Idiopathic gingival fibromatosis is a rare hereditary condition that has no definite cause. The pathogenesis of gingival fibromatosis is still unknown but an increase in proliferation of the gingival fibroblasts as well as increased collagen synthesis may be involved. In modern times, a mutation in the son of sevenless-1 (SOS-1) gene has been suggested as a possible cause of isolated (non-syndromic) gingival fibromatosis, but no definite linkage has been established. A twenty four year old patient presented with gingival enlargement since one year. Based on clinical and radiographic examination, laboratory tests, and thorough oral hygiene instructions, an external bevel gingivectomy was performed to remove excess gingival tissue. There was recurrence within two months following the surgery. The exact cause of the gingival enlargement was not identified, although amelioration of the fibrotic, enlarged gingiva was observed which recurred within two month after performing conventional gingivectomy. Recurrence of gingival fibromatosis in such patients can also be attributed to genetic predisposition.

Keywords: Gingiva, Fibromatosis, Dominant genetic conditions.

INTRODUCTION

Gingival fibromatosis may exist as isolated abnormality or as a part of a syndrome. Several types of gingival enlargement that differ from common lesions have been classified according to etiologic factors and histopathological findings. Drug-induced gingival enlargement, such as that produced by phenytoin, cyclosporine and calcium channel blockers as well as conditioned gingival enlargement associated with systemic disorders produced by hormonal factors, leukemia, vitamin C deficiency and idiopathic gingival fibromatosis have been reported. Investigations are in evolution to establish the genetic linkage and heterogeneity associated with it. This condition may manifest as an autosomal dominant or less commonly with an autosomal recessive mode of inheritance. Autosomal-dominant forms of gingival fibromatosis, which are usually non-syndromic, have been genetically linked to the chromosome 2p21-2p22 and 5q13-q22. In modern times, a mutation in the son of sevenless-1 (SOS-1) gene has been suggested as a possible cause of isolated (non-syndromic) gingival fibromatosis, but no definite linkage has been established. Idiopathic gingival fibromatosis is a gradually progressing benign enlargement that affects the marginal gingiva, attached gingival, and interdental papilla. The fibromatosis may potentially cover the exposed tooth surfaces, thereby hampering the functioning of the stomatognathic system. The gingival tissues are usually pink and non-haemorrhagic and have a firm and fibrotic consistency. Histopathologically, the bulbous increased connective tissue is relatively avascular, and has densely arranged collagen-fibre bundles and numerous fibroblasts. The present case is a...
A twenty four-year-old male reported to the institution with a chief complaint of massive swelling in the gums for the past one year. On intraoral examination, the hyperplastic gingiva covered the teeth labially. Especially at the lower anterior, hyperplasia covered the entire crown of the teeth. The enlarged gingiva was firm and fibrotic in consistency (Figure 1). The patient's oral hygiene was satisfactory, yet minimal debris was noted around involved area. Laboratory tests including complete blood investigation, urine analysis and hormonal profile was normal. The radiograph showed good bone support with minimal crestal bone loss (Figure 2). His chest x-ray was also normal.

**CASE REPORT**

A twenty four-year-old male reported to the institution with a chief complaint of massive swelling in the gums for the past one year. On intraoral examination, the hyperplastic gingiva covered the teeth labially. Especially at the lower anterior, hyperplasia covered the entire crown of the teeth. The enlarged gingiva was firm and fibrotic in consistency (Figure 1). The patient's oral hygiene was satisfactory, yet minimal debris was noted around involved area. Laboratory tests including complete blood investigation, urine analysis and hormonal profile was normal. The radiograph showed good bone support with minimal crestal bone loss (Figure 2). His chest x-ray was also normal.

**Treatment**

Informed consent of the patient was taken prior to the treatment after being approved by Institutional Ethical Committee. Treatment planning was done which included non-surgical periodontal
therapy followed by gingivectomy. The surgical intervention was carried out under local anaesthesia using external bevel incision to remove large increment of hyperplastic tissue (Figure 3a & 3b). The patient was prescribed an antibiotic for 24 hours before the surgery and five days postoperatively (Amoxicillin 500 mg, three times a day) to prevent postoperative bacteremia and an analgesic for three days postoperatively (Acelofenac 500 mg, twice a day) to relieve postoperative pain. Periodontal dressing (Coe-Pak) was placed. The patient was prescribed 0.2% chlorhexidine rinse twice a day post surgically. Patient was recalled after one week for check-up.

Histopathological investigation

The excised gingival mass showed mild infiltration of inflammatory cells, densely packed collagen bundles, few blood vessels and few lymphocytes. The overlying epithelium exhibited some hyperplasia (Figure 4).

With clinical and histopathological examination, the case was diagnosed as idiopathic gingival fibromatosis.

The patient reported after two months with recurrence of gingival enlargement (Figure 5).

DISCUSSION

Patient demonstrated extensive gingival enlargement with the clinical features of the enlarged gingiva being firm and fibrotic. The extensive gingival enlargement suggested that the patient may have been suffering from a syndrome associated with gingival fibromatosis, i.e., Murray-Puretic-Drescher syndrome, Rutherford’s syndrome, Leband syndrome, or Cross syndrome16. Clinically, the enlarged gingiva in hereditary or idiopathic gingival fibromatosis is firm in consistency. Patient was normal, without any symptoms of mental retardation. He did not suffer from epilepsy or hypertrichosis, nor did he have any tumors or corneal dystrophy. No skeletal deformities or defects of the skin or fingernails were observed. Thus, it was unlikely that the above mentioned syndromes were related to this gingival enlargement. Gingival enlargement also is a symptom of scurvy5 but the patient did not have any other signs of scurvy such as petechiae, ecchymoses, or spontaneous bruising of the extremities. The laboratory tests revealed no evidence of any systemic disorders such as leukemia, diabetes mellitus, or hormonal disorders, including disorders of the sex hormones. Considering the medical, prenatal and drug histories the diagnosis of idiopathic gingival fibromatosis was given. The precise mechanism of idiopathic gingival fibromatosis is unknown but it appears to confine to the fibroblasts which harbor in the gingivae. The hyperplastic response does not involve the periodontal ligament and occurs peripheral to the alveolar bone within attached gingiva17. The pathogenesis of gingival fibromatosis is still unknown but an increase in proliferation of the gingival fibroblasts as well as increased collagen synthesis may be involved. Idiopathic gingival fibromatosis is a rare hereditary condition that has no definite cause. Further investigations are necessary to establish the genetic linkage and heterogeneity associated with it18. Therefore a detailed medical history and physical examination should be carried out before a diagnosis of generalized gingival enlargement is given. The various types of acquired or hereditary generalized gingival enlargement should be considered in the differential diagnosis.

A surgical treatment is considered when the gingival enlargement is responsible for impairment of esthetics and interferes with function. The finest and suggested treatment modality for idiopathic gingival fibromatosis is gingivectomy. Surgical treatment improves the esthetics and enhances the function.

The patient should maintain good oral hygiene for stabilization of the effective treatment as the presence of inflammation and infection can be associated with the risk of recurrence of gingival enlargement. However, recurrence of gingival fibromatosis in such patients can also be attributed to genetic predisposition. Therefore, it is not possible to predict the long term results of gingival fibromatosis treatment even when associated with good oral hygiene.

CONCLUSION

Management of a patient with gingival enlargement should include a complete medical history and physical examination to rule out known causative agents and factors. A biopsy should be
performed to confirm the diagnosis and to rule out a neoplasm. Surgical management may require gingivectomy or total extraction of teeth, if required. In the case presented, external bevel gingivectomy was performed. There was recurrence of gingival enlargement seen in this patient at the within two month. It is very important to correlate the extraoral and intraoral clinical findings, family and medical history, medications, and results of histopathologic and laboratory tests to rule out known causative agents/factors and different pathologic conditions that may manifest themselves similar to the conditions seen in this case. Additionally, regularly scheduled maintenance visits and maintenance therapy will be necessary to prevent additional destruction of the periodontal tissues. Thus to conclude, etiological factors should be identified in cases of gingival fibromatosis and proper surgical intervention results in improving enhanced function and esthetic appearance.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES


