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Disrupting the Pattern: Treatment of Recurring Pyogenic Granuloma Using PRF – A Case Study

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Abstract: Pyogenic granuloma (PG) is an inflammatory proliferative lesion in the oral cavity usually caused by chronic low-grade irritation, trauma or hormonal influences. Clinically it presents as a smooth or lobulated exophytic growth, which may have a pedunculated or sessile base. In the present article, different treatment strategies for PG are discussed with special emphasis on the application of a platelet-rich fibrin (PRF) membrane to minimise recurrence. A female patient, 22 years old, with three recurrences of lesion in 2 years. Clinically, the lesion was sessile and PG was confirmed clinically and histopathologically. No recurrence was found at the 12-month follow-up.

Keywords: *Pyogenic Granuloma, Platelet-Rich Fibrin, Recurrence, Oral Lesion.*

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I. INTRODUCTION

Inflammatory hyperplasia is the name for nodular lesions of the oral mucosa that are enlarged and have a histological structure composed of fibrous and granulous tissue.[1]

Fibrous inflammatory hyperplasia includes clinical fibroma, epulis fissuratum, pulp polyp, palatal papillary hyperplasia, giant cell granuloma, pregnancy epulis and pyogenic granuloma (PG).[2]

Hullihen gave the first description of PG in 1844 describing it as a vascular lesion.[3] The term "granuloma pyogenicum" was introduced by Hartzell in 1904.[4]

PG is one of the common non-neoplastic lesions in the oral cavity. It is a reactive lesion, which is produced in response to chronic irritation or trauma which changes cell turnover and activity. Typically 2nd to 5th decades with female predilection.[5,6]

The higher prevalence in females, particularly in the second decade of life, is hypothesised to be related to the vascular effects of oestrogen and progesterone.[7] Approximately three quarters of oral PG cases present in the gingiva and are frequently associated with local irritants such as calculus or foreign bodies.[5]

In the gingival sulcus, irritation or trauma may result in proliferation of fibrovascular connective tissue into an overgrowth of granulation tissue. This occurs more frequently in certain predisposing conditions. Other conditions include hormonal changes (e.g., puberty or oral contraceptive use); medications (e.g., oral contraceptives and isotretinoin); bacterial or viral infections; trauma associated with natal or neonatal teeth; pregnancy tumours; abnormal development of natal, neonatal, or deciduous teeth or their supporting structures; and tooth irritation during the eruption sequence.[8]

The most frequent location of involvement includes lips, tongue, buccal mucosa, and hard palate. The lesions are more frequently located on the maxillary than on the

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mandibular gingiva and more frequently on the anterior than on the posterior segments of the gingiva. Lesions occur more frequently on the facial aspect than on the lingual aspect.

Clinically PG presents as a smooth lobulated lesion which may be sessile or pedunculated. It is a dumbbell shaped, compressible mass, often hemorrhagic and rarely. Lesions are described as sessile in 66% and pedunculated in 77%.[9]

The lesion is usually slow growing and asymptomatic. It can also, at times, grow rapidly before it settles down. Its size can range from few millimetres to few centimetres but usually does not exceed 2 cm in diameter.[1]

Histopathologic examination provides a diagnosis. Many treatment procedures have been described such as surgical excision most commonly used method of treatment. Complete excision of the lesion with a margin of 2mm of normal tissue is recommended. Removal of all possible irritants like plaque, calculus, foreign bodies or defective restorations is recommended.[10]

The differential diagnosis of PGCG includes peripheral giant cell granuloma, peripheral ossifying fibroma, peripheral odontogenic fibroma, haemangioma, hyperplastic gingival inflammation, conventional granulation tissue, Kaposi's sarcoma, bacillary angiomatosis, angiosarcoma, non-Hodgkin's lymphoma and metastatic tumours.[1]

PG is benign and does not undergo malignant transformation, but the recurrence rate of PG is relatively high (~15.8%) and re-excision is often required.[11]

Recurrence may be due to incomplete excision, persistence of etiological factors, re- injury, multiple satellite nodules around original lesion (Warner–Wilson Jones syndrome) are seen particularly in gingiva and hence frequent recurrence.Recurrent gingival lesions are the most common.[11]

II. CASE REPORT

A 22-year-old woman visited the Periodontics Department at Sharavathi Dental College and Hospital in Shimoga, complaining of swelling in the lower left front area of her jaw for the last two months. She mentioned that the swelling had reappeared at the same location, having been surgically removed thrice in the past two years. The previous removals were done with a scalpel, and histopathology had confirmed it as a pyogenic granuloma. The latest recurrence occurred about two months after the last excision.

Initially, the lesion was small but gradually grew larger. It was not painful but caused slight discomfort and bled when probed. An intraoral examination showed a lesion on the front lower jaw, stretching from the back of the canine to the front of the first premolar.[FIGURE 1] The lesion was about 7 mm by 5 mm, pink, with a lobulated surface and a broad base. The patient's medical and family histories were not relevant.

III. METHODOLOGY

The surgical area was numbed with 2% lignocaine containing 1:200,000 adrenaline. A full-thickness incision was made with a scalpel, extending from the distal aspect of canine to the mesial aspect of the first premolar, including a 2 mm margin of healthy gum tissue. The lesion was excised from its base, revealing the underlying bone.[FIGURE 2 & 3]

The removed tissue, measuring 7 mm by 5 mm[FIGURE 5], was preserved in 10% formalin was forwarded for histopathological analysis. At the same time, 10 ml of the patient's blood was drawn from the antecubital vein and centrifuged at 2700 rpm for 12 minutes using a REMI-4C centrifuge. The resulting clot was compressed between sterile gauze to create a platelet-rich fibrin (PRF) membrane, which was then placed over the surgical site.[12]

After thorough cleaning, the bone exposed was covered with the prepared PRF membrane according to the protocol by Choukron et al. in 2001.[FIGURE 4][13]

The membrane was secured with interrupted sutures placed mesially and distally.[FIGURE 6] A periodontal dressing was placed and postoperative care instructions were given. The patient was advised to use 0.12% chlorhexidine mouthwash.

Histopathological analysis [FIGURE 7] showed an ulcerated surface covered with fibrinous exudate. The epithelium displayed hyperplasia and edema, with inflammatory cell infiltration. Beneath the epithelium, numerous vascular channels lined with endothelium were arranged in a lobular pattern, indicating perivascular inflammation. The lesion shows a fibrous stroma with inflammatory infiltrate consisting of neutrophils, plasma cells and lymphocytes. These findings confirm the diagnosis of pyogenic granuloma.

At the two-week postoperative check-up[FIGURE 8], uneventful healing of the surgical site was noted. At the 12-month follow-up[FIGURE 9], no postitive indication of the lesion recurring was seen.


Fig 1 Pre-Operative View


Fig 2 Incisions Placed with No 15 Blade

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Fig 3 Immediate Post-Operative View

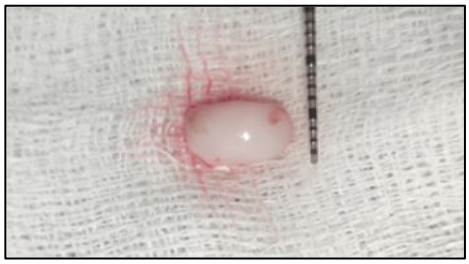
Fig 4 Biopsy Sample Measuring 7Mmx 5mm


Fig 5 Pre Placed WRT 33 and 34


Fig 6 Braided Black Sutures Placed


Fig 7 14 Day Post-Operative


Fig 8 12 Months Follow-Up

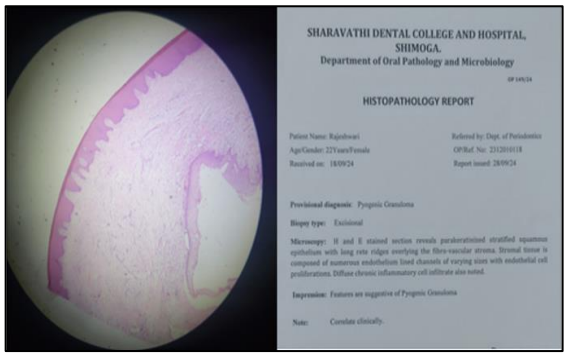

Fig 9 Histopathological Analysis

IV. DISCUSSION

Pyogenic granuloma is one of the commonly identified inflammatory hyperplastic lesion found in the oral cavity, representing around 1.85% of all oral pathologies, excluding caries and gingivitis. While it is usually without symptoms, slight trauma to the lesion can lead to considerable bleeding.[14]

This lesion often arises from persistent low-grade irritation, trauma, or hormonal fluctuations. It can pose aesthetic issues and disrupt functions like chewing, swallowing, and speaking. Its swift growth is linked to several biological mediators, such as inducible nitric oxide synthase, vascular endothelial growth factor (VEGF), basic fibroblast growth factor, and connective tissue growth factor.[7] In this case, the lesion reappeared thrice over two years at the same location, likely due to incomplete removal in earlier treatments.

Numerous treatment options are documented in the literature, with surgical excision being the most frequently used method. Other techniques include laser therapy, cryotherapy, and electrocautery, all of which have shown effectiveness. However, each method has its own pros and cons. While laser and electrosurgery offer a bloodless field and enhanced patient comfort, they might also harm surrounding tissues and slow down healing.[7]

The management of PG should be customized to the specific characteristics of each patient. Traditional surgical excision remains the preferred treatment, though less invasive methods like laser therapy, corticosteroid injections, cryosurgery, and sclerotherapy have also been proposed.[6,15]

Surgical treatment involved the complete excision of the lesion, extending to periosteum with a 2 mm margin of surrounding healthy tissue. In cases involving teeth, thorough supra- and subgingival cleaning is crucial to remove plaque and calculus. Eliminating local irritants, such as defective restorations or foreign objects, is vital to prevent recurrence.[16,17]

The rate of recurrence of PG is around 15%, often linked to incomplete excision of previous lesion or the failure to remove causative factors.[18,19] In this instance, the scalpel technique was chosen due to the lesion's sessile nature and

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history of recurrence. As reported by Asnaashari et al. (2014), recurrence rates following conventional surgical methods are approximately 16%.[11]

The procedure aimed for the complete removal of the lesion, including the underlying soft tissue, to reduce any chance of recurrence. However, this caused the exposure of the underlying alveolar bone. Wilderman (1964) noted that after full-thickness flap procedures, exposed bone undergoes superficial necrosis within 1–3 days, followed by peak osteoclastic activity at 4–6 days, resulting in minor bone loss.[20]

This underscores the importance of using PRF as a biological scaffold to cover the exposed bone and facilitate healing.[12]

V. CONCLUSION

Scalpel excision remains a reliable and effective method for addressing pyogenic granuloma, offering several clinical advantages. In cases of recurring lesions, it is essential to carefully plan the treatment to completely remove the underlying causes while preserving the surrounding soft and hard tissues. The use of an autologous platelet-rich fibrin (PRF) membrane is crucial due to its significant regenerative and healing properties, which greatly enhance treatment outcomes. The formation of PG is marked by intense inflammatory activity, resulting from an imbalance between pro-angiogenic and anti-angiogenic factors, as well as interactions between mast cells and fibroblasts. Future research focusing on genomic and molecular analyses may reveal specific mutations and pathways, potentially leading to more targeted and less invasive treatment options.

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