International Journal of Research in Medical Sciences
Guddeti L et al. Int J Res Med Sci. 2025 Sep;13(9):3843-3846
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20252804

Case Report

Amlodipine induced bullous pemphigoid: a case report

Likhitha Guddeti[1], Yashmeen Nikhat P.[2]*, G. Sai Kumar Reddy[3], Pavithra Mokkatla[4]

[1]Department of Pharmacy Practice, Jawaharlal Nehru Technological University, Ananthapur, Andhra Pradesh, India
[2]Department of Pharmacy Practice, Dr. K. V. Subbareddy Institute of Pharmacy, Kurnool, Andhra Pradesh, India
[3]Samahitha Research Solutions, Bangalore, Karnataka, India
[4]Cognizant Technology Solutions Corporation, Hyderabad, Telangana, India

Received: 28 April 2025
Revised: 06 May 2025
Accepted: 02 August 2025

***Correspondence:**
Dr. Yashmeen Nikhat P.,
E-mail: yashmeennikhat@gmail.com

ABSTRACT

Bullous pemphigoid (BP) is a common autoimmune blistering disease primarily affecting the elderly. It is often associated with itchy urticarial plaques and can be identified by the production of subepidermal blisters. Autoantibodies that target the proteins BP180 and BP230, which appear at the dermo-epidermal junction, formed as part of the pathogenesis. However, the exact reason for occurrence of this disease is still unknown, some drugs, such as antihypertensives like calcium channel blockers (CCBs), have been associated to the occurrence or aggravation of elevated blood pressure. The patient in this case study is a 65-year-old woman who was prescribed with amlodipine, a drug commonly prescribed to treat hypertension, for several years before developing BP-like lesions. The patient developed lesions on the face, chest, belly, and trunk; histological and clinical results supported the BP diagnosis. The patient's symptoms improved after stopping amlodipine and modifying her treatment plan. This case highlights the need for more cautious when prescribing medications to elderly patients, particularly those with comorbidities, who are at higher risk for drug-induced autoimmune conditions. When a patient presents with new-onset bullous lesions, particularly in the context of recent medication changes, the possibility of drug-induced BP (DIBP) should be considered.

Keywords: Bullous pemphigoid, Antihypertensive, Amlodipine, Lesions

INTRODUCTION

Bullous pemphigoid (BP) is the most common subepidermal bullous autoimmune disease, type II hypersensitivity reaction which mainly affects the elderly. It is characterized by generalized pruritic urticarial plaques and taut sub-epithelial blisters. BP auto-antibodies are primarily directed against two hemidesmosomal proteins, BP180 (also called type XVII collagen) and BP230, which are components of the dermoepidermal junction (DEJ).[1] In patients with BP, BP180 and BP230 antigens are taken up by antigen-presenting cells, processed, bound to major histocompatibility complex class II (MHC) and then exposed to the cell surface. Recognition of these epitopes by T lymphocytes induces the production of several cytokines and, subsequently, B lymphocytes are stimulated to produce autoantibodies (autoAbs). AutoAb isotypes against BP180 and BP230 are usually immunoglobulin G (IgG), but IgE is also involved.[2]

The incidence of BP ranges from 2 to 30 new cases per million people per year and has increased in recent year.[3] It also increases with age, with 190 to 312 new cases per million people per year among people over 80 years old. Some data suggest a higher overall prevalence of BP in women compared to men, but a higher prevalence in men after 70 years.[4]

The etiology and pathogenesis of BP remain largely unclear. In certain cases, the condition's onset or

worsening has been associated with various triggers, including medications, physical factors, vaccines, infections, and organ transplants. Further prospective studies with thorough patient histories are necessary to uncover other potential contributing factors. However, a specific trigger is "the last nail in the coffin." acting in concert with several predisposing factors, such as HLA, comorbidities and aging lead to the pathogenic cascade. Different classes of drugs that trigger the BP are antibiotics, anti-hypertensives [beta blockers, CCBs, diuretics], non-steroidal anti-inflammatory drugs (NSAIDs), salicylates and, more recently, anti-tumor necrosis factor (TNF)-α, dipeptidyl peptidase 4 inhibitors (DPP-4i), and immune checkpoint inhibitors targeting programmed cell death receptor 1 (PD-1) and its ligand (PD-L1).[5]

BP profoundly diminishes quality of life through visible lesions, intense itching, and pain that disrupts sleep. These symptoms may lead to poor self-image, social withdrawal, reduced independence. These may be associated with complications such as infections, and treatment side effects.[6]

DIBP has been previously reported in association with medications such as penicillamine, captopril, furosemide, and ampicillin. The highest frequency of BP occurrence has been observed in patients treated with diuretics and neuroleptic drugs.[7]

CASE REPORT

This 65-year-old female patient presents with a one-year history of fluid-filled blisters that have increased in number over the past month. Initially, the lesions appeared on the back of her trunk and later spread to her chest, face and abdomen. The blisters spontaneously ruptured, leaving raw areas that healed with post-inflammatory hyper-pigmentation. The patient denies any history of drug intake before the onset of the blisters and reports no associated itching.

Notably, she was on tablet prednisolone 40 mg daily, around the time of the initial onset of the blisters, which has helped alleviate the lesions. The patient reports a 20-year history of hypertension and is on a daily regimen of amlodipine 5 mg and atenolol 50 mg.

Dermatological examination

The patient presents with well-defined fluid-filled vesicles, with sizes ranging from 1×2 cm to 3×2 cm, which rupture easily. Multiple erosions are observed on the back of the trunk, and these lesions are healing with post-inflammatory hyperpigmentation without scarring. Crusting is noted on the scalp.

The overall appearance suggests an ongoing blistering process, with some lesions in the healing phase characterized by hyperpigmentation.

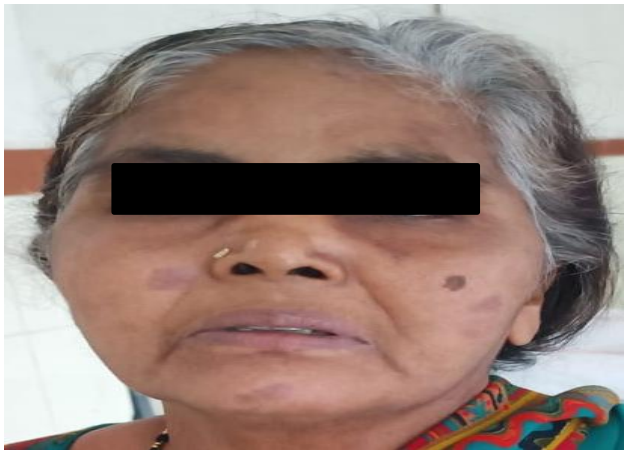

Figure 1: Localized hyperpigmentation at the site of blister rupture.

Figure 2: Scalp with fluid filled blisters.

Table 1: Investigations.

Parameters	Observed values
Hemoglobin	11.5 gm%
Total count	9000 cells/cumm
Differential count	
Neutrophils	53%
Lymphocytes	40%
Eosinophils	03%
Monocytes	04%
Platelets	2 lakh/cumm
Serum electrolytes	
Sr. sodium	150 mEq/l
Sr. potassium	4.5 mEq/l
HbSAg	Negative
HVC	Negative
FBS	100 mg/dl
Total bilirubin	0.8 mg/dl
Serum urea	19 mg/dl
Serum creatinine	0.7 mg/dl
Urine analysis	
Albumin	Traces
Sugar	Nil

Biopsy

A skin biopsy of the lesion demonstrates hyperkeratosis, while histopathological examination shows a subepidermal blister associated with a mixed inflammatory infiltrate predominantly composed of granulocytes.

Based on the clinical and histopathological findings, the patient was diagnosed with recurrent BP. Initial treatment included erythromycin, fusidic acid ointment, along with supplements of vitamin B and C, vitamin A and D capsules, zinc, and iron folic acid 100 mg. After 3 days, erythromycin was discontinued, and the treatment regimen was adjusted to include inj. chlorpheniramine maleate 2 CC, IM, BD, inj. ceftriaxone 1 g, IV, BD, inj. pantoprazole 40 mg, BD, inj. dexamethasone IV, 2 CC, BD, and tablet mycophenolate mofetil 500 mg, TID. The inj. dexamethasone dose was gradually tapered, starting from 2 CC to 1.5 CC, and then to 1cc after 1 week of administration. The patient's hypertension treatment was modified, with amlodipine 5 mg being replaced by telmisartan 40 mg.

After stopping the amlodipine, the patient symptoms of BP improved.

DISCUSSION

Amlodipine is associated with various skin reactions in less than 1 percent of patients. These include erythematous rash, maculopapular rash, skin discoloration, urticaria (hives), skin dryness, alopecia (hair loss), dermatitis, and erythema multiforme.[7] It has been known to cause linear IgA dermatosis as well as BP.[8] Although these skin eruptions can occur in response to amlodipine, there is no direct link between amlodipine and BP. BP is an autoimmune condition characterized by subepidermal blistering, which differs from common skin reactions such as rashes. While skin reactions like rashes may occur with amlodipine, BP involves a distinct immune mechanism. If BP is suspected in a patient taking amlodipine, the medication should be discontinued, and a dermatological evaluation is recommended for proper diagnosis and management.[7] The cutaneous manifestations of BP can be highly polymorphic, meaning they can present in a variety of ways. This variability has led to the identification of several clinical variants of BP. Despite these differences in presentation, direct immunofluorescence microscopy of a perilesional biopsy in all BP variants consistently reveals linear deposits of IgG and/or C3 at the DEJ, which is a hallmark of the disease. This immunological finding helps confirm the diagnosis of BP, regardless of the clinical variant.[9] In this case, there was a temporal relationship between the initiation of amlodipine therapy and the onset of skin lesions, strongly suggesting that amlodipine could have been a triggering factor. The clinical presentation, including tense bullae, erosions, and post-inflammatory hyperpigmentation, is consistent with BP. Histopathological findings, including a subepidermal blister with a mixed inflammatory infiltrate, further confirmed the diagnosis of BP.

Verheyden et al showed an uncertain association of amlodipine with the BP. Antihypertensive medications have been implicated as potential contributing factors in several cases of BP, including CCBs. Amlodipine plays a role in triggering or exacerbating BP in certain individuals, although the exact mechanisms remain unclear. CCBs, like amlodipine, have been associated with a range of dermatological reactions, and in rare instances, they may act as a precipitating factor for the development of BP.[10]

CONCLUSION

BP is a chronic autoimmune disorder, and it is crucial to suspect and diagnose DIBP to enable prompt management. Early recognition of the condition, particularly in patients with a history of antihypertensive use or other potential triggering medications, can facilitate the discontinuation of the causative drug and the initiation of appropriate treatment. This proactive approach helps minimize complications and improve patient outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Kasperkiewicz M, Zillikens D. The pathophysiology of bullous pemphigoid. Clin Rev Allergy Immunol. 2007;33(1-2):67-77.
2. Fania L, Caldarola G, Müller R, Brandt O, Pellicano R, Feliciani C, et al. IgE recognition of bullous pemphigoid (BP)180 and BP230 in BP patients and elderly individuals with pruritic dermatoses. Clin Immunol. 2012;143(3):236-45.
3. Bertram F, Bröcker E-B, Zillikens D, Schmidt E. Prospective analysis of the incidence of autoimmune bullous disorders in Lower Franconia, Germany. J Dtsch Dermatol Ges. 2009;7(5):434-40.
4. Werth VP, Murrell DF, Joly P, Ardeleanu M, Hultsch V. Bullous pemphigoid burden of disease, management and unmet therapeutic needs. J Eur Acad Dermatol Venereol. 2025;39(2):290-300.
5. Moro F, Fania L, Sinagra JLM, Salemme A, Di Zenzo G. Bullous Pemphigoid: Trigger and Predisposing Factors. Biomolecules. 2020;10(10):1432.
6. Werth VP, Murrell DF, Joly P, Ardeleanu M, Hultsch V, et al. Bullous pemphigoid burden of disease, management and unmet therapeutic needs. J Europ Academy Dermatol Venereol. 2025;39(2):290-300.
7. Park KY, Kim BJ, Kim MN. Amlodipine-associated bullous pemphigoid with erythema multiforme-like clinical features. Int J Dermatol. 2011;50(5):637-9.
8. Williams G, Goodwin R, Hughes D. Amlodipine as a cause of mucous membrane pemphigoid: first report of amlodipine as a causative agent in MMP. Eye. 2013;27:1425.

9. Mohan KR, Govindasamy Sugumar SP, Fenn SM, Thangavelu RP. Recurrent bullous pemphigoid: A case report and literature review. World Acad Sci J. 2023;5(2):185.
10. Verheyden MJ, Bilgic A, Murrell DF. A Systematic Review of Drug-Induced Pemphigoid. Acta Derm Venereol. 2020;100(15):adv00224.

Cite this article as: Guddeti L, Nikhat YP, Reddy GSK, Mokkatla P. Amlodipine induced bullous pemphigoid: a case report. Int J Res Med Sci 2025;13:3843-6.