5-2021

A Firm Mass of the Maxillary Gingiva

Brian P. Ford
University of Pennsylvania

Alhendi Fatmah

Faizan Alawi
University of Pennsylvania

Farshid Sanavi

Recommended Citation

Ford, B. P., Fatmah, A., Alawi, F., & Sanavi, F. (2021). A Firm Mass of the Maxillary Gingiva. *Journal of the American Dental Association, 152* (5), 402-407. http://dx.doi.org/10.1016/j.adaj.2020.04.028

A Firm Mass of the Maxillary Gingiva

Diagnostic Challenge

A firm mass of the maxillary gingiva

Brian P. Ford, DMD, MD; Fatmah Alhendi, DDS; Faizan Alawi, DDS; Farshid Sanavi, DMD, PhD; Eric T. Stoopler, DMD

THE CHALLENGE

A 57-year-old woman had a slow-growing mass affecting the maxillary right posterior gingiva of 2 years' duration. She denied initial trauma to the area and was asymptomatic. Her medical history was significant for gastroesophageal reflux disorder and attention deficit disorder. Surgical, family, and social histories were unremarkable. Medications included ranitidine hydrochloride, dexlansoprazole, dexmethylphenidate hydrochloride, lamotrigine, and citalopram hydrobromide. The patient reported no known drug allergies and food allergies associated with broccoli, cauliflower, and red dye. The review of systems was unremarkable. The extraoral examination revealed a well-nourished, well-developed woman with no evidence of lymphadenopathy, salivary gland enlargement, or thyromegaly. The intraoral examination revealed a pink-red, soft, nontender, 2-centimeter mass on the buccal gingiva in the area of tooth no. 2 with an intact mucosal surface (Figure 1). Tooth no. 2 responded normally to thermal testing. A periapical radiograph of tooth no. 2 (Figure 2A) and a cone-beam computed tomographic scan (Figure 2B) did not reveal dental or intrabony pathology in the area of the gingival lesion. An excisional biopsy was performed, and the soft-tissue defect was closed with a local mucoperiosteal advancement flap. The histopathologic examination revealed a soft-tissue lesion composed of haphazardly arranged, plump, uniform fibroblasts in an immature collagenous stroma with scattered metaplastic calcifications (Figures 3 and 4).

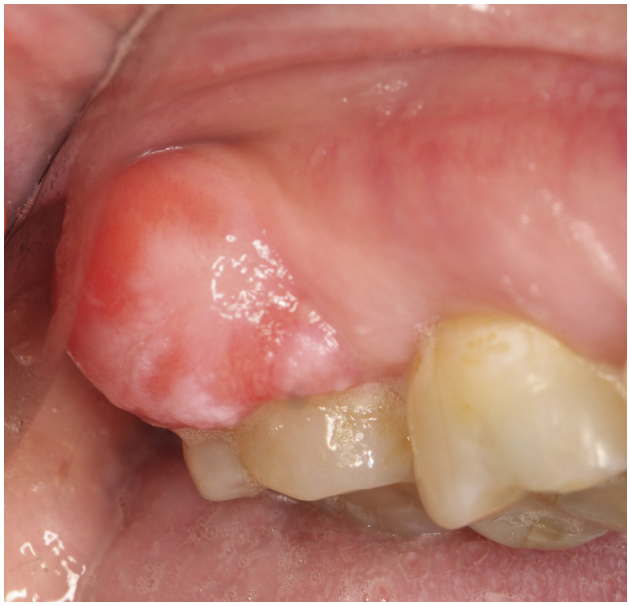

Figure 1. Pink-red, soft, nontender, 2-centimeter mass on the buccal gingiva in the area of tooth no. 2 with an intact mucosal surface.

(Please see next page for additional images.)

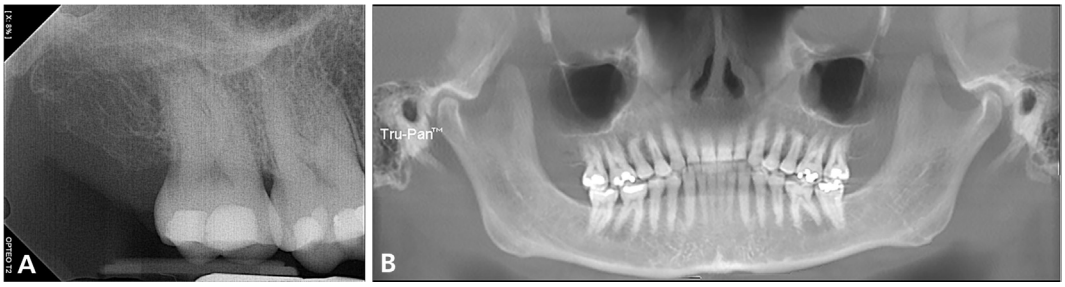

Figure 2. **A.** Unremarkable periapical radiograph of tooth no. 2. **B.** Unremarkable cone-beam computed tomographic scan.

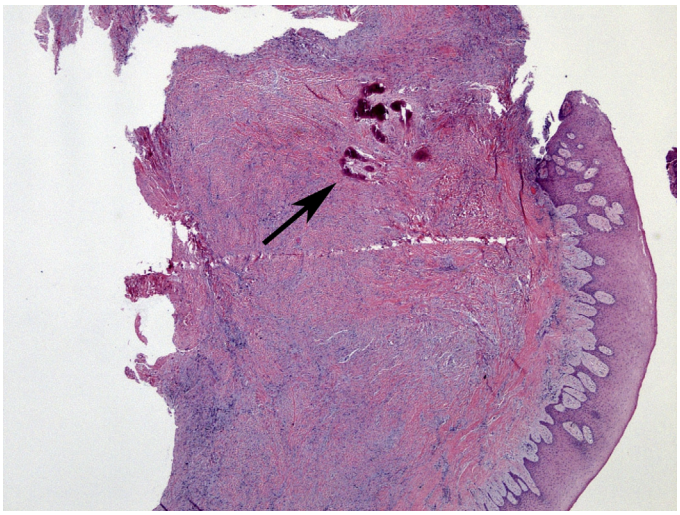

Figure 3. Fibrotic mass with metaplastic bone (arrow) (hematoxylin and eosin, magnification ×20).

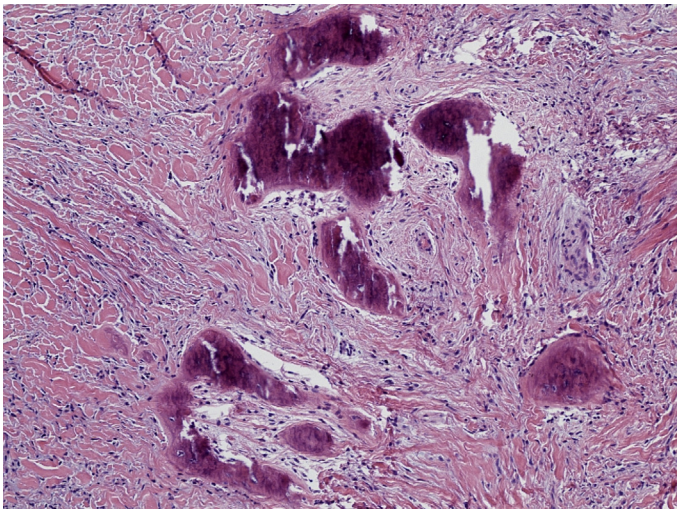

Figure 4. Scattered fibroblasts, collagen, and metaplastic bone (hematoxylin and eosin, magnification ×100).

Can you make the diagnosis?

A. Pyogenic granuloma
B. Peripheral giant cell granuloma
C. Peripheral ossifying fibroma
D. Fibroma
E. Peripheral ameloblastoma

The diagnosis:

C. Peripheral ossifying fibroma

Peripheral ossifying fibroma (POF) is a gingival growth with unclear pathogenesis. It is considered to be reactive rather than neoplastic in nature.[1] POF is a lesion predominantly found in teenagers and young adults, with peak prevalence from age 10 through 19 years. Almost two-thirds of all cases occur in females.[2] There is a slight predilection for the maxillary arch, and more than 50% of all cases occur in the incisor-canine region.[3] Usually teeth are unaffected by POF; rarely, there can be migration and loosening of adjacent teeth.

The etiology of POF is not well understood; there are 2 prevailing theories. The first is that a preexisting pyogenic granuloma (PG) becomes calcified, and the second is that POFs originate from an inflammatory hyperplasia in the cells of the periodontal ligament.[4] The second theory is generally more accepted with evidence of calcified matrix-rich oxytalan fibers included in the POF.[4,5] The inflammatory reaction in POF is secondary to local trauma, and this chronic irritation of the periosteum/periodontal ligament causes the formation of metaplastic bone, dystrophic calcifications, or both.[6]

Clinically, POF occurs exclusively on the gingiva as a pedunculated or sessile mass that has often been present for many weeks or months. The color ranges from pink to red. Most lesions are asymptomatic and less than 2 cm in size.[1] The microscopic appearance of a POF has a basic pattern of fibrous proliferation associated with the formation of a mineralized product. The deeper fibroblastic component often is cellular, especially in areas of mineralization.[1] The treatment of choice for POF is local surgical excision down to periosteum. The adjacent teeth should be thoroughly scaled to eliminate any possible irritants. Although excision is usually curative, a recurrence rate of 8% through 18% has been reported, mainly owing to incomplete removal.[7-11]

DIFFERENTIAL DIAGNOSIS

PG

PG is a tumorlike growth that is nonneoplastic in nature.[12] PGs manifest as a pedunculated pink, red, or purple mass, often with an ulcerated surface. PGs vary in size from a few millimeters to several centimeters in diameter. They are generally painless; however, because of their vascularity, bleeding is common. PGs may exhibit rapid growth, which may be alarming to the patient.[1,12] Although PG was originally thought to be caused by pyogenic organisms, it is now known to be unrelated to infection.[1] Instead, PG is thought to represent an exuberant tissue response to local irritation or trauma. PGs show a strong predilection for the gingiva, accounting for 75% through 85% of all cases.[1] When oral PGs manifest on the gingiva, the most common location is the marginal gingiva, with only 15% occurring on the alveolar portion.[12]

PGs can develop at any age; however, they are most common in children and young adults. Gingival PGs frequently develop in pregnant women, and the terms "pregnancy tumor" or "granuloma gravidarum" are commonly used to describe them. Development of these lesions in pregnancy may be related to fluctuating levels of estrogen and progesterone levels during pregnancy.[13] The histologic appearance of PGs is notable for a highly vascular proliferation that resembles granulation tissue. There are numerous endothelium-lined channels with red blood cells and a mixed inflammatory cell infiltrate. Older lesions may have areas with a more fibrous appearance.[1]

Treatment of PG consists of conservative surgical excision, which is usually curative. A recurrence rate of 3% through 15% has been reported.[14] For lesions that develop during pregnancy, treatment should be deferred unless significant functional or esthetic problems develop, as some lesions resolve spontaneously after parturition.[14]

Peripheral giant cell granuloma

Peripheral giant cell granuloma (PGCG) is a tumorlike growth of the oral cavity that is not a true neoplasm, but rather a reactive lesion caused by local irritation or trauma. It occurs exclusively on the gingiva and manifests as a red or red-blue mass.[1] PGCG can develop at almost any age with a mean age of diagnosis of 31 through 46 years.[15] Although PCGCs develop within soft tissue, resorption of the underlying alveolar bone is seen in almost one-third of cases.[15] PGCG bears a close

microscopic resemblance to central giant cell granuloma and may represent a soft-tissue counterpart of this intraosseous lesion. Treatment of PCGC consists of local surgical excision down to the underlying bone, and curettage or peripheral ostectomy may be beneficial. Approximately 10% of lesions are reported to recur.[15]

Fibroma

Fibroma is the most common lesion of the oral cavity and represents a reactive process resulting from local irritation or trauma.[1,16] A fibroma can occur anywhere in the mouth; the most common locations are the buccal mucosa, labial mucosa, tongue, and gingiva.[17] Gingival fibromas may represent fibrous maturation of a preexisting PG.[14] Fibromas typically appear as an asymptomatic sessile, smooth-surfaced, pink nodule. The surface may appear white as a result of hyperkeratosis from repeated irritation.[17] Fibromas vary in size but are generally less than 1.5 cm in diameter.

Microscopic examination reveals a nodular mass of fibrous connective tissue covered by stratified squamous epithelium. Lymphocytes and plasma cells may be present as the result of chronic inflammation.[1] Fibroma is treated by conservative surgical excision, and recurrence is rare.[17]

Peripheral (extraosseous) ameloblastoma

Peripheral ameloblastoma (PA) accounts for approximately 1% through 4% of all ameloblastomas.[1] The tumor has been observed in patients across a wide age range, but most are seen in middle-aged people, with an average reported age of 52 years.[18] PAs typically manifest as a painless, slow-growing, nonulcerated, sessile, or pedunculated gingival lesion. They are most commonly found on the posterior gingival and alveolar mucosa. Occasionally, the alveolar bone may have a superficial "cupping" erosion. Histopathologically, these lesions have the same features as the intraosseous form of the tumor, including the presence of peripheral palisading columnar cells exhibiting reverse nuclear polarization away from basement membrane, and stellate reticulum.[1] Compared with the intraosseous ameloblastoma, the PA is less aggressive and responds well to local surgical excision.[19]

CONCLUSIONS

Nodular lesions of the gingiva may present a diagnostic challenge. This case is unique because of the anatomic location of POF and patient's age at the time of diagnosis. Oral health care providers should be knowledgeable about POF to provide optimal health care. ■

https://doi.org/10.1016/j.adaj.2020.04.028

Dr. Ford is an assistant professor of oral and maxillofacial surgery/pharmacology, Department of Oral and Maxillofacial Surgery/Pharmacology, Penn Dental Medicine, Philadelphia, PA.

Dr. Alhendi is a resident in the Department of Oral Medicine, Penn Dental Medicine, Philadelphia, PA.

Dr. Alawi is an associate professor of pathology, Department of Basic and Translational Sciences, Penn Dental Medicine, Philadelphia, PA.

Dr. Sanavi is a clinical professor, Department of Periodontics, Kornberg School of Dentistry, Temple University, Philadelphia, PA.

Dr. Stoopler is a professor of oral medicine, Department of Oral Medicine, Penn Dental Medicine, 240 South 40th St, Room 206, Philadelphia, PA 19014, e-mail ets@upenn.edu. Address correspondence to Dr. Stoopler.

Disclosure. None of the authors reported any disclosures.

Diagnostic Challenge is published in collaboration with the American Academy of Oral and Maxillofacial Pathology and the American Academy of Oral Medicine.

1. Neville BW, Damm DD, Allen CM, Chi A. *Oral and Maxillofacial Pathology*. 4th ed. Philadelphia, PA: Saunders Elsevier; 2016.
2. Kenney JN, Kaugars GE, Abbey LM. Comparison between the peripheral ossifying fibroma and peripheral odontogenic fibroma. *J Oral Maxillofac Surg*. 1989;47(4):378-382.
3. Poon CK, Kwan PC, Chao SY. Giant peripheral ossifying fibroma of the maxilla: report of a case. *J Oral Maxillofac Surg*. 1995;53(6):695-698.
4. Manes RP, Ryan MW, Batra PS, Mendelsohn D, Fang YV, Marple BF. Ossifying fibroma of the nose and paranasal sinuses. *Int Forum Allergy Rhinol*. 2013;3(2):161-168.
5. Childers ELB, Morton I, Fryer CE, Shokrani B. Giant peripheral ossifying fibroma: a case report and clinicopathologic review of 10 cases from the literature. *Head Neck Pathol*. 2013;7(4):356-360.
6. Mishra AK, Maru R, Dhodapkar SV, Jaiswal G, Kumar R, Punjani H. Peripheral cemento-ossifying fibroma: a case report with review of literature. *World J Clin Cases*. 2013;1(3):128-133.
7. Gulati R, Khetarpal S, Ratre M, Solanki M. Management of a massive peripheral ossifying fibroma using diode laser. *J Indian Soc Periodontol*. 2019;23(2):177-180.
8. Akkara F, Chalakkal P, Boyapati C, Pavaskar R. Peripheral ossifying fibroma secondary to pulpoperiodontal irritation. *J Clin Diagn Res*. 2013;7(9):2076-2077.
9. Dahiya P, Kamal R, Saini G, Agarwal S. Peripheral ossifying fibroma. *J Nat Sci Biol Med*. 2012;3(1):94-96.
10. Mergoni G, Meleti M, Magnolo S, Giovannacci I, Corcione L, Vescovi P. Peripheral ossifying fibroma: a clinicopathologic study of 27 cases and review of the literature with emphasis on histomorphologic features. *J Indian Soc Periodontol*. 2015;19(1):83-87.

11. Franco-Barrera MJ, Zavala-Cerna MG, Fernández-Tamayo R, Vivanco-Pérez I, Fernández-Tamayo NM, Torres-Bugarín O. An update on peripheral ossifying fibroma: case report and literature review. *Oral Maxillofac Surg*. 2016;20(1):1-7.
12. Vilmann A, Vilmann P, Vilmann H. Pyogenic granuloma: evaluation of oral conditions. *Br J Oral Maxillofac Surg*. 1986;24(5):376-382.
13. Epivatianos A, Antoniades D, Zaraboukas T, et al., Pyogenic granuloma of the oral cavity: comparative study of its clinicopathological and immunohistochemical features. *Pathol Int*. 2005;55(7):391-397.
14. Jafarzadeh H, Sanatkhani M, Mohtasham N. Oral pyogenic granuloma: a review. *J Oral Sci*. 2006;48(4):167-175.
15. Chrcanovic BR, Gomes CC, Gomez RS. Peripheral giant cell granuloma: an updated analysis of 2824 cases reported in the literature. *J Oral Pathol Med*. 2018;47(5):454-459.
16. Torres-Domingo S, Bagan JV, Jiménez Y, et al., Benign tumors of the oral mucosa: a study of 300 patients. *Med Oral Patol Oral Cir Bucal*. 2008;13(3):161-166.
17. Bouqout JE, Nikai H. Lesions of the oral cavity. In: Gnepp DR, ed. *Diagnostic Surgical Pathology of the Head and Neck*. Philadelphia, PA: Saunders; 2001: 141-238.
18. Philipsen HP, Reichart PA, Nikai H, Takata T, Kudo Y. Peripheral ameloblastoma: biological profile based on 160 cases from the literature. *Oral Oncol*. 2001;37(1):17-27.
19. LeCorn DW, Bhattacharyya I, Vertucci FJ. Peripheral ameloblastoma: a case report and review of the literature. *J Endod*. 2006;32(2):152-154.