

Systemic Strongyloides Stercoralis Hyperinfection Presenting as Multifocal Transmural Enteritis in a Paediatric Patient: A Case Report

Dr. Nickle Sasidharan¹ Dr. Jyoti Goyal² Dr. Rajeev Chowdry³ Dr. Bhawesh Kumar Thakur⁴

¹ Department of Critical Care Medicine, Yatharth Super Speciality Hospital, Faridabad, Haryana, India

² Department of Internal Medicine, Yatharth Super Speciality Hospital, Faridabad, Haryana, India

***Corresponding Author:** Dr Jyoti Goyal, MD, IDCCM; Department of Critical Care Medicine, Yatharth Super Speciality Hospital, Sector 20, Faridabad, Haryana 121001, India.

Abstract & Background

Strongyloides Stercoralis is a soil-transmitted nematode capable of autoinfection and potentially fatal Hyperinfection. Paediatric cases presenting as multifocal transmural enteritis are infrequently reported in the literature. The Th2-polarised immune signature — markedly elevated serum immunoglobulin E and peripheral eosinophilia alongside normal acute-phase reactants — is a pivotal but under recognised diagnostic clue distinguishing helminthic infection from granulomatous, malignant pathology and allergic primary eosinophilic gastroenteritis (EoGE).

Case Presentation

A 12-year-old South Asian (Indian) male presented with a 2-day history of severe abdominal pain, vomiting, and generalised weakness following a soil-contaminated toe injury three weeks prior. Contrast-enhanced computed tomography of the abdomen demonstrated circumferential mural thickening from the second part of the duodenum to the ileocecal junction (4-5 mm), mesenteric lymphadenopathy, hepatosplenomegaly, and mild ascites. Laboratory investigations revealed peripheral eosinophilia (7.8%), markedly elevated serum immunoglobulin E at 1055 IU/mL (reference less than 100 IU/mL), microcytic hypochromic anaemia, and critically normal C-reactive protein (less than 6 mg/L) and erythrocyte sedimentation rate (8 mm/hr). Duodenal biopsy revealed mucosal eosinophilia of 30-40 per high-power field, with complete absence of granulomata and cellular atypia, histologically excluding intestinal tuberculosis and lymphoma. Routine stool microscopy was negative. Treatment comprised weight-based ivermectin (200 micrograms per kilogram per day for 7 days) and albendazole (400 mg per day for 3 days).

Conclusions

Colicky pain abdomen with triad of eosinophilia, markedly elevated immunoglobulin E, and normal acute-phase reactants is diagnostically strongly suggestive of systemic helminthic infection with relevant clinical background. Epidemiological timeline reconstruction is indispensable. Corticosteroids are absolutely contraindicated before exclusion of strongyloidiasis. Empiric weight-based antiparasitic therapy should not be delayed pending microbiological confirmation when clinical probability is high.

Keywords: *Strongyloides Stercoralis*; Strongyloidiasis; Hyperinfection; Eosinophilia; Immunoglobulin E; Transmural Enteritis; Paediatric; Case report.

Introduction

Strongyloides Stercoralis is estimated to infect more than 100 million individuals globally, with the highest prevalence in Southeast Asia, sub-Saharan Africa, and the Indian subcontinent [1]. Unlike other soil-transmitted helminths, *Strongyloides* is uniquely capable of autoinfection — converting rhabditiform larvae to infective filariform larvae within the host colon — enabling persistent infection lasting decades and, under certain conditions can lead to life-threatening Hyperinfection with dissemination to the lungs, central nervous system, and bloodstream [2].

Paediatric *Strongyloides* cases presenting as multifocal transmural enteritis, mimicking intestinal tuberculosis or inflammatory bowel disease, are rarely described in the published literature [3]. The key diagnostic clue — the Th2-polarised immune response characterised by elevated immunoglobulin E (IgE), eosinophilia, and paradoxically normal C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) — remains underappreciated in routine clinical practice [4]. We report a case of systemic *Strongyloides Stercoralis* Hyperinfection in a 12-year-old male presenting as multifocal transmural enteritis, in whom systematic integration of the epidemiological timeline, immunological profile and duodenal biopsy established the diagnosis and directed targeted antiparasitic therapy.

Case Presentation

A 12-year-old male (South Asian ethnicity; approximate weight 35 kg) presented to the emergency department of Yatharth Super Speciality Hospital, Faridabad, Haryana, India, with a 2-day history of severe diffuse abdominal pain, multiple episodes of vomiting, mild fever (38.1°C), and constipation. Three weeks prior he had sustained an injury to his right little toe with significant soil contact. Two weeks prior, he developed bilateral lower extremity pain, attributed at that time to a viral illness. There was no history of prior hospitalisation, corticosteroid use, immunosuppressive therapy, known tuberculosis contact, food allergy, or atopic disease. The family resided in a peri-urban area with access to open ground.

On examination, the abdomen was diffusely tender with voluntary guarding but without peritoneal signs. Bowel sounds were present. No organomegaly was clinically detectable. Vital signs were otherwise stable.

Investigations

Complete blood count revealed haemoglobin 11.6 g/dL (mean corpuscular volume 76.8 fL, mean corpuscular haemoglobin 24.1 pg, red cell distribution width 15.0%), consistent with microcytic hypochromic anaemia. Total leucocyte count was 8870 per cu mm with peripheral eosinophilia at 7.8% (absolute eosinophil count 691 per cumm (reference range <100 IU/mL), exceeding ten times the upper limit of normal. CRP was <6 mg/L (normal <10 mg/L) and ESR was 8 mm/hr (normal <20 mm/hr), both well within normal limits. Liver function tests were normal with albumin 4.7 g/dL. Serum IgE was markedly elevated at 1055 IU/mL. Results are summarised in Table 3. Routine stool examination for ova and cysts was negative. Standard direct microscopy has recognised limitations in detecting ***Strongyloides*** larvae, as the organism is excreted in larval rather than egg form, requiring specific concentration techniques.

Pathology and Microbiology

[5]. Duodenal biopsy obtained at upper gastrointestinal endoscopy (Histo No. 1264/26) revealed chronic nonspecific duodenitis with mucosal eosinophilia of 30–40 eosinophils per high-power field. Current literature defines eosinophilic gastrointestinal disease [5]. Duodenal biopsy obtained at upper gastrointestinal endoscopy (Histo No. 1264/26) revealed chronic nonspecific duodenitis with mucosal eosinophilia of 30–40 eosinophils per high-power field. Current literature defines eosinophilic gastrointestinal disease (EGID) in the duodenum as ≥ 30 eosinophils per high-power field in ≥ 3 high-power fields; a stricter transcriptomic threshold of >52 peak eosinophils per high-power field has also been described [6, 7]. Critically, granulomata and cellular atypia were completely absent (Table 1), histologically excluding intestinal tuberculosis and lymphoma. Absence of visible larvae on biopsy reflects the recognised sampling limitations of small-specimen forceps biopsy for an organism residing deep within intestinal crypts.

Imaging. Contrast-enhanced CT abdomen (Figure 1) demonstrated circumferential mural thickening of the D2–D3 duodenum (4–5 mm) and terminal ileum with ileocaecal junction involvement (4–4.5 mm), mucosal fold thickening of proximal jejunal loops, extensive mesenteric lymphadenopathy (5–8 mm nodes at the root of mesentery and right iliac fossa), mild free intraperitoneal fluid, hepatomegaly (15.6 cm), and splenomegaly (11.1 cm). No free intraperitoneal air was identified

UGI endoscopy:

Diagnostic Synthesis

Integration of the three-week illness timeline (Table 2), three-phase illness progression, and immunological profile established the working diagnosis of systemic *Strongyloides Stercoralis* Hyperinfection with probable co-infection with *Ancylostoma duodenale*. The differential diagnosis and exclusion rationale are presented in Table 2.

Management

Ivermectin was prescribed at 200 micrograms/kg/day as a single daily oral dose on an empty stomach for 7 days (extended hyperinfection protocol) [8]. The patient's weight was confirmed at 35 kg prior to dosing. Albendazole 400 mg once daily with a fatty meal for 3 days was co-administered for hookworm coverage along with supportive care with IV fluids and electrolyte corrections. Corticosteroids and stimulant laxatives were explicitly withheld (Table 4).

Outcome

Clinical improvement in abdominal pain and tolerance of oral intake was noted by day 3 of antiparasitic therapy. Follow-up serology and complete blood count were arranged at 2 and 4 weeks to confirm parasitological cure and eosinophil normalisation.

Discussion

This case illustrates the diagnostic value of systematically integrating epidemiological history, clinical timeline reconstruction, immunological profiling and finding of multifocal transmural enteritis in a child presenting with colicky abdominal pain. The radiological differential — intestinal tuberculosis, typhoid enteritis, Crohn's disease, and lymphoma — was excluded through convergent evidence: absence of granulomata and cellular atypia on duodenal biopsy, normal acute-phase reactants, and a classical Th2 immunological signature [4, 9]. *Strongyloides stercoralis* induces a Th2-polarised immune response characterised by interleukin 4-driven IgE class switching, interleukin 5-mediated eosinophil recruitment, and interleukin 10-mediated suppression of the Th1 axis [4]. This explains the paradox of severe intestinal inflammation with normal CRP and ESR. It is prudent to understand that this immunological signature is a positive diagnostic finding, not an incidental absence of inflammation. Clinicians who interpret normal acute-phase reactants as reassuring in the presence of eosinophilia and markedly elevated IgE risk catastrophic diagnostic delay. The three-week soil-exposure to admission timeline corresponds to the established biological phases of *Strongyloides* infection: filariform larval skin penetration at the site of the toe wound (week 0); transpulmonary migration through the musculoskeletal compartment with eosinophilic myositis during the Loeffler phase (weeks 1–2); and intestinal colonisation of the duodenum and jejunum causing transmural enteritis (days before admission) [2]. Portal circulation larval migration accounts for the hepatosplenomegaly identified on computed tomography [2]. Negative routine stool microscopy does not exclude *Strongyloides* infection, as the organism is shed as larvae rather than eggs, requiring specific larval concentration methods [5]. The Baermann technique with charcoal pre-incubation achieves substantially higher sensitivity than conventional Baermann, as demonstrated by Gelaye et al. [10]. Similarly, the absence of larvae on a small-specimen duodenal biopsy is consistent with the known sampling limitations of forceps biopsy for an organism residing deep within intestinal crypts [5]. Food allergy and primary eosinophilic gastroenteritis (EoGE) are important differentials in any child with duodenal eosinophilia. EoGE is characterised by elevated IgE, peripheral eosinophilia, and eosinophilic infiltration on biopsy — an

immunological overlap with helminthic infection [6, 7]. Distinguishing features favouring helminthic aetiology in this case included: soil-exposure epidemiological history, a three-phase febrile illness with bilateral myositis consistent with larval migration, acute systemic presentation rather than a chronic dietary-triggered course, and no personal or family history of atopy or food allergy. The biopsy count of 30–40 eosinophils per high-power field meets the EGID diagnostic threshold, underlining the importance of clinical context in interpreting duodenal eosinophilia. The absolute contraindication to corticosteroids in unexcluded *Strongyloides* infection is well established [9]. Geri et al. reported an intensive care unit mortality of 60.3% in a multicentre series of 133 patients with *Strongyloides* hyperinfection syndrome, with corticosteroid use present in 83.5% of cases [9]. Ivermectin at 200 micrograms/kg achieves cure rates of 93–96% in randomised controlled trials [8]. Weight-based dosing is essential in paediatric patients; adult fixed dosing risks underdosing in children below 60 kg [2].

Conclusions

Systemic *Strongyloides stercoralis* should be considered in any paediatric patient with multifocal enteritis, peripheral eosinophilia, and markedly elevated serum IgE — particularly following soil exposure in an endemic region. The Th2 immune signature with normal acute-phase reactants is diagnostically decisive and effectively excludes tubercular and bacterial aetiology. Empiric weight-based ivermectin therapy must not be delayed pending microbiological confirmation when clinical probability is high, and corticosteroids are absolutely contraindicated prior to exclusion of this diagnosis.

Declarations

Ethics approval and consent to participate: This case report was prepared in accordance with the October 2024 revision of the Declaration of Helsinki. As a case report describing routine clinical care, formal ethics committee approval was not required under institutional policy. Written informed consent for participation was obtained from the patient's parent/legal guardian.

Consent for publication: Written informed consent for publication of clinical data and images was obtained from the patient's parent/legal guardian prior to submission. A signed consent form is held by the corresponding author and is available for review by the Editor-in-Chief on request.

Availability of data and materials: All relevant data generated or analysed during this study are included within this published article and its supplementary information files.

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Authors' contributions: JG: Conceptualisation, clinical management, supervision, manuscript review and editing. NS: Data curation, formal analysis, investigation, writing — original draft, literature review. RC: Patient referral, clinical correlation, manuscript review and editing. All authors read and approved the final manuscript.

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Care Checklist: The CARE 2013 case report guideline checklist has been completed and is submitted as a supplementary file with this manuscript.

References

1. Puthiyakunnon S, Boddu S, Li Y, Zhou X, Wang C, Li J, et al. Strongyloidiasis—an insight into its global prevalence and management. *PLoS Negl Trop Dis*. 2014;8(8): e3018. doi: 10.1371/journal.pntd.0003018 [PMID: 25121962].
2. Gordon CA, Utzinger J, Muhi S, Becker SL, Keiser J, Khieu V, et al. Strongyloidiasis. *Nat Rev Dis Primers*. 2024;10(1):6. doi:10.1038/s41572-023-00490-x [PMID: 38272922].
3. Sankar J, Gupta S, Lingaiah R. Strongyloides stercoralis hyperinfection and intractable pulmonary haemorrhage. *J Pediatr*. 2020; 218:258. doi: 10.1016/j.jpeds.2019.10.052 [PMID: 31740146].
4. 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):458–463. doi: 10.1097/QCO.0b013e3283551dbd [PMID: 22691685].
5. Buonfrate D, Formenti F, Perandin F, Bisoffi Z. Novel approaches to the diagnosis of Strongyloides stercoralis infection. *Clin Microbiol Infect*. 2015;21(6):543–552. doi: 10.1016/j.cmi.2015.04.001 [PMID: 25887711].
6. Reed CC, Genta RM, Youngblood BA, Wechsler JB, Dellon ES. Mast cell and eosinophil count in gastric and duodenal biopsy specimens from patients with and without eosinophilic gastroenteritis. *Clin Gastroenterol Hepatol*. 2021;19(10):2102–2111. doi: 10.1016/j.cgh.2020.08.013 [PMID: 32801015].
7. Shoda T, Rochman M, Collins MH, Caldwell JM, Mack LE, Osswald GA, et al. Molecular analysis of duodenal eosinophilia. *J Allergy Clin Immunol*. 2023;151(4):1027–1039. doi: 10.1016/j.jaci.2022.12.814 [PMID: 36592704].
8. Sprecher VP, Hofmann D, Savathdy V, Xayavong P, Norkhankhame C, Huy R, et al. Efficacy and safety of moxidectin compared with ivermectin against Strongyloides stercoralis infection in adults in Laos and Cambodia: a randomised, double-blind, non-inferiority, phase 2b/3 trial. *Lancet Infect Dis*. 2024;24(2):196–205. doi:10.1016/S1473-3099(23)00507-8 [PMID: 37949090].
9. Geri G, Rabbat A, Mayaux J, Zafrani L, Chalumeau-Lemoine L, Guidet B, et al. Strongyloides stercoralis hyperinfection syndrome: a case series and a review of the literature. *Infection*. 2015;43(6):691–698. doi:10.1007/s15010-015-0799-1 [PMID: 26008854].
10. Gelaye W, Williams NA, Kepha S, Junior AM, Fleitas PE, Marti-Soler H, et al. Performance evaluation of Baermann techniques: the quest for developing a microscopy reference standard for the diagnosis of Strongyloides stercoralis. *PLoS Negl Trop Dis*. 2021;15(2): e0009076. doi: 10.1371/journal.pntd.0009076 [PMID: 33600434].

Tables

Table 1 Histopathological findings — duodenal biopsy

<u>Finding</u>	<u>Result</u>	<u>Interpretation</u>
Specimen size	0.3 × 0.3 × 0.2 cm (one block)	Small specimen: sampling limitations apply
Mucosal architecture	Duodenal mucosa present	Adequate for histological interpretation
Inflammatory infiltrate	Lymphocytes, plasma cells, neutrophils, eosinophils in lamina propria	Chronic active inflammation
Eosinophil count	30–40 per high-power field	Meets EGID diagnostic threshold ($\geq 30/\text{hpf}$ in ≥ 3 hpf [6]); below EoD transcriptomic threshold of $>52/\text{hpf}$ [7]
Granulomata	Absent	Intestinal tuberculosis effectively excluded
Cellular atypia	Absent	Lymphoma and malignancy excluded histologically
Parasitic larvae	Not identified	Does not exclude Strongyloides; biopsy sensitivity limited by small specimen size and superficial sampling
Pathologist impression	Chronic nonspecific duodenitis with slight increase in eosinophils	Clinical correlation advised

Histo No. 1264/26; specimen collected 28 February 2026; reported 2 March 2026. EGID, eosinophilic gastrointestinal disease; EoD, eosinophilic duodenitis; hpf, high-power field.

Table 2 Differential diagnosis and systematic exclusion

<u>Diagnosis</u>	<u>Expected features</u>	<u>Findings in this patient</u>	<u>Status</u>
Strongyloides hyperinfection	Elevated IgE, eosinophilia, normal CRP/ESR, soil exposure, three-phase illness timeline	IgE 1055 IU/mL, eosinophilia 7.8%, CRP/ESR normal, confirmed soil-contaminated toe wound 3 weeks prior	PRIMARY DIAGNOSIS
Intestinal tuberculosis	Elevated ESR/CRP, granulomata on biopsy	Normal CRP/ESR; granulomata absent on biopsy	Excluded
Typhoid enteritis	Elevated CRP, eosinopenia	Normal CRP; eosinophilia present	Excluded
Crohn's disease	Elevated CRP/ESR, granulomata	Normal CRP/ESR; granulomata absent	Excluded

<u>Diagnosis</u>	<u>Expected features</u>	<u>Findings in this patient</u>	<u>Status</u>
Food allergy / Eosinophilic gastroenteritis	Elevated IgE and eosinophilia; chronic dietary-triggered course; history of atopy; no fever	No atopy or food allergy history; acute febrile systemic illness; soil-exposure epidemiology inconsistent with dietary trigger	Considered; excluded by epidemiology and systemic features
Intestinal lymphoma	Elevated LDH/ESR, cellular atypia on biopsy	Normal uric acid/ESR; no atypia on biopsy	Excluded
HLH	Ferritin >10,000, cytopenias, elevated CRP	Normal CRP; no cytopenias	Excluded

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; HLH, haemophagocytic lymphohistiocytosis; IgE, immunoglobulin E; LDH, lactate dehydrogenase.

Table 3 Key laboratory investigations at admission

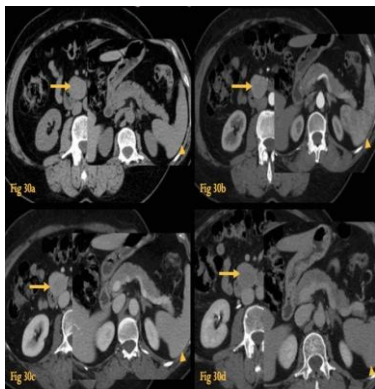
<u>Parameter</u>	<u>Value</u>	<u>Reference range</u>	<u>Significance</u>
HAEMATOLOGY			
Haemoglobin	11.6 g/dL	11.5–15.5 g/dL	Low-normal
MCV	76.8 fL	77–95 fL	Microcytic
MCH	24.1 pg	25.0–33.0 pg	Hypochromic
RDW	15.0%	11.6–14.0%	Elevated — iron-deficiency pattern
Eosinophils	7.8% (691/cumm)	0–3%	ELEVATED — diagnostically decisive for helminthic infection
BIOCHEMISTRY / IMMUNOLOGY			
Serum IgE	1055 IU/mL	<100 IU/mL	MARKEDLY ELEVATED >10× ULN — near-confirmatory for systemic helminthiasis
CRP	<6 mg/L (normal)	<10 mg/L	Normal — Th2 polarisation; excludes granulomatous/bacterial aetiology
ESR	8 mm/hr (normal)	<20 mm/hr	Normal — Th2 polarisation; excludes tubercular aetiology
Albumin	4.7 g/dL	3.5–5.0 g/dL	Normal

All investigations performed at Yatharth Super Speciality Hospital, Faridabad, 24–28 February 2026. AG, anion gap; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IgE, immunoglobulin E; IV, intravenous; MCH, mean corpuscular haemoglobin; MCV, mean corpuscular volume; RDW, red cell distribution width; ULN, upper limit of normal.

Table 4 Antiparasitic and supportive treatment protocol

<u>Agent</u>	<u>Dose</u>	<u>Route/timing</u>	<u>Duration</u>	<u>Indication</u>
Ivermectin	200 mcg/kg/day (weight confirmed 35 kg prior to dosing)	Oral; single daily dose; empty stomach	7 days [8]	Strongyloides stercoralis hyperinfection — extended protocol
Albendazole	400 mg once daily	Oral; with fatty meal	3 days	Ancylostoma duodenale co-infection coverage
WITHHELD — Corticosteroids	—	—	—	Absolute contraindication — risk of fatal hyperinfection amplification via ecdysone-like larval moulting stimulation [9]
WITHHELD — Stimulant laxatives	—	—	—	Contraindicated in transmural enteritis (perforation risk); prolongs colonic transit thereby worsening autoinfection cycle

Figure Legends

**Figure – 1****Figure – 2****Figure – 3**

Contrast-enhanced CT abdomen.

Axial and coronal reconstructions demonstrating: **(Figure 1)** circumferential mural thickening of D2–D3 duodenum (4–5 mm); **(Figure 2)** terminal ileal and ileocaecal junction mural thickening (4–4.5 mm) with proximal jejunal mucosal fold thickening; **“(Figure 3)** demonstrating multiple enlarged mesenteric lymph nodes at the root of mesentery and right iliac fossa. No free intraperitoneal air identified.