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Case Report

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A Rare Association of Idiopathic Gingival Enlargement with Plasma Cell Gingivitis and Generalized Chronic Periodontitis

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Abstract

Gingival fibromatosis is a clinical condition that is characterized by gingival overgrowth. It is often caused due to medication, hereditary reasons and other local factors. When the etiology is unknown, they are referred as Idiopathic gingival enlargement (IGE). IGE is a rare and is often occurs as a manifestation of an underlying syndrome or as a separate entity. They clinically appear as gingival overgrowth with firm consistency with both deciduous and permanent dentition equally affected and worsens during adolescence. IGE is a slowly growing benign growth affecting all anatomic parts of the gingiva leading to esthetic and functional problems with difficulty in speech, mastication and deglutition. This leads to plaque accumulation which complicates the previous gingival enlargements. There are many cases reporting the idiopathic gingival enlargement in the literature, but here we report a rare case of non-syndromic association of IGE, chronic periodontitis and plasma cell gingivitis in an older patient.

Keywords: Periodontitis, Gingival enlargement, Plasma cell gingivitis, Systemic syndrome, Dental.

INTRODUCTION

Gingival fibromatosis belongs to a group of disorders which are characterized by progressive enlargement of gingival apparatus. It can be localized or generalized. Generalized gingival enlargement with unknown etiology is termed as Idiopathic Gingival Enlargement [IGE]. IGE is a rare occurrence and either is associated with a syndrome or occurs as a separate entity [1]. Clinically they manifest as gingival over growth with firm consistency, with both deciduous and permanent dentition equally affected and worsens during adolescence. They are a slowly progressing benign growth affecting all of the gingival apparatus leading to esthetic and functional problems such as abnormal swallowing patterns and difficulty with speech. This results in the cumulative accumulation of materia alba and plaque, which further complicates the existing hyperplastic condition of the tissues [2].

Periodontitis is defined as the inflammatory disease of supporting tissues of teeth caused by a groups of specific microorganisms, that leads to progressive destruction of the periodontal ligament, cementum and alveolar bone with periodontal pocket formation, gingival recession or both and ultimately tooth loss [3,4]. Periodontal disease is a complex cross-talk between bacterial infection, host response, environmental factors, acquired risk factors and genetic susceptibility. It maybe localized involving one tooth or generalized chronic periodontitis (GCP), if it involves the whole dentition.

Plasma cell gingivitis (PCG) is a rare benign inflammatory condition of unknown aetiology. It is characterized clinically by a diffuse, erythaematous, papillary lesion and occasional desquamation of the gingiva [5]. Histopathologically, it is characterized by diffuse, massive infiltration of plasma cells into the connective tissue of the gingiva. Henceforth, the name plasma cell gingivitis. The etiology PCG is not clear understood yet. Some authors divide PCG into 1. Allergic 2. Neoplastic 3. Idiopathic.

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Biopsy remains the gold standard for the diagnosis of PCG as it may be mimicked by other serious systemic conditions like leukemia, plasmacytoma.

There are many cases reporting the idiopathic gingival fibromatosis and chronic periodontitis in the literature. But we report a rare case of non-syndromic association of IGE, Chronic periodontitis with PCG and in an older patient.

CASE REPORT

A 55-year-old female patient reported to the outpatient unit with a chief complaint of swelling and pain in the gums since 4 years. She had noticed the swelling 4 years back which got progressively increased to reach the present size. Pain had started a year later which was dull, intermittent and aggravates during mastication. Bleeding gums during brushing was reported. Medical history reveled that she is a known diabetic and hypertensive but is not under any medication. She also gives a history of oral prophylaxis 1 year back due to same problem which did not yield any significant relief. General examination reported her BP as 130/90 mm Hg.

Extra oral examination revealed incompetent lips and convex profile with chelitis on both sides. Intra oral examination shows massive generalized, diffuse, nodular enlargement of gingiva involving both arches. It was firm and lathery in consistency. Maxillary gingiva was pale in color while mandibular showed hyper pigmentation. Gingiva showed loss of stippling in all areas except 15. There was a generalized mild bleeding on probing and pus exudation. Gingiva was tender on palpation. Periodontal examination revealed pseudo pockets of varying depths from 7 to 10 mm. Gingival recession was seen on 11,13,21,23, 31, 41, 42 with teeth showing various degrees of mobility Grade I in 13, 23, 25, 33, 43, 44, Grade II in 15, 12, 11, 21, 22, 34, 32, 31, 41, 42 and Grade III in 17, 37, 35, 45, 47 and missing teeth in relation to 18, 17,16,14,24,26,28,38,36,46,48. Oral hygiene appeared to be fair. [Figure 1] [Figure 2] Orthopantamogram (OPG) showed multiple missing teeth in both arches with scanty plaques und calculus deposits. Generalized bone loss of both the arches with angular defects caused due to possible tooth drifting. Considering the clinical and radiographic findings, she was provisionally diagnosed with idiopathic gingival enlargement with generalized chronic periodontitis. [Figure 3] Blood examinations were insignificant with mild lymphocytosis. An Incisional biopsy was made from 25, 27 region and the tissue was sent for histopathological examination.

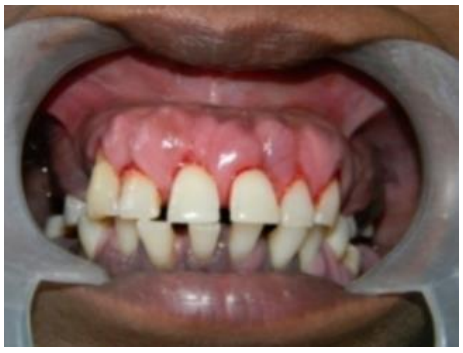

Figure 1: Clinical photograph showing Gingival enlargement with Bleeding gums

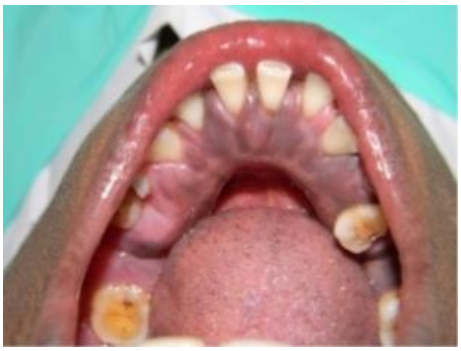

Figure 2: Occlusal view of mandibular arch showing Gingival enlargement

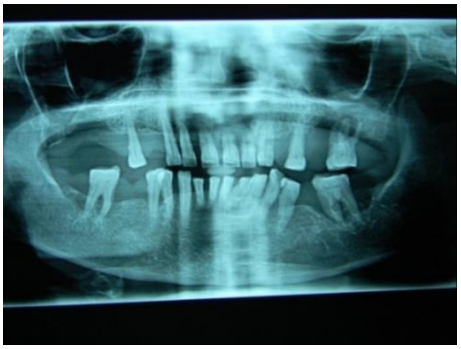

Figure 3: OPG showing generalized horizontal bone loss with missing teeth and angular bone defect created due to tooth drift

The H&E section shows a mild hyperplastic parakeratinized stratified squamous epithelium overlying a highly inflamed connective tissue stroma. The overlying epithelium shows flattening of rete pegs only in some areas. The connective tissue stroma is mild to moderately collagenous. Dense diffuse infiltration of mixed inflammatory cells, composed of plasma cells predominantly and few lymphocytes in the connective tissue. Vascularity is high suggestive of plasma cell gingivitis. Her treatment involved Oral prophylaxis, sub gingival scaling and root planing followed by gingivectomy and prosthetic rehabilitation. The patient is constant follow up and the enlargement has since not returned.

DISCUSSION

Idiopathic gingival fibromatosis is a slowly progressive, benign enlargement of the marginal gingiva, attached gingiva and the interdental papilla. The gingival overgrowth may expand to a size that it covers the exposed tooth surfaces causing esthetic and functional problems. [6]. The gingival tissues appear pink and non-hemorrhagic with a fibrotic consistency. IGF manifests due to congenital or hereditary causes which is not clearly understood. Some authors have proposed the mode of transmission as autosomal dominant or recessive, suggesting abnormal chromosome on phenotype 2p21. IGE are often associated with syndromes like Rutherford's syndrome, Laband syndrome, Jones' syndrome, Cross syndrome, Murray – Puretic Drescher syndrome, Cornelia de Lange Syndrome, Ramon's syndrome, hypothyroidism, chondrodystrophia and diffuse osteofibromatosis [7,8]. In our case, the patient doesn't exhibit any features of these syndromes. Blood tests and metabolic panels did not reveal any abnormality suggestive of any hormonal imbalance. Since the familial, medical, prenatal, and drug histories were non-contributory, it was termed as idiopathic gingival fibromatosis (IGF).

Chronic inflammatory periodontitis is associated with a number of systemic diseases and is a risk factor for cardiovascular diseases, cerebrovascular diseases, peripheral arterial disease, respiratory diseases, low birth weight and is a constant source of infection [9]. The pathogenesis of chronic periodontitis is multifactorial in nature. It

results from interaction between bacterial, environmental, immunologic and genetic factors [10,11]. The etiopathogenesis of periodontitis has been linked to a complex cascade of interactions of the bacterial colonies, environmental risk factors, host response and genetic susceptibility. Bacterial colonization is the primary etiological agent in periodontal disease, and it is estimated that there are more than 500 different bacterial species colonizing the human adult oral cavity [12]. Some of the high-risk bacteria associated with periodontal infection include *Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus, Campylobacter rectus, Actinobacillus actinomycetemcomitans* and *treponemes*.

They are often seen in the periodontal pockets which harbors a mixture of anaerobic bacteria [13]. A number of exotoxins and cytokines are released by the pathogenic bacteria which elitcits a B-cell mediated immune response from the hosts. This is characterized histologically by plasma cells and lymphocytes.

PCG is generally regarded as a hypersensitive reaction to an allergen present in chewing gum, flavouring dentifrice, spices or herbal products. Almost two decades ago, Gargiulo et al. classified PCG into three types: type 1 caused by an allergen, type 2 of neoplastic origin and type 3 as idiopathic [14]. It is a benign condition usually not associated with tissue destruction. Clinically, it manifests as bright red, oedematous gingival swelling usually localized and sharply demarcated from the mucogingival junction. But our case presented as typical fibrous gingival enlargement where the diagnosis of PCG was made by histopathological examination. Previously, different terminologies were used for PCG such as atypical gingivitis, plasma cell gingivostomatitis, plasmacytosis of the gingiva and allergic gingivitis.

The patient presented to us long time after the symptoms had started. Hence the exact association of PCG and GCP is still unclear. It's proven that the long-term periodontal destruction leads to increase plasma cell in the histopathological sections. As advanced periodontitis is marked with increased plasma cell population, their infiltration into the gingival connective tissue can be expected. This would result in oedematous swelling of the gingiva. Therefore, in the present case there is a possibility that GCP could have started earlier and its progression led to plasma cell infiltration into the sub-gingival stroma causing generalised gingival enlargement. But further studies are needed to prove this.

CONCLUSION

This case re-emphasizes the importance of clinical, medical history which gives pointers for diagnosing the varying spectrum of clinical conditions involving the gingiva. PCG is the oedematous swelling of the gingiva generally due to an allergic reaction. The presentation of PCG may be localised or generalised depending on the genetic and immune susceptibility of the individual in association with environmental factors. Further, assessment is needed to underpin the mechanism explaining concurrence of PCG with Idiopathic gingival enlargement and Generalized chronic periodontitis.

Conflicts of Interest

The authors declare no conflict of interest.

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