DOI: 10.7860/JCDR/2016/17311.7786

Management of Idiopathic Gingival Enlargement

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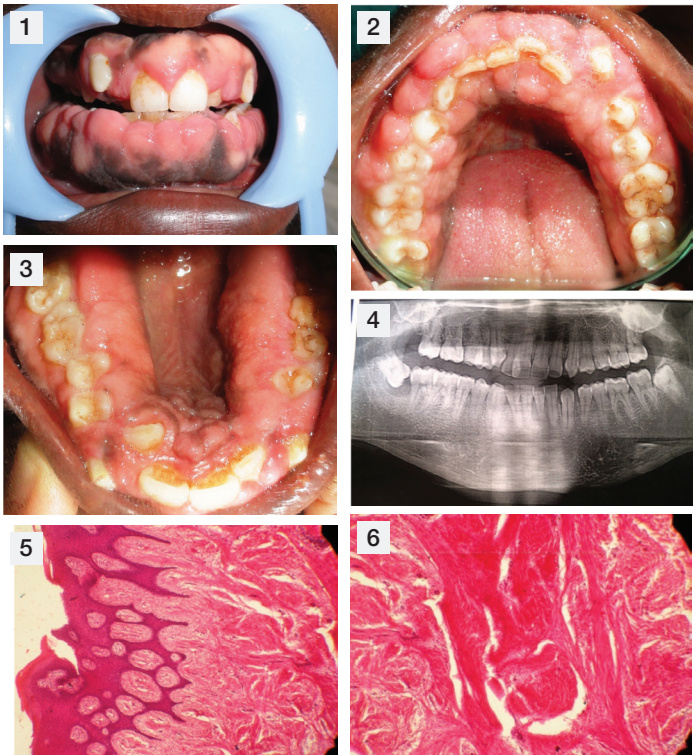
Keywords: Gingival hyperplasia, Gingival fibromatosis, Gingivectomy

A 25-year-old male reported to the department with a chief complaint of swollen gums since 10 years involving all the teeth. The swelling was aesthetically unpleasing and also causing difficulty in speech and mastication. Patient did not undergo any kind of dental treatment for the above problem. Patients dental, personal, medical, family histories were non-contributory. Patient was not under any anti-epileptic, anti-hypertensive, immunosuppressive medication and there was no history of fever, anorexia, weight loss, seizures, hearing loss or any physical or mental disorder.

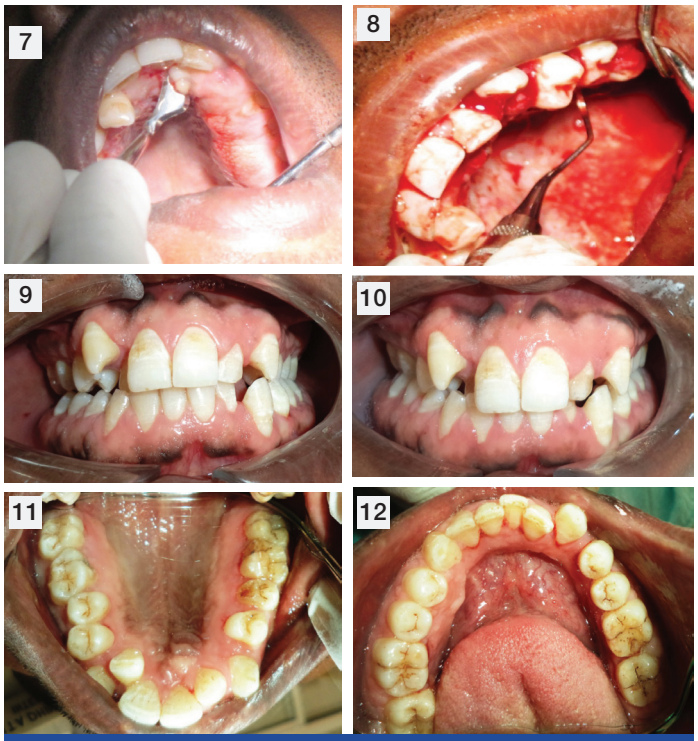
On extraoral examination the patient has incompetent lips with convex facial profile. Intraoral examination revealed generalized diffused nodular enlargement of gingiva on both buccal and lingual/palatal sides [Table/Fig-1-3]. Gingival enlargement score of grade III (Bokencamp 1994) [1] was given, that is enlargement covers three quarters or more of the crown. Gingiva was pink in colour with superimposed melanin pigmentation, firm and fibrous fibrous in consistency. The teeth were barely visible as enlargement extended up to incisal/occlusal third of all the teeth except 11, 21, 13 and 23. Stippling was absent and slight bleeding on probing was present with altered gingival contour. Gingival enlargement was not superimposed by any secondary inflammatory changes.



[Table/Fig-1]: Labial view. **[Table/Fig-2]** Mandibular arch. **[Table/Fig-3]:** Maxillary arch. **[Table/Fig-4]:** Panoramic view. **[Table/Fig-5]:** Histological picture hyperparakeratinized stratified squamous epithelium showing pseudoepitheliomatous hyperplasia. Haematoxylin and Eosin stain (Original maginification x 20). **[Table/Fig-6]:** Histological picture showing extensive collagen consisting of coarse collagen fibres. Haematoxylin and Eosin stain (Original maginification x 20).

Panoramic radiograph revealed no bone loss [Table/Fig-4]. Oclusal radiographs were not taken. Haematological investigation was done which was within normal limits. Histopathological examination revealed hyperparakeratinized stratified squamous epithelium showing pseudoepitheliomatous hyperplasia with thin elongated interconnecting retepegs [Table/Fig-5]. The underlying connective tissue showed extensive collagen consisting of coarse collagen fibres arranged in irregular fascicles [Table/Fig-6]. The fibroblasts were few with compressed blood vessels and scanty chronic inflammatory cell infiltrate. These findings were suggestive of fibrotic gingival enlargement.

On the basis of detailed case history and histological findings, the case was diagnosed as generalized idiopathic gingival enlargement. A treatment plan was devised for the patient Which included phase I therapy, followed by maintenance phase and phase II therapy. Adjunctive antimicrobial administration of systemic amoxicillin 500mg thrice daily and metronidazole 400mg twice daily for seven days was prescribed along with use of chlorhexidine mouthwash twice daily. The surgical phase included quadrant wise full mouth gingivectomy. Kirkl and knife was used for incisions on facial and lingual surfaces and Orban knife for interdental incisions [Table/Fig-7]. All granulation tissue was completely curetted and calculus remnants if any were scaled to make the areas clean [Table/Fig-8].



[Table/Fig-7]: Gingivectomy done with Kirkland knife. **[Table/Fig-8]:** Removal of calculus remnants with curette. **[Table/Fig-9]:** Postoperative view of after 1 month. **[Table/Fig-10]:** Postoperative view of after 2 months. **[Table/Fig-11]:** Postoperative maxillary occlusal view after 2 months. **[Table/Fig-12]:** Postoperative mandibular occlusal view after 2 months.

Patient was recalled after one month [Table/Fig-9] and two months [Table/Fig-10-12]. Healing was satisfactory.

DISCUSSION

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 [2].

Gingival hyperplasia can occur after treatment with drugs like Phenytoin and cyclosporine etc. [3]. Long term use of these drugs has to be ruled out. Gingival hyperplasia may be associated with physical development, retardation, and hypertrichosis [4]. Gingival enlargement may be due to nutritional and hormonal factors but they have not been completely substantiated. The tissue mass which increases constantly can result in delayed eruption and displacement of teeth [5].

When such massive gingival enlargement is responsible for aesthetic concern, interferes with normal mastication and maintenance of proper oral hygiene, a surgical intervention must be considered. In such cases, the most efficacious method is the conventional gingivectomy. Maintaining good oral hygiene is important as the presence of inflammation and infection can be associated with a risk of recurrence of the gingival enlargement. However, gingival fibromatosis recurrence is not only due to the presence of local factors, but also genetic predisposition. Therefore, it is not possible to predict the long-term results of gingival fibromatosis treatment even when associated with ideal oral hygiene.

Histologically increased thickening of mature collagen bundles in connective tissue stroma causes gingival hyperplasia. The thickened parakeratinized epithelium may cause nodular appearance.

CONCLUSION

In most of the cases recurrence could be expected within a few months after surgery and may return to the original condition within few years. So the patient may have to undergo repeated gingivectomy surgical procedures. In this case recurrence has occurred after three months even though the local factors were minimal. But the patient did not return back for further treatment due to some personal reasons. The psychological and functional benefits following surgical intervention should be kept in mind when compared to risk of recurrence.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Oct 13, 2015**
Date of Peer Review: **Dec 18, 2015**
Date of Acceptance: **Jan 14, 2016**
Date of Publishing: **May 01, 2016**