

Pseudotumoral Schistosomiasis Mimicking Locally Advanced Rectal Carcinoma: A Case Report

Yoseph Mulatu Habte

yosephmhabe1@gmail.com

Ethio Tebib Hospital

Binyam Mulatu Habte

Debre Berhan University

Philipos Shineda Areda

Ethio Tebib Hospital

Hanan Abdulkerim Fethudin

Ethio Tebib Hospital

Khalid Arebo Hassen

Ethio Tebib Hospital

Abubeker Fedlu Abdela

Ethio Tebib Hospital

Shimelis Ayalew Yimer

Ethio Tebib Hospital

Case Report

Keywords: Pseudotumoral schistosomiasis, Schistosomiasis, Schistosoma mansoni, Rectal carcinoma mimic, Case report, Ethiopia

Posted Date: July 7th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10180194/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at BMC Infectious Diseases on August 31st, 2026. See the published version at <https://doi.org/10.1186/s12879-026-14338-8>.

Abstract

Introduction and Importance:

Pseudotumoral rectal schistosomiasis is a rare complication of chronic *Schistosoma mansoni* infection characterized by mass-forming granulomatous inflammation and fibrosis. It may closely mimic locally advanced rectal carcinoma clinically and radiologically, creating significant diagnostic difficulty in endemic regions.

Case Presentation:

A 50-year-old Ethiopian man presented with rectal bleeding, lower abdominal pain, urinary intermittency, and weight loss. Laboratory investigations revealed leukocytosis with marked eosinophilia, microcytic anemia, renal impairment, and normal carcinoembryonic antigen levels. Contrast-enhanced CT and MRI demonstrated long-segment rectal wall thickening with mesorectal invasion, lymphadenopathy, and bilateral distal ureteric encasement, suggestive of cT4b rectal carcinoma. Colonoscopy showed a circumferential ulcerated rectal mass with luminal narrowing. Initial biopsies were non-diagnostic, revealing necrotic and inflammatory tissue only. Repeat deep biopsies demonstrated eosinophil-rich granulomatous inflammation with calcified *Schistosoma* ova and no malignancy, confirming pseudotumoral schistosomiasis. The patient developed obstructive uropathy with acute kidney injury requiring percutaneous nephrostomy and ureteric stenting and was treated with praziquantel with partial recovery.

Clinical Discussion:

This case highlights the diagnostic difficulty of pseudotumoral schistosomiasis due to its close resemblance to colorectal malignancy. Peripheral eosinophilia and discordant radiological–histopathological findings are key diagnostic clues. The condition may produce extensive fibrotic inflammation causing mass-forming lesions and adjacent organ compression. Deep biopsy is essential when superficial samples are non-diagnostic.

Conclusion

Pseudotumoral rectal schistosomiasis should be considered in the differential diagnosis of colorectal masses in endemic regions. Awareness of this entity and confirmation through adequate histopathology are essential to prevent misdiagnosis and unnecessary oncologic intervention.

Introduction

Schistosomiasis is a neglected tropical disease caused by blood flukes of the genus *Schistosoma*, with the highest burden in Sub-Saharan Africa [1, 2]. Intestinal schistosomiasis, most commonly caused by *Schistosoma mansoni*, results from deposition of eggs in the intestinal wall, leading to chronic granulomatous inflammation, fibrosis, and mucosal injury [3–5]. Clinical manifestations are often non-specific and may include abdominal pain and rectal bleeding, while chronic disease may occasionally progress to more advanced structural complications [1, 2, 5].

Pseudotumoral schistosomiasis is a rare manifestation of chronic intestinal schistosomiasis characterized by mass-forming granulomatous inflammation and fibrosis due to persistent egg deposition [6]. It may closely mimic colorectal malignancy clinically and radiologically, presenting with rectal bleeding, weight loss, bowel obstruction, and imaging findings such as bowel wall thickening, mesorectal involvement, and lymphadenopathy. Pseudotumoral colorectal schistosomiasis closely mimics colorectal malignancy and other inflammatory conditions, posing a diagnostic challenge in endemic regions and requiring histopathological identification of *Schistosoma* ova for definitive diagnosis [7, 8].

Here, we report a case of pseudotumoral rectal schistosomiasis in a 50-year-old man presenting with rectal bleeding and radiological findings suggestive of locally advanced rectal carcinoma, which was ultimately confirmed on repeat biopsy demonstrating *Schistosoma* ova with granulomatous inflammation. This case report has been written in compliance with CARE guidelines [9].

Case Presentation

A 50-year-old HIV-negative man presented with a one-month history of painless bright red bleeding per rectum during defecation. The bleeding was associated with intermittent crampy lower abdominal pain, urinary intermittency, decreased appetite, and significant but unquantified weight loss. He had previously been informed at another institution that he had rectal cancer and was advised to undergo surgical intervention; however, he declined treatment and subsequently sought reevaluation at our center because of progressive symptoms, particularly worsening urinary intermittency. He denied fever, chills, or changes in bowel habits. His past medical and family histories were unremarkable.

On physical examination, he appeared pale but was otherwise in no acute distress. His pulse rate was 110 beats/min, respiratory rate was 20 breaths/min, and oxygen saturation was 90% on room air. Pale conjunctivae and right costovertebral angle tenderness were noted. No palpable lymphadenopathy, abdominal mass, hepatosplenomegaly, or peripheral edema was identified. Digital rectal examination revealed irregular distal rectal mass. The remainder of the examination was unremarkable.

Initial laboratory investigations demonstrated leukocytosis ($22.74 \times 10^9/\text{L}$) with marked eosinophilia ($2.14 \times 10^9/\text{L}$; 9.4%), microcytic anemia (hemoglobin 9.8 g/dL, mean corpuscular volume 77.3 fL), thrombocytosis ($430 \times 10^9/\text{L}$), elevated serum creatinine (2.37 mg/dL), and elevated blood urea nitrogen (72.4 mg/dL). Carcinoembryonic antigen was within the normal range (1.27 ng/mL)(Table 1).

Table 1
Initial laboratory investigations and normal reference ranges

Laboratory parameter	Results	Normal Value
White Blood Cells	22.74 ×10 ³ /μL	4.0–10.0 ×10 ³ /μL
Hemoglobin	9.8 g/dL	12.5–15.5 g/dL
Platelet	430 ×10 ³ /μL	100–300 ×10 ³ /μL
Eosinophil Percentage	9.4%	0.5–5%
Lymphocyte Percentage	9.3%	15–50%
Neutrophil Percentage	76.8%	45–70%
Creatinine	2.37 mg/dL	0.67–1.17 mg/dL
Urea	72.4 mg/dL	17–43 mg/dL
Na ⁺	134.7 mmol/L	136–145 mmol/L
K ⁺	3.57 mmol/L	3.5–5.1 mmol/L
Cl ⁻	98.4 mmol/L	98–111 mmol/L
Aspartate Transaminase	41.6 U/L	2–50 U/L
Alanine Transaminase	49.3 U/L	1–50 U/L
C-reactive Protein	199 mg/L	0–5 mg/L
Erythrocyte Sedimentation Rate	140 mm/hr	0–20 mm/hr
Procalcitonin	1.21 ng/mL	0–0.05 ng/mL
Carcinoembryonic Antigen	1.27ng/mL	0–5.09 ng/mL
Hepatitis B Surface Antigen	Negative	Negative
Hepatitis C Virus Antibody	Negative	Negative
Rapid HIV Test	Negative	Negative
Venereal Disease Research Laboratory Test	Negative	Negative
Blood Film for Hemo parasites	Negative	Negative

Contrast-enhanced computed tomography (CT) of the abdomen and pelvis revealed long-segment asymmetric recto-anal wall thickening involving nearly the entire rectum with a maximal mural thickness of 2.3 cm. The lesion demonstrated extension into the mesorectum with marked mesorectal fat stranding and mesorectal fascia involvement. Multiple mesorectal, iliac, para-aortic, and aortocaval lymph nodes were identified, the largest measuring 1.26 cm in the right internal iliac region. Bilateral distal ureteric encasement resulted in mild-to-moderate hydroureteronephrosis with delayed right renal

excretion and absent left renal excretion. These findings were considered highly suggestive of locally advanced recto-anal malignancy (Fig. 1).

(A) Axial section showing asymmetric thickening of the anal canal (yellow arrow). (B) Sagittal section showing long-segment rectal wall thickening with mesorectal fat stranding (red arrow). (C) Axial section showing mesorectal involvement with associated mesorectal fat stranding (white arrows), suggestive of locally advanced rectal carcinoma.

Colonoscopy demonstrated a nearly circumferential ulcerated and necrotic distal rectal mass approximately 5 cm from the anal verge with marked luminal narrowing, precluding advancement of the colonoscope beyond the lesion. Multiple biopsies were obtained. Histopathological examination of the initial biopsy showed necrosis, granulation tissue formation, and mixed inflammatory infiltrates without evidence of malignancy. Because the specimen was considered inadequate for definitive diagnosis, repeat biopsy was recommended.

Pelvic magnetic resonance imaging (MRI) performed for local staging demonstrated an approximately 12-cm low rectal wall thickening with a distal margin located 3.5 cm from the anal verge. The lesion extended into the proximal anal canal and internal anal sphincter, with extension beyond the muscularis propria into the mesorectal fat and definite involvement of the mesorectal fascia. Apparent invasion of the right periprostatic fat and right prostatic apex was also identified. Extensive surrounding infiltrative fibrotic tissue resulted in bilateral distal ureteric narrowing with upstream hydroureter. The lesion was radiologically staged as cT4bN1 disease with circumferential resection margin involvement (Fig. 2).

(A) Axial image showing rectal wall thickening extending beyond the muscularis propria into the mesorectal fascia, with mesorectal fat stranding (red asterisks) and right periprostatic fat invasion (white arrow). **(B)** Sagittal image showing diffuse ~ 12-cm low rectal wall thickening (white arrow) extending into the proximal anal canal, with the distal margin 3.5 cm from the anal verge.

Given the discordance between the highly suspicious radiologic findings and the nondiagnostic initial pathology, repeat endoscopic biopsies were performed. Histopathological examination demonstrated extensive necrosis, eosinophil-rich inflammatory infiltrates, numerous epithelioid granulomas, and scattered calcified *Schistosoma* ova (Fig. 3). No dysplasia or malignancy was identified in multiple examined sections. The overall findings were diagnostic of schistosomiasis with granulomatous inflammation (pseudotumoral schistosomiasis). Subsequent stool examination for ova and parasites was negative.

(A) Epithelioid granuloma with Langhans giant cells and eosinophilic infiltrates (white arrow). (B) Dense eosinophilic inflammatory infiltrates involving the rectal mucosa and submucosa. (C) Multiple calcified *Schistosoma* ova within the rectal wall (white arrows). Findings are consistent with pseudotumoral schistosomiasis

During hospitalization, the patient developed worsening renal dysfunction secondary to bilateral distal ureteric obstruction, with serum creatinine rising to 7.27 mg/dL. Under ultrasonographic and fluoroscopic guidance, left percutaneous nephrostomy and antegrade double-J ureteric stent placement were performed. Antegrade urography demonstrated complete distal ureteric obstruction.

The patient was treated with praziquantel (1,400 mg orally twice daily for one day) in addition to supportive management. At follow-up, he reported improvement in flank pain and continued passage of stool without obstructive symptoms. Follow-up ultrasonography demonstrated persistent rectal wall thickening with mesorectal fat stranding, mild right-sided hydroureteronephrosis, and a left nephrostomy tube and double-J stent in situ. Renal function showed partial recovery, with serum creatinine decreasing from 7.27 mg/dL to 3.61 mg/dL. The patient was discharged in stable condition and scheduled for surgical and urological follow-up.

Discussion

Schistosomiasis is transmitted through exposure to contaminated freshwater and results from infestation by blood flukes belonging to the genus *Schistosoma*, affecting over 250 million individuals each year [1, 2, 10]. Approximately 90% of the global disease burden is concentrated in Sub-Saharan Africa, where the vast majority of annual cases are reported [2, 11]. Following Malaria, schistosomiasis is considered the second most significant parasitic disease worldwide in terms of overall public health burden [5, 11]. In this region, *S. haematobium* and *S. mansoni* are the principal human-infecting species [2], with the latter being the predominant cause of intestinal disease. Transmission is maintained through a complex life cycle involving freshwater snails as intermediate hosts and humans as definitive hosts [2, 10].

Schistosomiasis progresses through three clinical stages: an initial cercarial dermatitis occurring days after exposure, an acute Katayama syndrome characterized by systemic hypersensitivity manifestations including fever, gastrointestinal symptoms, and weight loss, and a chronic phase resulting from tissue deposition of eggs with subsequent long-term organ involvement [1–3]. Intestinal schistosomiasis, primarily due to *S. mansoni*, results from adult worms residing in the inferior mesenteric venous plexus where eggs are deposited and become trapped in the intestinal wall [4, 5, 12]. These eggs trigger granulomatous inflammation beginning in the submucosa, with progressive extension into the muscularis mucosa and eventual erosion of the overlying mucosa, resulting in mucosal ulceration, strictures, or hyperplastic mucosal changes [5]. Clinical manifestations arise from ongoing egg deposition and immune-mediated tissue injury driven by antigen release and inflammatory cell infiltration [3–5].

Schistosome eggs are highly immunogenic, and the release of soluble egg antigens drives a predominantly T-helper 2 (Th2)–mediated immune response characterized by eosinophilic infiltration and granuloma formation. With chronic antigen exposure, regulatory T-cell (Treg) responses contribute to immune modulation and persistence of inflammation over time [11]. The sustained host response to

trapped eggs leads to repeated cycles of tissue injury and repair, resulting in progressive fibrotic remodeling, architectural distortion, and transmural fibrosis. Over time, these changes may culminate in mass-forming, tumefactive lesions, while chronic inflammatory damage can also result in mucosal ulceration, necrosis, and hemorrhage [1, 2, 13]. In advanced disease, as seen in this case, progressive fibrosis and inflammatory extension may produce mass-forming lesions with apparent involvement or compression of adjacent structures, accounting for radiological findings such as mesorectal infiltration and ureteric obstruction.

Presentation of schistosomiasis in a pseudotumoral form is rare and has been previously documented in isolated case reports [6]. This manifestation may closely resemble a range of other pathological conditions, and similar diagnostic confusion has been reported in the literature. In particular, colonic schistosomiasis has been described as mimicking colorectal malignancy, polyps, and inflammatory bowel disease [7]. Furthermore, in a retrospective analysis of 46 patients with colonic schistosomiasis, 12 cases were initially misinterpreted as ulcerative colitis, Crohn's disease, and ischemic colitis, while 8 patients were subsequently diagnosed with colonic carcinoma [8]. In the present case, this process produced a long-segment rectal mass with mesorectal and pelvic extension, bilateral ureteric encasement, and radiological staging consistent with cT4b rectal carcinoma, illustrating the extreme end of the disease spectrum in which benign inflammatory pathology becomes radiologically indistinguishable from invasive malignancy.

Colonoscopy may demonstrate a spectrum of findings depending on disease stage. In early infection, features can include mucosal edema, superficial erosions, ulcerations, and areas of hemorrhage. In more advanced disease, structural changes such as intestinal strictures, polypoid lesions, and bowel wall thickening may be observed [7, 8].

Histopathological confirmation via endoscopic biopsy is considered the gold standard for diagnosing colonic schistosomiasis, with identification of schistosome ova within the lamina propria or submucosa being diagnostic. In chronic disease, these eggs may appear calcified or disrupted, and are typically associated with a chronic inflammatory infiltrate composed predominantly of lymphocytes and plasma cells within the lamina propria and submucosa [8]. In this case, initial superficial biopsies were non-diagnostic, showing only necrotic and inflammatory tissue, a known limitation in schistosomal lesions due to patchy egg distribution and deep submucosal involvement. Only repeat deep biopsies demonstrated the diagnostic triad of eosinophil-rich granulomatous inflammation, calcified *Schistosoma* ova, and absence of dysplasia. This underscores the critical importance of repeat tissue sampling when clinical and radiological findings are discordant, particularly in endemic regions.

Management is centered on antiparasitic therapy with a single dose of praziquantel, which remains highly effective and contributes to reducing the risk of long-term complications [3, 5, 10, 11, 14]. In addition, emerging evidence suggests that probiotics may have a beneficial role in the prevention, attenuation, and adjunctive treatment of schistosomiasis [4]. In this patient, treatment with praziquantel was combined with decompressive urological intervention, resulting in partial renal recovery and

symptomatic improvement. While praziquantel eliminates active infection, established fibrosis may persist, and long-term outcomes depend on the degree of irreversible structural damage at presentation.

In conclusion, pseudotumoral rectal schistosomiasis represents a rare but important diagnostic pitfall that can closely simulate colorectal carcinoma. Awareness of this entity, careful integration of clinical and laboratory clues, and insistence on definitive histological confirmation are essential to avoid misdiagnosis and ensure appropriate management.

Conclusion

This case highlights the diagnostic challenge posed by pseudotumoral schistosomiasis, a rare manifestation of chronic schistosomal infection that may closely mimic locally advanced colorectal carcinoma both clinically and radiologically, leading to potential misdiagnosis and inappropriate oncologic management. It also underscores that advanced pseudotumoral disease, despite its benign infectious etiology, may produce severe local complications including obstructive uropathy and acute kidney injury as a consequence of extensive inflammatory fibrosis. Careful integration of clinical clues, awareness of clinicoradiological–pathological discordance, and definitive histopathological confirmation through adequate deep biopsy remain essential for timely diagnosis and appropriate treatment. Recognition of this uncommon presentation is particularly important in endemic regions to prevent unnecessary radical intervention and avoid irreversible organ damage.

Abbreviations

CT

Computed tomography

MRI

Magnetic resonance imaging

Declarations

Ethics approval: Ethical approval by the ethics committee is not required for case report publication deemed not to constitute research as per the policies of the institution. This case report was prepared in accordance with the ethical principles outlined in the Declaration of Helsinki.

Consent to participate declaration: Not applicable

Consent for publication: Written informed consent has been obtained from the patient for publication of the case report and any accompanying images. A copy of the written consent is available for review upon request by the editor in chief of this journal.

Availability of data and materials: The data on this case report is available and will be provided to the editor in chief of the journal upon request.

Competing interests: None

Funding: The authors received no funding.

Authors' contributions:

Y.M.H.: Writing – original draft, writing – review and editing, conceptualization, data curation, and supervision. B.M.H.: Writing – original draft, writing – review and editing, conceptualization, data curation, and supervision. P.S.A.: Data curation and writing – review and editing. H.A.F.: Resources and writing – review and editing. K.A.H.: Data curation and writing – review and editing. A.F.A.: Resources and writing – review and editing. S.A.Y.: Writing – review and editing and supervision.

Acknowledgements: Not applicable

References

1. Ponzo E, Midiri A, Manno A, Pastorello M, Biondo C, Mancuso G. Insights into the epidemiology, pathogenesis, and differential diagnosis of schistosomiasis. *EuJMI*. 2024;14:86–96. 10.1556/1886.2024.00013.
2. Aula OP, McManus DP, Jones MK, Gordon CA. Schistosomiasis with a Focus on Africa. *TropicalMed*. 2021;6:109. 10.3390/tropicalmed6030109.
3. McManus DP, Bergquist R, Cai P, Ranasinghe S, Tebeje BM, You H. Schistosomiasis—from immunopathology to vaccines. *Semin Immunopathol*. 2020;42:355–71. 10.1007/s00281-020-00789-x.
4. Hong A, Umar A, Chen H, Yu Z, Huang J. Advances in the study of the interaction between schistosome infections and the host's intestinal microorganisms. *Parasites Vectors*. 2024;17:185. 10.1186/s13071-024-06245-1.
5. Elbaz T, Esmat G, Hepatic, Schistosomiasis I. *Rev J Adv Res*. 2013;4:445–52. 10.1016/j.jare.2012.12.001.
6. Segun AO, Alebiosu CO, Agboola AOJ, Banjo A. a. F. Schistosomiasis—An unusual cause of abdominal pseudotumor. *J Natl Med Assoc*. 2006;98:1365–8. PubMed PMID: 16916140; PubMed Central PMCID: PMC2569563.
7. Mulate ST, Nur AM, Tasamma AT, Annose RT, Dawud EM, Ekubazgi KW, et al. Colonic schistosomiasis mimicking cancer, polyp, and inflammatory bowel disease: Five case reports and review of literature. *World J Gastrointest Endosc*. 2024;16:472–82. 10.4253/wjge.v16.i8.472.
8. Cao J. Endoscopic findings and clinicopathologic characteristics of colonic schistosomiasis: A report of 46 cases. *WJG*. 2010;16:723. 10.3748/wjg.v16.i6.723.
9. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D, et al. The CARE guidelines: consensus-based clinical case reporting guideline development. *BMJ Case Rep*. 2013;2013:bcr2013201554. 10.1136/bcr-2013-201554.

10. Nelwan ML, Schistosomiasis. Life Cycle, Diagnosis, and Control. *Curr Therapeutic Res.* 2019;91:5–9. 10.1016/j.curtheres.2019.06.001.
11. Perera DJ, Koger-Pease C, Paulini K, Daoudi M, Ndao M. Beyond schistosomiasis: unraveling co-infections and altered immunity. *Messenger LA, editor. Clin Microbiol Rev.* 2024;37:e00098-23. 10.1128/cmr.00098-23
12. Alshoabi SA, Magram AO, Alkalady AH, Al-Maqtari RR, Almas KM, Al-Sayaghi KM, et al. *Schistosoma Mansoni* and *Haematobium*: Radiological Diagnostic Clues and Pathophysiology. *Pathogens.* 2026;15:536. 10.3390/pathogens15050536.
13. Wilson MS, Mentink-Kane MM, Pesce JT, Ramalingam TR, Thompson R, Wynn TA. Immunopathology of schistosomiasis. *Immunol Cell Biol.* 2007;85:148–54. 10.1038/sj.icb.7100014.
14. Aboagye IF, Addison YAA. Praziquantel efficacy, urinary and intestinal schistosomiasis reinfection – a systematic review. *Pathogens Global Health.* 2023;117:623–30. 10.1080/20477724.2022.2145070.

Figures

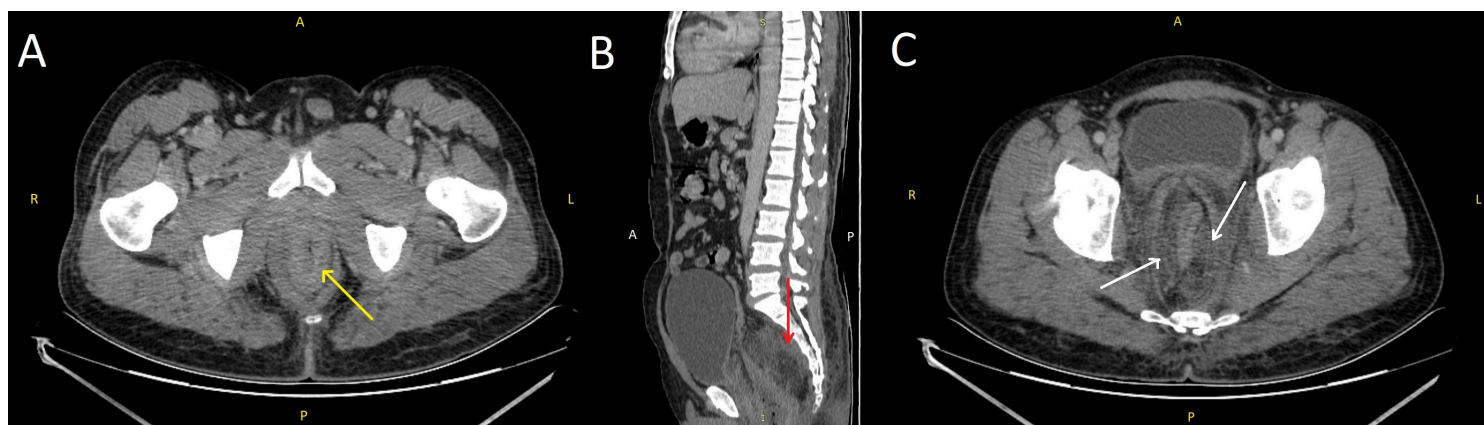


Figure 1

Portal venous phase contrast-enhanced abdominopelvic CT.

(A) Axial section showing asymmetric thickening of the anal canal (yellow arrow). (B) Sagittal section showing long-segment rectal wall thickening with mesorectal fat stranding (red arrow). (C) Axial section showing mesorectal involvement with associated mesorectal fat stranding (white arrows), suggestive of locally advanced rectal carcinoma.

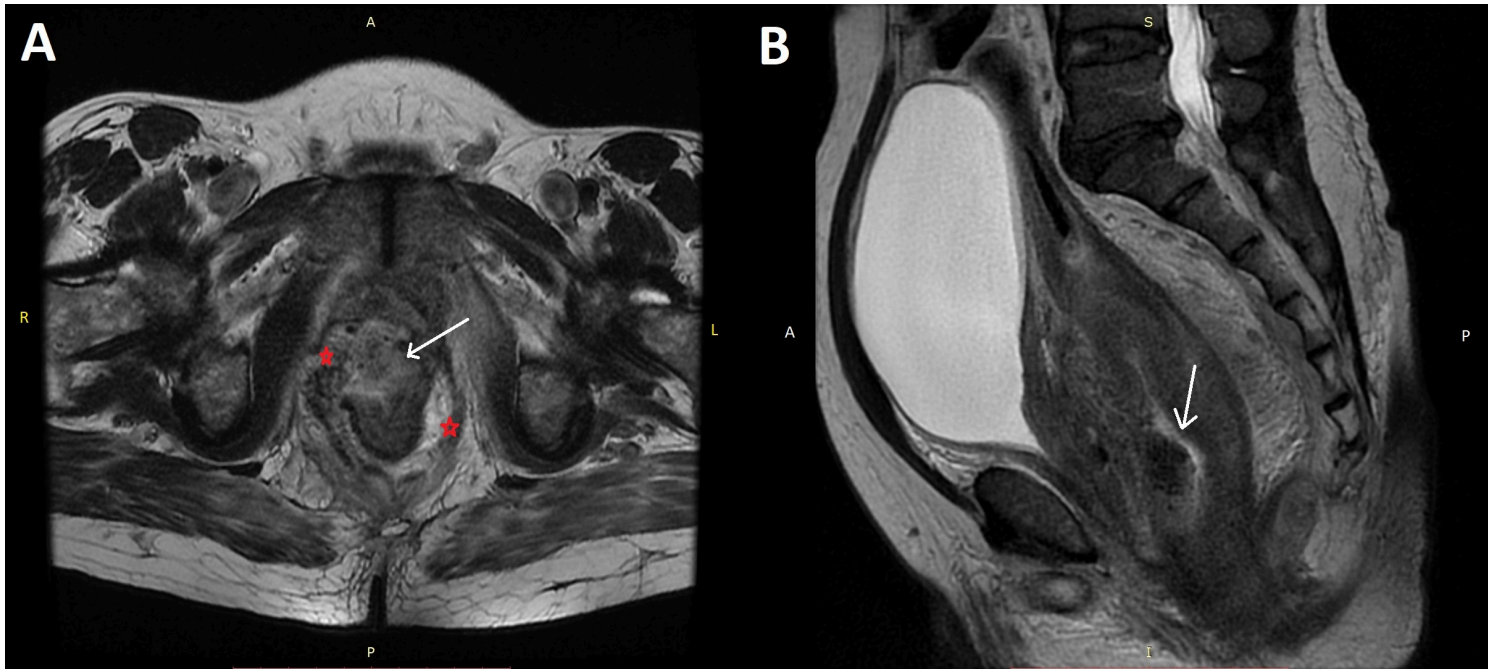


Figure 2

Pelvic MRI for local staging.

(A) Axial image showing rectal wall thickening extending beyond the muscularis propria into the mesorectal fascia, with mesorectal fat stranding (red asterisks) and right periprostatic fat invasion (white arrow). (B) Sagittal image showing diffuse ~12-cm low rectal wall thickening (white arrow) extending into the proximal anal canal, with the distal margin 3.5 cm from the anal verge.

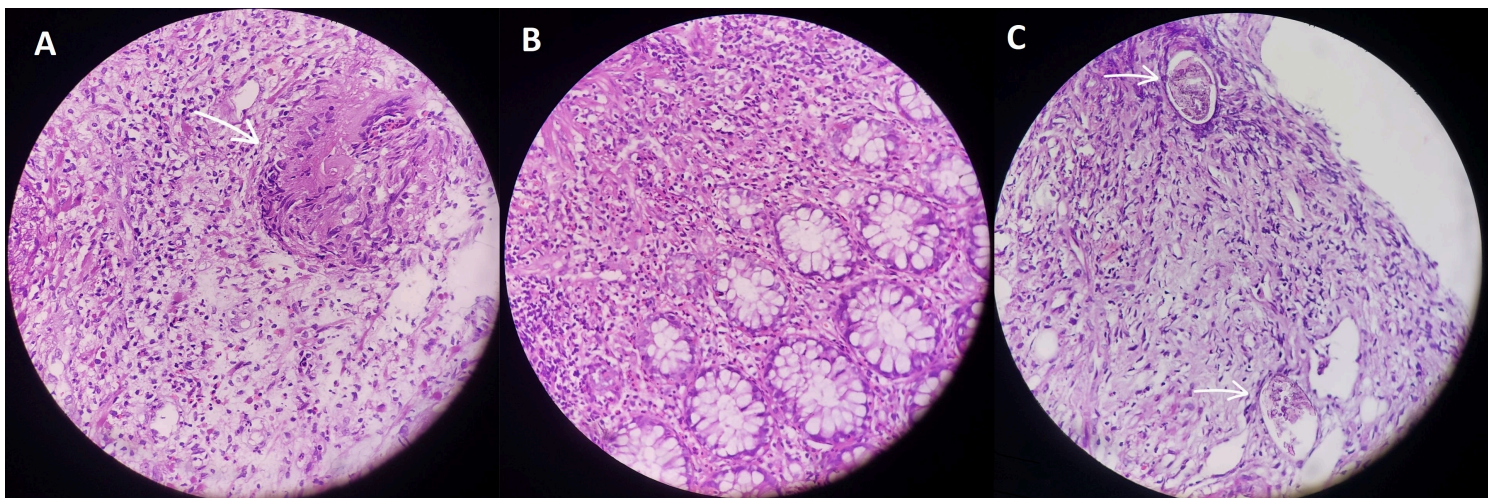


Figure 3

Histopathologic images of the rectal endoscopic biopsy.

(A) Epithelioid granuloma with Langhans giant cells and eosinophilic infiltrates (white arrow). (B) Dense eosinophilic inflammatory infiltrates involving the rectal mucosa and submucosa. (C) Multiple calcified granulomas (white arrows).

Schistosoma ova within the rectal wall (white arrows). Findings are consistent with pseudotumoral schistosomiasis