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When the Mouth Speaks First: Isolated Oral Pemphigus Vulgaris—A Case Report and Literature Review

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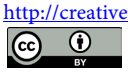

Abstract

Background: Pemphigus vulgaris (PV) is a rare autoimmune blistering disease in which oral lesions often precede cutaneous involvement. Early diagnosis may be delayed because of nonspecific presentations. **Case Presentation:** A 28-year-old woman presented with a one-year history of painful oral bullae and erosions without skin lesions. Clinical examination revealed multifocal erosions and a positive Nikolsky's sign. Histopathology demonstrated suprabasal clefting, acantholysis, and a "tombstone" basal layer, confirming PV. Systemic prednisone (60 mg/day) resulted in significant improvement within 10 days. Persistent inflammation related to impacted mandibular third molars was addressed surgically. By day 40, oral lesions had fully resolved; however, new-onset dysphagia raised concern for pharyngeal or esophageal involvement, necessitating referral to internal medicine. **Conclusion:** This case underscores the importance of early biopsy, recognition of local inflammatory triggers, and coordinated multidisciplinary care in isolated oral PV to prevent disease progression.

Subject Areas

Dentistry

Keywords

Pemphigus Vulgaris, Mouth Mucosa, Stomatitis, Autoimmune Diseases, Corticosteroids, Acantholysis, Immunofluorescence, Tooth Extraction

1. Introduction

Pemphigus vulgaris (PV) is a rare, chronic, potentially life-threatening autoimmune

bullous disease characterized by IgG autoantibodies directed against desmoglein 3 and, less frequently, desmoglein 1, leading to loss of keratinocyte adhesion and intraepithelial blister formation [1]. PV represents the most common subtype of pemphigus, accounting for approximately 70% of cases. It is also considered the most severe form of the disease [2] [3]. Oral lesions are often the first clinical manifestation [1]. According to a systematic review by Batistella *et al.*, the overall prevalence of oral involvement in pemphigus vulgaris patients was 90.3%, and that 50.8% of patients with PV presented exclusive oral manifestations [1]. Because early oral lesions present as nonspecific erosions or chronic stomatitis, diagnosis is commonly delayed, increasing the risk of disease progression and systemic complications [4] [5]. We report, by this paper, a case of isolated oral PV in a young female patient, highlighting the diagnosis challenges, the importance of early biopsy, and the benefits of comprehensive interdisciplinary management to prevent mucocutaneous extension.

2. Case Presentation

A 28-year-old female with no relevant medical or surgical history presented to the Dental Consultation and Treatment Center of Casablanca with chronic painful oral lesions evolving over more than one year. She reported recurrent episodes of fragile bullae affecting multiple oral sites, including the buccal and labial mucosa, lateral and ventral surfaces of the tongue, gingiva, vestibule, and floor of the mouth. The bullae ruptured rapidly, leaving painful erosions that interfered with mastication and speech, and caused dysphagia, resulting in a 10 kg weight loss over 8 months. No cutaneous lesions were noted. At baseline, the patient reported no involvement of extraoral mucosal sites, including ocular, nasal, pharyngeal, or genital regions. Additionally, she reported episodic pain in the region of the left mandibular third molar. No prior medical treatment had been administered.

Extraoral examination showed no facial swelling, no cutaneous lesions, and no cervical lymphadenopathy. Intraoral examination revealed multiple irregular erosions with erythematous borders distributed across the buccal and labial mucosa, lateral and ventral tongue, attached and free gingiva, vestibular mucosa, and floor of the mouth. Nikolsky's sign was positive on gentle pressure of the gingiva (**Figure 1**). A panoramic radiograph revealed impacted mandibular and maxillary third molars, suggesting a potential local inflammatory contribution (**Figure 2**). These clinical findings were suggestive of isolated oral pemphigus vulgaris.

A perilesional incisional biopsy was performed. Histological analysis showed suprabasal intraepithelial clefting with acantholysis and a "tombstone" appearance of basal keratinocytes, fibrinous exudate containing eosinophils, and a perivascular inflammatory infiltrate in the lamina propria composed of lymphocytes and polymorphonuclear cells. No evidence of viral cytopathic effect was observed (**Figure 3**). These findings were consistent with a diagnosis of pemphigus vulgaris in correlation with the clinical presentation. Direct immunofluorescence and serologic testing (indirect immunofluorescence or ELISA for anti-desmoglein 1 and

3 antibodies) were not performed due to financial constraints. Therefore, the diagnosis was established based on clinicopathological correlation.

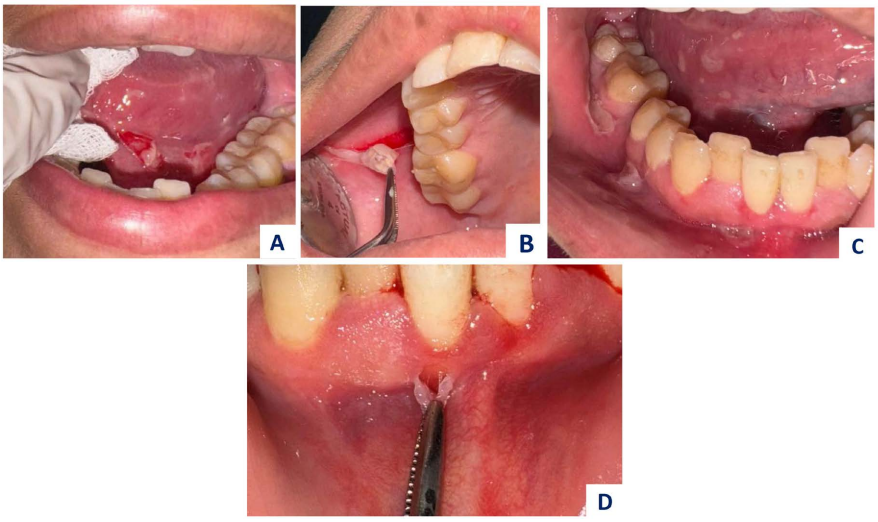

Figure 1. Intraoral manifestations of suspected oral pemphigus vulgaris. (A) Irregular erosions with erythematous borders involving the floor of the mouth. (B) Ruptured blister resulting in ulceration on the buccal mucosa. (C) Erosive lesions affecting the right lateral border of the tongue, vestibule, and adjacent gingiva. (D) Positive Nikolsky's sign on the gingiva, with epithelial detachment following gentle pressure.

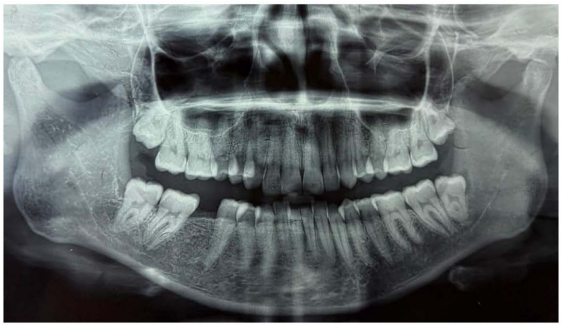

Figure 2. Panoramic radiograph showing impacted wisdom teeth.

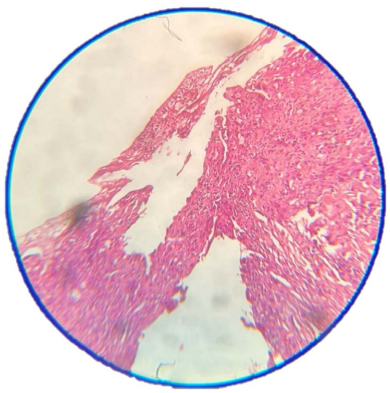

Figure 3. Histological section showing suprabasal clefting and acantholysis, consistent with pemphigus vulgaris.

The main differential diagnoses included mucous membrane pemphigoid, bullous pemphigoid, and erosive lichen planus. The combination of clinical presentation and histopathological findings, notably suprabasal acantholysis, strongly supported a diagnosis of pemphigus vulgaris. Viral infections were also considered but ruled out by the absence of cytopathic changes.

Systemic corticosteroid therapy was initiated with prednisone at a dose equivalent to 60 mg/day (3 tablets). Prior to treatment initiation, baseline evaluation included complete blood count, fasting blood glucose, and blood pressure assessment, and the patient was counseled regarding potential corticosteroid-related adverse effects. Preventive measures, including gastric protection and dietary advice, were also implemented, and clinical monitoring was scheduled during follow-up visits.

At the 10-day follow-up, most erosive lesions had markedly improved, except for a few persistent lesions on the right lateral border of the tongue (Figure 4). Given the suspicion of a persistent local inflammatory trigger, extraction of the mandibular and maxillary third molars was performed.

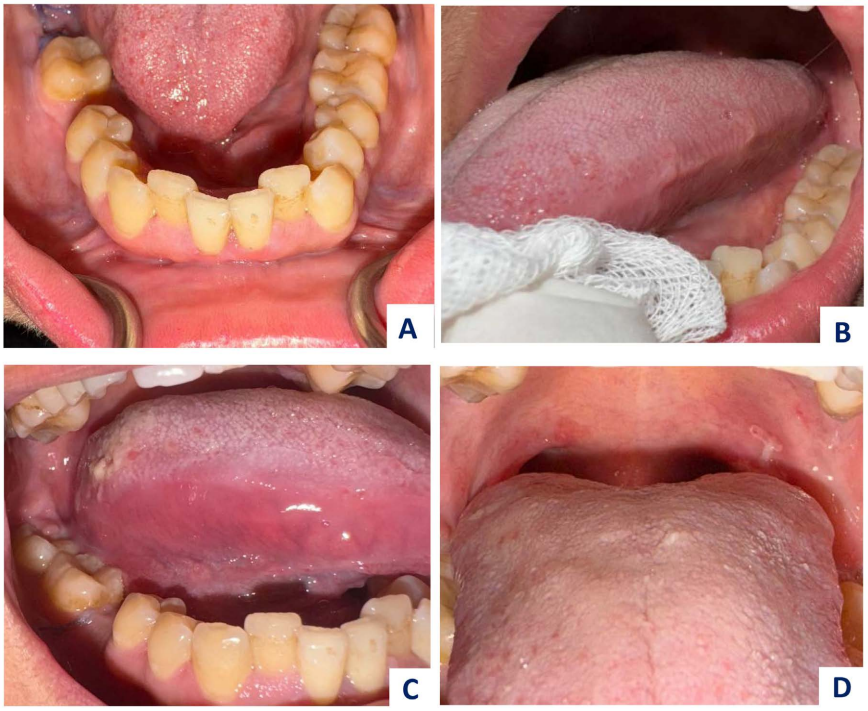

Figure 4. Follow-up of oral lesions after systemic corticosteroid therapy initiation. (A-B) General improvement of the lesions; (C-D) Persistent lesions on the right lateral border of the tongue and on the soft palate.

At day 30, the systemic corticosteroid dose was progressively tapered, as no new lesions had appeared during the initial two weeks, eventually reaching 20 mg/day (1 tablet). New oral lesions subsequently emerged; however, instead of increasing the systemic corticosteroid dose, a magistral preparation of dexamethasone in a Dologel base was prescribed for topical oral application, allowing good local con-

trol and stabilization of the lesions.

At the two-month follow-up, the oral lesions remained well controlled and the patient reported improved comfort. Nonetheless, lesions were noted on the soft palate, and the patient continued to report recurrent dysphagia, raising suspicion of possible pharyngeal or esophageal involvement. The patient was therefore referred to the internal medicine department for further evaluation and management. During this assessment, genital leukorrhea was identified, while upper gastrointestinal endoscopy has not yet been performed to confirm possible pharyngeal or esophageal involvement. No significant corticosteroid-related adverse effects were reported during the follow-up period.

3. Discussion

Pemphigus Vulgaris (PV) is a life-threatening, IgG-mediated autoimmune blistering disease characterized by acantholysis within the stratified squamous epithelium. The oral mucosa is the anatomical site that most frequently shows the first sign of PV, which appears in the form of erosions or ulcers. This often precedes cutaneous involvement by several months or even years. [1] [5]

This case report highlights an unusual presentation of PV in a 28-year-old female, who presented with chronic, painful oral erosions lasting over 4 months without any skin involvement. While PV has a slight female predominance, the typical age of onset is between 40 and 60 years [1] [6] [7]. The occurrence of this disease in a patient of only 28 years old is noteworthy and aligns with the minority of reports documenting juvenile or early-onset PV. The definitive diagnosis was established through histopathology, revealing classic intraepithelial clefting and acantholysis.

The pathogenesis of PV is well-defined by the autoimmune response against desmoglein (Dsg) proteins, which are critical components of the desmosomes responsible for keratinocyte adhesion. PV is primarily mediated by IgG autoantibodies targeting Desmoglein 3 (Dsg3), which is highly expressed in the oral mucosa, and/or Desmoglein 1 (Dsg1), which is predominantly expressed in the skin [2]. The binding of these antibodies to their respective targets activates a complex cascade of events, leading to the loss of cell-to-cell adhesion, a process known as acantholysis, which clinically manifests as suprabasal blisters and erosions [6] [8] [9]. In cases limited to the oral cavity, such as ours, the disease is generally characterized by a predominant anti-Dsg3 autoantibody profile [10].

A crucial aspect of this case is the potential identification of local inflammatory factors. The patient required the extraction of mandibular and maxillary wisdom teeth, which were identified as a possible local inflammatory triggering factor in the disease course. The literature increasingly recognizes that localized inflammation, trauma, or infection may act as crucial triggers or aggravating factors for autoimmune blistering diseases like PV [8] [11]-[13]. Specific oral triggers include local trauma, chronic infections such as pericoronitis or periodontitis, and alteration of the oral microbiome, which can induce inflammatory cytokines (IL-1, IL-

6, TNF-alpha) that contribute to disease activity. This case highlights the importance of a combined approach, integrating systemic corticosteroid therapy with the elimination of local dental foci to optimize disease control. Addressing both systemic and local contributors is essential, particularly as oral lesions tend to be persistent and slow to heal.

The establishment of a definitive PV diagnosis necessitates a systematic, multistep approach. The process begins with clinical assessment, where the presence of the Nikolsky sign, a key indicator of epithelial fragility, is highly suggestive of intraepithelial clefting [3] [14]. To provide a rapid, initial screening, a cytological smear (or Tzanck Test) is often performed, which involves scraping the base of a lesion to identify the characteristic presence of acantholytic cells (detached, rounded keratinocytes with large, hyperchromatic nuclei). However, cell specificity appears to be ambiguous when trying to distinguish the oral PV from an unexpected diagnosis of a malignancy [15] [16]. Definitive diagnosis relies on two principal methods: first, histopathology from a perilesional biopsy [17], which reveals the hallmark feature of suprabasal intraepithelial clefting with acantholysis; and second, direct immunofluorescence (DIF), considered the gold standard, which demonstrates the pathognomonic "fishnet" pattern of intercellular deposition of IgG and/or C3 within the epithelium. Finally, indirect immunofluorescence (IIF) is employed to detect and quantify circulating autoantibodies (Anti-Dsg3 and Anti-Dsg1) in the patient's serum, a step crucial not only for diagnosis but also for long-term monitoring of disease activity and therapeutic response [3] [4].

It is fundamental to confront the clinical data, including the presence of the Nikolsky sign, with these definitive laboratory and immunofluorescence results to effectively eliminate differential diagnoses, as the diagnosis of oral PV can be significantly delayed, when it's clinical presentations mimic more common chronic ulcerative conditions such as Erythema Multiforme (EM), Mucous Membrane Pemphigoid (MMP), or Erosive Lichen Planus (ELP) [6] [10] [11]. Czerninski *et al.* reported that the mean interval between the appearance of the first clinical sign and PV diagnosis ranged from 5.9 to 12 months, with an average delay of 6 months [18]. Moreover, although the patient in this case presented with strictly oral PV, it is important to recognize that this disease may occasionally coexist with other systemic or mucocutaneous disorders, which can modify its clinical presentation [19]. These unusual associations underscore the need for comprehensive intra and extraoral examination, even when the initial presentation appears limited to the oral cavity.

The primary goal of PV treatment is to halt blister formation, promote mucosal healing, and achieve clinical remission while minimizing treatment-related side effects [12]. The approach follows defined phases:

1) Induction Phase

The aim is rapid clearance, defined as the absence of new lesions and the healing of existing lesions (typically 80% re-epithelialization) within 2 weeks. Systemic

corticosteroids are the cornerstone of induction therapy [12] [20] [21].

Although corticosteroids are effective in treating PV, the optimal dosing is not known, but most clinicians start with 1 mg/kg per day equivalents of prednisolone. If doses greater than 1 mg/kg per day are required, delivery via IV pulses may be considered [20].

2) Maintenance Phase

Once control is achieved, corticosteroids are gradually reduced to a low maintenance dose (typically prednisone < 10 mg/day) or discontinued [21]. Immunosuppressive adjunctive agents (e.g., Azathioprine, Mycophenolate Mofetil, or Rituximab), also called steroïd-sparing treatments, are often introduced here to allow for the reduction of systemic corticosteroid dosage, thereby mitigating long-term adverse effects.

3) Cessation

Complete remission off therapy is possible, though rare before one year of treatment. Reported rates of complete remission after 3, 5, and 10 years are approximately 38%, 50%, and 75%, respectively [2].

Due to frequent relapses, long-term surveillance remains necessary, particularly given that oral lesions are often the most treatment-resistant sites.

For cases presenting with moderate disease limited exclusively to the oral cavity, such as the patient presented here, current international guidelines suggest a potentially lighter initial therapeutic approach [12]. For systemic corticosteroids, the induction dose may be lower (0.5 - 1 mg/kg/day) than that recommended for extensive mucocutaneous involvement (1 - 1.5 mg/kg/day) [10] [20]. Additionally, some authors suggest that high-potency topical corticosteroids may be sufficient to control mild and localized forms of the disease [12] [20]

It is important to note that while high-potency topical steroids (clobetasol or triamcinolone) are a standard treatment for localized PV, they are not always commercially or readily available in Morocco. In order to overcome this challenge, we used a magistral preparation combining dexamethasone with a suitable base (Dologel) for oral mucosal application, as recommended by the PNDS Pemphigus 2016 guidelines [22], leading to good local control and stabilization of the patient's oral lesions. This demonstrates the practical effectiveness of magistral formulations in settings where commercial preparations are unavailable.

Beyond conventional pharmacological approaches, modalities such as Low-Level Laser Therapy (LLLT) are emerging as valuable adjunctive treatments for PV. LLLT is proposed to facilitate wound healing, reduce pain, and decrease local inflammation, which is particularly beneficial for persistent and painful oral erosions that are refractory to systemic corticosteroids alone. A few studies, including randomized controlled trials, suggest that the local application of LLLT can promote the healing of oral lesions, positioning it as a valid supportive therapeutic option [23] [24].

Moreover, the healing kinetics of oral PV lesions are subject to notable variability influenced by both anatomic location and demographic variables. A study

by Nili *et al.* demonstrated site-specific differences in therapeutic response, reporting that lesions on the buccal mucosa and lower gingiva exhibited the most protracted healing course, in contrast to the posterior pharynx, which showed the fastest resolution. Furthermore, age emerged as a prognostic indicator, with slower improvement observed in the buccal mucosa and soft palate of older patients. Sex also imparted differential healing rates: female patients achieved a significantly higher rate of improvement in the soft palate, whereas male patients showed faster resolution of lesions on the lower gingiva [25].

While the management of PV primarily focuses on controlling oral and cutaneous lesions, it is imperative to maintain clinical vigilance for extension, particularly to the upper gastrointestinal tract (pharynx and esophagus). Such extensive involvement can lead to severe complications, including significant pain, nutritional deficits due to dysphagia, and aspiration risk. In this specific case, despite the successful initial control of the oral lesions following systemic corticosteroid induction, the patient's persistent dysphagia was a critical indicator necessitating a thorough investigation for esophageal or pharyngeal involvement. This persistent symptom prompted the urgent referral for specialized endoscopic evaluation and consideration of potential adjunctive therapies (e.g., Rituximab or other immunosuppressants). This case underscores that symptomatic improvement in one area (e.g., oral cavity) does not preclude activity in other less visible sites, and highlights the essential role of interdisciplinary collaboration in the comprehensive management of PV.

4. Conclusion

This case of early-onset, chronic, oral PV serves as a vital reminder to consider this rare but severe diagnosis when evaluating patients with persistent, non-healing oral erosions. Prompt and accurate diagnosis, confirmed by histopathology and immunofluorescence, allows for early initiation of treatment and improvement of the patient's prognosis and quality of life. Furthermore, meticulous local care, including the eradication of potential inflammatory or infectious foci such as periodontitis or pericoronitis, is a critical step that should be integrated into the comprehensive management plan to achieve remission and prevent aggravation and extension.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Batistella, E.Â., Sabino da Silva, R., Rivero, E.R.C. and Silva, C.A.B. (2021) Prevalence of Oral Mucosal Lesions in Patients with Pemphigus Vulgaris: A Systematic Review and Meta-Analysis. *Journal of Oral Pathology & Medicine*, **50**, 750-757. https://doi.org/10.1111/jop.13167

[2] Porro, A.M., Seque, C.A., Ferreira, M.C.C. and Enokihara, M.M.S.E.S. (2019) Pemphigus Vulgaris. *Anais Brasileiros de Dermatologia*, **94**, 264-278.

https://doi.org/10.1590/abd1806-4841.20199011

[3] Venugopal, S.S. and Murrell, D.F. (2011) Diagnosis and Clinical Features of Pemphigus Vulgaris. *Dermatologic Clinics*, **29**, 373-380. https://doi.org/10.1016/j.det.2011.03.004

[4] Dridi, S., Lutz, C.M., Gaultier, F., Bellakhdar, F., Jungo, S. and Ejeil, A.L. (2024) Oral Biopsy in Mucous Membrane Pemphigoid and Pemphigus Vulgaris with Gingival Expression: The Optimal Site. A Systematic Review and Meta-Analysis. *BMC Oral Health*, **24**, Article No. 1093. https://doi.org/10.1186/s12903-024-04853-y

[5] Kuriachan, D., Suresh, R., Janardhanan, M. and Savithri, V. (2015) Oral Lesions: The Clue to Diagnosis of Pemphigus Vulgaris. *Case Reports in Dentistry*, **2015**, Article ID: 593940. https://doi.org/10.1155/2015/593940

[6] Saccucci, M., Di Carlo, G., Bossù, M., Giovarruscio, F., Salucci, A. and Polimeni, A. (2018) Autoimmune Diseases and Their Manifestations on Oral Cavity: Diagnosis and Clinical Management. *Journal of Immunology Research*, **2018**, Article ID: 6061825. https://doi.org/10.1155/2018/6061825

[7] Cura, M.J., Torre, A.C., Cueto Sarmiento, K.Y., Bollea Garlatti, M.L., Riganti, J., Puga, M.C., *et al.* (2020) Pemphigus Vulgaris: A Retrospective Cohort Study of Clinical Features, Treatments, and Outcomes. *Actas Dermo-Sifiliográficas* (*English Edition*), **111**, 398-407. https://doi.org/10.1016/j.adengl.2019.10.009

[8] Tavakolpour, S. (2017) Pemphigus Trigger Factors: Special Focus on Pemphigus Vulgaris and Pemphigus Foliaceus. *Archives of Dermatological Research*, **310**, 95-106. https://doi.org/10.1007/s00403-017-1790-8

[9] Martinez, A.B., Corcuera, M.M., Ilundain, C.B. and Gómez, G.E. (2010) Oral Manifestations of Pemphigus Vulgaris: Clinical Presentation, Differential Diagnosis and Management. *Journal of Clinical & Experimental Dermatology*, **1**, Article ID: 1000112. https://doi.org/10.4172/2155-9554.1000112

[10] van Beek, N., Holtsche, M.M., Atefi, I., Olbrich, H., Schmitz, M.J., Pruessmann, J., *et al.* (2024) State-Of-The-Art Diagnosis of Autoimmune Blistering Diseases. *Frontiers in Immunology*, **15**, Article 1363032. https://doi.org/10.3389/fimmu.2024.1363032

[11] Hashimoto, T., Qian, H., Ishii, N., Nakama, T., Tateishi, C., Tsuruta, D., *et al.* (2023) Classification and Antigen Molecules of Autoimmune Bullous Diseases. *Biomolecules*, **13**, Article 703. https://doi.org/10.3390/biom13040703

[12] Filho, S.R.C., da Silva, L.A.M., Maia, C.R., de Andrade Santos, P.R., Alves, P.M. and de Andrade Santos, P.P. (2024) Oral Pemphigus Vulgaris Diagnostic Characteristics and Treatment: A Systematic Review. *Medical Molecular Morphology*, **58**, 1-22. https://doi.org/10.1007/s00795-024-00414-y

[13] Zorba, M., Melidou, A., Patsatsi, A., Poulopoulos, A., Gioula, G., Kolokotronis, A., *et al.* (2021) The Role of Oral Microbiome in Pemphigus Vulgaris. *Archives of Microbiology*, **203**, 2237-2247. https://doi.org/10.1007/s00203-021-02199-5

[14] Subadra, K., Sathasivasubramanian, S. and Aravind Warrier, S. (2021) Oral Pemphigus Vulgaris. *Cureus*, **13**, e18005. https://doi.org/10.7759/cureus.18005

[15] Kondo, S., Kawashima, J., Kobata, K., Ohgawara, T., Tanaka, S., Nabeshima, K., *et al.* (2017) Oral Pemphigus Vulgaris: Liquid-based Cytological Findings and Pitfalls. *Diagnostic Cytopathology*, **46**, 63-66. https://doi.org/10.1002/dc.23792

[16] Kuyama, K., Sun, Y., Endo, H., Kaneda, E., Morikawa, M., Wakami, M., *et al.* (2012) Pemphigus Vulgaris Macroscopically and Cytologically Resembling Oral Squamous Cell Carcinoma. *Open Journal of Stomatology*, **2**, 33-38. https://doi.org/10.4236/ojst.2012.21006

[17] Zeng, Q., Liu, J., Mu, J., Yang, J., Gao, Q., Wu, F., *et al.* (2023) Optimal Biopsy Site for the Diagnosis of Oral Pemphigus Vulgaris and Mucous Membrane Pemphigoid: A Systematic Review and Meta-Analysis. *International Journal of Oral and Maxillofacial Surgery*, **52**, 1162-1172. https://doi.org/10.1016/j.ijom.2023.05.005

[18] Czerninski, R., Abu Elhawa, M., Cleiman, M., Keshet, N., Haviv, Y. and Armoni-Weiss, G. (2025) Oral Involvement and Pain among Pemphigus Vulgaris Patients as a Clinical Indicator for Management—An Epidemiological and Clinical Study. *Applied Sciences*, **15**, Article 10145. https://doi.org/10.3390/app151810145

[19] Saddik, A., Hali, F., Chiheb, S., Elkhalifa, k. and Benyahya, I. (2025) Grinspan Syndrome Associated with Pemphigus Vulgaris Induced by COVID-19 Vaccination. *Cureus*, **17**, e88255. https://doi.org/10.7759/cureus.88255

[20] Geng, R.S.Q. and Sibbald, R.G. (2025) Pemphigus Vulgaris: Clinical Aspects and Treatments. *Advances in Skin & Wound Care*, **38**, 232-238. https://doi.org/10.1097/asw.0000000000000307

[21] Hussain, M.H., Tanweer, F., Sakagiannis, G., Mair, M., Mahmood, S. and Ashokkumar, S. (2021) Pemphigus Vulgaris and Bullous Pemphigoid of the Upper Aerodigestive Tract: A Review Article and Novel Approaches to Management. *ORL*, **83**, 395-403. https://doi.org/10.1159/000515229

[22] Centres de Référence des Maladies Bulleuses Auto-Immunes (2016) Protocole National de Diagnostic et de Soins (PNDS): Pemphigus. Ministère des Affaires Sociales et de la Santé. https://www.sfdermato.org/upload/recommandations/pnds-pemphigus-98f40f13cc0af3ec994b244ef35ee158.pdf

[23] Amadori, F., Bardellini, E., Veneri, F. and Majorana, A. (2022) Photobiomodulation Laser Therapy in Pemphigus Vulgaris Oral Lesions: A Randomized, Double-Blind, Controlled Study. *Stomatologija*, **24**, 80-84.

[24] Yousefi, M., Mansouri, P., Partovikia, M., Esmaili, M., Younespour, S. and Hassani, L. (2017) The Effect of Low Level Laser Therapy on Pemphigus Vulgaris Lesions: A Pilot Study. *Journal of Lasers in Medical Sciences*, **8**, 177-180. https://doi.org/10.15171/jlms.2017.32

[25] Nili, A., Karimi, S., Salehi Farid, A., Molhem Azar, P., Farimani, Z., Shahbazian, H., *et al.* (2022) Factors Associated with the Healing Time of Pemphigus Vulgaris Oral Lesions: A Prospective Study. *Oral Diseases*, **29**, 2248-2255. https://doi.org/10.1111/odi.14236