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Gastrointestinal Basidiobolomycosis: A Diagnostic Dilemma in Clinical Practice – A Case Report

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ABSTRACT

The following is a case report of a 17-year-old immunocompetent female with no significant past medical history, who presented to King Fahad Hospital after multiple previous hospital visits. She was complaining of unexplained abdominal pain, fever, weight loss, and diarrhea. Initial investigations raised concerns about colon malignancy or inflammatory bowel disease. However, further evaluation, including endoscopy and biopsy, revealed the diagnosis of gastrointestinal basidiobolomycosis through histopathological study. Treatment with antifungals started for 6 months, and the patient showed remarkable improvement during clinical stay and follow-up.

Keywords

Gastrointestinal basidiobolomycosis, Fungal colitis, Crohn's disease mimicry, Splendore–Hoeppli phenomenon.

Introduction

Basidiobolomycosis is a type of Zygomycosis, an infection caused by fungi from the orders Entomophthorales and Mucorales within the class Zygomycota. The specific fungus responsible for Basidiobolomycosis is Basidiobolus ranarum, which occurs saprophytically in decaying plant material. This fungus has frequently been isolated from the intestines of amphibians and reptiles, such as frogs, toads, garden lizards, and chameleons. However, the exact mechanism of transmission remains poorly understood [1]. B. ranarum is primarily known for causing subcutaneous Zygomycosis, a condition that is widespread in tropical and subtropical regions, including parts of Africa, Asia, the United States, and Latin America. While subcutaneous infections are well-documented, there has been an increase in reported gastrointestinal manifestations in recent years, whereas extraintestinal manifestations are infrequent [2]. The first case of gastrointestinal Basidiobolomycosis was documented in Nigeria in 1964 [3] and since then, additional cases have been recorded. Gastrointestinal Basidiobolomycosis is a complex and poorly understood disease that mimics various gastrointestinal pathologies, including malignancies and chronic granulomatous diseases like tuberculosis and Crohn's disease, making diagnosis very challenging [4]. Our case report highlights the challenges we faced in accurately diagnosing gastrointestinal Basidiobolomycosis at King Fahad Hospital, emphasizing the clinical importance of expanding the differential diagnosis in clinical practice.

Case Presentation

A 17-year-old female with no significant past medical history presented to the emergency room (ER) with complaints of severe, vague, periumbilical abdominal pain of a colicky nature that had been ongoing for three months. The pain was associated with subjective fever, nausea, vomiting of food content, and watery, non-bloody diarrhea that had lasted for the last two months. She also reported significant weight loss, loss of appetite, and an inability to tolerate oral intake. During this period, she sought explanations from various hospitals and was treated without a professional diagnosis with a triple therapy for Helicobacter

pylori infection, but her symptoms did not improve. Suspicions of colon malignancy or inflammatory bowel disease (IBD) were raised after a computed tomography (CT) scan of the abdomen revealed colon masses with features suggestive of malignancy. She then decided to visit our hospital for a second opinion before undergoing a colonoscopy. On physical examination, the patient was hemodynamically stable and afebrile but underweight, with a BMI of 17, and exhibited tachycardia. Her abdomen was soft and lax, with mild to moderate tenderness upon palpation of the epigastric area, and no masses were appreciated. A per-rectum (PR) exam revealed no melena; however, perianal skin tags were found. Laboratory studies indicated leukocytosis with neutrophilia and eosinophilia. Inflammatory markers, including erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), were elevated (See Table 1). Absorption studies showed nutritional deficiencies in iron, vitamin D, folate, and vitamin K. The autoimmune panel was negative for anti-nuclear antibody (ANA) and anti-neutrophil cytoplasmic antibodies (ANCA), but positive for anti-saccharomyces cerevisiae antibody (ASCA). Celiac disease testing was negative, as was fecal calprotectin (Table 1). A stool ova and parasite smear, as well as a Clostridium difficile PCR, were also negative, while the TB QuantiFERON gold test returned negative. The CT scan of the abdomen and pelvis revealed thickening of the terminal ileum, cecum, and ascending colon, with significant surrounding inflammatory changes and lymphadenopathy (See Figure 1). Thick-walled abscesses were noted, the largest measuring 2.6 x 2.7 cm. The radiological findings suggested severe atypical infections, such as gastrointestinal tuberculosis or atypical fungal infections, were more likely than malignancy, although gastrointestinal lymphoma remained a differential diagnosis. An upper endoscopy showed a normal esophagus and stomach, and the cardia and gastric fundus appeared normal on retroflexion. Both the first and second parts of the duodenum were normal. During colonoscopy, an ulcerated non-obstructing large mass was identified in the descending colon, at the splenic flexure, and in the transverse and hepatic flexures (See Figure 2). A tissue biopsy, with deep tissue samples (See Figure 3) taken from the large intestine specifically at the cecum, confirmed a Basidiobolomycosis infection through a positive tissue culture. The biopsy, performed with hematoxylin and eosin stain (H&E), reveals extensive necrosis with granulomatous inflammation. A thin, broad hypha with optically clear centers was observed in the necrotic areas, surrounded by eosinophilic material known as the Splendor–Hoeppli phenomenon. The Gomori methenamine silver (GMS) stain highlights the fungal wall. Additionally, the adjacent colon mucosa shows heavy eosinophilic infiltration, with no evidence of chronic inflammatory changes, dysplasia, or malignancy. Superficial cultures of the mass were also positive for Staphylococcus haemolyticus and Pseudomonas aeruginosa. An AFB culture showed no growth. There was no evidence of dysplasia or malignancy in the tissue samples. The patient was initially unable to tolerate oral medication, so intravenous posaconazole was started. She was later transitioned to oral posaconazole to complete a six-month course of antifungal treatment, resulting in significant improvement. Fortunately, surgical intervention was not needed. Later on, the patient demonstrated notable clinical improvement during the follow-up period, with stable vital signs and an overall well-appearing status. Physical examination revealed no concerning findings, and the patient reported resolution of previous symptoms with no new complaints. Laboratory parameters returned to normal limits, supporting her clinical stability. The patient was compliant with her medication, contributing to favorable outcomes. Overall, her condition was well-maintained, and her recovery rate showed a positive response to the management plan initiated during an ongoing follow-up evaluation that lasted for eight months.

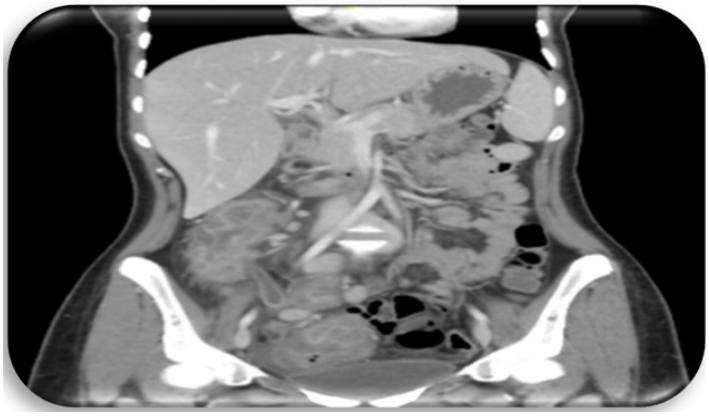

Image 1: Coronal contrast-enhanced CT of the abdomen demonstrated diffuse edematous wall thickening of the cecum and ascending colon with adjacent fat stranding, multiple enlarged mesenteric lymph nodes, and thick-walled rim-enhancing collections; findings are consistent with an aggressive granulomatous infectious process, later confirmed as gastrointestinal basidiobolomycosis.

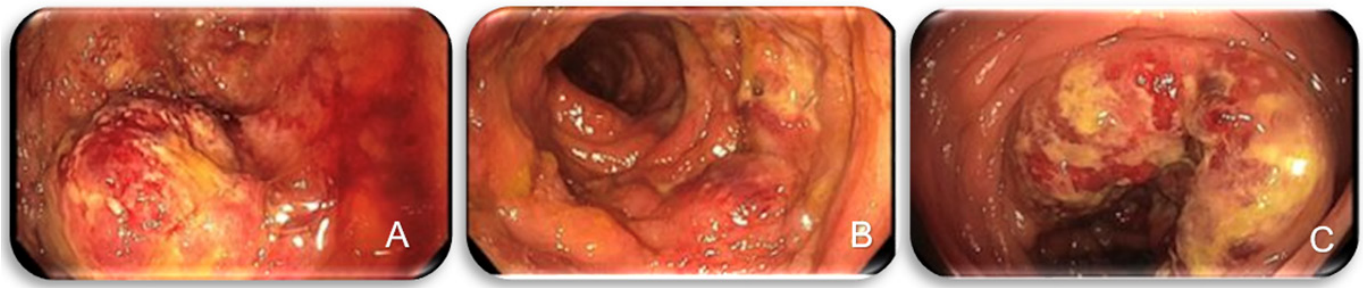

Image 2: A large non-circumferential, ulcerated, and non-obstructing mass with no bleeding was found in the cecum (A) and the ileocecal valve. In addition, the mass was found in the transverse colon along with the hepatic flexure (B) and splenic flexure (C), as well as in the descending colon.

Table 1: Laboratory Finding.

Category	Analysis	Result	Normal Range	Category	Analysis	Result	Normal Range
CBC	**WBC**	18.20 (10^3/UL)	3.90 - < 11.00	**Anemia Profile**	**Iron**	3.2 (umoL/L)	9 – 30
	Neutrophilis	8.4 (10^3/UL)	1.35 - <7.5		**TIBC**	26.2 (umoL/L)	Dec-55
	Esinophils	4.26 (10^3/UL)	0.25 - < 1		**Feritin**	244 (ug/L)	5 – 67
	Hemoglobin	8.9 (g/dL)	11 - <16		**Folate**	16 (nmoL/L)	18 – 46
	Hematocrit	27%	42 - < 47%		**B12**	329 (pmoL/L)	150 – 599
	MCV	73.5 fL	75- <95	**Coagulation Profile**	**INR**	1.86	0.87 – 1.16
	MCH	24.2pg	24 - <30		**APTT**	52.1	28.7 – 39.7
	Platelet	743 (10^3/UL)	150-450	**Infectious Disease Screening & Cultures**	Blood x2, Stool x2, Respiratory & Urine Culture.	Negative	
Inflammation & Autoimmune Markers	**ESR**	> 98 mm/H	0-30		Ova&Parasite x3 (Microscopic examination)	No ova, cysts, or parasite seen	
	CRP	178 mg/L	<5.0 mg/L				
	ANA	NEGATIVE			Tissue Culture (Cecum)	Pseudomonas aeruginosa Abnormal MDR	
	ANCA	NEGATIVE					
	CCP	NEGATIVE		**PCR Analyses**	PCR CLOSTRIDIUM DIFFICILE (CDF)	Negative	
	RF	NEGATIVE			PCR HIV-1	quantitative -> negative	
	anti-dsDNA AB	NEGATIVE			PCR viral panel	unremarkable	
	C3 C4	NEGATIVE			MRSA PCR	NEGATIVE	
Celiac Profile	(TTG) IGA AB	0.49	<4 U/ml		HSV PCR	NEGATIVE	
	GLIADIN IGA AB	Negative		**Urinalysis**	Urine examination		
	GLIADIN IGG AB	Negative			WBC	22/HPF	
ASCA	ASCA IGG	(73.52)Positive	< 20 Units		RBC	443/HPF	
	ASCA IGA	(18.06) Negative	< 20 Units		Bacteria in urine	positive	
Fecal calprotectin		Negative					
Mycoplasm Examination (Sputum, Tissue)	AFB Culture	No Growth after 6 Weeks					
	PCR MBT	Target Not Detected					

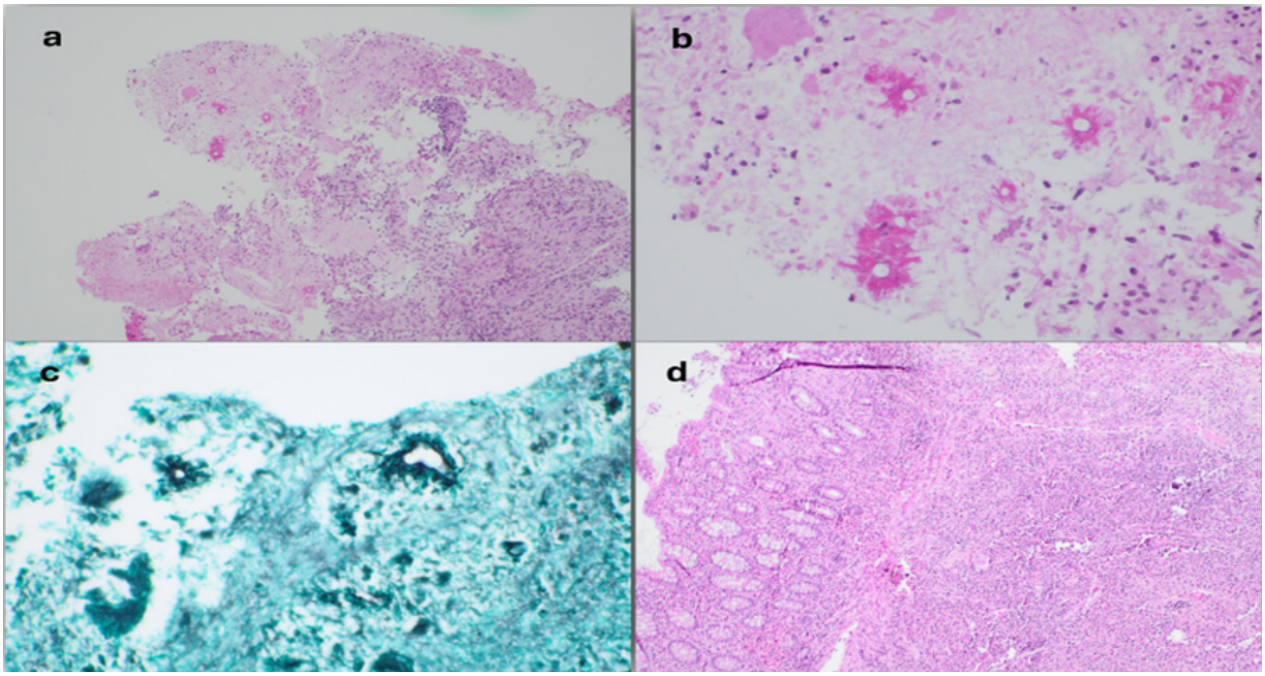

Image 3: Histopathology of the colon biopsies showed granulomatous inflammation with extensive necrosis. B) The organisms are broad, have optically clear centers, and are surrounded by a striking Splendore-Hoeppli reaction. C, GMS stain highlights the fungal organisms (d) and adjacent colon with eosinophilic infiltration and no chronic inflammatory changes or malignancy.

Discussion

Gastrointestinal basidiobolomycosis (GIB) is a rare but serious fungal infection caused by the organism Basidiobolus ranarum, which belongs to the class Zygomycota. While it is predominantly known for causing subcutaneous infections, recent reports highlight its alarming potential for visceral gastrointestinal involvement. The exact mechanism of pathogenesis of basidiobolomycosis remains unclear; however, it is believed that the infection is contracted through ingestion of contaminated food and soil, leading to infection within the submucosa and muscularis layers of the bowel, eventually resulting in granulomatous lesions and inflammatory masses [1,5]. Notably, a significant majority of documented cases involved male children and young adults, which may suggest gender and age-related susceptibility factors [6]. Disturbingly, 37.2% of basidiobolomycosis cases have been reported from Saudi Arabia, establishing it as the country with one of the highest incidences globally. The majority of these cases originate from the southwestern and southern regions, particularly Aseer and Jazan, where the subtropical climate and specific environmental conditions could foster the growth of Basidiobolus species [6,7]. Gastrointestinal basidiobolomycosis can present with a wide range of symptoms. Common symptoms include abdominal pain, abdominal masses, constipation, and fever, Other gastrointestinal symptoms that have been observed include diarrhea, vomiting, lower gastrointestinal bleeding, and hepatomegaly [5-7]. This variety of symptoms, combined with the absence of specific risk factors, poses significant challenges for physicians in clinical practice, often leading to misdiagnoses. Reports have shown that gastrointestinal basidiobolomycosis is frequently misidentified as other conditions such as malignancy, inflammatory bowel disease, appendicitis, and lymphoma [5,6]. Imaging studies often reveal abdominal masses and focal bowel thickening, which can further confuse the condition with neoplasms or other bowel pathologies, Additionally, B. ranarum is located deep beneath the mucosa, which can lead to unrepresentative biopsy results. Biopsies may reveal nonspecific features such as inflammatory changes and granulomatous formation [5,6]. Histopathology of biopsy specimens can sometimes confirm the diagnosis by displaying characteristics like thin-walled, broad hyphae often surrounded by the characteristic Splendore–Hoeppli phenomenon, which consists of radiating, intensely eosinophilic material, which can be visualized using hematoxylin and eosin staining [1,5,6]. These fungal features can resemble those of mucormycosis; however, angioinvasion, commonly seen in mucormycosis, is rare in gastrointestinal basidiobolomycosis. While the Splendore–Hoeppli phenomenon may aid in recognizing fungal involvement, it is non-specific and may occur in response to a variety of microorganisms or inert materials. Therefore, the use of Periodic Acid-Schiff (PAS) and Gomori's Methenamine Silver (GMS) stains is highly appreciable, as it can enhance the visibility of the fungal cell wall and reveal zygospores, which closely resemble the trophozoites of amoebae [1,6,8]. Consequently, all of these difficulties and challenges faced during clinical practice can, eventually, lead to delays in both diagnosis and treatment.

Although there are no specific guidelines for the treatment of GIB, the treatment often involves both medical and surgical approaches. Antifungal medications should be given after surgical removal of the affected part of the bowel [5-7]. Notably combined surgical and antifungal therapy remains the most commonly reported treatment strategy, some reports, including our case, suggest that selected patients may respond to antifungal therapy alone [6,9]. While GIB

typically has a favorable prognosis, certain studies suggest that it may result in serious outcomes, such as spreading to different organs, worsening infections, and causing bowel perforation. Additionally, it has been noted to carry a significant mortality risk even in healthy individuals [6,9,10]. As a result, this increases awareness of considering GIB as a differential diagnosis, as failure to do so may lead to undesirable and potentially fatal outcomes during clinical practice.

Conclusion

GIB is a challenging emerging disease. Its non-specific symptoms make the diagnosis difficult and can lead to misdiagnosis. Although it is a curable disease, its confusing nature can result in serious outcomes. Our case report highlights the importance of considering GIB in the differential diagnosis early, as this can lead to more favorable outcomes and potentially avoid the need for surgical intervention.

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