Review Article

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Types and characteristics of gingival enlargements

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ABSTRACT

One of the common symptoms of gingival disorders is gingival hypertrophy. However, the practitioner finds it difficult to diagnose these entities because of their diverse manifestations. According to their etiology and pathogenesis, site, volume, severity, etc., they can be grouped. A differential diagnosis can be made using the available information and clinical experience. The practitioner then provides a definitive diagnosis or a diagnosis of elimination following a thorough examination. The care of these lesions and the control of their recurrence entirely rely on an accurate diagnosis, so it is crucial. Further, a proper diagnosis of these enlargements may save the patient's life or at the very least initiate therapy early and enhance their quality of life in situations when gingival enlargement (GE) may be the first indicator of possibly deadly systemic disorders. When making a differential diagnosis of local (isolated, discrete, regional) or widespread GE, one can maintain a broad perspective by being conscious of the presence of typical and uncommon GE forms.

Keywords: Gingival hyperplasia, GE, Gingival diseases

INTRODUCTION

The effective treatment of GE, which is a frequent occurrence in clinical practice hinges on correctly identifying the etiology. The interdental papillae appear to be most frequently connected with the most prevalent type of GE, which is caused by plaque-induced inflammatory changes of the surrounding gingival tissues (inflammatory hyperplasia). This inflammation can be isolated or widespread. Such GE may be exacerbated by hormonal changes, such as those associated with puberty and pregnancy, as well as by some systemic drugs. Particularly when the gingival tissue is edematous,

plaque-induced inflammatory hyperplasia should go away with removal of plaque and calculus and enhanced dental hygiene. If the gingival tissue is fibrotic, the GE may not resolve, leaving periodontal pockets that make it difficult to maintain good dental hygiene. In this situation, a more thorough evaluation and a longer-term care strategy that maps the degree of gingival and maybe periodontal affliction are required. It can be necessary to perform surgery to remove swollen tissue and increase the patient's accessibility for dental hygiene. Other kinds of GE exist in addition to plaque-induced GE, spanning from malignant illness to the bland gingival fibrous nodule and retrocuspid papilla.^{2,3} In the past,

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pedunculated or sessile GE have been referred to as epulides, which is the term used to describe isolated GEs.⁴ However, since epulides is a topographical concept and cannot be utilized to describe a particular lesion histologically, the phrase "reactive lesion of the gingiva" has frequently been employed in its place.⁵ Reactive GE that are localized which make up a category of epulides, differ from plaque-induced inflammatory GEs in terms of a variety of clinically significant ways. ^{5,6} With this differentiation, a medical diagnosis is possible and a care plan that aims to reduce recurrence is defined.⁷ The origin of these few epulides from the suprabony fibers of the periodontium and their fundamental reactive and non-inflammatory character are their two distinguishing characteristics. These enable a logical justification for their clinical picture and behavior. Particularly, these epulides are not just an GE of the frequently inflammatory interdental papilla because they do not arise from the gingival edge. They can develop anywhere on the free gingival margin, and they often have a gingival margin that is cervically displaced and develop out of sulcus of gingiva. Natural free gingival margin, which is often seen stretching across abnormalities, indicates lesion's source, predominant growth pattern (supra or subgingival), and likelihood that attached gingiva and mucogingival junction will be disrupted during any later operative procedure. Overall demographic characteristics of number of sizable case studies have been reported, and they are consistent in that peripheral fibroma (PF) is most common condition seen, followed by angiogranuloma, peripheral fibroma with calcification, and peripheral giant (AG) cell granuloma (PGCG).^{5,6} Although there is significant variation in male to female ratio, majority of investigations show a ratio between 1:1.31 for PF and 1:1.99 for AG and 1:1.5 for PGCG.⁶ Position and dimension also vary but maxilla is predominant presenting location and dimension ranges from 0.5-1.5 cm.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on December 5, 2022, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about the types and characteristics of GEs. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

Isolated reactive lesions of the gingiva

The phrase "localized enlargement of gingiva," also known as "epulis" historically, pertains to any

isolated/discrete, pedunculated or sessile growths of the gingiva without a specific histological disease being identified.⁸ For this group of growths, the correct terminology "reactive lesion of the gingiva" seems more fitting.⁵

Fibrous epulis/peripheral fibroma

Adults usually present with this growth as a solid, pinkish, non-inflamed lump that appears to originate underneath the free gingival margin or interdental papilla. The growth is typically pain free. Additional trauma from biting, flossing, or brushing may cause injury. Histopathologically, the fibroma may also exhibit centers of cementicles (peripheral cementifying fibroma), osseous trabeculae (peripheral ossifying fibroma), or more foci of mineralization (peripheral calcifying fibroma).

Angiogranuloma/pyogenic granuloma

Adults experience this condition as a smooth-surfaced mass that often has ulcers and emerges from the gingival margin. These extremely vascular, collapsible, and potentially bleeding masses are red/blue in appearance. They generally grow quickly for the initial weeks, then gradually. Although osseous erosion is rare, the lump may extend beyond the interdental region and appear as a bilobular (buccal and lingual) bulge linked through the col region. Angiogranulomas that develop in pregnancy are known as granuloma gravidarum or pregnancy epulis/tumor. The stratified squamous epithelial layer is swollen histologically, has extensive rete pegs, some edema inside the cells and externally, conspicuous intercellular bridging, as well as the leukocytic involvement.

Peripheral giant cell granuloma

In young patients or individuals with mixed dentition, they are more common in the anterior portion of the mouth. These lesions are extremely aggressive and have a large growth potential. These growths have a high vascularity, which is why they hemorrhage often and are reddish-purple in color. Additionally, they frequently burrow between teeth, eroding nearby bone and separating neighboring teeth.

Gingival cysts

These are uncommon odontogenic cysts. They are typically discovered in women in their 50s or 60s. These are more prevalent on the lower anterior dentition's facial attached gingiva. They may have a blue tint from the fluid present, and compression from them may cause the labial bone to resorb. Its radiolucency on radiographs might occasionally cause misunderstanding with a lateral periodontal cyst. The optimum treatment for these lesions is excision and biopsy.⁹

Neoplastic

Discrete epulis-like lesions can also be divided into benign and neoplastic categories. Myoblastoma, PGCG, central giant cell granuloma, papilloma, leukoplakia, nevus, hemangioma, neurilemoma, neurofibroma, and ameloblastoma are examples of innocuous growths. 9,10 Melanoma or squamous cell carcinoma are examples of cancerous tumors. Kaposi's sarcoma is the most prevalent sarcoma, while fibrosacroma, lymphosarcoma, and reticulum cell sarcoma are less commonly noted. 11,12

Acute

Different abscesses, including gingival, periodontal, periapical, or pericoronal, might manifest as the acute type of localized GE. It may be widespread and make up a sizeable amount of the attached gingiva, or it may be situated close to the gingival margin or papilla. The lesion may have developed from an endodontic condition (periapical abscess/endo-perio lesion) when the tooth with which it is related is not alive. The pericoronal flap that covers the distalmost lower teeth may frequently swell and become inflammatory. If inflammation doesn't go away, these pericoronal folds may ultimately create an abscess. The suppurative core in the connective tissue of an abscess originating in the of a gingiva, periodontium, or pericoronal area may be accompanied by diffuse polymorphonuclear leukocyte infiltrate, edematous tissue, and vascular engorgement on histological evaluation. Leukocyte penetration, intracellular and extraneous edema, and occasionally ulceration is present in varying degrees on the surface epithelium.

Characteristic presentation of generalized GE

Most frequently gingival disease appears as localized/generalized GE, which belong to one of several forms.

Inflammatory GE

These are inflammatory reactions to nearby irritations connected to the gingiva. Microbiological buildups (plaque and calculus), cracked teeth, overhanging fillings, improperly placed prosthetics, orthodontic brackets, etc. could all be the cause of the irritation. Based on where the irritation is, the manifestation starts as a small bulging of the papilla or marginal gingiva. The swelling may gradually grow larger and spread out to become more noticeable. They could seem blue or intense red in the clinic. They commonly hemorrhage easily and have a smooth, shiny appearance that is friable and delicate. A hard, tenacious, pinkish, and fibrotic GE with many fibroblasts and collagen fibers can sometimes accompany persistent inflammatory GE.

GE in mouth breathers

The precise process of GE in mouth breathers is unclear, despite the fact that it is thought to be inflammatory. It is

believed to be caused by the gingival surface being alternately wet and dry. The gingiva has a dappled glossy appearance and appears reddish and edematous. A distinguishing characteristic of this sort of GE would be the absence of posterior involvement and the occurrence of considerable GE in the upper and lower anterior regions. The palatal aspect of the upper anteriors and the facial aspect of the lower anteriors will be enlarged in a classic bimaxillary protrusion situation. Individuals who have a pattern of breathing via their mouths may have a small upper lip, an overactive labii superioris, proclination of incisors, rhinitis, or another condition.

Fibrotic GE

enlargement: Drug induced Numerous immunosuppressants, calcium channel blockers, and antiepileptics can all cause different manifestations of gingival hypertrophy. Within 2-4 months of starting a clinical medication regimen, manifestations there is no pain at the GE appear. Typically, first presentation. The interdental papilla first enlarges in a bead-like appearance, and subsequently the marginal gingiva may be affected. The GE has a mulberry appearance, is firm, pinkish, and resilient with small lobulations, and does not hemorrhage easily. All teeth may be affected, but the upper and lower anteriors show it more visibly. It would not exist in edentulous places and will subside in regions where teeth have undergone extraction. Secondary infection results in a growth of the GE that already exists as well as the addition of inflammatory GE's distinctive features. The most prominent drug-induced GE cases are those brought on by immunosuppressants like cyclosporine, which seem more vascularized than those brought on by phenytoin.¹³ It is unclear which symptoms should be assigned to this diagnosis in patients who are receiving combined treatment, in which two or more medications are associated with gingival hypertrophy. In these situations, consulting the patient's doctor and asking him to replace/terminate each medication one at a time, beginning with the one which would have the slightest impact on the patient's normal schedule, is one method for arriving at a judgment. Blood pressure medications, antiepileptics, and immunosuppressants are commonly discussed in the patient's medical history, although the enlargement has just been seen recently. In these situations, it becomes challenging to relate the timeframe of the GE's incidence to a relevant medication history. To link the two, a specific question about a recent shift in medication kind or dosage will be helpful.

Genetic disorders linked with enlargement: Based on their pathogenesis, clinical characteristics, and histopathology, the four basic types of genetic diseases linked to GE can be identified. Specifically, those linked to classic dental anomalies, lysosomal storage conditions, vasculopathies, and idiopathic GE. Other names for idiopathic GE include elephantiasis, hereditary familial fibromatosis, gingivomatosis, and idiopathic fibromatosis.

It manifests as an atypical local or widespread fibrotic gingival hypertrophy. A positive familial history of GE can help in diagnosis. The emergence of the deciduous or permanent teeth typically marks the beginning of it. A common observation could be the existence of dense, hard gingiva that is exclusively present in the second and third molar portions of the maxilla and mandible. On palpation, the enlarged mass may feel hard or nodular and may be pinkish or red. Although pseudo-pockets and trouble keeping up with oral care can cause periodontal issues, alveolar bone is infrequently damaged. Large overgrowths may cause the patient to experience aesthetic and functional issues.

Conditioned enlargement

Hormonal: Hormonal fluctuations that modify the responses to regional irritants throughout pregnancy and puberty affect widespread gingival hyperplasia. More obviously enlarged than the facial and/or lingual surfaces is the interdental gingiva. The enlarged gingiva often has a smooth, shiny appearance, is delicate and friable, and is vivid crimson or magenta in color. Hemorrhage can happen spontaneously or in response to minor provocation. After delivery, the bulge might naturally shrink, but full removal might necessitate the operative removal of all nearby irritants and any fibrotic residues.

Vitamin C deficiency: When the concentration of serum ascorbic acid is less than 2 g/mL, a vitamin C deficiency is diagnosed. Tobacco, stress, and diabetes mellitus are the three most frequently mentioned risk factors for mild vitamin C insufficiency. The bluish crimson, delicate, and friable gingiva of vitamin C deficiency-related enlargement has a smooth, shining appearance. Hemorrhage can happen on its own or after a minor irritant. Also frequently observed are superficial necrosis and pseudomembrane formation. High-sensitivity C-reactive Protein (hs-CRP) concentrations were found to be negatively correlated with serum vitamin C levels, according to Kubota et al suggesting that these patients may have raised hs-CRP blood levels. 15

Plasma cell gingivitis: Although the cause of this condition is unclear, it is thought to be a hypersensitive reaction with abundant plasma cells as revealed histologically. Common allergies that have been linked to this lesion include toothpaste, culinary products, especially cinnamon, chewing gum, and substances of uncertain cause. It could bleed if provoked. Patients frequently complain about burning after consuming hot or spicy food. The look is often red in hue, nearly entirely composed of attached gingiva, and with a slightly granular surface.

GE in systemic conditions

In leukemia, the widespread influx of leukemic cells in the gingival connective tissue is the cause of the widespread gingival hypertrophy linked to leukemia. Clinically, it might resemble an inflammatory source. Other characteristics that may be present in addition to enlarged gingiva include oral ulcers, sudden gingival bleeding, petechiae, mucosal pallor, herpes infections, and thrush. Infrequently, unusual characteristics like chin numbness and/or tooth ache have been reported. Leukemia is the most severe disorder in this group related to gingival hypertrophy. Ecchymoses, night sweats, recent infections, and tiredness are some indicators of bone marrow failure that it may be connected with. An easy full blood count can quickly provide a diagnosis. Additionally, a rare instance of acute lymphoblastic leukemia-related gingival hyperplasia has been documented. Leukemia-related gingival hyperplasia has been documented.

Wegener's Granulomatosis is characterized by strawberry gingivitis, a red-purplish exophytic GE with petechial hemorrhages. Due to their persistence for a significant period of time before multiple organ involvement, oral lesions may be extremely helpful in the early detection of this possibly deadly disorder. ^{18,19} For a diagnosis, at least two of the below-mentioned criteria must be met: (1) ulcerations of the oral mucosa or nasal bleeding or inflammation; (2) nodules, fixed infiltrates, or cavities on chest imaging; (3) aberrant urinary sedimentation; and (4) granulomatous inflammation on biopsy. ¹⁹

In Crohn's disease, the gingiva is pinkish, tough, almost leathery in nature, and has a distinctive texture that is finely pebbly. These people need to be closely monitored for any indicators of these illnesses. It frequently goes hand in hand with lip puffiness, gastrointestinal problems, fever, and ulceration.

Sarcoidosis is a condition where lung infiltration and hilar lymphadenopathy, as well as cutaneous and eye lesions, make up the most typical presentation. However, oral presence is unusual.²⁰ For sarcoidosis, there are no specialized testing. Excluding other disorders that do not produce non-caseating granulomas and doing additional laboratory studies are the primary components of a sarcoidosis diagnosis.^{20,21} Both serum angiotensin converting enzyme levels (appropriate less than 670 nkat/L) and eosinophil count may be considerably elevated (normal range 0-4%). Hilar lymphadenopathy may be visible on a chest X-ray.²²

Tuberculous GE is a primary oral tuberculous lesion which is extremely uncommon. When they do develop, they frequently affect children. In the majority of instances, the lesions themselves are asymptomatic, but caseation of the dependent lymph nodes may be visible. ^{23,24} Additionally, primary tuberculosis that just causes GE is incredibly uncommon and can be identified by a history of pyrexia, lethargy, decreased appetite, and weight loss. ²⁵ Histologically, a complete blood count, and a polymerase chain reaction can all be used to confirm the diagnosis. ²⁵ While the frequency is higher in elderly

people, secondary oral TB can be detected in 0.05% to 1.5% of cases. ^{26,27}

CONCLUSION

Despite having a wide range of causes, GEs can frequently be identified by a thorough history, site, or clinical manifestations. Localized irritations (plaque and calculus) may be the primary reason or a contributing factor in GEs. Plaque reduction is therefore a crucial component of patient management in general. For some rare cases of GE, a hematological or histological investigation, an excision or incision biopsy, or both may be required to make the accurate diagnosis. Before making a conclusive diagnosis of the problem at hand, the doctor should have an open mind and take all potential diagnoses into account.

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REFERENCES

- 1. Seymour RA. Effects of medications on the periodontal tissues in health and disease. Periodontology. 2000. 2006;40(1):120-9.
- 2. Giunta JL. Gingival fibrous nodule. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol. 1999;88(4):451-4.
- 3. Savage N, Daly C. Gingival enlargements and localized gingival overgrowths. Aust Dental J. 2010;55:55-60.
- 4. Cooke B. The fibrous epulis and the fibroepithelial polyp: their histogenesis and natural history. Br Dent J. 1952;93:305-9.
- 5. Kfir Y, Buchner A, Hansen LS. Reactive lesions of the gingiva: a clinicopathological study of 741 cases. J Periodontol. 1980;51(11):655-61.
- 6. Zhang W, Chen Y, An Z, Geng N, Bao D. Reactive gingival lesions: a retrospective study of 2,439 cases. Quintessence Int. 2007;38(2).
- 7. Bosco AF, Bonfante S, Luize DS, Bosco JMD, Garcia VG. Periodontal plastic surgery associated with treatment for the removal of gingival overgrowth. J Periodontol. 2006;77(5):922-8.
- 8. Ingles E, Rossmann JA, Caffesse RG. New clinical index for drug-induced gingival overgrowth. Quintessence Int. 1999;30(7).
- 9. Giunta JL. Gingival cysts in the adult. J Periodontol. 2002;73(7):827-31.
- 10. Allen R, Bruce K. Nevus of the gingiva; report of case. J Oral Surg. 1954;12(3):254-6.
- 11. Lager I, Altini M, Coleman H, Ali H. Oral Kaposi's sarcoma: a clinicopathologic study from South Africa. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol. 2003;96(6):701-10.
- 12. Ponnam SR, Srivastava G, Jampani N, Kamath V. A fatal case of rapid gingival enlargement: Case report

- with brief review. J Oral Maxillofacial Pathol. 2014;18(1):121.
- 13. Seymour R, Smith D, Rogers S. The comparative effects of azathioprine and cyclosporin on some gingival health parameters of renal transplant patients: A longitudinal study. J Clin Periodontol. 1987;14(10):610-3.
- 14. Omori K, Hanayama Y, Naruishi K. Gingival overgrowth caused by vitamin C deficiency associated with metabolic syndrome and severe periodontal infection: a case report. Clin Case Rep. 2014;2(6):286-95.
- 15. Kubota Y, Moriyama Y, Yamagishi K. Serum vitamin C concentration and hs-CRP level in middle-aged Japanese men and women. Atherosclerosis. 2010;208(2):496-500.
- 16. Güngör N, Koçak Ü. Tooth Pain and Numb Chin as the Initial Presentation of Systemic Malignancy.
- 17. Patil S, Kalla N, Ramesh D, Kalla A. Leukemic gingival enlargement: a report of two cases. Arch Orofac Sci. 2010;5(2):69-72.
- Shiboski CH, Regezi JA, Sanchez HC, Silverman Jr
 Oral lesions as the first clinical sign of microscopic polyangiitis: a case report. Oral Surg
 Oral Med Oral Pathol Oral Radiol Endodontol. 2002;94(6):707-11.
- 19. Stewart C, Cohen D, Bhattacharyya I. Oral manifestations of Wegener's granulomatosis: a report of three cases and a literature review. J Am Dental Ass. 2007;138(3):338-48.
- 20. Fernandez-Faith E, McDonnell J. Cutaneous sarcoidosis: differential diagnosis. Clin Dermatol. 2007;25(3):276-87.
- LS N. Rose CS, Maier LA. Sarcoidosis N Engl J Med. 1997;3:1224-34.
- 22. Kadiwala SA, Dixit MB. Gingival enlargement unveiling sarcoidosis: Report of a rare case. Contemporary Clin Dentistr. 2013;4(4):551.
- 23. Smith WR, Mason K, Davies D, Onions J. Intraoral and pulmonary tuberculosis following dental treatment. Lancet. 1982;319(8276):842-4.
- 24. Nwoku LA, Kekere-Ekun TA, Sawyer DR, Olude OO. Primary tuberculous osteomyelitis of the mandible. J Maxillofacial Surg. 1983;11:46-8.
- 25. Varadhan Karthikeyan B, Raju Pradeep A, Dileep Sharma C. Primary tuberculous gingival enlargement: a rare entity. J Can Dental Asso. 2006;72(7).
- 26. Woolfe M. Secondary tuberculous ulceration of the tongue. A case report. Bri Dental J. 1968;125(6):270-71.
- 27. Weaver RA. Tuberculosis of the tongue. JAMA. 1976;235(22):2418.

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