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Bullous Pemphigoid in a Young Female: A Case Report

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ABSTRACT

Introduction: Bullous pemphigoid (BP) is the most common autoimmune subepidermal blistering disease, mediated by autoantibodies against hemidesmosomal proteins BP180 and BP230, which activate the complement system, recruit eosinophils and neutrophils, and produce subepidermal bullae. Although BP typically affects older adults, it can occasionally arise in young patients, and prompt recognition and objective monitoring of disease activity are important.

Case Description: A 22-year-old woman presented with a two-week history of pruritic, fluid-filled tense bullae over the trunk, axillae, forearms, palms, and thighs, which were unresponsive to topical betamethasone and cetirizine. Examination revealed tense, clear, and hemorrhagic bullae on erythematous macules with a negative Nikolsky's sign, and the initial Bullous Pemphigoid Disease Area Index (BPDAI) was 23 (moderate). Histopathological examination revealed subepidermal bullae with eosinophil- and neutrophil-rich infiltrates, confirming the BP diagnosis. Laboratory testing revealed thrombocytopenia underlying the hemorrhagic lesions, and oral mucosal involvement was observed during follow-up. She was treated with systemic and topical corticosteroids, an antihistamine, and supportive care. The BPDAI progressively decreased to 17 at two weeks and 7 at three weeks, with no new lesions.

Conclusion: Oral and topical corticosteroids were effective in treating moderate bullous pemphigoid in this patient. Serial BPDAI scoring provides a practical and objective measure of treatment response and supports timely dose adjustment.

Keywords

Autoimmune Blistering Disease, BPDAI, Bullous Pemphigoid, Corticosteroid, Young Female

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INTRODUCTION

Bullous pemphigoid (BP) is mediated by autoantibodies directed against a transmembrane protein (BP180) and hemidesmosomal plaque protein (BP230), which activate the complement system, recruit inflammatory cells (mainly eosinophils and neutrophils), and produce subepidermal bullae. Linear IgG and C3 deposits along the basement membrane zone can be demonstrated using direct immunofluorescence (DIF) [1,2]. BP most often manifests around the age of 80 years; however, it can also affect young people, particularly in drug-induced BP and children. Women aged < 75 years are more frequently affected than men, although men continue to have a higher incidence of BP [3]. The diagnosis of bullous pemphigoid is based on clinical features, histology, and immunofluorescence [4]. A comprehensive dermatological examination of the skin and mucosa forms part of the clinical evaluation of patients with suspected BP, and disease activity is commonly quantified using the Bullous Pemphigoid Disease Area Index (BPDAI) [5].

Treatment depends on several factors, including the extent of the disease and patient comorbidities. Localized bullous pemphigoid can be treated successfully with topical corticosteroids, whereas more extensive or generalized lesions usually require systemic corticosteroids, which remain the mainstay of therapy.

Although BP is often self-limiting, it may persist for several years, generally fewer than five years. Pruritus can substantially impair the quality of life, and extensive erosive lesions place patients at risk of severe fluid and electrolyte loss, impaired thermoregulation, and infection [5]. We report a case of a 22-year-old woman diagnosed with bullous pemphigoid, in whom the BPDAI score decreased after three weeks of treatment.

CASE DESCRIPTION

A 22-year-old woman presented to the Dermatology, Venereology, and Aesthetics Polyclinic of Universitas Sumatera Utara Hospital on 4 December 2023 with a chief complaint of fluid-filled blisters accompanied by itching on the abdomen, axillae, forearms, palms, and thighs for the preceding two weeks. She reported that one to two blisters had initially appeared suddenly and healed spontaneously, after which red blisters developed on the left wrist, some of which ruptured due to scratching. She had previously been treated at a primary health center with topical betamethasone twice daily and cetirizine 10 mg once daily, without improvement. She denied any history of drug allergy, family history of similar complaints, or previous episodes of the same disorder.

On physical examination, the patient was moderately ill but fully conscious, with good nutritional status: body weight, 55 kg; height, 153 cm; blood pressure, 120/80 mmHg; temperature, 36.5°C; pulse, 88/min; and respiratory rate, 20/min. Dermatological examination revealed erythematous macules with multiple tense, clear fluid-filled bullae in the right and left axillary regions, the left shoulder, the right anterior forearm, and the right thigh; tense hemorrhagic bullae in the left anterior forearm; erythematous macules with vesicles on the plantar surface of the hand; and erythematous macules with papules on the abdomen (Figure 1). Nikolsky's sign was also negative.

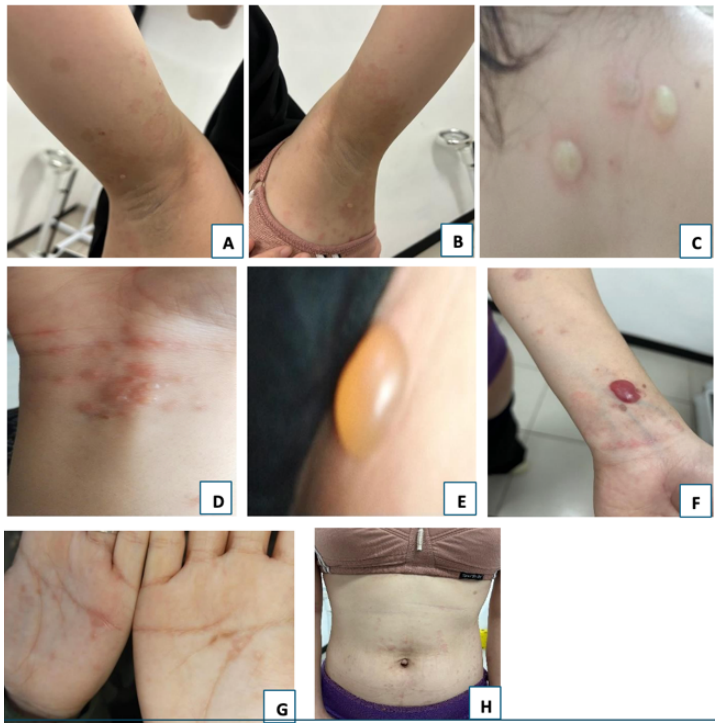

Figure 1. Clinical photographs were taken during the first visit. Erythematous macules with multiple tense, clear fluid-filled bullae in the right and left axillary regions, left shoulder, right anterior forearm, and right thigh (A–E); tense hemorrhagic bullae in the left anterior forearm (F); erythe macules with vesicles on the plantar surface of the hand (G); and erythe macules with papules on the abdomen (H).

The differential diagnoses were bullous pemphigoid, linear IgA bullous dermatosis, and dermatitis herpetiformis. The calculated BPDAI score was 23, corresponding to moderate disease, and a working diagnosis of moderate bullous pemphigoid was made. An excisional biopsy for histopathology and laboratory investigations was planned (Table 1). While awaiting histopathology, the patient received cetirizine 10 mg once daily, gentamicin ointment twice daily on dried bullae and eroded areas, and 0.9% NaCl compresses for

15 minutes every 6 hours on intact bullae. She was advised not to scratch, to adhere to therapy, and to return in one week.

Table 1. Laboratory Examination Results

No	Examination	Result	Reference Range
1	Hemoglobin	9.10 g/dL	12–16 g/dL
	Hematocrit	31.6%	36–47%
	Erythrocytes	5.19 ×10⁶/μL	3.8–5.2 ×10⁶/μL
	Leukocytes	7,290 /μL	4,000–11,000 /μL
	Platelets	110,000 /μL	150,000–450,000 /μL
2	Random blood glucose	100 mg/dL	<100 normal; 100–199 indeterminate; ≥200 suggests diabetes

Note: The results show anemia and thrombocytopenia, the latter of which underlies the hemorrhagic bullae observed clinically.

At the first follow-up, one week later, the complaint had not improved, and new tense hemorrhagic bullae had appeared on the tongue, while the lesions on the left wrist and several others had deflated and dried. Histopathology showed a supradermal (subepidermal) vesicle in the sub-basal region with an empty lumen; elsewhere, among fibrocollagenous connective-tissue stroma, hair follicles and cutaneous adnexal glands showed normal nuclear morphology. There was no evidence of malignancy, and the findings were consistent with those of bullous pemphigoid (Figure 2).

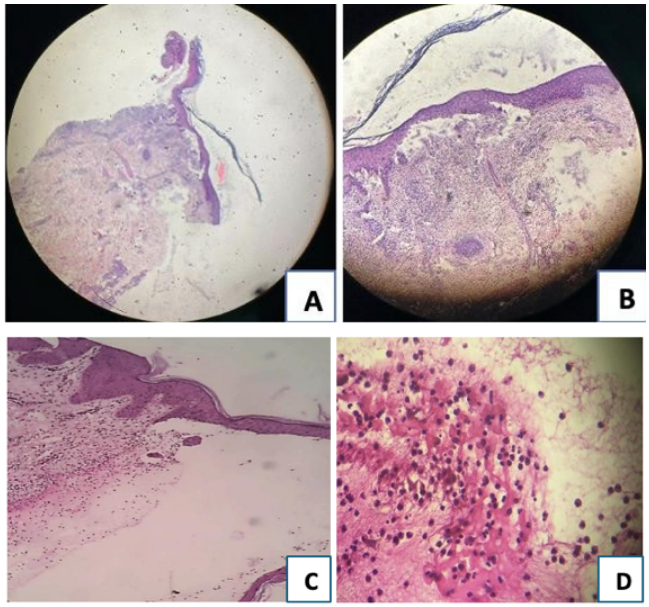

Figure 2. Histopathology of the lesional skin. The specimen comprised the epidermis and dermis (A). A supradermal vesicle is visible in the sub-basal region with an empty lumen (B). Subepidermal bullae separate the epidermis from the dermis (C). Within the bullous lumen, eosinophils and neutrophils were observed (hematoxylin–eosin, ×400) (D).

Dermatological examination at this visit revealed multiple hemorrhagic bullae on the right side of the tongue, erythematous macules with partial hyperpigmentation, erosion, and excoriation in both axillae, erythematous macules with bullae on the left shoulder, erythematous macules with partial hyperpigmentation on the anterior forearms and thighs, erythematous macules with papules, erosion, and excoriation on the plantar hand, and hyperpigmented macules with papules on the abdomen (Figure 3). Skin scraping with 10% potassium hydroxide revealed no hyphae or spores, and a Tzanck smear of the vesicles showed no multinucleated giant cells. Based on the history, physical and dermatological examination, and supporting investigations, the patient was diagnosed with moderate bullous pemphigoid (BPDAI score 23). She was then treated with methylprednisolone 4 mg (6–5–0 tablets) for seven days, followed by tapering, cetirizine 10 mg once daily, desoximetasone 0.25% cream twice daily, and gentamicin ointment twice daily on the ruptured bullae and eroded areas. At the second follow-up, two weeks after therapy, the complaints had improved, with

no new blisters, deflation and drying of existing lesions, reduced itching, and no pain. The BPDAI score at the time of this visit was 17. Therapy was continued with methylprednisolone 4 mg (5–5–0 tablets) for seven days, followed by tapering, cetirizine 10 mg once daily, desoximetasone 0.25% cream twice daily, and gentamicin cream twice daily on the ruptured blisters and eroded areas. The prognosis was quo ad vitam dubia and bonam, quo ad functionam dubia and bonam, and quo ad sanationam dubia and bonam.

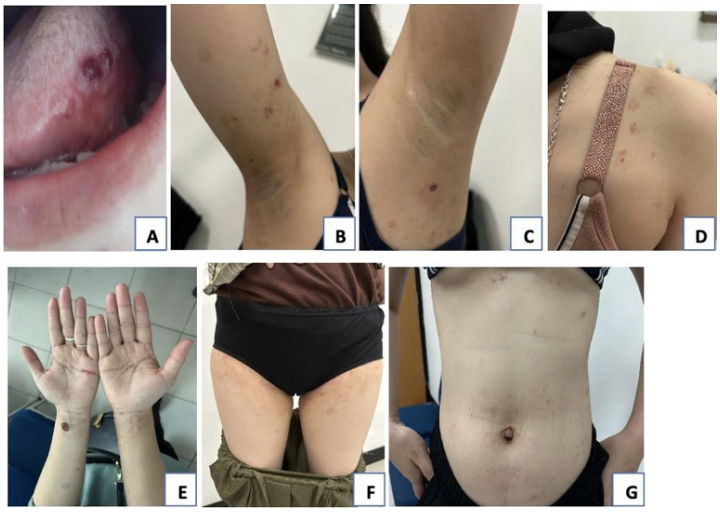

Figure 3. Clinical photographs were taken at the first follow-up (one week after therapy). Multiple hemorrhagic bullae on the right side of the tongue (A), erythematous macules with partial hyperpigmentation, erosion, and excoriation in the right and left axillary regions (B, C), erythematous macules with blisters on the left shoulder (D), erythematous macules with partial hyperpigmentation on the anterior forearms and thighs (E, F), and hyperpigmented macules with papules on the abdomen (G).

DISCUSSION

Bullous pemphigoid is a classic example of an antibody-mediated disease. The principal targets of pathogenic IgG1 and IgG4 autoantibodies are hemidesmosomal proteins BP180 and BP230 within the dermoepidermal junction. The binding of autoreactive IgG to BP180 and BP230 disrupts hemidesmosomes, exposes neo-antigens, and initiates a cascade that releases cytokines, proteases, and lysosomal enzymes. Complement activation, mast cell degranulation, and the release of leukotrienes, platelet-activating factor, tumor necrosis factor, and other cytokines are thought to result from the synergy between autoimmune IgE and self-reactive IgG1 and IgG4. Proteolytic enzymes released by neutrophils and eosinophils recruited to the lesion degrade adhesion molecules and produce subepidermal blisters [6].

Young-onset bullous pemphigoid can be triggered by factors such as medications, infections, environmental exposure, stress, autoimmune diseases, and genetic predisposition. In young adults, changes in the immune system, such as increased reactivity or regulatory dysfunction, can lead to the formation of autoantibodies against self-tissues, which may be precipitated by infection, stress, or other autoimmune disorders. Hormonal fluctuations during youth may also modulate the immune response and contribute to an increased risk of developing autoimmune diseases [7]. Antibiotic use can also trigger BP in young patients through incompletely understood mechanisms. For example, sulfhydryl-containing drugs can directly impair T-suppressor cell activity, promoting autoantibody production and immune dysregulation. Thiol drugs may cause non-immunogenic injury at the dermoepidermal interface, exposing the immune system to novel antigens [8]. Under these triggers, lesions may arise in unexpected locations [9,10]. Tense bullae and severe generalized pruritus are common features of immunobullous diseases, although bullous pemphigoid has a broad clinical spectrum [11]. The initial manifestation is usually a widespread, itchy bullous eruption [12]. The bullous stage is characterized by vesicles and bullae arising predominantly on erythematous skin, along with urticarial papules and occasionally annular plaques. Atypical, non-bullous variants of generalized BP have also been described, including non-bullous (erythrodermic) BP, papular or nodular lesions without blisters, intertriginous vegetative plaques (pemphigoid vegetans), grouped symmetrical tense vesicles

resembling dermatitis herpetiformis (vesicular pemphigoid), and toxic epidermal necrolysis-like BP [12]. These features are consistent with the findings of the present study.

This patient also exhibited hemorrhagic bullae. In bullous pemphigoid, vesicles and bullae may be hemorrhagic and appear in clusters or be widespread [3]. Other studies have noted that hemorrhagic bullous lesions are uncommon and, in most clinical settings, signal serious disease because they are associated with severe coagulation or platelet disorders, the underlying cause of which —often an autoimmune disorder or infection—is not always readily identifiable [13]. This patient's laboratory results showed thrombocytopenia, which provided the basis for the hemorrhagic bullae. The diagnosis of bullous pemphigoid can be confirmed by direct or indirect immunofluorescence, with a specificity approaching 100% [1]. The patient was diagnosed with moderate BP based on a BPDAI score of 23. The score then decreased steadily, indicating a good therapeutic response, reaching 17 (mild) at the second visit. The BPDAI severity classification comprises three grades: mild (BPDAI ≤ 19), moderate (BPDAI 20–56), and severe (BPDAI ≥ 57) [14]. Glucocorticoids are the cornerstone of treatment; high-dose systemic corticosteroids are used to induce remission, followed by dose tapering [15]. Steroid-sparing adjuvants (including azathioprine, doxycycline, mycophenolate mofetil or mycophenolic acid, methotrexate, dapsone, cyclosporine, and rituximab) may be added when glucocorticoid monotherapy fails to control the disease or when relapse occurs during dose reduction [4]. At the one-week follow-up, new tense hemorrhagic bullae appeared on the tongue, while the other lesions deflated and dried. Mucous membrane pemphigoid (MMP) is a rare vesiculobullous condition with varied clinical manifestations, and its variants overlap with bullous and cicatricial (benign mucous membrane) pemphigoid [16]. Mucosal lesions occur in approximately 10% of patients with bullous pemphigoid and are almost always confined to the oral mucosa [2]. Systemic corticosteroid therapy was administered at an initial dose of 0.5 mg/kg/day for seven days, followed by tapering. Three weeks after starting therapy, the patient reported no new lesions, and the recalculated BPDAI was 7 (mild).

Early mortality in untreated patients has been reported to be as high as 25%. The factors behind the mortality differences between Europe and the United States remain unclear; recent studies have shown a slow, steady increase in mortality over two decades in the United States that cannot be explained by disease severity alone [2].

CONCLUSION

Oral and topical corticosteroid therapies appear to be effective in treating moderate bullous pemphigoid, even in young patients. Serial BPDAI scoring offers a practical, objective measure of disease activity and treatment response and can guide timely dose adjustments.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

C.P.H. was responsible for the study concept, patient management, data interpretation, and critical revision of the manuscript for important intellectual content. E.W. contributed to patient management, data acquisition,

the literature review, and drafting of the initial manuscript. Both authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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