

Primary Cutaneous Mucormycosis Caused by *Apophysomyces elegans* in an Immunocompetent Child: A Rare Pediatric Case Report

Kokab Jabeen,¹ Faiqa Arshad,² Muhammad Suliman Sajid³

ABSTRACT

BACKGROUND & OBJECTIVE: Primary cutaneous mucormycosis is an uncommon invasive fungal infection that typically occurs in immunocompromised persons. *Apophysomyces elegans* is a member of order Mucorales and is an emerging pathogen that can cause severe soft tissue infections in immunocompetent individuals following minor trauma. Pediatric cases in immunocompetent children are exceedingly rare.

CASE PRESENTATION: We describe a healthy boy aged 9 years who presented with a progressive painful swelling and black discoloration of the right lower limb after a minor incident outdoors. The first treatment with oral antibiotics didn't work. On examination, it was found that there was necrotic eschar with surrounding erythema and tenderness. Laboratory exams were normal and HIV testing was negative. MRI showed soft tissue involvement in the absence of bone invasion. Direct potassium hydroxide (KOH) mount showed broad aseptate hyphae and fungal culture produced the growth of *Apophysomyces elegans*. Urgent surgical debridement and intravenous (IV) liposomal amphotericin B were performed. He improved markedly clinically and was discharged on oral posaconazole for 6 weeks. At 3-month follow-up, there were no recurrences.

CONCLUSION: This case illustrates that invasive fungal infections can be encountered even in immunocompetent children after minor trauma. Prompt and aggressive combined medical-surgical management and early suspicion is vital to survival.

KEY WORDS: Pediatrics, mucormycosis, immunocompetent .

How to cite: Jabeen K, Arshad F, Sajid SM. Primary Cutaneous Mucormycosis Caused by *Apophysomyces elegans* in an Immunocompetent Child: A Rare Pediatric Case Report. *J Allam Iqbal Med Coll.* 2026; 24(2): 69-72

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Mucormycosis is an invasive and life-threatening fungal infection caused by fungi that belongs to order Mucorales.^{1,2} The disease is characterized by rapid angioinvasion, thrombosis, tissue infarction, and extensive necrosis, and often results in significant morbidity and mortality if diagnosis and treatment are delayed. Individuals with uncontrolled diabetes mellitus, hematological malignancies, neutropenia, and those who are receiving immunosuppressive therapies are at the highest risk of developing this disease. Among the various clinical manifestations of mucormycosis, cutaneous involvement accounts for approximately 10–20% of reported cases. Primary cutaneous mucormycosis develops when fungal spores are directly introduced into disrupted skin caused by trauma, burns, surgical procedures, insect bites, and contaminated wounds. Although cutaneous disease generally carries a better prognosis than disseminated forms, delayed diagnosis may permit rapid extension into deeper soft tissues, fascia, muscle, and bone which leads to severe clinical outcomes.

Apophysomyces elegans is a thermotolerant environmental fungus that is mainly found in soil and decaying vegetation in tropical and subtropical regions.^{3,4} In contrast to other mucormycetes, *Apophysomyces elegans* mostly affects healthy individuals. It is increasingly recognized as a cause of aggressive cutaneous and soft tissue infection caused by trauma. The organism is particularly prevalent in tropical and subtropical regions where environmental conditions favor its growth. Since its first description as a human pathogen, *A. elegans* has increasingly been recognized as an important cause of necrotizing cutaneous and subcutaneous infections.⁵ Pediatric cases of primary cutaneous mucormycosis due to *A. elegans* are exceedingly rare, especially in immunocompetent children. Early clinical manifestations are often non-specific and presentations often mimic bacterial cellulitis or necrotizing soft tissue infection that results in delayed diagnosis. We report here an interesting and rare case of primary cutaneous mucormycosis caused by *A. elegans* in a previously healthy 9-years old boy following minor trauma. This case highlights the diagnostic challenges associated with this uncommon pathogen and emphasizes the critical role of early microbiological diagnosis, prompt surgical intervention, and aggressive antifungal therapy in achieving favorable clinical outcomes.

Correspondence:

Dr. Kokab Jabeen
Professor of Microbiology,
Allama Iqbal Medical College, Lahore
Email: kaukab_jabeen@yahoo.com

- * Received for Publication: April 11, 2026
- * Revision Received: May 15, 2026
- * Accepted for Publication: June 02, 2026

CASE PRESENTATION

A 9-year-old boy presented in our hospital with a 5-day history of progressively worsening pain, swelling, and black discoloration over the anterior aspect of right leg. The patient reported severe localized pain disproportionate to the apparent extent of injury. These symptoms developed after a minor fall when the boy was playing outdoors that resulted in a superficial abrasion. Initial treatment was carried out at a local health care facility, but it could not clinically improve the patient.

Physical examination revealed marked swelling of right shin with a necrotic tissue surrounded by redness, warmth, and tenderness. No regional lymphadenopathy was identified, and the patient had no fever. The patient's medical history was unremarkable. He had no known chronic illnesses, no history of corticosteroid, and immunosuppressive therapy, and his vaccination status was up to date. Laboratory investigations demonstrated mild leukocytosis ($12,800/\text{mm}^3$) and elevated C-reactive protein levels. Blood glucose levels, renal and liver function tests were within normal limits, and HIV serology was negative. MRI of the affected limb revealed extensive subcutaneous edema with no abscess formation and osseous involvement. Microscopic examination of tissue samples using potassium hydroxide preparation revealed broad aseptate fungal hyphae suggestive of mucormycosis. Subsequent fungal culture yielded growth of *Apophysomyces elegans*, which was confirmed by characteristic microscopic morphology using lactophenol cotton blue staining.

A multidisciplinary workup was established. The patient underwent urgent surgical removal of all necrotic tissue and started on intravenous liposomal amphotericin B at a dose of 5 mg/kg/day. Serial wound assessments were performed, and additional debridement procedures were performed as necessary to achieve adequate source control. Marked clinical improvement was observed within one week of therapy. There was reduction in pain and inflammation and the development of healthy granulation tissue. Following stabilization, treatment was changed to oral posaconazole for six weeks. At three-month follow-up, complete wound healing had occurred without functional impairment or evidence of recurrent infection.

DISCUSSION

Primary cutaneous mucormycosis represents a relatively uncommon manifestation of mucormycosis and results from direct inoculation of fungal spores into the skin and underlying soft tissues.⁶ In contrast to rhinocerebral and pulmonary forms, which are mainly observed in immunocompromised patients, cutaneous disease may occur in healthy individuals due to traumatic disruption of the skin barrier. Nevertheless, the disease remains highly invasive and can rapidly progress to deep tissue destruction if not recognized quickly. Among the causative organisms, *Apophysomyces elegans* occupies a unique position because of its ability to infect

immunocompetent hosts. The fungus is commonly found in soil and decaying vegetation, particularly in tropical and subtropical climates. Traumatic implantation of fungal spores during outdoor activities, road traffic accidents, natural disasters, or minor injuries has been implicated as the primary mechanism of infection. In the present case, a seemingly trivial abrasion sustained during outdoor play served as the likely portal of entry, ultimately leading to an invasive fungal infection despite the absence of traditional risk factors.

The pathogenesis of mucormycosis is largely attributed to the organism's angioinvasive nature. Due to tissue invasion, fungal hyphae penetrate blood vessel walls that results in vascular thrombosis, ischemia, and extensive tissue necrosis.⁷ Clinically, this process manifests rapidly progressive swelling, severe pain, and the characteristic black necrotic tissue observed in our patient. The presence of pain that is disproportionate to physical findings should alert clinicians to the possibility of an invasive necrotizing process rather than uncomplicated bacterial cellulitis. Diagnosis of cutaneous mucormycosis remains challenging because the initial presentation frequently resembles common bacterial skin infections. Consequently, many patients receive empirical antibacterial therapy before the correct diagnosis is established. In our patient, failure to respond to oral antibiotics, combined with progressive tissue necrosis, prompted further investigation. Direct microscopic examination using potassium hydroxide preparation demonstrated broad aseptate hyphae consistent with mucormycosis, while fungal culture confirmed *Apophysomyces elegans*. These findings underscore the importance of obtaining microbiological specimens early in cases of atypical or treatment-resistant soft tissue infections.

Imaging studies play an important supportive role in determining the extent of disease spread and guides surgical planning. Magnetic resonance imaging in our patient demonstrated extensive subcutaneous involvement without evidence of osseous extension and allowed timely intervention before deeper dissemination occurred. Although imaging findings are generally nonspecific, they are valuable for assessing disease progression and identifying complications. Current management strategies for mucormycosis rely upon three fundamental principles: early diagnosis, aggressive surgical debridement, and prompt initiation of systemic antifungal therapy. Surgical removal of all necrotic tissue is particularly important because vascular thrombosis may limit penetration of antifungal agents into infected areas. Liposomal amphotericin B remains the recommended first-line antifungal treatment owing to its potent activity against *Mucorales* species and improved safety compared with conventional amphotericin B formulations. In our patient, the combination of urgent surgical debridement and intravenous liposomal amphotericin B resulted in rapid clinical improvement and successful eradication of infection. Subsequent oral

posaconazole therapy provided effective consolidation treatment and contributed to sustained remission.

The favorable outcome observed in this case contrasts with the historically high morbidity and mortality associated with mucormycosis. Delayed diagnosis has been identified as one of the most important predictors of adverse outcomes, with studies demonstrating significantly increased mortality among patients in whom antifungal therapy is postponed. The absence of underlying immunosuppression, early recognition of disease progression, prompt microbiological confirmation, and timely multidisciplinary management likely contributed to the successful outcome in our patient. This case adds to the limited body of literature describing *Apophysomyces elegans* infections in immunocompetent pediatric patients. Given the rarity of this presentation, each additional case contributes valuable clinical information regarding risk factors, diagnostic approaches, treatment strategies, and outcomes. Increased awareness among clinicians is essential to facilitate earlier recognition and reduce the potentially devastating consequences of delayed intervention.

LIMITATIONS

Several limitations of this case report must be acknowledged. First, as a single case report, the findings cannot be generalized to a broader pediatric population, and the rarity of this presentation precludes definitive conclusions regarding epidemiology, optimal treatment duration, or long-term outcomes. Second, molecular identification techniques such as internal transcribed spacer (ITS) sequencing were not performed to confirm the species identification of *Apophysomyces elegans* beyond conventional morphological characterization; molecular typing would have strengthened the microbiological diagnosis and provided additional epidemiological data. Third, the exact inoculum size and precise mechanism of traumatic implantation could not be determined, limiting understanding of the minimum infective dose required to establish cutaneous mucormycosis in an immunocompetent host. Fourth,

although the patient demonstrated no clinical or laboratory evidence of immunodeficiency, comprehensive immunological profiling including lymphocyte subset analysis, immunoglobulin levels, and complement function was not performed, leaving open the possibility of an undetected subtle immune defect. Fifth, the follow-up period of three months, while sufficient to document complete wound healing, may not be adequate to exclude late recurrence, particularly given that prolonged antifungal suppression may be required in certain presentations of mucormycosis. Finally, the absence of histopathological examination with special fungal stains on the surgical specimen represents a diagnostic gap, as tissue histology would have provided additional confirmation of angioinvasion and corroborated the microbiological findings.

LITERATURE REVIEW

Primary cutaneous mucormycosis caused by *Apophysomyces elegans* is an uncommon infection that mainly affects individuals living in tropical and subtropical regions. In contrast to other members of the order Mucorales, *A. elegans* has been increasingly recognized as a pathogen capable of causing severe soft tissue infections in immunocompetent individuals following traumatic inoculation. The majority of published reports describe infections occurring after road traffic accidents, agricultural injuries, natural disasters, burns, or major penetrating trauma. Pediatric cases are considerably less common, and reports involving otherwise healthy children are particularly rare. Delayed diagnosis is frequent because the initial clinical presentation often mimics bacterial cellulitis or necrotizing soft tissue infection, leading to inappropriate antibiotic treatment and progression of tissue necrosis. A review of the available literature demonstrates that successful outcomes are strongly associated with early diagnosis, prompt surgical debridement, and initiation of amphotericin B-based antifungal therapy. Nevertheless, significant morbidity, including extensive tissue loss, multiple surgeries, limb amputation, and occasional mortality, continues to be reported. The present case

Aspect	Previously Known	Present Case
Typical Host	Immunocompromised adults	Immunocompetent child
Organism	Rare <i>Apophysomyces elegans</i> infection	Confirmed <i>A. elegans</i> infection
Age Group	Predominantly adults	9-year-old child
Risk Factor	Major trauma, burns, disasters	Minor superficial abrasion
Initial Presentation	Cellulitis-like lesions	Progressive necrotic lesion
Diagnosis	Often delayed	Early microbiological confirmation
Treatment	Debridement + Amphotericin B	Debridement + Amphotericin B + Posaconazole
Outcome	Variable; significant morbidity reported	Complete recovery without recurrence
Clinical Message	Rare invasive infection	Highlights need for suspicion in healthy children

contributes to the limited literature describing *Apophysomyces elegans* infection in an immunocompetent pediatric patient. The occurrence of invasive mucormycosis after a seemingly trivial outdoor injury, combined with early microbiological diagnosis and favorable outcome without permanent disability, highlights important clinical lessons for pediatricians, dermatologists, surgeons, and infectious disease specialists.^{6,7}

CONCLUSION

This case report describes a rare and successfully managed presentation of primary cutaneous mucormycosis caused by *Apophysomyces elegans* in a previously healthy 9-year-old child following minor outdoor trauma. The case underscores several critical clinical lessons. Mucormycosis is no longer exclusively a disease of immunocompromised patients; clinicians encountering rapidly progressive necrotizing soft tissue infections in otherwise healthy children must maintain a high index of suspicion for invasive fungal etiology, even in the absence of traditional predisposing conditions. Early and accurate microbiological diagnosis, achieved in this case through direct KOH microscopy and fungal culture, was instrumental in guiding appropriate therapy and avoiding further delay caused by futile antibacterial treatment.

The favorable outcome in this patient was attributed to the convergence of several favorable factors: early recognition of disease progression, prompt microbiological workup, timely surgical debridement to achieve adequate source control, and aggressive antifungal therapy with liposomal amphotericin B followed by oral posaconazole consolidation. These elements collectively highlight the indispensable role of multidisciplinary collaboration among pediatricians, infectious disease specialists, microbiologists, and surgeons in managing this potentially fatal condition. Physicians practicing in tropical and subtropical regions, where *Apophysomyces elegans* is endemic in the environment, should be particularly vigilant about the possibility of cutaneous mucormycosis even following seemingly trivial injuries in healthy children. Increased awareness, timely tissue sampling for fungal studies, and an aggressive combined medical-surgical approach remain the cornerstones of successful management and are essential to reduce the morbidity and mortality associated with this devastating infection.

Ethical Approval: Not Applicable

Patient consent: Written informed consent was obtained from the patient for publication of this case report and associated details

Conflict of Interest: Authors declare no conflict of interest.

Financial Disclosure: None

REFERENCES

1. Farmakiotis D, Kontoyiannis DP. Mucormycoses. *Infect Dis Clin North Am.* 2016 Mar;30(1):143–63. doi:10.1016/j.idc.2015.10.011
2. Ibrahim AS, Spellberg B, Walsh TJ, Kontoyiannis DP. Pathogenesis of mucormycosis. *Clin Infect Dis Off Publ Infect Dis Soc Am.* 2012 Feb;54 Suppl 1(Suppl 1):S16–22. doi:10.1093/cid/cir865
3. Guarro J, Chander J, Alvarez E, Stchigel AM, Robin K, Dalal U, et al. *Apophysomyces variabilis* infections in humans. *Emerg Infect Dis.* 2011 Jan;17(1):134–5. doi:10.3201/eid1701.101139
4. Landré V, Klingebiel FKL, van Niftrik CHB, Goetze E, Speck RF, Hübner CT, et al. Mucormycosis Caused by *Apophysomyces elegans*-A Case Report and Systematic Review of the Literature of Rhino-Orbito-Cerebral Cases of the Genus *Apophysomyces*. *J Fungi.* 2025 May 9;11(5):368. doi:10.3390/jof11050368
5. Lakshmi V, Rani TS, Sharma S, Mohan VS, Sundaram C, Rao RR, et al. Zygomycotic necrotizing fasciitis caused by *Apophysomyces elegans*. *J Clin Microbiol.* 1993 May;31(5):1368–9. doi:10.1128/jcm.31.5.1368-1369.1993
6. Simbli M, Hakim F, Koudieh M, Tleyjeh IM. Nosocomial post-traumatic cutaneous mucormycosis: a systematic review. *Scand J Infect Dis.* 2008;40(6–7):577–82. doi:10.1080/00365540701840096
7. Ibrahim AS, Spellberg B, Walsh TJ, Kontoyiannis DP. Pathogenesis of mucormycosis. *Clin Infect Dis Off Publ Infect Dis Soc Am.* 2012 Feb;54 Suppl 1(Suppl 1):S16–22. doi:10.1093/cid/cir865

Authors' Contributions:

KJ & FA: Conceptualization & study design.

MSS: Data Collection and manuscript drafting.

MSS, KJ, FA: Supervision & Manuscript drafting & proof reading.

All authors have read and approved the final version of the manuscript and are responsible and accountable for the accuracy and integrity of the work.

-
1. Kokab Jabeen
Professor of Microbiology,
Allama Iqbal Medical College, Lahore
 2. Faiqa Arshad
Associate Professor of Microbiology,
Allama Iqbal Medical College, Lahore
 3. Muhammad Suliman Sajid
MBBS Student
Allama Iqbal Medical College, Lahore