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CASE REPORT

A RARE CASE OF FATAL GASTROINTESTINAL MUCORMYCOSIS IN A TODDLER DIAGNOSED AT MEDICO-LEGAL AUTOPSY: A CASE REPORT

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ABSTRACT

Introduction: Gastrointestinal (GI) mucormycosis is a rare, rapidly progressive, and life-threatening angio-invasive fungal infection caused by fungi of the order *Mucorales*. It predominantly affects immunocompromised individuals and is associated with high mortality, particularly in infants and neonates. Owing to its nonspecific clinical presentation, diagnosis is frequently delayed or established post-mortem.

Case Presentation: We report the case of an 18-month-old toddler with a history of marginal prematurity, intrauterine growth restriction, malnutrition, and iron-deficiency anaemia who presented with abdominal distension, vomiting, and diarrhoea. The child was on a balanced diet with oral iron supplementation. He was initially managed for acute viral gastroenteritis; however, his clinical condition rapidly deteriorated, with the development of blood-stained stools and subsequent sudden cardiac arrest. Resuscitation was unsuccessful. A medico-legal autopsy revealed inflamed bowel loops and multiple gastric ulcers without perforation. Histopathological examination of the stomach demonstrated ulcerated mucosa with extensive necro-inflammatory debris and numerous broad, aseptate, ribbon-like fungal hyphae with angio-invasion. Grocott methenamine silver staining confirmed features consistent with GI mucormycosis.

Conclusion: This case highlights GI mucormycosis as a rare but fatal cause of acute gastrointestinal illness in children, particularly in the presence of predisposing factors such as malnutrition and iron deficiency anaemia on top of marginal prematurity. The nonspecific clinical features may mimic common gastrointestinal conditions, leading to missed or delayed diagnosis. Prompt and appropriate medical treatment, together with early histopathological confirmation in cases of presumptive or suspected mucormycosis, is essential to facilitate timely intervention for optimal outcomes.

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INTRODUCTION

Gastrointestinal (GI) mucormycosis is a rare, life-threatening, and rapidly progressive angio-invasive fungal infection caused predominantly by fungi of the order *Mucorales*. Based on the organ system involved, mucormycosis is classified into pulmonary, cutaneous,

gastrointestinal, central nervous system, and disseminated forms[1]. GI mucormycosis is considerably less common than other clinical forms, accounting for approximately 5–15% of reported cases[2]. Ingestion of contaminated food is considered the source of primary infection, and the stomach, small intestine, and ileum are the most commonly affected sites[3]. The infected patients typically present with abdominal pain, abdominal distension, and bloody diarrhoea, and may be complicated by ulceration and perforation.

Immunosuppression is a major predisposing factor for GI mucormycosis in all age groups[4]. In paediatric patients, particularly those with a history of prematurity, autoimmune disorders requiring immunomodulatory therapy, corrective abdominal or congenital cardiac surgeries, or admission to paediatric intensive care units, there is an increased risk of developing this lethal fungal infection[5,6]. Although rare, gastrointestinal mucormycosis has also been reported in premature neonates, where it may clinically mimic neonatal necrotizing enterocolitis[7]. We report a rare case of gastrointestinal mucormycosis in a toddler with a history of prematurity and malnutrition, which was diagnosed during post-mortem examination.

CASE PRESENTATION

An 18-month-old male toddler was admitted to the hospital with a two-day history of abdominal distension, vomiting, and loose stools. On admission, he was clinically dehydrated and given emergency intravenous fluid resuscitation. According to the history provided by the mother, the child was born with marginal prematurity and intrauterine growth restriction at 34 weeks of gestation following premature rupture of membranes. According to the history and clinical records, his birth weight was 1.85 kg, and he had received intensive care during the first 2 weeks for neonatal sepsis. Since then, his weight gain had been poor and was significantly below the expected weight-for-height percentile. He was under regular follow-up at a general paediatric clinic for malnutrition and iron-deficiency anaemia. The parents were advised regarding a balanced diet, and oral iron supplementation was prescribed.

He has been fed a combination of breast milk, supplemental formula milk, and a mixed diet. The child's meals were prepared by the mother, ensuring proper hygiene. The child was initially managed for acute viral gastroenteritis. However, despite fluid resuscitation and other supportive medical treatment, his clinical condition deteriorated with an increased frequency of bowel movement that subsequently became blood-stained. Laboratory investigations were suggestive of a recent viral infection, with a white blood cell count of 4.2 k/µL and a low haemoglobin level of 9 g/dL. After 36 hours of admission, the child suffered a sudden cardiac arrest, and resuscitation efforts were unsuccessful. A medico-legal autopsy was performed the next day.

Post-mortem examination revealed a thin, pale, and markedly underweight body (weight 6.9 kg, below -3SD at Weight for Age chart) with no external or internal congenital abnormalities. On internal examination, the bowel loops appeared inflamed but showed no evidence of necrosis or perforation. The stomach was inflamed, with congested mucosa and multiple ulcers (Figure 1). Blood-stained stool was present throughout the lower intestine (Figure 2) with inflammation and epithelial necrosis of the intestinal mucosa.

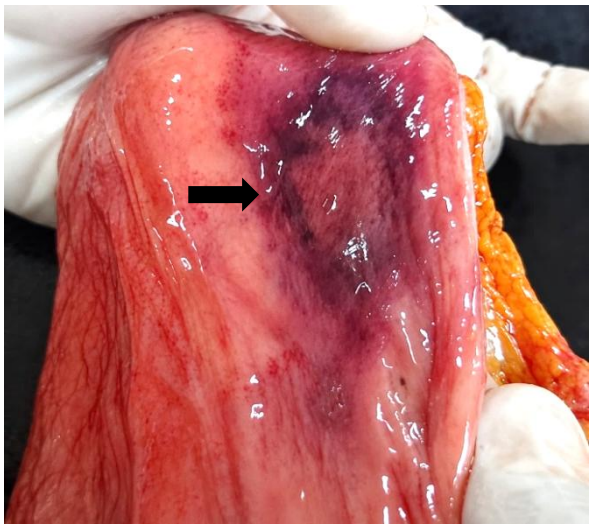

Figure 1: Specimen of the stomach demonstrating a gastric ulcer (arrow) with an inflamed, congested mucosal margin and a pale base.

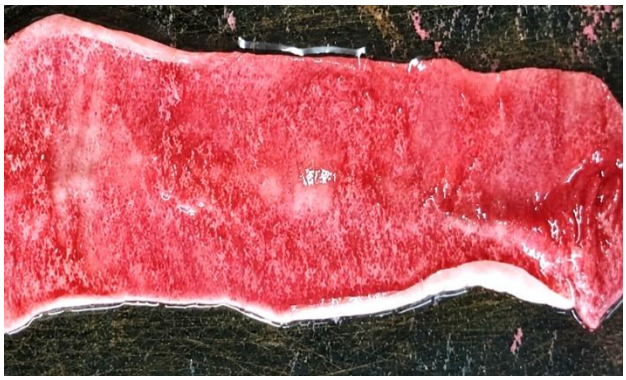

Figure 2: Specimen of the small intestine demonstrating inflamed intestinal mucosa.

Postmortem samples were obtained for toxicology, microbiology, and histopathology studies. The samples were negative for common poisons, and no organisms were isolated. Histopathology of the stomach revealed ulcerated mucosa with necroinflammatory debris (Figure 3) and numerous aseptate broad ribbon-like, irregular branching hyphae at right angles (Figure 4). Multiple foci of angioinvasion were noted in the submucosal vessels (Figure 5). Morphology of the hyphae was compatible with mucormycosis.

The gastrointestinal lesion was stained with Grocott Methenamine Silver (GMS) stain, which demonstrated fungal hyphae stained in black consistent with mucormycosis (Figure 6). Microscopic examination of the liver, lungs, brain, and heart revealed features of multiorgan failure.

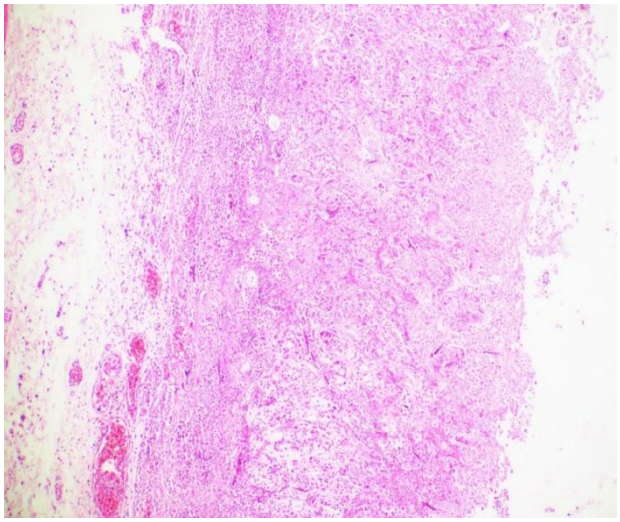

Figure 3: Ulcerated gastric mucosa covered by necroinflammatory debris (H&E, 4x10).

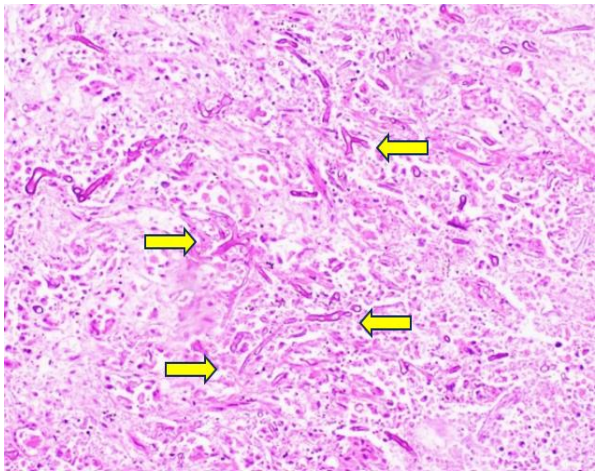

Figure 4: Numerous fungal hyphae (yellow arrows) in the gastric mucosa (H&E, 10x10).

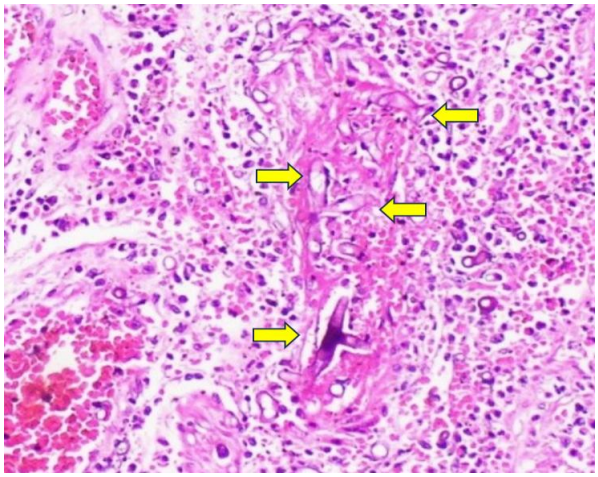

Figure 5: Fungal hyphae (yellow arrows) invading the gastric submucosal vessels (angioinvasion) with surrounding inflammatory cell infiltrates (H&E, 10x10).

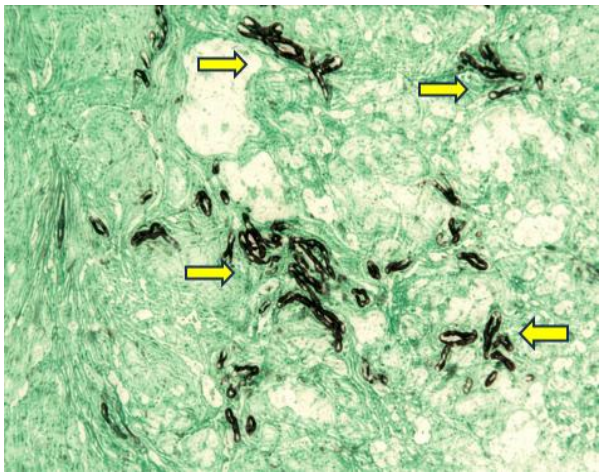

Figure 6: Gastrointestinal lesion, showing mucormycosis fungal hyphae (yellow arrows) stained in black (Grocott Methenamine Silver stain, 40X10).

DISCUSSION

Mucormycosis is a severe, opportunistic, angio-invasive fungal infection mainly observed in immunocompromised paediatric patients. Ingestion of Mucorales spores from contaminated food or water predisposes patients to invasive mucormycosis, particularly in the presence of impaired mucosal defence mechanisms. Among the affected systems of the body, GI involvement may represent the sole manifestation of mucormycosis, accounting for approximately 7% of reported cases[8]. In paediatric GI mucormycosis, the stomach is the most commonly affected organ, followed by the colon, small intestine, and oesophagus[9,10]. Clinical manifestations are nonspecific and vary according to the site involved, with abdominal pain, distension, vomiting, and gastrointestinal bleeding being common features. Most reported cases describe massive GI bleeding and perforation as the cause of death. In our case, the disease manifested with bloody diarrhoea without intestinal perforation at the time of death, possibly due to the short duration of illness and rapid clinical deterioration leading to death. GI haemorrhage, bowel perforation, and local infarction often necessitate urgent surgical resection and systemic antifungal therapy with amphotericin B[11,12].

According to the World Health Organization classification, birth at 34 weeks of gestation is considered a late preterm delivery (range 34 – 36+6 weeks of gestational age at birth), while a foetal weight of 1850 g at 34 weeks is most likely due to intrauterine growth restrictions [weight below 10th centile][13]. Therefore, this represents a case of marginal prematurity associated with intra-uterine growth restriction. The child's birth weight was 1.85 kg, which was below the −3 SD centile according to the weight-for-age chart in the Child Health Development Record (CHDR). The body weight recorded at autopsy was 6.9 kg and remained below the −3 SD centile, indicating severe malnutrition. Antemortem investigations revealed moderate anaemia, with a haemoglobin level of 9 g/dL. Although the child had gained weight over time, adequate catch-up growth was not achieved despite receiving a balanced diet and supplementary iron therapy.

Considering the child's age of 18 months and the associated predisposing factors, malnutrition was likely the most significant contributor to the virulence and invasive potential of Mucorales. Protein-energy malnutrition in toddlers is a recognized major risk factor for invasive fungal infections, particularly GI mucormycosis. Malnutrition impairs both innate and adaptive immunity, weakens mucosal barrier integrity, reduces phagocytic activity, and predisposes to opportunistic fungal infections. Proposed pathophysiological mechanisms include phagocytic dysfunction, impaired chemotaxis, and defective intracellular killing of Mucorales, particularly within an acidic gastric environment[13]. Iron is an essential growth factor for Mucorales, and increased availability of free iron enhances fungal proliferation, angioinvasion, and virulence, thereby contributing to the pathogenesis of mucormycosis[14].

Clinically, it is difficult to diagnose GI Mucormycosis due to its nonspecific presentation and low clinical suspicion. Most cases are diagnosed accidentally during surgery or autopsy examination. Biopsy of the affected site within the GI tract remains the gold standard for establishing the histological diagnosis of GI mucormycosis[15]. Microscopic identification of broad, irregular, aseptate hyphae with a right-angle branching pattern and evidence of angio-invasion is important. In some cases, a positive fungal culture or polymerase chain reaction (PCR) result from the biopsy specimen is required to confirm the diagnosis[16]. In the index autopsy case, incidental findings suggestive of mucormycosis were identified primarily through histopathological examination. A definitive diagnosis could have been further confirmed by positive fungal culture or PCR testing. Considering the clinical history, autopsy findings, and ancillary investigation results, the cause of death was concluded to be GI mucormycosis complicated by dehydration and probable electrolyte imbalance secondary to fluid loss, in the background of malnutrition and history of prematurity.

CONCLUSIONS

GI mucormycosis is a rare but highly aggressive fungal infection associated with significant mortality, particularly in vulnerable paediatric populations. This case highlights the diagnostic challenge posed by its nonspecific clinical presentation, which may closely mimic common conditions such as acute gastroenteritis or necrotizing enterocolitis, leading to delayed or missed diagnosis. Marginal prematurity, malnutrition, iron deficiency anaemia, and prior neonatal sepsis were important predisposing factors in this toddler. A high index of clinical suspicion, especially in at-risk children with rapidly deteriorating gastrointestinal symptoms, is essential. Histopathological examination and fungal cultures in combination with genetic material analysis (PCR) remain the cornerstone for definitive diagnosis. Early recognition, Prompt and appropriate systemic antifungal therapy treatment, together with early histopathological confirmation in cases of presumptive or suspected mucormycosis, is essential to facilitate timely intervention and improve outcomes.

LIMITATIONS

Definitive species-level identification could not be achieved because fungal culture and molecular diagnostic techniques were not performed. While these ancillary investigations may enhance diagnostic confirmation, their availability remains limited in routine practice in Sri Lanka.

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None.

CONFLICTS OF INTEREST

The authors declared no conflicts of interest.

ETHICAL ISSUES

The presented case was conducted for medicolegal purposes, and the findings were used for academic purposes, according to the institutional guidelines, without divulging the identity of the individual.

SOURCES OF SUPPORT

None.

AUTHOR CONTRIBUTIONS

UGBJ: Conception and design of the work; the acquisition, analysis, and interpretation of data for the work; drafting the work and revising it critically for important intellectual content; final approval of the version to be published; and agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. **KBR:** Conception and design of the work; the acquisition, analysis, and interpretation of data for the work; drafting the work and revising it critically for important intellectual content; final approval of the version to be published; and agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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