World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

Idiopathic gingival enlargement: A brief review and case report

Deepa Anumala *, Kishore kumar Avula, Mohan kumar Pasupuleti, Naga Jyothi Chadalavada, Siva sai Prasad Reddy Dandu and Ravindra reddy Nagireddy

Department of periodontics, Sibar Institute of dental sciences, Takkellapadu, Guntur, India.

World Journal of Advanced Research and Reviews, 2022, 16(03), 789–793

Publication history: Received on 12 November 2022; revised on 21 December 2022; accepted on 24 December 2022

Article DOI: https://doi.org/10.30574/wjarr.2022.16.3.1410

Abstract

Idiopathic gingival enlargement is a heterogenous group of disorders characterized by progressive enlargement of the gingiva caused by an increase in submucosal connective tissue elements. Many cases are iatrogenic and some are inherited or idiopathic. We present a case of idiopathic gingival enlargement to highlight the etiological factors, radiographic and histological features

Keywords: Inflammation; Gingival enlargement; Dental Plaque; Pseudo pockets; Gingivectomy

1. Introduction

Gingival enlargements are quite common and may be either inflammatory, non-inflammatory or a combination of both. Idiopathic gingival enlargement is a rare condition of undetermined etiology described variously as Fibromatosis gingivae [1], Gingivaematosis, Hereditary gingival fibromatosis[2], idiopathic fibromatosis[3], familial elephantiasis[4] and diffuse fibroma[5].

Idiopathic gingival hyperplasia was first reported in 1856 by Goddard and Gross and described as "fungus excrescence of the gingiva ". Wynne and colleagues in 1995 reported a new syndrome of Heriditary Gingival Fibromatosis(HGF)occurring with hearing deficiencies, hypertelorism and supernumerary teeth. Machuda in 1996 described a gene for HGF that has recently been localized to chromosome 2p21-p22 in a large Brazilian family, segregating for an autosomal-dominant form of Heriditary Gingival Fibromatosis. HGF is usually identified as an autosomal dominant condition although recessive forms are described in the literature [6,7]. Gingival fibromatosis may exist as an isolated abnormality or as a part of syndrome. Idiopathic gingival enlargement is also found to be associated with syndromes like Cross syndrome, Rutherford syndrome, Ramen syndrome, Zimmerman Laband syndrome and Juvenile hyaline syndrome.[8]

Most of the patients with idiopathic enlargement presents with generalized diffuse gingival enlargement involving the maxillary and mandibular arches extending on buccal and lingual/palatal surfaces and covering incisal / occlusal third of the tooth resulting in difficulty in speech and mastication. The condition is not painful until the tissue enlarges to partially cover the occlusal surfaces and becomes traumatized during mastication. All these factors will favor accumulation of materia alba and plaque, which further complicates the existing hyperplastic tissue. Acute pain associated with gingival hyperplasia leads to loss of masticatory ability and difficulty in swallowing food. Therefore, these patients have a tendency to swallow partially crushed food which eventually causes gastric disturbances. Thus, idiopathic gingival enlargement is a proliferative fibrous lesion of the gingival tissue that causes esthetic and functional problems. Idiopathic Gingival Fibromatosis (IGF) has no causative agent and a family history is always lacking. A

*Corresponding author: Deepa Anumala
Department of periodontics, Sibar Institute of dental sciences, Takkellapadu, Guntur, India.

Copyright © 2022 Author(s) retain the copyright of this article. This article is published under the terms of the Creative Commons Attribution Liscense 4.0.

genetically[9, 10] and pharmacologically induced[11] form of gingival enlargement has also been reported. Here we present a non-syndromic case of IGF which highlights clinical, radiographic and histophathological features.

2. Case report

A 25 year old male patient reported to the outpatient department of C.k.S Teja institute of dental sciences, Tirupathi, with the chief complaint of swelling in the gums. The patient had no history of seizures, drug intake, fever. There was no family history of a similar illness. Any mental impairment was not noticed on this patient and his weight and height seemed within normal limits. There were no relavent systemic diseases involvement also. The gingival enlargement was extensive, involving almost all maxillary and mandibular teeth. On extraoral examination no abnormalities detected in the TMJ and lymph nodes and the lips were incompetent. On intraoral examination enlarged gingiva is pink, firm and almost leathery in consistency and has a characteristic plebbled surface with abundant stippling.

On clinical examination color of the gingiva appeared normal. The patient presented with generalized diffuse gingival enlargement involving the maxillary and mandibular arches extending on buccal and lingual/palatal surfaces and covering incisal / occlusal third of the tooth resulting in difficulty in speech and mastication. Mild inflammatory signs were present. In addition, halitosis was accentuated. Bleeding and suppuration were noticed in most areas. Dental plaque and calculus were found with moderate levels. Pseudo pockets ranging from 6mm to 8 mm were observed. The patient was severed from poor oral hygiene and did not utilize any kind of tooth paste and/or other types of oral health care because of enlargement. Maintaining oral hygiene was difficult due to the gum overgrowth. Figure 1 shows clinical picture of the gingival enlargement.

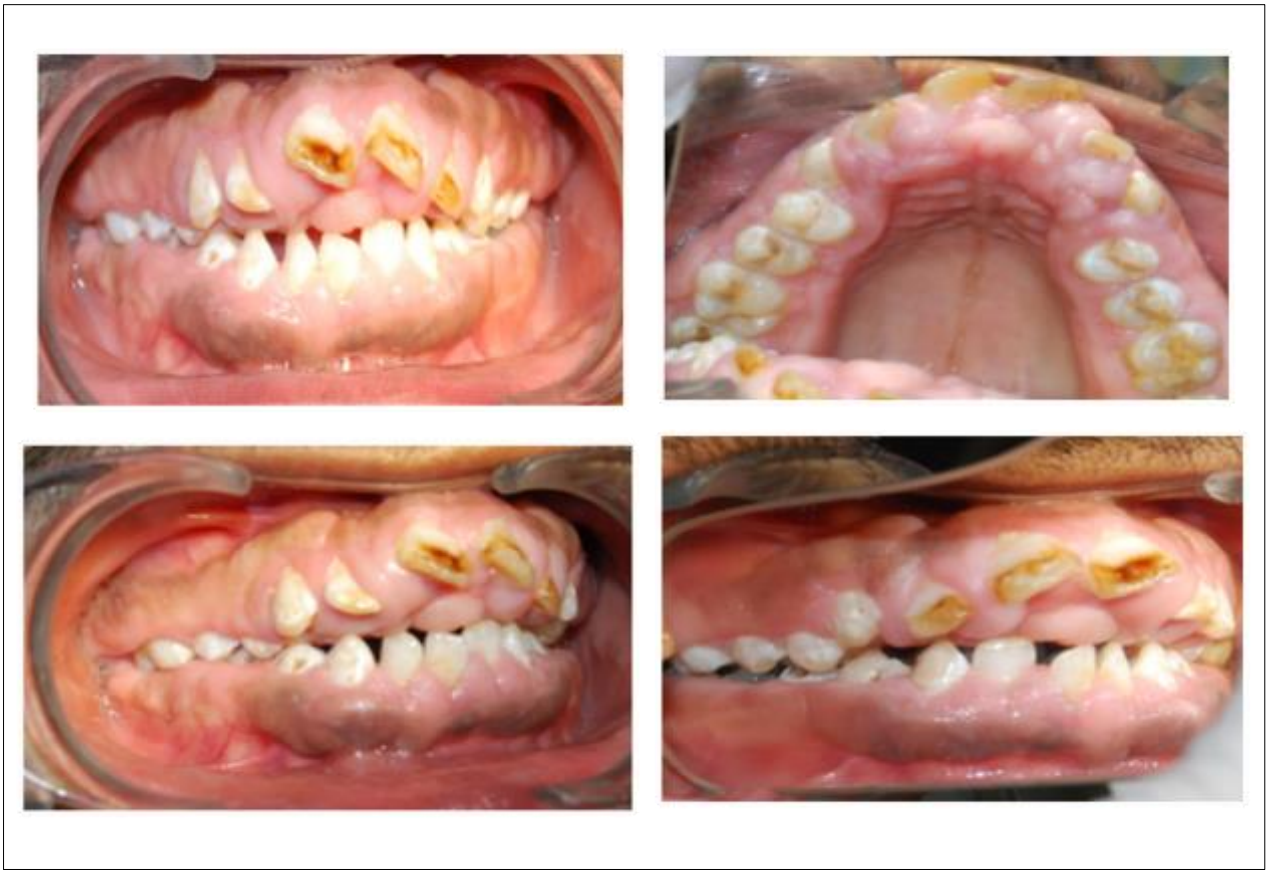

Figure 1 Clinical picture showing Idiopathic Gingival Enlargement

On radiographic examination malalignment of teeth is seen in anterior teeth region of both maxilla and mandible with widening of interdental space. Bone loss is seen in left lower posterior teeth region especially in relation to 36 tooth. Figure 2 shows OPG of root coverage of facial bones and teeth including the TMJ

On histological examination there is para keratinized squamous epithelium with long slender rete ridges. Underlying connective tissue showing dense collagen fibers arranged into bundles intermixed with chronic inflammatory cell

infiltrate chiefly lymphocytes and plasma cells. Figure 3 shows histologic picture of associated gingival tissue. Based on the above clinical, radiographic and histological findings the case is diagnosed as idiopathic gingival enlargement.

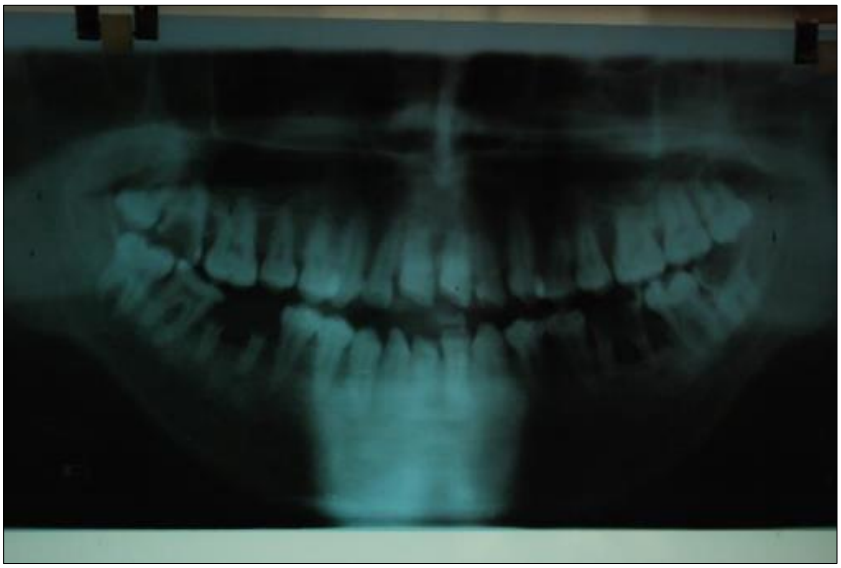

Figure 2 OPG of root coverage of facial bones and teeth including the TMJ

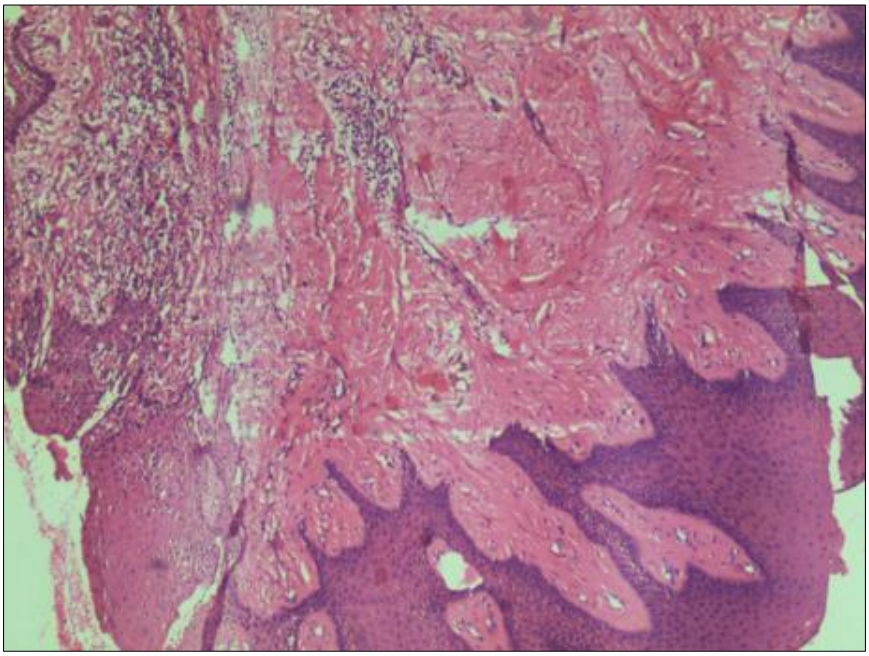

Figure 3 Histologic picture of associated gingival tissue

3. Discussion

Gingival hyperplasia has been classified based on etiological factors and associated pathological changes as inflammatory, drug-induced hyperplasia, associated with systemic diseases, neoplastic, and either idiopathic or hereditary gingival fibromatosis. Drug induced gingival enlargement (DIGE) can occur after treatment with drugs like Phenytoin and cyclosporine etc [11]. Long term use of these drugs has to be ruled out. Gingival hyperplasia may be associated with physical development, retardation, and hypertrichosis[12]. Gingival enlargement may be due to nutritional and hormonal factors but they have not been completely substantiated. The tissue mass which increases constantly during eruption of permanent teeth can result in delayed eruption and displacement of teeth [13]. Idiopathic

gingival enlargement may be congenital or hereditary. Though the genetic mechanism is not well understood, majority of the reported cases have attributed the condition of fibrous enlargement of gingiva to hereditary factors[14].

Idiopathic gingival hyperplasia is the rarest variety among all forms, with unknown etiology. It is clinically classified as the symmetric form and the nodular form. The most common is the symmetric form characterized by uniform enlargement of the gingiva, whereas the nodular form shows the multiple enlargements in the gingiva. In the present case, the gingival hyperplasia did not represent any relation to hereditary, syndromes, drugs, conditions, or endocrine problems; hence a diagnosis of idiopathic gingival hyperplasia was made[15].

Idiopathic gingival fibromatosis usually presents with no identified causative agent. This condition sometimes develops with the eruption of deciduous dentition and rarely present at birth. The hyperplastic gingiva in idiopathic gingival fibromatosis usually presents a normal color and has a firm consistency with abundant stippling. The idiopathic gingival enlargement may occur alone or as part of a syndromes. Due to massive gingival enlargement, an affected subjects usually develop an abnormal swallowing pattern and experiences difficulty with speech and mastication. Along with these features, there may be some interference with maintenance of oral hygiene and mastication. All these factors will favor accumulation of plaque, which further complicates the existing hyperplastic tissue. More severe lesions may cover the dental crowns, resulting in both aesthetic and functional problems. Therefore, these patients have a tendency to swallow partially crushed food which eventually causes gastric disturbances[16, 17].

The present case of idiopathic gingival overgrowth may be a typical condition, characterized by progressive, large masses of firm, dense, resilient insensitive growth that covers the alveolar ridges and extends over the teeth. The condition is not painful until the tissue enlarges to partially cover the occlusal surface of the molars and become traumatized during mastication, which was observed in the present case. Attributable to massive gingival enlargement, the affected patient usually develops abnormal swallowing pattern and experiences difficulty in speech and mastication. Along with these features, there may be some interference with the oral hygiene measures and normal mastication. All these will favour accumulation of material alba and plaque, which further complicates the existing hyperplastic tissue. Grade III score was given for the enlargement that covered three quarters or more of the crown (Bokencamp 1994)[18].

Gagliano et al.,suggested that gingival hyperplasia of different etiologies may have different mechanisms of overgrowth. These include an increase in proliferation of resident tissue fibroblasts, a reduced level of metalloproteinase synthesis (matrix metalloproteinases (MMP)-1 and MMP-2), resulting in low levels of extracellular matrix degrading, an increase in collagen type I production, heat-shock protein 47 (hsp47) production, and other extracellular matrix components.[19, 20]. In the present case histologically, epithelium appears hyperplastic with elongated rete pegs. There is a marked increase in the amount of connective tissue and shows bundles of collagen fibers running in all directions with numerous fibroblasts. These features are as reported by previous studies [21]. The etiology and pathogenesis of gingival hyperplasia are still not well established; however it could be directly linked to three factors: individual susceptibility, local factors (dental plaque, caries, and iatrogenic factors) and the action of chemical substances and their metabolites.

3.1. Treatment

Treatment methods for idiopathic gingival enlargement are dependent on the severity of the lesion. When the enlargement is minimal and possible to maintained by the patient, scaling and root planning along with proper maintenance of oral hygiene may be sufficient but when enlargement is massive, surgical intervention such as external bevel gingivectomy, electrocautery, and lasers can be used with professional cleaning and home care maintenance [22,23].

4. Conclusion

This was a case of idiopathic gingival enlargement not associated with any syndrome. The patient was properly diagnosed based on clinical examination and proper correlation with radiological and histopathological examination. Even though recurrence cannot be predicted, the psychological and functional benefits far outweigh the risk of recurrence. Oral hygiene and the superimposition of plaque accumulation have a crucial effect on the prognosis of gingival enlargement. Long-term follow-up will be required to evaluate the predictability of the different surgical techniques.

Compliance with ethical standards

Acknowledgments

We thank J. Divakar for his support on this work.

Disclosure of conflict of interest

No conflict of interest.

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors'.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Ball EI. Case of gingivamatosis or elephantiasis of the gingiva. J Periodontol 1941, 12: 26.

[2] Laskin J, Weisberger D. Hereditary gingival fibromatosis. JOral Surg 1961, 14: 828.

[3] Thukral PP. Idiopathic hyperplasia. J Ind Dent Assoc 1972; 44:109.

[4] Raman Y, Berman, Bubbis JJ. Gingival fibromatosis combined with cherubism. Oral Surg 1967, 24:455.

[5] Buckner HJ. Diffuse fibroma of the gums. J Am Dent Assoc 1937, 27:2003.

[6] Emerson TG. Hereditary gingival hyperplasia. A family pedigree of four generations. Oral Surg 1965, 19:1.

[7] Jorgenson RJ, Cocker ME. Variations in the inheritance and expression of the gingival fibromatosis. J Periodontol 1974, 45:472-477.

[8] Aldred MJ, Bartold PM. Genetic disorders of the gingivae and periodontium. Periodontol 2000; 18: 7–20.

[9] Salinas CF. Orodental findings and genetic disorders, Birth Defects. 1982; 18: 79–120.

[10] Shapiro SD, Jorgenson RJ. Heterogeneity in genetic disorders that affect the orifices. Birth Defects. 1983; 19(1):155–166.

[11] Angelopoulos AP, Goaz PW. Incidence of diphenylhydantoin gingival hyperplasia. Oral Surg Oral Med Oral Pathol. 1972, 34:898-906.

[12] Shafer WG, Hine MK, Levy BM. 4. Developmental disturbances of the perioral structures. 4th ed. (Philadelphia) A Prisma Indian; 1993. pp. 23-24.

[13] Mcdonald RE, Avery DR. Gingival and periodontal diseases. Dentistry for the Child and Adolescent. 7th ed. USA: Mosby Company; 2000.

[14] Cortelli JR. Evidence of genetic heterogenecity for hereditary gingival fibromatosis. J Dent Res. 2000, 79:1758-64.

[15] S. Bhatnagar, S. Galgali, G. Gontiya, U. Mohandas, Laser-assisted gingivectomy in pediatric patients: a novel alternative treatment, J. Indian Soc. Pedodont. Prev. Dent. 29 (3) (2011) 264

[16] Salinas CF. Orodental findings and genetic disorders. Birth Defects. Orid Artic Ser 1982; 18: 79-120.

[17] Fatema S, ModaPreeti M. Idiopathic gingival fibromatosis associated with generalized aggressive periodontitis combined with plasma cell gingivitis: A

[18] Michael G. Newman, Henry H. Takei, Perry R. Klokkevold, Fermin A. Carranza. Clinical Periodontology 10 ed, saunders (an imprint of elsevier).

[19] Gagliano N, Moscheni C, Dellavia C, Masiero S, Torri C, Grizzi F, et al. Morphological and molecular analysis of idiopathic gingival fibromatosis: A case report. J Clin Periodontol 2005, 32:1116-21.

[20] Coletta RD, Graner E. Hereditary gingival fibromatosis: A systematic review. J Periodontol 2006, 77:753-64.

[21] Yadav VS, Chakraborty S, Tewari S, Sharma RK. An unusual case of idiopathic gingival fibromatosis. Contemp Clin Dent 2013; 4: 102-104.

[22] V. Pant, S. Pandey, A rare case report of grade III gingival enlargement associated with chronic periodontitis: comparison of two treatment techniques, Natl. J. Maxillofac. Surg. 6 (1) (2015) 119.

[23] Idiopathic Gingival Hyperplasia: A Case Report Merve Sirin, Meral Unur Department of Maxillofacial Surgery, Istanbul University, Turkey, 2020.