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Case Report

Bullous Pemphigoid Simulating Pseudoporphyria Case Report

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Abstract: Bullous pemphigoid is an autoimmune disease that predominantly affects older adults. It is characterized by autoantibodies against the hemidesmosome proteins BP180 and/or BP230, and elevated levels of serum IgE, eosinophils, and chemokines in skin lesions. It typically presents pruritic, eczematous, excoriated, urticarial-like lesions; the treatments include high-potency topical corticosteroids, oral prednisone, immunosuppressants, doxycycline, dapsone, B-cell depletion therapy, intravenous immunoglobulin, as well as biologic drugs. In this report, we present the case of 63- year-old patient with a previous diagnostic of systemic arterial hypertension who developed multiple tense vesicle and bullas at photo exposed skin areas.

Keywords: Dermatology, Bullous Pemphigoid, Pseudoporphyria.

INTRODUCTION

Bullous pemphigoid is an autoimmune disease that predominantly affects older adults. It is characterized by autoantibodies against the hemidesmosome proteins BP180 and/or BP230, type 2 inflammation, and elevated levels of serum IgE, eosinophils, and chemokines in skin lesions [1, 2].

The disease manifests with a non-bullous prodromal phase characterized by pruritic, eczematous, excoriated, urticarial-like lesions that may progress to a generalized pruritic bullous eruption; however, some patients remain in the eczematous and urticarial stage without developing blisters [2].

The drug-associated subtype shares clinical and histological features identical or similar to those of the idiopathic form of bullous pemphigoid; the use of gliptins, PD-1/PD-L1 inhibitors, loop diuretics, penicillin, and derivatives has been linked [3, 4].

Some of the unusual variants of bullous pemphigoid include: Dyshidrosiform pemphigoid, vegetans pemphigoid, nodular pemphigoid, vesicular pemphigoid, papular pemphigoid, eczematous pemphigoid, erythrodermic pemphigoid, and lichen planus pemphigoid.

Recommended treatments include high-potency topical corticosteroids, oral prednisone, immunosuppressants, doxycycline, dapsone, B-cell depletion therapy, intravenous immunoglobulin, and biologic agents [2].

CASE PRESENTATION

A 63-year-old male with a history of systemic arterial hypertension and chronic lumbago. He had smoking history of 15 years, 3 cigarettes per day. His current condition begin with itching and flaking skin on the back of both hands; He applied lemon and coconut cream, and washed with chlorine-based disinfectant cleaners and seawater, with no improvement for approximately 1 week, afterwards he developed multiple tense vesicle and bullas in the same area, some with clear contents, others hemorrhagic, with a tendency to merge on an erythematous base (Figure 1and 2); sparing the trunk, palms, and soles (Figure 3).

A 0.5 mm skin punch biopsy was taken from his left hand, revealing evidence of a subepidermal blister with eosinophilic infiltrate and histopathological changes consistent with bullous pemphigoid (Figure 4,5,6).

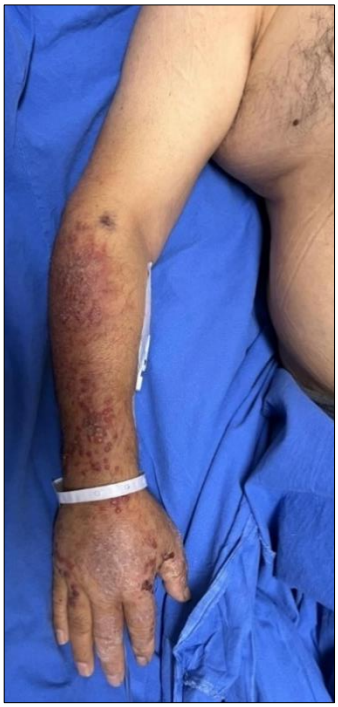

Figure 1: Clinical image of the right arm showing vesicles, erythema and dried blood crusts

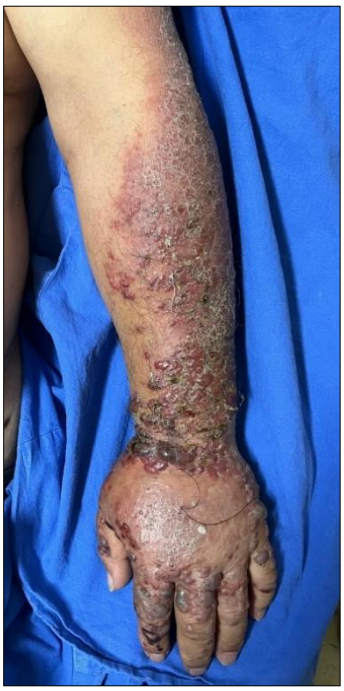

Figure 2: Clinical image of the left arm showing vesicles, erythema and dried blood crusts

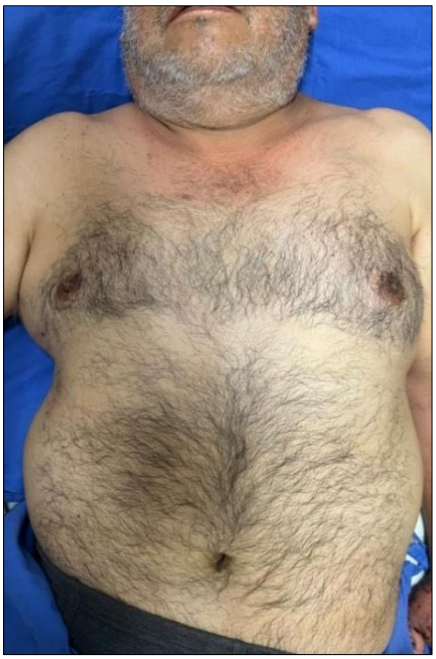

Figure 3: Clinical image of the chest and abdomen without skin lesions, only with photodamage at the V of the neck

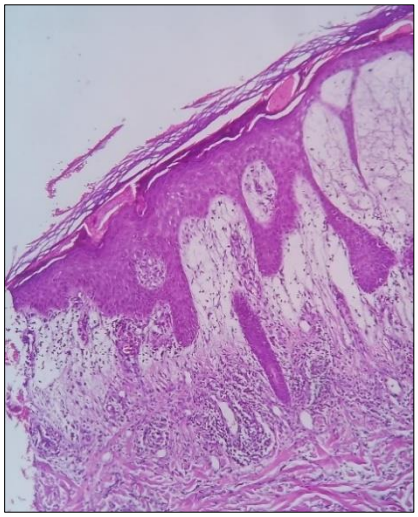

Figure 4: Histopathological features of skin punch biopsy that's show subepidermal blister with internal inflammation. The epidermis shows mild spongiosis. (Hematoxylin & eosin 10x)

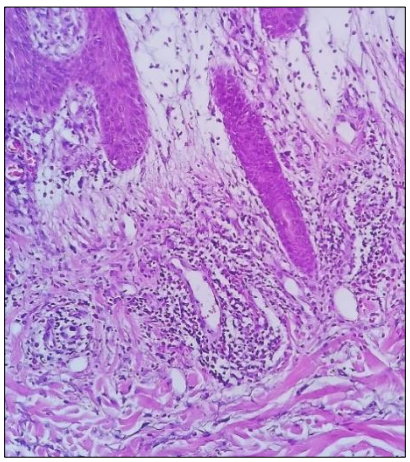

Figure 5: Perivascular inflammatory infiltrate with the presence of eosinophils. (Hematoxylin & eosin 20x)

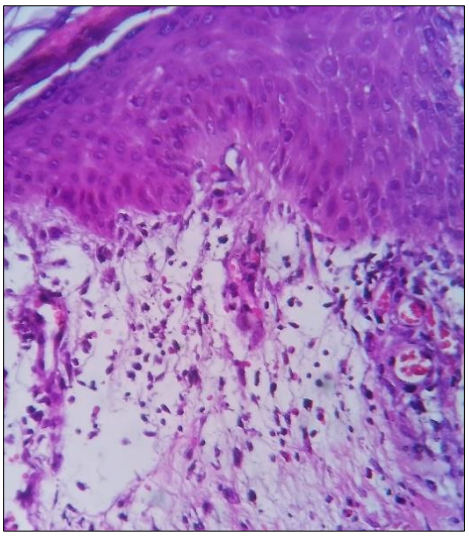

Figure 6: Subepidermal vesicle with presence of eosinophils (Hematoxylin & eosin 40x)

DISCUSSION

Bullous pemphigoid (BP) is an autoimmune blistering disease with elements of type 2 inflammation that primarily affects older people. BP is mainly characterized by IgG autoantibodies to BP180 and/or BP230, which are components of hemidesmosomes involved in dermoepidermal cohesion [1-4].

In most patients with PB, the disease manifests with a non-bullous prodromal phase characterized by pruritic, eczematous, excoriated, urticarial-like lesions that may progress to a generalized pruritic bullous eruption; however, some patients remain in the eczematous and urticarial stage without developing blisters [2, 3].

Infiltration of inflammatory cells (including mast cells, neutrophils, and eosinophils) is a consistent feature of cutaneous lesions in BP, and studies suggest that these cells play an important role in blister formation in BP [3-5].

Studies show that eosinophils are required for anti-BP180 IgE-mediated skin blister formation, and that eosinophils participate in dermoepidermal junction cleavage through the generation of reactive oxygen species, the release of eosinophilic granules, and the formation of eosinophil extracellular traps [6-9].

The natural history of BP associated with drugs is relatively unclear, although two divergent courses have been identified [10-12]. These include an acute, self-limited form, characterized by definitive resolution after withdrawal of the suspected drug [12].

Pseudoporphyria is a relatively rare skin disease that usually occurs in the context of significant sun exposure [6]. Most cases described in the literature are related to the use of various drugs, especially non-steroidal anti-inflammatory drugs (NSAIDs) derived from propionic acid (ibuprofen, ketoprofen, oxaprozin, naproxen, and nabumetone), antibiotics (nalidixic acid, tetracyclines), and diuretics, particularly sulfur-containing ones such as chlorthalidone, bumetanide, and furosemide. Other examples of drug-induced disease include retinoids (isotretinoin and etretinate), 5-fluorouracil, flutamide, oral contraceptives, diclofenac, and vitamin B6 [7]. Differential diagnoses should be considered due to clinical findings in the non-bullous phase of pemphigoid can be nonspecific and often resemble dermatoses such as drug reactions, contact dermatitis, prurigo (simple and nodular), urticarial dermatosis, and scabies. This group of disorders is usually distinguished based on clinical history, pathological features, and negative immunofluorescence findings [9]. The presence of bullae increases the possibility of arthropod bites, allergic contact dermatitis, Stevens-Johnson syndrome, bullous drug eruptions, paraneoplastic pemphigoid, dermatitis herpetiformis, among others.

CONCLUSIONS

This is particularly relevant because this is a possible case of atypical bullous pemphigoid, as it presented on sun-exposed skin, mimicking pseudoporphyria. This raises the question of whether it is related to other subepidermal blistering diseases with eosinophilic infiltrates, such as epidermolysis bullosa and EAS. Detecting these cases will allow for the implementation of potentially better targeted therapies for patients.

Conflict of Interest: The authors declare that there are no conflicts of interest at the time of publication of this article.

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