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CASE REPORT

Case Report on Gastrointestinal Basidiobolomycosis Mimicking Inflammatory Bowel Disease: Insights and Review of Saudi Literature

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Abstract: Gastrointestinal basidiobolomycosis (GIB) is a rare invasive fungal infection of immunocompetent hosts that is endemic to Saudi Arabia and the wider Gulf region. Its presentation overlaps with inflammatory bowel disease (IBD), intestinal tuberculosis and malignancy, and many patients reach a definitive diagnosis only after surgery. We report a 39-year-old Saudi woman who presented with a 7-day history of right lower quadrant pain and non-bloody watery diarrhea. Initial bloods showed an elevated C-reactive protein of 21.5 mg/L, an erythrocyte sedimentation rate of 44 mm/hour and an absolute eosinophil count of $0.54 \times 10^3/\mu L$. Contrast-enhanced computed tomography (CT) demonstrated segmental wall thickening of the terminal ileum, cecum and proximal transverse colon, with a 3×5 cm intramural cecal collection. Colonoscopy revealed ulcerated congested mucosa and an ileocecal stricture; targeted biopsies showed an eosinophil-rich granulomatous inflammation with broad, sparsely septate fungal hyphae demonstrating the Splendore–Hoeppli phenomenon, positive on Grocott methenamine silver and periodic acid–Schiff stains; and tissue culture grew *Basidiobolus* spp. Interventional radiology and surgical drainage were considered but were technically not feasible because of the intramural location, so the patient was managed medically with oral itraconazole at 200 mg every 8 hours for 3 days followed by 200 mg once daily, with monthly liver enzyme monitoring. Surgery was avoided. A repeat CT scan at 2 months showed near complete resolution of wall thickening, and follow-up colonoscopy at 5 months was normal. The patient self-discontinued itraconazole at 9 months because of symptom resolution and remained asymptomatic at follow-up 2 months later. Clinicians working in endemic regions should consider GIB in any patient with an ileocecal mass or abscess, eosinophilia and an IBD-like presentation, because early biopsy with fungal stains and prompt azole therapy can avert surgery.

Keywords: gastrointestinal basidiobolomycosis, fungal infection, immunocompetent, itraconazole, case report, Saudi Arabia, inflammatory bowel disease mimicker, *Basidiobolus*

Introduction

Basidiobolus ranarum is a saprophytic fungus of the order Entomophthorales that lives in soil, decaying plant matter, and the gastrointestinal contents of amphibians, reptiles and insectivorous bats.[1] Human infection most commonly takes the form of a chronic subcutaneous mycosis of the limbs or trunk in healthy hosts in tropical and subtropical regions. Gastrointestinal involvement is rare but increasingly recognized, with Saudi Arabia and the southwestern USA accounting for most reported cases worldwide.[2,3] Within Saudi Arabia, the southern Jazan and Asir regions remain the principal endemic foci, although adult cases from the central region around Riyadh are now well documented.[2,4,5]

The clinical phenotype of gastrointestinal basidiobolomycosis (GIB) is what makes it so often misread on first encounter. Patients typically present with right lower quadrant abdominal pain, a palpable or radiographically defined ileocecal mass, peripheral eosinophilia and elevated inflammatory markers. The radiological appearance of segmental bowel wall thickening

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with a localized mural collection further overlaps with Crohn's disease, intestinal tuberculosis and ileocecal malignancy. In the 2001 Arizona case–control study by Lyon et al,[6] longer Arizona residence and ranitidine use were the two exposures most strongly associated with GIB, with reported odds ratios of approximately 6.0 for ranitidine and 2.1 for each additional 20 years of smoking, although the small sample limits precision.[6] The 2012 Mayo Clinic series by Vikram et al confirmed the surgical and antifungal management pattern and documented mortality of around 36% when diagnosis was delayed.[7]

These aspects matter for everyday gastroenterology practice because GIB sits inside a wider differential of inflammatory bowel disease (IBD) mimickers that clinicians in endemic regions must actively rule in or out before starting immunosuppression. Other infectious mimics include intestinal and hepatobiliary tuberculosis, which can produce identical ileocecal masses, biliary strictures and weight-loss patterns;[8] endemic mycoses such as paracoccidioidomycosis, which has been reported to mimic IBD with right lower quadrant ileitis and granulomatous histopathology;[9] and gastrointestinal histoplasmosis. Non-infectious mimics include drug-induced colitis, exemplified by sodium polystyrene sulfonate, mycophenolate mofetil and immune checkpoint inhibitor colitis, which can reproduce both the endoscopic and histological appearance of IBD;[10] vasculitides such as eosinophilic granulomatosis with polyangiitis; and rare neoplasms such as indolent T-cell lymphoproliferative disorder of the gut.[11] Misdiagnosis carries real cost: empirical corticosteroids or biologics for presumed Crohn's disease can accelerate untreated fungal or mycobacterial disease and have been associated with disseminated infection and death.[11]

We describe a Saudi woman with cecal GIB whose presentation mimicked Crohn's disease and intestinal tuberculosis. Source control by interventional radiology and surgery was attempted but not feasible because of the intramural anatomy of the collection. The patient was treated medically with itraconazole; she achieved radiological and endoscopic remission, and surgery was avoided. We also summarize the published Saudi adult and pediatric GIB literature and discuss how this case fits the IBD mimicker framework.

Case Report

A 39-year-old Saudi woman, known to have polyarteritis nodosa (not on treatment) and a history of smoking, as well as a history of liposuction, laparoscopic cholecystectomy and uncomplicated open ventral hernia repair, was referred from a private community hospital with a 7-day history of cramping right lower quadrant abdominal pain and watery non-bloody diarrhea. She denied fever, weight loss, night sweats, vomiting, cough or known tuberculosis contact, and reported no recent antibiotic or proton pump inhibitor use.

On arrival at our tertiary center she was alert, afebrile (37.0°C), normotensive (blood pressure 122/74 mmHg) and tachycardic, at 96 beats per minute. The abdomen was soft, with a tender, ill-defined mass in the right iliac fossa and no peritoneal signs. A faint reticulated rash on the lower limbs was noted, consistent with her known background of polyarteritis nodosa. Initial laboratory results from the referring hospital showed an ESR of 37 mm/hour and a CRP of 38.1 mg/L. The results of repeat laboratory testing at our center are shown in Table 1; the absolute eosinophil count was

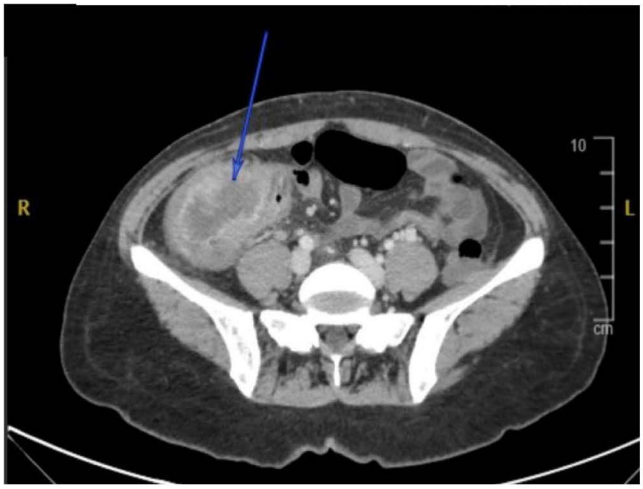
Table 1 Initial Laboratory Investigations at Presentation to the Tertiary Center

Laboratory Test	Patient's Value	Reference Range
White blood cell count	7.2×10^3/μL	4.0 to 10.0×10^3/μL
Hemoglobin	11.0 g/dL	11.6 to 15.0 g/dL
Platelets	227×10^3/μL	150 to 400×10^3/μL
Absolute neutrophil count	4.74×10^3/μL	2.0 to 7.0×10^3/μL
Absolute lymphocyte count	1.40×10^3/μL	1.5 to 4.3×10^3/μL
Absolute eosinophil count	0.54×10^3/μL	0.05 to 0.50×10^3/μL
Erythrocyte sedimentation rate	44 mm/hour	2 to 28 mm/hour
C-reactive protein	21.5 mg/L	0.0 to 5.0 mg/L

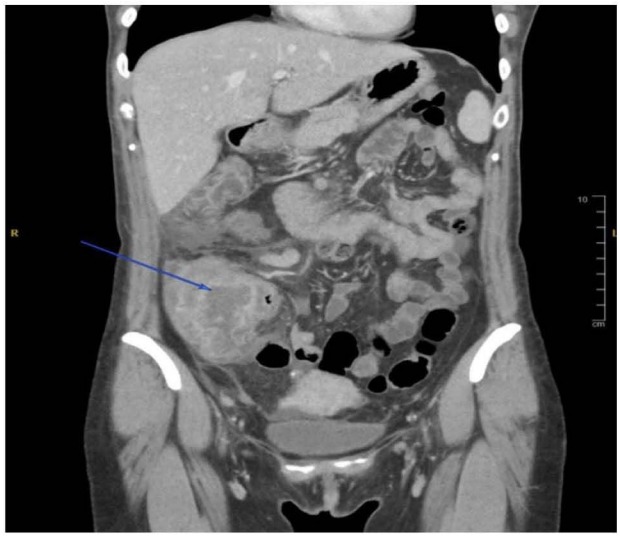
$0.54\times10^3/\mu L$ (above the upper reference limit), with mild lymphopenia, an ESR of 44 mm/hour and a CRP of 21.5 mg/L. Liver and renal panels, serum albumin and glycosylated hemoglobin (HbA_{1c}) were within normal limits. Stool studies for ova, cysts and parasites, *Clostridioides difficile* toxin and routine bacterial pathogens were negative. QuantiFERON TB Gold was negative, and screening for HIV, hepatitis B and hepatitis C was negative.

A contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis (Figure 1A) demonstrated long-segment circumferential wall thickening of the terminal ileum, cecum, and ascending and proximal transverse colon, with surrounding mesenteric fat stranding, free pelvic fluid and mesenteric lymphadenopathy. Within the cecum, there was a 3×5 cm intramural fluid collection consistent with an abscess (Figure 1B), without luminal obstruction or pneumoperitoneum.

She was admitted for inpatient evaluation and continued on intravenous piperacillin tazobactam and metronidazole started at the referring hospital, along with intravenous metoclopramide and oral omeprazole for symptom control. The clinical differential at this point was Crohn's disease with a phlegmon, ileocecal tuberculosis and a perforated cecal neoplasm. Colonoscopy on day 2 of admission revealed a localized area of severely congested, inflamed and ulcerated



(A) Axial view



(B) Coronal view

Figure 1 Contrast-enhanced computed tomography of the abdomen at presentation. (**A**) Axial image showing right body segmental circumferential wall thickening of the terminal ileum (blue arrow indicates the thickened segment). (**B**) Coronal image showing a 3×5 cm right body intramural fluid collection within the cecal wall (blue arrow indicates the collection).

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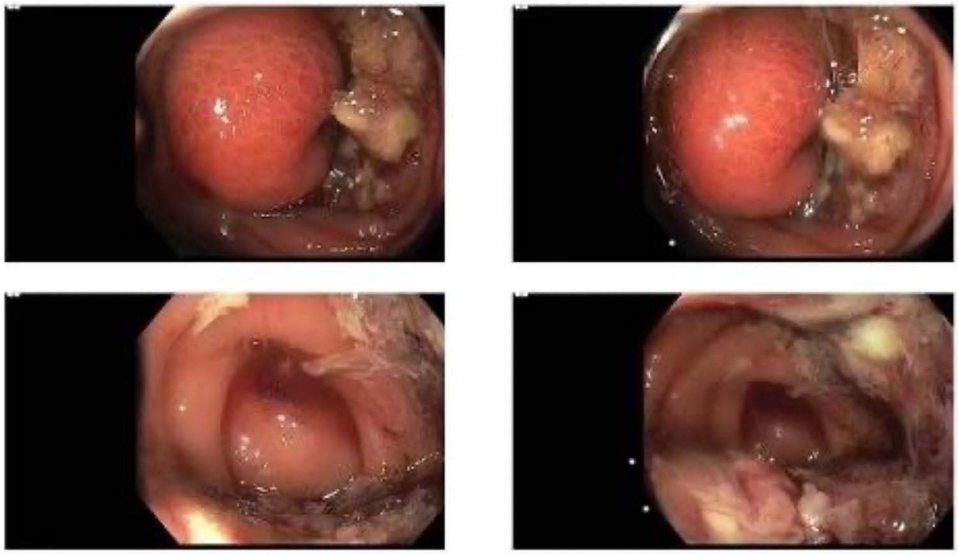

Figure 2 Colonoscopy at admission. Four representative images of the cecum showing a localized area of severely congested, inflamed and ulcerated mucosa with friable exudate, and a tight non-traversable stricture at the ileocecal valve.

mucosa in the cecum, with a tight non-traversable stricture at the ileocecal valve (Figure 2). Multiple biopsies were taken from the cecal ulcers and the structured ileocecal valve.

Histopathology, reviewed by two consultant gastrointestinal pathologists, demonstrated a dense eosinophil-rich inflammatory infiltrate of the colonic crypts and lamina propria, with crypt abscesses (Figure 3A) and broad, irregular, sparsely septate fungal hyphae with right-angled branching surrounded by eosinophilic material consistent with the

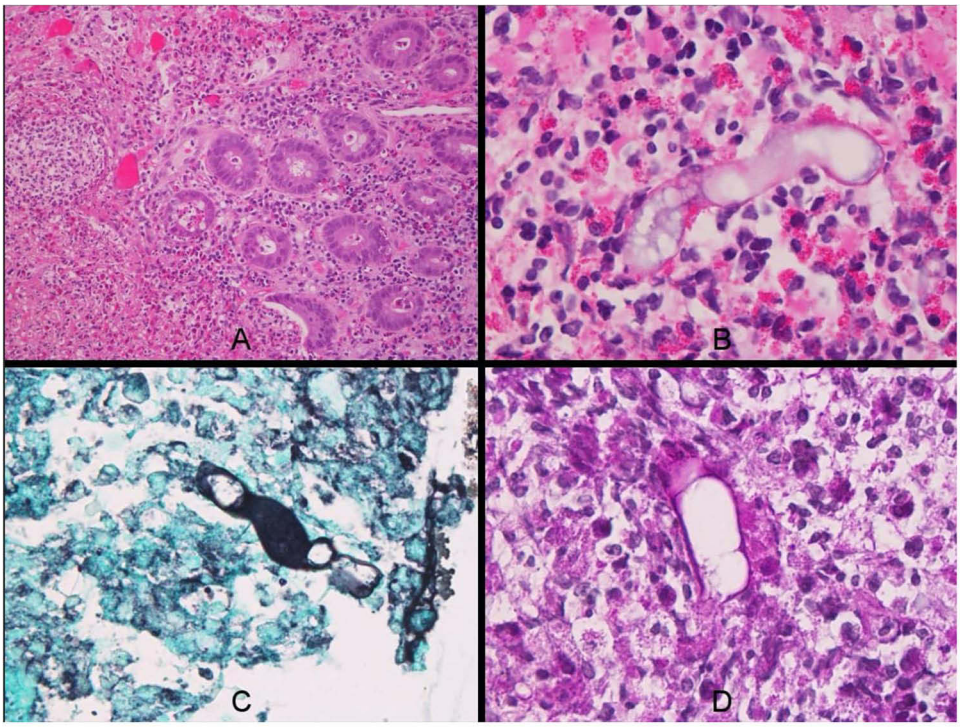

Figure 3 Histopathology of the cecal biopsy. (**A**) Dense eosinophilic infiltration of the colonic crypts and lamina propria (hematoxylin and eosin, original magnification 100×). (**B**) Broad, irregular, sparsely septate fungal hyphae with right-angled branching, surrounded by eosinophilic Splendore–Hoeppli material (hematoxylin and eosin, 400×). (**C**) Positive staining of the fungal hyphae with Grocott methenamine silver (400×). (**D**) Positive staining of the fungal hyphae with periodic acid–Schiff (400×).

Splendore–Hoeppli phenomenon (Figure 3B). The hyphae stained positively with Grocott methenamine silver (Figure 3C) and periodic acid–Schiff stains (Figure 3D). Tissue culture from the cecal biopsy grew *Basidiobolus* spp.; species-level identification was not performed because the genus-level diagnosis is sufficient to guide therapy and the laboratory did not have molecular identification on site. Acid-fast bacillus (AFB) stain and mycobacterial culture were negative on both biopsy and stool. Together with the negative QuantiFERON, this excluded intestinal tuberculosis; and the histopathological appearance, in particular the broad sparsely septate hyphae with right-angled branching and the Splendore–Hoeppli reaction, excluded paracoccidioidomycosis (which typically shows multiple budding ship-wheel yeasts on Grocott) and Crohn's disease (which is characterized by non-caseating granulomas without fungal elements).[9]

Interventional radiology and colorectal surgery were consulted to consider source control. Percutaneous CT-guided drainage was deemed not feasible because the collection was contained within the bowel wall itself rather than in a separate accessible cavity, and any percutaneous tract would have crossed normal bowel, with a high risk of enterocutaneous fistulization or perforation, an established limitation of intramural abscess drainage. Surgical drainage would, in effect, have required right hemicolectomy, which the patient preferred to defer pending a trial of medical therapy.

Oral itraconazole was started at 200 mg every 8 hours for the first 3 days as a loading regimen, followed by 200 mg once daily as maintenance therapy. Itraconazole was selected over voriconazole because of its established Saudi case experience in GIB,[2,4,12] its reasonable activity against Entomophthorales and a more favorable side-effect profile for prolonged use. The patient was counseled on the importance of taking the capsule with food and an acidic beverage to maximize absorption, and the existing omeprazole was discontinued. Liver function tests were monitored at baseline, week 2, week 4 and monthly thereafter, and remained within normal limits throughout. Routine therapeutic drug monitoring of itraconazole trough levels was not available at our institution; the decision to continue therapy was based on clinical and radiological response. The patient was reviewed in the colorectal surgery clinic with a planned repeat CT at 2 months and repeat colonoscopy at 5 months, with surgical resection held in reserve as a rescue strategy.

A follow-up CT at 2 months showed near-complete resolution of the wall thickening in the terminal ileum, cecum and ascending colon, and resolution of the intramural collection. Follow-up colonoscopy at 5 months showed a normal cecum and a healed ileocecal valve. The patient was advised to complete a 12-month course of itraconazole but elected to self-discontinue at 9 months citing full symptom resolution and a preference to stop chronic medication. A telephone follow up at 2 months after discontinuation confirmed that she remained asymptomatic; she was discharged from clinic with instructions to return for repeat imaging and inflammatory markers if symptoms recurred.

Discussion

This case anchors three teaching points for clinicians who care for patients with IBD-like presentations in endemic regions. First, GIB can be successfully managed with antifungal therapy alone when surgical or percutaneous source control is not feasible. Second, a structured exclusion of other IBD mimickers, in particular intestinal tuberculosis, is essential before any immunosuppression is started. Third, intramural collections behave differently to free pericolic abscesses, both in terms of drainability and in terms of the antifungal response that can be expected.

The Saudi GIB literature was reviewed by searching PubMed and Google Scholar on 14 November 2025 using the terms basidiobolomycosis, *Basidiobolus*, Saudi Arabia and gastrointestinal, with no date restriction, supplemented by hand searching the reference lists of included articles. Reports describing adult or pediatric patients with histopathologically or microbiologically confirmed GIB diagnosed in Saudi Arabia were eligible, and we extracted age, sex, presenting symptoms, eosinophilia status, primary anatomical site, treatment and outcome. The accumulated Saudi experience is summarized in Table 2. Recurring features across these reports include male predominance (particularly in pediatric cases from the Jazan region), right lower quadrant abdominal pain, palpable abdominal mass, peripheral eosinophilia in the majority of cases, elevated ESR and CRP, and an ileocecal site of disease.[2,4,5,12–14] The most recent bicentric retrospective series from Jazan and Riyadh, by Dhayhi et al in 2025, reported 42 pediatric cases over a 12-year period, and confirmed the persistent male predominance and the central role of itraconazole- or voriconazole-based regimens.[15] Most adult Saudi cases have undergone surgical resection at diagnosis, but a small number have been managed medically without operation, including the 2025 report by Alsulaimi et al of a 32-year-old Saudi man with

Table 2 Summary of Published Saudi Reports of Gastrointestinal Basidiobolomycosis

Reported Region	Age (Years)/Sex	Clinical Presentation	Duration	Palpable Mass	Laboratory Findings	Treatment	Outcome
Central Region (Riyadh)[12]	58/F	Persistent non-bloody diarrhea, vomiting, diffuse abdominal pain	4 weeks	Yes (lower right quadrant)	Anemia, eosinophilia, high alkaline phosphatase, hypokalemia, elevated BUN, high calprotectin, elevated CA 125, mild hypoalbuminemia, elevated ESR and CRP	Itraconazole 200 mg once daily	Diarrhea and abdominal pain resolved in 2 weeks. Emergency laparotomy for perforated colon after 1 month. Extended right hemicolectomy and end ileostomy
Southern Region (Asir)[5]	8/M	Perianal pain, constipation, gluteal swelling, low-grade fever	3 months	Yes (firm erythematous gluteal mass)	Leukocytosis, neutrophilia, monocytosis, lymphocytosis, eosinophilia, basophilia, markedly elevated ESR, CRP and creatinine	Prolonged course of voriconazole	Excellent response. Marked improvement in blood and kidney function. Complete resolution of bladder and gluteal masses
Central Region (Riyadh)[12]	12/M	Abdominal pain, intermittent fever	2 months	Yes (tender right upper quadrant mass, scrotal swelling)	Leukocytosis, eosinophilia, elevated ESR, low hemoglobin	Oral itraconazole for 2 years, right hemicolectomy	Asymptomatic on follow-up
Central Region (Riyadh)[12]	12/M	Abdominal pain, intermittent fever	3 months	Yes (hepatomegaly)	Eosinophilia, mildly low hemoglobin, markedly elevated ESR	Itraconazole for 1 year, biliary duct stenting	Clinical and radiological improvement
Central Region (Riyadh)[12]	9/M	Abdominal pain, intermittent fever	2 months	Yes (abdominal mass)	Leukocytosis, eosinophilia, markedly elevated ESR	Itraconazole for 2 years, right hemicolectomy	Clinical and radiological improvement
Central Region (Riyadh)[12]	4/M	Fever, abdominal pain	1 month	Yes (massive hepatomegaly)	Leukocytosis, eosinophilia, markedly elevated ESR	Intravenous itraconazole	Asymptomatic on oral itraconazole
Central Region (Riyadh)[12]	3/M	Fever, abdominal pain	2 months	No	Leukocytosis, eosinophilia, markedly elevated ESR	Amphotericin B	Died within 2 days of diagnosis
Central Region (Riyadh)[12]	7/M	Fever, abdominal pain, progressive abdominal distension	Not stated	Yes (hepatosplenomegaly)	Leukocytosis, eosinophilia, markedly elevated ESR	Intravenous itraconazole	Died at a local hospital with massive gastrointestinal bleeding
Central Region (Qassim)[12]	12/M	Sudden-onset abdominal pain with diffuse rigidity and distension	Not stated	Not stated	Not stated	Hemicolectomy and itraconazole	Improved within 1 month and remained well on long-term follow-up
Central Region (Riyadh)[12]	10/M	Severe recurrent right iliac fossa pain, vomiting, fever	3 weeks	Yes (firm to hard right iliac fossa mass)	Leukocytosis, eosinophilia, high platelet count, slight elevation in ESR and CRP	Right hemicolectomy, intravenous itraconazole	Transient postoperative improvement for 7 days, deterioration, then improvement within 10 days of intravenous itraconazole. Complete resolution at 6 months
Central Region (Riyadh)[13]	69/M	Left lower abdominal pain, intermittent fever, weight loss	2 weeks (pain), 1 year (fever and weight loss)	No	Leukocytosis, eosinophilia, neutrophilia, high platelet count, markedly elevated CRP	Adhesiolysis, total colectomy, end ileostomy, liposomal amphotericin B, itraconazole, modification of antibacterial agents	Deteriorated and died of refractory septic shock

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Central Region (Riyadh)[12]	23/M	Constipation, weight loss, rectal bleeding	Not stated	Not stated	Eosinophilia	Itraconazole 200 mg twice daily for 6 months	Good response. No evidence of disease at 6 months
Central Region (Riyadh)[12]	33/F	Vague abdominal pain, weight loss	Long-standing	Not stated	Leukocytosis, eosinophilia	En bloc resection of terminal ileum, appendix, cecum, ascending colon, plus itraconazole 200 mg twice daily for 6 months	Good response. No evidence of disease at 8 months
Central Region (Qassim)[12]	39/F	Progressive severe left abdominal pain, weight loss	2 months	No	Eosinophilia, elevated ESR, CRP, IgG subclasses	Oral itraconazole 200 mg twice daily	Excellent response on CT at 5 months. Laboratory parameters improved
Central Region (Riyadh)[12]	65/M	Lower abdominal pain, constipation, weight loss	1 month	Yes (palpable right-sided abdominal mass)	Leukocytosis, eosinophilia, microcytic hypochromic anemia, high platelet count	Right hemicolectomy, intravenous vancomycin, voriconazole	Postoperative fever and abdominal pain treated with voriconazole and drainage. Liver resection at 2 months. Discharged on voriconazole, made full recovery
Central Region (Riyadh)[12]	11/M	Abdominal pain	3 weeks	Yes (firm mobile mass in right iliac fossa)	Mild anemia, elevated ESR	Oral voriconazole 150 mg twice daily	Laboratory findings normalized in the first month with continued improvement. Voriconazole discontinued after 1 year
Central Region (Riyadh)[12]	12/M	Diffuse abdominal pain, non-bilious vomiting, poor appetite, weight loss	2 months	Right upper quadrant tenderness	Leukocytosis, eosinophilia, markedly elevated ESR and CRP	Right hemicolectomy, itraconazole 100 mg twice daily	Complete clinical and radiological improvement. Treatment discontinued after 12 months
Central Region (Riyadh)[12]	21/F	Colicky epigastric pain, nausea, weight loss, rectal bleeding	3 months (pain, nausea); 1 month (weight loss, bleeding)	Firm ill-defined left epigastric mass	Leukocytosis, hypoalbuminemia	Subtotal colectomy, itraconazole 200 mg daily	Remained well with normalization of laboratory findings on follow-up
Central Region (Riyadh/ Qassim)[14]	32/M	Right lower quadrant abdominal pain, ileocecal mass mimicking malignancy	2 months	Yes (ileocecal mass on imaging)	Eosinophilia, elevated inflammatory markers	Voriconazole, switched to posaconazole because of hepatotoxicity. No surgery	Full clinical and radiological resolution after 4 months. Medical management only
Central Region (Riyadh) – Present case	39/F	Right lower quadrant pain, non-bloody watery diarrhea, IBD-like presentation	7 days	Yes (tender ill-defined right iliac fossa mass)	Eosinophilia (0.54×10^3/μL), ESR 44 mm/hour, CRP 21.5 mg/L, mild lymphopenia	Oral itraconazole 200 mg every 8 hours × 3 days, then 200 mg once daily for 9 months. No surgery. Drainage not feasible (intramural collection)	Near-complete radiological resolution at 2 months. Normal colonoscopy at 5 months. Asymptomatic at follow-up 2 months after self-discontinuation

Abbreviations: IBD, inflammatory bowel disease; BUN, blood urea nitrogen; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IgG, immunoglobulin G.

ileocecal GIB treated successfully with antifungal therapy alone.[14] Our patient adds to this small but growing experience of medical management.

Eosinophilia is one of the most consistent laboratories clues and was the feature that prompted us to broaden the differential beyond Crohn's disease. Across the largest published series, peripheral eosinophilia is reported in approximately 85% of cases.[3,5] In our patient, the absolute eosinophil count of $0.54\times10^3/\mu L$ was modestly elevated above the upper reference limit, in keeping with this pattern, and tissue eosinophilia was the dominant histopathological feature. Eosinophilia is not specific, however, and overlaps with eosinophilic gastroenteritis, parasitic infection (*Strongyloides* in particular), eosinophilic granulomatosis with polyangiitis and hypereosinophilic syndrome, all of which were considered and excluded in our patient through stool studies, vasculitis serology and clinical evaluation.

Intestinal tuberculosis is the single most important diagnostic competitor in this clinical setting. Caseating granulomas with central necrosis and AFB-positive bacilli on biopsy distinguish tuberculosis from GIB, and a structured workup with QuantiFERON or T SPOT.TB testing, mycobacterial culture and AFB staining of biopsy material is mandatory. In our patient, QuantiFERON was negative, AFB staining was negative, mycobacterial culture grew no organisms after 6 weeks, and histopathology showed broad sparsely septate hyphae with the Splendore–Hoeppli phenomenon rather than caseating granulomas. The diagnostic overlap between intestinal and hepatobiliary tuberculosis and other infectious mimickers has been thoroughly reviewed by Esguerra-Paculan and Soldera, who emphasize that histological demonstration of *Mycobacterium tuberculosis* is the definitive diagnostic step.[8] Paracoccidioidomycosis was also considered because it can present with right lower quadrant ileitis and granulomatous histopathology in immunocompetent hosts, although it is essentially restricted to Latin America and is morphologically distinguishable by its multiple budding yeast forms on Grocott stain.[9] Drug-induced colitis from sodium polystyrene sulfonate, mycophenolate or checkpoint inhibitors was considered and excluded by medication history.[10] Polyarteritis nodosa, which our patient carried as a background diagnosis, can itself cause gastrointestinal ischemia and ulceration that may mimic Crohn's disease,[11] but the absence of mesenteric arterial findings on CT, the absence of systemic flare and the presence of fungal hyphae on biopsy made this an unlikely contributor to the colonic lesion. A 2025 narrative review by Bakkaloğlu and Çelik provides a useful framework for systematically working through these IBD mimickers in clinical practice.[11]

The decision to manage this patient medically rather than surgically was driven by the intramural nature of the collection. Free pericolic or pelvic abscesses larger than 3–4 cm are typically drained percutaneously under CT or ultrasound guidance, with success rates above 80%, but collections contained entirely within the bowel wall are not readily accessible without traversing healthy bowel and risking perforation or fistula formation. For this anatomical subset, antibiotic and antifungal management with close radiological follow-up, or formal bowel resection, are the two principal options. Itraconazole was selected over voriconazole because the published Saudi adult experience with itraconazole-based regimens has been favorable,[2,4,12] and because voriconazole carries a higher risk of hepatotoxicity, visual disturbance and skin photosensitivity during prolonged use.[13] Liver function tests were monitored monthly and remained normal. Therapeutic drug monitoring of itraconazole trough levels is recommended in international guidance for invasive fungal infection because of variable oral absorption, although it was not available at our institution; the clinical and radiological response served as a pragmatic substitute. The optimal duration of therapy in GIB is uncertain, and has ranged from 6 to 12 months in published Saudi and international series.[3,4,12] Our patient adhered to 9 months of therapy and remained well at short-term follow-up; longer follow-up would be needed to confirm durable remission.

Limitations

This report has several limitations. As a single case, it cannot support generalizable conclusions about medical management of intramural GIB collections. Species-level identification of the *Basidiobolus* isolate was not performed, so we cannot confirm *Basidiobolus ranarum* as the causative species, and the diagnosis rests on the genus-level culture together with the characteristic histopathology. Source control by drainage was not achieved, which complicates any direct comparison with the standard surgical literature. Treatment duration was 9 months rather than the planned 12 months, and follow-up after discontinuation has so far been limited to 2 months, so the possibility of late relapse cannot be excluded. Finally, the risk factor odds ratios mentioned in the Introduction are taken from a small case–control study with wide confidence intervals,[5] and should be interpreted as hypothesis generating rather than as established causation.

Conclusion

In endemic regions, an IBD-like presentation with right lower quadrant pain, an ileocecal mass or abscess on imaging and peripheral eosinophilia should prompt early endoscopic biopsy with fungal stains and culture, and a structured exclusion of intestinal tuberculosis, paracoccidioidomycosis and drug-induced colitis before any immunosuppressive therapy is started. Early itraconazole-based antifungal therapy can avert surgery when source control is not technically feasible, as in our patient with an intramural cecal collection. The optimal duration of antifungal therapy in GIB remains uncertain, and a structured follow-up plan with symptom-triggered repeat imaging and inflammatory markers is advisable after treatment discontinuation.

Data Sharing Statement

The data and materials supporting this case report are available upon request from the corresponding author.

Ethical Approval and Consent for Publication

Institutional approval to publish the case details was obtained from the Institutional Review Board of King Fahad Medical City, Riyadh, Saudi Arabia (log number 24-582). Written informed consent for publication of the clinical details and accompanying images was obtained from the patient.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest concerning the materials or methods used in this study or the findings specified in this paper.

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