. The ileum was not inflamed and the gross appearance was normal. Duodenal (D2) biopsy

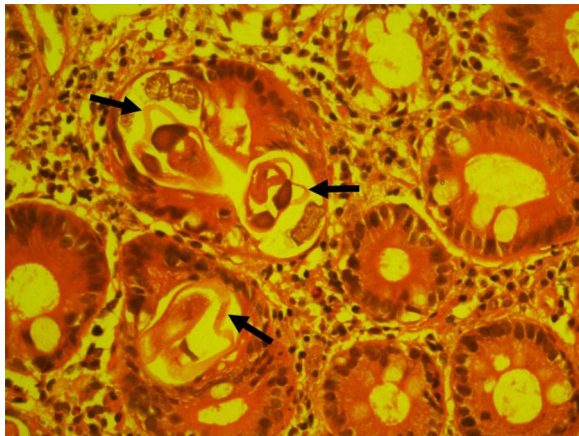


Fig. 1 Intraepithelial nematodes are indicated with arrowheads observed in the duodenal (D2) biopsy within the lumina of glands in the duodenum. Duodenal biopsy—arrowheads indicate cross sections of intraepithelial nematodes (*S. stercoralis* larvae) within the lumina of glands in the duodenum

revealed numerous intraepithelial nematodes within the lumina of glands in the duodenum, and the appearance was that of parasitic infestation favoring *Strongyloides* spp. (Fig. 1). Biopsies taken from the ileum, caecum, and the rest of the colon were normal. Fecal wet smear revealed numerous *S. stercoralis* L1 rhabditiform larvae (Fig. 2a). Coproculture using the Harada–Mori technique yielded L3 filariform larvae, which confirmed the species identification by its long esophagus and the tail with a notched tip (Fig. 2b). Larval tracks were seen in the agar plate culture of feces. The patient was diagnosed with strongyloidiasis and was treated with albendazole 400 mg twice a day for 7 days according to the CDC, USA treatment guidelines.

Outcome and follow-up

With this treatment, his appetite improved and he was started on a high-protein diet. Oral hydrocortisone (daily dose 35 mg) and fludrocortisone 100 µg daily were continued. Fecal wet smear and agar plate culture repeated after treatment with albendazole were negative for *S. stercoralis*. The patient was discharged on 20 July 2020 on oral hydrocortisone (daily dose 25 mg). One month later, in August 2020, his condition was reviewed. The repeated fecal wet smear and culture did not reveal *S. stercoralis* larvae. Fecal smears and agar plate cultures carried out on the patient's wife and child were also normal. Patient continues to be followed up every 3–6 months as he is on lifelong steroid therapy. He was also advised to use footwear.

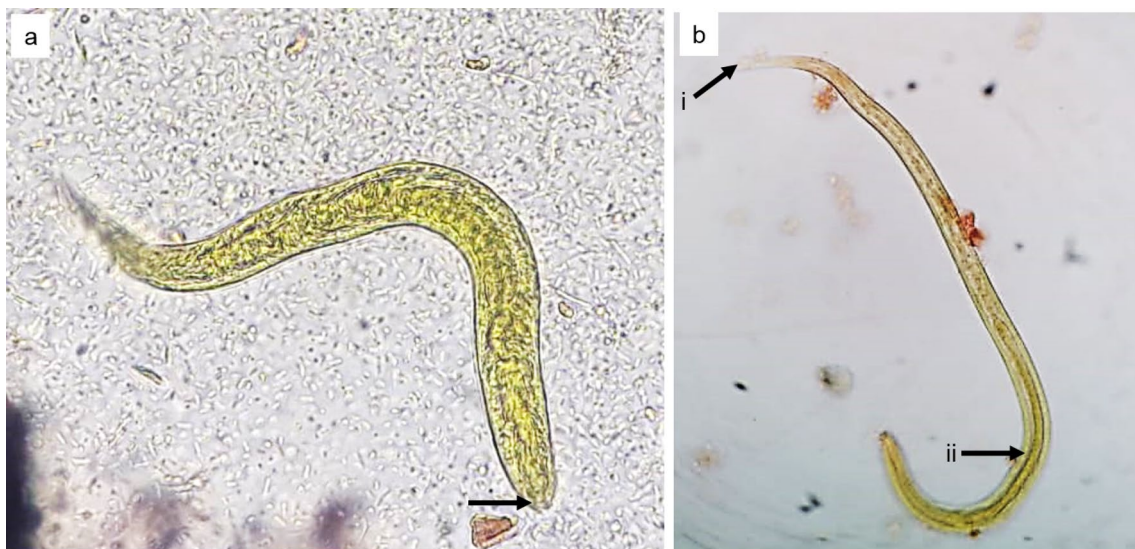


Fig. 2 **a** *S. stercoralis* L1 rhabditiform larva seen in the fecal wet smear. The arrowhead indicates shallow buccal cavity. **b** L3 filariform larva of *S. stercoralis* seen in the Harada Mori culture. Arrowheads indicate (i) the notched tip of the tail and (ii) the oesophagus which is half of the body length of the larva

Discussion and conclusions

Strongyloides stercoralis is a unique intestinal helminth species with a self-perpetuating life cycle. This is responsible for its potential lifelong infection and capacity to kill its human host. It is estimated that 30–100 million people are infected worldwide [1]. Potentially life-threatening disseminated strongyloidiasis in immunocompromised patients results in mortality rates of up to 85% [8]. Strongyloidiasis is usually asymptomatic and often only indicated by a raised eosinophil count. However, it can present with dermatological or gastrointestinal symptoms [3].

Steroids administered to treat underlying conditions represent the main trigger predisposing to *Strongyloides* hyperinfection syndrome (SHS) and disseminated strongyloidiasis [9, 10]. SHS represents the increased number of larvae within the traditional reproductive route (skin, GI tract, lungs), while disseminated strongyloidiasis involves widespread dissemination of larvae outside the GI tract and lungs (liver, heart, brain, urinary tract) [10]. Increased number of larvae in stool and sputum is the hallmark of SHS [8]. The gastrointestinal manifestations include severe abdominal cramping, pain, watery diarrhea, nausea, vomiting, weight loss, occasionally gastrointestinal bleeding, small bowel obstruction, and pulmonary manifestations with cough, wheezing, pulmonary hemorrhage, pleural effusion, and acute respiratory failure [2]. Bacteria carried with the larvae into the bloodstream can lead to gram-negative sepsis which may be fatal, especially in immunosuppressed patients [11].

This patient is on 25 mg hydrocortisone daily. A high dose of steroids is defined as more than 15–20 mg for longer than 2–4 weeks [12]. However, dose, duration, or route of administration of drugs is not related to SHS, as even a short course of steroids (6–17 days) in immunocompetent patients can be associated with fatal SHS [8].

Previous cases reporting *Strongyloides* infection in patients with leprosy undergoing steroid treatment have been identified from Asia [13–15]. Patients with leprosy and *S. stercoralis* infection have developed SHS due to steroids being administered to patients with leprosy [16, 17]. The patient that we report in this case discontinued his leprosy treatment, including prednisolone, owing to his severe gastrointestinal symptoms. Thereafter, he developed adrenocortical insufficiency. The resulting low cortisol levels probably protected him from developing SHS. Thus, suffering a complication of leprosy protected this patient from complications due to infection with *S. stercoralis*. Subclinical adrenocortical insufficiency has been reported in patients with leprosy [18]. This patient, however, developed severe adrenocortical insufficiency with reduced volume of bilateral adrenal glands, which necessitated long-term steroid therapy.

Strongyloides stercoralis can cause chronic eosinophilia in humans owing to auto-infection within the human host [19]. Although this patient had increased neutrophils, his eosinophil count was normal. Corticosteroids can reduce the levels of circulating eosinophils by inhibiting their proliferation and increasing apoptosis [20].

Fecal examination for *Strongyloides* larvae and enzyme-linked immunosorbent assay (ELISA) for IgG antibodies, as well as eosinophilia in peripheral blood, are recommended to screen kidney donors and recipients. Since *S. stercoralis* can be dormant in an infected person and can cause SHS when the patient becomes immunocompromised, patients with strongyloidiasis should be treated before transplantation and commencement of immunosuppressive therapy [21, 22]. Therefore it is important to continue investigating for strongyloidiasis in this patient, as he is on lifelong treatment for adrenocortical insufficiency and longstanding chronic infection is well recorded [23–25].

Strongyloidiasis is mainly diagnosed by the detection of larvae found in feces. Coproculture techniques such as Harada–Mori filter paper, charcoal, and agar plate cultures are more labor intensive but have a higher sensitivity than direct fecal smears [26]. These techniques are used to specifically differentiate *S. stercoralis* infection from other intestinal nematode infections [27, 28]. In this case the diagnosis of strongyloidiasis was delayed for more than 3 months. Fecal examination for *S. stercoralis* was done only after the duodenal (D2) biopsy showed intraepithelial nematodes within the lumina of glands in the duodenum (Fig. 1).

This patient was treated with albendazole 400 mg twice a day for 7 days while continuing his steroid therapy. Several drugs have been recommended for the treatment of strongyloidiasis, such as albendazole, mebendazole, thiabendazole, and ivermectin. Ivermectin is more effective for SHS, primarily when associated with predisposing conditions such as acquired immunodeficiency syndrome [29]. A previous case [5] in Sri Lanka highlighted the importance of careful follow-up of cases after treatment. The patient in this case was reviewed after 1 month and the repeated fecal wet smear and culture were normal. We plan to continue to follow-up with him every 3 months, as he will be on lifelong steroid therapy owing to adrenocortical insufficiency.

This is the first case of strongyloidiasis diagnosed in a patient with borderline lepromatous leprosy from Sri Lanka. Strongyloidiasis was not considered in the differential diagnosis until the duodenal biopsy showed nematode larvae. If there is greater awareness of strongyloidiasis among clinicians, the patient could have been treated in the rural hospital where he was initially

admitted. As it is, he had to suffer for 3 months until he was finally transferred to a hospital 170 km away.

This case also highlights an interesting situation where, in spite of the delay in diagnosis of strongyloidiasis, the patient was protected from disseminated disease owing to developing adrenocorticoid insufficiency as a complication of leprosy.

Although Sri Lanka is an endemic country for strongyloidiasis, the recorded prevalence is very low [4]. The Centers for Disease Control and Prevention (CDC), USA guidelines state that physicians should screen for strongyloidiasis prior to starting corticosteroids or other immunosuppressants [30]. This case emphasizes the need for such screening.

Abbreviations

LIF: Left iliac fossa; CRP: C-reactive protein; CDC: Centers for Disease Control and Prevention; Na⁺: Sodium ion; K⁺: Potassium ion; AST: Aspartate aminotransferase test; ALT: Alanine aminotransferase test; INR: International normalized ratio; HIV: Human immunodeficiency viruses; CECT: Computed tomography; IgG: Immunoglobulin G; IgM: Immunoglobulin M; IgA: Immunoglobulin A; UGIE: Upper gastrointestinal endoscopy; SHS: Strongyloides hyperinfection syndrome; ELISA: Enzyme-linked immunosorbent assay.

Acknowledgements

We would like to thank Mr. Harsha Wickramanayake and Mr. M. Sangar in the Department of Parasitology, Faculty of Medicine, University of Peradeniya for their assistance.

Author contributions

Management of the patient was carried out by AM and KM. Histology of duodenal biopsy was checked by NA, SA, NG, and RW. Diagnosis of strongyloidiasis by fecal examination was done by RM, GW, LB, and SW. Article was initially drafted by MTG, AA, and RM. All authors reviewed and contributed to the final version of this case report. All authors read and approved the final manuscript.

Funding

None.

Availability of data and materials

The low prevalence rate of strongyloidiasis reported in Sri Lanka is due to poor diagnosis. Physicians do not suspect strongyloidiasis and therefore do not test for it. This is clearly shown in this case report where the patient suffered for six months and was transferred a long distance away to a tertiary hospital before he was diagnosed. If he was screened prior to starting immunosuppressive therapy this would not have happened.

Declarations

Ethics approval and consent to participate

Obtained written consent from patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

Authors declare that they have no competing interests.

Author details

¹Department of Parasitology, Faculty of Medicine, University of Peradeniya, Peradeniya, Kandy, Sri Lanka. ²Department of Medicine, Faculty of Medicine, University of Peradeniya, Peradeniya, Kandy, Sri Lanka. ³Department

of Pathology, Faculty of Medicine, University of Peradeniya, Peradeniya, Kandy, Sri Lanka.

Received: 13 December 2020 Accepted: 7 November 2022

Published online: 09 December 2022

References

- Schär F, Trostorf U, Giardina F, Khieu V, Muth S, Marti H, Vounatsou P, Odermatt P. *Strongyloides stercoralis*: global distribution and risk factors. *PLoS Negl Trop Dis*. 2013;7(7): e2288.
- Keiser PB, Nutman TB. *Strongyloides stercoralis* in the immunocompromised population. *Clin Microbiol Rev*. 2004;17(1):208–17.
- Greaves D, Coggie S, Pollard C, Aliyu SH, Moore EM. *Strongyloides stercoralis* infection. *BMJ*. 2013;347: f4610.
- Sorensen E, Ismail M, Amarasinghe DK, Hettiarachchi I, Dassanaike TS. The prevalence and control of soil-transmitted nematode infections among children and women in the plantations in Sri Lanka. *Cey Med J*. 1996;41(2):37–41.
- Rodrigo KM, Premaratna R, Chandrasena TG, de Silva NR, de Silva HJ. Marked eosinophilia due to intestinal strongyloidiasis in an immunocompetent patient. *Cey Med J*. 2012;57(4):172–3.
- Britt DP, Khan HA, Florentino AO. Strongyloides infection in an immunosuppressed patient. *Med Princ Pract*. 1989;1(1):63–4.
- Gunathilaka N, Chandrasena N, Wijerathna T, Fuji Y, Gunasekara D, Gunatilaka RP, Premaratna R. Descriptive investigation of strongyloidiasis infection and characterization of *Strongyloides stercoralis* using morphological and molecular-based methods. *Case Rep Infect Dis*. 2020. <https://doi.org/10.1155/2020/5431491>.
- Mejia R, Nutman TB. Screening, prevention, and treatment for hyperinfection syndrome and disseminated infections caused by *Strongyloides stercoralis*. *Curr Opin Infect Dis*. 2012;25(4):458.
- Buonfrate D, Requena-Mendez A, Angheben A, Muñoz J, Gobbi F, Van Den Ende J, Bisoffi Z. Severe strongyloidiasis: a systematic review of case reports. *BMC Infect Dis*. 2013;13(1):78.
- Fardet L, Gènereau T, Poirot JL, Guidet B, Kettaneh A, Cabane J. Severe strongyloidiasis in corticosteroid-treated patients: case series and literature review. *J Infect*. 2007;54(1):18–27.
- Shorman M, Al-Tawfiq JA. *Strongyloides stercoralis* hyperinfection presenting as acute respiratory failure and gram-negative sepsis in a patient with astrocytoma. *Int J Infect Dis*. 2009;13:e288–91.
- Shroff A, Mertz D. Infectious diseases risk while on chronic, high-dose corticosteroids. *Can J Gen Intern Med*. 2017;12(1):162.
- Leang B, Lynen L, Tootill R, Griffiths S, Monchy D. Death caused by strongyloides hyperinfection in a leprosy patient on treatment for a type II leprosy reaction. *Lepr Rev*. 2004;75:398–403.
- Darlong J. Strongyloides hyper infection in a steroid dependent leprosy patient. *Lepr Rev*. 2016;87(4):536–43.
- Prabha N, Chhabra N. Prevention of *Strongyloides stercoralis* hyperinfection in leprosy patients on long-term steroid therapy. *Indian J Dermatol Venereol Leprol*. 2018;84(6):709.
- Younger B, Michaud RM, Fischer M. Leprosy: our southeast Asian refugee experience. *Arch Dermatol*. 1982;118(12):981–4.
- Mutreja D, Sivasami K, Tewari V, Nandi B, Nair GL, Patil SD. A 36-year-old man with vomiting, pain abdomen, significant weight loss, hyponatremia, and hypoglycemia. *Indian J Pathol Microbiol*. 2015;58(4):500.
- Gupta A, Sharma PK, Garga UC, et al. Adrenal cortical function and adrenal volume in leprosy: a study of 40 cases. *Lepr Rev*. 2018;89(2):148–57.
- Nutman TB. Human infection with *Strongyloides stercoralis* and other related *Strongyloides* species. *Parasitology*. 2017;144(3):263.
- Corrigan CJ. Cellular effects of glucocorticoids. *Immunol Allergy Clin North Am*. 1999;19:671–82.
- Abdhalamid BA, Al Abadi AN, Al Saghier MI, Joudeh AA, Shorman MA, Amr SS. *Strongyloides stercoralis* infection in kidney transplant recipients. *Saudi J Kidney Dis Transpl*. 2015;26(1):98.
- Altintop L, Cakar B, Hokelek M, Bektaş A, Yildiz L, Karaoglanoglu M. *Strongyloides stercoralis* hyperinfection in a patient with rheumatoid arthritis and bronchial asthma: a case report. *Ann Clin Microbiol Antimicrob*. 2010;9(1):27.

23. Gill GV, Beeching NJ, Khoo S, Bailey JW, Partridge S, Blundell JW, Luksza AR. A British Second World War veteran with disseminated strongyloidiasis. *Trans R Soc Trop Med Hyg.* 2004;98(6):382–6.
24. Gill GV, Bell DR, Reid HA. Strongyloidiasis in ex-Far East prisoners of war. *BMJ.* 1977;1(6067):1007.
25. De Silva S, Saykao P, Kelly H, MacIntyre CR, Ryan N, Leydon J, Biggs BA. Chronic *Strongyloides stercoralis* infection in Laotian immigrants and refugees 7–20 years after resettlement in Australia. *Epidemiol Infect.* 2002;128(3):439–44.
26. Requena-Méndez A, Chiodini P, Bisoffi Z, Buonfrate D, Gotuzzo E, Muñoz J. The laboratory diagnosis and follow up of strongyloidiasis: a systematic review. *PLoS Negl Trop Dis.* 2013;7(1): e2002.
27. De Kaminsky RG. Evaluation of three methods for laboratory diagnosis of *Strongyloides stercoralis* infection. *J Parasitol.* 1993;79:277–80.
28. Morel R, Ekanayake S, Abeykoon S. *Strongyloides stercoralis* isolated by agar plate culture. *Cey Med J.* 2007;52:150.
29. Orem J, Mayanja B, Okongo M, Morgan D. *Strongyloides stercoralis* hyperinfection in a patient with AIDS in Uganda successfully treated with ivermectin. *Clin Infect Dis.* 2003;37(1):152–3.
30. https://www.cdc.gov/parasites/strongyloides/health_professionals/index.html#tx.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more biomedcentral.com/submissions

