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Case Report

A Rare Presentation of the Deck-Chair Sign in a Patient with Bullous Pemphigoid: A Case Report

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Abstract: The deck-chair sign (DCS) is a pattern of skin-fold sparing in the setting of generalized erythematous papules or plaques. Since its description in 1984, the DCS was believed to be unique to papuloerythroderma of Ofuji. While infrequent, there have been reports of the DCS in a variety of other systemic and dermatologic conditions. Herein, we report a case of the DCS in a 79-year-old male with bullous pemphigoid.

Keywords: deck-chair sign; bullous pemphigoid; papuloerythroderma of Ofuji

1. Introduction

Bullous pemphigoid (BP) is an autoimmune subepidermal blistering disorder characterized by pruritic urticarial and eczematous plaques followed by tense bullae [1–3]. While the diagnosis of BP is largely based on a combination of clinical, histopathologic, and immunologic criteria, atypical variants exist, emphasizing the importance of clinical suspicion despite possible non-classic clinical presentations [1,3]. In 1984, the deck-chair sign (DCS) was reported as a clinical finding characterized by sparing of skin folds in the setting of generalized erythematous plaques and was initially believed to be unique to papuloerythroderma of Ofuji [4,5]. However, reports suggest this clinical sign can be observed in a broader range of systemic and dermatologic conditions [4,5]. To the best of our knowledge, two prior cases of BP presenting with a DCS-like eruption have been reported [6,7].

2. Case Report

A 79-year-old male presented with a 2-year history of recurrent, clinically suspected BP without clear histopathologic confirmation. Previous flares had been empirically managed with clobetasol propionate ointment, rituximab, prednisone, and azathioprine. His medical history was otherwise significant for hypertension controlled with atenolol.

Cutaneous examination revealed a generalized, symmetric eruption of confluent, pruritic erythematous papules coalescing into plaques on the trunk and extremities with well-demarcated sparing of the inframammary and axillary skin folds, reminiscent of the DCS (Figure 1A). Additionally, there were dozens of vesicles and bullae on the palms (Figure 1B) and medial and plantar aspects of the feet (Figure 1C). At the time of maximum skin involvement, the total body surface area (BSA) was approximately 54%, with involvement of the back (18% BSA), chest (7% BSA), abdomen (7% BSA), bilateral upper extremities (4% BSA), and lower extremities (18% BSA).

Based on clinical findings, the differential diagnosis included papuloerythroderma of Ofuji, BP, linear immunoglobulin A bullous dermatosis (LABD), other neutrophilic dermatoses, epidermolysis bullosa acquisita, contact dermatitis, or an infectious etiology. While papuloerythroderma of Ofuji was suspected based on the presence of the DCS, the patient denied any constitutional symptoms and there was no known history or symptoms of lymphoma, leukemia, solid organ malignancy, human immunodeficiency virus, or hepatitis C.

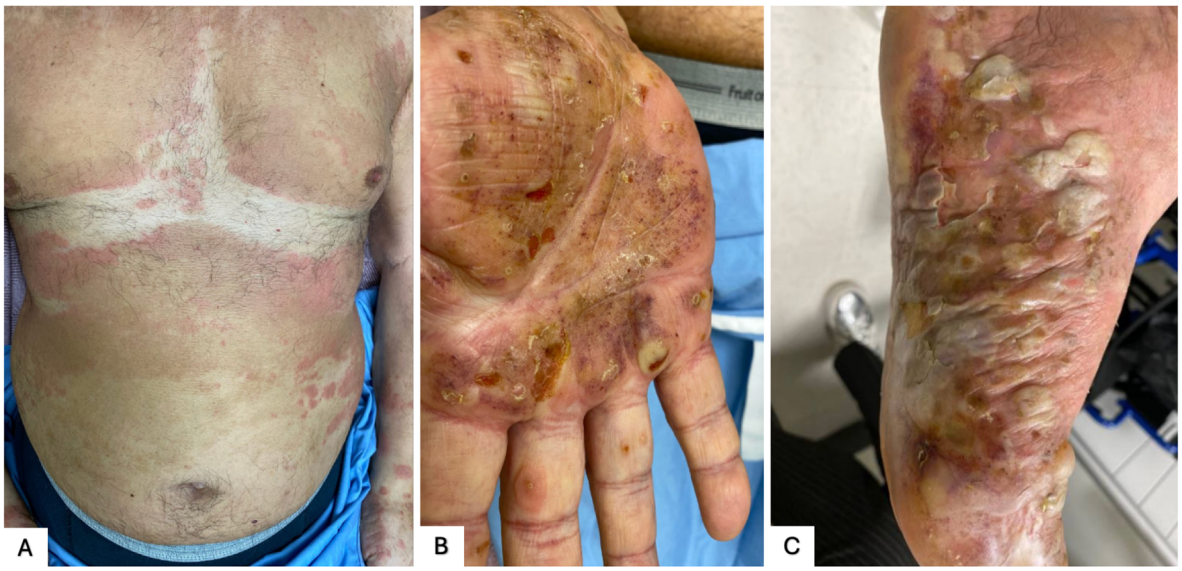

Figure 1. (**A**) Well-demarcated sparing of the inframammary folds, resembling the deck-chair sign. (**B**) Multiple vesicles and small bullae in various stages of evolution on the hands. (**C**) Multiple bullae in various stages of evolution on the medial and plantar surfaces of the feet.

A wound swab for bacterial culture was negative and two punch biopsies from the right arm were obtained. Histopathologic examination demonstrated spongiotic dermatitis and focal subepidermal bulla with prominent eosinophilic infiltration, compatible with BP. Direct immunofluorescence (DIF) was unfortunately negative on two occasions due to active treatment and processing error, respectively. Laboratory investigations revealed an eosinophil count of 4.8×10^9/L (normal reference range: 0–0.7×10^9/L) and a total serum immunoglobulin E (IgE) level of 4005 kU/L (normal reference range: <120 kU/L). Enzyme-Linked Immunosorbent Assay (ELISA) was also completed, demonstrating positive BP180 antibodies (810 IU/mL) and positive BP230 antibodies (110 IU/mL). Although DIF was negative twice, the overall clinical presentation, histopathologic and laboratory findings, and serologic profile remained most consistent with BP. The patient was initiated on oral dexamethasone, intravenous immunoglobulin, and clobetasol propionate ointment, resulting in near complete resolution of symptoms and skin findings at one month follow-up.

3. Discussion

Since its description in 1984, the DCS was thought to be unique to papuloerythroderma of Ofuji, a rare dermatologic condition characterized by diffuse erythrodermic papules coalescing into plaques with sparing of the natural skin folds [4,5]. Several hypotheses have been proposed to explain the possible etiology of the DCS, including an association with age-related skin laxity where mechanical compression of skin folds influences apoptosis and the release of inflammatory mediators, sparing these regions from the eruption [4,8]. Papuloerythroderma of Ofuji is most often seen in patients over the age of 55 and has been linked to a variety of triggering factors including underlying malignancies, infectious processes, and pharmacological agents [4]. In this case, the patient had no clinical history suggestive of malignancy, infection, or drug trigger for papuloerythroderma of Ofuji. Furthermore, the clinical presentation, histopathologic and laboratory findings, and serologic profile was more in keeping with BP.

While infrequently reported, the DCS has been recently described across a spectrum of dermatologic conditions including inflammatory dermatoses, cutaneous lymphomas, autoimmune disorders, infections, drug-induced reactions, acanthosis nigricans, Waldenström macroglobulinaemia, and idiopathic cases [4,5]. In our case, the DCS was observed in the context of BP, an autoimmune subepidermal blistering disorder caused by autoantibodies against BP180 and/or BP230 [1–3]. Typically the diagnosis of BP relies on clinical morphology and compatible histopathology, supported by DIF demonstrating linear deposits of immunoglobulin G (IgG) and/or complement component 3 (C3) along the dermal-epidermal junction [1–3]. These can be complemented by serologic studies such as ELISA to detect autoantibodies against BP180 and/or BP230 and indirect immunofluorescence (IIF) for IgG anti-basement membrane zone antibodies in salt-split human skin [1–3]. Additionally, eosinophilia and elevated serum IgE have been reported as useful investigations due to their role in the pathogenesis of BP [1,9]. While IIF was not completed in our case, ELISA did demonstrate positive BP180 and BP230 antibodies and the patient had eosinophilia with an elevated serum IgE.

Two prior reports suggest that BP can present with a fold-sparing eruption reminiscent of the DCS. In 2017, Hayakawa-Asai et al. [7] presented the case of a 75-year-old male with a history of gastric cancer presenting with a papuloerythroderma of Ofuji-like eruption with edematous, erythematous, vesicular papules sparing the skin folds on the trunk, compatible with the DCS. A diagnosis of BP was later confirmed with histopathological examination, chemiluminescent enzyme immunoassay, direct immunofluorescence, and a peripheral blood smear. More recently in 2023, Bhasin et al. [6] reported the DCS in a 77-year-old male presenting with multiple large tense bullae, urticarial lesions, and hyperpigmentation with well-demarcated sparing of abdominal folds, also resembling the DCS. A diagnosis of BP was later confirmed on histopathological examination and DIF. Our case further highlights the variability of clinical manifestations in the context of BP and reinforces the importance of considering BP as a potential diagnosis when a skin eruption presents with the DCS.

4. Conclusions

In conclusion, this rare presentation of the DCS in BP further expands its clinical spectrum and supports a broader possibility of differential diagnoses beyond papuloerythroderma of Ofuji. By contributing to the limited existing literature on the DCS in BP, this report highlights the importance of including BP among the conditions capable of exhibiting the DCS.

Author Contributions

K.B.: data collection, literature review, writing—original draft; C.J.: conceptualization, writing—reviewing and editing, supervision. Both authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

Ethical review and approval were waived for this case report due to its nature as a single-patient case report using de-identified clinical information.

Informed Consent Statement

Written informed consent was obtained from the patient for the publication of this case and accompanying images.

Data Availability Statement

All relevant data supporting the findings of this case report are included within the article.

Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

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