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CASE REPORT

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Case report on an unusual pathological triad of oral pyostomatitis vegetans, oral melanoacanthosis, and eosinophilic gastroenteritis in a pediatric patient

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Abstract

Background: Pyostomatitis vegetans (PV) is a rare chronic mucocutaneous disorder that is often associated with inflammatory bowel disease, and comparatively, oral melanoacanthosis is a benign reactive pigmentation of the oral mucosa. Eosinophilic gastroenteritis (EGE) is an uncommon gastrointestinal disorder characterized by eosinophilic infiltration of the gastrointestinal wall. To our knowledge, the coexistence of PV, oral melanoacanthosis, and EGE has not been previously documented. This report explores a potential immunological link among these three diseases and their overlapping inflammatory and clinical manifestations. **Case**: A 9-year-old Saudi girl presented with oral pigmentation. Her medical history was notable for variable allergies, with symptoms of dysphagia and abdominal pain. Intraoral examination revealed multiple brown macules on the lips, labial mucosa, gingiva, and tongue, as well as cobblestone-like folds in the buccal mucosa. Laboratory investigations demonstrated low iron levels and neutrophil counts, with elevated eosinophil counts and antinuclear antibody levels. Buccal mucosal biopsy revealed features consistent with PV and melanoacanthosis-like changes with melanocytic hyperplasia. These findings supported a diagnosis of PV with atypical melanocytic involvement and suspected gastrointestinal disease. Supportive measures for lip dryness (including lip care and hydration) were recommended, and the patient was referred to a pediatric gastroenterologist. Gastrointestinal work-up included a barium swallow (normal) and endoscopy, which showed moderate esophageal and gastric inflammation, and the biopsies confirmed EGE. The patient was subsequently started on dietary modification instructions, together with proton pump inhibitor therapy and oral corticosteroids, resulting in sustained clinical improvement and stable growth over a 1-year follow-up. **Conclusions**: This case describes a previously unreported triad of PV, oral melanoacanthosis-like changes, and EGE in a pediatric patient. The overlapping immunological and inflammatory features across these conditions raised the possibility of a common pathogenic mechanism, and awareness of this potential association may facilitate early multidisciplinary diagnosis and management in similar presentations.

Keywords

Pyostomatitis vegetans; Eosinophilic gastroenteritis; Inflammatory bowel disease; Melanoacanthosis; Buccal mucosa; Oral mucosa

1. Introduction

Pyostomatitis vegetans (PV) is a rare chronic inflammatory condition of the oral mucosa that is strongly associated with inflammatory bowel disease (IBD), particularly ulcerative colitis and Crohn's disease [1]. Clinically, PV presents as erythematous thickened oral mucosa that is often covered with multiple small pustules that may rupture to form the characteristic superficial erosions or ulcerations often described as "snail-track" lesions [2]. The buccal and labial mucosa and the gingiva are most commonly affected, whereas involvement of the soft and hard palate, lip vermillion, and tongue is reported less frequently [3]. Histopathologically, PV is characterized by intraepithelial or subepithelial abscesses containing eosinophils, typically accompanied by acanthosis and spongiosis [2, 4]. Although the pathogenesis of PV remains poorly understood, current evidence supports a role for immune dysregulation and chronic inflammation [4]. In this context, recent studies have highlighted the contribution of innate immune pathways, including the presence of neutrophil and eosinophil extracellular traps within lesional tissues [5]. Moreover, interleukin-36 (IL-36) accumulation in the affected

areas suggests a potential autoinflammatory mechanism that may amplify local inflammation and promote disease persistence or progression [5]. Based on this, PV is typically managed using immunosuppressive therapy to control both oral lesions and underlying systemic diseases [5]. Corticosteroids, administered either topically or systemically, are commonly used as first-line treatment, while patients with PV associated with active IBD may require biological agents, such as infliximab and adalimumab, to achieve remission of both oral and intestinal symptoms [2, 3].

Oral melanoacanthosis is a benign pigmented lesion histologically characterized by the presence of dendritic melanocytes within the acanthotic epithelium. Although melanoacanthosis-like changes are not commonly associated with PV, they may represent a reactive response secondary to chronic inflammation or trauma. Alternatively, this histopathological profile could indicate a distinct histopathological variant that warrants further investigation [5, 6].

Eosinophilic gastroenteritis (EGE) is a rare gastrointestinal disorder in children, characterized by eosinophilic infiltration of the stomach and/or intestines, typically presenting with nonspecific symptoms such as abdominal pain, vomiting, diarrhea, and failure to thrive. Recent studies have highlighted its association with allergic conditions (*e.g.*, food allergies and asthma) and have emphasized the pathogenic roles of dietary triggers and Th2-mediated immune dysregulation [7, 8]. A diagnosis of EGE relies on endoscopic biopsy demonstrating >20 eosinophils per high-power field and the absence of parasitic infections or malignancies [7, 9]. Peripheral eosinophilia and elevated immunoglobulin E levels are commonly observed and can support clinical suspicion, although neither is consistently present across cases [10]. First-line management includes dietary modifications (*e.g.*, elimination diets) and corticosteroid therapy; however, relapse rates remain high (21%–33% in steroid-administered patients) [10]. Emerging evidence further suggests that children with relatively low levels of eosinophilic infiltration (<14%) often respond well to methylprednisolone or montelukast, whereas those with severe infiltration (>14%) or elevated C-reactive protein (CRP) levels may require budesonide for sustained remission [7].

To the best of our knowledge, based on currently available literature, this study represents the first documented pediatric case of PV associated with EGE and concurrent oral melanoacanthosis-like changes. In this report, we primarily emphasize the diagnostic pathway and the distinctive clinicopathological features of this unusual triad.

2. Case presentation

A 9-year-old Saudi girl, accompanied by her father, was referred from her family's dental clinic to an oral medicine clinic for assessment of oral pigmentation. She was unaware of the pigmentation, which was identified incidentally during a dental examination. Her medical history was significant for allergic asthma of several years' duration, for which she was prescribed antihistamines on an as-needed basis. She also reported dysphagia to solid food intake and frequent abdominal pain. The parents were consanguineous, and her family had a history of lactose intolerance and gluten sensitivity, without other systemic diseases. She was allergic to fava beans, lentils, nuts, peanuts and sesame, and exposure triggered anaphylaxis that required emergency department management or administration of an epinephrine autoinjector (EpiPen; Meridian Medical Technologies, St. Louis, MO, USA).

2.1 Extraoral findings

Upon examination, the patient appeared underdeveloped (10th percentile in terms of growth), her skin was pale and dry, her lips were dry, and the oral commissures were fissured and crusted, with multiple brown macules on the upper lip (Fig. 1).

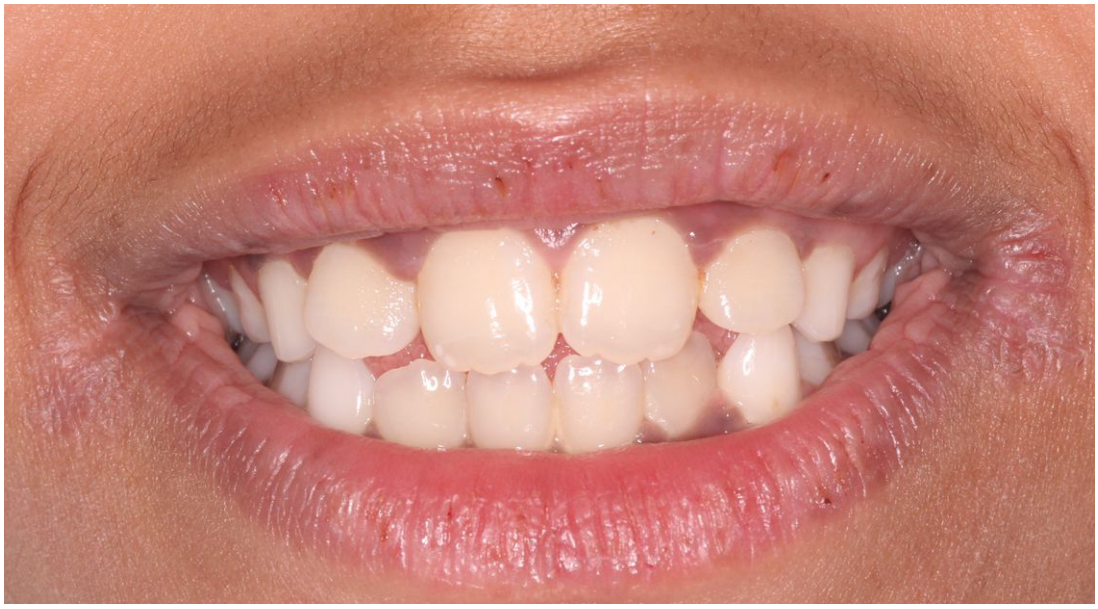

FIGURE 1. Extraoral findings. Clinical examination showing pale dry skin and dry lips with fissured and crusted commissures consistent with angular cheilitis. Multiple brown macules can also be observed on the upper lip.

2.2 Intraoral findings

Intraoral examination revealed multiple dark brown flat discolorations involving the labial mucosa, gingiva, and tongue (Fig. 2a). The buccal mucosa displayed multiple folds bilaterally, producing a cobblestone-like appearance (Fig. 2b,c). In addition, the dorsal surface of the tongue was fissured (Fig. 2d).

2.3 Diagnoses

Based on the cobblestone-like folds of the buccal mucosa, the initial differential diagnosis included inflammatory bowel disease-associated oral manifestations and focal epithelial hyperplasia (Heck's disease). However, the presence of multiple brown pigmented macules confined to the oral mucosa also raised consideration of systemic causes of mucosal hyperpigmentation, including Addison's disease or Peutz-Jeghers syndrome. The patient and her parents denied having skin or genital pigmentation or lesions. Laboratory investigations revealed low iron levels and a low neutrophil count, high eosinophil count, and high antinuclear antibody (ANA) levels (Table 1). All other parameters, including complete blood count with differential, cortisol, T4, adrenocorticotropic hormone (ACTH), and CRP levels, and erythrocyte sedimentation rate, were within normal limits.

2.4 Management and follow-up

A punch biopsy specimen was obtained from the left buccal mucosa for histopathological and immunohistochemical analyses. Hematoxylin and eosin staining revealed characteristic features consistent with PV, accompanied by melanoacanthosis-like changes (Fig. 3). Immunohistochemical staining with Melan-A markers revealed melanocytic hyperplasia in the basal cell layer and melanin-laden dendritic melanocytes distributed through the epithelium (Fig. 4). Taken together, the clinical presentation and histopathological findings, supported by peripheral blood eosinophilia, were consistent with a diagnosis of PV; however, the concomitant melanocytic hyperplasia represented an unusual feature in this context.

The patient and her family were reassured and instructed on appropriate lip care measures, including regular moisturization and adequate hydration. A topical preparation of nystatin combined with triamcinolone acetonide was prescribed to manage the angular cheilitis, and oral iron supplementation was recommended for the underlying iron deficiency.

Given the clinical context, she was also referred to a pediatric gastrologist to evaluate for concomitant gastrointestinal disease, which involved a fluoroscopic barium swallow test and upper gastrointestinal endoscopy with multiple biopsies. The fluoroscopic barium swallow test showed unremarkable results, whereas the endoscopy showed moderate inflammation and erosion with white patches and longitudinal furrows over the esophageal and gastric mucosal linings, with slight inflammation and few erosions in the duodenum. Histopathological assessment of the gastrointestinal biopsies supported a diagnosis of EGE.

The patient was initially managed by her immunologist with a dietary plan, which resulted in partial symptomatic improvement. After comprehensive gastroenterology evaluation, proton pump inhibitors (PPIs) were initiated at a dose of 20 mg twice daily for 3 months. However, as there was no clinical improvement with PPI, therapy was escalated to budesonide inhalation suspension at a dose of 1 mg/2 mL, resulting in considerable clinical improvement.

At the 1-year follow-up, the patient remained clinically stable, reported episodic abdominal pain and oral symptoms, and showed an improved growth parameter (50th percentile). She was advised to continue regular follow-up with her gastroenterologist and to promptly report any changes, such as increased abdominal pain and alterations in bowel habits. Reassessment by an oral medicine specialist was also recommended if the oral lesions became symptomatic or if any new changes were noted.

3. Discussion

This case describes an unusual presentation of PV associated with EGE and concurrent oral melanoacanthosis-like changes.

3.1 Process of diagnosis

The clinical presentation of cobblestone-like lesions in the buccal mucosa in a pediatric patient, along with widespread oral pigmentation, initially led to a broad range of differential diagnoses, including IBD, focal epithelial hyperplasia, Addison's disease, and Peutz-Jeghers syndrome [11].

Oral lesions associated with IBD may be classified as specific or non-specific and can precede, coincide with, or follow gastrointestinal symptoms. In particular, a cobblestone appearance on the buccal mucosa is recognized as a unique oral sign of Crohn's disease and, when considered alongside gastrointestinal complaints such as abdominal pain and diarrhea, appropriately places IBD high on the differential list.

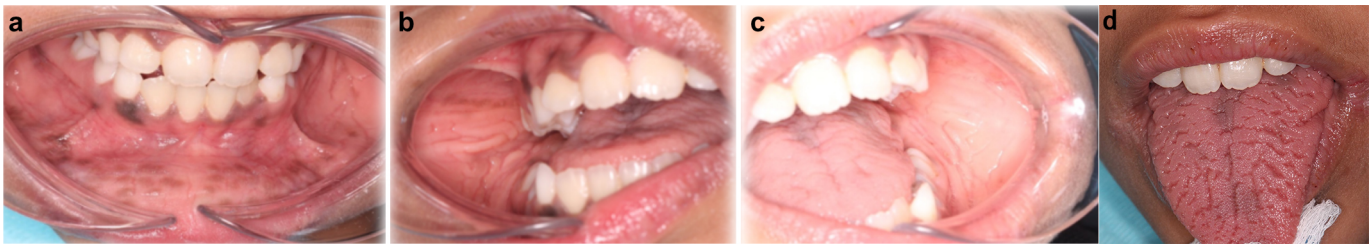

FIGURE 2. Intraoral findings. (a) Intraoral examination showing multiple dark brown, flat discolorations involving the labial mucosa, gingiva, and tongue. (b,c) The buccal mucosa with multiple bilateral mucosal folds, creating a cobblestone-like appearance. (d) The dorsal surface of the tongue appeared fissured.

TABLE 1. Laboratory results with normal range used.

Test	Result	Normal range
WBC	7.930	(4500–13.500)
RBC	5.4	(4.0–5.5)
Hgh	142.0	(115.0–145.0)
Hct	44.3	(35.0–45.0)
MCV	82.8	(80.0–91.0)
MCH	26.5	(23.0–31.0)
MCHC	321.0	(320.0–360.0)
RDW	13.4	(11.5–14.5)
Platelet	329.0	(140.0–450.0)
MPV	10.4	(7.2–11.1)
Neutrophil auto[#]	1.4 (L)	(1.5–8.0)
Neutrophil auto %	18.2 (L)	(40.0–55.0)
Lymphocyte auto[#]	3.3	(1.5–8.0)
Lymphocyte auto %	41.7	(38.0–42.0)
Monocyte auto[#]	0.6 (L)	(0.7–1.5)
Monocyte auto %	7.3	(3.0–9.0)
Eosinophil[#]	2.50 (H)	(0.20–0.80)
Eosinophil %	31.5 (H)	(0.0–6.0)
Basophil[#]	0.10	(0.00–0.20)
Basophil %	1.30 (H)	(0.00–1.00)
ESR	11	(0–17)
Iron	6.19 (L)	(9.0–22.0)
Ferritin	69.0	(13.0–130.0)
Folate	28.5	(10.40–78.90)
Vitamin B12	318.0	(145.0–569.0)
ANA	1.80 (H)	<1.160
ANA pattern	Fine speckled	
CRP	1.360	<3.0
Cortisol	479.0	(166.0–507.0)
ACTH	5.24	(5.0–60.0)
T4 free	17.30	(12.0–22.0)

ANA: antinuclear antibody; ACTH: adrenocorticotropic hormone; CRP: C-reactive protein; WBC: white blood cell count; RBC: red blood cell count; T4 free: free thyroxine; Hgh: hemoglobin; Hct: hematocrit; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; RDW: red cell distribution width; MPV: mean platelet volume; ESR: erythrocyte sedimentation rate. [#]: in numbers.

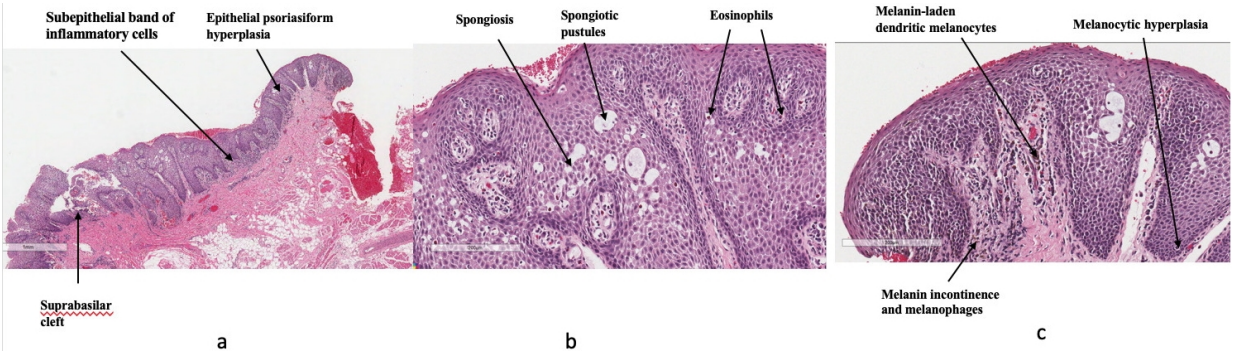

FIGURE 3. Histopathological examination. Hematoxylin and eosin-stained sections obtained from the left buccal mucosa through punch biopsy showing microabscesses. (a) Epithelium with psoriasiform hyperplasia and a suprabasilar cleft with a subepithelial band of inflammatory cells. (b) On high-power view, the epithelium demonstrates spongiosis, spongiotic pustules, and eosinophilia. (c) Melanocytic hyperplasia with melanin-laden dendritic melanocytes scattered throughout the epithelium, along with melanin incontinence and melanophages. Hematoxylin and eosin staining, original magnification ×40.

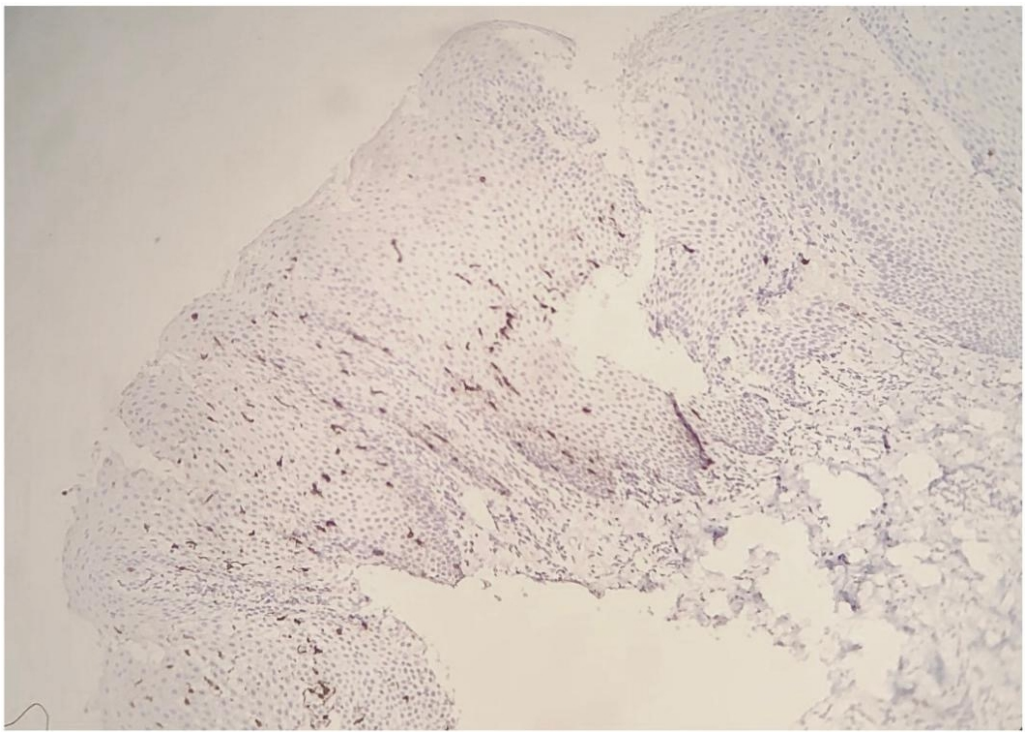

FIGURE 4. Immunohistochemical analysis. Specimen from the buccal mucosa showing melanin-laden dendritic melanocytes distributed throughout the epithelium. Melan-A, original magnification ×40.

At the same time, focal epithelial hyperplasia may clinically mimic a cobblestone-like mucosal pattern, and tissue biopsy is, therefore, essential for reliable distinction.

Focal epithelial hyperplasia is a benign human papillomavirus (HPV)-related growth of the epithelium with papillomatosis and hyper/parakeratosis. The epithelium typically shows broad, anastomosing rete ridges, prominent koilocytosis, "mitosoid" keratinocytes, minimal cytologic atypia, and a mild chronic inflammatory infiltrate. Cytopathic virus changes, such as koilocytosis in the top layers of epithelial cells and unique "mitosoid" keratinocytes with nuclei that look like they are dividing, are evident, but without observable dysplasia.

In regard to the multiple, asymptomatic brown macules confined to the oral mucosa, post-inflammatory pigmentation was considered; however, there was no reported history of preceding ulceration, mucosal irritation, or clinically apparent inflammatory triggers on oral examination. Peutz-Jeghers syndrome classically presents with focal/lentiginous mucocutaneous pigmentation in association with gastrointestinal polyposis, whereas Addison's disease typically manifests as more diffuse pigmentation accompanied by evidence of adrenal insufficiency. In this patient, normal cortisol/ACTH levels and absent extraoral pigmentation strongly favored neither diagnosis. Importantly, histopathological findings of epithelial acanthosis with dendritic melanocytic proliferation (Fig. 4) supported an oral melanoacanthoma diagnosis and helped refine the diagnostic interpretation [12].

Overall, this case underscores the importance of incorporating a comprehensive assessment of extra-oral manifestations during the diagnostic work-up, rather than limiting evaluation solely to intra-oral findings, which are essential for achieving an accurate and well-informed diagnosis. Given the presence of abdominal symptoms and histopathological findings, the provisional diagnosis favored IBD-related oral lesions in particular PV with oral melanoacanthoma-like changes.

3.2 Oral pyostomatitis vegetans and oral melanoacanthoma

PV most commonly presents with a characteristic snail-track appearance, which has been reported in up to 95% of cases, and it is strongly associated with inflammatory bowel disease (IBD), particularly ulcerative colitis [3, 7, 8]. Although the clinical presentation in this case was atypical, the histopathological findings of eosinophilic infiltration and intraepithelial microabscesses were consistent with PV. However, the additional finding of melanocytic hyperplasia is unusual, as concurrent pigmented oral lesions have not been emphasized in PV-associated disease [13, 14].

Oral melanoacanthosis is currently regarded as a reactive lesion, rather than a true neoplasm, commonly triggered by chronic irritation or inflammation. Histopathologically, it is characterized by epithelial acanthosis, intraepithelial dendritic melanocytes, and a chronic inflammatory infiltrate with melanophages [2, 15]. In our present case, pigmented lesions developed within areas affected by chronic pustular and erosive disease and showed features consistent with reactive oral melanoacanthosis (Figs. 2,3).

Moreover, our patient had pigmented oral lesions developed within areas already involved by chronic pustular and erosive disease (Fig. 2), and their distribution and microscopic appearance closely paralleled published descriptions of reactive oral melanoacanthosis, including acanthosis, intraepithelial dendritic melanocytes, and a background of chronic inflam-

mation (Fig. 3). Accordingly, two plausible explanations can be considered. First, the melanoacanthosis-like pigmentation may represent a secondary reactive phenomenon induced by long-standing inflammatory mucosal disease in the context of PV and EGE. Second, it may represent an incidental, unrelated pigmented lesion occurring in a child who also happens to develop PV and EGE. The first interpretation appears more likely because the timing (pigmentation emerging after or alongside intense mucosal inflammation), location (confined to clinically inflamed gingival/buccal mucosa), and histology (reactive pattern superimposed on chronic inflammatory infiltrate) all favor a shared inflammatory milieu driving both pustular and pigmented components. Nevertheless, we explicitly acknowledge that causality cannot be definitively established in a single case report, and this association should be viewed as hypothesis-generating, rather than proof of a unified pathophysiologic triad. Collectively, these observations underscore the importance of careful clinicopathological correlation when evaluating complex oral lesions in patients with suspected or confirmed gastrointestinal inflammatory disease.

3.3 EGE and oral melanoacanthosis

GE and related eosinophilic gastrointestinal diseases have been increasingly recognized in children, most often in the context of atopy or food allergy; however, reported oral manifestations remain uncommon and, when present, are usually nonspecific (*e.g.*, aphthous-like ulcers or nonspecific stomatitis). In contrast, oral melanoacanthosis is most often reported as an isolated mucosal finding or in association with local trauma, chronic irritation, or hormonal factors, and is not considered a typical feature of EGE. Notably, a review of available case reports and series did not reveal any pediatric patients in whom EGE and oral melanoacanthosis have been documented together as part of a single clinical entity [15, 16].

3.4 EGE and IBD

The patient's history of allergic asthma, dysphagia, and abdominal pain, coupled with peripheral blood eosinophilia, raised concern for an underlying systemic condition. The elevated ANA levels indicated an autoimmune component consistent with the association between PV and IBD [7, 8]. The patient exhibited an unusual co-occurrence of EGE and PV, which have not been directly linked in previous studies. Both EGE and PV are associated with IBD and exhibit similar immune dysregulation and eosinophil activation. Importantly, up to 40% of patients with EGE may subsequently be diagnosed with IBD [17]. In our patient, the coexistence of PV and EGE alongside marked eosinophilic infiltration illustrates this clinical overlap, reinforcing the importance of considering underlying IBD, such as ulcerative colitis, when such histopathological and immunological features are present.

3.5 Absence of combined PV, EGE, and oral melanoacanthosis in children

Across the accessible literature on PV (including large case compilations), eosinophilic gastrointestinal disorders, and oral melanoacanthosis, published cases typically described only one or, at most, two of these conditions coexisting in the same individual, most often PV with IBD or other neutrophilic dermatoses [13, 14]. No reports were found that describe the simultaneous presence of PV, EGE, and oral melanoacanthosis in a pediatric patient.

3.6 Immunological mechanism

The coexistence of PV, melanoacanthosis, and EGE underlines the clinical relevance of systemic manifestations of immune-mediated diseases. Although PV is well-documented as an extraintestinal manifestation of IBD [11], its relationship to oral melanoacanthosis has not been well characterized, and the present findings raise the possibility that chronic inflammation may serve as a unifying driver of melanocyte activation and eosinophilic infiltration under these conditions. Recent immunohistochemistry studies have shown that IL-36 is more active in the lesional mucosa of PV, especially in places where neutrophils and eosinophils actively infiltrate. IL-36 has been implicated in promoting the formation of Neutrophil and eosinophil extracellular traps (NETs and EETs) by neutrophils and eosinophils, consequently inducing autoinflammatory responses and perpetuating localized tissue damage in PV. This mechanism is associated with the activation and recruitment of supplementary immune cells and the secretion of extra pro-inflammatory chemicals [5].

In EGE, the expression of IL-36α and other IL-36 family members is similarly upregulated in inflamed gastrointestinal tissues, where they stimulate epithelial and stromal cells to secrete chemokines (such as C-X-C motif chemokine ligand 1 (CXCL1) and CXCL8) and other cytokines, thereby recruiting and activating eosinophils and neutrophils. This cytokine-driven amplification loop is thought to contribute to disease activity in EGE, leading to both epithelial barrier failure and chronic mucosal inflammation [18].

Beyond IL-36, both PV and EGE share an eosinophil-rich inflammatory profile with Th2-skewed inflammation as a central pathogenic mechanism. In PV, oral mucosal biopsies typically demonstrate mixed neutrophilic-eosinophilic microabscesses within a background of chronic inflammation, with elevated tissue eosinophils (>20/hpf) reported in up to 80% of cases and peripheral eosinophilia in most patients [19]. Similarly, EGE is defined by dense eosinophilic infiltrates (>30/hpf) predominantly affecting the lamina propria and muscularis mucosae of gastrointestinal tissues, and are often accompanied by Th2 cytokines (*i.e.*, IL-4, IL-5, IL-13) that promote eosinophil recruitment and activation. In addition to this adaptive immune profile, shared innate immune features have been proposed, including NETs and dysregulated keratinocyte/mucosal responses. Notably, recent studies have implicated IL-36 family cytokines (IL-36α, IL-36γ) in amplifying epithelial inflammation in both oral neutrophilic disorders like PV and eosinophilic gastrointestinal diseases. Collectively, these observations support the hypothesis that IL36 may synergize with Th2 cytokine pathways to sustain eosinophil recruitment, activation, and epithelial injury across oral and gastrointestinal mucosae, providing a mechanistic framework for the observed clinical overlap between PV and

EGE. Further translational work is required to validate IL36 as a therapeutic target in this shared disease spectrum [20, 21].

Our patient's prominent eosinophilic infiltrates, documented in both oral biopsies (mixed neutrophilic-eosinophilic pustules with Fig. 3) and gastrointestinal specimens (lamina propria eosinophilia consistent with EGE), align closely with these overlapping immunologic profiles, prompting a conceptual link between PV and EGE, potentially mediated by shared Th2-skewed inflammatory signaling within an IL-36-modulated pathways that may sustain mucosal and enteric inflammation across tissue barriers. However, we emphasize that this remains a hypothesis generated by the current case, supported by histologic parallels, but not by direct mechanistic proof (*e.g.*, cytokine assays or functional studies), and requires validation through larger series or experimental models.

3.7 Management

The management of similar cases should involve a multidisciplinary approach, including gastroenterology, to manage EGE and determine its association with oral PV [3, 8, 9]. Long-term follow-up is essential for monitoring the development of systemic symptoms and managing oral manifestations with topical/systemic corticosteroids, immunosuppressants, biologics, or a combination of these.

4. Limitations

The follow-up of our patient was only about one year, which restricts long-term conclusions on related disease progression, recurrence rates, and long-term outcomes associated with coexistent PV, melanoacanthosis, and EGE. Additionally, comprehensive molecular and genetic analyses, incorporating longitudinal monitoring and advanced molecular profiling, are needed to completely elucidate the shared immunological mechanisms.

5. Conclusions

Oral lesions in patients with systemic diseases should be regarded as clinically meaningful diagnostic—and, in some contexts, prognostic signs, rather than incidental findings. Early recognition of these manifestations may expedite diagnosis of underlying conditions, guide timely appropriate therapy and reduce associated morbidity.

In pediatric patients, dental practitioners serve a pivotal frontline role by identifying atypical, persistent, or otherwise unexplained oral lesions—particularly pustular or pigmented changes—that may indicate a systemic or gastrointestinal pathology. This role extends to prompt referral to relevant specialists (including oral medicine, dermatology, and pediatric gastroenterology) and active participation in multidisciplinary collaboration to support timely diagnosis and coordinated management. In parallel, careful elicitation and assessment of extraoral symptoms remain essential, as systemic features frequently provide critical diagnostic context and may prevent diagnostic delay.

Further investigations into the pathologic and immunologic interrelationships among PV, oral melanoacanthosis, and EGE are warranted to clarify shared mucosal immune dynamics and to inform future theranostic approaches. Until such evidence is available, comprehensive multidisciplinary care involving gastroenterologists, dermatologists, pediatric dentists, and oral medicine specialists remains essential for optimal patient outcomes.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

AUTHOR CONTRIBUTIONS

MAA—conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing-original draft, writing-review & editing, visualization, supervision, and project administration.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Institutional Review Board and Ethics Committee of the Dental College at King Saud University (IRB approval no. E-25-9792) and performed in accordance with the tenets of the Declaration of Helsinki. Written informed consent was obtained from the patient's father for the publication of this report with the accompanying images.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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