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# WHEN EOSINOPHILS MISLEAD: GASTROINTESTINAL BASIDIOBOLOMYCOSIS INITIALLY MISDIAGNOSED AS EOSINOPHILIC COLITIS

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## ABSTRACT

*Introduction:* Gastrointestinal basidiobolomycosis (GIB) is a rare fungal infection caused by *Basidiobolus ranarum* that predominantly affects immunocompetent individuals in tropical and subtropical regions. It frequently mimics colonic malignancy or inflammatory bowel disease, leading to significant diagnostic delays.

*Case description:* A 36-year-old previously healthy Indian male presented with a 1-month history of abdominal pain, bleeding per rectum, and weight loss. Computed tomography scan revealed circumferential wall thickening of the ascending colon from the ileocecal junction to the hepatic flexure with loco-regional and para-aortic lymphadenopathy. Colonoscopy demonstrated a large circumferential inflammatory mass causing significant luminal narrowing. Initial histopathology from endoscopic biopsies was interpreted as eosinophilic colitis. However, expert review identified broad, pauciseptate fungal hyphae surrounded by eosinophilic material consistent with the Splendore-Hoeppli phenomenon, confirming GIB. The patient was commenced on oral itraconazole 200 mg twice daily with significant clinical, biochemical, and radiological improvement at 3-month follow-up, without the need for surgical intervention.

*Discussion:* This case highlights the diagnostic pitfall of misinterpreting GIB as eosinophilic colitis on superficial endoscopic biopsies and demonstrates successful non-surgical management with itraconazole monotherapy.

*Conclusion:* GIB should be considered in the differential diagnosis of obstructing colonic masses in immunocompetent patients with eosinophilia. When initial biopsies suggest eosinophilic colitis in the context of a mass lesion, expert histopathological review with attention to fungal elements is essential.

## KEYWORDS

Basidiobolomycosis, gastrointestinal fungal infection, eosinophilic colitis, Splendore-Hoeppli phenomenon, colonic mass

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## LEARNING POINTS

- Gastrointestinal basidiobolomycosis (GIB) can closely mimic colonic carcinoma or inflammatory bowel disease endoscopically and radiologically; a high index of suspicion is essential in immunocompetent patients presenting with obstructing colonic masses and peripheral eosinophilia in tropical and subtropical regions.
- Superficial endoscopic biopsies in GIB frequently yield dense eosinophilic infiltration misdiagnosed as eosinophilic colitis; expert histopathological review focusing on the Splendore-Hoeppli phenomenon on haematoxylin and eosin stain can be diagnostic, and special stains (Grocott-Gomori methenamine silver, periodic acid-Schiff) should be requested when available to further confirm the diagnosis.
- Early diagnosis of GIB enables curative antifungal therapy and may avert unnecessary surgical resection, underscoring the importance of including this entity in the differential diagnosis of colonic masses with inconclusive initial histopathology.

## INTRODUCTION

Gastrointestinal basidiobolomycosis (GIB) is an uncommon but emerging fungal infection caused by *Basidiobolus ranarum*, an environmental saprophyte belonging to the order Entomophthorales. Although *B. ranarum* is found worldwide in soil, decaying vegetation, and the gastrointestinal tracts of amphibians and reptiles, GIB is predominantly reported from tropical and subtropical regions, including Saudi Arabia, the southwestern United States, Iran, and Kuwait[1,2]. The infection characteristically affects immunocompetent individuals and most commonly involves the cecum and ascending colon[3].

The clinical presentation of GIB is non-specific, typically featuring abdominal pain, fever, weight loss, and a palpable abdominal mass, frequently leading to an initial misdiagnosis of colonic malignancy, inflammatory bowel disease, or intestinal tuberculosis[4,5]. The histopathological hallmark is the Splendore-Hoeppli phenomenon: broad, thin-walled, pauciseptate fungal hyphae surrounded by intensely eosinophilic material within a granulomatous and eosinophil-rich inflammatory background, identifiable on haematoxylin and eosin (H&E) staining and further confirmed by special stains such as Grocott-Gomori methenamine silver (GMS) and periodic acid-Schiff (PAS) when available[3]. However, the fungal elements typically reside in the submucosal and muscular layers, meaning that superficial endoscopic biopsies frequently yield only dense eosinophilic infiltration that can be misinterpreted as eosinophilic colitis[5,6].

We report a case of GIB involving the ascending colon in a 36-year-old Indian male, initially misdiagnosed as eosinophilic colitis on endoscopic biopsy and successfully treated with itraconazole monotherapy without surgical intervention.

## CASE DESCRIPTION

A 36-year-old previously healthy Indian male presented with a 1-month history of progressive abdominal pain, bleeding per rectum, and unintentional weight loss of approximately 5 kg. He reported several episodes of fresh blood mixed with stool, along with alternating loose stools and constipation. He denied fever, night sweats, or prior similar episodes. His past medical history was unremarkable with no history of immunosuppression or family history of malignancy or inflammatory bowel disease.

On examination, the patient appeared pale. Vital signs were stable. Abdominal examination revealed right-sided tenderness with a vague fullness in the right hypochondrium. There was no hepatosplenomegaly, ascites, or peripheral lymphadenopathy. Laboratory investigations

| Parameter | Result | Reference range |
|---|---|---|
| **Haemoglobin (g/dl)** | 10.8 | 13.0–17.5 |
| **White blood cell count (×10⁹/l)** | 14.2 | 4.0–11.0 |
| **Eosinophils (%)** | 22.6 | 1–6 |
| **Absolute eosinophil count (×10⁹/l)** | 3.21 | 0.04–0.44 |
| **Platelets (×10⁹/l)** | 486 | 150–400 |
| **ESR (mm/hr)** | 68 | 0–15 |
| **C-reactive protein (mg/l)** | 78.4 | <5 |
| **Albumin (g/dl)** | 3.1 | 3.5–5.0 |
| **ALT / AST (U/l)** | 28 / 32 | <40 / <40 |
| **Creatinine (mg/dl)** | 0.9 | 0.7–1.3 |
| **CEA (ng/ml)** | 2.1 | <5.0 |
| **CA 19-9 (U/ml)** | 12.4 | <37 |
| **HIV serology** | Negative | — |
| **Stool culture** | Negative | — |
| **Stool ova and parasites** | Negative | — |

***Table 1.*** *Laboratory investigations at presentation.*
*Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CA 19-9, carbohydrate antigen 19-9; CEA, carcinoembryonic antigen; ESR, erythrocyte sedimentation rate; HIV, human immunodeficiency virus.*

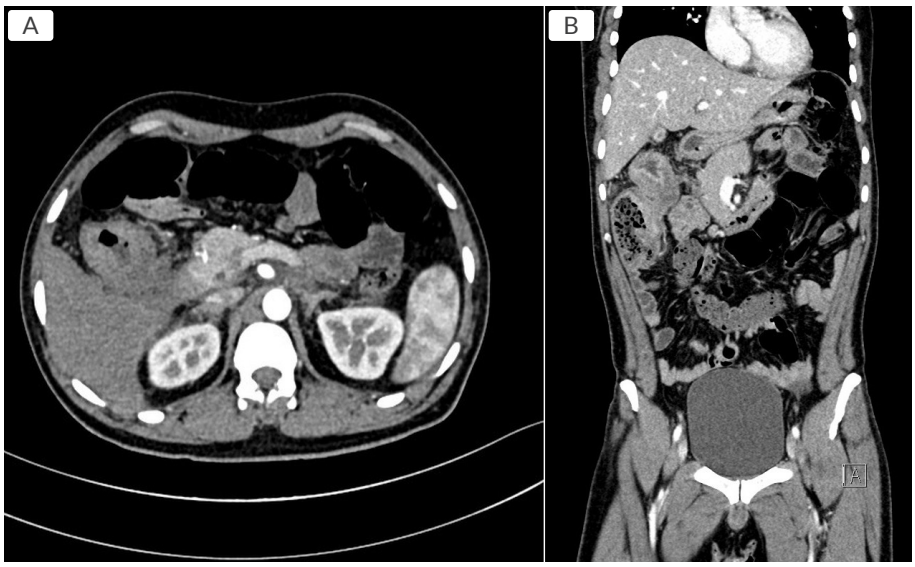



*Figure 1. Contrast-enhanced computed tomography scan of the abdomen. A) Axial view demonstrating circumferential wall thickening of the ascending colon with luminal narrowing and pericolonic fat stranding. B) Coronal view showing the extent of the mural thickening from the ileocecal junction to the hepatic flexure, with mesenteric lymphadenopathy.*

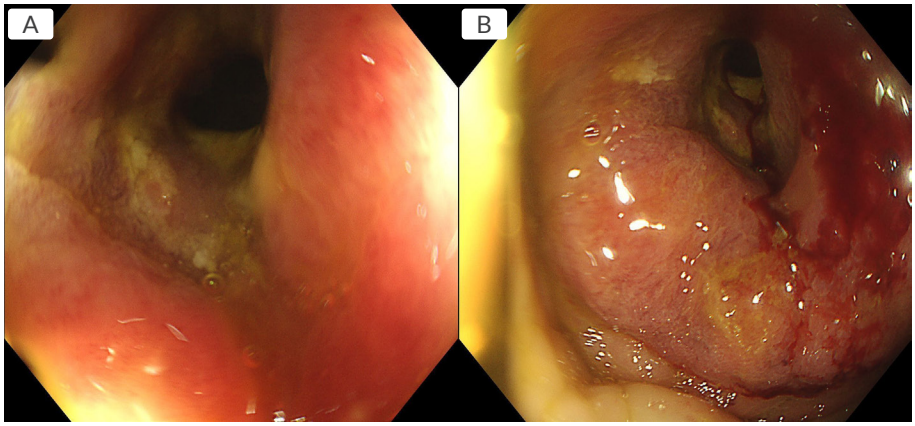



*Figure 2. Colonoscopic images of the lesion at the hepatic flexure. A) Circumferential inflammatory mucosal thickening with significant luminal narrowing, preventing passage of the colonoscope. B) Close-up view demonstrating a friable, oedematous, and ulcerated mass-like lesion with irregular nodular inflammatory mucosa and contact bleeding.*

are summarized in *Table 1*. Tumour markers including carcinoembryonic antigen and carbohydrate antigen 19-9 were within normal limits. Human immunodeficiency virus serology was negative.

Contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis revealed circumferential mural wall thickening involving the ascending colon from the level of the ileocecal junction to the hepatic flexure, measuring approximately 10 cm in length and 18 mm in thickness, with mild post-contrast enhancement. The thickening was associated with mesenteric fat stranding, peritoneal thickening, and loco-regional pathological lymphadenopathy (largest 2 cm in short axis). Para-aortic lymph nodes were also noted. Subsequent dilatation of the terminal ileum reaching 3 cm in maximum diameter was seen, suggesting an element of mechanical small bowel obstruction. The liver, pancreas, spleen, kidneys, and urinary bladder were normal in appearance *(Fig. 1)*. Colonoscopy was subsequently performed. The colonoscope was advanced to the hepatic flexure, where a large circumferential inflammatory mass-like lesion was identified causing significant luminal narrowing with partial obstruction. The lesion appeared friable, oedematous, and ulcerated, with irregular nodular inflammatory mucosa and contact bleeding on gentle manipulation *(Fig. 2)*. The scope could not be advanced beyond the lesion. Multiple biopsies were obtained.

Initial histopathological examination of the endoscopic biopsies demonstrated a dense inflammatory infiltrate rich in eosinophils, with scattered multinucleated giant cells and areas of mucosal ulceration. No malignant cells were identified. The findings were initially interpreted as eosinophilic colitis. However, in view of the clinical presentation—a young male with an obstructing colonic mass, weight loss, and marked peripheral eosinophilia—and the endoscopic appearance suggesting an inflammatory or infectious aetiology rather than primary eosinophilic disease, GIB was considered in the differential diagnosis. Expert histopathological review of the H&E-stained biopsies identified broad, thin-walled, pauciseptate fungal hyphae surrounded by intensely eosinophilic, amorphous radiating material consistent with the Splendore-Hoeppli phenomenon, confirming the diagnosis of GIB *(Fig. 3)*. Tissue sent for fungal culture on Sabouraud dextrose agar at 30°C did not yield growth after 3 weeks of incubation.

The patient was commenced on oral itraconazole 200 mg twice daily with regular monitoring of hepatic function. Within 2 weeks, he reported significant improvement in abdominal pain and cessation of rectal bleeding. At 1-month follow-up, his appetite had improved and he had regained 2 kg of weight. Laboratory investigations at 3 months showed normalization of the eosinophil count (4.2%), reduction in

erythrocyte sedimentation rate (18 mm/hr) and C-reactive protein (6.2 mg/l), and improvement in haemoglobin (12.4 g/dl). A follow-up CT abdomen at 3 months demonstrated marked interval reduction in the wall thickening with resolution of the pericolonic fat stranding and reduction in the lymphadenopathy. The patient remains on itraconazole therapy with a planned total duration of 12 months, with ongoing clinical and laboratory surveillance.

## DISCUSSION

This case underscores the critical diagnostic pitfall of misinterpreting GIB as eosinophilic colitis on endoscopic biopsies. The fungal elements in GIB typically involve the submucosal and muscular layers of the bowel wall, meaning that standard mucosal biopsies may capture only the dense eosinophilic infiltration without the diagnostic fungal hyphae[6,7]. This pitfall was recently corroborated by Al Hassani et al. (2025), who described a 52-year-old South Asian male in whom colonoscopic biopsies initially suggested eosinophilic colitis before the diagnosis of GIB was established[5]. Similarly, Meeralam et al. (2024) reported that endoscopic biopsies showed only non-specific inflammation in most of their five cases, with the Splendore-Hoeppli phenomenon identified on initial biopsy in only one patient[6]. In our case, expert re-review of the H&E-stained biopsies with a heightened clinical suspicion identified the characteristic fungal morphology. These findings reinforce the importance of considering GIB whenever dense eosinophilic infiltration is encountered in the context of a colonic mass lesion. Where available, special stains such as GMS and PAS can further confirm the diagnosis by highlighting the fungal wall[7].

Our patient demonstrated an excellent clinical, biochemical, and radiological response to itraconazole monotherapy without surgical intervention, adding to the growing evidence supporting medical management in uncomplicated GIB[5,6]. The negative fungal culture is consistent with the published literature, where cultures are negative in the majority of cases and the diagnosis is established on histopathological grounds[6-8]. A limitation of this report is that special stains (GMS, PAS) were not performed; however, the characteristic H&E morphology of broad, pauciseptate fungal hyphae with the Splendore-Hoeppli phenomenon, combined with the clinical context and sustained treatment response, provides strong diagnostic support. Molecular diagnostic techniques such as pokymerase chain reaction may complement histopathology in equivocal cases[3].

GIB has been reported in South Asian expatriates residing in the Gulf region, including cases from Kuwait described by Khan et al.[4]. Whether our patient acquired the infection in his country of origin or in Kuwait remains uncertain, but this case adds to the limited literature and highlights the importance of considering GIB in expatriate populations in endemic regions, particularly given the increasing globalization of travel and migration[9,10].

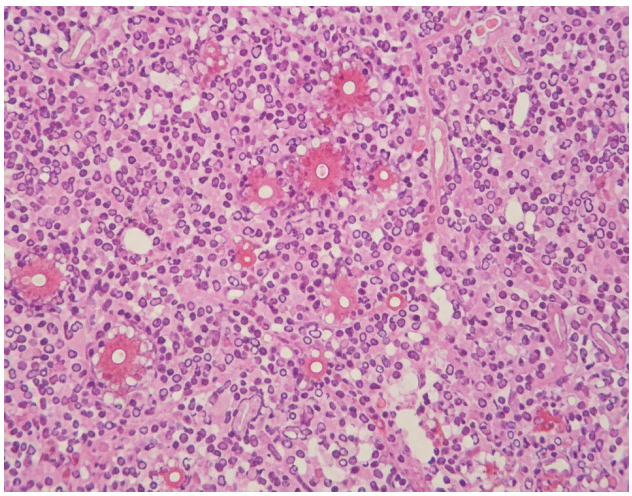


*Figure 3. Histopathology of the colonic biopsy (haematoxylin and eosin stain). Dense eosinophilic inflammatory infiltrate with broad, thin-walled, pauciseptate fungal hyphae surrounded by intensely eosinophilic amorphous material consistent with the Splendore-Hoeppli phenomenon. Scattered multinucleated giant cells and inflammatory debris are seen in the background.*

Awareness of this rare but curable infection, combined with expert histopathological review when initial biopsies are inconclusive, is key to avoiding diagnostic delays and unnecessary surgical intervention.

## CONCLUSION

GIB is a rare but potentially curable fungal infection that closely mimics colonic malignancy and inflammatory bowel disease. This case highlights the importance of maintaining a high index of suspicion for GIB in immunocompetent patients presenting with obstructing colonic masses and peripheral eosinophilia. When endoscopic biopsies demonstrate dense eosinophilic infiltration in the context of a mass lesion, expert histopathological review with attention to the Splendore-Hoeppli phenomenon is essential before attributing the findings to eosinophilic colitis. Special stains (GMS, PAS) should be requested when available. Early recognition enables targeted antifungal therapy and may avert unnecessary surgical intervention.

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