

## A Case Report on Bullous Pemphigoid

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### Abstract

Bullous pemphigoid (BP) is an auto-immune sub-epidermal blistering disorder that results in large tense bullae, which is induced by autoantibodies against the dermo epidermal junction of the skin and adjacent mucous membrane. It is more commonly observed in the elderly (over 80 years of age), mostly affects the people over 50 years of age. The pathogenesis of BP is characterized by tissue bound and circulating IgG autoantibodies against two components of the hemidesmosome of stratified epithelia, BP 230 KD (BPAg1) and BP 180 KD (BPAg2, COL17). We reported a 58 year of male patient who was diagnosed bullous pemphigoid on the basis of clinical findings and confirmed by skin biopsy show atrophic epidermis with basal cell layer vacuolization with fluid like material. Treatment includes corticosteroids, antibiotics, proton pump inhibitor, vitamin supplements. The patient was symptomatic-free 2 weeks after therapy was initiated.

**Keywords:** Bullous Pemphigoid; Autoantibodies; Proton Pump Inhibitor, Sub-Epidermal; Hemidesmosome, Antibiotics; Corticosteroids

### Abbreviations

BP: Bullous Pemphigoid; Igg: Immunoglobulin; Bpag1: Bullous Pemphigoid Antigen1; Bpag2: Bullous Pemphigoid Antigen2; COL17: Collagen Type17

### Introduction

Bullous pemphigoid (BP) is an autoimmune, sub-epidermal blistering disorder that results in large tense bullae, which is triggered by autoantibodies against the dermo epidermal junction of the skin and adjacent mucous membrane [1]. It majorly affects geriatric population in the fifth to seventh decade of life, with average age of onset being 65 years [2].

There is equal occurrence in both genders, and there are no known racial predilections [1]. A drug or an injury or skin infection can activate the onset of disease. About 1 in 10,000 people are affected by this condition in the UK each year. In India the incidence of sub-epidermal auto immune disease is comparatively low [2]. The incidence rate of BP was high in swiss population by relating with other countries due to demographic characteristics of Switzerland population [4].

The occurrence of BP were tense bullae with clear fluid or erosions, may initiate as erythematous urticarial, pruritic plaques, which are generalized on the areas like lower legs, fore arms, thigh, groin, abdomen, but not mucosa. The condition is finally stated by light sub epidermal blisters with mixed superficial redness [1].

Histological examination of skin biopsy from a bulla reveals a sub epidermal blister with superficial dermal inflammation consisting of eosinophils, histiocytes, and lymphocytes [1]. Although their presence is not an accurate diagnosis [2]. Therapy involves in systemic prednisone alone or in combination with a steroid-sparing agent such as azathioprine, mycophenolate mofetil and tetracycline. Patient with mild symptoms may require topical corticosteroids. The patients with severe disease who can't tolerate the treatment with prednisone may use methotrexate [1].

Though BP last from months to years, it is a self-limited disease. It is rarely lethal, and even without corticosteroid therapy, the symptoms can be relived. The disease can be controlled by the therapy, yet there is no permanent cure [1].

### Case Report

A 58 yrs old male patient was admitted in dermatology department in a tertiary care hospital with chief complaints of fluid filled lesions and raw areas over both upper limbs, lower limbs, chest and back for 25 days. Patient was apparently normal till 25 days back and then he developed these symptoms. Patient has taken for outside treatment i.e. Inj. Monocef and then lesions have been subsided. The lesions aggravated for 3 days with pain. He is a known case of HTN on treatment Amlodipine 5 mg for 5 years and he was known alcoholic.

On cutaneous examination A multiple fluid filled fragile bullae with erythematous base present over both extremities, lateral side of abdomen and back. Lesions are present in multiple raw areas over back, upper limbs.

Few crusted lesions present in palms and soles. Nikolsky sign is negative. On pathology examination section study shows atrophic epidermis with basal cell layer vacuolization with fluid like material. No evidence of malignancy in the sections studied correlate clinically. Treatment includes corticosteroids, antibiotics, proton pump inhibitor, vitamin supplements. Patient was symptoms free and discharged.

### Discussion

The epidemiology study of BP in U.K. estimated an incidence of 1.4 per 100000 - person years [2]. Every year the incidence seems to be almost seven per million in France and Germany [1]. In Malaysia one - hundred and forty - eight with bullous diseases were seen over a period of 15 years. The epidemiological information from India is not sufficient. In India the most commonly seen skin disease is Pemphigus vulgaris when compared to BP. The incidence of BP was highest in the Indians of Malaysian origin, and they are also more likely to develop pemphigus vulgaris than any other ethnic group [3].

The pathophysiology of BP is categorized by tissue bound and circulating IgG autoantibodies against 2 components of the hemidesmosome of stratified epithelia, that is BP 230 KD (BPAg1) and BP 180 KD (BPAg2, COL17). BPAg1 is a cytoplasmic protein involves in the rooting of intermediate filaments to the cytoskeleton. BPAg2 is an illustrated as a transmembrane adhesion molecule which consist of various with collagenous extracellular domains. The blister formation in sub epidermal layer is normally due to the antibodies related to BPAg2 which is mandatory. BPAg1 has no any vivid function in the disease progression although it has secondary role. In BP autoantibodies found are against alpha 6 integrin and laminin - 5, besides two other skin basement membrane components.

The complement and inflammatory mediators are activated when IgG autoantibodies attaches to the basement membrane. After the Activation of the complement system the inflammatory cells are attracted to the basement membrane which is a crucial step. The blister formation is due to the degradation of hemidesmosomal proteins which is occurred one inflammatory cells release proteases. There are other elements which play a major role like chemokines including eotaxin, cytokines, IL16 and IL2 in blister formation.

Factors like influenza vaccination, UV light, Radio therapy for breast cancer, injection or an adhesive dressing etc. are the some of the causes which aids in the progression of the disease condition.

The drugs like furosemide, enalapril, phenacetin, ibuprofen, sulfa pyridine on chronic therapy can cause BP, in addition the viruses like cytomegalovirus, Epstein-Bar virus, HHV-6, HHV-8, Hepatitis B and C viruses, *Helicobacter pylori* and *Toxoplasma gondii* may also induces BP. Stress is also one of the condition that trigger the symptoms of BP [6].

The clinical manifestations of this disease and the patient was experienced are same, the symptoms are large blisters that don't easily rupture when touched. The blisters consist of the fluid which is clear even though presented with blood. The normal areas affected with the blisters are lower abdomen, groin, upper thighs and arms. Blisters are often located in the folds of skin. The affected areas can feel severe itchiness after the infection.

Mainly the treatment involves in the reduction of the blister formation and redness without or with minimal adverse events, so that the quality of life of the patient to be improved by the healing of blisters and erosions on the skin [5]. After BP was diagnosed in the patient the treatment was initiated with corticosteroids, antibiotics, proton pump inhibitors, vitamin supplements. Patient was relieved by the symptoms. The recurrence can be seen after six years in mostly 50% of the patients who are cured as we know that there is known permanent cure.

### Conclusion

Bullous pemphigoid is a rare disease in India as it has low prevalence in our country. It is an auto immune disease, there is no permanent cure, but treatment helps to control the disease. Patient quality of life can be improved by appropriate diagnosis and treatment. BP without therapy is often a self-limiting but it may last from months to years. Even though it is self - limiting certain medical care should be taken in case of severe condition.

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