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Research Article

CASE REPORT: VILDAGLIPTIN INDUCED BULLOUS PEMPHIGOID

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ABSTRACT

Bullous pemphigoid (BP) is an acquired autoimmune disorder characterized by chronic blistering of the subepidermal skin. BP is classified as type II hypersensitivity reactions and can be induced by drugs such as furosemide, nonsteroidal anti-inflammatory agents, DPP-4 inhibitors, captopril, pencillamine and antibiotics. Case of a 85 year old male is presented here. The patient was presented with the complaints of blisters on right and left leg for 2 days and also had a history of ulceration. The clinical diagnosis of BP was confirmed pathologically. After switching vildagliptin to insulin, remission was achieved.

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INTRODUCTION

Bullous pemphigoid (BP) is an acquired autoimmune disorder characterized by chronic blistering of the subepidermal skin.[1]. It involves formation of mildly itchy welts to severe blisters and infections, with the production of two antigens (BPAG 1 and 2). It is mostly seen in the elderly aged 70 years and over and has increased risk for long term morbidity and mortality. [2]

BP is classified as type II hypersensitivity reactions and can be induced by drugs such as furosemide, nonsteroidal anti-inflammatory agents, DPP-4 inhibitors, captopril, pencillamine and antibiotics.[3] There are many reports on the development of BP in patients treated with dipeptidyl peptidase inhibitors plus metformin used for the treatment of type 2 Diabetes mellitus. However, it is still unknown whether gliptins alone or combination with metformin are accountable for the production of BP.[4]

Case Report

A 85 year old male, admitted to the casualty department presented with the complaints of blisters on right and left leg for 2 days and also had a history of ulceration. The patient is a known case of Type 2 Diabetes mellitus, hypertension, CVA and cellulitis in both legs. He was on regular treatment with injection insulin, rosuvastatin + aspirin, vildagliptin + metformin for past 9 months.

On examination the patient was conscious and oriented with BP 130/70 mm Hg, pulse rate 96 beats/minute, respiratory rate 20 breaths/minute and SPO $_2$ 98%. Routine chemistry report showed elevated levels of glucose random {163 mg/dl}. Physical examination showed diffused erythematous tense bullae. Most lesions were ruptured, pruritic and painful.

The clinical diagnosis of BP was confirmed pathologically. He was treated with antibiotics, chymotrypsin and antihistamine. The rashes continued to worsen even after the treatment with antibiotics. After switching vildagliptin to insulin, remission was achieved.

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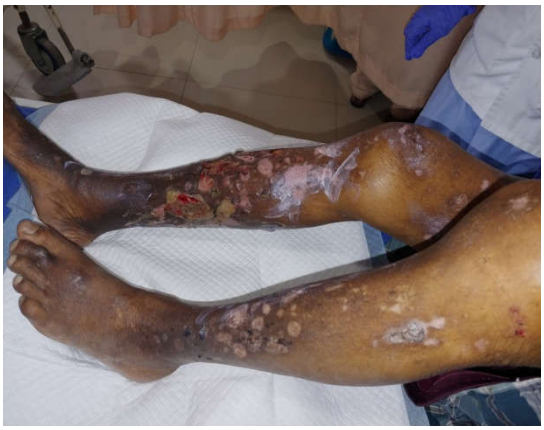

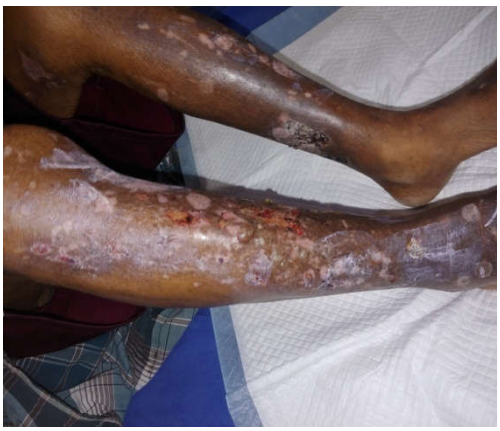

DISCUSSION

Bullous Pemphigoid, an autoimmune disease characterized by blister formation in the subepidermal region of the skin typically seen in the elderly and rarely among children and young adults.[5] The biopsy taken from the cleavage lined grey brown soft tissue of the lesion revealed maculopapular lesions and microscopy of the section showed granuloma annulare and hyperkeratotic acanthotic stratified squamous epithelium. Koilocytes were seen in the epithelium. The subepithelial region showed fibrocollagenous, fibrofatty tissue admixed with sweat glands. These observations were consistent with the diagnosis of Bullous Pemphigoid in this patient.

Dipeptidyl Peptidase 4, otherwise known as CD26, expressed throughout the body including skin, brain, heart, intestine, kidney, lungs, lymphocytes is a cell surface glycoprotein with intrinsic enzyme activity.[6] Eosinophil activation which is responsible for blister formation by CCL11/exotaxin mediated mechanism is promoted by the inhibition of dipeptidyl peptidase IV by gliptins. As a result there is an increased level of transforming growth factor beta-1 (TGF beta 1) in T cells leading to its extracellular secretion.[7]

Lesions appeared as tense bullae on an erythematous or even urticarial base. Patient experienced one episode of lesions. The bullae were filled with clear fluid and were hemorrhagic. There was no oral and ocular mucosal involvement. The bullae usually heal with post inflammatory pigmentary changes, and there is no scarring or milia formation.

Oral antidiabetics were discontinued and the patient was followed up with Insulin alone. The lesions suppressed after cessation of the drug.

CONCLUSION

The histopathological report of the disease and history of received medications set a possible diagnosis. After cessation of the suspected medication, patient responded rapidly to treatment. The use of vildagliptin needs to be carefully evaluated, mainly in high-risk patients such as males and those age 80 years or older.

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