A CASE REPORT ON THE PRESENTATION OF PSORIASIS WITH BULLOUS PEMPHIGOID

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ABSTRACT

Psoriasis, a chronic autoimmune skin condition, can coexist with bullous pemphigoid, a rare autoimmune blistering disorder. This comorbidity presents challenges in diagnosis and management due to overlapping symptoms. Treatment involves a multidisciplinary approach, including topical corticosteroids for psoriasis plaques and systemic corticosteroids or immunosuppressants for bullous pemphigoid blisters. Close monitoring and individualized therapy are crucial to balance control of both conditions while minimizing adverse effects. A compelling case study of a 63-year-old male with diabetes and hypertension presented with scalp psoriasis and bullous pemphigoid. Echocardiogram revealed severe aortic stenosis. Skin biopsy confirmed bullous pemphigoid. Treatment included medications for pruritus, inflammation, pain relief, skin protection, infection prevention, and autoimmune control. The patient's progress was closely monitored for effective management of both skin conditions and associated comorbidities, emphasizing the necessity of comprehensive care in complex dermatological cases.

Keywords: *Psoriasis, bullous pemphigoid, corticosteroids, immunosuppressants, Echocardiogram, Aortic stenosis.*

INTRODUCTION

Psoriasis is a persistent, immune-mediated skin disease[1], allied with complex genetic susceptibility[2,9]. The exact etiology is unknown, but it is considered an autoimmune disease mediated by T lymphocytes [6].

Although rare, psoriasis can coexist with immune-mediated conditions like bullous pemphigoid (BP), a disorder often affecting older adults, presenting with pruritus and bullae. The mechanisms that trigger bullous pemphigoid in patients with psoriasis remain poorly understood [12]; however, most cases are linked to factors such as genetics, drug intake, viral infections, physical agents, and diet, which can disrupt the skin's basement membrane zone[13].

The coexistence of BP and psoriasis poses unique clinical challenges[10,11]. Psoriasis can develop at any age, often peaking at 20–30 and 50–60 years. Around 30% of patients have a first-degree relative with the condition, and risk increases with more affected family members [5].

The global prevalence is around 2%, varying by region, with lower rates in the Asian and African populations[4]. Moreover, comorbidities—including psoriatic arthritis, mental health disorders, heart disease, and liver conditions—are frequently observed[3]. The most common form of psoriasis vulgaris (plaque-type), though distinctions exist among its clinical subtypes[4], typically presents on the trunk, extremities, and scalp[6].

Immunopathogenesis involves the upregulation of inflammatory pathways that drive excessive skin cell growth. T helper (Th) cells release pro-inflammatory cytokines, with IL-23 activating Th17 cells to produce TNF-α, IL-17, and IL-22. IL-17 promotes keratinocyte hyperproliferation and cytokine production[8].

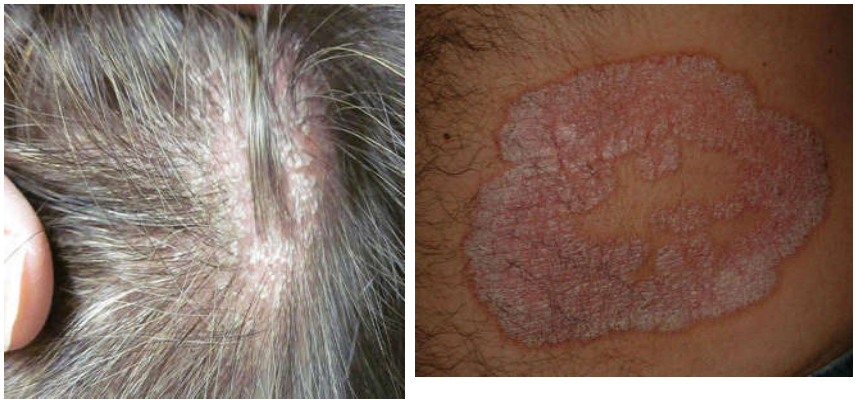

Fig 1 [2] (A) psoriasis on the scalp as white flakes. (B) psoriasis with red, silver scaling on skin.

Psoriasis was primarily diagnosed based on its characteristic appearance of red, scaly plaques, often itchy or painful. A skin biopsy was typically not required for a standard diagnosis. The plaques were usually round or oval and appeared symmetrically. Signs like Auspitz (bleeding spots when the scale was scraped) and the Koebner phenomenon (lesions

forming after skin injury) helped confirm the diagnosis. A biopsy was considered only if the presentation was unusual or unclear [6,19].

Individuals with psoriasis often have a diminished quality of life due to symptoms such as pruritus, flaking, and joint discomfort, along with the financial and emotional challenges associated with the condition. These factors contribute to issues like low self-worth, social stigma, and negative emotions such as frustration, shame, and humiliation[7].

Bullous pemphigoid is an uncommon rare skin disorder characterized by itchy welts resembling hives or fluid-filled blisters[14]. The blisters may emerge in specific regions or extend throughout the body, often presenting in areas prone to skin flexion like the armpits, groin, or abdomen. In more severe instances, blisters can also form on mucous membranes including the mouth, tongue, throat, esophagus, and eyes[15].

Bullous pemphigoid is identified as an autoimmune disorder that targets the layer of tissue beneath the outermost skin layer. This immune-related inflammation leads to the separation of skin layers, ultimately causing the formation of blisters[16].

Bullous pemphigoid typically presents in individuals aged 50 and above, showing a greater prevalence. The condition often resolves on its own within a few months, but in some instances, this resolution may take several years[17] [18]. The important medications that are linked to bullous pemphigoid are PD1-inhibitor (Programmed cell death protein-1) immunotherapies, like pembrolizumab and nivolumab, which are used for treating metastatic melanoma and various cancers[14].

Furthermore, dipeptidyl peptidase-4 inhibitors, commonly referred to as 'gliptins' and used in diabetes management, including medications like sitagliptin, linagliptin, and notably vildagliptin, have been linked to the development of bullous pemphigoid[18].

Exposure to light and radiation can also trigger bullous pemphigoid, with ultraviolet light therapy for skin conditions and radiation therapy for cancer serving as potential instigators. Certain medical conditions such as psoriasis, lichen planus, diabetes, rheumatoid arthritis, ulcerative colitis, and multiple sclerosis have been associated with precipitating bullous pemphigoid[15].

Bullous pemphigoid manifests as severe itching and the presence of typically large, tense bullae (fluid-filled blisters) that rupture, leading to crusted erosions. Prior to blister formation, a non-specific rash may appear several weeks earlier. Others may exhibit red skin to urticaria. The presence of ring-shaped lesions, termed annular lesions, can also be observed. Smaller blisters (vesicles) may form in addition to pemphigoid nodularis, which manifests as prurigo nodules. Blister fluid can vary in color and consistency, appearing clear, cloudy, yellowish, or bloodstained. Small blisters or sores may also develop on mucous membranes, such as inside the mouth (benign mucous membrane pemphigoid[15] [18].

Bullous pemphigoid is associated with human leukocyte antigen (HLA), indicating a genetic predisposition to the disorder. In certain instances, bullous pemphigoid can be associated with acquired hemophilia, stemming from developing anti-factor VIII antibodies. The production of IgG autoantibodies targeting hemidesmosomal proteins like BPAG1 (BP antigen 230) and

BP antigen 180 (BPAG2 or type XVII collagen) plays a crucial role in the pathogenesis of bullous pemphigoid[17] [18].

A confirmed diagnosis typically requires a skin biopsy of a developing blister. Direct immunofluorescence staining on skin tissue close to the blister reveals antibodies lining the basement membrane that divides the epidermis and dermis. Blood examinations may include indirect immunofluorescence tests to identify circulating pemphigoid BP180 antibodies, alongside immunoblotting[14] [15] [18].

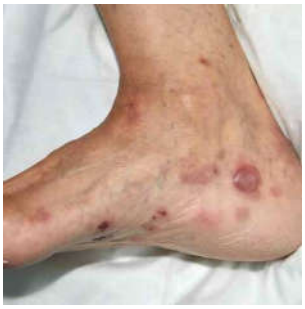

Fig 2. Bullous Pemphigoid blisters on right foot [18].

TREATMENT FOR BULLOUS PEMPHIGOID**:**

The primary goals of treating Bullous Pemphigoid are to prevent skin damage, promote healing, and relieve pruritus. As most patients are elderly, treatment should consider comorbidities and self-care abilities to minimize complications and improve outcomes. Management should focus on effective disease control with well-tolerated therapies suited to the patient's condition [20].

Systemic corticosteroids

Probably the most universally recommended treatment for adequate management of bullous pemphigoid is oral prednisolone (PSL) at 0.5 to 1 mg/kg/day. It must be administered in full doses as a suppressive treatment. [21 - 23]

Topical corticosteroids

The topical corticosteroids, including 0.05% Clobetasol propionate cream, can be given in a daily dose of 10-40 g according to Clinical Practice Guidelines for Bullous Pemphigoid[26]. Furthermore, it is supposed that in-patient topical corticosteroids are superior than oral corticosteroids for the treatment of severe bullous pemphigoid, particularly to patients who manifested minimal bullous pemphigoid [24, 25, 27].

Non-Immunosuppressive Agents When steroids are appropriate, but partial or inadequate response is expected, non-exactly immunosuppressive agents may be added. Tetracycline (0.5-2 g/day), doxycycline (200-300 mg/day) and nicotinamide (500 mg-2.5 g/day) have anti-inflammatory actions and can be used in cases of moderate or moderate Bullous Pemphigoid, also sensitizes to steroids [26, 27]. Sulfone (50-100 mg/day, 0.5-2 mg/kg/day in children) has the

drug action of attaching neutrophils; response rates are 15-45% but produces anaemias and methemoglobinemia's [26, 28, 29].

Methotrexate is an immunosuppressive agent used as monotherapy and also in conjunction with topical steroid. The starting dose is in the range of 2.5 to 15 mg per week [26]. Cyclosphamide is a second line agent reserved for older patients. It is good even in sub therapeutic doses: 50 to 100 mg daily oral or IV [26].

Intravenous Immunoglobulins (IVIG) For BP which is caused by drugs which are unresponsive IVIG is useful and it has a low rate of adverse effects but it is very expensive. It is given for 24 months and 15 cycles [30, 31].

Therapeutic Plasma Exchange

It is usually used as a last option in which autoantibodies and cytokines are stepwise disposition with an appraisal of rapid improvement having the effect that follows them. They are costly and must be extremely regulated for their complications [26, 32].

TREATMENT FOR PSORIASIS:

It is one of the immune-mediated chronic diseases characterized by always red scaling plaques. Its treatment depends upon how wide the disease will be, what quality of life it will impair, and how well the individual responds to treatment. Some of the treatments include topical therapies, systemic agents, phototherapy, biologics, and even new treatments targeting patients with refractory cases.

Topical therapies:

Topical corticosteroids are first-line for mild psoriasis but lose efficacy with long-term use. Adding vitamin D analogues or retinoids improves outcomes. Phototherapy (narrowband UVB, PUVA) is effective for moderate to severe cases, with excimer laser suitable for localized disease (<10% body surface area) [33, 34].
New agents include Tapinarof (AhR modulator), suppressing IL-17A and oxidative stress with sustained efficacy and minimal local reactions [35]. Roflumilast (PDE-4 inhibitor) reduces inflammatory cytokines, achieving significant skin clearance in 40% of patients within eight weeks, with mild side effects like diarrhea and headache [35].

Of these, Tapinarof, Roflumilast, and Deucravacitinib significantly contribute to the arena of safe and effective therapy for psoriasis, even in such cases otherwise recalcitrant, by clearly pushing ahead the prospects for safe effective treatment. [35]

For the treatment of psoriasis with bullous pemphigoid, the patient was prescribed methotrexate orally at 10 mg/week for managing psoriasis and Bullous pemphigoid, which is an agent associated with targeting systemic inflammation in the latter. Topical cream 0.3% betamethasone was also used to manage bullous pemphigoid's inflammatory and blistering manifestations. The combined approach then targeted the overlapping pathologies that both conditions have [36] .

CASE REPORT:

A 63-year-old male presented to a tertiary care hospital with a history of persistent scalp pruritus and psoriatic skin involvement for the past year. In addition, he developed tense vesicles and bullae, along with raw, eroded areas over his hands and body for the last 20 days. These lesions were associated with localized itching. The patient has a known history of long-standing diabetes mellitus and hypertension, both of which have been managed for the past four years. He was admitted for a comprehensive evaluation of his chronic, progressively worsening dermatologic condition. His current medications included Telma (Telmisartan) 40 mg once daily in the morning after breakfast for hypertension, Glucophage (Metformin) 20 mg once daily in the morning after breakfast for diabetes management, Omeprazole (capsule form) taken before breakfast, and Trica (Tramadol 0.5 mg and Paracetamol) for pain relief.

On admission, the patient's vital signs were stable: blood pressure 120/70 mmHg, heart rate 80 beats per minute, respiratory rate 20 breaths per minute, and oxygen saturation 97% on room air. He was afebrile at the time of the examination.

A dermatological examination suggested a differential diagnosis of bullous pemphigoid or psoriasis vulgaris. A skin biopsy was promptly obtained for histological analysis. A skin biopsy was collected for histological evaluation. Diagnostic tests were performed to assess the patient's overall health. The hepatic function tests indicated normal liver function and a balanced lipid profile. Renal function was stable, with serum glucose measured at 105 mg/dL, plasma urea at 9 mg/dL, blood urea nitrogen at 4 mg/dL, creatinine at 0.81 mg/dL, and uric acid at 4.5 mg/dL. Bilirubin levels were also within normal limits, recorded at 0.6 mg/dL. Additional tests indicated that the albumin-to-globulin ratio, serum calcium, and serum electrolytes were in normal ranges. The prealbumin level was 19 mg/dL, and amylase was 47 U/L. The patient's ESR (erythrocyte sedimentation rate) was 45.4 mm/hr, indicating the presence of inflammation.

The echocardiogram revealed significant calcific aortic stenosis accompanied by minor aortic regurgitation. The aortic jet velocity was measured at 4.0 m/s, with maximum and mean pressure gradients of 63 mmHg and 40 mmHg, respectively, indicating severe obstruction of the aortic valve. Left ventricular hypertrophy (LVH) was observed, likely as a response to the increased pressure caused by the aortic stenosis. Both the left and right ventricles are functioning normally, with an ejection fraction of 60% and no signs of pulmonary arterial hypertension. The skin biopsy revealed a denuded epidermis with a dense infiltration of neutrophils, eosinophils, and a few lymphocytes.

The clinical presentation and skin biopsy findings lead to the diagnosis of bullous pemphigoid, along with a history of scalp psoriasis for the past year. Following the diagnosis of bullous pemphigoid and the co-existing scalp psoriasis, the patient was initiated on a treatment regimen to focus on both conditions. the patient was initiated on Atarax (Hydroxyzine hydrochloride) 2.5 mg orally three times a day (TID) to control pruritus, and Doxet SL (Doxycycline) 100 mg orally twice daily (BD) to address the skin lesions. Nico

Glow (Nicotinamide) 250 mg orally twice daily (BD) was included in the treatment for skin protection, trying to enhance overall well-being.

Halox-F (a mixture of Fluocinolone Acetonide and Clotrimazole) ointment was given to the scalp and body twice daily to manage the lesions associated with psoriasis and bullous pemphigoid. the patient received Hifenac (Nimesulide) 100 mg orally twice daily (BD) for pain and Metrogyl (Metronidazole) 400 mg twice daily (BD), AMO K (Potassium) 500 mg three times per day (TID), Nexpro (Esomeprazole) 40 mg orally once a day (OD) and Wysolone (Prednisolone) 20 mg orally once a day (OD) was given to reduce inflammation and manage the immune-mediated reaction which is caused by bullous pemphigoid and psoriasis. The patient's progress was monitored and focused on managing both conditions.

DISCUSSION:

Patient came to hospital with complaints of few tense vesicle and bullae along with raw eroded skin lesions and vesicles over hands and body since 20 days and itching at the site of lesions. The patient has a known history of long-standing diabetes mellitus and hypertension, both of which have been managed for the past four years persistent scalp pruritus and psoriatic skin involvement for the past year. He came to hospital for further management. Physician was advised investigations of Liver function test, Complete blood picture, Kidney function test, Thyroid profile, Complete lipid profile, Serum Electrolytes, Blood sugar levels and Echocardiogram. A skin biopsy was promptly performed for histopathological examination. Skin biopsy showed s/o Bullous Pemphigoid. Following the diagnosis of bullous pemphigoid and the co-existing scalp psoriasis, patient was treated with Atarax 2.5mg PO, TID, Doxet SL100mg PO BD, Nico Glow 250mg PO BD. For topical therapy, Halox-F ointment was applied to the scalp and body twice daily, Hifenac 100mg PO BD, Metrogyl 400mg PO BD, AMO K (Potassium) 500mg PO TID, Nexpro 40mg PO OD, Wysolone 20mg PO OD. The patient’s progress was closely monitored, with treatment focused on managing both conditions. The patient and his care takers were given advice on how to proceed with their treatment.And also counseled for drug adherence.

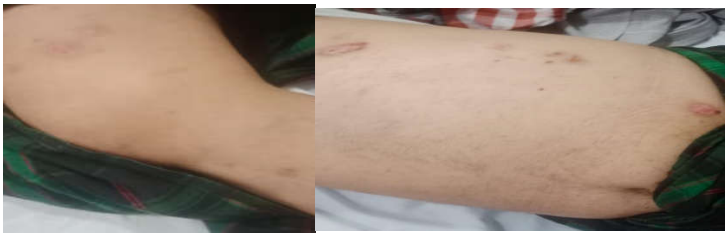

Fig (A) lower legs showing bullous blisters and plaques. **(B)** Anterior chest to upper abdomen showing multiple blisters over the skin.

CONCLUSION:

The patient presented to the hospital with chief complaints of tense vesicles and bullae along with raw, eroded areas over his hands and body for the last 20 days. These reasons were associated with localized itching. The patient had a history of persistent scalp psoriasis and a

long history of diabetes mellitus and hypertension. A dermatology opinion was sought. The patient was suspected of having bullous pemphigoid and psoriasis vulgaris. A skin biopsy was sent. Skin biopsy was s/o Bullous pemphigoid. Dermatology advice was followed. The Patient was managed with topical and systemic corticosteroids, NSAID antibiotics, antihistamines, and potassium supplements. There was a prognosis observed with the use of steroids in bullous Pemphigoid and scalp psoriasis. The patient was hemodynamically stable while discharged.

Conflict of Interest: No potential conflicts of interest

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