J. Oral Diagn 2025;10:e302

CASE REPORT

Gerardo Gilligan[1]*
Guillermina Marchetti[1]
René Panico[1]
Betiana del Valle Nieva[2]
Hernán Arce[2]
Roberto Navarro Guitart[3]
Mónica Herrero[4]
María Elsa Giovo[2]

# Childhood pemphigus vulgaris: case report and literature review

## Abstract:

Childhood Pemphigus Vulgaris is a rare entity, with an estimated incidence of 0.1–0.5 cases per 100,000 inhabitants per year, occurring between the ages of 13 and 19 years, with clinical and pathological features resembling those seen in adults. Clinical manifestations include the presence of flaccid, painful blisters, and secondary erosions affecting the skin and mucous membranes, particularly the oral mucosa. To date, only about 50 cases of this entity have been reported in the literature. The diagnosis relies on clinical assessment, complemented by histopathological examination and direct immunofluorescence to identify the presence of antibodies. We report the case of a 3-year-old patient with mucocutaneous lesions of 8 months duration, highlighting the clinical features, interdisciplinary diagnostic approach, and the use of rituximab as a first-line therapeutic agent in Childhood Pemphigus Vulgaris. In addition, a concise review of the literature from the past decade was conducted, focusing on the clinical characteristics of the disease presentation, with particular emphasis on oral involvement.

**Keywords:** Pemphigus, Pediatrics, Vesiculobullous.

## INTRODUCTION

Pemphigus vulgaris (PV) is a chronic blistering disease of autoimmune aetiology that affects the skin and mucous membranes. Among the various subtypes of pemphigus, PV and pemphigus foliaceus are the most common[1]. PV is considered rare in the general population, particularly among individuals under 18 years of age[2]. Typically, PV predominantly affects adults aged between 40 and 60 years. Childhood PV (CPV) is a rare entity, with an estimated incidence of 0.1–0.5 cases per 100,000 inhabitants per year. The onset of CPV typically occurs between the ages of 13 and 19 years, with clinical and pathological features resembling those seen in adult PV[3]. Clinical manifestations include the presence of flaccid, painful blisters, and secondary erosions affecting the skin and mucous membranes, particularly the oral mucosa, although lesions may also be found in the mucosa of the eyes, nose, oesophagus, genitals, cervix, and anus[4].

**Statement of Clinical Significance**
This case highlights the importance of early identification of oral lesions associated with Childhood Pemphigus Vulgaris in a 3-years old child. This report and literature review provides insights into the clinical spectrum of this entity and emphasizes the need for collaborative care involving other fields of medicine.

The pediatric variant of PV shares clinical, histopathological, and immunological similarities with adult PV. It encompasses CPV (0–12 years) and Juvenile Pemphigus Vulgaris (JPV) (13–18 years)[5]. While epidemiological investigations on the incidence of pediatric pemphigus are lacking, it is suggested that 1.4–2.9% of all PV are pediatric cases[6]. The rarity of CPV poses significant challenges

[1]Universidad Nacional de Córdoba, Facultad de Odontología, Oral Medicine Department – Córdoba, Argentina.
[2]Private Centre of Dermatology – Córdoba, Argentina.
[3]Sanatorio Allende, Hemato-Oncology Service – Córdoba, Argentina.
[4]Hospital Córdoba, Pathology Service – Córdoba, Argentina.
***Correspondence to:** Email: ggilligan@unc.edu.ar
Received on December 29, 2024. Accepted on March 17, 2025.
https://doi.org/10.5327/2525-5711.302

in understanding its etiopathogenesis and triggers of the autoimmune response associated with the disease. To date, only about 50 cases of CPV have been reported in the literature[5].

The diagnosis of CPV relies on clinical assessment, complemented by histopathological examination and direct immunofluorescence to identify the presence of antibodies[5]. This study aims to present a case of CPV in a 3-year-old patient with mucocutaneous lesions of 8 months duration, highlighting the clinical features, interdisciplinary diagnostic approach, and the use of rituximab as a first-line therapeutic agent in CPV. Furthermore, a concise review of the literature from the past decade was conducted, focusing on the clinical characteristics of CPV presentation, with particular emphasis on oral involvement.

## CASE REPORT

A 3-year-old female patient presented with highly painful oral lesions persisting for two months. Additionally, she exhibited skin manifestations on her arms, legs, abdomen, face, and superciliary area for the past 8 months, which had been unsuccessfully treated with antibiotics and oral acyclovir. The patient had no comorbidities or relevant family medical history.

During the examination, the patient displayed cooperation and motivation. Notable involvement of the lips was observed, with painful hematic and serohematic blisters (Figure 1A). The labial and buccal mucosa exhibited multiple ulcerative lesions with a whitish, necrotic appearance and visible detachment of superficial

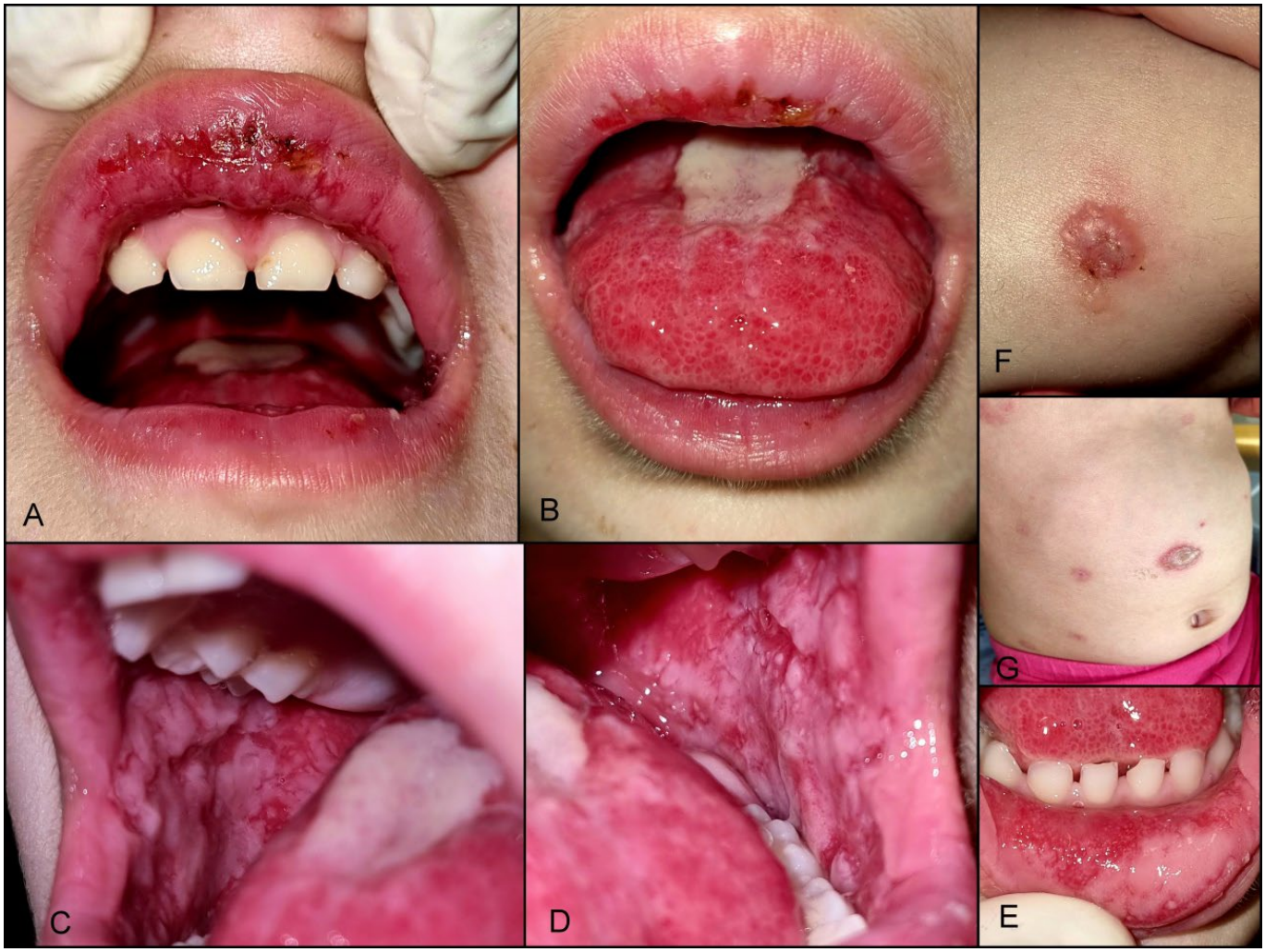


**Figure 1.** Lesions at the initial consultation. A) Erosive lesion covered by a hematous and serohematous crust on the upper labial mucosa. B) Strawberry tongue appearance, with marked inflammation and hypertrophy of the fungiform papillae. In the center of the dorsal surface, a very painful ulcer with necrotic base is observed. C and D) Multiple ulcers extending along both buccal mucosae, covered by a white necrotic-looking surface, with irregular appearance and visible detachment of superficial mucosal layers. E) Extensive ulcerative lesion on the lower labial mucosa, covered by a whitish necrotic-looking membrane. F) Serous blistering lesion on the arm skin. G) Multiple serous blistering lesions on the abdominal skin, with firm roof and non-inflamed base.

layers. The tongue showed significant inflammation with increased fungiform papillae, resembling a strawberry tongue. A painful necrotic ulcer was observed in the central part of the tongue, causing discomfort and compromised intake (Figure 1B). No submandibular lymphadenopathy was noted. The clinical presentation of oral lesions accompanied by genital and cutaneous involvement prompted consideration of several differential diagnoses, including Crohn's disease, Behcet's disease, Kawasaki-like disease, and other conditions associated with vesiculobullous lesions, such as herpetic gingivostomatitis, erythema multiforme, and linear IgA deposition dermatosis.

The patient was referred to a Pediatric Dermatology Service, where a physical examination revealed firm blisters with a well-consistent roof on non-inflamed skin surfaces (Figures 1F and 1G). Blistering lesions were also found in the vulva and perineum region. Laboratory tests showed elevated C-reactive protein (+++ 48) and erythrocyte sedimentation rate (40 mm/1 hour), but other values were within normal ranges(Red blood cells: 4,880,000/mm$^3$, hemoglobin: 14.80%, white blood cells: 8,700/mm$^3$, platelets: 225,000/mm$^3$, and blood glucose: 95 mg/100 ml). A biopsy of healthy and diseased skin from the arm was performed for histopathological examination and immunofluorescence. Histologically, an unroofed suprabasal intraepidermal blister was observed, with acantholytic cells or Lapidary cells attached to the basement membrane. A slight perivascular inflammatory infiltrate consisting of lymphocytes and eosinophils was present in the underlying dermis. Immunofluorescence revealed immune deposits in the intercellular substance, forming a characteristic lace or honeycomb pattern indicative of PV (Figure 2). The patient was hospitalized

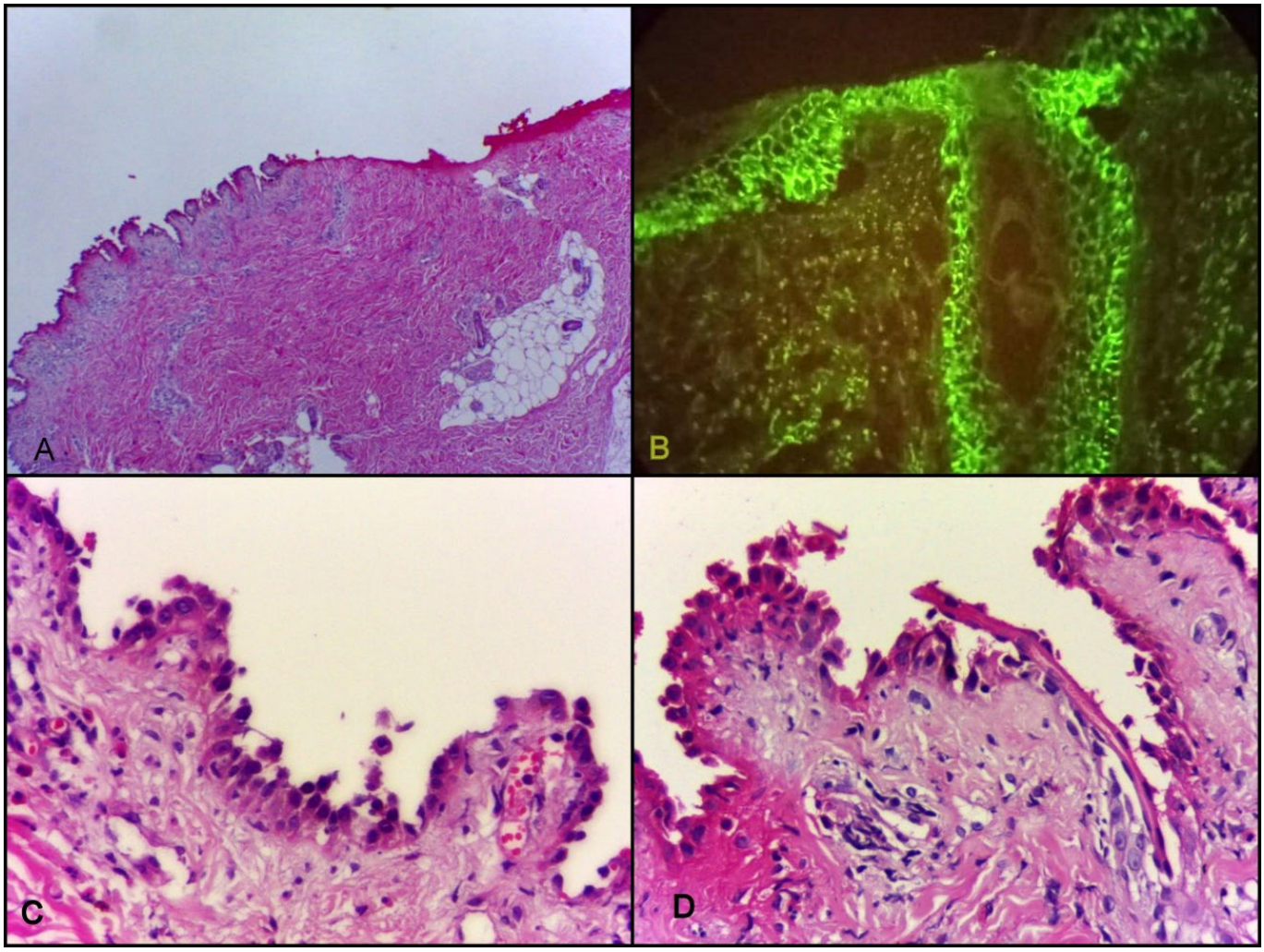


**Figure 2.** Histopathology and direct immunofluorescence. A) 10X H/E – Suprabasal intraepidermal blister with a slight perivascular inflammatory infiltrate consisting of lymphocytes in the underlying dermis. B) IFD – Immune deposits of Ig in the intercellular substance forming a characteristic honeycomb or chicken wire pattern indicative of PV. C and D) 40X H/E Basal cells with acantholytic cells attached to the basement membrane.

for 2 days to manage the systemic and oral condition with a corticosteroid pulse.

Treatment was initiated with Prednisone 1 mg/day, gradually tapered, and supplemented with Rituximab, an anti-CD20 monoclonal antibody, administered at a dose of 375 mg/m$^2$/dose, given in two doses separated by 15 days, with repeat administration every 6 months. Both skin and mucosal lesions resolved promptly. A year after diagnosis, the patient showed no oral lesions, although a fissured tongue, not previously noted, was evident during follow-up (Figures 3 and 4). The patient is currently being treated with Rituximab administered every 6 months.

### Literature research

A comprehensive literature search was conducted using PubMed, and MEDLINE, to identify articles related to childhood pemphigus vulgaris with oral manifestations, published between 2014 and January 2024. Full-article review was performed for eligible articles based on title and abstract. Manual searching of reference lists and previous literature searches in other databases was also performed. The review included case reports and case series publications, with exclusion criteria for articles without full-text access. A total of 41 articles were identified, of which 9 met the inclusion criteria. The reviewed articles provided data on patient demographics, clinical presentation, evolution, skin and oral mucosal involvement, diagnostic methods, and initial clinical suspicion. Table 1[3,5,7-13] summarizes the analyzed articles.

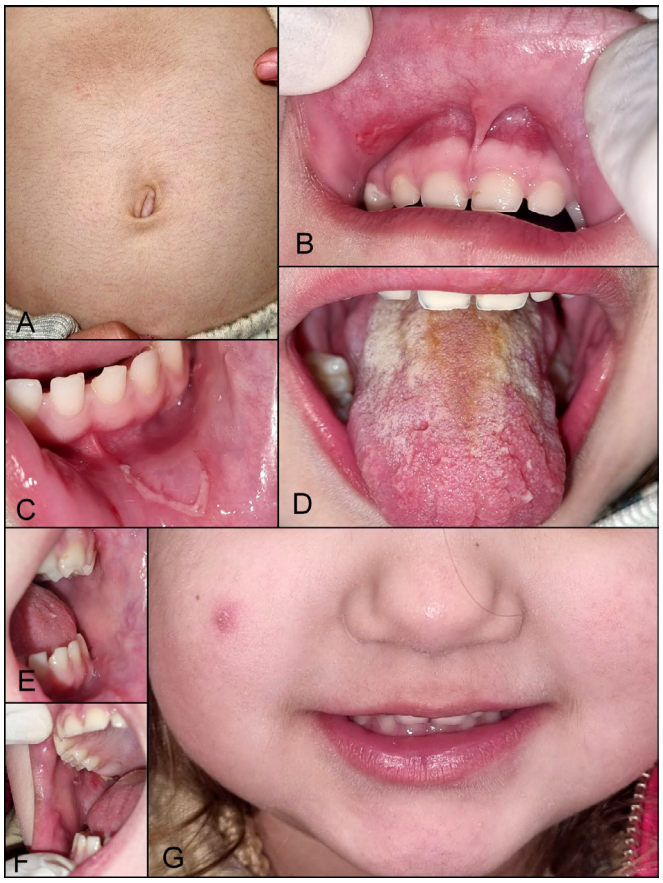


**Figure 3.** Evolution of lesions 6 months after the first application of Rituximab. A) Absence of multiple blisters in the abdominal area. B) Healthy upper lip mucosa. C) Partial remission of lesions in the lower lip mucosa; a superficial desquamation area persists. D) Dorsal surface of the tongue without inflammation and with resolution of the ulcerated lesion. E and F) Complete remission of multiple ulcerated lesions in the right and left buccal mucosa. G) Absence of clinical lesions on extraoral examination.

## DISCUSSION

PV is an autoimmune blistering cutaneous disease primarily affecting adults between 40 and 60 years of age[3]. Among the pediatric variants of PV, CPV affects children under 12 years of age and represents between 1.4% and 3.7% of all PV cases. CPV is a rare disease, with the first reported case dating back to 1955, and approximately 50 cases published since then[14]. According to our literature search, less than 20 cases of CPV with oral manifestations have been reported in the last 10 years.

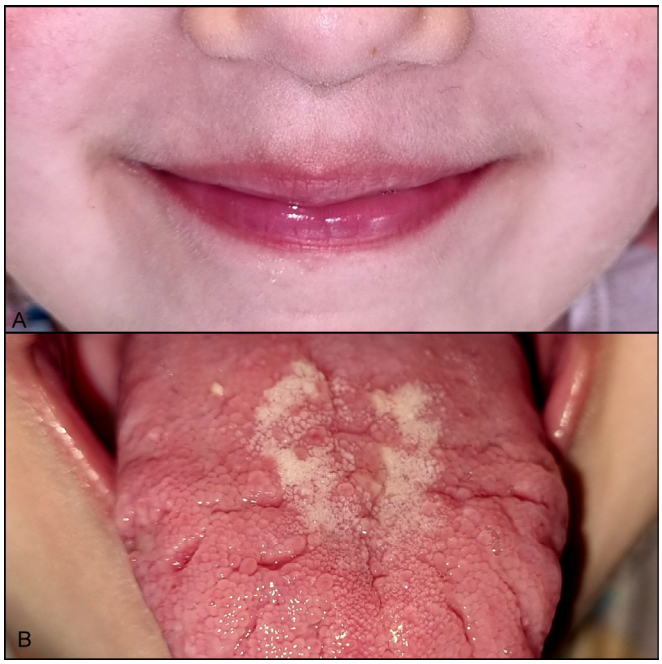


**Figure 4.** 1 year post Rituximab. A) Absence of clinical lesions in the perioral area. B) At the one-year follow-up after Rituximab treatment, nearly complete remission of oral blistering lesions was observed. Attention was drawn to the development of a fissured or cerebriform tongue with a centrally hyperpigmented area not reported in previous control visits. The lesion was asymptomatic.

**Table 1.** Childhood pemphigus vulgaris – literature review of clinical cases or case series from the last 10 years with emphasis on clinical characteristics.

| | Origin | Patients | Age | Gender | Chief complaint | Evolution | Skin involvement | Oral lesions | First clinical diagnostic hypothesis | Diagnostic method | Treatment |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Virtoso et al.[7] | Portugal | 1 | 10 | Male | Painful oral lesions | 3 months | No | Widespreads superficial erosions located on tongue, labial and buccal mucosa, and sialorrhea. | Infection ? | Biopsy, histopathology and DIF | Corticosteroids and azathioprin |
| Belleli et al.[8] | Argentina | 1 | 12 | Female | Painful oral lesions | 2 months | Skin and genital involvement | Extensive erosive lesions on the tongue and lips. | Herpes, Behcet disease, Herpetiform dermatitis, Paraneoplastic pemphigus. | Skin biopsy, histopathology and DIF | Prednisone and azathioprine. |
| Kincaid et al.[9] | Canada | 1 | 4 | Male | Painful bullous eruption | 5 days | Skin and genital involvement | Bullous and painful erosion on the tongue, lips, and palate. | Bullous multiform erythema | Skin biopsy, histopathology and DIF. Also, Indirect immunofluorescence with mokey esophagus substrate was positive for pemphigus (1:120). Circulating desmoglein 1 and 3 autoantiboidies were positive (1:320). | Rituximab |
| Patil et al.[11] | India | 1 | 11 | Female | Multiple eruptions and blisters all over the mouth | 3 months | Skin involvement | Tongue surface was eroded and erythematous, causing extreme discomfort. Also, crusty erosive surfaces with watery discharge were seen on the labial vermillion. | - | Skin, biopsy, histopathologyd and DIF. | Systemic treatment regime (dexamethasone, roxythromycin, prednisolone, hematopoietics, Na Cl saline). |
| Surya et al.[3] | India | 2 | #1: 10<br>#2: 12 | Male<br>Male | Painful oral lesions<br>Painful oral lesions and odinophagia | 10 months<br>6 months | No<br>No | Extensive superficial erosive lesions on the bilateral buccal mucosae, tongue, labial mucosa, gingiva, and palate. A single ulcer was noticed on the dorsal surface of the tongue. Small ulcers with slough on the buccal and labial mucosa, ventral surface of the tongue, and floor of the mouth. | Herpetic gingivostomatitis and Pemphigus<br>Pemphigus | Smear, biopsy and DIF<br>Tzank test (showing large round darkly eosinophilic acantholytic cells with hypertrophic nuclei). | Betamethasone and topical traiamcinolone<br>Betamethasone and topical traiamcinolone |
| Antonucci et al.[13] | Italy | 1 | 9 | Male | Skin and mucosal blisters | 2 months | Skin and genital involvement | Multiple oral lesions (not well-described) | - | Antidesmoglein (Dsg) 1 and anti-Dsg3 antibodies were both increased (196.57 RU/ml and 349.00 RU/ml, respectively). Biopsy and histopathologial diagnosis. DIF. | Predinone with azathioprine and three courses of Rituximab were ineffective. Mycophenolate mophetil with prednisone controlled the disease. |
| Khaddour et al.[14] | Syria | 1 | 14 | Female | Complete skin, mucosal lesions. Refractory aggressive bullous disease. | 3 months | Blisters and erosions all over her body and in the oral mucosa | Multiple oral lesions (not well-described) | - | Histopathology and DIF. | Refracory to conventional treatment. Plasmapheresis. (life-threatening case) |
| Lins et al.[5] | Brazil | 1 | 8 | Black male | Skin and oral lesions | Several years | Skin and genital involvement | Opening limitation, sialorrhea, and ulcers in the oral mucosa (upper and lower lip mucosa, the tongue's ventral surface, hard palate, soft palate, oral commissures, lower alveolar mucosa, and upper and lower gums). | - | Biopsy, histopathology and DIF | Prednisione and dapsone |
| Erdoğan et al.[10] | Turkey | 1 | 5 | White female | Non-healing oral lesions | 20 days | Skin and genital involvement | Crusted lesions on the lips and eroded lesions onthe buccal mucosa, and gingiva. | - | Biopsy, histopathology and DIF. Skin biopsy. Indirect immunofluorescence with monkey esophagus; positive results for cell surface antibodies (titer≥1 : 20) | Systemic methylprednisolone, oral dapsone. Given the ongoing disease activity, the patient received rituximab (clinical improvement without adverse events). |

Initially, CPV typically presents with significant involvement of the oral mucosa, including multiple blisters and painful erosions, as seen in our case. A notable feature in our patient was the appearance of a strawberry tongue, characterized by inflammation and increased fungiform papillae. Over time, other mucous membranes and skin may become affected, leading to symptoms such as weight loss, dehydration, fatigue, and dysphagia[4]. Although our patient initially presented with skin lesions, the oral mucosa became involved later. Despite the presence of oral lesions, the patient did not experience weight loss, dehydration, or fatigue.

The literature review focused on recent cases of CPV, comprising a total of 10 patients aged between 4 and 14 years, with a mean age of approximately 10 years (Table 1). The gender distribution was almost balanced, with six males and four females. The most common clinical manifestation was painful oral blisters. These lesions were often associated with extensive erosions and ulcers located on the tongue, labial mucosa, buccal mucosa, palate, and gingiva. Some patients also experienced sialorrhea, crusted lesions, or opening limitation of the mouth due to the severity of the mucosal involvement. Skin and genital involvement were noted in several cases, particularly those presenting with more severe or widespread disease. The primary diagnostic methodologies included biopsy, histopathological examination, and DIF, which were consistently used across all reported cases. In some instances, indirect immunofluorescence was performed, which demonstrated positive results for pemphigus-related antibodies. Furthermore, elevated levels of desmoglein 1 and 3 autoantibodies were detected in several cases, confirming the diagnosis of CPV. Several aspects of the cases reviewed in the literature search were comparable to the case presented in this report, particularly regarding the challenges in establishing a definitive diagnosis and the history of prior treatments based on incorrect diagnoses, which consequently delayed the final diagnosis. Notably, it was of interest to analyze the clinical presentation of a strawberry tongue in our case, as this manifestation has not been previously associated with CPV in earlier descriptions of this entity.

Diagnosing CPV can be challenging due to its rarity in children and its similarity to other blistering and ulcerative diseases affecting the oral cavity[2]. The differential diagnosis for CPV includes recurrent aphthous stomatitis, herpetic gingivostomatitis, erythema multiforme, linear IgA deposition dermatosis, paraneoplastic pemphigus, cicatricial pemphigoid and erosive lichen planus, and other conditions[2,15].

In our case, delayed diagnosis was attributed to the initial misdiagnosis and treatment of recurrent impetigo and herpetic stomatitis. Additionally, the presence of a strawberry tongue led to the consideration of other diagnoses. Strawberry tongue (or raspberry tongue) is a distinctive enanthem of the dorsum of the tongue characterized by the prominence of inflamed and hypertrophic fungiform papillae together with hyperemia. While it is a diagnostic feature of disorders such as Kawasaki disease and scarlet fever, it can also be seen in various other conditions (streptococcal pharyngitis, toxic shock syndrome, recurrent toxin-mediated perianal erythema, recalcitrant erythematous desquamating disorder, Yersinia pseudotuberculosis, yellow fever), being a useful diagnostic indicator[16]. In our case, this particular clinical finding led to other differential diagnoses such as scarlet fever and Kawasaki disease. The diagnosis of scarlet fever (associated with strawberry tongue) was ruled out because the patient's blistering skin lesions and clinical course (with lesions present for many months) were inconsistent with the acute course typically seen in scarlet fever, along with fever and other characteristic cutaneous manifestations of this disease. Moreover, the irregular, cobblestone appearance of the buccal mucosa observed in our patient has been described in systemic conditions such as ulcerative colitis and Crohn's disease[17]. Although this irregular appearance (cobblestone) raised suspicion about Crohn's disease, the skin biopsy findings and the presence of intraepithelial blisters ruled out Crohn's disease, so additional tests and studies for this condition were not pursued. As reported in the literature, there are numerous instances where patients are not accurately diagnosed with CPV, leading to delays in initiating appropriate treatment. CPV poses a significant diagnostic challenge for both dermatologists and oral medicine specialists.

CPV shares clinical, histological, and immunological characteristics with PV in adults, as their etiopathogenesis is similar[14]. However, CPV may exhibit a higher incidence of genital and conjunctival lesions compared to adult PV. Diagnostic tests for PV and CPV are similar[2,18], and the prognosis for children appears to be better. Deaths from CPV are usually caused by massive skin disease (greater than 70%), sepsis, pneumonia and altered serum electrolytes[2,3,7]. Adverse effects of chronic corticosteroid therapy are common, necessitating modifications to the therapeutic regimen.

Regarding treatment, our review revealed that therapeutic regimens varied across cases, commonly involving corticosteroids (e.g., prednisone, methylprednisolone), immunosuppressants (e.g., azathioprine,

mycophenolate mofetil), and biologics (e.g., rituximab). While some patients responded well to conventional treatments, others required more aggressive interventions, such as plasmapheresis, particularly in life-threatening cases. Overall, the findings suggest that CPV remains a challenging condition to diagnose and treat, especially in severe cases involving extensive skin and mucosal involvement. Immunosuppressive drugs are the mainstay of treatment for CPV, and their doses must be adjusted according to the age, weight, severity of the disease and side effects with prednisolone being the drug of choice in most cases. When high doses of steroids are required for prolonged periods, adjuvants such as mycophenolate mofetil and azathioprine may be added[3]. Some patients with CPV are refractory to conventional treatments and require additional therapies. Rituximab, a monoclonal antibody against CD20, has shown long-term safety and efficacy in CPV patients refractory to conventional treatments[2,19,20]. In our case, prednisone was initiated at 1 mg/kg/day with gradual tapering, in combination with Rituximab 375 mg/m2/dose, given in two doses separated by 15 days, and repeated every 6 months. Rituximab is primarily chosen due to its effectiveness in inducing remission in cases of refractory or recalcitrant CPV. Corticosteroids are the standard treatment, but their prolonged use is associated with significant adverse effects in the pediatric population, such as Cushing's syndrome, growth retardation, elevated liver enzyme levels, hypertrichosis, and oedema[21,22]. Moreover, several studies have shown high rates of complete or partial remission (97.8%) in patients treated with RTX, with adequate follow-up indicating a good prognosis in most cases. In general, the adverse effects reported with Rituximab in children with pemphigus vulgaris have been minor compared to conventional treatments. The most common side effects include infusion reactions (fever, urticaria, tachycardia, angioedema) and infections (Herpes zoster infection, upper respiratory tract infections). No long-term effects on growth and development have been reported. However, a systematic review on this topic demonstrated the need for more studies addressing its use in CPV[22]. In our case, the patient showed excellent progress, with no adverse effects and rapid remission of the lesions, along with the absence of recurrences. This treatment regimen led to significant clinical improvement after the initial applications, highlighting the safety and efficacy of Rituximab in very young patients. While Rituximab has been used for CPV treatment, our case is the first to report its use in the youngest patient to date.

An important limitation of this case report on CPV is that isolated or individual case reports, while valuable for understanding rare or unusual manifestations of the disease, do not provide a strong level of scientific evidence. These studies lack the breadth and generalizability that characterize controlled clinical trials or cohort studies. However, this particular case is of significant relevance due to its unusual clinical presentation, which mimics other oral diseases, challenging the differential diagnosis, and it is one of the youngest cases reported in the literature. Early identification of specific clinical signs, such as the combination of oral and cutaneous lesions, is crucial to avoid misdiagnosis. Furthermore, this case presents both diagnostic and therapeutic challenges, given the use of Rituximab at a young age — a treatment typically administered to adult patients — adding complexity to the management of this rare disease in the pediatric population.

## CONCLUSIONS

The case presented here posed a significant diagnostic challenge, with the definitive diagnosis being reached only through interdisciplinary collaboration among various medical specialities. This collaboration proved essential not only for accurate diagnosis but also for the effective treatment and ongoing management of the patient. Moreover, this case underscores the importance of reporting novel findings, such as the manifestation of a strawberry tongue in CPV, a feature not previously described in the literature.

Persistent cutaneous and mucosal vesicular-bullous lesions in infants, unresponsive to short-term treatments and characterized by recurrent episodes, should raise early suspicion for autoimmune diseases such as CPV.

## AUTHORS' CONTRIBUTIONS

GG: Conceptualization, Data curation, Formal análysis, Methodology, Project administration, Resources, Visualization, Writing – original draft, Writing – review & editing. GM: Conceptualization, Data curation, Resources, Visualization, Writing – original draft, Writing – review & editing. RP: Conceptualization, Supervision, Visualization, Writing – original draft, Writing – review & editing. BVN: Resources, Supervision, Visualization, Writing – original draft, Writing – review & editing. HA: Resources, Supervision, Visualization, Writing – original draft, Writing – review & editing. RNG:

Resources, Supervision, Visualization, Writing – original draft, Writing – review & editing. MH: Conceptualization, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing. MEG: Conceptualization, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing,

## CONFLICT OF INTEREST STATEMENT

**Funding:** The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

**Competing interests:** The authors have no relevant financial or non-financial interests to disclose.

**Ethics approval:** The parents of the patient provided informed consent for the publication of the clinical case. All examination and diagnostic procedures were conducted in accordance with health bioethics standards and the Declaration of Helsinki.

## REFERENCES

1. Kridin K, Schmidt E. Epidemiology of pemphigus. JID Innov. 2021;1(1):100004. https://doi.org/10.1016/j.xjidi.2021.100004
2. Vezzoli P, Parietti M, Carugno A, Di Mercurio M, Benaglia C, Zussino M, et al. Childhood pemphigus vulgaris during COVID-19 outbreak successfully treated with prednisone and azathioprine: a case report and literature review. J Clin Med. 2022;11(22):6858. https://doi.org/10.3390/jcm11226858
3. Surya V, Kumar P, Gupta S, Urs AB. Childhood pemphigus vulgaris: report of two cases with emphasis on diagnostic approach. Contemp Clin Dent. 2018;9(Suppl 2):S373-6. https://doi.org/10.4103/ccd.ccd_461_18
4. Malik AM, Tupchong S, Huang S, Are A, Hsu S, Motaparthi K. An updated review of pemphigus diseases. Medicina (Kaunas). 2021;57(10):1080. https://doi.org/10.3390/medicina57101080
5. Lins GT, Barbosa NLS, Abreu EMV, Costa KVT, Meneses KCB, Silva RN, et al. Childhood pemphigus vulgaris is a challenging diagnosis. Autops Case Rep. 2021;11:e2021267. https://doi.org/10.4322/acr.2021.267
6. Bilgic-Temel A, Özgen Z, Harman M, Kapıcıoğlu Y, Uzun S. Rituximab therapy in pediatric pemphigus patients: a retrospective analysis of five Turkish patients and review of the literature. Pediatr Dermatol. 2019;36(5):646-50. https://doi.org/10.1111/pde.13926
7. Virtuoso J, Ribeiro JF, Silva IS, Santos S, Fernandes P, Cabral F. Pemphigus vulgaris: a rare disease in childhood. J Paediatr Child Health. 2022;58(9):1661-3. https://doi.org/10.1111/jpc.15866
8. Bellelli AG, Mantero NM, Rueda ML, Navacchia D, Cao G, De Lillo L, et al. Childhood pemphigus vulgaris, a case report. Arch Argent Pediatr. 2016;114(6):e457-60. https://doi.org/10.5546/aap.2016.e457
9. Kincaid L, Weinstein M. Rituximab therapy for childhood pemphigus vulgaris. Pediatr Dermatol. 2016;33(2):e61-4. https://doi.org/10.1111/pde.12744
10. Erdoğan B, Meral EN, Topkarci Z, Hatipoğlu N, Kavak A. Long-term remission with rituximab therapy in a five-year-old patient with pemphigus vulgaris. J Dtsch Dermatol Ges. 2024;22(2):292-4. https://doi.org/10.1111/ddg.15299
11. Patil RU, Anegundi RT, Gujjar KR, Indushekar KR. Childhood occurrence of pemphigus. Int J Clin Pediatr Dent. 2017;10(2):196-200. https://doi.org/10.5005/jp-journals-10005-1434
12. Antonucci R, Locci C, Biondi G, Manconi A, Mannazzu R, Abis L, et al. Mycophenolate mofetil in the treatment of childhood pemphigus vulgaris. Pediatr Int. 2020;62(9):1123-4. https://doi.org/10.1111/ped.14327
13. Khaddour HH, Zaher D, Kassem T, Hasan A. Aggressive refractory pemphigus vulgaris that responded to plasmapheresis: a case report. J Med Case Rep. 2020;14(1):109. https://doi.org/10.1186/s13256-020-02421-w
14. Santoro FA, Stoopler ET, Werth VP. Pemphigus. Dent Clin North Am. 2013;57(4):597-610. https://doi.org/10.1016/j.cden.2013.06.002
15. Fuertes I, Guilabert A, Mascaró Jr JM, Iranzo P. Rituximab in childhood pemphigus vulgaris: a long-term follow-up case and review of the literature. Dermatology. 2010;221(1):13-6. https://doi.org/10.1159/000287254
16. Adya KA, Inamadar AC, Palit A. The strawberry tongue: what, how and where? Indian J Dermatol Venereol Leprol. 2018;84(4):500-5. https://doi.org/10.4103/ijdvl.IJDVL_57_17
17. Merkourea SS, Tosios KI, Merkoureas S, Sklavounou-Andrikopoulou A. Pyostomatitis vegetans leading to Crohn's disease diagnosis. Ann Gastroenterol. 2013;26(2):187. PMID: 24714872.
18. Gürcan H, Mabrouk D, Razzaque Ahmed A. Management of pemphigus in pediatric patients. Minerva Pediatr. 2011;63(4):279-91. PMID: 21909064.
19. 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 (EADV). J Eur Acad Dermatol Venereol. 2020;34(9):1900-13. https://doi.org/10.1111/jdv.16752
20. Kanwar AJ, Sawatkar GU, Vinay K, Hashimoto T. Childhood pemphigus vulgaris successfully treated with rituximab. Indian J Dermatol Venereol Leprol. 2012;78(5):632-4. https://doi.org/10.4103/0378-6323.100587
21. Kianfar N, Dasdar S, Mahmoudi H, Tavakolpour S, Balighi K, Daneshpazhooh M. Rituximab in childhood and juvenile autoimmune bullous diseases as first-line and second-line treatment: a case series of 13 patients. J Dermatolog Treat. 2022;33(2):869-74. https://doi.org/10.1080/09546634.2020.1788702
22. Shrivastava P, Mariam S, Abid L, Buch SA, Ahmad SA, Mansoori S, et al. Rituximab in childhood and juvenile pemphigus vulgaris: a systematic review. Cureus. 2024;16(4):e58288. https://doi.org/10.7759/cureus.58288