International Journal of Enhanced Research in Medicines & Dental Care (IJERMDC),
ISSN: 2349-1590, Vol. 12 Issue 10, October 2025, Impact Factor: 8.325

Pyogenic Granuloma in an Adolescent: A Case Report

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ABSTRACT

Pyogenic granuloma (PG), or lobular capillary hemangioma, is a benign vascular lesion of the skin and mucous membranes that commonly develops in response to irritation, trauma, or hormonal changes. It is characterized by rapid growth and a tendency to bleed easily. Although its name suggests an infectious process, PG is noninfectious and represents a reactive proliferation of capillaries.

Keywords: Pyogenic granuloma, Lobular capillary hemangioma, Angiogenesis, Oral mucosa

INTRODUCTION

Pyogenic granuloma (PG) is a common benign vascular lesion that often occurs on the skin and oral mucosa. It presents as a rapidly growing, reddish, and friable papule or nodule that bleeds easily. Despite its misleading name, PG is neither pyogenic nor granulomatous; instead, it represents an excessive reactive proliferation of capillaries in response to various stimuli (1).

The etiology of pyogenic granuloma is multifactorial. Local irritation and trauma, such as chronic friction, foreign bodies, or dental plaque, are frequent triggering factors (2). Hormonal influences, particularly during pregnancy, play a major role—elevated estrogen and progesterone levels increase vascular permeability and angiogenic activity, resulting in granuloma gravidarum (3). Drug-induced PG has been reported with retinoids, indinavir, and certain antineoplastic agents (4). Some cases develop without an identifiable cause, suggesting genetic or molecular predisposition (5).

PG represents an exaggerated tissue repair process characterized by excessive angiogenesis. Histopathologically, it shows lobular aggregates of proliferating capillaries within an edematous stroma and inflammatory infiltrate (6). Elevated levels of vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), and angiopoietins play a crucial role in promoting endothelial proliferation and vascular remodeling (7). Clinically, the lesion appears as a red to reddish-purple nodule that may ulcerate and bleed easily. Oral lesions are common on gingiva, lips, and tongue, while cutaneous lesions often occur on hands and face (8).

Management depends on lesion size, site, and symptoms. The standard treatment is surgical excision with curettage of the base, which allows histopathologic confirmation and minimizes recurrence (9). Laser excision (CO_2, pulsed-dye, Nd:YAG), electrocautery, and cryotherapy are effective alternatives, especially in cosmetically sensitive areas (10). Topical beta-blockers, particularly 0.5% timolol, are emerging as non-invasive options for small or pediatric lesions due to their anti-angiogenic effect (11). In pregnancy-associated PG, conservative management is preferred unless there is significant bleeding or discomfort (3). Recurrence mainly results from incomplete excision or persistent local irritants, emphasizing the need for their elimination and maintenance of good oral hygiene (2,8).

CASE REPORT

A 14-year-old female patient presented to the Department of Periodontics with a chief complaint of swelling in the lower right anterior region for the past six months. The lesion initially appeared as a small nodule but gradually increased in size.

Intraoral examination revealed a reddish-pink, smooth, pedunculated growth involving the interdental gingiva in the region of teeth 42 and 43(Fig. A). Malalignment of the teeth was also noted. On palpation, the lesion was soft, non-tender, and non-reducible. Bleeding was observed on gentle probing.

Phase I periodontal therapy was completed(Fig. B), and routine hematological investigations were within normal limits. Following adequate local anesthesia, the lesion was excised completely using a 15C surgical blade(Fig. C). Postoperative care included prescription of chlorhexidine mouthwash twice daily for 15 days, amoxicillin 500 mg three times daily for five days, and ibuprofen 400 mg as needed for discomfort.

The excised tissue was submitted for histopathological examination, which confirmed the diagnosis of pyogenic granuloma. At the 3-months follow-up, healing was uneventful, with no evidence of recurrence(Fig. D). Patient was advised of orthodontic treatment for malaligned teeth.

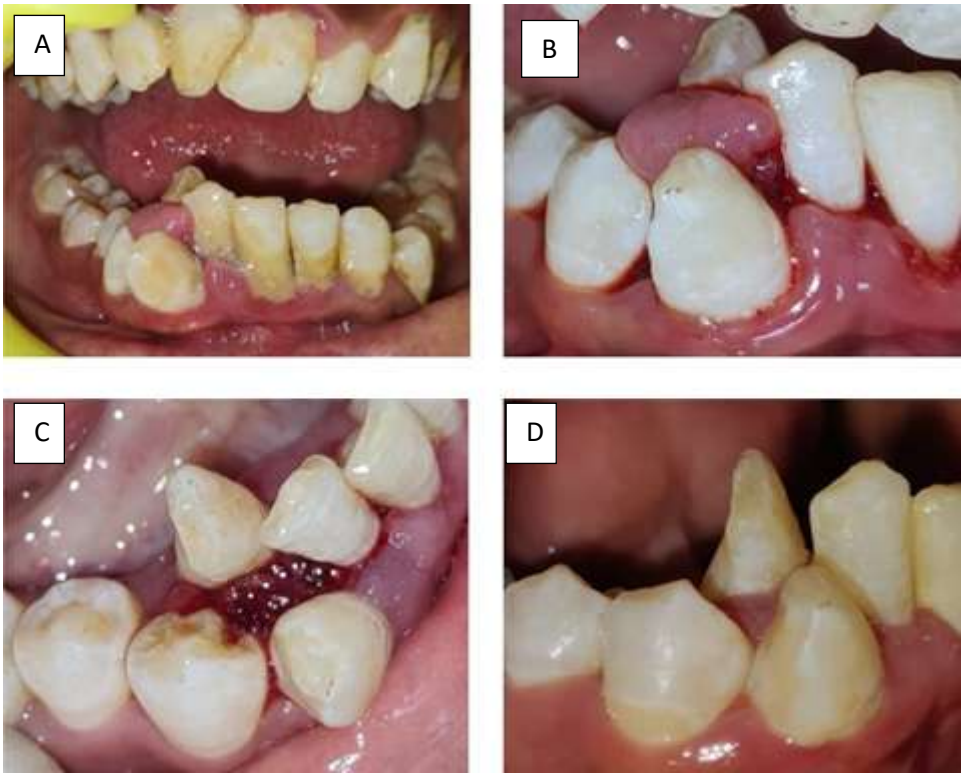

Figure: A) Pre-operative photo, B) Immediate after scaling, C) Post excision, D) 3-months follow-up

DISCUSSION

Pyogenic granuloma represents a reactive vascular proliferation rather than a true neoplasm, resulting from an exaggerated reparative process following minor trauma or irritation (12). The lesion's rapid growth, friability, and tendency to bleed often raise suspicion for more aggressive pathologies such as hemangioma, peripheral giant cell granuloma, or even Kaposi's sarcoma, making histopathologic evaluation indispensable for accurate diagnosis (13). Histopathologically, PG typically exhibits a lobular capillary architecture composed of proliferating endothelial cells embedded in a loose fibromyxoid stroma with variable inflammatory infiltrate (14). Ulceration of the overlying epithelium is common, especially in oral lesions subjected to recurrent trauma. With maturation, the lesion becomes more fibrotic and less vascular, correlating with its clinical transition from a soft, red nodule to a firmer, pinkish mass (15). Immunohistochemical studies have demonstrated overexpression of vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), and angiopoietin-2, which collectively promote endothelial proliferation and capillary formation (16).

Clinically, PG must be differentiated from a range of hyperplastic and neoplastic lesions including peripheral ossifying fibroma, hemangioma, peripheral giant cell granuloma, and metastatic carcinoma (17). The presence of a history of rapid development and spontaneous bleeding favors PG, but definitive differentiation requires histopathologic confirmation (18). Intraoral PGs frequently arise on the gingiva due to chronic local irritation from plaque, calculus, or ill-fitting restorations, emphasizing the importance of meticulous oral hygiene both pre- and postoperatively (19). Surgical excision remains the mainstay of treatment, with complete removal of the lesion and underlying irritants to minimize recurrence (20). The excised specimen should always be submitted for histopathologic evaluation to confirm the diagnosis and rule out malignancy. Minimally invasive modalities such as laser excision (CO_2, Nd:YAG, diode) and electrocautery have gained popularity due to advantages such as reduced intraoperative bleeding, faster healing, and better cosmetic outcomes (21). Emerging therapies, including topical beta-blockers like 0.5% timolol, exert anti-angiogenic effects by downregulating VEGF and may serve as a conservative alternative for small or pediatric lesions (22). Recurrence rates have been reported between 5% and 16%, primarily resulting from incomplete excision or

persistence of local irritants (23). In pregnancy-associated cases, lesions may regress spontaneously after delivery as hormonal levels normalize; however, persistent or symptomatic lesions may require conservative surgical management (24). Close postoperative monitoring is essential to ensure complete healing and to detect potential recurrence early.

CONCLUSION

Pyogenic granuloma is a common reactive vascular lesion resulting from trauma, hormonal changes, or drug influences. Although benign, it often causes functional and aesthetic concerns due to bleeding and recurrence. Proper diagnosis, removal of causative factors, and complete excision or appropriate conservative treatment lead to successful outcomes.

ACKNOWLEDGMENT
The authors would like to acknowledge the Department of Periodontology, PGIDS, Rohtak, for their support and cooperation.

CONFLICT OF INTEREST
The authors declare no conflicts of interest related to this study.

REFERENCES

[1]. Neville BW, Damm DD, Allen CM, Chi AC. Oral and Maxillofacial Pathology. 4th ed. Elsevier; 2016.
[2]. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: A review. J Oral Sci. 2006;48(4):167–175.
[3]. Marla V, Shrestha A, Goel K, Shrestha S. The histopathological spectrum of pyogenic granuloma: A case series. Case Rep Dent. 2016;2016:1323798.
[4]. Reddy V, Saxena S, Aggarwal P. Drug-induced pyogenic granuloma: A case report and review. J CutanAesthet Surg. 2014;7(3):188–191.
[5]. Rossi E, Mandato F, et al. Pyogenic granuloma: Diagnosis and management—a practical review. Dermatol Ther (Heidelb). 2023;13(4):1127–1139.
[6]. Saravana GH. Oral pyogenic granuloma: A review of 137 cases. Br J Oral Maxillofac Surg. 2009;47(4):318–319.
[7]. Yuan K, Jin YT, Lin MT. Expression of vascular endothelial growth factor and angiopoietin-2 in pyogenic granuloma. J Periodontol. 2000;71(5):702–709.
[8]. Kamal R, Dahiya P, Puri A. Oral pyogenic granuloma: Various concepts of etiopathogenesis. J Oral Maxillofac Pathol. 2012;16(1):79–82.
[9]. Eversole LR. Clinical Outline of Oral Pathology. 4th ed. Lea &Febiger; 2002.
[10]. Lee J, Sinno H, Tahiri Y, Gilardino MS. Treatment options for cutaneous pyogenic granulomas: A review. J Plast ReconstrAesthet Surg. 2011;64(9):1216–1220.
[11]. Patra AC, et al. Efficacy of topical 0.5% timolol in pyogenic granuloma: A randomized controlled trial. Indian Dermatol Online J. 2022;13(3):352–356.
[12]. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: a review. J Oral Sci. 2006;48(4):167–175.
[13]. Neville BW, Damm DD, Allen CM, Chi AC. Oral & Maxillofacial Pathology, 5th ed. Elsevier; 2022.
[14]. Kamal R, Dahiya P, Puri A. Oral pyogenic granuloma: Various concepts of etiopathogenesis. J Oral Maxillofac Pathol. 2012;16(1):79–82.
[15]. Parisi E, Glick PH. Histopathologic spectrum of pyogenic granuloma. Am J Dermatopathol. 2018;40(8):561–566.
[16]. Yuan K, Jin YT, Lin MT. Expression of VEGF and bFGF in pyogenic granuloma. J Periodontol. 2000;71(5):701–709.
[17]. Regezi JA, Sciubba JJ, Jordan RCK. Oral Pathology: Clinical-Pathologic Correlations, 7th ed. Elsevier; 2017.
[18]. Esmeili T, Lozada-Nur F, Epstein J. Common oral lesions: differential diagnosis and management. Dent Clin North Am. 2005;49(1):55–70.
[19]. Gomes SR, Shakir QJ, Thaker PV, Tavadia JK. Pyogenic granuloma of the gingiva: A misnomer? – A case report and review. J Indian Soc Periodontol. 2013;17(4):514–519.
[20]. Bhaskar SN, Jacoway JR. Pyogenic granuloma—Clinical features, incidence, histology, and result of treatment: Report of 242 cases. J Oral Surg. 1966;24(5):391–398.
[21]. Raulin C, Greve B, Hammes S. The combined continuous-wave/pulsed carbon dioxide laser for treatment of pyogenic granuloma. Br J Dermatol. 2002;146(6):1042–1045.
[22]. Gupta S, Kumar B. Use of topical timolol for the treatment of pyogenic granuloma. J Am Acad Dermatol. 2014;70(4):e85–e86.
[23]. Ojanotko-Harri A, Harri MP, Hurttia HM. Factors affecting recurrence of oral pyogenic granuloma after surgical excision. Acta Odontol Scand. 1990;48(5):377–381.
[24]. O'Neil T, Sandor GKB, Carmichael RP. Pregnancy tumor: Oral pyogenic granuloma associated with pregnancy. J Can Dent Assoc. 2010;76:a74.