

# Head Injury Exacerbates Tinea Corporis!!

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A 32-year-old, 67 kg male patient was admitted to the neurosurgical intensive care unit (ICU) for the management of severe traumatic brain injury (TBI) (left frontoparietal contusion with midline shift) and cervical spine injury. On admission to ICU, his Glasgow Coma Scale (GCS) was E2VTM5 and he had associated right-sided humerus fracture. He did not have any other comorbid illness/was not receiving any medication and he did not have any apparent skin lesions at the time of admission to ICU. His routine investigations and other systemic examinations were normal. The patient had a downhill course in the next 24 hours and required inotropic support (inj. dopamine). Along with dopamine, patient was receiving intravenous fluid: mannitol, fentanyl, midazolam, and levetiracetam. On the third day of admission, we noticed erythematous scaly lesions with central clearing in the right forearm and shoulder, and axillary lesions (**►Fig. 1**). The lesions progressed fast to involve the upper half of the chest within next 48 hours. The lesions were diagnosed to be tinea corporis on the basis of clinical and microbiological findings (potassium hydroxide mount examination), and tablet terbinafine 100 mg twice daily was started. Patient's clinical condition improved (GCS improved to E3 V5 M6) in next 8 days, and the skin lesions started to subside. Terbinafine continued for total 3 weeks and the lesions completely subsided.

Drug-induced cutaneous eruption might have been a possibility in our cases. The common drugs implicated to cause skin lesions are phenytoin, carbamazepine, oxcarbazepine, phenobarbitone, primidone, zonisamide, and lamotrigine.<sup>1</sup> Our patient was receiving levetiracetam and drug-induced cutaneous eruption is very rare with levetiracetam. Since the lesion was confirmed both by clinical and microbiological examination and the lesions responded to antifungal medication, possibility of a drug-induced skin lesion was ruled out. Tinea corporis is a common superficial fungal infection and is contagious. It remains unnoticed very often in an intensive care setup.<sup>2</sup> Our patient did not have any manifestation of tinea corporis at the time of admission and our ICU did not have a known source during that period. However, diseases or conditions leading to disorder of cell-mediated immunity are known to cause progression of superficial fungal infections. Factors such as immunodeficiency syndrome,

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immunosuppression, treatment with corticosteroids or cytotoxic medication, and malnutrition can lead to tinea corporis infection.<sup>3</sup> Nevertheless, except for head injury, our patient did not have any other predisposing factors.



**Fig. 1** Tinea corporis lesions.

Head injury can lead to immune system abnormality. Severe TBI can result in significant decrease in the percentage and absolute number of circulating T lymphocytes, leading to reduction in both CD4<sup>+</sup> T helper cells and CD8<sup>+</sup> cytotoxic T cells. This can subdue cell-mediated immunity.<sup>4,5</sup> This decrease can occur even within 24 hours of injury.<sup>4</sup> In our case, most probably immune suppression secondary to TBI was the predisposing factor causing exacerbation of latent tinea corporis. Extensive tinea corporis requires treatment with systemic antifungal agents and terbinafine is one of the effective oral antifungal medications for tinea corporis.<sup>6</sup> So we treated our patient with terbinafine and the lesions subsided.

So, to conclude, TBI-induced immune suppression can cause rapid progression of tinea corporis in critically ill patients and knowledge about this fact may lead to its effective management in an ICU setting.

#### **Conflict of Interest**

None declared.

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