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Case Report

Lobular capillary hemangioma subterfuge of pyogenic granuloma: A case report of a recurrent stubborn lesion with 2-year outcomes

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Abstract

Lobular capillary hemangioma (LCH), a histological form of pyogenic granuloma (PG), is a frequent benign skin and mucous membrane vascular connective tissue anomaly. PG is neither pyogenic nor granuloma histologically, despite its misleading moniker. It is usually reactive proliferation in reaction to local irritation, and it bleeds less than PG, which is hormonal immune-inflammatory. It may create discomfort, esthetic concerns, or functional challenges depending on its size and position.

A 39-year-old woman presented with a recurring lobular capillary hemangioma from the connective tissue in the intra-furcal granulation tissue and associated attached gingiva of maxillary posterior region. Despite conventional therapy, the lesion recurred three times over a period of two years, underscoring the diagnostic challenges, therapeutic difficulties, and the need for long-term follow-up.

Keywords: Lobular capillary hemangioma, Pyogenic granuloma, Pregnancy tumour, Benign vascular lesion, Arterio-venous proliferation.

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1. Introduction

Lobular capillary hemangioma (LCH) is characterized by a proliferation of capillaries arranged in a lobular pattern.[1] Term Pyogenic Granuloma (PG) and LCH has been used interchangibly. In the 2017 WHO classification of head and neck tumours, LCH and PG were categorized as synonyms and classified under hemangiomas. Conversely, the 2018 International Society for the Study of Vascular Anomalies (ISSVA) classifies both as granuloma.[2] Clinically, LCH commonly presents as a rapidly growing, friable, red-to-purple mass on the gingiva. It is not a true neoplasm but rather a reactive proliferation of capillaries and granulation tissue, usually triggered by local irritation, trauma, or hormonal changes, such as those occurring during pregnancy.[3]

In the context of chronic periodontitis (CP), LCH frequently arises due to plaque and calculus-induced inflammation, complicating gingival health[4] while in aggressive periodontitis (AgP), its rapid growth may mimic malignancy, necessitating careful diagnosis.[5]

Pyogenic granuloma (PG) was first described by Poncet and Dor in 1897 as “botryomycosis hominis,” mistakenly assuming an infectious origin.[6] Later, in 1904, Hartzell introduced the term “pyogenic granuloma”, which, though still widely used.[7]

2. Case Report

A 39-year-old woman complained of gingival overgrowth (at attached gingiva and margins) in the maxillary left posterior region, specifically tooth #27. (**Figure 1**) The patient stated that the lesion started as a small, pimple-sized, flat swelling and grew to the size and shape of an almond over two months.

Clinical examination revealed a well-circumscribed, single, lobulated mass from the connected and marginal gingiva next to tooth #27. The lesion, measuring 16 × 10 mm, was oblong and orientated corono-apically. It had a

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corrugated surface with erythematous and pale white patches. (**Figure 1**) Firm, non-tender, and blanching on pressure, the lesion did not bleed.

In 1980, pathologists Stacey E. Mills, Philip H. Cooper, and Robert E. Fechner proposed the term "lobular capillary hemangioma (LCH)" after studying 73 cases of PG, all of which demonstrated a distinct lobular arrangement of proliferating capillaries embedded in a fibrovascular stroma.[8]

2.1. Epidemiology

LCH has a 26.8%–32% prevalence among reactive oral lesions, showing a 2:1 female predilection, often attributed to hormonal factors.[9] It most commonly affects the gingiva, particularly the interdental papilla, accounting for over 70% of oral cases.[4] In contrast, capillary hemangiomas, though histologically similar, are true vascular neoplasms, constituting about 7% of benign tumors in children, with a 3:1 female predominance and predilection for head and neck sites (e.g., lips, tongue, cheeks).[10]

Histologically, the overlap between LCH and other vascular proliferations such as capillary hemangioma, epithelioid hemangioma, and bacillary angiomatosis can complicate diagnosis. Therefore, accurate identification requires a combination of clinical context and histopathologic correlation.[11]

Generalized hard and soft deposits were present especially around the upper left posterior and lower anterior teeth, suggested inadequate dental hygiene. Probing #26 and #27 revealed a 4 mm pocket. Local irritants were removed using full-mouth scaling as well as root planning. The patient was educated and motivated to maintain proper oral hygiene.

Routine hematological investigations were conducted and found to be within normal limits. After seven days of non-surgical periodontal therapy (NSPT) but size of lesion not reduced, the gingival overgrowth was surgically excised under local anesthesia (LA) in toto using a scalpel. (**Figure 2**) After obtaining a written informed consent and a Coe pack was given. (**Figure 3**)

Histopathology was performed on excised tissue promptly. In (**Figure 4**) Low-power 10X Lobules of tiny capillaries. Parakeratinized epithelium with fibrinopurulent membrane, loose and edematous stroma, proliferating endothelial cells, fibrous septa, inflammatory infiltrate. Lobular capillary hemangioma was confirmed by delicate collagen fibers and acute and chronic inflammatory cells, mostly neutrophils and lymphocytes. (**Figure 5**)

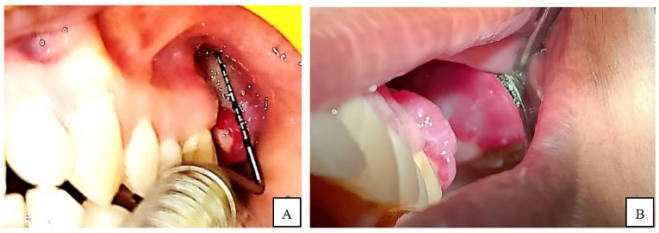

Figure 1: **A)**: solitary, pedunculated mass around 16×10 mm observed on attached gingiva of maxillary posterior region i.r.t. tooth # 27. **B):** Shows an erythematous pale white area

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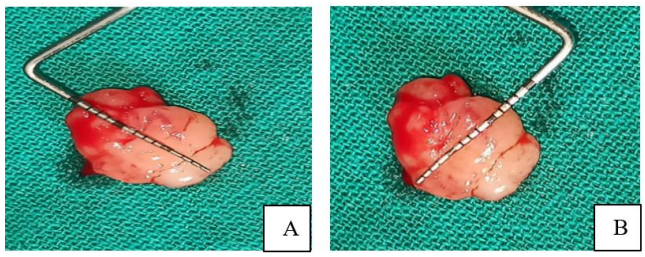

Figure 2: Solitary mass around 15×12 mm excised with scalpel and blade first time from gingival margin of maxillary posterior teeth w.r.t 27 and low level laser therapy (LLLT) given

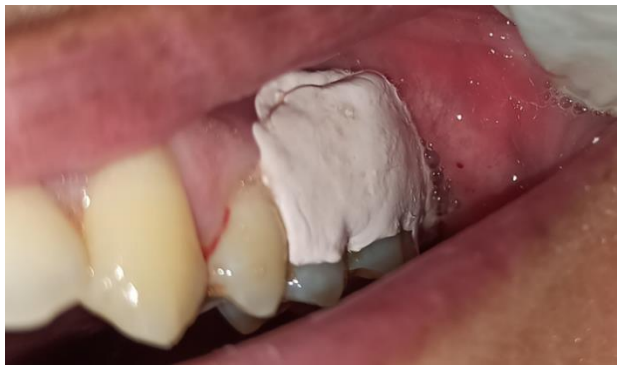

Figure 3: Coe-pack placed post-surgical excision

Histopathology was performed on excised tissue promptly. In (**Figure 4**) Low-power 10X Lobules of tiny capillaries. Parakeratinized epithelium with fibrinopurulent membrane, loose and edematous Stroma, proliferating endothelial cells, fibrous septa, inflammatory infiltrate.

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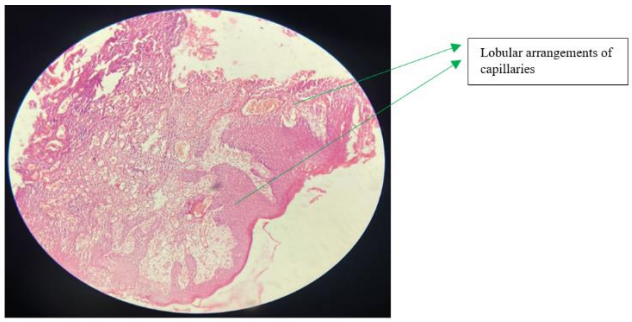

Figure 4: Low-power 10X view showing ulcerated surface with connective tissue comprising numerous proliferating blood vessels with chronic inflammatory cells (H and E, ×10) numerous small capillaries arranged in lobules. Proliferating endothelial cells, fibrous septa, inflammatory infiltrate, parakeratinized epithelium with squamous epithelium ulceration and fibrinopurulent membrane. Loose and edematous stroma, inflammatory infiltrate, granulation tissue, stroma, neutrophis, macrophage, fibrous septa, collagen bundle, fibroblast

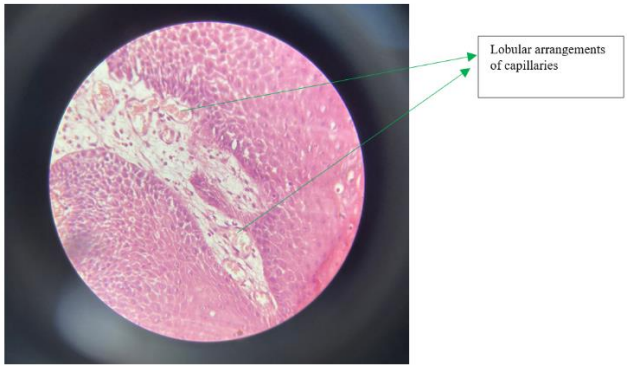

Figure 5: High-power 40 X view showing blood vessels lined by endothelial cells, filled with RBCs, proliferating into the connective tissue (H and E, ×40)

2.2. Recalcitrant lesion treatment

Following four months post-excision of the growth, the patient reported with a recurrence of 4 × 3 mm growth. Patient was educated and motivated to maintain hygiene. An interproximal brush advised and recalled after one weak. Patient hygiene improved, but growth size increased. It was spherical, edematous, and moist with shining mucosa, measuring 6x5 mm. (**Figure 6**)

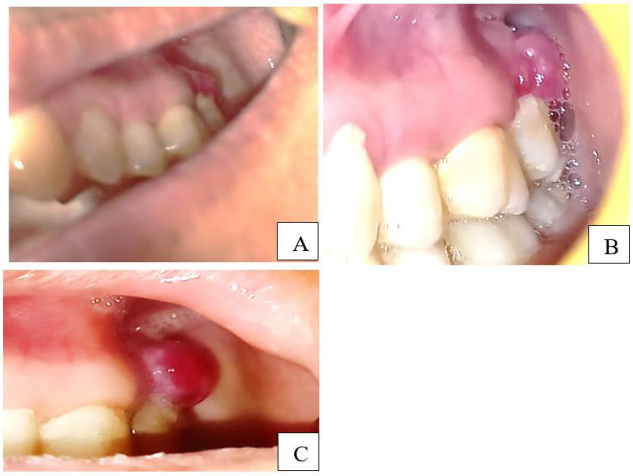

Figure 6: Showing progressive enlargement of 2nd time recurrent growth w.r.t# 27

The patient noted that her growth size has increased after one week despite proper dental and oral hygiene. OPG and IOPA were ordered (**Figure 7**), and patient diagnosed with generalized stage II grade A periodontitis. [Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification. J Clin Periodontol. 2018;45 (Suppl 20):S1–S8.] Under local anesthesia (2% lignocaine with 1:80,000 adrenaline), access flap surgery (Kirland flap) was performed in the 2nd quadrant to completely remove growth with healthy 1 mm marginal tissue and extensively debride roots. (**Figure 8**)

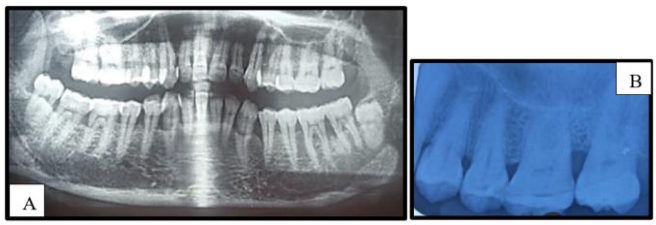

Figure 7: A): (OPG / Orthopantomogram) The red arrow points to an area of generalized horizontal bone loss around the molar region. In generalized horizontal bone loss, the alveolar crest is uniformly reduced in height, running parallel to the cemento-enamel junction (CEJ) but at a greater distance than normal (normally 1–2 mm). This pattern of bone loss is characteristic of generalized periodontitis rather than localized bone defects. Stage II → bone loss extending into the coronal third of the root (15–33%). Grade-A → slow rate of progression, no additional risk factors (like smoking or diabetes). **B):** (IOPA / Intraoral Periapical Radiograph) The red arrow shows horizontal bone loss between adjacent teeth. Alveolar crest appears flattened and lowered in relation to the CEJ. This confirms the localized manifestation of the generalized bone loss seen in the OPG.

The excised growth was (10 × 8 mm) sent for histopathological examination [Figure 9]. Upon histopathology exanination in reports hyperplastic epithelium with heavy connective tissue inflammatory cell infiltration and perivascular inflammation (10 X H & E) seen. (40 X H & E) Lobular capillaries, endothelial cell proliferation, vascular dilation, fibrous septa, inflammatory cell infiltrate. (**Figure 10** A,B)

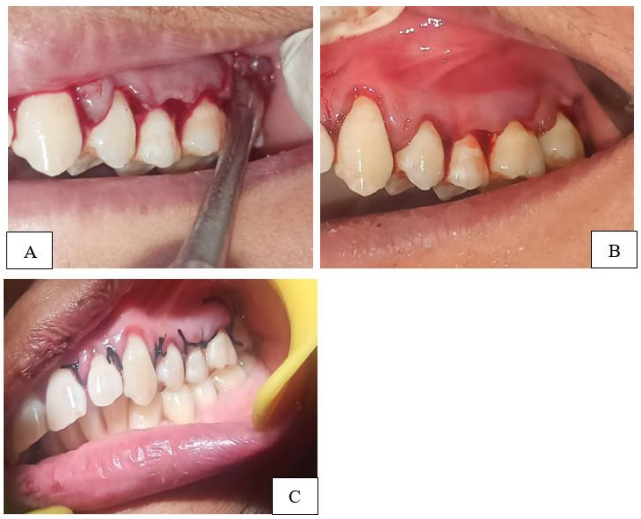

Figure 8: A): Access Flap Elevation) A periodontal elevator used to reflect gingival flap. The purpose of this access flap is to gain access to the underlying alveolar bone and root surfaces. The gingival overgrowth excised in to (completely) to ensure complete removal; **B):** (Post-Excision View) The surgical site after excision shows healthy marginal gingiva with removal of the pathological growth. The aim here is to restore the gingival contour and complete debridement of local irritating factors on root and tooth surfaces while maintaining adequate access for debridement; **C):** (Post-Excision View) The surgical site after excision shows healthy marginal gingiva with removal of the pathological growth. The aim here is to restore the gingival contour and complete debridement of local irritating factors on root and tooth surfaces while maintaining adequate access for debridement

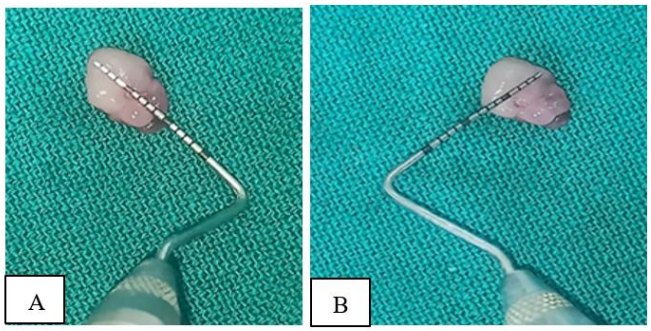

Figure 9: Solitary mass around 10×8 mm excised in to 2nd time from attached gingiva of maxillary posterior teeth w.r.t 27 tooth after flap surgery

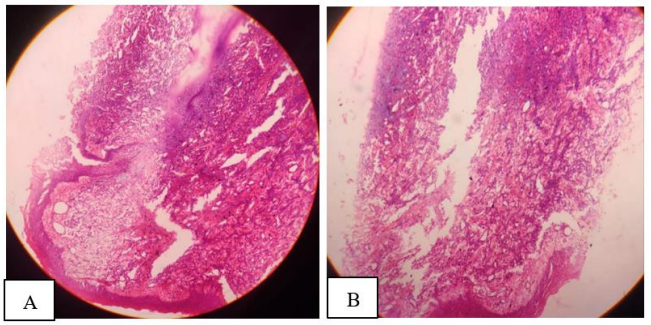

Figure 10: A): (10 X H & E) On histopathological examination hyperplastic epithelium,dense inflammatory cell infiltrate in the connective tissue and perivascular inflammation. **B):** (40 X H & E), lobular arrangement of capillaries, proliferating endothelial cells, dilated vascular spaces, fibrous septa, inflammatory cell infiltrate.

The patient reappeared to Periodontics after 10 months with an recalcitrant growth of 6 x 5 mm w.r.t #27. It was dome-shaped, oblong coronally-apical, pale white with erythematous regions, with corrugated surface, firm to the touch, non-tender, and with no bleeding. (**Figure 11**)

Figure 11: Pre-extraction image of recurring growth attached to gingival margin and attached mucosa w.r.t. #27 teeth seen on 3rd occasion

After 11 months patient reported to the department of periodontics with a 2nd recurring growth measuring approximately 6 x 5 mm. The growth was dome shaped in external form, oblong in shape in coronally-apical direction, pale white with erythematous areas, with lobulations and corrugated surface, firm to the touch, non-tender, and with no bleeding.

Because the recurrence rate was so high, Patient decided to extract the corresponding tooth. The tooth was extracted under local anaesthesia (**Figure 12**) and sent for histolpathological analysis along with the growth. Histopathological study Low-power10 X revealed thin parakeratinized stratified squamous epithelium and connective tissue with haphazardly oriented thick collagen fibers and many endothelial cell-lined blood vessels. Additionally, chronic inflammatory cells were found. These observations suggested lobular capillary Hemengioma. (**Figure 13**)

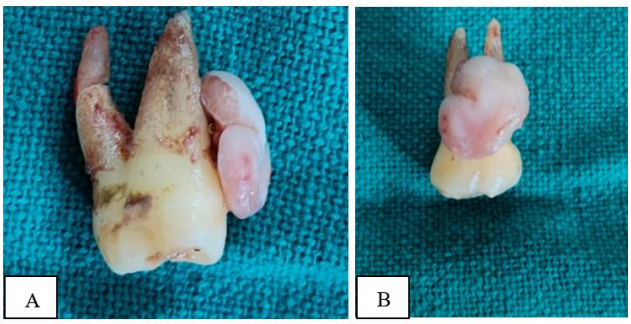

Figure 12: A): Extracted tooth along with dome shape growth with its peduncle attached to intra-furcal connective tissue proximal view. **B):** Facial view

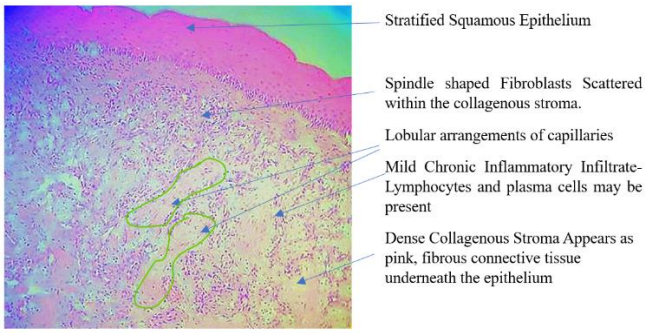

Figure 13: On histopathological examination (Low-power 10 X), a thin parakeratinized stratified squamous epithelium and underlying connective tissue with haphazardly arranged dense collagen fibers with numerous proliferating blood vessels lined by endothelial cell were seen. Chronic inflammatory cells were also noted. These findings were suggestive of lobular capillary Hemengioma

3. Pathogenesis

In contrast, LCH often lacks inflammatory infiltration or obvious inciting events, indicating that its development is predominantly spontaneous. Nevertheless, the pathogenic pathways of PG an LCH are likely to intersect. At the molecular level, LCH may arise from local irritation (e.g., plaque in CP, bacteria in AgP, trauma).[1] Activation of TLR2/TLR4 by irritants induces the production of cytokines (IL-1β, TNF-α), PGE2, and VEGF, which promote lobular capillary proliferation, edema, and granulation tissue formation. Recruitment of neutrophils, lymphocytes, and macrophages via the bloodstream sustains inflammation and leads to matrix metalloproteinase (MMP)-mediated tissue damage. Genetic factors, such as IL-1B polymorphisms, may further enhance susceptibility to LCH in patients with chronic periodontitis.[12]

Although the name suggests an infectious aetiology, the cause of both LCH & pyogenic granuloma (PG) is unknown. Most theories on pathogenesis consider LCH & PG as a hyperplastic, neovascular response to an angiogenic stimulus driven by imbalance between angiogenic promoters and inhibitors.[1]

Theories of pathogenesis of lobular capillary hemangioma-

1. Infectious (Historical) Theory: This theory suggested a mycotic or bacterial origin. Bacteria, such as those associated with Chronic Periodontitis (CP), were thought to indirectly trigger inflammation.[7]
2. Reactive/Hyperplastic Theory: This theory proposes that gingival lesions result from an exaggerated response to irritation (e.g., plaque, trauma). It remains the most widely accepted explanation for gingival lesions in CP.[4]
3. Hormonal Influence Theory: Estrogen and progesterone increase vascularity, which explains the occurrence of the "pregnancy tumor" in CP.[9]
4. Angiogenic Dysregulation Theory: Vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), and hypoxia-inducible factor 1-alpha (HIF-1α) drive lobular capillary proliferation, contributing to rapid growth in CP and aggressive periodontitis (AgP).[3]
5. Immunological Theory: Toll-like receptor (TLR)-driven cytokines, such as interleukin-1 beta (IL-1β) and prostaglandin E2 (PGE2), along with immune cells via blood circulation, sustain inflammation. This mechanism is relevant in both CP and AgP.[12]
6. Genetic Susceptibility Theory: Polymorphisms in cytokine genes and TLRs may enhance inflammatory responses and predispose individuals to increased risk of CP.[12]

The varied clinical and histological presentations of both diseases suggest different pathogenic processes. PGs exhibit a distinct predilection for pregnant women and are characterized by a significant inflammatory infiltration, features that contribute to their development.

Lobular Capillary Hemangioma (LCH) arises from local irritation (e.g., plaque in CP, bacteria in AgP, trauma).[1] Blood delivers neutrophils, lymphocytes, and macrophages, sustaining inflammation and MMP-mediated tissue damage. At molecular level TLR2/TLR4 activation by irritants induces cytokines (IL-1β, TNF-α), PGE2, and VEGF, promoting lobular capillary proliferation, edema, and granulation tissue formation. Genetic factors (e.g., IL-1B polymorphisms) may enhance susceptibility to LCH in chronic periodontitis population.[12]

Gene expression studies of LCH and PGs have revealed embryonic stem cell markers in endothelial cells, with interstitial cells exhibiting a more differentiated phenotype. This suggests the possibility of de novo vasculogenesis originating from primitive stem cells.

Angiogenic growth factors, such as VEGF and decorin, also play key roles. Decorin is a small proteoglycan found in connective tissue, normally involved in collagen organization and regulation of cell growth. When overexpressed, it may interact with growth factors such as Transforming Growth Factor-beta (TGF-β) and VEGF, thereby enhancing

neovascularization and modulating inflammation and angiogenesis.

Furthermore, transcription factors such as phosphorylated Activating Transcription Factor 2 (pATF2), activated by inflammation and cellular stress, and phosphorylated Signal Transducer and Activator of Transcription 3 (pSTAT3), activated by IL-6 and VEGF, promote angiogenesis, cell proliferation, and immune responses.[13]

Proteins of the Mitogen-Activated Protein Kinase (MAPK) signaling pathway are also overexpressed in PGs, although their exact role remains unclear. In a whole-exome sequencing study of 40 PG lesions, HRAS somatic mutations were detected in four cases, implicating the RAS–MAPK pathway in PG pathogenesis.[14] Activation of the MAPK/ERK pathway has also been directly demonstrated in oral LCH lesions.[13]

4. Discussion

Based on the histological appearance, two distinct forms of Pyogenic Granuloma (PG) have been recognized: LCH type and the non-LCH type. Evidence of clusters of multiplying blood vessels organized in a lobular architecture is used to identify the LCH type. Epivatianos et al. distinguished between the two forms: the non-LCH PG manifests as a pedunculated soft-tissue growth, whereas the LCH PG more commonly presents as a sessile lesion.[2]

The histopathology of LCH differs from that of congenital hemangiomas.In LCH, capillary lobules are separated by thick bands of fibrous tissue, whereas congenital hemangiomas reveal lobular clusters of thin-walled vessels with a central stellate-shaped vessel.[11] Contributing factors to such connective tissue proliferations include local trauma, hormonal influences, and poor oral hygiene, which irritate and inflame the gingiva, triggering lesion development.[15]

Clinically, LCH seen as a red to purple nodular growth, with colour variations depending on duration and vascularity. The lesion surface may exhibit erythema or ulceration, often caused by impingement from adjacent teeth during mastication or speech.

While clinical diagnosis may suffice in typical classic cases, atypical presentations necessitate biopsy to exclude malignancies or vascular neoplasms.[5]

Conservative management involves surgical excision followed by oral prophylaxis. Addressing underlying causes such as trauma or irritation is essential. Recurrence may occur due to incomplete excision, persistence of local irritating factors, or reinjury. For gingival lesions, excision down to the periosteum and elimination of etiologic factors are necessary to minimize recurrence.[16]

Histologically, non-proliferative gingival enlargements must be differentiated from proliferative hemangiomas, as they can mimic less common lesions such as capillary hemangioma, epithelioid hemangioma, or epithelial cell histiocytoma.[5]

In the present case report, a two-year follow-up revealed complete wound healing post-tooth extraction, with no recurrence observed.

4.1. Recurrence: A clinical challenge

Recurrent LCH poses a major clinical dilemma. The recurrence rate after simple excision can range from 5% to 16%, and is often higher if predisposing factors persist.[1] In the present case, the lesion recurred twice following initial excision and nonsurgical periodontal therapy. Definitive resolution was achieved only after flap surgery, complete excision with healthy tissue margins, or by tooth extraction, emphasizing the importance of thorough debridement and removal of all local irritants—even at the cost of tooth sacrifice.[16]

Alternative approaches such as laser excision, cryotherapy, and sclerotherapy have been suggested, particularly in esthetically sensitive areas.[17,18] However, surgical excision remains the gold standard, especially for recurrent lesions.

Recent evidence reinforces these findings. A 2024 practical review reported that surgical excision achieved nearly complete resolution (success rate ~96%), outperforming modalities such as lasers and injectables that often require multiple sessions and have variable outcomes. Recurrence and postoperative complications averaged around 15%, underscoring the importance of robust surgical management.[19]

Advancements in adjunctive treatments have also been explored. A 2023 randomized controlled trial comparing diode laser to sclerotherapy in oral PG found both to be effective; however, the diode laser approach offered better intraoperative control with minimal bleeding.[20] Similarly, a 2022 comparative study demonstrated that both diode laser (980 nm) and sclerotherapy are viable options with satisfactory outcomes and safety profiles.[21]

5. Conclusion

Lobular capillary hemangioma, although benign, may behave aggressively when persistent local factors are present. This case underscores the importance of a comprehensive clinical, surgical, and histological approach to ensure effective treatment and minimize recurrence. This case reinforces the importance of combining surgical excision with periodontal therapy to minimize recurrence. Persistent or recurrent lesions must be re-biopsied to exclude malignancies or vascular tumors such as capillary hemangiomas or epithelioid

hemangiomas. Patient education, plaque control, and frequent follow-up are critical for long-term success.

6. Source of Funding

Nil.

7. Conflicts of Interest

There are no conflicts of interest.

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