Case Report

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Coexistence of Psoriatic Arthritis, Vitiligo and Bullous Pemphigoid: A Case Report

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Abbreviations: BP: Bullous Pemphigoid; TSH: Thyrotropin; TG: Thyroglobulin; TT4: Total Thyroid Hormone; TT3: Total Triiodothyronine; FT4: Free Thyroxine Determination; FT3: Free Triiodothyronine; TNF: Tumour Necrosis Factor; Th: T-Helper

ABSTRACT

Psoriasis, bullous pemphigoid and vitiligo are all clinically well-characterized chronic, inflammatory skin diseases. Many case reports have described the coexistence of psoriasis and bullous pemphigoid or vitiligo. However, it is a rare case of bullous pemphigoid in a patient who has a concomitant and colocalized presentation of psoriasis and vitiligo. We experienced a 67-year-old male psoriatic patient who also had vitiligo and developed bullous lesions and erosions all over the body. The histopathology of a blistering lesion did not only reveal psoriasis-like hyperplasia model of the epidermis, but that al-so showed the subepidermal blisters and infiltration of eosinophils around the dermal superficial blood vessels. His direct immunofluorescence demonstrated linear deposits of immunoglobulin IgG and C3 at the basement membrane zone. Moreover, the patient had a colocalized presentation of psoriasis and vitiligo in the trunk. At last, joint ultra-sound revealed that there were synovitis, tenosynovitis, bone erosion and joint deformation in finger and wrist joints.

Diagnoses of psoriatic arthritis, vitiligo and bullous pemphigoid were made. The patient was treated with methylprednisolone (80mg/d), cyclophosphamide (600mg/week), and immunoglobulin pulse therapy after his admission. Three weeks after admission, the patient's lesions gradually subsided. To our knowledge, this is first report of coexistence of psoriatic arthritis and vitiligo with bullous pemphigoid.

Keywords: Psoriatic Arthritis; Vitiligo; Bullous Pemphigoid

Introduction

Psoiasis is a chronic, systemic, inflammatory, multigenic skin disease characterized by abnormal epidermal growth and differentiation, presenting as red, scaly patches, papules, and plaques. This common disease has a variable prevalence of approximately 2% [1]. Although the primary cause of psoriasis remains unclear, autoimmunity and a strong genetic basic have clear roles. In contrast, bullous pemphigoid (BP) represents a distinct autoimmune disease characterized by the presence of variable numbers of tension bullaes all over the body. The cause of the disease is not yet clear, but the role of autoantibodies against basement membrane antigens is well established. Moreover, vitiligo is a depigmenting disorder characterized by progressive epidermal melanocyte destruction. This autoimmune disease affects approximately 0.3% to 0.5% of the population and is induced by multiple genetic and environmental factor [2]. Therefore, immune factors are more or less involved in the pathogenesis of these three diseases. Many case reports have shown a significantly increased risk of BP or vitiligo in patients with psoriasis [3,4]. Here, we present a rare case of BP eruption in a psoriatic patient, who also had a history of vitiligo.

Case Report

A 67-year-old man with a 15-year history of psoriasis vulgaris and a 3-year history of psoriatic arthritis presented with an extensive eruption that had started one week previously with

disseminated erythemato squamous plaques, tense bullaes, and erosions partially superimposed on pre-existing psoriatic plaques in the double lower limb area and on the intact skin (Figure 1a & 1d). mucosal involvement was also observed (Figure 1c). In addition, the patient stated that there was obvious itching. Moreover, this patient had typical psoriatic plaques on the trunk and limbs as well as vitiliginous macules and patches for 5 years duration on the trunk and double upper limbs (Figure 2). The lesions of psoriasis and vitiligo have overlapping in the trunk. The patient did not admit to having a family history of psoriasis and vitiligo. And he had never received PUVA or narrow band UVB therapy. At last, the patient visited our hospital because of psoriatic arthritis 3 months before admission and treated with methotrexate (10mg/week). However, he had stopped the treatment of methotrexate by himself due to the improvement of psoriasis 2 months prior.

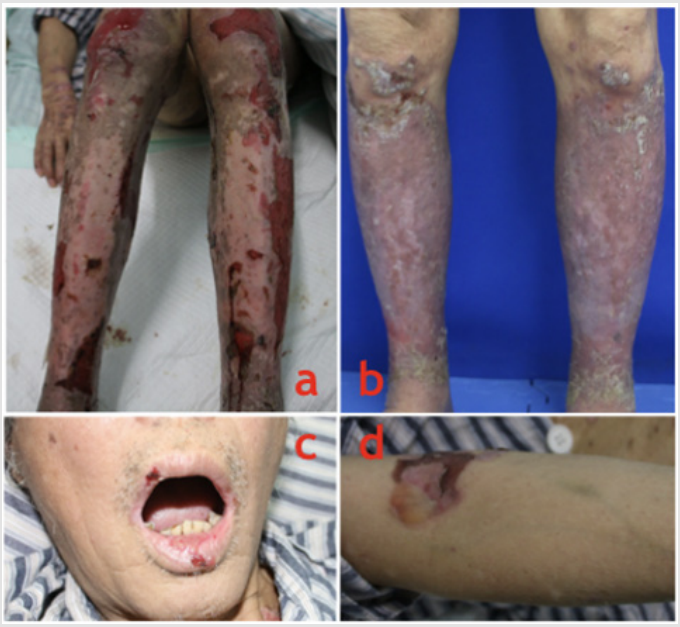

Figure 1: (a, d) Disseminated erythematosquamous plaques, tense bullaes, and erosions on the double upper and lower limb area.
(b) Disseminated erythematosquamous plaques on the double lower limb area 3 months before admission.
(c) Tense bullaes and erosions on the oral mucosa.

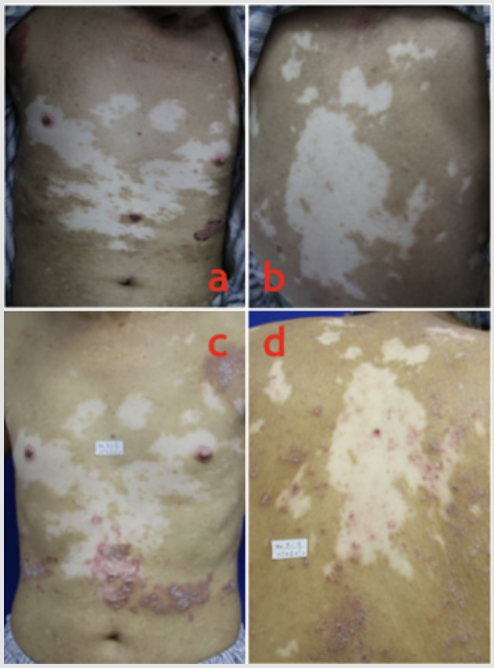

Figure 2: (a, b) Vitiliginous macules and patches on the trunk on admission.
(c, d) the lesions of vitiligo and psoriasis on the trunk 3 months before admission.

Laboratory data disclosed the following value: white blood cell count of 5860/mm^3; hemoglobin level of 12.8g/100mL; neutrophil absolute value of 4600/mm^3; lymphocyte absolute value of 850/mm^3. T cells and subpopulations: CD3+ T cells of 81.9%; CD4+ T cells of 30.5%; CD8+ T cells of 49.5%; Th/Ts ratio of 0.620; B cells of 4.17%; natural killer (NK) cells of 10.90%; NK-like T cells of 1.16%; CD3+ T cells absolute count of 896/ul, CD4+ T cells absolute count of 333/ul, CD8+ T absolute count of 542/ul. High sensitivity C reactive protein 337.06mg/L; IgG 11.29g/L; IgA 2.42g/L; IgM 0.63g/L; complement C3 1.18g/L; complement C4 0.27g/L; rheumatoid factor 11.5IU/ml; antistreptolysine O 20.0IU/mL. Thyrotropin (TSH) 1.390mLU/l; thyroglobulin (TG) 0.04ng/ml; thyroglobulin antibody 165.60IU/ml; anti-thyroid peroxidase antibody 152.90IU/mL; total thyroid hormone (TT4) 79.4nmol/L; total triiodothyronine (TT3) 0.9nmol/Ll; free thyroxine determination (FT4) 14.93pmol/L; free triiodothyro-nine (FT3) 2.14pmol/L. A biopsy specimen taken from one of the crusted lesions on the left leg on admission did not only reveal hyperkeratosis, parakeratosis, spine thickening and psoriasis-like hyperplasia model of the epidermis, but that also showed the formation of the sub epidermal blisters. The dermis showed an inflammatory infiltrate, comprising most of eosinophils, which was perivascular (Figure 3a & 3b). And direct immunofluorescence revealed that IgG and C3 deposits linearly in the basement membrane (Figure 3c & 3d). Joint ultrasound revealed that there was synovitis, tenosynovitis, bone erosion and joint deformation in finger and wrist joints. The patient was treated with methylprednisolone (80mg/d), cyclophosphamide (600mg/week), and immunoglobulin pulse therapy after his admission. Three weeks after admission, the patient's lesions gradually subsided.

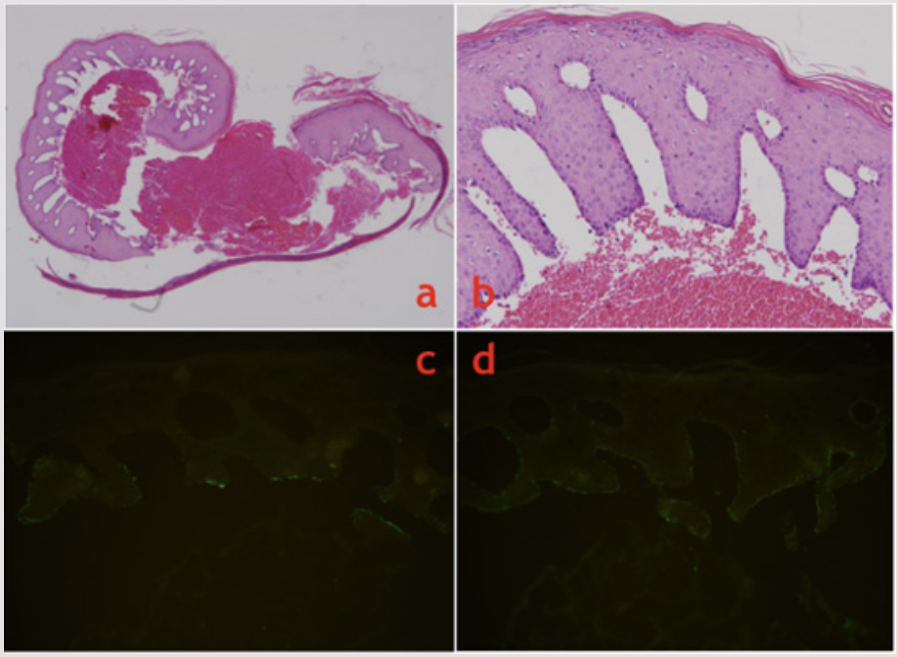

Figure 3: (a, b) Hyperkeratosis, parakeratosis, spine thickening and psoriasis-like hyperplasia model of the epidermis, and subepidermal blisters in an area of the epidermis. Dermis perivascular inflammatory infiltrate contained most of eosinophils. (c, d) IgG and C3 deposits linearly in the basement membrane. a*40. b*200.

Discussion

In our case, the histopathology of a blistering lesion showed the subepidermal blisters and infiltration of eosinophils around the dermal superficial blood vessels. His direct immunofluorescence demonstrated linear deposits of immunoglobulin IgG and C3 at the basement membrane zone. These results corresponded with BP. In addition, the patient had a 3-year history of psoriatic arthritis and a 5-year history of vitiligo. To the best of our knowledge, coexistence of psoriasis vulgaris, vitiligo and BP was described in the literature [5,6]. However, the report of coexistence of psoriatic arthritis and vitiligo with bullous pemphigoid is the first case. It is not clear whether the occurrence of three conditions is a true association. It may be suggested that psoriasis as a chronic inflammatory disease provides a particular predisposition of the immune system that, under certain circumstances, leads to an autoimmune response [7].

It has been postulated that abnormal psoriatic epidermis during the treatment with UVB radiation induces production of BP autoantibodies in susceptible psoriatic patients [8]. Muramatsu et al. [9] found that UVB radiation of the skin decreased BP antigen expression. They suggested that BP antigen is more susceptible to UVB radiation, which probably lead to configurational changes in antigen or as a secondary phenomenon, to changes resulting from damage or degenerative changes in basal cells. Moreover, the relevance of immunologic factors in psoriasis is supported by the clinical response of this disease to T-cell suppressive and modulatory therapy. The dysregulation of T-cell activity in psoriasis might result

in the induction of specific antibodies to basement membrane antigens. [10] In addition, autoimmune diseases induced by tumour necrosis factor (TNF)-targeted treatments have also been reported [11]. Although the etiopatho-genesis of psoriasis is unknown, an important role of TNF-α-inducible plasmacytoid dendritic cells and T-helper 17 lymphocytes has been described [12]. In anti-laminin c1 pemphigoid, TNF-α had been reported to stimulate neutrophils to produce and release proteolytic enzymes, including matrix (MMP)-9, which degrades matrix proteins [13]. Thereafter, the pathogenic epitope of these proteins may be exposed and may induce autoimmunity. This pathological cross-talk was also proposed as the mechanism in concurrence of the psoriasis and BP. In most patients, psoriatic lesions were treated with a broad spectrum of therapies, in combination or sequentially. Therefore, it is difficult to incriminate a single agent as a potentially causative factor in the development of BP. In our patient, he did not receive PUVA or narrowband UVB therapy, and he did not also receive the treatment of biological agents. However, it was clear that this patient had a history of suddenly interrupting the treatment of methotrexate 2 months before admission. On admission, significantly decrease in B cells (4.17% vs. 9.02-14.2%), which revealed the normal cell immunity had been destroyed, may be considered to be related with the formation of BP. In addition, considerably reduced T4:T8 ratio (0.620 vs. 1.05-2.03) involved in the clinical remission phase was consistent with clinical manifestations of our patient. To the best of our knowledge, relatively few reports of concomitant and colocalized psoriasis and vitiligo are available. The majority of case reports describe initial vitiligo followed by the development of psoriasis. However, our patient presents with vitiligo following a longstanding history of psoriasis. therefore, the colocalized nature of the concomitance and the similar origins of these diseases suggest the possibility for a relationship.

Several theories have been proposed to explain the cooccurrence of vitiligo and psoriasis. Reports of concomitant disease often describe underlying autoimmune conditions such as arthritis and thyroiditis, suggesting that these diseases may develop through similar autoimmune mechanisms. In our patient, BP had been proved by histopathology and direct immunofluorescence. In addition, thyroglobulin antibody (165.60IU/ml vs. 0-115 IU/ml) and anti-thyroid peroxidase antibody (152.90IU/ml vs. 0-34 IU/ml) showed significantly increase. Moreover, T-helper (Th)-17 cells, had been confirmed to have an important role in psoriasis and some autoimmune diseases, along with Th-1, Th-2, and regulatory T-cells (Tregs), are made from naive CD4+ helper T cells. In a study of 30 vitiligo patients and 20 matched controls, IL-17A levels in serum and lesional skin were significantly higher for vitiligo patients, for whom levels correlated with disease extent antibody. Furthermore, a recent immunohistochemical analysis revealed increased numbers of IL-17A-producing cells in colocalized psoriasis-vitiligo lesions [14]. In our case, when CD4+ T cells absolute counts (333/ul vs. 550-1440/ul) were significantly reduced, the lesions of psoriasis in the overlapping parts turned into that of vitiligo. The phenomenon revealed that vitiligo may have a more complex pathogenesis.

Conclusion

This is a rare presentation of concomitant and colocalized vitiligo and psoriasis with BP. Apart from Th-17 cells, recent hypotheses suggest a genetic factor for susceptibility to the occurrence of multiple autoimmune disorders. Further work is needed to advance knowledge on the pathophysiological mechanism of this phenomenon.

Acknowledgement

There are no conflicts of interest to declare.

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