

Case Report

Pyogenic Granuloma of the Buccal Mucosa Mimicking an Atypical Vascular Tumor: A Case Report

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Abstract

Background: Pyogenic granuloma (PG) is an uncommon vascular proliferation that, when it arises intraorally, is predominantly gingival; buccal mucosal involvement is rare. Extralingual PG in elderly patients may mimic malignant vascular tumors. **Methods:** We report a 69-year-old woman with a 1.0 cm ulcerated mass of the left lower buccal mucosa that enlarged after biting trauma. The excised lesion was examined histopathologically and by immunohistochemistry for CD31, CD34, D2-40, S100, human herpesvirus 8 (HHV-8), Ki-67, and p53. Reports of buccal mucosal PG published up to December 2025 were reviewed. **Results:** The initial pathology report described an atypical vascular tumor with involved margins, requiring exclusion of hemangioendothelioma, angiosarcoma, and Kaposi sarcoma. Immunohistochemistry showed diffuse CD31 and CD34 positivity and preserved S100-positive perilesional nerve bundles, with absent HHV-8, a low Ki-67 index, and no aberrant p53 expression; the diagnosis was formally amended to PG. No recurrence occurred at 9 months despite involved margins. Among 11 previously reported cases, recurrence was documented in 6, none of which recurred. **Conclusions:** Buccal mucosal PG may mimic vascular malignancy in elderly patients, and immunohistochemistry can be decisive. Whether prognosis differs by site remains an open question requiring larger series.

Keywords: pyogenic granuloma; buccal mucosa; immunohistochemistry; angiosarcoma; case report

1. Introduction

Pyogenic granuloma (PG), also termed lobular capillary hemangioma, is a benign reactive proliferation of capillaries that arises in response to local irritation, trauma, or hormonal influences [1]. Although it is among the more familiar reactive lesions of the oral mucosa, PG is an infrequent finding overall, and the great majority of intraoral lesions are localized to the gingiva; extralingual sites such as the lips, tongue, palate, and buccal mucosa are involved far less often [2,3]. In the largest reported series, comprising 293 cases, the gingiva accounted for 83% of lesions whereas the buccal mucosa accounted for only 0.8% [4]. Occurrence at this site is therefore rare, and fewer than a dozen cases have been documented in the English-language literature.

Site appears to influence not only frequency but also behavior. Reported recurrence rates for oral PG range from approximately 3% to 23% [4], and because the great majority of reported lesions are gingival, these figures largely reflect gingival disease. By contrast, recurrence has not been documented among the reported buccal mucosal lesions. This



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difference has not previously been examined as a distinct phenomenon, and its anatomic basis has not been articulated.

Diagnosis becomes more difficult when PG occurs at an atypical site or in an atypical age group. Rapid enlargement, surface ulceration, and prominent endothelial proliferation may closely mimic malignant vascular tumors, particularly angiosarcoma and Kaposi sarcoma. In most cases these entities are distinguishable on routine histopathology, and immunohistochemistry serves as an ancillary technique reserved for diagnostically equivocal lesions [5,6]. We report a buccal mucosal PG in a 69-year-old woman that was initially reported as an atypical vascular tumor, and use this case together with the published literature to examine whether buccal mucosal PG carries a more favorable prognosis than its gingival counterpart.

2. Case Presentation

A 69-year-old woman was referred for evaluation of an enlarging mass of the left lower buccal mucosa near the oral commissure. She had first noticed the lesion approximately 2 years earlier, shortly after implant placement in the left posterior mandible; it was small at that time and remained stable thereafter. The lesion itself was painless, but she described mild tingling and discomfort of the perioral region. One month before presentation, the lesion enlarged after she accidentally bit it while eating. The lesion had therefore been present for approximately 2 years before this event, which is best understood as the stimulus for its rapid terminal enlargement rather than as the initiating cause. Her medical history included hypertension, hyperlipidemia, and daily aspirin use. She denied tobacco and alcohol consumption.

Extraoral examination was unremarkable. Intraorally, a well-circumscribed, sessile, reddish-pink nodule measuring approximately 1.0 cm in diameter was present on the left lower buccal mucosa (Figure 1). The surface was irregular and showed focal central ulceration with exposed granular red tissue, rimmed by a yellowish-white fibrinopurulent membrane (Figure 1); the lesion was soft, compressible, and bled readily on manipulation. No spontaneous bleeding, tenderness, or cervical lymphadenopathy was noted. Given the vascular appearance and recent rapid enlargement in an elderly patient, the clinical differential diagnosis included PG, Kaposi sarcoma, and low-grade angiosarcoma. Excisional biopsy was performed under local anesthesia. On the advice of the patient's physician, aspirin had been discontinued 7 days before the procedure; hemostasis was achieved by local measures, and no intraoperative or postoperative bleeding complications occurred.

The excised specimen measured $1.2 \times 1.0 \times 0.5$ cm. Tissue was fixed in formalin, embedded in paraffin, and stained with hematoxylin and eosin. Immunohistochemistry was subsequently performed for CD31, CD34, D2-40, S100 protein, human herpesvirus 8 (HHV-8), Ki-67, and p53, with appropriate positive and negative controls.

Histologic examination at low power showed a polypoid, dome-shaped lesion (Figure 2a). The exophytic mass comprised a lobular proliferation of capillary-sized vessels situated beneath a hyperplastic stratified squamous epithelium with elongated rete ridges. In the plane of section illustrated, the surface epithelium was intact; the clinically observed focal ulceration was not represented in this section. At higher magnification, the lobules comprised thin-walled, blood-filled capillaries lined by plump endothelial cells set in granulation tissue with an edematous, fibromyxoid stroma containing a mixed infiltrate of lymphocytes and neutrophils (Figure 2b). Prominent endothelial tufting and intraluminal projections were noted and initially raised suspicion for a vascular neoplasm.

The initial surgical pathology report characterized the lesion as an atypical vascular tumor with involvement of the resection margins, stated that differential diagnosis with hemangioendothelioma, angiosarcoma, and Kaposi sarcoma was required, and recom-

mended sequential immunohistochemical examination for confirmation. The case had been reviewed in intradepartmental consultation by a second pathologist, who concurred. The patient was informed of this report together with our clinical opinion that the appearance of the lesion—well circumscribed, superficial, soft, and 1.0 cm in diameter, without induration, fixation, or cervical lymphadenopathy—appeared inconsistent with a malignant vascular neoplasm. Re-excision for the involved margin was planned but deferred pending immunohistochemical confirmation, since the wound was healing uneventfully and definitive management depended on the final diagnosis.



Figure 1. Clinical photograph of the lesion. A well-circumscribed, sessile, polypoid reddish-pink nodule of the left lower buccal mucosa. The surface shows focal ulceration with exposed granular red tissue, bordered by a yellowish-white fibrinopurulent membrane.

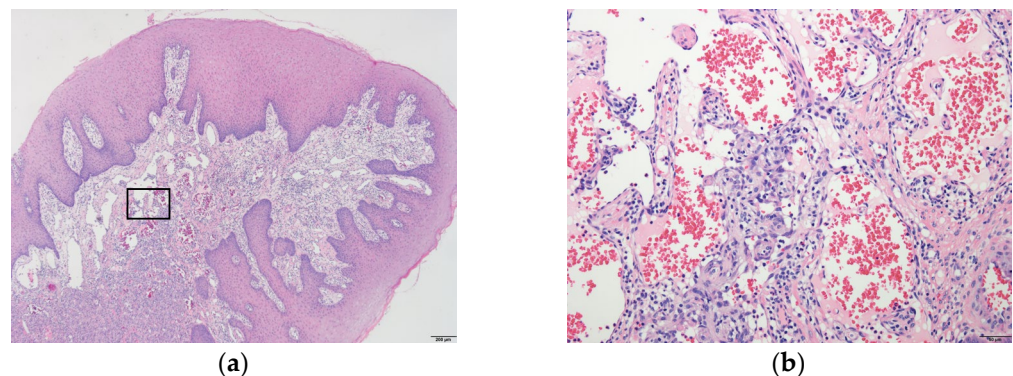


Figure 2. Histopathologic findings (hematoxylin and eosin). (a) Polypoid, dome-shaped lesion showing a lobular proliferation of capillary-sized vessels beneath a hyperplastic stratified squamous epithelium with elongated rete ridges; the surface epithelium is intact in this plane of section (scale bar = 200 μ m). The black frame indicates the area shown at higher magnification in the image on the right. (b) Thin-walled, blood-filled capillaries lined by plump endothelial cells within granulation tissue containing a mixed inflammatory infiltrate (scale bar = 50 μ m).

Immunohistochemically, the endothelial cells were diffusely positive for CD31 and CD34, confirming vascular lineage (Figure 3a,b), and negative for D2-40, excluding a lymphatic phenotype. S100 protein highlighted nerve bundles preserved adjacent to the lesion, with negative endothelium (Figure 3c). HHV-8 was negative (Figure 4a), the Ki-67 proliferative index was low (Figure 4b), and p53 showed no aberrant expression (Figure 4c).

Because angiosarcoma is itself characteristically CD31-positive, the endothelial markers served to confirm vascular lineage rather than to discriminate between the alternatives; the findings that argued against malignancy were the absence of cytologic atypia, the preserved S100-positive perilesional nerve bundles indicating non-infiltrative growth, the low Ki-67 index, and the wild-type p53 pattern, together with the absence of HHV-8. Following submission of these results, the diagnosis was formally amended to PG in a supplementary report issued 2 weeks after the original. As the lesion was benign and reactive, the planned re-excision was not performed. The postoperative course was uneventful, and no recurrence was observed at the 9-month follow-up despite the documented margin involvement.

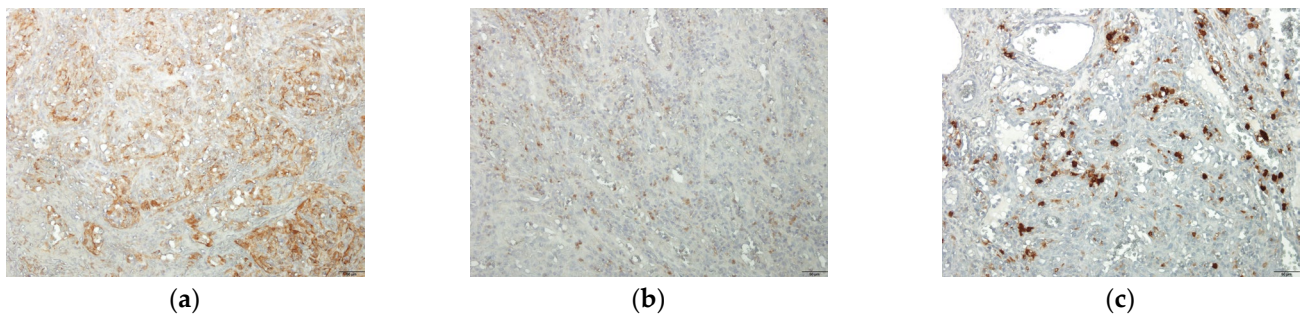


Figure 3. Immunohistochemical findings supporting the diagnosis (scale bars = 50 μ m; Mayer hematoxylin counterstain). (a) CD31 shows diffuse cytoplasmic and membranous positivity in the endothelium lining the proliferating capillaries. (b) CD34 shows endothelial positivity within the capillary lobules. (c) S100 protein highlights preserved nerve bundles and dendritic cells adjacent to the lesion, with negative endothelium.

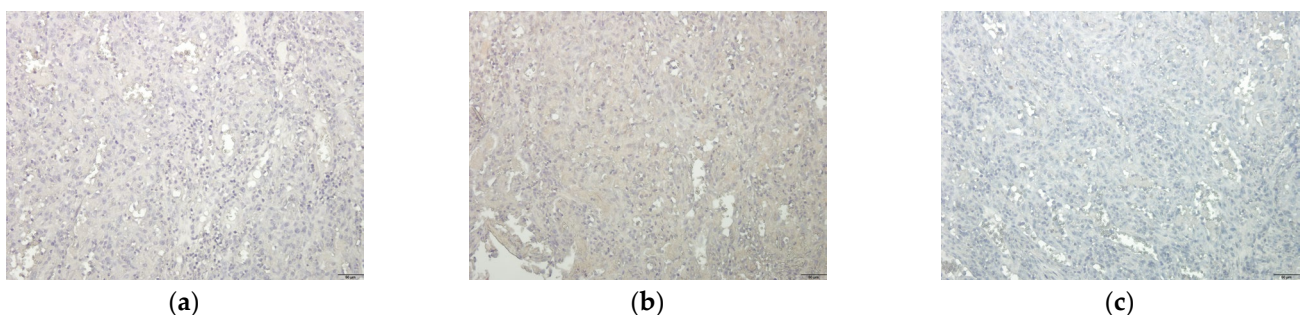


Figure 4. Immunohistochemical findings excluding malignancy (scale bars = 50 μ m; Mayer hematoxylin counterstain). (a) Human herpesvirus 8 is negative. (b) Ki-67 shows only scattered nuclear labeling, indicating a low proliferative index. (c) p53 shows no aberrant expression, consistent with a wild-type pattern.

3. Discussion

Oral PG is an infrequent lesion that is overwhelmingly gingival in location, and buccal mucosal involvement is rare. In a series of 293 oral PGs, the gingiva accounted for 220 of 262 lesions with a recorded site (83%) whereas only 2 lesions (0.8%) arose on the buccal mucosa [4]; a smaller series reported a higher proportion of buccal lesions, at 15% to 20% depending on histological subtype [7], indicating that estimates vary with case mix and referral pattern. Oral PG occurs most commonly in women, with a female-to-male ratio of 2.38:1 and a mean age of 27 years in the largest series [4], typically in response to chronic irritation, trauma, or hormonal influences including pregnancy [1]. In the present case the lesion appeared shortly after implant placement and remained small and stable for approximately 2 years; no persistent local irritant was documented during that period. The patient was postmenopausal and taking no hormonal medication, so a hormonal contribution is unlikely. Extragingival lesions have been attributed almost exclusively to

biting injury, whereas gingival lesions are associated with recently extracted teeth, calculus, dental appliances, and overhanging restoration margins [7]. The perioral tingling reported by this patient was most likely mechanical in origin, since the preserved S100-positive nerve bundles excluded perineural infiltration. This combination of advanced age, absence of a persistent irritant, rapid growth, and surface ulceration raised clinical suspicion of malignancy and made histopathologic confirmation essential.

PG is characterized by lobulated capillary growth, plump but cytologically bland endothelium, and non-infiltrative margins that preserve adjacent stromal structures, including peripheral nerves [1,7]. The principal histopathologic differential diagnoses in this case were Kaposi sarcoma and angiosarcoma. Kaposi sarcoma shows slit-like vascular spaces lined by spindled endothelial cells with extravasated erythrocytes and hemosiderin deposition [8], and is reliably identified by immunohistochemical detection of HHV-8 latency-associated nuclear antigen [9]; the absence of HHV-8 expression and of spindle-cell morphology excluded this diagnosis. Angiosarcoma may share rapid enlargement and easy bleeding with PG [10], but characteristically demonstrates cytologic atypia, infiltrative and destructive growth, frequent mitoses, and disruption of normal structures including nerves and vessels [11]. In the present case, intact S100-positive perilesional nerve bundles, absent cytologic atypia, a low Ki-67 index, and wild-type p53 expression together excluded malignancy. These markers are of particular value in elderly patients, in whom the pretest probability of a vascular malignancy is higher and the consequences of misdiagnosis greater.

The histopathological diagnosis of PG rests on four features: a polypoid architecture; a lobular proliferation of capillary-sized blood vessels; granulation tissue accompanied by an inflammatory cell infiltrate; and surface ulceration, classically covered by fibrin and a purulent membrane. All four were present in this lesion. The polypoid architecture, the lobular capillary proliferation, and the granulation tissue with inflammatory infiltrate are demonstrated in Figure 2. Surface ulceration with an overlying fibrinopurulent membrane was clearly evident clinically and is shown in Figure 1, although it was not captured in the plane of histological section illustrated; ulceration is characteristic of PG but is not invariable, being present in 27 of 30 (90%) lobular capillary hemangioma-type lesions in one comparative series [7]. It was rather the superimposed features—prominent endothelial tufting with intraluminal projections in an elderly patient—that obscured this otherwise typical picture and prompted the initial interpretation of an atypical vascular tumor.

Squamous cell carcinoma also merits consideration, particularly in an elderly patient with an ulcerated, rapidly enlarging exophytic mass. Marked pseudoepitheliomatous hyperplasia at the margin of an ulcerated PG may superficially resemble invasive squamous epithelium in a superficial biopsy. The distinction rests on the absence of cytologic atypia, abnormal mitoses, and true stromal invasion, and on recognition of the underlying lobular capillary architecture; adequate depth of sampling is important, as a superficial specimen may show only the reactive epithelial component.

Among reactive and inflammatory lesions, the principal differential diagnoses are peripheral giant cell granuloma, peripheral ossifying fibroma, irritation (traumatic) fibroma, and epulis granulomatosa. Peripheral giant cell granuloma is typically bluish-purple and gingival, and is distinguished by multinucleated giant cells in a hemorrhagic stroma. Peripheral ossifying fibroma is firmer, gingival in origin, and contains mineralized material. Irritation fibroma is pale, firm, and non-hemorrhagic, reflecting a collagenous rather than vascular composition. Epulis granulomatosa arises specifically within a healing extraction socket. In a clinicopathological series of lesions diagnosed clinically as PG, 9.5% proved to be peripheral giant cell lesions and 8.8% fibrous hyperplasia, indicating that clinical diagnosis alone is unreliable and that histopathological confirmation is required [4].

Several features may lead a PG to be regarded as atypical: an extralingival location, particularly the buccal mucosa, palate, or floor of mouth; presentation outside the usual second-to-fifth-decade range, especially in elderly patients; occurrence in the absence of any identifiable local irritant; rapid enlargement or a size exceeding approximately 2 cm; surface ulceration with a firm rather than soft consistency; recurrence after apparently complete excision; and histological features overlapping with vascular neoplasms, such as prominent endothelial tufting, intraluminal projections, or plump endothelium with hyperchromatic nuclei. The present case exhibited four of these. Clinically, oral PG most often appears as a red, reddish-purple, or pink nodule, either pedunculated or sessile, which bleeds readily on gentle manipulation; color and consistency vary with lesional age, early highly vascular lesions being soft, compressible, and deep red, whereas older lesions undergoing fibrous maturation become firmer, paler, and bleed less readily. In the largest reported series, the lesions were predominantly red (73.2%), soft (62.3%), and pedunculated (61.1%), with a mean diameter of 1.3 cm [4]. The present lesion was reddish-pink, soft, compressible, and 1.0 cm in diameter.

PG is ordinarily diagnosed on routine histopathology, and immunohistochemistry is properly regarded as an ancillary technique rather than a routine requirement. The present case illustrates the circumstances under which that generalization does not hold. The lesion was not merely difficult to interpret; it was formally reported as an atypical vascular tumor with involved margins, with a differential encompassing hemangioendothelioma, angiosarcoma, and Kaposi sarcoma, and the reporting pathologist recommended sequential immunohistochemistry for resolution. The morphological features responsible were prominent endothelial tufting and intraluminal projections in an ulcerated, rapidly enlarging lesion in a 69-year-old patient, a combination lying well outside the usual presentation of PG. Had the initial interpretation stood, management would have proceeded toward wide re-excision with consideration of adjuvant therapy for a lesion that was in fact benign and reactive. We would not advocate this work-up for morphologically typical lesions; where the differential includes a high-grade vascular sarcoma, however, immunohistochemistry is not confirmatory but determinative.

To place this case in context, we searched PubMed, Scopus, and Google Scholar for English-language reports of buccal mucosal PG, without date restriction, up to 31 December 2025. The PubMed strategy combined (“pyogenic granuloma” OR “lobular capillary hemangioma” OR “oral pyogenic granuloma”) with “buccal” in title or abstract, and equivalent strategies were adapted for the other databases. Case reports and case series were eligible if they described a histopathologically confirmed PG with explicitly documented buccal mucosal involvement and reported individual patient data, including age, sex, treatment, and follow-up; institutional retrospective series were excluded where these data were not reported separately for the buccal cases, as were review articles. Reference lists of included reports were screened for additional cases. Nine studies describing 11 cases were identified [12–20]; with the present patient, 12 cases have been reported (Table 1). Despite the absence of a lower date limit, no qualifying report was identified before 2010.

Two observations emerge. First, in every reported case treatment consisted of surgical excision. Recurrence status was documented in 6 of the 11 previously reported cases, none of which recurred over follow-up periods ranging from 1 month to 5 years; in the remaining 5 cases recurrence was not reported, and no inference can be drawn from them. Reported recurrence rates for oral PG range from approximately 3% to 23%, with a rate of 8.2% in the largest published series [4]; because gingival lesions dominate these series, the figures principally describe gingival disease. Second, the demographic distribution differs: although most adult cases occurred in women, the reported pediatric

cases were predominantly male, with several arising after bone marrow transplantation under intensive immunosuppression [18].

Table 1. Reported cases of buccal mucosal pyogenic granuloma identified in the English-language literature to December 2025.

Study	No. of Cases	Surgical Method	Follow-Up	Recurrence
Sangamesh et al. [12]	1	Excisional biopsy	6 mo	No
Selvamuthukumar et al. [13]	1	Conservative excision	NS	NS
Sulabha et al. [14]	1	Excisional biopsy	1 y	No
Sachdeva [15]	1	Surgical excision	3 y	No
Pandey et al. [16]	1	Complete excision	1 mo	No
Marla et al. [17]	2	Excision	NS	NS
Cheney-Peters and Lund [18]	2	Surgical excision	1 y and 5 y	No
Hamit et al. [19]	1	Excision with margins	12 mo	No
Sharma et al. [20]	1	Excisional biopsy	1 wk	NS
Present case	1	Excisional biopsy	9 mo	No

mo, months; NS, not stated; y, years; wk, week.

We propose that the site-dependent difference in recurrence is primarily anatomic rather than biologic, and that it is governed by whether the inciting irritant can be eliminated along with the lesion. Gingival PG develops in a site continuously exposed to plaque, calculus, and prosthetic or restorative trauma. In one comparative series, gingival lesions were attributed to recently extracted teeth, calculus, dental appliances, overhanging restoration margins, and trauma, whereas extragingival lesions were attributed almost exclusively to biting injury [7]. These gingival irritants are chronic and commonly persist after excision unless specifically addressed; the gingiva also affords restricted surgical access, and excision is frequently limited by the need to preserve attached mucosa and interdental architecture, favoring incomplete removal. The buccal mucosa, by contrast, is readily accessible, permits excision with an adequate margin under local anesthesia, and is subject to an isolated traumatic event rather than sustained mechanical irritation. On this view, the low recurrence rate at the buccal site reflects removal of both the lesion and its inciting stimulus in a single procedure, rather than any intrinsic difference in the lesional tissue.

Two observations from the literature are consistent with this interpretation, although neither constitutes direct evidence for it. First, the presence of an identifiable etiological factor differs markedly by histological subtype: etiological factors were recorded in 84.6% of non-lobular PG but in only 20% of lobular capillary hemangioma-type lesions, and the authors of that study concluded that in the former the causative factors must be identified and removed to prevent recurrence or continued growth [7]. Recurrence therefore appears to track with the persistence of a stimulus rather than with the lesional tissue itself. Second, and as a clinical rather than an empirical observation, excision to the underlying periosteum with removal of predisposing factors has been recommended to prevent recurrence of gingival lesions [4], an approach that implicitly attributes recurrence to retained local irritants. The present lesion was of lobular type and followed a single biting injury without any persistent irritant, a combination that on this model would predict cure by excision alone, as was observed. Notably, the resection margins were involved, yet no recurrence occurred over 9 months. This is difficult to reconcile with completeness of excision as the principal determinant of recurrence and is consistent with the interpretation advanced here; it should not, however, be taken to imply that margin status is unimportant in the management of oral PG.

This proposal should be interpreted with appropriate caution, and the comparison must be regarded as exploratory. With no recurrences among 6 evaluable published cases, the exact 95% confidence interval for the recurrence rate at this site is 0–46% (0–41% including the present case), an interval that encompasses the entire published range for oral PG (3–23%). The available data are therefore compatible with a lower recurrence rate at the buccal mucosa but do not demonstrate one. The published cases are few, follow-up durations are variable and, in several instances, unstated, and negative findings are subject to reporting bias. Published site distributions also vary considerably between series [4,7], and most reported recurrence rates are not stratified by site, so the gingival comparator is inferred from the predominance of gingival lesions rather than measured directly. Prospective documentation of extragingival PG with standardized follow-up, and reporting of recurrence by site and histological subtype, would be required to establish the difference with confidence.

4. Conclusions

Buccal mucosal PG is an uncommon presentation of an already infrequent, predominantly gingival lesion, and in elderly patients it may closely mimic malignant vascular tumors. The present case was initially reported as an atypical vascular tumor requiring exclusion of hemangioendothelioma, angiosarcoma, and Kaposi sarcoma, and the diagnosis was resolved only by immunohistochemistry. Recognition of lobular architecture, absent cytologic atypia, preserved peripheral nerves, and low proliferative activity, supported by a focused immunohistochemical panel, permits accurate diagnosis. Excision was curative in this patient despite involved margins, and no recurrences have been documented among the small number of previously reported buccal mucosal cases. We propose, as a hypothesis for future testing rather than an established finding, that any site-dependent difference in recurrence reflects whether the inciting irritant is removed together with the lesion; the present data are too few to establish such a difference, and prospective series reporting recurrence by site are required.

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Institutional Review Board Statement: This study was conducted in accordance with the Declaration of Helsinki. The Institutional Review Board of Gangneung-Wonju National University Dental Hospital (now Kangwon National University Dental Hospital) reviewed the study and determined that formal ethical approval was not required given the retrospective nature of the report (protocol code GWNUDH-IRB2025-A011; date of determination: 11 December 2025).

Informed Consent Statement: The Institutional Review Board granted exemption from formal approval and waived the requirement for patient consent, given the retrospective chart-review design (protocol code GWNUDH-IRB2025-A011). Notwithstanding this waiver, written informed consent for publication of the case details and clinical images was subsequently obtained from the patient. All patient identifiers were removed, and the clinical images contain no identifiable features.

Data Availability Statement: All data supporting the findings of this case report are included within the article. Additional material is unavailable for sharing because it contains identifiable patient information.

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References

1. Lomeli Martinez, S.M.; Carrillo Contreras, N.G.; Gómez Sandoval, J.R.; Zepeda Nuño, J.S.; Gomez Mireles, J.C.; Varela Hernández, J.J.; Mercado-González, A.E.; Bayardo González, R.A.; Gutiérrez-Maldonado, A.F. Oral pyogenic granuloma: A narrative review. *Int. J. Mol. Sci.* **2023**, *24*, 16885. [\[CrossRef\]](#) [\[PubMed\]](#)
2. Jafarzadeh, H.; Sanatkhan, M.; Mohtasham, N. Oral pyogenic granuloma: A review. *J. Oral Sci.* **2006**, *48*, 167–175. [\[CrossRef\]](#) [\[PubMed\]](#)
3. Kamal, R.; Dahiya, P.; Puri, A. Oral pyogenic granuloma: Various concepts of etiopathogenesis. *J. Oral Maxillofac. Pathol.* **2012**, *16*, 79–82. [\[CrossRef\]](#) [\[PubMed\]](#)
4. Gordón-Núñez, M.A.; Carvalho, M.V.; Benevenuto, T.G.; Lopes, M.F.F.; Silva, L.M.M.; Galvão, H.C. Oral pyogenic granuloma: A retrospective analysis of 293 cases in a Brazilian population. *J. Oral Maxillofac. Surg.* **2010**, *68*, 2185–2188. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Flucke, U.; Karanian, M.; ten Broek, R.W.; Thway, K. Soft tissue special issue: Perivascular and vascular tumors of the head and neck. *Head Neck Pathol.* **2020**, *14*, 21–32. [\[CrossRef\]](#) [\[PubMed\]](#)
6. Leung, A.K.C.; Barankin, B.; Hon, K.L. Pyogenic granuloma. *Clin. Mother Child Health* **2014**, *11*, e106. [\[CrossRef\]](#)
7. Epivatianos, A.; Antoniadis, D.; Zaraboukas, T.; Zairi, E.; Pouloupoulos, A.; Kiziridou, A.; Iordanidis, S. Pyogenic granuloma of the oral cavity: Comparative study of its clinicopathological and immunohistochemical features. *Pathol. Int.* **2005**, *55*, 391–397. [\[CrossRef\]](#) [\[PubMed\]](#)
8. Cantisani, C.; Di Guardo, A.; Ardigò, M.; Suppa, M.; Gonzalez, S.; Longo, C.; Taliano, A.; Rovaldi, E.; Cinotti, E.; Pellacani, G. Non-invasive diagnostic imaging in Kaposi sarcoma evaluation. *Diagnostics* **2025**, *15*, 1665. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Patel, R.M.; Goldblum, J.R.; Hsi, E.D. Immunohistochemical detection of human herpesvirus 8 latent nuclear antigen-1 is useful in the diagnosis of Kaposi sarcoma. *Mod. Pathol.* **2004**, *17*, 456–460. [\[CrossRef\]](#) [\[PubMed\]](#)
10. Antonescu, C. Malignant vascular tumors—An update. *Mod. Pathol.* **2014**, *27*, S30–S38. [\[CrossRef\]](#) [\[PubMed\]](#)
11. Cao, J.; Wang, J.; He, C.; Fang, M. Angiosarcoma: A review of diagnosis and current treatment. *Am. J. Cancer Res.* **2019**, *9*, 2303–2313. [\[PubMed\]](#)
12. Sangamesh, N.C.; Poornima, B.; Vidya, K.C.; Sakri, S.B. Extralingival pyogenic granuloma: A rare case report. *J. Sci. Soc.* **2013**, *40*, 49–51. [\[CrossRef\]](#)
13. Selvamuthukumar, S.C.; Ashwath, N.; Anand, V. Unusual presentation of pyogenic granuloma of buccal mucosa. *J. Indian Acad. Oral Med. Radiol.* **2010**, *22*, S45–S47. [\[CrossRef\]](#)
14. Sulabha, A.N.; Choudhari, S.; Suchitra, G. Pyogenic granuloma—Report of three cases in rare sites. *Bangladesh J. Med. Sci.* **2013**, *12*, 445–448. [\[CrossRef\]](#)
15. Sachdeva, S.K. Extralingival pyogenic granuloma: An unusual clinical presentation. *J. Dent.* **2015**, *16*, 282–285.
16. Pandey, R.; Gupta, R.; Rawat, S. Pyogenic granuloma of buccal mucosa mimicking traumatic fibroma in pregnancy. *Indian J. Dent. Adv.* **2016**, *8*, 172–175. [\[CrossRef\]](#)
17. Marla, V.; Shrestha, A.; Goel, K.; Shrestha, S. The histopathological spectrum of pyogenic granuloma: A case series. *Case Rep. Dent.* **2016**, *2016*, 1323798. [\[CrossRef\]](#) [\[PubMed\]](#)
18. Cheney-Peters, D.; Lund, T.C. Oral pyogenic granuloma after bone marrow transplant in the pediatric/adolescent population: Report of 5 cases. *J. Pediatr. Hematol. Oncol.* **2016**, *38*, 570–573. [\[CrossRef\]](#) [\[PubMed\]](#)
19. Hamit, B.; Bilici, S.; Yiğit, Ö.; Bozkurt, E.R. Pyogenic granuloma: Retrospective analysis of eleven cases with literature data. *Istanbul Med. J.* **2019**, *20*, 446–449. [\[CrossRef\]](#)
20. Sharma, S.R.; Mehra, A.; Karjodkar, F.R.; Sansare, K.P. Atypical extralingival occurrence of lobular capillary hemangioma: A report of two cases. *J. Indian Soc. Periodontol.* **2021**, *25*, 254–257. [\[CrossRef\]](#) [\[PubMed\]](#)

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