

Case Report

Progressive external nasal destruction caused by presumed invasive sinonasal aspergillosis in an apparently immunocompetent infant: a case report

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ABSTRACT

Invasive sinonasal aspergillosis is a life-threatening infection classically seen in immunocompromised patients. It has occurred in infants with grade II protein-energy malnutrition without typical immunocompromising conditions. External cutaneous nasal extension further distinguishes this presentation from previously described paediatric cases. An 18-month-old girl with grade II protein-energy malnutrition presented with a 7-8-month history of progressive ulceration, crusting, and destruction of the external nose. Diagnostic nasal endoscopy under general anaesthesia revealed extensive black necrotic fungal debris within both nasal cavities. Histopathological examination of the biopsy specimen revealed slender septate fungal hyphae with acute-angle branching, consistent with *Aspergillus* species. There were no known immunocompromising conditions, although a full immunological workup was limited. The caregivers declined further evaluation and definitive treatment, and the child left against medical advice. This case demonstrates that presumed invasive sinonasal aspergillosis with external cutaneous extension can occur in infants even in the absence of known immunocompromising conditions. Early nasal endoscopy and prompt tissue biopsy are essential for timely diagnosis. Although grade II protein-energy malnutrition may have contributed to host susceptibility, its causal role cannot be established from a single case.

Keywords: Invasive aspergillosis, Sinonasal aspergillosis, Invasive fungal rhinosinusitis, Infant, Malnutrition, Cutaneous extension, Nasal endoscopy, Case report

INTRODUCTION

Progressive destructive lesions of the external nose in infancy are uncommon and can be difficult to diagnose. Although bacterial infections, granulomatous disorders, and neoplasms are common considerations, invasive fungal rhinosinusitis should also be included in the differential diagnosis because delayed recognition may result in extensive tissue destruction and life-threatening complications.^{1,2}

Invasive sinonasal aspergillosis is an aggressive form of fungal rhinosinusitis characterized by fungal invasion of the sinonasal mucosa and adjacent tissues.^{1,2} It primarily occurs in individuals with known immunocompromising

conditions like uncontrolled diabetes mellitus, hematological malignancies, prolonged corticosteroid therapy, or post-transplant immunosuppression.¹ Reports of this condition in infants without these classical risk factors are exceptionally rare, and external cutaneous involvement of the nose has only infrequently been described in the pediatric literature.^{3,4}

We report a case of presumed invasive sinonasal aspergillosis presenting as progressive external nasal destruction in an 18-month-old infant with grade II protein-energy malnutrition but no classical immunocompromising condition. This case highlights the importance of maintaining a high index of suspicion for invasive fungal disease in infants with destructive nasal

lesions. It also emphasizes the diagnostic value of early nasal endoscopy and tissue biopsy.

CASE REPORT

An 18-month-old girl was brought to outpatient department of otorhinolaryngology with a 7-8-month history of progressively enlarging ulcerative lesions over external nose. Lesions began as a small boil over nasal tip and gradually progressed to involve nasal dorsum and philtrum, causing noticeable nasal deformity despite repeated symptomatic treatment at peripheral health facilities. There was no history of DM, prolonged corticosteroid/ antibiotic use, recurrent severe infections/ any family history of primary immune-deficiency.

The child weighed 7.2 kg (expected weight for age approximately 10 kg), which was consistent with grade II protein-energy malnutrition (weight-for-age Z-score between -2 and -3 SD). General examination revealed pallor but no hepatosplenomegaly, generalised lymphadenopathy, or cutaneous stigmata of primary immunodeficiency. Bilateral cervical lymphadenopathy was present, likely reactive in nature.

Local examination revealed multiple nodulo-ulcerative lesions with crusting and areas of pink-to-black discoloration involving the nasal tip, dorsum, and philtrum (Figure 1 A-C). The lesions were friable and bled on contact. Nasal examination was limited because of marked external swelling.

Routine laboratory investigations revealed severe anaemia (haemoglobin 7.0 g/dL) with thrombocytosis

(platelet count $11.66 \times 10^5/\mu\text{L}$). Random blood glucose, HIV ELISA, and Mantoux test were negative, and no routine haematological evidence of immunodeficiency was identified. Chest radiography was normal. Plain radiography of the paranasal sinuses demonstrated bilateral maxillary sinus haziness.

Diagnostic nasal endoscopy performed under sedation revealed extensive black necrotic fungal plaques, friable nasal mucosa, and multiple nodular lesions involving both nasal cavities. Advancement of the endoscope was limited because of contact bleeding. Multiple biopsy specimens were obtained from the necrotic tissue.

Histopathological examination of the biopsy specimens demonstrated necrotic tissue heavily infiltrated by slender septate fungal hyphae with characteristic acute-angle branching, features consistent with *Aspergillus* species (Figure 2 A-D).

No fungal culture was done prior to discharge. Based on the combined endoscopic and histopathological findings, a diagnosis of presumed invasive sinonasal aspergillosis with cutaneous extension was established. The use of the term 'presumed' reflects the lack of culture or molecular species confirmation.

Further radiological evaluation, fungal culture, formal immunological workup, and definitive antifungal therapy could not be undertaken because the caregivers declined all further investigations and treatment and left the hospital against medical advice. All available diagnostic information was shared with the family, along with the risks of untreated invasive fungal disease.



Figure 1 (A-C): Clinical presentation at the time of initial assessment. (A) Frontal view demonstrating a large nodulo-ulcerative lesion involving the nasal tip with extensive crusting and areas of black necrosis. (B) Left lateral view showing marked expansion and deformity of the external nose. (C) Right lateral view demonstrating ulceration and crusting of the nasal tip and ala with surrounding inflammatory induration.

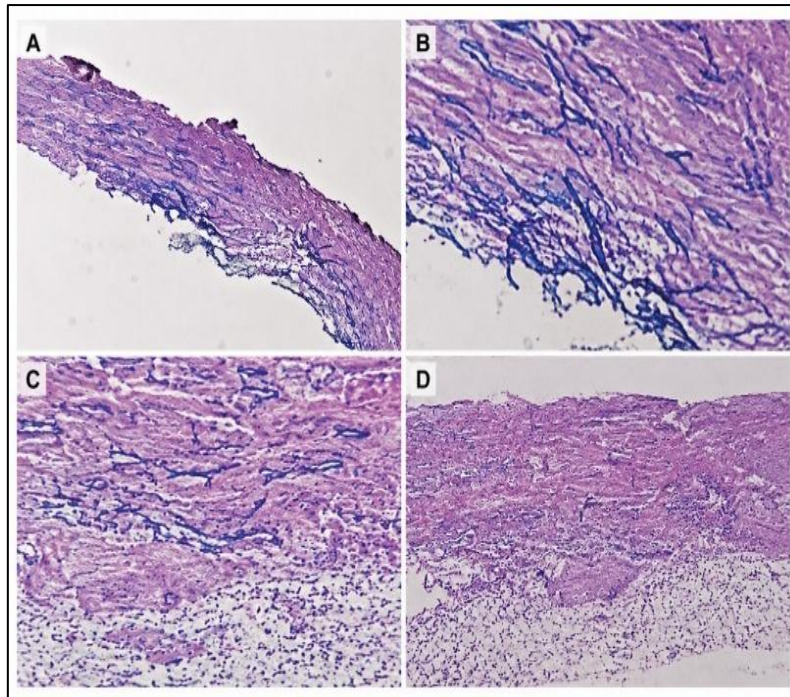


Figure 2 (A-D): Histopathological examination of the nasal biopsy specimen (Special stain; A-D). (A) Low-power view demonstrating areas of necrotic tissue. (B-D) High-power views showing slender, regular, septate fungal hyphae with characteristic acute-angle (approximately 45°) branching, features consistent with *Aspergillus* species.
 *Species identification by culture or molecular testing was not performed.

DISCUSSION

Invasive sinonasal aspergillosis is an uncommon but potentially life-threatening form of fungal rhinosinusitis characterized by tissue invasion and angioinvasion, leading to vascular thrombosis, tissue ischemia, and progressive necrosis.^{1,2} It is encountered predominantly in patients with well-recognized immunocompromising conditions such as uncontrolled diabetes mellitus, haematological malignancies, prolonged corticosteroid therapy, or transplantation.¹ Consequently, the diagnosis may not be considered early in infants without these classical risk factors, resulting in delayed treatment and significant tissue destruction.¹

The present case is noteworthy for several reasons. First, the patient was only 18 months old, making her among the youngest reported children with presumed invasive sinonasal aspergillosis. Second, unlike previously published paediatric reports that primarily presented with orbital involvement, our patient had progressive destruction of the external nose with notable cutaneous extension.

Third, the diagnosis was established by early nasal endoscopy and histopathological examination despite the absence of fungal culture or molecular identification. Comparison with previously published paediatric cases is summarized in Table 1.^{3,4}

Table 1: Published paediatric reports of invasive aspergillosis involving the sinonasal region in apparently immunocompetent children.

Authors	Year	Age/sex	Initial presentation	Site of disease	External nasal involvement	Histopathology	Culture	Treatment	Outcome
Chowdhary et al ⁴	2024	10-year-old/ Female	Orbital swelling with fever	Ethmoid sinus with orbital subperiosteal abscess	No	Yes	Yes	Surgical drainage + voriconazole	Complete recovery
Rafizadeh et al ³	2025	4-year-old/ Female	Eyelid swelling mimicking orbital tumour	Sino-orbital disease with frontal bone erosion	No	Yes	Yes (<i>A. fumigatus</i>)	Surgical excision + voriconazole	Disease-free

Continued.

Authors	Year	Age/ sex	Initial presentation	Site of disease	External nasal involvement	Histopathology	Culture	Treatment	Outcome
Present case	2026	18-month-old/ Female	Progressive boil over nasal tip progressing to ulceration and nasal deformity	Sinonasal disease with external nasal destruction	Yes	Yes	No	LAMA before definitive treatment	Unknown

Instances of Invasive fungal disease has occasionally been reported in apparently immunocompetent infants, including a 17-month-old child with mixed orbital aspergillosis and mucormycosis reported by Habroosh et al.⁵ However, that case differed substantially from the present report in terms of anatomical site, microbiology, and clinical presentation.

Although no classical immunocompromising condition was identified, the child had grade II protein-energy malnutrition and severe anaemia. Routine investigations excluded common acquired risk factors, including diabetes mellitus and HIV infection; however, comprehensive immunological evaluation was not performed. Therefore, the patient is more appropriately described as apparently immunocompetent. We cannot determine whether malnutrition alone predisposed to invasive aspergillosis based on a single case, although impaired nutritional status may have contributed to reduced host defense.⁶⁻⁸

The principal differential diagnosis was mucormycosis, which shares similar clinical features, including black eschar, tissue necrosis, and rapidly progressive facial destruction. Histopathological examination demonstrated slender septate fungal hyphae with acute-angle branching, findings that favour *Aspergillus* species over the broad pauciseptate hyphae with right-angle branching characteristic of Mucorales.^{1,2} Although fungal culture was unavailable, the histomorphological findings were sufficiently characteristic to establish presumed invasive aspergillosis.

This case also highlights the importance of early tissue diagnosis. Progressive nasal ulceration, crusting, or external nasal destruction in infants should prompt urgent nasal endoscopy and biopsy rather than repeated empirical antimicrobial therapy. Histopathological confirmation remains the cornerstone for demonstrating tissue invasion, while fungal culture and molecular techniques provide species-level identification whenever feasible.^{1,2} Early recognition is vital, as delayed diagnosis may result in extensive local destruction and increased morbidity.

This report has several limitations. Fungal culture, molecular identification, comprehensive immunological investigations, and cross-sectional imaging were not performed because the caregivers declined further

evaluation and treatment. Thus, we must describe the diagnosis as presumed invasive sinonasal aspergillosis, with the exact extent of disease and long-term clinical outcome remaining uncertain. Nevertheless, the combination of characteristic clinical presentation, endoscopic findings, and histopathological evidence provides convincing support for the diagnosis and emphasizes the need to consider invasive fungal disease in infants with progressive destructive nasal lesions, even without classic immunocompromising conditions.

CONCLUSION

This report highlights that presumed invasive sinonasal aspergillosis, although exceptionally rare, should be considered in infants presenting with progressive destructive lesions of the external nose, even in the absence of classical immunocompromising conditions. Early nasal endoscopy with prompt tissue biopsy is essential for establishing the diagnosis and facilitating early diagnosis and appropriate management. Although the patient had grade II protein-energy malnutrition, its contribution to disease susceptibility cannot be established from a single case and warrants further investigation. Increased awareness of this uncommon presentation may help minimize diagnostic delay and prevent extensive local tissue destruction.

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