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Pediatric gastrointestinal basidiobolomycosis

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Basidiobolomycosis is an unusual fungal infection that manifests in the skin and rarely involves other systems including the gastrointestinal tract. We retrospectively reviewed records of six pediatric patients (≤14 years of age) diagnosed with gastrointestinal basidiobolomycosis from March 2000 to March 2002. Four patients came from the same region, suggesting environmental exposure. Basidiobolomycosis should be considered in the differential diagnosis in pediatric patients presenting with abdominal mass and eosinophilia.

INTRODUCTION

Basidiobolomycosis is a rare fungal infection caused by *Basidiobolus ranarum*, an environmental saprophyte found worldwide.[1] *B. ranarum* is a member of the order Entomophthorales of the class Zygomycetes.[1]

Basidiobolomycosis usually presents as a subcutaneous infection that affects mostly young males and is thought to be transmitted through traumatic inoculation.[1, 2] Most cases have been reported from tropical and subtropical regions.[1, 3, 4]

To our knowledge only 15 cases of gastrointestinal basidiobolomycosis (GIB) have been reported worldwide: 8 cases from the United States; 4 cases from Brazil; 2 cases from Nigeria; and 1 case from Kuwait. Most of the reported cases were adults[5–8] with only 3 pediatric patients (2 from Brazil and one from Nigeria).[6, 7, 9]

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Key words: Basidiobolomycosis, fungal; *Basidiobolus ranarum,* Splendore-Hoeppli phenomenon, tissue eosinophilia.

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Because this is a rare disease, risk factors, clinical presentation and treatment of GIB are poorly understood. We report the clinical features of six Saudi pediatric patients with GIB and review the literature.

MATERIALS AND METHODS

Case definition. Each case was diagnosed based on typical histopathologic features (granulomatous inflammation with tissue eosinophilic infiltration and broad based hyphae on Gomori methenamine silver (GMS) stain with or without isolation of *B. ranarum* from surgical specimens.

Medical record review. Health records of six pediatric patients (≤14 years of age) with GIB were retrospectively reviewed. Those patients were diagnosed between March 2000 and March 2002 at three tertiary care centers in Riyadh (King Faisal Specialist Hospital and Research Centre, King Khalid University Hospital and Security Forces Hospital), Saudi Arabia. Data collected included demographic data, symptoms, signs, laboratory, radiologic studies, procedures, treatment and outcome.

Culture and histopathology confirmation. Specimens were inoculated onto Sabouraud dextrose and brain-heart infusion agars. Plates or tubes were incubated at 28–30°C and checked for growth every 48 h of incubation for the first week of the culture. Plates were inspected for growth of buff to gray, waxy, flat to folded colonies on both culture media for 3 to 7 days. Lactophenol aniline blue mount preparations were examined under the microscope. Identification of *Basidiobolus* by observing broad hyphae with sparse septations and thick walled, round intercalary zygospores with conjugation beak on one side which are characteristics of *B. ranarum*.

Pathologic tissues were examined for evidence of granulomatous inflammation, tissue eosinophilia, necrosis, fungal elements consistent with zygomycosis and structures showing the Splendore-Hoeppli phenomenon (radiating, intensely eosinophilic granular material surrounding the fungal elements).

RESULTS

Clinical and laboratory features. Table 1 provides detailed clinical, laboratory and surgical features

of all patients included. All six patients were male; four (67%) were from Jizan (Southern part of Saudi Arabia). All patients had abdominal pain and fever as presenting symptoms. All were misdiagnosed initially: two as appendicitis with appendicular mass; two as abdominal tuberculosis; and two as lymphoma. All patients had leukocytosis with significant eosinophilia and high erythrocyte sedimentation rate (ESR). Liver function tests were initially normal in all patients (first case had impaired liver function tests in late stage). Immunologic evaluation that included quantitative serum immunoglobulins, nitroblue tetrazolium (NBT) test and leukocyte markers was normal, and serology for HIV was negative. Abdominal CT scan findings revealed gastrointestinal tract masses that in most cases involved adjacent organs. Three patients (Cases 1, 2 and 3) underwent partial surgical resection of fungal masses, followed by itraconozole therapy. *B. ranarum* was isolated in four patients, and histopathologic findings were consistent with features of Entomophthorales infection. They responded well to itraconozole and became asymptomatic. Leukocytosis, eosinophilia and high ESR resolved after surgical resection of infected bowel and treatment with antifungal therapy. One patient (Case 4) had no surgery performed and was treated medically. He responded after prolonged hospitalization (>4 months). The two patients who died (Cases 5 and 6) presented at a young age (2½ and 3 years) and were diagnosed 6 months after initial presentation. Both had disseminated inoperable fungal masses.

Case 1. In April 2000 a previously healthy 12-year-old Saudi boy from Jizan presented to a local hospital with abdominal pain and intermittent fever of 2 months duration. Examination revealed a febrile child with a temperature of 38.8°C. He was thin and chronically ill looking. He had a tender right upper quadrant mass extending 12 cm below the right costal margin and right scrotal swelling.

Laboratory data as follows: White blood cell count (WBC) of 16 000/mm^3 with 68% polymorphonuclear leukocyte and 15% eosinophils (absolute eosinophil count, 2400/mm^3); hemoglobin 7.9 g/dl; platelets 556 000/mm^3; ESR 76 mm/h; liver function tests, normal; NBT test, quantitative immunoglobulins and leukocyte markers normal; HIV test nonreactive. An abdominal computerized tomography (CT) scan showed a large right upper quadrant mass with involvement of the mesentery and the liver. The right kidney was swollen and showed diminished contrast enhancement and mild hydronephrosis (Fig. 1). Urine for fungal culture was negative. The patient underwent laparotomy at which a large firm mass arising from the ileocecal and right colon region was found. The mass infiltrated the anterior abdominal wall and extended retroperitoneally to the right kidney. The mass was partially resected with right hemicolectomy done with ileocolic anastomosis.

Histopathology showed granulomatous inflammation with eosinophilia and fungal elements with Splendore-Hoeppli phenomenon. Culture grew *B. ranarum*. The patient did well postoperatively and was treated with itraconazole. He was readmitted 6 months later with poor compliance of medication and relapse of disease involving mainly the porta hepatis. Biliary obstruction was manifested by fever, abdominal pain and jaundice. Alanine transaminase was 203 IU/l, aspartate transaminase 937 IU/l, gamma-glutamyltransferase 197 IU/l, total bilirubin 179 μmol/l and direct bilirubin 135 μmol/l. He underwent stenting of the biliary duct through endoscopic retrograde cholangiopancreatography. Subsequently liver enzymes and bilirubin normalized. He was treated with oral itraconozole for >2 years, and he is asymptomatic.

Case 2. In June 2001, a 12-year-old Saudi boy from Jizan presented with a 3-month history of abdominal pain and intermittent fever. He was hospitalized and operated on for the possibility of acute appendicitis. He had extensive lesions involving the terminal ileum, cecum and ascending colon. A large portion of the intestine was resected, with end to end anastomosis. Histopathology from the resected bowel showed fungal hyphae; therefore he was treated with amphotericin B and then referred to this hospital for further management.

Physical examination revealed a thin patient with hepatomegaly but no splenomegaly. Laboratory data as follows: WBC 11 000/mm^3 with 44% neutrophils, 26% lymphocytes and 20% eosinophils, (2200); hemoglobin 9.8 g/dl, platelets 512 000/mm^3; ESR 114 mm/h. Liver function tests were normal. Immunologic workup including NBT test, quantitative serum immunoglobulins lymphocyte markers (enumeration tests) and lymphocyte blastogenesis were normal, and an HIV test was nonreactive. CT of the abdomen showed a mass involving the right liver lobe. FNA biopsy from the liver revealed fungal elements and tissue eosinophilia with a Splendore-Hoeppli phenomenon. He was treated with oral itraconazole for 1 year with clinical and radiologic improvement.

Case 3. In October 2001 a 9-year-old Saudi boy from Jizan was seen initially at a local hospital with a history of intermittent fever and abdominal pain for 2 months. He was operated on for the possibility of acute appendicitis with appendicular mass and had an abdominal mass involving the intestine and enlargement of surrounding lymph nodes. The patient was referred to Security Forces Hospital, Riyadh, Saudi Arabia for further evaluation.

He was febrile and underweight with severe right iliac fossa tenderness. Laboratory data were as follows: WBC was 17,800/mm^3 with 56% polymorphonuclear

TABLE 1. Clinical and laboratory features of six patients with GIB

Patient	Age	Sex	Date	Method of Diagnosis	Clinical Presentation	WBC Count × 10^3 Cells/ml	% Eosinophils	ESR (mm/h)	Immunological Workup	Radiologic Findings	Surgical Procedures	Treatment	Follow-up
1	12	M	May 2000	Culture + Histopath +	Abdominal pain, fever × 2/12	16	15	76	Normal	Huge abdominal mass occupying the right upper part of the abdomen	Right hemicolectomy	Ampho-B	Asymptomatic
					Abdominal distension and right sided scrotal swelling × 1/12					Liver lesions	Billary duct stenting	Itraconazole	Itraconazole po
					Started on anti-TB treatment						Retroperitoneal with right kidney involvement		
2	12	M	June 2001	Histopath +	Abdominal pain, fever C3/12	16	20.6	114	Normal	Right liver lobe mass	Resection of terminal ileum, cecum and ascending colon	Ampho-B	Asymptomatic
					Operated as appendicitis					Right kidney mass		Itraconazole	Itraconazole po
													Resolved renal and liver lesions
3	12	M	Oct 2001	Culture + Histopath +	Abdominal pain, fever × 2/12	17	19.7	85	Normal	Right iliac fossa mass engulfing cecum with surrounding lymphadenopathy liver mass	Right hemlcolectomy	Ampho-B	Asymptomatic
					Operated as appendicitis with appendicular mass						Retroperitoneal extension	Itraconazole	Itraconazole
4	4	M	March 2002	Culture + Histopath +	Fever, abdominal pain × 1/12	30	30	138	Normal	Hepatomegaly	FNA liver lesion	Ampho-B	Asymptomatic
					Massive hepatomegaly					Hepatic lesion with an area of necrosis		Itraconazole	Itraconazole po
5	3	M	August 2001	Culture + Histopath +	Abdominal pain, fever × 3/12	24	18	105	Normal	Liver mass	FNA liver lesion	Ampho-B	Died within 2 days of diagnosis
					Abdominal distension, weight loss × 2/12					Extensive lesions involving liver, spleen, gallbladder, mesentery		5FC	
					Anti-TB treatment							Ambisome	
										Llsser sac compressing IVC Right kidney not seen			
6	7	M	August 2000	Histopath +	Abdominal pain, distension, fever, vomitting, jaundice and weight loss	16.9	17	96	Normal	Liver lesion	Laparotomy → extensive highly vascular inoperable lesions	Ampho-B	Died at referred hospital with massive GI bleeding
					Hepatosplenomegaly GI bleeding				Hepatosplenomegaly	Endoscopy → eosophageal varices + duodenobiliary fistula	Itraconazole		
									Retroperitoneal mass				

Culture +, culture-positive; Histopath +, histopathology-positive; TB, tuberculosis, Ampho-B, amphotericin B, 5FC, flucytosine; po, orally; IVC, inferior vena cava; GI, gastrointestinal.

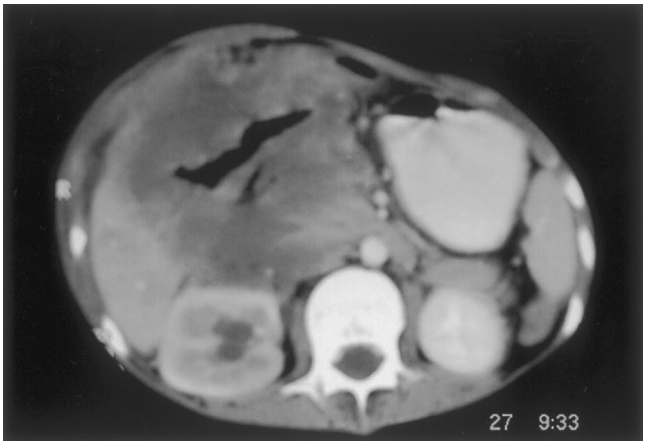

FIG. 1. Contrast-enhanced CT scan. Irregular hypodense right upper quadrant mass enveloping bowel with involvement of mesentery, liver and anterior abdominal wall. Right kidney showed mild hydronephrosis, some swelling and diminished contrast enhancement.

leukocyte, 19.8% eosinophils (AEC 3524) and ESR 85 mm/hr. Immunologic work-up including quantitative serum immunoglobulins, NBT test, lymphocytes markers and lymphocyte blastogenesis was normal, and an HIV test was non reactive. Abdominal CT showed right iliac fossa mass engulfing the cecum with enlarged lymph nodes surrounding this mass. It also showed a nonenhancing liver mass. Hemicolectomy with excision of the retroperitoneal mass and removal of extrahepatic masses was done. Histopathology showed necrotizing granulomatous inflammation with profuse tissue eosinophilia and fungal elements. The fungal culture grew *B. ranarum*.

The patient had a good response to itraconazole and was discharged in good condition. During follow-up, he showed continuous clinical and radiologic improvement. He is currently receiving itraconazole.

Case 4. On March 2002, a 4-year-old Saudi boy was referred to King Khalid University Hospital, Riyadh, Saudi Arabia with a 1-month history of fever and abdominal pain and was found to have massive hepatomegaly.

Laboratory data were as follows: complete blood count showed WBC 30 000/μl with 30% eosinophils, absolute eosinophil count of 9000 and ESR 138 mm/h. Liver function tests were normal. The immunologic workup including quantitative serum immunoglobulin, NBT test, lymphocyte markers and lymphocyte blastogenesis was normal, and an HIV test was nonreactive. An abdominal CT scan showed an enlarged liver with liver lesions and an area of necrosis. Liver biopsy was consistent histopathologically with basidiobolomycosis, and culture was positive for *B. ranarum*. The patient was treated with amphotericin B (1 mg/kg/day), but he did not respond given that he had expanding hepatomegaly and lung and intestinal dissemination. Amphotericin B was therefore replaced with intravenous itraconazole with prompt improvement. This patient was not operated on, but his hospitalization was prolonged for more than 4 months. The patient was discharged receiving oral itraconazole with continued clinical and radiologic improvement. He is currently asymptomatic and taking oral itraconazole.

Case 5. In May 2001, a 3-year-old Saudi boy who was living in Jeddah, Saudi Arabia, was seen at a private hospital with a 2-month history of abdominal pain and fever. An abdominal CT scan showed a mass 4 by 4 by 3.6 cm anterior to the main portal vein. WBC was 24 500/mm^3 with 18% eosinophils (4410) and ESR 105 mm/h. FNA from the mass was negative for bacteria by Gram stain and culture. The patient was treated with cefotaxime, amoxicillin/clavulanate and metronidazole. He left the hospital against medical advice. He was readmitted in July 2001 to a different private hospital with fever, vomiting, abdominal pain and progressive abdominal distension. He was ill-looking and emaciated with abdominal distension, hepatomegaly and ascites. The radiologic studies showed a liver lesion, and an FNA from the lesion was again negative for bacteria by Gram stain and culture. The patient was treated with antituberculous medications. His clinical condition deteriorated, and he was referred to our hospital for further management. An abdominal CT scan again showed a liver mass. A biopsy specimen showed necrotic material suggestive of fungal infection. Histopathology showed septate hyphae, tissue eosinophilia and Splendore-Hoepplie phenomenon. Fungal culture grew *Basidiobolus ranarum.* He had a normal basic immunologic workup. The extensive disease and critical condition did not allow for surgical resection. The patient was treated with amphotericin B and then liposomal amphotericin B and flucytosine, but he developed multiorgan failure and died within 2 days of admission before histopathology and culture results were known.

Case 6. A 7-year-old boy from Jizan was evaluated initially at the age of 30 months because of fever, vomiting, abdominal pain, progressive abdominal distension and hepatosplenomegaly. He was admitted for the possibility of lymphoma. Liver biopsy showed septated fungal hyphae, tissue eosinophilia and Splendore-Hoepplie phenomenon. He was treated with amphotericin B and then itraconazole with initial improvement during the year of therapy. However, the patient missed follow-up at a local hospital and was referred to this hospital in August 2000 at age of 7 years with fever, abdominal pain and progressive abdominal distension. Complete blood count showed WBC 16 900/mm^3 with 17% eosinophils (2873). ESR was 96 mm/h, and immunologic workup was normal. Abdominal CT scan showed extensive masses involving most of the abdominal organs. Upper gastrointestinal

study showed a duodenobiliary fistula. Endoscopy showed esophageal varices and a duodenobiliary fistula. Laparotomy showed unresectable extensive intra-abdominal masses, which were highly vascular with diffuse adhesions. The patient died at a local hospital with massive gastrointestinal bleeding despite treatment with intravenous itraconazole for 2 weeks.

Histopathologic findings. Histopathologic material was available for all patients. Low power examination showed extensive necrotizing inflammation. Cellular areas composed of numerous clusters of eosinophils and some neutrophils were interspersed with paucicellular zones of necrosis. A prominent feature was the presence of empty spaces within the inflammatory infiltrate, the outlines of which were those of fungal hyphae. These spaces were found mostly in areas of necrosis but were also present in zones of intense inflammation. At first glance the spaces were thought to represent small blood vessels, but on closer inspection they were devoid of an endothelial lining. They stained positively with GMS stain and showed stout, septate branching hyphae having somewhat variable diameter (Fig. 2). Degenerating and partially necrotic fungal hyphae could be identified on hematoxylin and eosin, stain and some of these had a peripheral halo of amorphous eosinophilic material (the Splendore-Hoeppli phenomenon). (Fig. 3) Peripherally located Langerhans and foreign body-type giant cells were present; in some of these it was possible to identify degenerating fungal hyphae.

DISCUSSION

Entomophthoromycosis is a rare form of zygomycosis. Two principal species responsible for the majority of these infections are *Conidiobolus coronatus* and *B. ranarum*.[10, 11] They have been known to cause skin and soft tissue infections in otherwise healthy individuals in tropical and subtropical areas of Africa, South America and Asia. Visceral involvement is unusual and thus far has been reported only in association with *Basidiobolus*. *B. ranarum* was first isolated in 1955 from decaying plants in the United States and subsequently has been found in soil and vegetations throughout the world.[12] *B. ranarum* is sometimes present as a commensal in the intestinal tracts of frogs, toads, turtles, chameleons, horses and dogs.[13, 14] The first recognized human case of infection caused by *B. ranarum* was one of subcutaneous mycosis reported in 1956 in Indonesia,[15] and other cases subsequently occurred in India,[16] Africa[17] and South America.[6] In 1978 the first culture-proved case of invasive basidiobolomycosis of the maxillary sinus and the palate was reported in the United States.[18]

Because of nonspecific presenting signs and symptoms, GIB may masquerade as other clinical entities defying definitive diagnosis and treatment. However, several clinical characteristics should raise suspicion for GIB. Patients in this study had subacute onset of symptoms, usually beginning with abdominal pain and fever, but failed to respond to the initially prescribed therapy. Most patients were apparently in good health before acquiring the infection. All our patients were misdiagnosed initially either as acute appendicitis with appendicular mass, abdominal tuberculosis or malignancy.

Intraabdominal masses were found on imaging studies, and during surgical exploration these masses were suggestive of either malignancy or inflammatory lesions, but histopathologic tests revealed inflammatory changes with marked tissue eosinophilia. All patients had elevated WBC counts with eosinophilia, which had been associated with systemic mycosis such as coccidioidomycosis,[19] but rarely with gastrointestinal malignancies.

In this series, which represents the largest reported series in pediatric GIB, the small intestine and liver were involved in all six patients followed by the colon in five and the kidneys in three. One patient had biliary obstruction caused by extension of the disease and

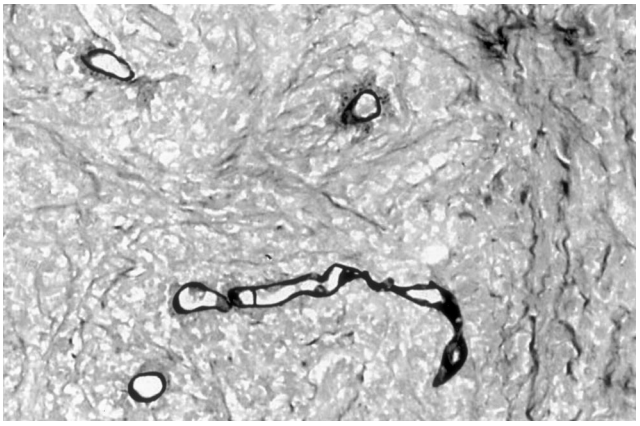

FIG. 2. Silver stain showing branching septate hyphae of variable but generally wide diameter.

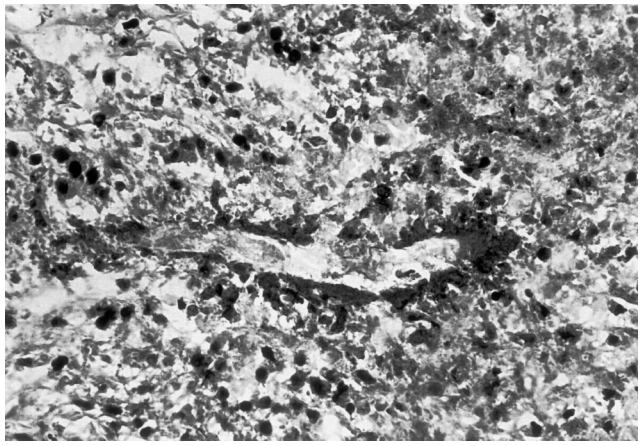

FIG. 3. Degenerating fungal hypha ringed by eosinophilic deposits (Splendore-Hoeppli phenomenon).

required biliary duct stenting. Two patients had extensive involvement of most of the intraabdominal organs and were inoperable; both died from their disease. One of these two patients developed portal hypertension, esophageal varices and duodenobiliary fistula and died with massive gastrointestinal bleeding.

B. ranarum can be isolated from surgical specimens[1]; however, media should be inoculated soon after resection because *B. ranarum* does not survive at 4°C.[1] Sabouraud agar is an adequate medium, and visible growth is usually present 3 to 4 days after inoculation at 25–30°C.[1] Colonies appear white or pale gray and have radial folds.[1] Silver stain of surgically resected specimens will show fungal elements which appear as broad, pleomorphic, sparsely septated hyphae, the walls of which stain faintly with fungal preparations (GMS or periodic acid-Schiff stain).[1–8]

On hematoxylin and eosin staining, the fungal elements often appear as empty spaces surrounded by intensely staining eosinophilic material (Splendore-Hoeppli phenomenon).[1, 5, 8] Vascular invasion was not seen in any of these cases. The abundant eosinophilic inflammation in affected tissues helps to distinguish basidiobolomycosis from mucormycosis, which usually is characterized by a neutrophilic infiltrate and vascular invasion with thrombosis.[8] In the cases presented the histopathology and fungal morphology, were characteristic of Entomophthorales infection. Because the four culture-proved cases have histopathologic features similar to those for whom no culture was performed, it is reasonable to assume that all cases represent *basidiobolus* infection. Our patients were immunologically grossly normal and hence did not have obvious risk for a fungal infection. Mortality was high (40%) and was observed in the very young patients who were diagnosed late with dissemination of the infection.

The observation that most of our cases were from the same region, southern part of Saudi Arabia (Jizan), which has warm, humid climate, which may enhance the growth of the fungus, suggest environmental contamination. Although the portal of entry of the fungus into the host is unknown, involvement of the small intestine in all cases may indicate ingestion of the fungus through contaminated soil or vegetables.

Our cases share many features with those previously reported. Three male pediatric GIB were reported before, two from Brazil (4 and 13 years old) and one (6 years old) from Nigeria.[6, 7, 9] All have similar presentation in the form of abdominal pain and fever and similar laboratory, radiologic and surgical findings. All died of the disease (Table 2).

Whether this entity is a new disease in Saudi Arabia or an old one that had been misdiagnosed and missed is unclear at this time.

Appropriate treatment of GIB has not been outlined. Surgical resection of the inflammatory masses seems to help in curing the disease in conjunction with the administration of systemic antifungal agents. The best choice of antifungal agents is not clear, but the use of Itraconazole seems to be reasonable, based on our experience and literature. Treatment failure has been described in association with amphotericin B.[8, 20–23] Potassium iodide has been successful in treatment of subcutaneous basidiobolomycosis,[2, 6, 24] but experience is lacking for invasive disease.

GIB is an emerging infection that causes substantial morbidity and mortality and diagnostic confusion. The current experience of treating patients is limited; however, it seems reasonable to say that surgical resection of the infected tissue and prolonged treatment with itraconozole offer the best chance for curing this disease; diagnosis of GIB requires high index of suspicion. Increased awareness and inclusion of this rare entity in the differential diagnosis in patients with abdominal masses and eosinophilia help reach an early diagnosis and prompt starting treatment. In such presentations obtaining specimens for fungal culture and histopathologic examination with special fungal stains are crucial.

TABLE 2. Summary of presumed or culture-proved cases of pediatric GIB from the literature

Reference Country	Patient's Age (yr)	Sex	Risk Factor	Symptoms and Signs	Site(s) Involved	Diagnostic Method	Therapy	Outcome
Brazil[7]	4	M	None	Abdominal pain, fever, sweats and diarrhea, epigastric mass	Stomach, transverse colon	*Basidiobolus,* random culture, histology	Surgery	DOD 2.5 mo after presentation
Brazil[6]	13	M	None	Abdominal pain, weakness, fever, anorexia, memory loss	Stomach, duodenum, transverse colon, pancreas, liver, biliary system, intestinal obstruction	Histology	None	DOD without therapy, 4 mo after presentation
Nigeria[9]	6	M	None	Skin infection, rectal obstruction	Skin, ileum, transverse colon, rectum, bladder	Histology	Antibiotics, iodide therapy	DOD 1 yr after sc presentation

DOD, died of disease.

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REFERENCES

1. Kwon-Chung KJ, Bennett JE. Medical mycology. Philadelphia: Lea & Febiger, 1992.
2. De Leon-Bojorge B, Ruiz-Maldonado R, Lopez-Martinez R. Subcutaneous phycomycosis caused by *Basidiobolus haptosporus*: a clinicopathologic and mycologic study in a child. Pediatr Dermatol 1988;5:33–6.
3. Antonelli M, Vignetti P, Dahir M, Mohamed MS, Favah AA. Entomophthoromycosis due to *Basidiobolus* in Somalia. Trans R Soc Trop Med Hyg 1987;81:186–7.
4. Vismer HF, de Beer HA, Dreyer L. Subcutaneous phycomycosis caused by *Basidiobolus haptosporus* (Drechsler, 1945). S Afr Med J 1980;58:644–7.
5. Lyon GM, Smilack JD, Komatsu KK, Gastrointestinal basidiobolomycosis in Arizona: clinical and epidemiological characteristics and review of the literature. Clin Infect Dis 2001; 32:1448–55.
6. Bittencourt AL, Londero AT, Araujo MS, Mendonca N, Bastos JL. Occurrence of subcutaneous zygomycosis caused by *Basidiobolus haptosporus* in Brazil. Mycopathologia 1970;68: 101–4.
7. De Aguiar E, Moraes WC, Londero AT. Gastrointestinal entomophthoromycosis caused by *Basidiobolus haptosporus*. Mycopathologia 1980;72:101–5.
8. Yousef OM, Smilack JD, Kerr DM, Ramsey R, Rosati L, Colby TV. Gastrointestinal basidiobolomycosis: morphologic findings in a cluster of six cases. Am J Clin Pathol 1999;112:610–16.
9. Edington GM. Phycomycosis in Ibadan, Western Nigeria: two post-mortem reports. Trans R Soc Trop Med Hyg 1964;58: 242–5.
10. Rinaldi MG. Zygomycosis. Infect Dis Clin North Am 1989;3: 19–41.
11. Espinel-Ingroff A, Oakley LA, Kerkering TM. Opportunistic zygomycotic infections. Mycopathologia 1987;97:33–41.
12. Greer DL, Friedman L. Studies on the genus *Basidiobolus* with recalcification on the species pathogenic to man. Sabouraudia 1966;4:231–41.
13. Okafor JI, Te Strake D, Mushinsky HR, Yango BG. A *Basidiobolus* species and its association with reptiles and amphibians in Southern Florida. Sabouradia 1984;22:47–51.
14. Zahari P, Hirst RG, Shipton WA, Campbell RS. The origin and pathogenicity of *Basidiobolus* species in Northern Australia. J Med Vet Mycol 1990;28:461–8.
15. Joe L, Eng NIT, Pohan A, Van Der Muelen H, Emmons CW. *Basidiobolus* ranarum as a cause of subcutaneous mycosis in Indonesia. Arch Dermatol 1956;74:378–83.
16. Dasgupta LR, Agarwal SC, Varma RA, Bedi BMS, Chatterjee H. Subcutaneous phycomycosis from Pondicherry, South India. Sabouraudia 1976;14:123–7.
17. Mugerwa JW. Subcutaneous phycomycosis in Uganda. Br J Dermatol 1976;94:539–44.
18. Dworzack DL, Pollok AS, Hodges GR, Barnes WG, Ajello L. Zygomycosis of the maxillary sinus and palate caused by *Basidiobolus haptosporus*. Arch Intern Med 1978;138: 1274–6.
19. Wardlaw AJ, Kay AB. Eosinopenia and eosinophilia. In: Beutler E, Lichtman MA, Coller BS, Kipps TJ, eds. Williams hematology. 5th ed. New York: McGraw-Hill, 1995:844–52.
20. Khan ZU, Prakash B, Kapoor MM, Madda JP, Chandy R. Basidiobolomycosis of the rectum masquerading as Crohn's disease: case report and review. Clin Infect Dis 1998;26: 521–2.
21. Schmidt JH, Howard RJ, Chen JL, Pierson KK. First culture-proven gastrointestinal entomophthoromycosis in the United States: a case report and review of the literature. Mycopathologia 1986;95:101–4.
22. Zavasky D, Loftus T, Segal H, Carroll K. Gastrointestinal zygomatic infection caused by *Basidiobolus ranarum*: case report and review. Clin Infect Dis 1999;28:1244–8.
23. Guaro J, Aguilar C, Pujol I. *In-vitro* anti-fungal susceptibilities of *Basidiobolus* and *Conidiobolus* spp. strains. J Antimicrob Chemother 1999;44:557–60.
24. Bittencourt AL, Araujo MG, Fontoura MS. Occurrence of subcutaneous zygomycosis (entomophthoromycosis basidiobolae) caused by *Basidiobolus haptosporus* with pulmonary involvement. Mycopathologia 1980;71:155–8.