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**Case Report**
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# Case Report on Bullous Pemphigoid

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**ABSTRACT**

Bullous Pemphigoid is an autoimmune blistering skin disease that largely affects the older generation. The clinical hallmark comprises extensive blisters associated with itchy lesions. However, the triggering factors are unknown. Diseases such as Parkinson's disease, Diabetes Mellitus and Psoriasis are some independent triggers of Bullous Pemphigoid. Recently DPP4 inhibitors are found to be a risk factor for the development of BP. The diagnosis of BP is done by direct immunofluorescence and light microscopic skin biopsy. The general treatment pattern for BP depends on the severity of the disease. This includes innumerable topical and systemic anti-inflammatory and immunosuppressive agents.

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## INTRODUCTION

Bullous Pemphigoid [BP] is an orphan autoimmune disease condition that mainly affected the skin and mucous membranes in the geriatric population. The hallmark of the same is indicated by large blisters with objective evidence of granular disposition of IgG and C3 in the dermal-epidermal junction. BP is commonly seen in the elder population at an average age of between 66 and 83 years, but in countries including India, childhood cases are also reported. Childhood BP is rare and most cases occurred in children generally those older than 8 years. The exact reason for BP in childhood was unknown, more studies are needed to identify the triggering agents. The condition is not inherited so it cannot be spread to other childrens[1].

Neurological diseases like Multiple sclerosis, Parkinson's disease, and malignancy conditions are the risk factors for BP[2]. Recently patients with diabetes mellitus and under the treatment of Dipeptidyl peptidase inhibitors especially Vildagliptin associated as the element for the development of BP[3].Injury to the tissues and destruction of the dermal-epidermal junction happens due to the release of inflammatory mediators and turns to form blister formation[4].

The antigenic targets of BP are BP 180 and BP 230. BP 180 is a glycoprotein in the transmembrane and it is highly immunodominant in BP. BP 230 consists of 2649 amino acids, it is a cytoskeletal linker protein and is spotted as a target autoantigen for BP. So, in persons with BP, the immune system release antibodies to the epidermis and dermis layer of the skin leads to the production of inflammatory mediators and finally turns to blister formation. The blisters are tense and oval or round. The affected area of the skin will be slightly raised and have clear fluid inside[5]. Blisters may form anywhere but generally develop on flexural areas of skin like armpits, groin area, abdomen, etc., in some cases, blisters were seen in mucous membranes, mouth, throat, tongue, esophagus, and eyes. If untreated BP can be serious and it leads to deeper skin infection finally turns to sepsis.

## CASE REPORT

77 aged women presented with complaints of pain in the throat and bleeding for 3 months. On physical examination, she was conscious more over no facial puffiness and pedal edema. Neurological, chest, and GIT findings are normal, during the admission time the vitals especially pulse rate and blood pressure were found to be 72 beats/ minute & 130/70 mm Hg respectively.

The patient had a medical history of Diabetes Mellitus, Hypertension and was treated with Inj. Human Actrapid (40-40-30 units), Tab. Bisoprolol fumarate+ Amlodipine Besilate (Tab. Concor AM) and Tab. Vildagliptin for the respective disease conditions. She had no known neurological diseases, malignancy, liver disease, and history of blood transfusions.

The endoscopy report findings showed mucosal erosions in the sublingual region and hard palate. The mucosal erosions in the epiglottis were detected as edematous and covered with slough. This explains the reasons behind the throat pain and bleeding **Fig. 1a.**

During the time of admission, skin rashes were spotted on the surface of both hands. It begins as pruritic small papules and turns to form itchy blisters but painless **Fig.1b-1d**. IgG& C3 showed a positive result in IFA. From the skin biopsy, sections of skin show subepidermal blister with numerous inflammatory cell infiltrations within the blister cavity.

The patient was treated under the expert guidance of a general practitioner and dermatologist at the tertiary care hospital. The blood sugar level was managed with the patient's medication. The BP was managed with Tab. Prednisolone 40 mg (Omnacortil), Inj. Cefapreazone sulbactam and Mupirocin ointment. On the 6$^{th}$ day, she was discharged with stable condition, blisters also partially healed. Oral antibiotic Tab. Cefixime 200 mg BD for 5 days and Tablet Omnacortil 40 mg were prescribed as discharge medication until the next OP visit. No recurrence was noticed at the follow-up of 1 month.

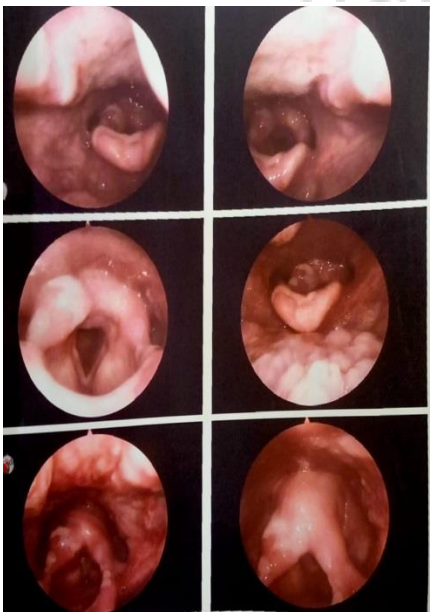


**FIG. 1a**

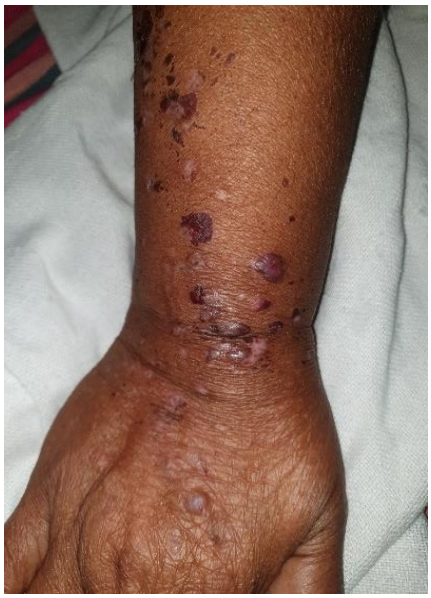
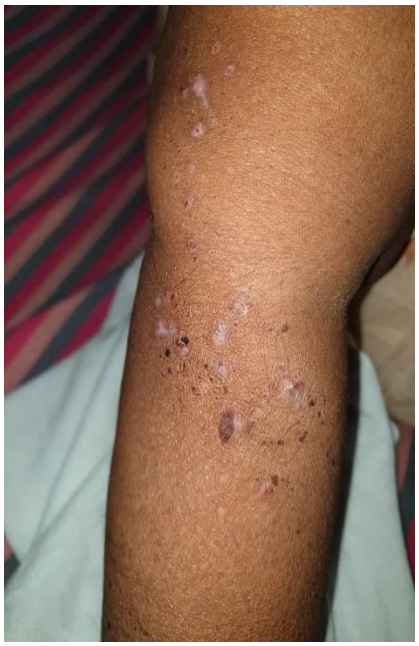


**FIG. 1b**



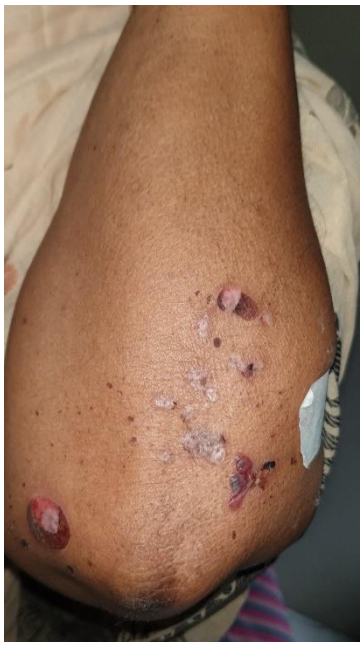


**FIG. 1c** **FIG. 1d**

**FIGURE 1: BULLOUS LESIONS OVER LARYNX (1a) AND ON SURFACE OF BOTH HANDS (1b,1c, and 1d)**

## DISCUSSION

BP is a rare autoimmune disease portrayed as blister formation with inflammatory cell infiltration. Based on the critical analysis the risk factors for BP in this patient include female sex, age, type II diabetes mellitus, and Dipeptidyl peptidase inhibitors (Vildagliptin). Diabetes patients have a higher chance of BP because it increases the chance of skin rupturing due to elevated blood sugar levels. Furthermore, some classes of drugs may induce the chance of developing BP including Diuretics (Furosemide, Hydrochlorothiazide), Antihypetensives (Amlodipine), NSAID (Ibuprofen), Antidiabetics (Vildagliptin, Sitagliptin) and vaccines (Varicella, Pneumococcus). Currently, the patient was under the treatment of DPP4 inhibitors i.e., Vildagliptin. So, it indicates that the development of BP is associated with the use of Vildagliptin but not proven.

Generally, lesions appear in the lower abdomen, thigh, and forearm nonetheless they may develop extensively[5]. Here blisters are observed on the surface of both hands, and mucosal erosions are spotted in the epiglottis and hard palate.

Typically, BP was identified by immunopathological testes. In the majority of cases, IgG antibodies and C3 were positive at the basement membrane. There are two scores available to measure the BP activity i.e., BP Disease Area Index (BPDAI) and Auto Immune Bullous Skin Disorder Intensity Score (ABSIS)[6]. In this case, also IgG antibodies and C3 were strongly positive. Mucosal involvement was identified with the help of specialties like ENT and skin biopsy. The management of BP aims to slacken the episode of blisters and heal the current erosions. The choice of therapy must be according to specific patients. Localized blisters are managed by topical steroids (Clobetasol propionate) but in advanced cases should be managed by systemic or oral corticosteroids (Prednisolone) and immunosuppressive agents (Azathioprine, Mycophenolate Mofetil)[7]. Here BP blisters are managed by oral corticosteroids Tablet Omnacotil (Prednisolone) and Injection Cefaperazone Sulbactam. Skin lesions are managed with antibiotic (Mupirocin) ointment.

## INFORMED CONSENT

Before taking this case the patient and their families were informed and informed consent was acquired.

**CONFLICTS OF INTEREST**: The authors have no conflicts of interest to declare.

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