



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Case Study

A Case Report On Pemphigus Vulgaris Presenting With Extensive Mucocutaneous Erosions, Oral Candidiasis, And Electrolyte Disturbance

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ARTICLE INFO

Published: 1 Oct. 2026

Keywords:

Pemphigus vulgaris;
autoimmune blistering disease;
suprabasal acantholysis; oral
erosions; mucocutaneous
disease; corticosteroids;
mycophenolate mofetil.

DOI:

10.5281/zenodo.23074897

ABSTRACT

Pemphigus vulgaris (PV) is a rare, chronic, potentially life-threatening autoimmune blistering disorder characterized by the production of pathogenic autoantibodies against desmoglein-3 and desmoglein-1, resulting in intraepithelial acantholysis and blister formation. The disease commonly affects middle-aged individuals and frequently presents initially with painful oral erosions before progressing to involve the skin and other mucosal surfaces. Delay in diagnosis is common because early lesions mimic recurrent aphthous ulcers, candidiasis, or other inflammatory oral disorders. Histopathological examination remains the gold standard for diagnosis and demonstrates suprabasal clefting with acantholytic keratinocytes and the characteristic "tombstone" appearance of basal cells. This case highlights the importance of early recognition of persistent oral ulcerations, prompt histopathological confirmation, multidisciplinary management, and individualized immunosuppressive therapy for improving patient outcomes and preventing disease progression.

INTRODUCTION

Pemphigus vulgaris is a chronic autoimmune mucocutaneous blistering disorder characterized

by the production of IgG autoantibodies directed primarily against desmoglein-3 and, in many patients, desmoglein-1. These adhesion molecules are essential components of desmosomes

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



responsible for maintaining keratinocyte cohesion within stratified squamous epithelium. Autoantibody-mediated destruction of these proteins results in loss of cell-to-cell adhesion (acantholysis), leading to intraepidermal blister formation and painful erosions. Pemphigus vulgaris is an uncommon disease with an estimated annual incidence ranging from 0.5 to 5 cases per million population worldwide. The disease most commonly affects individuals between 40 and 60 years of age and occurs equally in both sexes, although slight female predominance has been reported in several epidemiological studies. Oral lesions represent the initial manifestation in nearly 70–90% of patients and may precede cutaneous involvement by several weeks or months. Painful oral erosions involving the buccal mucosa, palate, tongue, lips, and gingiva often interfere with eating, swallowing, speech, and oral hygiene, thereby significantly impairing quality of life. Because these lesions resemble aphthous ulcers, candidiasis, lichen planus, Behçet disease, and other ulcerative disorders, diagnosis is frequently delayed. Diagnosis relies on careful clinical examination together with histopathological confirmation and direct immunofluorescence. Histology typically demonstrates suprabasal cleft formation, intraepithelial acantholysis, and the characteristic "row of tombstones" appearance of basal keratinocytes. Direct immunofluorescence reveals intercellular deposition of IgG and complement C3 throughout the epidermis.

Systemic corticosteroids remain the cornerstone of therapy. Steroid-sparing immunosuppressive agents including azathioprine, mycophenolate mofetil, cyclophosphamide, methotrexate, rituximab, and intravenous immunoglobulin are used according to disease severity and treatment response. Early diagnosis and multidisciplinary management have substantially improved survival and long-term outcomes.

CASE PRESENTATION

PATIENT INFORMATION

A 64-year-old female was admitted with multiple painful oral ulcers of 10 days' duration. The lesions were associated with severe localized pain and a marked reduction in oral food intake. She also reported worsening upper abdominal discomfort and heartburn. She was discharged after a 9-day hospital stay and subsequently underwent outpatient dermatology evaluation following the biopsy result.

PAST MEDICAL HISTORY

The patient had long-standing type 2 diabetes mellitus managed with oral hypoglycemic therapy, systemic hypertension, coronary artery disease, bronchial asthma requiring inhaled/nebulized therapy, and parkinsonism treated with antiparkinsonian medications. Her baseline HbA1c was 11%. Chronic medications included sitagliptin/metformin-based therapy as documented, telmisartan, aspirin for coronary artery disease, inhaled budesonide/formoterol and levosalbutamol, Syndopa Plus, and pramipexole.

CLINICAL EXAMINATION

On admission, the patient was clinically stable. [9] Blood pressure was 130/80 mmHg, pulse rate ranged from 78 to 90 beats/minute, respiratory rate was 20–26 breaths/minute, and oxygen saturation was 98% on room air. Cardiovascular examination revealed normal S1 and S2 without murmurs or gallops. Respiratory examination revealed normal vesicular breath sounds with focal bilateral basal crepitations. The abdomen was soft with mild epigastric discomfort. No acute focal neurological deficit was noted apart from chronic neurological impairment related to parkinsonism.

Oral examination demonstrated extensive, severe, painful ulcerations involving the palatal mucosa, floor of the mouth, and upper and lower labial mucosa extending into the vestibular region. A



yellowish pseudomembranous discoloration was present over the dorsum of the tongue, clinically consistent with oral candidiasis. Swallowing and phonation were significantly painful.

INVESTIGATIONS

Baseline hematological investigations demonstrated a gradual decline in hemoglobin concentration from 13.9 g/dL on admission to 10.5 g/dL at discharge, with recovery to 13.2 g/dL during follow-up. Total leukocyte count and platelet count remained within normal limits throughout hospitalization, suggesting the absence of significant systemic bacterial infection or bone marrow suppression. Inflammatory activity was evidenced by persistently elevated C-reactive protein (CRP), which decreased progressively from 27.8 mg/L on admission to 16.9 mg/L at discharge, indicating a favorable response to treatment. Serum electrolyte analysis revealed progressive hyponatremia during hospitalization, with sodium levels declining from 136 mmol/L to 127 mmol/L before correction following therapeutic intervention. Mild hypokalemia was also documented during follow-up, with serum potassium decreasing to 3.0 mmol/L. Renal function remained preserved throughout admission, with serum urea and creatinine values remaining within normal reference ranges. These findings suggested electrolyte disturbances related to poor oral intake and systemic illness rather than intrinsic renal dysfunction.

Immunological investigations were negative for antinuclear antibodies (ANA), rheumatoid factor, anti-cyclic citrullinated peptide antibodies, cANCA, and pANCA, thereby excluding several autoimmune connective tissue disorders. Viral screening for HIV, hepatitis B surface antigen, and hepatitis C antibody was non-reactive. *Helicobacter pylori* IgG yielded an indeterminate result. Vitamin B12 levels were elevated secondary to supplementation, while thyroid

function remained normal. Transthoracic echocardiography demonstrated preserved left ventricular systolic function without regional wall motion abnormalities despite the presence of chronic bilateral pedal edema.

DIFFERENTIAL DIAGNOSIS AND MULTIDISCIPLINARY EVALUATION

Because of the severity and atypical nature of the oral lesions, a multidisciplinary approach was undertaken involving specialists from Dental Surgery, Cardiology, Rheumatology, Gastroenterology, Internal Medicine, and Dermatology. Dental Surgery evaluation identified extensive palatal, sublingual, and labial mucosal ulcerations associated with secondary oral candidiasis. Initial management included chlorhexidine mouthwash, topical triamcinolone oral paste, and clotrimazole mouth paint. Owing to persistent ulceration and suspicion of an autoimmune vesiculobullous disorder, an incisional biopsy was recommended.

Cardiology consultation optimized antihypertensive therapy and temporarily discontinued aspirin because of the anticipated biopsy procedure and increased risk of mucosal bleeding. Valsartan, dapagliflozin, and torsemide were initiated for better cardiovascular and volume management. Rheumatology evaluated the patient for recurrent oral ulceration associated with polyarthritis, and a pathergy test was planned to exclude Behçet's disease. Gastroenterology recommended outpatient upper gastrointestinal endoscopy because of persistent dyspepsia and severe epigastric discomfort.

HISTOPATHOLOGICAL EXAMINATION

An incisional biopsy obtained from the lower labial mucosa represented the diagnostic turning point in the patient's clinical course. Microscopic examination demonstrated non-keratinizing stratified squamous epithelium exhibiting



suprabasal cleft formation with numerous acantholytic keratinocytes. The basal keratinocytes remained attached to the basement membrane, producing the classical "tombstone" appearance. Mild lymphocytic infiltration with occasional eosinophils was identified within the underlying connective tissue. These characteristic histopathological findings established the diagnosis of **Pemphigus Vulgaris**.

CLINICAL EVOLUTION

Following discharge, the patient underwent Dermatology review. Despite improvement in oral

symptoms, disease progression was evident with the development of multiple erosive lesions involving the neck, anterior chest wall, and genital mucosa. Progressive dysphagia resulting from extension of oral lesions into the pharynx significantly compromised nutritional intake. These findings confirmed systemic progression of pemphigus vulgaris and justified initiation of definitive systemic immunosuppressive therapy.

THERAPEUTIC MANAGEMENT

Initial Inpatient Supportive Management

Table -1

Medication	Dose / Route	Schedule	Clinical Indication
Tolvaptan (Natrise)	15 mg PO	Once daily for 4 days	Correction of persistent hyponatremia
Fluconazole	150 mg PO	Once daily for 7 days	Treatment of oral candidiasis
Sompraz HP Kit	Oral combination therapy	Twice daily for 12 days	Gastric/anti-secretory management
Amoxicillin	250 mg PO	Twice daily for 12 days	Protection against secondary bacterial infection as documented [2]
Valsartan / Torsemide	80 mg / 10 mg PO [3]	Valsartan at night; torsemide in morning [4]	Blood pressure and fluid-overload management
Syndopa Plus	Standard oral dose	$\frac{1}{2}$ – $\frac{1}{2}$ – $\frac{1}{2}$	Maintenance therapy for parkinsonism

Initial management focused on stabilization of the patient's general condition, treatment of secondary infections, correction of electrolyte imbalance, and improvement of nutritional status.

Hyponatremia was managed with oral tolvaptan for four days. Oral candidiasis was treated using oral fluconazole together with topical clotrimazole mouth paint. Gastric symptoms were managed

with Sompraz HP Kit, while amoxicillin was prescribed for prophylaxis against secondary bacterial infection. Existing therapies for hypertension, coronary artery disease, bronchial asthma, diabetes mellitus, and Parkinsonism were optimized during hospitalization.

Definitive Dermatology Management

Table -2

Medication	Dose / Route	Frequency / Duration	Therapeutic Rationale
Prednisolone (Omnacortil)	30 mg PO	Once daily for 14 days	Systemic immunosuppression to



			control autoimmune blistering [5]
Pantoprazole	40 mg PO	Once daily for 14 days	Gastroprotection during corticosteroid therapy
Desloratadine	5 mg PO	Once nightly for 14 days	Symptomatic relief of cutaneous symptoms
Mucaine gel	5 mL PO	Three times daily, before meals	Mucosal coating and symptomatic relief of dysphagia [6]
Kenacort oral paste	Triamcinolone 0.1%	Topical, three times daily	Local anti-inflammatory treatment
Candid mouth paint	Clotrimazole	Topical, twice daily	Treatment/prevention of oral candidiasis
Mupirocin ointment	2% topical	Twice daily	Management of localized skin erosions

Following confirmation of pemphigus vulgaris, systemic corticosteroid therapy was initiated with oral prednisolone (30 mg daily). Pantoprazole was prescribed for gastrointestinal protection, while desloratadine was added for symptomatic relief of cutaneous manifestations. Topical therapy included triamcinolone oral paste for active oral erosions and clotrimazole mouth paint to prevent steroid-induced fungal overgrowth. Mucaine gel was prescribed before meals to reduce odynophagia and improve oral intake. Mupirocin ointment was applied to cutaneous erosions over the neck and chest.

Considering the patient's advanced age and multiple metabolic comorbidities, Dermatology planned to introduce **mycophenolate mofetil** during follow-up as a steroid-sparing immunosuppressive agent to minimize long-term corticosteroid toxicity.

CLINICAL OUTCOME AND FOLLOW-UP

The patient experienced gradual symptomatic improvement following initiation of comprehensive medical management. Oral pain decreased considerably, allowing improvement in food intake and hydration. Oral candidiasis responded well to antifungal therapy, and

correction of hyponatremia was successfully achieved prior to discharge. The patient was discharged in stable condition with instructions for close dermatological follow-up, blood glucose monitoring, blood pressure monitoring, electrolyte assessment, and gradual introduction of long-term immunosuppressive therapy. Continued surveillance was recommended for disease activity, corticosteroid-related adverse effects, electrolyte disturbances, and progression of mucocutaneous lesions.

DISCUSSION

Pemphigus vulgaris (PV) is a chronic autoimmune blistering disorder characterized by the production of pathogenic autoantibodies against desmosomal proteins, predominantly desmoglein-3 and, in patients with mucocutaneous disease, desmoglein-1. Disruption of desmosomal adhesion results in intraepidermal acantholysis and formation of fragile vesicles and bullae, which subsequently rupture and produce painful erosions. Oral involvement is particularly common in PV and may precede cutaneous manifestations. The present case demonstrated extensive mucocutaneous involvement associated with severe oral ulcerations, secondary oral candidiasis,



poor oral intake, and electrolyte disturbance, highlighting the complexity of managing PV in a patient with multiple comorbidities.

The clinical presentation of the present patient was comparable to the case reported by Janumpally Varshitha Thanmai et al.,^[21] in which a 55-year-old female presented predominantly with painful oral ulcerations, dysphagia, and burning sensation. Histopathological evaluation supported the diagnosis of pemphigus vulgaris, and the patient was treated with systemic corticosteroid therapy along with topical triamcinolone. In the reported case, prednisolone was initiated at a relatively higher dose of 60 mg/day and was subsequently tapered over a period of approximately six months. In comparison, the present 64-year-old female presented with extensive and painful oral ulcerations involving the palatal, sublingual, and labial mucosa, accompanied by dysphagia, poor oral intake, epigastric discomfort, and subsequent progression to the neck, chest, genital region, and pharyngeal mucosa. Prednisolone was initiated at 30 mg/day in the present case, along with topical and supportive therapies. The differences in corticosteroid dose and clinical progression may reflect differences in disease extent, comorbidities, clinical assessment, and treatment strategy.

An important additional finding in the present case was the presence of **secondary oral candidiasis**. The development of candidiasis was considered multifactorial rather than attributable to a single factor. The patient had extensive oral mucosal erosions secondary to PV, which resulted in disruption of the normal mucosal barrier. Such mucosal damage can facilitate colonization and proliferation of opportunistic organisms, including *Candida* species. Furthermore, extensive oral lesions were associated with pain, dysphagia, and reduced oral intake, which may have further compromised the patient's nutritional and general physiological status.

A particularly important contributing factor was the patient's **poorly controlled type 2 diabetes mellitus**, as evidenced by an HbA1c level of **11%**. Poor glycaemic control is associated with increased susceptibility to opportunistic infections, including candidal infections. Hyperglycaemia may promote fungal growth and can impair several components of host immune defence. Therefore, the markedly elevated HbA1c in the present patient represents an important predisposing factor for the development and persistence of oral candidiasis. In this context, the candidiasis should not be considered an isolated complication of PV but rather the result of the interaction between extensive mucosal injury, metabolic dysfunction, and medication-related factors.

Another relevant factor was the patient's treatment with an **inhaled corticosteroid-containing regimen, budesonide/formoterol**, for bronchial asthma. Inhaled corticosteroids are well-recognized risk factors for oropharyngeal candidiasis because deposition of corticosteroid particles in the oral cavity can produce local immunosuppressive effects and alter the local oral environment. The risk may be increased when inhaler technique is inappropriate or when patients do not adequately rinse and gargle the mouth after administration. In the present case, the concurrent use of inhaled budesonide, extensive oral mucosal erosions, and poorly controlled diabetes could have acted synergistically to increase susceptibility to *Candida* overgrowth. Therefore, inhaled corticosteroid exposure represents an important medication-related consideration in interpreting the occurrence of oral candidiasis in this patient.

The relationship between corticosteroid therapy and candidiasis is also clinically important because the patient subsequently required systemic prednisolone for control of pemphigus vulgaris. Systemic corticosteroid therapy can suppress



immune responses and potentially increase susceptibility to secondary infections. Thus, once candidiasis had developed, continued corticosteroid therapy required careful monitoring for persistence or worsening of fungal infection. In the present case, antifungal therapy with oral fluconazole and topical clotrimazole mouth paint was provided, and the oral candidiasis subsequently showed clinical improvement. The use of antifungal therapy alongside corticosteroid treatment illustrates the importance of balancing effective immunosuppression for autoimmune disease with prevention and management of opportunistic infections.

The subsequent initiation of dapagliflozin during hospitalization is also clinically relevant to the medication history. Dapagliflozin is an SGLT2 inhibitor and is associated particularly with an increased risk of genital mycotic infections because of increased urinary glucose excretion. However, its initiation occurred during the hospital course and should not be assumed to be responsible for the oral candidiasis that was already clinically evident. Therefore, temporal association should be considered when attributing adverse effects or infection risk to individual medications.

The presence of oral candidiasis in this patient is particularly important because oral pain and mucosal ulceration can substantially impair nutritional intake. The patient already experienced painful oral lesions and dysphagia, and superimposed candidiasis could further increase discomfort and interfere with eating and drinking. This may contribute to dehydration, nutritional compromise, and disturbances in electrolyte balance. In the present case, electrolyte abnormality, particularly hyponatremia, was observed and subsequently corrected during treatment. Although the electrolyte disturbance cannot be attributed solely to candidiasis, the combined effects of poor oral intake, systemic

illness, comorbid conditions, and medication-related factors may have contributed to the patient's metabolic imbalance.

The management of oral candidiasis with **fluconazole 150 mg once daily for seven days** together with **clotrimazole mouth paint** resulted in clinical improvement. This response supports the clinical assessment that the oral fungal infection was an important component of the patient's oral symptoms. The addition of topical antifungal therapy was particularly relevant because the patient had extensive oral mucosal involvement. The case also emphasizes the importance of monitoring for fungal overgrowth when systemic or inhaled corticosteroids are used. Clotrimazole was incorporated during treatment to reduce the risk of persistent or recurrent fungal involvement while corticosteroid therapy was continued. The present case also demonstrates the importance of differentiating primary PV lesions from secondary infectious complications. The painful erosions were primarily related to the autoimmune mucocutaneous disease, whereas the yellowish pseudomembranous discoloration of the tongue and other clinical findings were consistent with superimposed oral candidiasis.

Failure to recognize secondary infection may lead to inappropriate escalation of immunosuppressive treatment, potentially worsening the infection. Therefore, careful oral examination and early recognition of superimposed fungal infection are essential components of comprehensive PV management.

The extensive mucocutaneous progression observed in the present patient further emphasizes the need for continuous clinical assessment. In addition to oral lesions, involvement of the neck, chest, genital region, and pharyngeal mucosa indicated progressive disease activity. Pharyngeal involvement is particularly significant because it may worsen dysphagia and compromise oral intake. The planned introduction of



mycophenolate as an additional immunosuppressive strategy reflects the need for longer-term disease control while minimizing prolonged dependence on systemic corticosteroids where clinically appropriate.

Supportive treatment was another important component of management. Mucaïne gel was used to provide symptomatic relief of gastrointestinal and mucosal discomfort, while Kenacort oral paste and topical triamcinolone-related therapy provided local management of oral lesions. Desloratadine was used for symptomatic relief, and mupirocin was included for local management of skin lesions where indicated. These interventions demonstrate that treatment of PV extends beyond immunosuppression and requires a multidisciplinary approach involving dermatology, dental/oral care, medicine, nursing, and clinical pharmacy.

From a clinical pharmacy perspective, the case highlights several opportunities for medication optimization and patient counselling. In patients receiving inhaled corticosteroids, correct inhaler administration should be reinforced to minimize unnecessary oropharyngeal drug deposition. Patients should be advised to rinse and gargle the mouth with water after inhaled corticosteroid administration and avoid swallowing the rinse. Proper inhaler technique should also be assessed regularly, particularly in older patients and those with multiple comorbidities. In the present patient, these measures are especially relevant because inhaled budesonide represented an additional risk factor for oropharyngeal candidiasis. The coexistence of poorly controlled diabetes and corticosteroid exposure also emphasizes the importance of glycaemic monitoring. An HbA1c of 11% indicates substantial chronic hyperglycaemia and may increase vulnerability to infections and adversely affect recovery.

Corticosteroid therapy can further increase blood glucose concentrations, making monitoring of capillary blood glucose and appropriate adjustment of antidiabetic therapy clinically important during hospitalization and subsequent follow-up.

Overall, this case differs from the previously reported case by Janumpally Varshitha Thanmai et al. not only in age and disease progression but also in the presence of significant comorbidities, poorly controlled diabetes mellitus, secondary oral candidiasis, electrolyte disturbance, and the need for management of concomitant respiratory therapy. While both cases demonstrate the importance of systemic corticosteroids and topical therapy in controlling PV, the present case illustrates the additional challenges created by secondary infection and metabolic abnormalities. The occurrence of oral candidiasis in the present patient was therefore most appropriately interpreted as a **multifactorial complication**, with major clinically relevant contributors including extensive pemphigus-related mucosal disruption, poorly controlled diabetes mellitus with an HbA1c of 11%, inhaled corticosteroid exposure, and subsequent systemic corticosteroid therapy. Early recognition and treatment of candidiasis with antifungal therapy resulted in clinical improvement and allowed continued management of the underlying autoimmune disease. The case emphasizes the importance of comprehensive medication review, monitoring for treatment-related complications, optimization of inhaler technique, glycaemic control, oral hygiene counselling, and multidisciplinary follow-up in patients with pemphigus vulgaris and multiple comorbidities.

CONCLUSION

Pemphigus vulgaris should be considered in the differential diagnosis of persistent, painful oral ulcerations, particularly when lesions are



extensive, recurrent, and associated with dysphagia or nutritional compromise. In the present case, comprehensive multidisciplinary evaluation followed by histopathological examination established the diagnosis after exclusion of other autoimmune and infectious disorders. The coexistence of oral candidiasis, progressive hyponatremia, and multiple chronic comorbidities increased the complexity of management and highlighted the importance of individualized treatment. Early initiation of systemic corticosteroid therapy, together with appropriate antifungal treatment, correction of electrolyte imbalance, topical corticosteroids, and supportive care, resulted in clinical stabilization. Planned introduction of mycophenolate mofetil as a steroid-sparing immunosuppressive agent represents an evidence-based long-term strategy to maintain disease remission while minimizing corticosteroid-related adverse effects. This case highlights the importance of early biopsy in persistent oral lesions, multidisciplinary collaboration, continuous monitoring for treatment-related complications, and active involvement of clinical pharmacists in optimizing medication therapy and patient counseling. Early diagnosis and individualized immunosuppressive treatment remain fundamental to improving functional outcomes, preventing disease progression, and enhancing quality of life in patients with pemphigus vulgaris.

REFERENCES

1. Kasperkiewicz M, Ellebrecht CT, Takahashi H, Yamagami J, Zillikens D, Payne AS, et al. Pemphigus. *Nat Rev Dis Primers*. 2017;3:17026.
2. Murrell DF, Peña S, Joly P, Marinovic B, Hashimoto T, Diaz LA, et al. Diagnosis and management of pemphigus: recommendations of an international panel of experts. *J Am Acad Dermatol*. 2020;82(3):575-585.e1.
3. Joly P, Horvath B, Patsatsi A, Uzun S, Bech R, Beissert S, et al. Updated S2K guidelines on the management of pemphigus vulgaris and foliaceus initiated by the European Academy of Dermatology and Venereology. *J Eur Acad Dermatol Venereol*. 2020;34(9):1900-1913.
4. Schmidt E, Sticherling M, Sárdy M, Eming R, Goebeler M, Hertl M, et al. S2k guidelines for the treatment of pemphigus vulgaris/foliaceus and bullous pemphigoid: 2019 update. *J Dtsch Dermatol Ges*. 2020;18(5):516-526.
5. Joly P, Maho-Vaillant M, Prost-Squarcioni C, Hebert V, Houivet E, Calbo S, et al. First-line rituximab combined with short-term prednisone versus prednisone alone for the treatment of pemphigus (Ritux 3): a prospective, multicentre, parallel-group, open-label randomised trial. *Lancet*. 2017;389(10083):2031-2040.
6. Harman KE, Brown D, Exton LS, Groves RW, Hampton PJ, Mohd Mustapa MF, et al. British Association of Dermatologists guidelines for the management of pemphigus vulgaris 2017. *Br J Dermatol*. 2017;177(5):1170-1201.
7. Hertl M, Jedlickova H, Karpati S, Marinovic B, Uzun S, Yayli S, et al. Pemphigus: S2 guideline for diagnosis and treatment-guided by the European Dermatology Forum. *J Eur Acad Dermatol Venereol*. 2015;29(3):405-414.
8. Amagai M. Autoimmune and infectious skin diseases that target desmosomes. *Proc Jpn Acad Ser B Phys Biol Sci*. 2010;86(7):694-704.
9. Amagai M, Tsunoda K, Zillikens D, Nagai T, Nishikawa T. The clinical phenotype of pemphigus is defined by the anti-desmoglein autoantibody profile. *J Am Acad Dermatol*. 1999;40(2 Pt 1):167-170.



10. Harman KE, Seed PT, Gratian MJ, Bhogal BS, Challacombe SJ, Black MM. The severity of cutaneous and oral pemphigus vulgaris correlates with desmoglein 1 and 3 antibody levels. *Br J Dermatol.* 2001;144(4):775-780.
11. Scully C, Mignogna M. Oral mucosal disease: pemphigus. *Br J Oral Maxillofac Surg.* 2008;46(4):272-277.
12. Robinson JC, Lozada-Nur F, Frieden I. Oral pemphigus vulgaris: a review of the literature and a report on the management of 12 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997;84(4):349-355.
13. Mihai S, Sitaru C. Immunopathology and molecular diagnosis of autoimmune bullous diseases. *J Cell Mol Med.* 2007;11(3):462-481.
14. Schmidt E, Zillikens D. The diagnosis and treatment of autoimmune blistering skin diseases. *Dtsch Arztebl Int.* 2011;108(23):399-405.
15. Langan SM, Smeeth L, Hubbard R, Fleming KM, Smith CJP, West J. Bullous pemphigoid and pemphigus vulgaris--incidence and mortality in the UK: population based cohort study. *BMJ.* 2008;337:a180.
16. Kridin K. Pemphigus group: overview, epidemiology, mortality, and comorbidities. *Immunol Res.* 2018;66(2):255-270.
17. Kridin K, Schmidt E. Epidemiology of pemphigus. In: *Autoimmune Bullous Diseases*. Cham: Springer; 2020. p. 1-13.
18. Werth VP. Treatment of pemphigus vulgaris. *UpToDate*. Waltham (MA): Wolters Kluwer; 2024.
19. Payne AS, Hanakawa Y, Amagai M, Stanley JR. Desmosomes and disease: pemphigus as a model of desmosome dysfunction. *J Invest Dermatol.* 2004;122(5):1239-1250.
20. Kasperkiewicz M, David M, Schmidt E, Zillikens D. Current treatment of autoimmune blistering diseases. *Curr Drug Discov Technol.* 2009;6(4):270-280.
21. Janumpally Varshitha Thanmai J, Ramlal G, Tejaswi K, Mounica IL. Pemphigus vulgaris: a case report. *Pan Afr Med J.* 2022;42:184. doi:10.11604/pamj.2022.42.184.34184.

HOW TO CITE: Krishna sooraj, Grace N. Raju*, M. Shahbaz Zailu, Shaiju S. Dharan, A Case Report On Pemphigus Vulgaris Presenting With Extensive Mucocutaneous Erosions, Oral Candidiasis, And Electrolyte Disturbance, *Int. J. of Pharm. Sci.*, 2026, Vol 4, Issue 10, 135-144. <https://doi.org/10.5281/zenodo.23074897>

