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Case Report Open Access

A Case of Psoriasis Vulgaris Coexisting with Bullous Pemphigoid and Eosinophilia in a 69-year-old Male with Diabetes Mellitus and Chronic Renal Failure

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Abstract

A 69-year-old male with CRF due to DM developed PV. This patient suddenly developed bullas and erosions on his arms and thighs which spread to his trunk, neck, limbs, hands and feet after a 4-year history of PV. Our diagnosis was PV coexisting with BP from the results of a skin biopsy and the results of anti-BP 180 and 230 antibodies detection. We started the systemic administration of steroid tablets, but we did not elect immunosuppressive therapies (cyclosporine and methotrexate) because of his hypertension and the risk of malignancy. The treatment of the patient's DM was modified from oral medicine to insulin injections. After three weeks of treatment, his laboratory results showed the eosinophilia (41.3%), and we increased the dose of steroids (betamethazone 2 mg/day) and anti-histamine tablets. After the 1.5 months of his hospitalization, his cutaneous condition and eosinophilia improved. There could be as many as 100 cases of PV coexisting with BP, and this is a rare case of PV complicated with BP and eosinophilia.

Keywords: Psoriasis vulgaris; Bullous pemphigoid; Eosinophilia; Coexisting; Diabetes mellitus; Chronic Renal failure

Introduction

Psoriasis vulgaris (PV) is a Th1-dependent chronic cutaneous disease. The worldwide morbidity rate of PV is approximately 2% [1]. PV is often treated using ultraviolet (UV) radiation, but this is a trigger of bullous pemphigoid (BP). BP is a subepidermal blistering disease [2] that generally occurs in elderly patients (more than 60 years old) [3]. BP is related to a variety of diseases, and it can be difficult to treat due to comorbidities.

PV and BP are very often seen coexisting. Over 80 cases of PV coexisting with BP have been observed [4], and there could be as many as 100 cases of this comorbidity at present. Here, we show a rare case of PV complicated with BP and eosinophilia in a patient with diabetes mellitus (DM) and chronic renal failure (CRF).

Case

A 69-year-old male developed PV on his arms, legs, trunk and scalp when he was 65 years old. This patient has been treated with renal dialysis for CRF due to DM since he was 66 years old. We had treated his PV using Dovobet ointment (calcipotriol hydrate and betamethasone dipropionate compounding ointment), steroid

ointment and steroid lotion without using tumor necrosis factor (TNF)-alpha antagonists or UV radiation for 6 months, and his PV was under good control. He suddenly developed bullas and erosions (Figure 1) on his arms and thighs which spread to his trunk, neck, limbs, hands and feet after a 4-year history of PV. The results of a skin biopsy (affected bulla area) showed a subepidermal blister with infiltration of eosinophils, neutrophils and lymphocytes into the dermis (Figure 2). The results of anti-BP 180 and 230 antibodies detection showed more than 1000 (<9) and 11 (<9), respectively. Our diagnosis was PV coexisting with BP in a 69-year-old male with DM and CRF. We started the systemic administration of steroid tablets (methylprednisolone 12 mg/day) while the patient was hospitalized after the patient was informed of the adverse side effects of oral steroids. At first, we did not elect immunosuppressive therapies (cyclosporine and methotrexate) because of his hypertension and the risk of malignancy. The treatment of the patient's DM was modified from oral medicine to insulin injections. After three weeks, his laboratory results showed an increase in the number of eosinophils to 41.3% (0-6%), and we increased the dose of steroids (betamethazone 2 mg/day) and anti-histamine tablets. There were no significant changes in his chest x-ray results. During the 1.5 months of his hospitalization, no adverse side effects such as secondary infection, postural psoriasis, or others due to the oral steroid were shown. He was discharged from the hospital after his cutaneous condition and eosinophilia improved.

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Figure 1: Clinical findings of bullous pemphigoid on his right arm.

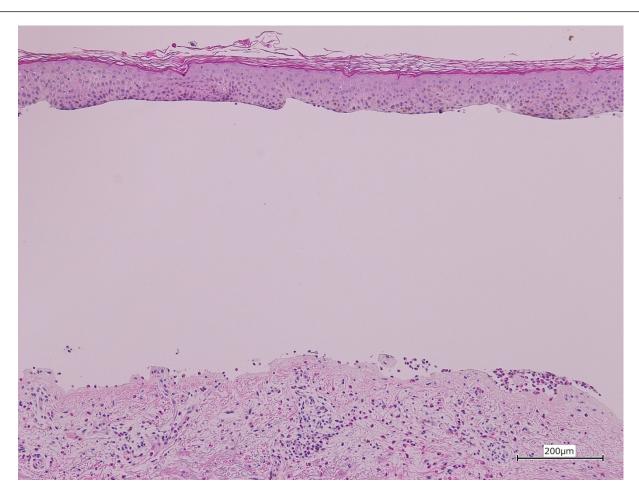


Figure 2: Histopathological findings of his bulla.

Discussion

The etiology of BP in PV patients is unknown, but their associations with autoantibodies against type 17 collagen [5] and a 200-kDa protein [6] of the lower lamina lucida have been reported. Both 180-kDa and 230-kDa IgG antibodies against hemidesmosome are detected in more than 90% patients with BP. The reasons of developing these antibodies are unknown, but these antibodies are useful for the diagnosis and follow-up of BP.

A previous report found that DM, rheumatoid arthritis, ulcerative colitis, and dermatomyositis increase the prevalence of BP, and that there is no relation between BP and malignant conditions [7]. Wilczek [8] and Mehdi [9] found that the diseases that can be comorbid with BP are psoriasis, autoimmune diseases, DM, dementia cerebrovascular events and Parkinson's disease. Mehdi [9] reported that psoriasis-induced BP was a sign of psoriasis activity. Psoriasis precedes BP, but the opposite diagnosis is rare [8]. The triggers of BP are trauma, insect bite, injection, radiation, burn, topical steroid, and others, but none of these triggers were identified in our patient. Ren [10] reported that BP was associated with renal failure. Other reports [11,12] found that BP was related with dialysis or renal complications. The cellulose membrane used for dialysis may be a factor in causing BP by activating eosinophils of the blood and the skin lesion [13]. Our case is an elderly patient and has developed DM and psoriasis and undergoes dialysis for

CRF, all of which are closely related to the appearance of BP. His PV is under good control, and the triggers of his BP are unknown, although his PV might be in an active phase beyond what is shown by the clinical findings and eosinophilia. The risk factors for developing BP in psoriasis cases include being an elderly person, developing DM and renal complications, an increase in the number of eosinophils in the blood, infiltration of the skin, and high titers of both 180 kDa and 230 kDa IgG antibodies.

TNF-alpha antagonists used for the treatment of psoriasis can cause eosinophilia [14]. Sodemoto [13] reported that the cellulose membrane used for dialysis may be related with the activation of eosinophils of the blood and the skin lesion in BP. Previous reports showed that eosinophilia is associated with dialysis [15-18].

In this case, eosinophilia occurred under treatment with steroids, although this patient did not experience any of the triggers for increasing eosinophils. Mansur [19] reported that the prevalence of psoriasis patients with eosinophilia was significantly higher than that of basal cell carcinoma, and eosinophils are associated with the pathogenesis of types or severe forms of psoriasis. Rifaioglu [20] found that the number of eosinophils in BP patients was significantly higher than that in control subjects. Messingham [21] reported that the number of eosinophils is related with the levels of BP 180 IgE than with that of BP180 IgG, and the BP180 IgG level is correlated with

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disease severity. His BP could be associated with PV and DM, and the high titers of anti-BP 180 antibodies and eosinophilia related with dialysis affected the severity of his PV and BP. His eosinophilia might have been caused by the high BP 180 IgG level and the severity of the PV. The number of eosinophils and the serum level of BP 180 IgG can be indicators of BP in elderly PV patients. Elderly individuals with PV in combination with DM or renal complications should undergo a skin biopsy and blood tests (eosinophil number and BP180 and 230-kDa IgG levels).

Oral steroids, azathioprine, methotrexate, intravenous Ig, mycophenolate mofetil, and rituximab have been demonstrated to be effective against BP [22]. It is difficult to treat cases such as ours, in which the treatment is limited by the advanced age of the patient and the presence of DM, hypertension and CRF. In our case, not only PV and BP were present, but so was eosinophilia, and we used oral steroids after informing the patient about their possible adverse side effects and receiving his informed consent. In the future, decreasing the dose of oral steroids will depend on the improvement of his BP and adverse side effects of the oral steroids (acne, osteoporosis, infection, postural psoriasis, DM and others). Our case did not develop pustular psoriasis during the treatment with the oral steroid, but combination therapy (cyclosporine and steroids) is necessary to treat his PV and BP. In case of more developing his BP, it needs plasma exchanging therapy.

Conclusion

This is a rare case of PV complicated with BP and eosinophilia in a 69-year-old male with DM and CRF.

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