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Case Report
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Bullous Pemphigoid in a Patient with Psoriasis and Acquired Reactive Perforating Collagenosis

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Abstract

A 57-year-old female suffering from plaque psoriasis for 25 years developed acquired reactive perforating collagenosis and then bullous pemphigoid three months later. Although there have been several reports of bullous pemphigoid in patients with psoriasis, the coexistence of all three autoimmune skin diseases in a patient has not been described. The aim of this article is to present the clinical case of the 57-year-old female with these three autoimmune skin diseases and to emphasise the need for a treatment plan that takes into account the different immunopathogenesis of each condition, so that treating one disease will not result in exacerbation of the others.

Keywords: Bullous Pemphigoid, Psoriasis, Acquired Reactive Perforating Collagenosis, Autoimmune Skin Disease.

Introduction

Bullous pemphigoid (BP) is the most common subepidermal autoimmune bullous skin disease that affects mostly people above the age of 70. The incidence is 2,5-42,8 new cases/ million/year [1]. The disease can be triggered by factors such as drugs, infections and vaccines. Of great importance in the BP pathogenesis are the T helper (Th) 2 axis that enhances the production of antibodies against proteins of the dermoepidermal junction (BP180/BP230) and the Th17 axis that produces interleukin (IL)-17 and IL-23. The cytokine production then leads to complex activation, mast-cell degranulation, neutrophil chemotaxis and protease release, resulting in dermoepidermal separation and blister formation [1, 4]. Histopathological examination, immunofluorescence and titration of BP180/BP230 autoantibodies by enzyme-linked immunosorbent assay are used for the diagnosis of BP. Local and systemic corticosteroids are the first-line treatment of BP [1].

Psoriasis is a chronic inflammatory skin disease that affects around 2% of the worldwide population and usually presents during the 3rd or 6th decade. Drugs, infections and trauma can trigger the disease. Psoriasis is characterised by increased expression of Th1 and Th17 cytokines, tumor necrosis factor (TNF)-α, IL-17 and IL-23 and T regulatory cell dysfunction. Plaque psoriasis is the most common type, whereas chronic inflammation is found in up to 75% of patients with psoriasis, leading to comorbidities, such as psoriatic arthritis. Diagnosis of psoriasis is mainly clinical [3, 5]. Therapeutic options include systemic agents such as acitretin and biological agents and topical treatments such as steroid creams [6].

Acquired reactive perforating collagenosis (ARPC) is a rare skin disease that usually affects adults with diabetes mellitus or renal disease. Although the exact mechanism of the disease has not yet been elucidated, overexpression of transforming growth factor-

beta 3 and extracellular matrix proteins such as fibronectin and transepidermal collagen elimination have been noticed in ARPC patients [7, 8]. Clinically, ARPC presents as pruritic hyperkeratotic papules in any area of the body, mainly following skin trauma. Histopathology may help with the diagnosis. The disease may resolve after 6-8 weeks leaving hyper- or hypopigmented areas but relapses and remissions usually follow. An important aim of the treatment is to reduce pruritus and to consult patient to avoid skin trauma [7, 8]. Although there are no specific therapeutic guidelines for severe cases that do not resolve or relapse often, various treatment options have been used in individual cases, such as cyclosporine, acitretin and methotrexate [9].

This case report documents an unusual combination of three autoimmune skin diseases in a single patient (plaque psoriasis, ARPC and BP) and may offer insight for further research into the pathophysiology of the coexistence and the management of these conditions. Of great importance is the development of a therapeutic plan that may address all three diseases and also minimise the relapse risk and the side-effects, ultimately improving the quality of life of such patients.

Case Report

A 57-year-old female was admitted to our department due to painful pruritic generalised rash with pustules, papules and erosions, blisters on wrists and ankles and hyperkeratotic plaques on palms and soles (shown in Fig. 1). The rest of the clinical examination was normal.

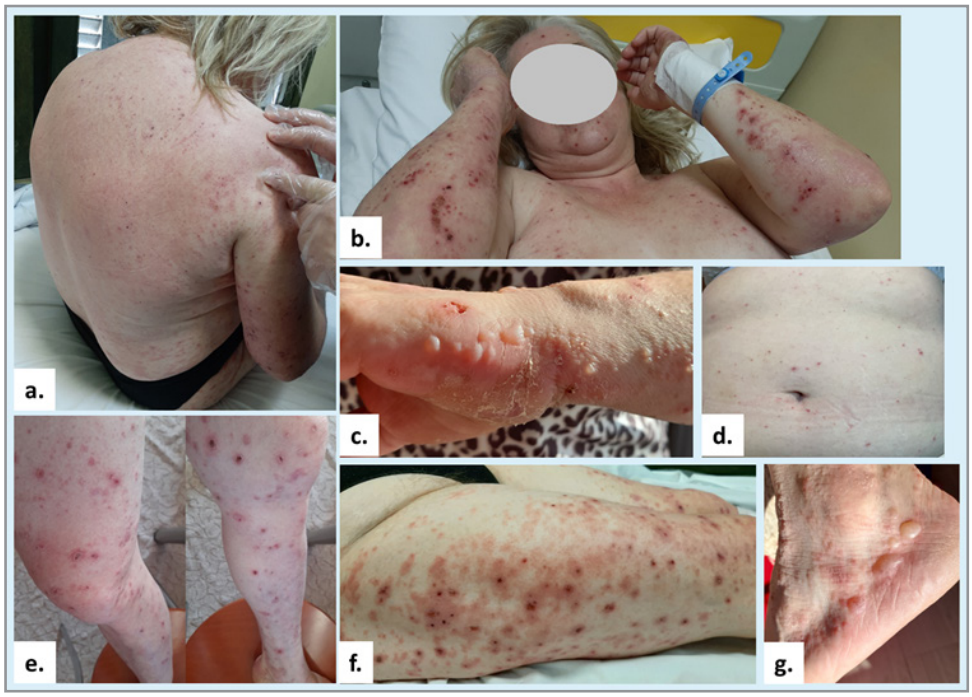

Figure 1: Skin lesions of patient on 1st day – admission to hospital
a, b, d, e, f. whole-body distribution of scattered papules and erosions on erythematous base; c, g. BP blisters on wrists and ankles

Dermatological History: 24 years before admission to our clinic – psoriatic plaques in wrists, ankles and elbows presented – treated with topical steroid creams; 14 years before admission – methotrexate was prescribed – improved lesions but was discontinued due to increase in liver enzymes, so only used topical steroid creams; 3 years before admission – psoriatic lesions worsened and arthritis appeared on psoriatic lesion sites and patient was prescribed brodalumab, risankizumab and adalimumab sequentially, with no clinical improvement; one month before admission – pruritic hyperkeratotic papules appeared all over the body – patient was examined in outpatient clinic of our department – histopathological examination of skin biopsy showed ARPC – patient was treated with cyclosporine but due to no improvement, acitretin and bilastine were prescribed instead; day of admission – blisters appeared mainly in extremities, so patient was admitted to our clinic.

Medical History: hypertension, dyslipidemia, generalised anxiety disorder, psoriasis, psoriatic arthritis, iridocyclitis, ARPC, two normal labors (38 & 33 years before) and appendectomy-tonsillectomy during childhood.

Medical Treatment at Home (Prior to Admission): fluoxetine, alprazolam, venlafaxine, olmesartan/amlodipine/hydrochlorothiazide, atorvastatine, calcium folinate, acitretin, bilastine, mometasone.

Laboratory Tests in Clinic: increased erythrocyte sedimentation rate and C-reactive protein serum levels, positive antinuclear antibodies in serum, negative Tzanck smear test. Histological Examination of Skin Biopsy (Containing a Blister): BP.

Medical Treatment in Our Clinic: per os (po) methylprednisolone 24 mg daily for 2 weeks, po acitretin 25 mg daily for 1 week, po doxycycline 100 mg daily for 2.5 weeks, po levocetirizine 5 mg twice per day, po omeprazole 20 mg twice per day, po

calcium-D3 (500 mg/440 IU) daily, cream fluocinonide thrice daily in palms and soles and mixed with hydrophilic base (1:1) in whole body, vaseline cream with 10% salicylic acid daily in hyperkeratotic plaques of soles, emollient cream for whole body moisturisation; also continued with her prior-to-admission medical treatment for non-skin diseases.

Patient Response To Treatment: no new blisters after 6th day in the clinic and significant reduction in pruritus; po acitretin reduced to 10 mg daily after 1st week; po methylprednisolone reduced to 20 mg daily after 2nd week.

Patient Discharge: on 25th day; significant improvement of skin lesions (shown in Fig. 2) and pruritus.

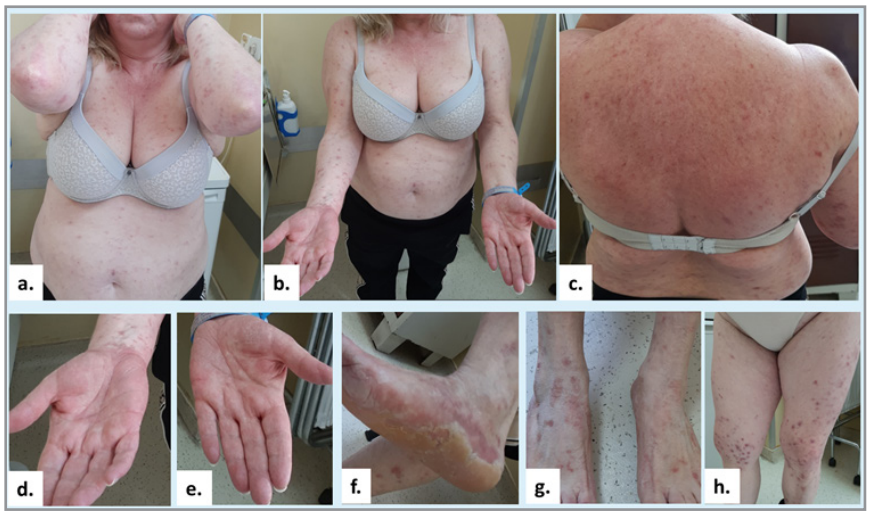

Figure 2: Improvement of skin lesions of patient on 25th day – discharge from hospital
a, b, c, h. improvement of whole-body skin lesions; d, e, g. no new blisters on wrists and ankles; f. improvement of hyperkeratotic plaques

Medical Treatment After Discharge: po methylprednisolone 16 mg daily, po acitretin 10 mg daily, po omeprazole 20 mg daily, vaseline cream with 10% salicylic acid daily in hyperkeratotic plaques of soles, emollient cream for whole body moisturisation; also continued with her prior-to-admission medical treatment for non-skin diseases.

First Follow-up After One Month: no increase in pruritus; significant improvement of hyperkeratotic plaques in palms and soles (shown in Fig. 3); no new psoriasis or BP lesions; 5-6 pruritic hyperkeratotic papules in arms and legs (ARPC lesions), no change in the post-inflammatory skin hyperpigmentation (shown in Fig. 3); will continue with same treating plan until 2nd follow-up after one month.

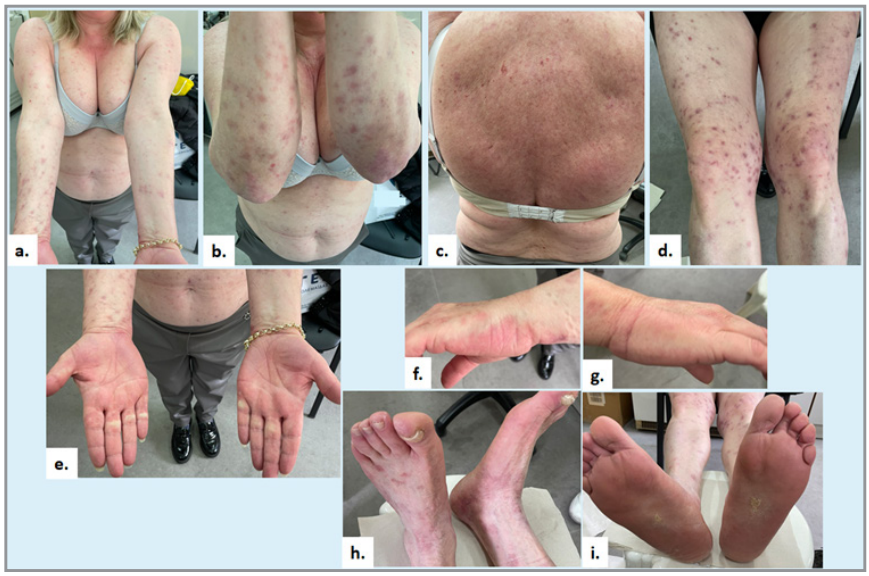

Figure 3: Skin lesions of patient on 1st follow-up, one month after discharge from hospital
a-d. no regression of residual skin hyperpigmentation; e-h. no new BP blisters; i. almost complete remission of hyperkeratotic plaques

Discussion

This is the first published case report of a patient with psoriasis that developed ARPC and BP. The literature has found that prevalence of psoriasis is higher in BP patients compared to control group, with a male predisposition [10]. The epitope spreading phenomenon has been suggested to interpret the coexistence of

BP and psoriasis, according to which, psoriasis leads to chronic inflammation of the dermoepidermic junction, increasing the exposure of antigens such as BP180 to immune system, leading to auto-antibody production [3]. A study reported that a patient with psoriasis developed BP after treating psoriasis with psoralen and ultraviolet-A light (which causes a shift from Th1 axis to Th2). Psoriasis then relapsed after successfully treating BP [11]. Biological agents have been reported to either induce or treat BP [12]. A study searched articles published in English between 2009-2022 and found reports of successful treatment of psoriasis and BP with IL-12/23, IL-17 and TNF-α inhibitors in three, one and three cases respectively [12]. Nevertheless, the same study also found seven, one and seventeen cases that used IL-12/23, IL-17 and TNF-α inhibitors respectively to treat conditions such as rheumatoid arthritis, inflammatory bowel disease, or psoriasis, which reported a new onset of BP after initiation of biological agents [12]. Although the mechanism of these phenomena has not yet been fully elucidated, it seems that changes in the balance of Th1/Th2 axis and IL-4/interferon-γ levels may play a role in the effect of a biological agent on BP course [12].

With regards to coexistence of BP and ARPC, the literature has reported only a few cases of BP, usually within two years of development of ARPC. These patients were mostly men, above the age of 60, with diabetes mellitus and some were on dipeptidyl peptidase-4 inhibitors (DDP-4) or had renal failure. Treatment with po prednisolone and discontinuation of DDP-4 inhibitors improved BP but not ARPC in all cases [13, 14]. Pruritus and tissue damage in ARPC is proposed to increase exposure of isolated antigens such as BP180 to immune system. This increased antigen exposure, combined with the predisposition of diabetes mellitus to autoimmunity due to chronic inflammation, may lead to production of autoantibodies against basal membrane proteins and BP development [14].

Lastly, a few cases of coexistence of ARPC and psoriasis have been reported. Two studies have found underlying psoriasis in approximately 7% of ARPC patients [15, 16]. Although no potential mechanism for ARPC in patients with psoriasis has been described, a study about perforating folliculitis and psoriasis hypothesised that both psoriasis and psoriasis treatments such as corticosteroids may increase skin keratinisation, which could result in collagen perforation [17].

During the treatment of our patient all coexisting diseases were taken into account. A few weeks before admission, the patient, who was already diagnosed with plaque psoriasis and arthritis, was examined in our outpatient clinics due to development of pruritic hyperkeratotic papules all over body. Although there is no standard treatment for ARPC, therapies such as cyclosporine, acitretin and methotrexate have been successfully used in separate cases [9]. Cyclosporine was initially prescribed, targeting ARPC, psoriasis and psoriatic arthritis [18]. As ARPC showed no improvement and the arthritis of the patient was in remission and, according to the rheumatologist of the patient, in no need for treatment, cyclosporine was replaced with acitretin, which would target both ARPC and psoriasis [1, 9]. However, blisters developed, which raised the suspicion of a bullous disease, therefore the patient was admitted to our clinic. After BP was confirmed by histological examination, the patient was treated with po methylprednisolone, which is the first-line treatment for BP and po doxycycline, which was used as steroid-sparing agent, due to its anti-inflammatory properties [19]. Moreover, acitretin would target ARPC and is also a treatment option for psoriasis [1, 9]. Although patient BP responded to treatment, methylprednisolone tapering-off was slow, so that psoriasis would not relapse.

The aim of this case report was to describe a patient with three autoimmune skin diseases and analyse the complexity of the immunological mechanisms. Due to the fact that each disease has different immunopathology and each patient has different immunological profile and different response to therapeutic agents, it is of great importance to carefully design a treatment plan so that treating one disease will not lead to exacerbation of another condition.

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Statement of Ethics

Written informed consent was obtained from the patient for the publication of this case report.

Conflict of Interest

The authors declare no conflict of interest.

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No financial support was received for this case report.

Author's Contribution

V. E. Pappa was the dermatology resident in charge of the patient in the clinic, she examined her daily and kept track of the course of the disease and her medical treatment; K. Efthimiadis and S. Arampatzi were the dermatology specialists who supervised the medical treatment of the patient and reviewed this article; T. Sidiropoulos is the head of the clinic and the dermatology specialist in charge of the patient, who examined the patient before admission and designed her medical treatment plan and supervised and reviewed this article; V. E. Pappa is the author of this case report.

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