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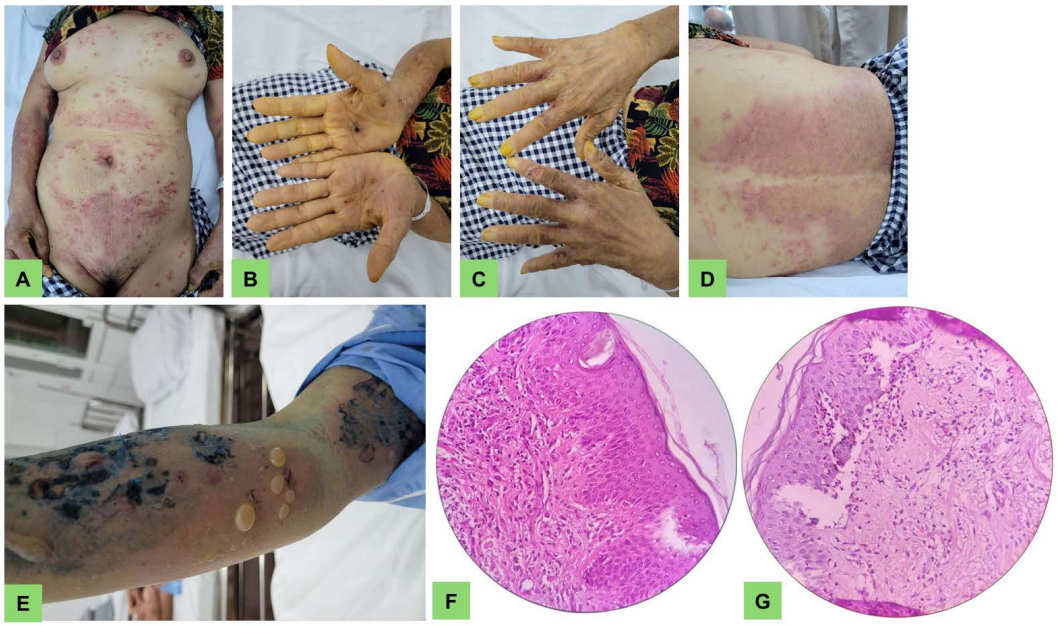

Figure 1. Clinical and Histopathological Presentation on Admission. Disseminated, intensely pruritic, erythematous plaques with yellowish crusts distributed on the chest and abdomen, including the periumbilical region (A). The palms and interdigital spaces of the fingers (B, C), and the back (D). (E) Tense, opaque-white bullae developing on an erythematous base located on the forearm. (F) Histopathology from a burrow (H&E staining x20) revealing epidermal hyperplasia, spongiosis, an intracorneal mite (blue arrow), and scattered dermal eosinophilic infiltrate. (G) Histopathology from a bulla (H&E staining x20) demonstrating a subepidermal blister containing predominantly eosinophil-rich infiltrate within the blister cavity and scattered throughout the dermis.

An 80-year-old female patient, with a history of stroke sequelae and poorly controlled type II diabetes mellitus, presented with a sudden onset of symptoms one month prior. The condition began with the appearance of red, pruritic papules with yellowish crusts. These lesions subsequently disseminated, increased in size to form large plaques, and spread to multiple locations, including the interdigital finger spaces, hands, interdigital toe spaces, back, chest, buttocks, and groin (Figures 1A-D). The eruption was accompanied by development of numerous tense bullae on the erythematous base of the papules and plaques (Figure 1E).

The patient reported intense pruritus, with significant nocturnal exacerbation. Persistent scratching led to excoriations and rupture of the bullae, resulting in erosions.

A skin biopsy taken from a scaly, erythematous plaque near the umbilicus revealed significant spongiosis and an intracorneal structure consistent with a mite, along with scattered dermal infiltrate of eosinophils (Figure 1F). A second biopsy from an inflamed bulla on the arm demonstrated a subepidermal blister with prominent infiltration of eosinophils within the blister cavity and throughout the dermis (Figure 1G).

Bullous scabies, also known as scabies-induced bullous pemphigoid-like eruption, is a rare variant of scabies, with

What is your diagnosis?
See the next page for your diagnosis.

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Photoclinic Diagnosis

Bullous Scabies

only approximately 60 cases reported in the literature.[1] This rarity has limited both the understanding of its diagnosis and clinical management experience. The clinical presentation of bullous scabies combines the characteristic scabies lesions and those of pemphigoid.[2] Firstly, it typically affects elderly patients with chronic comorbidities or those with compromised ability to maintain personal hygiene due to illness or advanced age.[3] In our case, the patient had a history of stroke, which caused mobility difficulties and impaired self-hygiene, along with poorly controlled pre-existing type II diabetes mellitus. Our patient also reported intense pruritus, particularly at night. The lesions were located in characteristic scabies distributions, such as the intertriginous folds, specifically the interdigital spaces, periumbilical area, and genitocrural region. These areas are potential sites for identifying scabies via dermoscopy or histopathological biopsy.[3] Secondly, the patient also exhibited tense, pemphigoid-like bullae, arising on the erythematous skin. In our case, they were distributed across the trunk and limbs, but were most prominent and primarily located on the limbs. These bullae are considered an id reaction to the scabies infestation.[4]

Histopathological examination with Hematoxylin and Eosin (H&E) staining was sufficient for diagnosis in our case. Two biopsies were taken: one from a potential scabies burrow and one from a tense bulla. The first showed epidermal spongiosis and the presence of mites, confirming scabies. Meanwhile, the second revealed a subepidermal blister with dense inflammatory infiltrate composed predominantly of eosinophils, both within the bulla and scattered throughout the dermis, confirming a pemphigoid-like process.

The pathogenesis of bullous scabies is not fully elucidated, but several mechanisms have been proposed. The first is superinfection with *Staphylococcus aureus*, leading to bullous impetigo. The second theory proposes that enzymes from the scabies mite damage the basement membrane, leading to the exposure of hidden human BP antigens. This exposure triggers the body to produce antibodies that bind to the basement membrane zone, activating the classical complement cascade and recruiting inflammatory cells such as eosinophils and lymphocytes. The subsequent release of proteases by these cells then amplifies the damage, leading to dermo-epidermal cleavage and the clinical formation of bullae. In this model, the autoimmune response is a secondary consequence of mite-induced tissue damage. The third mechanism is the compelling theory of molecular mimicry. This proposes that antigens from the scabies mite share structural epitopes with human BP180 and BP230. The host's immune response against the mite produces antibodies that cross-react with the patient's own basement membrane, initiating an autoimmune blistering cascade. This theory is supported by Western blot studies that have detected circulating antibodies against BP180 and BP230 in patients with bullous scabies.[4]

As these bullae are subepidermal, they typically heal without scarring, leaving only post-inflammatory hypopigmentation, as seen in our patient.

As the bullous eruption is considered secondary to the scabies infection, the primary treatment focus is on eradicating the mites. We applied the classic scabies treatment protocol long used in our hospital: oral ivermectin 12 mg per week for two weeks; topical 5% permethrin spray applied from the neck down; topical diethyl phthalate applied to scabies lesions on the limbs and trunk, but excluding the genital area; and daily baths with diluted potassium permanganate. For the pemphigoid-like bullae, tense blisters were aspirated with an aseptic technique, and a potent topical corticosteroid, betamethasone dipropionate 0.064% ointment, was applied to these lesions to reduce local inflammation and promote healing. The scabietic lesions and pruritus began to improve significantly from the second week of treatment. However, new pemphigoid-like bullae continued to appear on other areas (Figure 2A), a finding inconsistent with most previous reports where anti-scabies therapy alone resolves the blistering.

Consequently, we initiated systemic methylprednisolone at 40 mg per day. The dose was rapidly tapered to 8 mg per day once the patient stopped developing new bullae. During

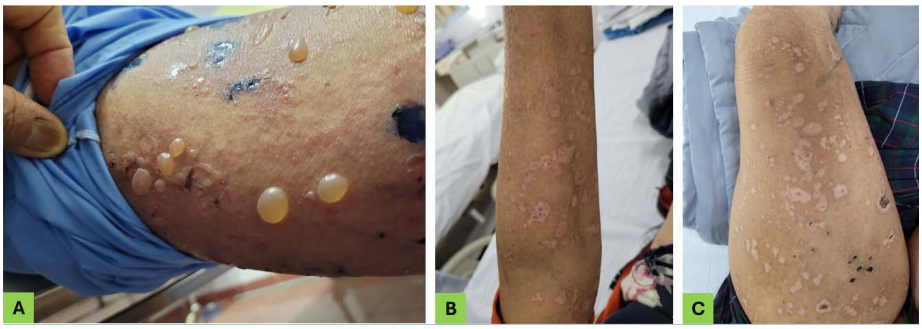

Figure 2. (A) Tense Bullae on an Erythematous Base Distributed on the Forearm, Appearing Secondarily Despite Significant Improvement of Scabies Lesions; (B, C) Post-inflammatory hypopigmented macules without scarring following bullae resolution with topical and systemic glucocorticoid therapy.

the taper and after discontinuation, there was no recurrence of bullae, which contrasts with the typical course of other autoimmune blistering diseases like classic pemphigoid or pemphigus. After 14 days of treatment, the patient achieved complete resolution, with no new bullae and existing lesions healing with hypopigmented macules but no scarring (Figures 2B & C). The use of systemic glucocorticoids in a diabetic patient required careful consideration. However, we proceeded with immunosuppression while actively managing blood glucose levels with subcutaneous insulin.

We propose that, due to the secondary immune mechanisms triggered by scabies, systemic glucocorticoids are necessary to control the bullous eruption. It is noteworthy that our patient had type II diabetes mellitus, a condition that may cause hesitation among dermatologists regarding glucocorticoid use. However, we did not delay immunosuppressive therapy and managed blood glucose levels effectively with subcutaneous insulin therapy.

In conclusion, bullous scabies should be suspected when clinical features of both scabies and pemphigoid are present. Histopathological examination with H&E staining can aid in diagnosis through biopsies of both suspected scabies burrows and bullae. Targeted scabies treatment does not always lead to the resolution of the pemphigoid-like bullae, topical and systemic glucocorticoid therapy should be considered to control persistent blistering even in complicated patient populations.

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Competing Interests

No conflict of interest to declare.

Ethical Approval

We obtained informed consent from the patient to publish this case report.

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