Pediatric Gastrointestinal Basidiobolomycosis: A Retrospective Review of Three Cases from Oman

Maather Al Naeemi[1*], Nidaa Al Naamany[2] and Hilal Al Hashami[3]

[1]Medical Student At Sultan Qaboos University, Muscat, Sultanate of Oman

[2]General Pediatrics Specialist, Al Nahda Hospital, Muscat, Sultanate of Oman

[3]Consultant Pediatrics Infectious Diseases, Royal Hospital, Muscat, Sultanate of Oman

Received: 04 March 2025

Accepted: 17 June 2025

*Corresponding author: misn2882000@gmail.com

DOI 10.5001/omj.2028.30

Abstract

Introduction: Basidiobolomycosis is a rare fungal infection, which primarily affects immunocompetent individuals. Infection with this fungus can lead to infections of the skin and subcutaneous tissues. Gastrointestinal basidiobolomycosis (GIB) infection is rare, and difficult to diagnosis as it mimics the clinical presentation of other illnesses such as neoplasia or inflammatory bowel disease. **Methods:** This retrospective chart review case series presents the clinical presentation, investigations, management, and outcome of three pediatric patients. **Results:** All patients presented with an abdominal mass that was either initially diagnosed clinically or radiologically. The initial working diagnosis was malignancy for two patients, the third was suspected to have inflammatory bowel disease. The diagnosis of GIB was confirmed by biopsy in two cases, and from radiological findings and high eosinophil count in the peripheral blood in the third. All three patients responded well to the voriconazole treatment. One of the cases underwent surgical resection of the gastrointestinal mass. **Conclusion:** A diagnosis of GIB should be considered in cases where eosinophilia, elevated inflammatory markers, and gastrointestinal symptoms are present, especially those who reside in mountainous and farm areas with possible exposure to basidiobolomycosis.

Keywords: Basidiobolomycosis, Gastrointestinal infection, children

Introduction

The pathogenic fungus *Basidiobolus Ranarum* is be found in amphibian droppings, soil, reptiles, and insects in tropical and subtropical areas. When the fungus enters the body via contact with open skin such as an insect bite, it can lead to an uncommon infection called basidiobolomycosis.[1]

The first reported infection with *Basidiobolus Ranarum* was described in 1956 as subcutaneous swelling in an Indonesian boy.[2] Extracutaneous infections—namely gastrointestinal basidiobolomycosis (GIB)—have been reported to occur at a higher rate in adults than children, with the largest populations of reported cases occurring in the Kingdom of Saudi Arabia.[3] The global prevalence of this condition and mortality rate are not well documented.

Gastrointestinal infection is rare and presents similarly to other gastrointestinal conditions such as inflammatory bowel disease (IBD) and neoplasms, thus making diagnosis and treatment challenging prior to obtaining a biopsy.[3,4] Most reported cases initially present with abdominal pain and fever; however, coexistence of constipation, gastrointestinal bleeding, and diarrhea have also been reported.[5-7] Majority of the reported cases are of normal host with no underlying immunodeficiencies.[3,8,9]

Radiological findings of GIB on ultrasound (US) and computed tomography (CT) scans include intestinal wall thickening and gastric, intestinal, or abdominal masses.[3,10] The preferred method of diagnosis is histopathology and culture, as a previous study involving 32 cases revealed that 50% of cultures returned positive results.[11] The pathological diagnosis was consistent across all cases, with all exhibiting intensely radiating eosinophilic granular material surrounding the fungal elements, Splendore-Hoeppli bodies, and numerous eosinophils.[12]

The present case series presents our retrospective chart review of three cases of GIB admitted to the child health department at the Royal Hospital, Muscat, Sultanate of Oman. Verbal consent was obtained from the parents of the patients to report their clinical cases.

Case Report

Case one

A previously well 1-year-old boy presented to our department in July 2021. ***The child presented with*** fever since 3 days previously, abdominal pain, and distension. He had a 2-day history of vomiting, which resolved upon presentation to our emergency department. He was admitted to a regional hospital where he received intravenous ceftriaxone, and abdominal Ultrasound (US) revealed possible intussusception. The parents signed a Leave Against Medical Advice form and presented to our hospital's emergency department. ***the childs past medical history was*** unremarkable.

Physical assessment revealed tachycardia (heart rate of 170/min), fever of 38.9°C, and dehydration. Two boluses of intravenous fluids were administered. Abdominal examination revealed distended abdomen and mild tenderness with no organomegaly or masses.

Initial laboratory investigations revealed hemoglobin (Hb): 9.4 g/dL, white blood cell count (WBC): 26 x 10^9 g/L, neutrophils: 14 x 10^9 g/L, eosinophils: 3 x 10^9 g/L, and C-reactive protein (CRP): 298 mg/L.

Abdominal US was repeated on admission, which revealed evidence of intussusception in the left upper quadrant area with minimal free fluid. Further evaluation with CT was performed. Large circumferential heterogenous wall thickening/mass of the proximal transverse colon involving part of the hepatic flexure was observed, giving the appearance of aneurysmal dilatation with no evidence of obstruction. Findings were considered to likely be related to infectious/inflammatory/neoplastic etiology.

Triple antibiotics were started on admission following the results of examinations; intravenous ampicillin 25mg/kg every 6 hours, gentamicin 2.5mg/kg every 8 hours, and oral metronidazole 7.5mg/kg every 8 hours. An air enema was performed under fluoroscopy monitoring, confirming the suspected intussusception. The mass was reduced under fluoroscopy monitoring with pressure maintained at <120 mm Hg. Air reflux to the small bowel was noted, no pneumoperitoneum was present. The procedure went smoothly with no immediate complications.

The patient was admitted to the pediatric surgical ward, where abdominal distension persisted at 1-day postadmission, with no palpable mass on examination. Therefore, US was repeated, which showed significant circumferential bowel wall thickening of a bowel loop in the epigastric region. The whole-bowel wall thickening/mass (with bowel loop) measured 6.8 × 3.1 cm and the single- wall thickening measured 2.5 cm. Associated moderate ascites were observed, mainly in the pelvis. There was no definite hepatosplenomegaly. The appearance suggested possible infective/inflammatory/neoplastic etiology. Lymphoma/leukemia could not be ruled out at this stage.

The lesion did not appear amenable for percutaneous biopsy due to the overlying bowel loops; thus, further evaluation with CT was performed. Findings were considered to likely be related to infectious/inflammatory/neoplastic etiology. Histopathology correlation was recommended. The rest of the chest and abdominal CT was normal.

The hematology–oncology team was consulted, and recommended tissue biopsy for histopathological examination. The gastroenterology team was then consulted, biopsy by colonoscopy was deferred after discussion

with surgeons, in order to obtain a deeper biopsy of the lesion if malignancy was suspected. The patient suffered another fever, and so antibiotics were upgraded to piperacillin-tazobactam (Tazocin) 90mg/kg every 8 hours and metronidazole 7.5mg/kg every 8 hours. Surgery was performed 7 days after admission involving laparotomy, resection of the cecum with the appendix and the mass with the ascending and transverse colon up to the distal transverse and primary ileocolonic anastomosis.

The histopathology findings 2 days postoperatively were as follows: Gross findings; A segment of the colon was received with appendix attached and orientated with a stitch in the proximal margin. The colonic segment measured 20.5 cm proximal to distal and 0.6 cm in diameter. The appendix measured 4.2 × 0.5 cm. A mass measuring 11.0 cm proximal to distal and 6.0 cm medial to lateral was identified. Proximal to the mass, the bowel was dilated about 9.0 cm proximal to distal, 3–5 cm in diameter. Distal to the mass, a defect measuring 3.2 × 2.0 cm was noted. On opening, the mass had a heterogeneous, cut surface and was located 9 cm from the proximal margin and 3–5 cm from the distal margin. Background mucosa was unremarkable.

Microscopic findings revealed colonic mucosa with dense transmural mixed inflammation composed predominantly of eosinophils, neutrophils, lymphocytes, and plasma cells involving all layers of the colon wall. Scattered broad septated, thick-walled organisms were identified surrounded by eosinophilic material (Splendore-Hoeppli phenomenon). No dysplasia or malignancy were identified. Excision margins were normal. The background colonic mucosa and appendix exhibited no significant pathology. The histopathological features were thus suggestive of GIB.

The pediatric infectious disease team was consulted, and advised intravenous voriconazole (9 mg/kg twice daily) for the treatment of GIB during admission. Once discharged, the patient was to continue oral voriconazole (9 mg/kg twice daily). The patient was discharged after 16 days of admission in a stable condition with no abdominal pain or fever, to continue with outpatient followup care under the pediatric infectious disease team.

Two months after discharge, laboratory results were as follows: WBC, 26.5 × 109 g/L; eosinophils, 14 × 109 g/L; CRP, 80 mg/L. Abdominal US was reassuring with no recurrence of the lesions, and the patient was gaining weight slowly. The dose of voriconazole was increased to 10 mg/kg twice daily. One month later, CRP and eosinophil levels were normalized, and the patient continued to gain weight normally. The patient completed 9 months of voriconazole treatment, which was stopped prematurely at the decision of the parents due to the development of photosensitivity. He remained asymptomatic on subsequent followup visits.

Case two

A previously healthy 2-year-old boy was referred to our department from the regional hospital (Al Rustaq Hospital). He came in for evaluation of fever reaching 39°C and abdominal pain lasting for 1 month.

Further medical history revealed that he was reviewed at a local outpatient clinic 1 month prior, which reported failure to thrive. His weight was 8.9 kg, and laboratory investigations showed features of anemia. He was readmitted 1 week later after referral from a private clinic following the discovery of an epigastric mass on abdominal US. We performed abdominal CT, which revealed a suspicious epigastric lesion crowded by collapsed small bowel loops, which appeared thick-walled, with no oral contrast or gas detected. These findings were suggestive of an infectious process, and workup on the lesion was advised by the hematology-oncology team, as was magnetic resonance imaging if clinically indicated.

Fever was present inconsistently for 3 weeks after presentation at our department, after which it was contiguous for 1 week. The fever exhibited no specific timings, association with chills or rigors, or abnormal movement, and was partially relieved with paracetamol.

The patient complained of intermittent abdominal pain, which was not radiating and had no specific location, and was relieved by passing gas and stool. No vomiting or abdominal distention were noted, and appetite was normal.

The patient was confirmed as being positive for COVID-19 infection 2 weeks prior to presentation, with full recovery. There were no symptoms of upper respiratory tract infection or other focus of infection (e.g., vomiting/diarrhea, rash, urinary symptoms, ear discharge), and there was no history of travel.

The child past medical history was unremarkable.

Physical examination revealed the child to be active and pale, the abdomen to be soft with a swelling in the umbilical area. The swelling was difficult to describe as the child was crying when examined, and bowel sounds were present.

Laboratory investigations revealed: Hb, 6.06 g/dL; total WBC, 27 × 109 g/L; absolute neutrophils, 13 × 109 g/L; eosinophils, 3.35 × 109 g/L; transferrin saturation, 3.2%; ferritin, 107 ug/L; CRP, 210 mg/L; erythrocyte sedimentation rate (ESR), 105 mm/h; lactate dehydrogenase, 252 iU/L; positive stool occult blood. Peripheral smear was performed twice, revealing eosinophilia, active mild thrombocytosis, anisocytosis, microcytes, hypochromia, and polychromasia. At the time of admission to our institution, laboratory findings were as follows: Hb, 7.8 g/dL; WBC, 21.6 × 109 g/L; neutrophils, 13.9×10^9 g/L; eosinophils, 1.2×10^9 g/L; CRP, 205 mg/L.

Intravenous ceftriaxone 80mg/kg once a day, vancomycin 10mg/kg every 8 hours, and metronidazole 7.5mg/kg every 8 hours had been initiated in the regional hospital as empirical treatment to cover for infectious causes, and was continued due to the continuing fever.

Abdominal US revealed a right subhepatic bowel mass lesion with a heterogeneous echotexture and a small amount of surrounding free fluid with no associated bowel obstruction. The lesion was in close proximity to the gallbladder and liver. No significant intraabdominal lymphadenopathy nor aneurysmal dilation of the bowel were observed. Findings were considered to be suggestive of basidiobolomycosis infection.

After the US findings were considered, a multidisciplinary meeting was held with the general pediatrics team, infectious disease team, hematologists, radiologists, and surgeons. Because of the risk of bowel resection secondary to bowel adhesion, it was decided that surgical biopsy should be performed to avoid major complications.

All antibiotics were ceased, and intravenous voriconazole (9 mg/kg twice daily) was initiated. During admission, the patient required a blood transfusion for a low hemoglobin (6.6 g/dL), after which he improved dramatically. The patient was discharged after 7 days of intravenous voriconazole, to continue with oral voriconazole (9 mg/kg twice daily).

The first outpatient follow up visit a month after admission, revealed the patient to be asymptomatic, have gained weight, and to have an eosinophil count of 0.4 × 109 g/L. Abdominal US showed the previously observed right subhepatic mass to be resolved. At the second outpatient visit, which was 5 months after discharge, he was diagnosed with a pinworm infection, which was treated with albendazole along. His family members were all treated. His parents reported improved appetite with weight gain in the patient. Repeated US abdomen showed complete resolution of the previously seen right subhepatic mass with no new masses detected. There was no evidence of enlarged lymph nodes in the abdomen, and laboratory tests showed CRP of four and eosinophil count of 0.6 × 109 g/L. After completing 1 year of voriconazole treatment, the patient remained asymptomatic and returned to normal activities.

Case three

A 2-year-old male from the Al Sharqiya region of the Sultanate of Oman who lived in an area near farms, presented to the gastroenterology team. He presented with recurrent bloody stool for 5 months. The stool was well-formed, with no mucus, passing 2–3 times/day, with no history of diarrhea or constipation, and was not associated with vomiting or abdominal pain.

Systematic review showed two episodes of mouth ulcers, which had been treated symptomatically. There were no hematuria or urinary symptoms, good urine output, no history of fever or cough, no history of joint pain, and no recent weight loss.

Medical history revealed preterm birth at 33 weeks via elective lower-segment cesarean section, requiring 2 weeks of neonatal intensive care unit admission. A history of mild neonatal hyperbilirubinemia was noted. The patient had a history of asthma, treated with fluticasone and salbutamol inhalers. He was admitted to the regional hospital (Ibri hospital) at the age of 3 months for a chest infection, and had been admitted a month ago in regional hospital with a complaint of bloody stool.

Development was appropriate for age; the child was able to actively walk and communicate. Weight was 11.1 kg at the time of admission. Physical examination revealed an enlarged liver, 4–5 cm, palpable below the costal margin. His labs revealed HB: 8.7 g/dL, WBC: 20×10^9 g/L, Eosinophile 6.6×10^9 g/L

Following review by the gastroenterology team, the patient underwent colonoscopy and liver biopsy to evaluate the bloody stools and hepatomegaly. The endoscopic diagnosis was patchy colitis, possibly indicating very early onset IBD and eosinophilic-related colitis.

Histopathological examination showed features strongly suggestive of GIB. The esophageal biopsy revealed moderate eosinophilia, and the ileal biopsy revealed mild ileitis with eosinophilia. Cecum, sigmoid, and right and left colon biopsies revealed markedly increased eosinophils. Liver biopsy showed notable eosinophilia in the few portal tracts. The Splendore-Hoeppli phenomenon was observed in the transverse colon. The patient was negative for cytomegalovirus, human immunodeficiency viruses, and *Clostridium difficile*. Feces culture were positive for *Shigella sonnei*, for which 5 days of ciprofloxacin was implemented.He was discharged after a 4 days inpatient stay.Itraconazole treatment was initiated (3.5 mg/kg twice daily) based on histopathological findings.

At an outpatient clinic followup around 1 month after discharge, he was passing stool once to twice a day but still had bleeding daily; however, it was significantly better. The abdomen was soft and non-tender. Laboratory tests were as follows: Hb, 9 g/dL; WBC, 31.4 × 109 g/dL; lymphocytes, 13.9 × 109 g/dL. Abdominal US showed mild hepatomegaly and thickening in the rectosigmoid wall surrounding the multiple lymph nodes.

Intermittent abdominal pain persisted after 2 months of itraconazole treatment. After 3 months, itraconazole was changed to voriconazole (9 mg/kg twice daily). After 1 month, symptoms were improved, with no blood in the stool, abdominal pain, fever, or weight gain reported.

Followup colonoscopy was performed 7 months later and advanced to the ileocecal valve. Findings were completely normal. Multiple biopsies were performed, with no significant morphologic abnormalities detected.

Voriconazole was continued for 1.5 years. At this time, eosinophil levels were still high (1.1 × 109 g/L); therefore, treatment was repeated. Colonoscopy and biopsy were planned due to the reoccurrence of bloody stools and high eosinophil count. No other diagnoses were revealed, and voriconazole was planned to continue for 2 years with regular followup. The patient completed 2 years of voriconazole and returned to normal state, with no abdominal pain or bleeding from the rectum.

Find Summary of all three case presentations and treatment on Table 1.

Table 1: Summary of cases.

Case	Age	Sex	Clinical presentation	Duration of the symptoms	Region
1.	1 year	Male	Fever, abdominal pain, and distension	3 days	Al Sharqiya
2.	2 years	Male	Fever and abdominal pain	1 month	Al Rustaq

3.	2 years	Male	Recurrent bloody stool	5 months	Al Sharqiya

Case	Laboratory on presentation				Radiology	Culture
	WBC	Hb	Eosinophils	CRP		
1.	26×10^9 g/L	9.4 g/dL	3 × 109 g/L	298 mg/L	CT abdomen: Large circumferential heterogenous wall thickening/mass of the proximal transverse colon, no evidence of obstruction. Findings were considered to likely be related to infectious/inflammatory/neoplastic etiology	None
2.	27×10^9 g/L	6.06 g/dL	3.35×10^9 g/L	210 mg/L	US abdomen: right subhepatic bowel mass with a heterogeneous echotexture and a small amount of surrounding free fluid no associated bowel obstruction. Findings were considered to be suggestive of basidiobolomycosis infection.	None
3.	20×10^9 g/L	8.7 g/dL	6.6×10^9 g/L	<4 mg/L	Nil	None

Case	Intervention (surgical)	Medical treatment	Histopathology	Complication
1.	Laparotomy, resection of the cecum with the appendix and the mass with the ascending and transverse colon up to the distal transverse and primary ileocolonic anastomosis.	9 months of voriconazole	Colonic mucosa with dense transmural mixed inflammation composed predominantly of eosinophils, neutrophils, lymphocytes, and plasma cells involving all layers of the colon wall. Scattered broad septated, thick-walled organisms were identified surrounded by eosinophilic material (Splendore-Hoeppli phenomenon). No dysplasia or malignancy were identified.	-Intussusception -Photosensitivity secondary to voriconazole
2.	Nill	1 year of voriconazole	-	-
3.	Nil	2 years of voriconazole	Esophageal biopsy: moderate eosinophilia, and the ileal biopsy: mild ileitis with eosinophilia.	Lower GI bleed

	Cecum, sigmoid, and right and left colon biopsies: markedly increased eosinophils. Liver biopsy: notable eosinophilia in the few portal tracts. The Splendore-Hoeppli phenomenon was observed in the transverse colon.

Discussion

It is believed that the primary route of entry of basidiobolomycosis into the gastrointestinal tract is via ingestion of soil or foods contaminated by amphibian or lizard droppings.[1] Infection develops in the submucosal and muscular layers of the bowel, which leads to the formation of granulomatous lesions and inflammatory masses.[8] The majority of individuals who are affected by the fungus are immunocompetent,[9] and cases of GIB are more prevalent in dry areas such as Arizona and the Middle East.[13] This distribution suggests that the saprophyte may be cycling in reptiles; an assumption that is supported by the number of cases originating from the Sultanate of Oman's neighboring country, the Kingdom of Saudi Arabia.[3,14,15]

The cases presented here are all males, which is in line with the majority of reported cases of GIB om the current literature.[3,14,16] One study investigating the risk factors of developing gastrointestinal infection following exposure to the fungus revealed that most affected individuals to had exposure to livestock at home, frogs, or have walked barefoot among organic manure.[14] Furthermore, all participants in the study reported geckos existing in their place of residence, and the majority resided in village or mountain areas.[14] The report also indicated age and gender to be susceptibility factors.

Gastrointestinal complaints are common in pediatric patients, and GIB is an uncommon differential for some of these, making diagnosis challenging in many cases. Furthermore, GIB can masquerade with a range of symptoms, commonly including abdominal pain, palpable abdominal mass, weight loss, and fever.[3] All cases in the present study experienced abdominal pain, and an abdominal mass was detected through either clinical or radiology investigations. Our initial considerations were malignancy and IBD; however, the presence of peripheral eosinophilia led us to suspect gastrointestinal fungal infection. Early diagnosis and treatment are important to improve prognosis, but lack of specific clinical findings often results in misdiagnosis and late treatment. Our study indicates that the presence of peripheral eosinophilia, along with other nonspecific laboratory findings including leukocytosis and elevated ESR, could indicate fungal infection and further investigations should precede with this in mind.[17,18]

The second case of the present report was diagnosed and treated as GIB based on clinical features and radiological findings, and later confirmed by resolution of radiological findings and improvement in clinical features after treatment. However, most reported cases involve nonspecific radiological findings such as inflammatory changes and thickening of the bowel wall, with or without the presence of abscesses.[16,19] Confirming the diagnosis of GIB based solely on clinical and radiological findings is reported to be extremely challenging.[16] This is further complicated by the fact that the organism itself lies deep under the mucosa, often rendering endoscopic biopsies nonrepresentative and, therefore, not diagnostic.[20] A definitive diagnosis of GIB can be made on the finding of positive culture growth[4]; also, characteristic postoperative pathologic findings, along with clinical and laboratory investigations, can be diagnostic,[16] as in the first and third cases of the present study.

None of the cases in the present report required surgical resection. Historically, surgery was the first-line treatment for GIB; however, a preference for medical treatment has arisen due to the risk of complications.[6,14] However, early diagnosis and prompt treatment are still crucial, if surgical intervention is to be avoided, and complications of GIB itself—including GI obstruction—remain a risk. Several studies have indicated voriconazole to be particularly useful for the treatment of pediatric patients with GIB[14,15]; indeed, clinical and radiological improvements were observed in three of our patients in response to this treatment. Itraconazole is historically the most commonly used antifungal for GIB[21]; however, voriconazole has been shown to be a safer and more effective option in multiple studies, including the present.[14,15,22] There is currently no consensus on the duration of treatment of GIB,[14,15,21-23] and so duration is usually tailored to patient response.

Conclusion

Gastrointestinal basidiobolomycosis is an emerging fungal infection in the Gulf region. The diagnosis of GIB should be kept in mind as a differential diagnosis for any child presenting with prolonged fever, abdominal pain, and abdominal mass. More specific indicators include high peripheral eosinophilia and raised inflammatory markers.

Disclosure

Consent was taken verbally from the patients' parents and it was approved by the research committee of the hospital.

Acknowledgements

We would like to acknowledge the patients and their parents for their participation.

References

<jrn>1. Vilela R, Mendoza L. Human Pathogenic Entomophthorales. Clin Microbiol Rev 2018 Aug;31(4):31. PMID: 30158298 DOI: 10.1128/CMR.00014-18. </jrn>

<jrn>2. Kian Joe L, Pohan A, Tjoei Eng NI, Van Der Meulen H. Basidiobolus ranarum as a cause of subcutaneous mycosis in Indonesia. AMA Arch Derm 1956 Oct;74(4):378-383. PMID: 13361511 DOI: 10.1001/archderm.1956.01550100046008. </jrn>

<jrn>3. El-Shabrawi MH, Kamal NM, Kaerger K, Voigt K. Diagnosis of gastrointestinal basidiobolomycosis: a mini-review. Mycoses 2014 Dec;57(Suppl 3):138-143. PMID: 25186791 DOI: 10.1111/myc.12231. </jrn>

<jrn>4. Mendoza L, Vilela R, Voelz K, Ibrahim AS, Voigt K, Lee SC. Human Fungal Pathogens of Mucorales and Entomophthorales. Cold Spring Harb Perspect Med 2014 Nov;5(4):5. PMID: 25377138 DOI: 10.1101/cshperspect.a019562.</jrn>

<jrn>5. Rabie ME, El Hakeem I, Al-Shraim M, Al Skini MS, Jamil S. Basidiobolomycosis of the colon masquerading as stenotic colon cancer. Case Rep Surg 2011;2011:685460. PMID: 22606589 DOI: 10.1155/2011/685460. </jrn>

<jrn>6. Mandhan P, Hassan KO, Samaan SM, Ali MJ. Visceral basidiobolomycosis: An overlooked infection in immunocompetent children. Afr J Paediatr Surg 2015;12(3):193-196. DOI: 10.4103/0189-6725.170218. </jrn>

<jrn>7. Saadah OI, Farouq MF, Daajani NA, Kamal JS, Ghanem AT. Gastrointestinal basidiobolomycosis in a child; an unusual fungal infection mimicking fistulising Crohn's disease. J Crohns Colitis 2012 Apr;6(3):368-372. PMID: 22405176 DOI: 10.1016/j.crohns.2011.10.008. </jrn>

<jrn>8. Hussein MR, Musalam AO, Assiry MH, Eid RA, El Motawa AM, Gamel AM. Histological and ultrastructural features of gastrointestinal basidiobolomycosis. Mycol Res 2007 Aug;111(Pt 8):926-930. .</jrn>

<jrn>9. Geramizadeh B, Foroughi R, Keshtkar-Jahromi M, Malek-Hosseini SA, Alborzi A. Gastrointestinal basidiobolomycosis, an emerging infection in the immunocompetent host: a report of 14 patients. J Med Microbiol 2012 Dec;61(Pt 12):1770-1774. .</jrn>

<jrn>10. 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 Feb;26(2):521-523. PMID: 9502495 DOI: 10.1086/517107. </jrn>

<jrn>11. Shetty S, Mambatta AK, Penmatsa KR, Venkatakrishnan L. Ileal mucormycosis: a rare cause of lower gastrointestinal bleeding. Ann Gastroenterol 2014;27(2):169. PMID: 24733229.</jrn>

<jrn>12. Wasim Yusuf N, Assaf HM, Rotowa NA. Invasive gastrointestinal Basidiobolus ranarum infection in an immunocompetent child. Pediatr Infect Dis J 2003 Mar;22(3):281-282. PMID: 12664879. </jrn>

<jrn>13. 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 Nov;112(5):610-616. PMID: 10549247 DOI: 10.1093/ajcp/112.5.610. </jrn>

<jrn>14. Ghazwani SM, Arishi HM, Dhayhi NS, Shami MO, Gosadi IM, Rajab M, et al. Pediatric Gastrointestinal Basidiobolomycosis: A Retrospective Study from Jazan Province, Saudi Arabia. Infect Drug Resist 2023 Jul;16:4667-4676. PMID: 37484908 DOI: 10.2147/IDR.S416213. </jrn>

<jrn>15. Al Jarie A, Al Azraki T, Al Mohsen I, Al Jumaah S, Almutawa A, Mohd Fahim Y, et al. Basidiobolomycosis: Case series. J Mycol Med 2011 Mar;21(1):37-45. PMID: 24451502 DOI: 10.1016/j.mycmed.2010.11.002. </jrn>

<jrn>16. Geramizadeh B, Heidari M, Shekarkhar G. Gastrointestinal Basidiobolomycosis, a Rare and Under-diagnosed Fungal Infection in Immunocompetent Hosts: A Review Article. Iran J Med Sci 2015 Mar;40(2):90-97. PMID: 25821287.</jrn>

<jrn>17. Lyon GM, Smilack JD, Komatsu KK, Pasha TM, Leighton JA, Guarner J, et al. Gastrointestinal basidiobolomycosis in Arizona: clinical and epidemiological characteristics and review of the literature. Clin Infect Dis 2001 May;32(10):1448-1455. .</jrn>

<jrn>18. Meeralam Y, Basfar AM, Alzanbagi A, Tashkandi A, Al Harthi W, Saba F, et al. Gastrointestinal Basidiobolomycosis: A Case Series. Cureus 2024 Feb;16(2):e55008. PMID: 38414515 DOI: 10.7759/cureus.55008.</jrn>

<jrn>19. Flicek KT, Vikram HR, De Petris GD, Johnson CD. Abdominal imaging findings in gastrointestinal basidiobolomycosis. Abdom Imaging 2015 Feb;40(2):246-250. PMID: 25099255 DOI: 10.1007/s00261-014-0212-z. </jrn>

<jrn>20. Al Jarie A, Al-Mohsen I, Al Jumaah S, Al Hazmi M, Al Zamil F, Al Zahrani M, et al. Pediatric gastrointestinal basidiobolomycosis. Pediatr Infect Dis J 2003 Nov;22(11):1007-1014. PMID: 14614376 DOI: 10.1097/01.inf.0000095166.94823.11. </jrn>

<jrn>21. Almoosa Z, Alsuhaibani M, AlDandan S, Alshahrani D. Pediatric gastrointestinal basidiobolomycosis mimicking malignancy. Med Mycol Case Rep 2017 Aug;18:31-33. PMID: 28970974 DOI: 10.1016/j.mmcr.2017.08.002. </jrn>

<jrn>22. Al Harthy F, Al Kalbani O, Al Masqri M, Al Dhuhli A, Al Said K. Gastrointestinal Basidiobolomycosis in a Child: Unusual Fungal Infection Mimicking Eosinophilic Gastrointestinal Diseases-A Case Report and Review of the Literature. JPGN Rep 2021 Jul;2(3):e109. PMID: 37205942 DOI: 10.1097/PG9.0000000000000109. </jrn>

<jrn>23. Paredes-Sabja D, Sarker MR. Host serum factor triggers germination of Clostridium perfringens spores lacking the cortex hydrolysis machinery. J Med Microbiol 2011 Dec;60(Pt 12):1734-1741. PMID: 21799201 DOI: 10.1099/jmm.0.031575-0. </jrn>