Refractory tinea caused by Aspergillus niger

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DESCRIPTION

A 4-year-old boy presented to the clinic with a persistent rash despite ongoing management for fungal dermatitis. His foster mother for the past year described the presence of a large, raised and scaly scalp lesion when he first came under her custody. Though he had previously been neglected, there was no known history of frequent infections, atopic disease or other significant medical issues. His prior treatment regimen had included topical antifungal medications, ketoconazole shampoo and a 14-week course of two times per day, high-dose (20–25 mg/kg/day) oral griseofulvin, but he had only mild improvement initially. Most recently, the rash appeared to be worsening and spreading to his neck and torso.

On physical examination, several circular and irregular patches, with raised, erythematous borders and central hypopigmentation, were prominent on his scalp, neck, abdomen and back (figures 1 and 2); the lesions were all clinically consistent with typical tinea capitis and corporis. The rest of his examination was normal.

A skin scraping grew Aspergillus niger on Sabouraud agar (fungal culture). He had already been placed on terbinafine, but dermatitis only progressed during 2 weeks of this therapy. Based on superior clinical effectiveness against Aspergillus, he was switched to voriconazole by infectious diseases specialists. The patient initially showed a strong response, especially with improvement on his scalp and his body lesions becoming less erythematous and smaller. However, after the first few weeks of therapy, lapses from continuous voriconazole treatment led to a recrudescence of the tineaconsistent rash. He ultimately was felt to also have



Figure 1 Patient's neck lesions.



Figure 2 Patient's truncal lesions.

secondary immunologic dermatophytid (id) reaction lesions on his body, which responded to topical corticosteroids.

Recent international reports note changing aetiology of superficial mycoses, with the emergence of non-dermatophyte fungi, particularly Aspergillus species, causing tinea capitis and onychomycosis. 12 One study in India found that 14.5% (9/62) of the superficial (skin/nails) fungal isolates were nondermatophyte moulds including Aspergillus spp.³ In a 2018 report from Ethiopia, 46% (76/164) of the superficial fungal isolates were non-dermatophyte moulds with Aspergillus spp commonly isolated.4 Additionally, a 2019 case report from China described a kerion-type scalp mycosis caused by Aspergillus protuberus in a 5-year-old child, which responded to oral terbinafine treatment.⁵ A. niger underlying prolonged scalp tinea with alopecia, similar to our patient, has also been reported in two children in Brazil.²

Learning points

- ► There are increasing reports internationally of non-dermatophyte fungi, including *Aspergillus*, causing tinea capitis and onychomycosis.
- ► In cases of tinea capitis which respond poorly to conventional therapy such as griseofulvin, consider moulds as the potential aetiology, for which terbinafine and systemic azole antifungals are likely to be effective.



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Aspergillus species are a leading causative organism of non-dermatophyte mould onychomycosis (NDMO). In a review of international studies from 1974 to 2017, over 50% (23/42) included NDMO cases isolating Aspergillus. NDMO caused by Aspergillus spp responds well to systemic agents, and in vitro, itraconazole performs better than terbinafine. A Cochrane review found terbinafine to be more effective than griseofulvin for onychomycosis, and terbinafine combined with an azole was more effective than terbinafine alone, without any increase of adverse events. This case of Aspergillus underlying a superficial mycosis represents an unusual but potentially increasing presentation around the globe. In paediatric patients with tinea capitis who fail to respond to conventional therapy, non-dermatophyte fungal aetiologies and alternative systemic antifungals should be considered.

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