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FROM VESICLES TO FLAMES: VARICELLA INDUCED TOXIC EPIDERMAL NECROLYSIS

Dermatology

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ABSTRACT

Background: Toxic epidermal necrolysis (TEN) is a rare, life-threatening mucocutaneous disorder characterized by widespread epidermal necrosis, skin detachment, and multisite mucosal involvement. Drugs are the most commonly implicated triggers; however, infections, particularly viral infections such as varicella- zoster virus (VZV) are rare but increasingly recognized etiological factors. TEN following varicella infection in immunocompetent adults is uncommon and poses diagnostic and therapeutic challenges. **Case Presentation:** A 32-year-old immunocompetent male presented with burning sensation, extensive skin peeling, oral erosions causing dysphagia, and ocular redness. Fifteen days prior, he developed fever, headache, and vesicular eruptions beginning over the trunk and progressively involving the face and extremities. The lesions evolved into papules, pustules, crusts, followed by widespread desquamation. There was no history of recent drug intake. A significant family history revealed varicella infection in the patient's mother 10 days before symptom onset in the patient. Patient had no similar history during childhood. Cutaneous examination revealed epidermal detachment involving more than 30% body surface area with positive pseudo-Nikolsky sign and mucosal involvement. Histopathology confirmed full- thickness epidermal necrosis consistent with TEN. VZV serology showed markedly elevated IgG (2026 mIU/mL) with low IgM levels, supporting recent infection. **Conclusion:** This case highlights varicella-zoster virus as a rare infectious trigger of TEN in immunocompetent adults. Early recognition, exclusion of drug causes, and prompt supportive management are crucial for favorable outcomes.

KEYWORDS

Toxic Epidermal Necrolysis; Varicella-Zoster Virus; Viral-Induced TEN; Mucocutaneous Involvement

INTRODUCTION

Toxic epidermal necrolysis (TEN) is a rare, life-threatening mucocutaneous disorder characterized by extensive epidermal necrosis, skin detachment, and involvement of multiple mucosal surfaces. It represents the severe end of the spectrum of Stevens–Johnson syndrome (SJS) and TEN, differentiated primarily by the percentage of body surface area (BSA) involved [1,2]. The condition is most commonly triggered by medications, including anticonvulsants, NSAIDs, and antibiotics; however, infectious etiologies have also been implicated, particularly viral infections such as herpes simplex virus, Mycoplasma pneumoniae, and less commonly varicella zoster virus (VZV) [3,4]. TEN results from an immune-mediated cytotoxic response causing massive keratinocyte apoptosis. Cytotoxic T lymphocytes and natural killer cells release perforin, granzyme B, and granulysin, while tumor necrosis factor-α and Fas–Fas ligand interactions further propagate epidermal cell death. [5,6].

Varicella zoster virus infection is generally a self-limiting illness in immunocompetent individuals; however, in adults it may present with severe complications, including pneumonia, encephalitis, and rarely severe cutaneous adverse reactions such as TEN [7]. The association between varicella infection and TEN is uncommon and sparsely reported in the literature, making such presentations diagnostically challenging. In some cases, TEN may develop during the recovery phase of varicella, when vesicular lesions begin to crust and heal, suggesting an immune-mediated post-infectious phenomenon rather than direct viral cytopathic damage [8].

Laboratory findings in TEN varies according to etiology, including lymphopenia, elevated inflammatory markers, and electrolyte imbalance from skin loss[9]. In the absence of drug exposure, elevated IgG antibodies to Varicella-zoster virus support a recent infectious trigger[10]. Management of TEN requires early recognition, withdrawal of the offending trigger, and intensive supportive care similar to burn management, including fluid resuscitation, wound care, infection prevention, and meticulous monitoring of vital parameters [11]. The use of topical emollients, sterile non-adherent dressings, and supportive ophthalmic and oral care are essential components of therapy. Despite advances in supportive management, TEN continues to be associated with significant morbidity and mortality, emphasizing the importance of identifying rare triggers such as varicella infection [12].

Case Presentation

A 32-year-old male presented to our dermatology opd with acute onset of painful, rapidly progressive rash with peeling of skin, oral erosions, difficulty in swallowing, and redness of both eyes. The patient was apparently healthy 15 days prior to presentation, then he developed fever, body ache, and headache, followed by the appearance of fluid filled lesions over the trunk(Fig.4). Over the next few days, the lesions progressively involved the upper extremities, face, and subsequently the lower extremities.

The vesicular lesions gradually evolved into pustules, which later crusted over with surrounding erythema. As the lesions began to heal, the patient noticed extensive peeling of skin associated with pain and discomfort. Concurrently, he developed erosions in the oral cavity causing dysphagia, along with conjunctival redness.

There was no history of drug intake prior to the onset of symptoms, including antibiotics, NSAIDs, anticonvulsants, or native medications. The patient denied any past history of similar illness. On eliciting family history, it was noted that the patient's mother had suffered from an episode of varicella infection approximately 10 days prior to the onset of the patient's symptoms, suggesting a possible exposure. The patient had no known comorbidity.

On general physical examination, the patient was conscious and oriented. Cutaneous examination revealed widespread areas of erythema with sheet-like epidermal detachment involving more than 30% of the body surface area over the back of trunk (Fig.1) Multiple areas of denuded skin were noted over the upper chest with hyperpigmented superficial crusting (Fig.2) and similar lesions over extremities and face.

Axillary area involved a large erosion exting to the back (Fig.3) A pseudo-Nikolsky sign was positive. Mucosal involvement was evident in the form of painful erosions over the oral cavity and conjunctival congestion , genital mucosa was normal.

Based on the extent of epidermal detachment and mucosal involvement, a clinical diagnosis of toxic epidermal necrolysis was considered. The SCORTEN score was 2.

A skin biopsy was performed from the lesional area. Histopathological examination revealed features characteristic of toxic epidermal necrolysis, including full-thickness epidermal necrosis with minimal dermal inflammation.

Laboratory investigations showed hemoglobin of 12.5 g/dL and a total leukocyte count of 10,000/mm³ with evidence of lymphopenia. Inflammatory markers including ESR and CRP were elevated. Serum urea was 38mg/dL. Serological tests for HIV, hepatitis B and C, and VDRL were negative. Varicella zoster virus serology demonstrated a markedly elevated IgG level of 2026 mIU/mL, indicating a more than four-fold rise from reference values, while IgM levels were 0.69, supporting recent exposure or infection.

The patient was managed in intensive care unit. Intravenous fluid resuscitation was initiated according to percentage of body surface area involved using Parklands formula. Broad-spectrum systemic antibiotics ceftriaxone 1 g intravenously twice daily) were administered prophylactic-ally.Patient was started on oral cyclosporine 5mg/kg dose for 2weeks until disease progression stopped and then continued on tapering dose for 1 month . NSAIDs were given for pain management. Cutaneous care included barrier nursing, application of paraffin dressings and autoclaved banana leaves to minimize skin trauma. Strict monitoring of temperature, urine output, and fluid balance was maintained. Adequate protien intake was ensured through diet.

Oral mucosal involvement was managed with chlorhexidine mouthwash,lignoicaine gel and triamcinolone gel applied twice daily. Ocular care included warm saline compresses and carboxymethyl-cellulose eye drops. The patient showed gradual improvement with stabilization of skin lesions and mucosal healing on continued supportive management.

DISCUSSION

Toxic epidermal necrolysis (TEN) is a condition on the severe end of drug hypersensitivity reactions spectrum, however infections particularly viral infections, have increasingly been recognized as potential triggers. In the present case, a 32-year-old immunocompetent male developed TEN with more than 30% body surface area involvement and significant mucosal manifestations following recent exposure to varicella, supported by serological evidence.

Chahal et al., in their systematic review of vaccine-induced SJS/TEN, reported that among 29 published cases, only four were TEN, emphasizing the rarity of non-drug- induced TEN [13]. Although their focus was vaccine-related cases, the review reinforces the concept that immune activation—whether by vaccines or infections—can precipitate TEN. Importantly, the authors advocated for considering non-drug triggers when classical etiologies are absent, a principle directly applicable to the present case.

Varicella-zoster virus is known to cause a spectrum of cutaneous and systemic manifestations. Although varicella more commonly results in benign vesicular eruptions, severe immune-mediated reactions have been documented. Vassia et al. reported a fatal disseminated VZV infection in an immunosuppressed patient, underscoring the aggressive potential of VZV in triggering systemic inflammatory responses, even in the absence of classic cutaneous signs [14]. While our patient was immunocompetent and did not develop visceral dissemination, the case illustrates that VZV can act as a potent immunological trigger capable of inducing severe cutaneous adverse reactions such as TEN.

Gupta et al. described an atypical case of disseminated hemorrhagic varicella mimicking TEN in an immunocompetent adult, highlighting the diagnostic challenge posed by overlapping clinical features of viral exanthems and TEN[15]. In contrast, our patient demonstrated histopathological confirmation of TEN, thereby excluding a TEN-like varicella presentation and reinforcing the diagnosis of true epidermal necrolysis secondary to viral infection. The presence of lymphopenia, elevated ESR and CRP in our patient further supports an intense inflammatory and immune-mediated process.

The absence of antecedent drug exposure strongly supports an infectious etiology in this patient. Fetriani and Zakiawati systematically reviewed ten studies of virus-associated SJS/TEN, identifying herpes simplex virus, influenza virus, HIV, COVID-19, and varicella-zoster virus as implicated triggers[16]. They noted that viral infections occurring within one week prior to rash onset may precipitate severe epidermal necrolysis, underscoring the importance of clinical vigilance. The close temporal relationship between household varicella exposure and symptom onset in our patient, supported by serological confirmation, is consistent with these findings.

Gungor et al. reported necrotizing scleritis secondary to VZV infection, emphasizing that ocular involvement in varicella can be severe and vision-threatening if not promptly recognized and treated [17]. Our patient exhibited conjunctival redness and irritation, necessitating early ophthalmic care, which likely contributed to the favorable outcome.

CONCLUSION

Toxic epidermal necrolysis (TEN) is a rare, potentially fatal dermatological emergency requiring prompt recognition and aggressive supportive care. While drugs are the most common trigger, Varicella-zoster virus infection can rarely precipitate TEN, even in immunocompetent adults. The overlap between viral exanthems and TEN poses diagnostic challenges, highlighting the importance of detailed history, targeted investigations, and early biopsy. Prompt supportive management—including fluid resuscitation, meticulous skin and mucosal care, and close monitoring—was critical to a favorable outcome. Clinicians should maintain a high index of suspicion for non-drug triggers, particularly viral infections, to enable early diagnosis reduce TEN-related morbidity and mortality.

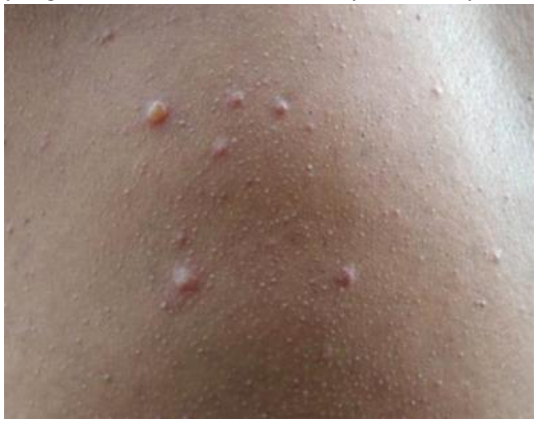

Figure 1: Multiple Discrete Vesicles and Few Papules Seen Over Back of Trunk

Figure 2: Multiple Areas of Denuded Skin With Superficial Hyperpigmented Crusting

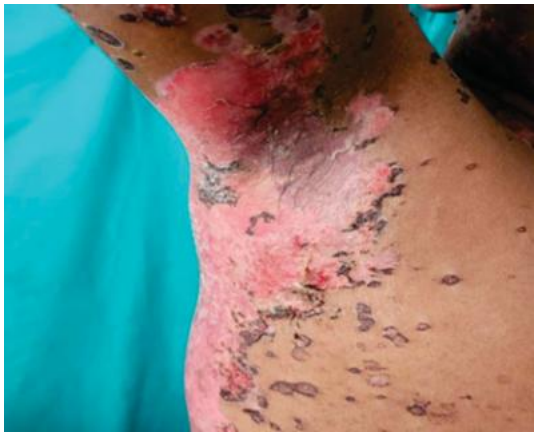

Figure 3: Large Erosion in the Axillary Area Extending to the Back

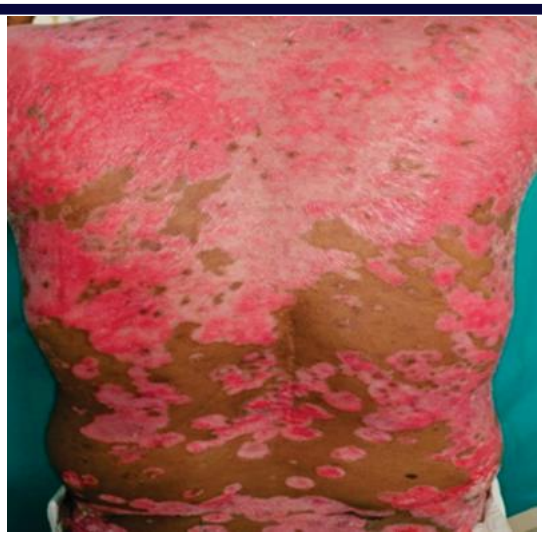

Figure 4: Large Areas of Raw Erosions and Erythematous Plaques Covering >30% Back of Trunk.

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