



Case Study

Recurrent Bullous Pemphigoid During Corticosteroid Tapering in A Patient with Parkinsonism: A Case Report

Vishnumaya A. M.¹, Angitha Binu¹, Drishya L.*², Shaiju S. Dharan³

¹PharmD Intern, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram

²Assistant Professor, Department of Pharmacy Practice Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram

³Principal/HOD, Department of Pharmacy Practice Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram

Background: Bullous pemphigoid is an autoimmune subepidermal blistering disorder predominantly affecting older adults. Systemic corticosteroids are effective for disease control; however, recurrent disease activity during corticosteroid tapering can make long-term management challenging. Steroid-sparing immunosuppressive agents such as mycophenolate mofetil may be considered in patients with recurrent disease activity. **Case Presentation:** A 59-year-old man with parkinsonism receiving Tab. Syndopa presented with sudden-onset vesicles and bullae over the trunk and upper limbs for one week. Skin biopsy demonstrated a subepidermal split containing fluid and inflammatory cells, including eosinophils, supporting a diagnosis of bullous pemphigoid. Direct immunofluorescence and BP180/BP230 antibody testing were not performed. The patient initially improved with oral prednisolone but developed new lesions during repeated corticosteroid tapering. Clinical improvement with healing of lesions was observed at subsequent visits; however, new lesions recurred when prednisolone was reduced to lower doses, including 7.5 mg/day. Mycophenolate mofetil was subsequently introduced as a steroid-sparing agent. After discontinuing medication for approximately two months, recurrent bullous pemphigoid was documented, requiring reinitiation of prednisolone and mycophenolate mofetil. At the final documented follow-up, two new lesions were present while older lesions were healing. **Conclusion:** This case highlights the challenges of maintaining disease control in bullous pemphigoid during systemic corticosteroid tapering. Recurrent lesion development at lower corticosteroid doses and after treatment discontinuation emphasizes the importance of careful longitudinal monitoring and individualized adjustment of therapy. The case also illustrates the potential role of mycophenolate mofetil as a steroid-sparing treatment in recurrent disease. The absence of direct immunofluorescence and BP-specific serological testing represents an important diagnostic limitation.

Keywords: Bullous pemphigoid, Corticosteroid tapering, Prednisolone, Mycophenolate Mofetil; Parkinsonism, Autoimmune blistering disease.

INTRODUCTION

Bullous pemphigoid (BP) is the most common autoimmune subepidermal blistering disorder and predominantly affects older adults. It is characterized by the development of tense vesicles and bullae, commonly involving the trunk and extremities, and results from an autoimmune response directed against structural proteins of the dermoepidermal junction [1]. The clinical presentation may vary considerably,

but the presence of tense bullae in an older patient is an important clinical feature suggestive of BP. Diagnosis of BP is based on the combination of clinical findings, histopathological examination, and immunopathological studies. Histopathology typically demonstrates a subepidermal blister with an inflammatory infiltrate frequently containing eosinophils. Direct immunofluorescence (DIF) is an

important confirmatory investigation, typically demonstrating linear deposition of immunoglobulin G and/or complement component 3 along the basement membrane zone. Serological detection of BP180 and BP230 autoantibodies can provide additional diagnostic support [2, 3]. Systemic corticosteroids are an effective treatment for moderate-to-severe BP. However, prolonged corticosteroid therapy can be associated with significant adverse effects, and some patients develop renewed disease activity during dose reduction [4]. In patients with recurrent disease or difficulty maintaining disease control during corticosteroid tapering, steroid-sparing immunosuppressive agents such as mycophenolate mofetil may be considered as part of an individualized treatment strategy [5]. Neurological disorders, including Parkinson disease and other neurodegenerative conditions, have also been reported in association with BP. Although the mechanisms underlying this association remain incompletely understood, the coexistence of BP and neurological disease has been increasingly recognised [6]. Here, we report a case of bullous pemphigoid in a 59-year-old man with parkinsonism who demonstrated an initial response to systemic corticosteroid therapy but experienced recurrent lesion development during corticosteroid tapering and following discontinuation of treatment. The case highlights the challenges of maintaining disease control during corticosteroid dose reduction and the subsequent use of mycophenolate mofetil as a steroid-sparing agent.

CASE PRESENTATION

A 59-year-old man presented to the dermatology outpatient department on 30 April 2025 with a history of sudden-onset vesicles and bullae over the trunk and upper limbs for approximately one week. There was no associated fever or history of upper respiratory tract infection. The patient had a history of parkinsonism and was receiving treatment with Syndopa. No other new medications were reported at the time of presentation. No mucosal involvement was documented. A skin biopsy was performed at the initial visit. Histopathological examination of a blister-edge biopsy demonstrated a subepidermal split, with separation of the epidermis from the dermis, containing fluid and inflammatory cells

including eosinophils. Based on the clinical presentation and compatible histopathological findings, a diagnosis of bullous pemphigoid was made. Direct immunofluorescence was not performed, and serum BP180 and BP230 autoantibody testing was not undertaken. At the initial visit, the patient was prescribed oral prednisolone (TAB. OMNACORTIL) 20 mg twice daily for one week, along with amoxicillin–clavulanic acid 625 mg twice daily, pantoprazole 40 mg, desloratadine 5 mg once daily, a calcium/vitamin D3-containing nutritional supplement, topical mupirocin twice daily, and an omega-3 fatty acid supplement containing eicosapentaenoic acid and docosahexaenoic acid. A review was advised after one week. At follow-up on 27 May 2025, the patient reported having discontinued the prescribed treatment after one week. Despite the interruption in treatment, he was clinically better. Oral prednisolone was prescribed at a dose of 10 mg with a planned tapering regimen of 3-0-2 tablets daily for two weeks, followed by 3-0-1 tablets daily for one week and 3-0-0 tablets daily for one week. Pantoprazole 40 mg twice daily, azithromycin 500 mg once daily for five days, and the calcium/vitamin D3-containing supplement were also prescribed. On 19 June 2025, the patient was clinically better with no new lesions. Serum sodium was 138, potassium 4.58, and random blood sugar 109. Prednisolone 10 mg was continued with further tapering, initially at 2½-0-1 tablets daily for 15 days, followed by 2-0-1 tablets daily for one week and then 1½-0-0 tablets daily. Pantoprazole 40 mg once daily and the calcium/vitamin D3-containing supplement were continued. At the next follow-up on 23 July 2025, new lesions were reported while the corticosteroid dose was being tapered to 15 mg/day. Serum sodium was 140, potassium 3.98, and random blood sugar 139. The prednisolone regimen was adjusted to 2-0-0 tablets daily for two weeks, followed by 1½-0-0 tablets daily for two weeks. Pantoprazole 40 mg once daily and the calcium/vitamin D3-containing supplement were continued. Potassium chloride supplementation (SYP. POTKLOR 2.4 mL diluted in half a glass of water three times weekly) was also prescribed. On 23 August 2025, no new lesions were reported and the previously existing lesions were healing. Serum potassium measured after two weeks was 3.58, while subsequent sodium and potassium values were 141 and 3.75, respectively.

Prednisolone was further tapered to 10 mg once daily for three weeks, followed by 7.5 mg once daily for one week. Pantoprazole 40 mg once daily and the calcium/vitamin D3-containing supplement were continued, with a review planned after one month. At follow-up on 27 September 2025, the patient developed two to three new lesions after tapering prednisolone to 7.5 mg/day. Serum sodium and potassium were 141.4 and 4.14, respectively. Prednisolone 7.5 mg once daily was continued for two weeks followed by 5 mg once daily for two weeks. In view of the recurrent lesion development during corticosteroid tapering, mycophenolate mofetil (TAB. MMF) 500 mg once daily at night was introduced for four weeks. Topical betamethasone with fusidic acid (FUCIBET CREAM L/A) twice daily, pantoprazole 40 mg once daily, the calcium/vitamin D3-containing supplement, and potassium chloride 2.5 mL diluted in half a glass of water twice weekly were also prescribed. The patient subsequently returned on 10 March 2026, when the condition was documented as recurrent bullous pemphigoid. He had been off medication for approximately two months. Serum sodium was 138 and potassium was 4.2. Treatment was restarted with prednisolone 30 mg once daily for two weeks and MMF 500 mg twice daily for two weeks. Pantoprazole 40 mg once daily and calcium 500 mg once daily were also prescribed, with a review planned after two weeks. At the final documented follow-up on 24 March 2026, two new lesions were present, while the older lesions were healing. Prednisolone was prescribed at 25 mg once daily for two weeks, and MMF 500 mg twice daily was continued for two weeks. Pantoprazole 40 mg once daily and calcium 500 mg once daily were continued. Potassium chloride 2.5 mL diluted in half a glass of water twice weekly was also prescribed, with a further review planned after two weeks. Overall, the patient demonstrated an initial clinical response to systemic corticosteroid therapy, followed by repeated development of new lesions during corticosteroid dose reduction. Mycophenolate mofetil was introduced during the later course as a steroid-sparing immunosuppressive therapy. Following two months without medication, recurrent bullous pemphigoid was documented, requiring reinitiation of higher-dose corticosteroid therapy and MMF. At the last available follow-up, older lesions were healing, although two new lesions had developed.

DISCUSSION

Bullous pemphigoid (BP) is an autoimmune subepidermal blistering disorder that predominantly affects older adults. The present case describes a 59-year-old man with parkinsonism who developed vesicles and bullae over the trunk and upper limbs and subsequently experienced recurrent disease activity during systemic corticosteroid tapering. The prolonged follow-up demonstrated an initial clinical response to prednisolone, followed by repeated development of new lesions during dose reduction, eventual introduction of mycophenolate mofetil (MMF), and recurrence after discontinuation of treatment. The diagnosis in this patient was supported by the characteristic clinical presentation and histopathological findings [2]. The biopsy demonstrated a subepidermal split containing fluid and inflammatory cells including eosinophils, which is compatible with the typical histopathological pattern of BP. Direct immunofluorescence (DIF) is an important investigation in the diagnosis of autoimmune blistering disorders and commonly demonstrates linear deposition of IgG and/or C3 along the basement membrane zone in BP [4]. However, DIF was not performed in this patient, and BP180 and BP230 autoantibody testing was also not undertaken. Therefore, the diagnosis in this case should be described as clinically and histopathologically supported BP rather than immunofluorescence-confirmed disease. The absence of immunopathological and serological confirmation represents an important limitation of the case. The patient's subsequent clinical course illustrates the difficulty of maintaining disease control during systemic corticosteroid tapering. Following initiation of prednisolone, the patient showed progressive clinical improvement, with no new lesions documented on 19 June 2025 and healing of existing lesions by 23 August 2025. However, new lesions appeared during subsequent dose reduction, first when the dose was being tapered to 15 mg/day in July 2025 and later when it was reduced to 7.5 mg/day in September 2025. This repeated pattern suggests that disease activity remained sensitive to corticosteroid dose reduction. Long-term management of BP requires balancing adequate suppression of disease activity against the risks associated with prolonged systemic corticosteroid exposure, making

individualised tapering and close clinical monitoring important [4]. In September 2025, MMF 500 mg once daily was introduced while the patient continued on a low dose of prednisolone after the development of two to three new lesions during tapering. MMF is an immunosuppressive agent that has been used as a steroid-sparing treatment in BP. Its use may be considered when prolonged corticosteroid therapy is undesirable or when adequate disease control is difficult to maintain during corticosteroid reduction [5]. In this patient, MMF was subsequently restarted at 500 mg twice daily when recurrent BP was documented in March 2026 after approximately two months without medication. At the final available follow-up, two new lesions were present although older lesions were healing, and treatment with prednisolone and MMF was continued. An additional feature of this case is the presence of parkinsonism. An association between BP and neurological disorders has been described in several observational studies. A systematic review and meta-analysis [7] involving more than 23,000 patients with BP found a significant association between BP and Parkinson disease, with a pooled relative risk of 3.42 compared with controls. Other population-based and case-control studies have similarly reported increased odds of Parkinson disease among patients with BP. These findings support an association between neurological disease and BP, although they do not establish a direct causal relationship [6]. The patient's use of Tab. Syndopa is also noteworthy when considering possible medication-related associations. A systematic review and meta-analysis have reported an association between certain dopaminergic medications and BP [8]. However, this evidence does not establish that Syndopa was responsible for BP in the present patient. In this case, no other new medications were reported at the initial presentation, but the available information is insufficient to establish a drug-induced aetiology. Therefore, the patient's parkinsonism and dopaminergic treatment should be considered relevant clinical context rather than a proven cause of the blistering disorder. During the treatment course, serum potassium showed a decline, with a value of 3.58 documented in August 2025. Potassium chloride supplementation was subsequently prescribed and later continued during follow-up. Although corticosteroid therapy may contribute to electrolyte disturbances in some

patients, the available case records do not establish corticosteroids as the definite cause of the potassium reduction [9]. Therefore, the potassium changes are best reported as an observed laboratory abnormality that was monitored and managed with supplementation. An important strength of this case is the longitudinal documentation of treatment response and recurrence over approximately eleven months. The repeated relationship between corticosteroid dose reduction and the appearance of new lesions provides a clinically relevant picture of the challenges involved in maintaining disease control. The introduction and subsequent continuation of MMF also demonstrate an attempt to reduce reliance on systemic corticosteroids. However, the case has important limitations. DIF and BP-specific autoantibody testing were not performed, and therefore immunopathological confirmation was unavailable. The clinical records also do not provide standardized disease-activity scores or detailed quantitative assessment of lesion burden at each visit. Furthermore, the final available follow-up was on 24 March 2026, at which time two new lesions were still present; therefore, complete remission cannot be claimed. Overall, this case emphasizes the importance of careful follow-up in patients with BP undergoing corticosteroid tapering. The development of new lesions at lower corticosteroid doses may indicate inadequate disease suppression and should prompt reassessment of the treatment strategy. In patients requiring prolonged systemic therapy, steroid-sparing approaches such as MMF may be incorporated into individualized management. The coexistence of parkinsonism further highlights the importance of considering neurological comorbidities when evaluating older patients with BP.

CONCLUSION

This case highlights the recurrent and treatment-dependent course of bullous pemphigoid in an older patient with Parkinsonism. Although the patient initially responded well to oral prednisolone, new lesions developed repeatedly during corticosteroid tapering, necessitating adjustment of therapy and the introduction of mycophenolate mofetil as a steroid-sparing agent. Recurrence after a period of treatment discontinuation further emphasizes the importance of individualized treatment strategies and close clinical monitoring during corticosteroid tapering and

withdrawal. The absence of direct immunofluorescence and BP180/BP230 antibody testing represents an important diagnostic limitation. Nevertheless, the characteristic clinical presentation, supportive histopathological findings, and longitudinal treatment response provide clinically relevant insight into the management challenges of recurrent bullous pemphigoid.

REFERENCES

1. Miyamoto D, Santi CG, Aoki V, Maruta CW. Bullous pemphigoid. *A Bras Dermatol*. 2019;94(2):133-46.
2. Amber KT, Valdebran M, Kridin K, Grando SA. The Role of Eosinophils in Bullous Pemphigoid: A Developing Model of Eosinophil Pathogenicity in Mucocutaneous Disease. *Front Med (Lausanne)*. 2018; 5:201.
3. Grochowska-Rak M, Kulig K, Grabowska A, Karlińska-Jonkisz V, Racisz P, Czop M, Pas HH, Lesiak A, Barasińska P, Matych M, Narbutt J. Sensitivity and Specificity of ELISA Tests Detecting BP180 and BP230 Antibodies in the Diagnosis of Bullous Pemphigoid: A Systematic Review and Meta-Analysis. *Int J Dermatol*. 2026;65(7):1367-75.
4. Borradori L, Van Beek N, Feliciani C, Tedbirt B, Antiga E, Bergman R, Böckle BC, Caproni M, Caux F, Chandran NS, Cianchini G, Daneshpazhooh M, De D, Didona D, Di Zenzo GM, Dmochowski M, Drenovska K, Ehrchen J, Goebeler M, Groves R, Günther C, Horvath B, Hertl M, Hofmann S, Ioannides D, Itzlinger Monshi B, Jedličková J, Kowalewski C, Kridin K, Lim YL, Marinovic B, Marzano AV, Mascaro J M, Meijer JM, Murrell D, Patsatsi K, Pincelli C, Prost C, Rappersberger K, Sárdy M, Setterfield J, Shahid M, Sprecher E, Tasanen K, Uzun S, Vassileva S, Vestergaard K, Vorobyev A, Vujic I, Wang G, Wozniak K, Yayli S, Zambruno G, Zillikens D, Schmidt E, Joly P. Updated S2 K guidelines for the management of bullous pemphigoid initiated by the European Academy of Dermatology and Venereology (EADV). *J Eur Acad Dermatol Venereol*. 2022;36(10):1689–704.
5. Liang S, Cohen J, Soter N. The Use of Mycophenolate Mofetil in the Treatment of Bullous Pemphigoid. *J Drugs Dermatol*. 2022;21(2):151-5.
6. Brick KE, Weaver CH, Savica R, Lohse CM, Pittelkow MR, Boeve BF, Gibson LE, Camilleri MJ, Wieland CN. A population-based study of the association between bullous pemphigoid and neurologic disorders. *J Am Acad Dermatol*. 2014;71(6):1191-7.
7. Lai YC, Yew YW, Lambert WC. Bullous pemphigoid and its association with neurological diseases: a systematic review and meta-analysis. *J Eur Acad Dermatol Venereol*. 2016;30(12):2007-15.
8. Liu S, Chen W, Chi C. Association Between Medication Use and Bullous Pemphigoid: A Systematic Review and Meta-analysis. *JAMA Dermatol*. 2020;156(8):891–900.
9. Zoungrana L, Traoré F, Ouédraogo MS, Ouédraogo B-WJC, Tapsoba P, Traoré S, Bognounou R, Ouédraogo NCJ, Ouédraogo NB, Korsaga NS. Profile of adverse effects of corticosteroid therapy in pemphigus and bullous pemphigoid according to the Common Terminology Criteria for Adverse Events (CTCAE) at the Yalgado Ouédraogo University Hospital in Ouagadougou. *World J Adv Res Rev*. 2026;29(1):1925-37.

Cite: Vishnumaya A. M., Angitha Binu, Drishya L.*, Shaiju S. Dharan, Recurrent Bullous Pemphigoid During Corticosteroid Tapering in A Patient with Parkinsonism: A Case Report, *Int. J. Med. Pharm. Sci.*, 2026, 2 (10), 14-18. <https://doi.org/10.5281/zenodo.23089051>