Diagnostic and treatment dilemma of gingival enlargement: a report of 3 cases

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Abstract

Gingival enlargement is a common feature of periodontal disease which is occasionally associated with medication use for systemic conditions such as hypertension and epilepsy. However, other local and systemic factors have been implicated in the aetiopathogenesis of this condition which could pose diagnostic and therapeutic challenge for clinicians. This paper presents the management of three cases of gingival enlargement recently seen at the Dental Centre of the Lagos University Teaching Hospital, Lagos. The association of unusual secondary aetiological factors is emphasized.

Key words: Gingival Enlargement, Diagnosis, Treatment.

Introduction

Gingival enlargement, the currently accepted terminology can be defined as an increase in the size of the gingiva[1] or it can be defined as an overgrowth of gingival tissue[2]. This is strictly a clinical description of the condition and avoids erroneous pathologic terms used in the past such as gingival hyperplasia or hypertrophy[2]. They usually present as submucosal enlargement covered by normal epithelium. The lesions can be localized or generalized[2]. According to the American Academy of Periodontology 1999 Classification of Periodontal Diseases and Conditions, gingival enlargement can occur because of pregnancy-associated hormonal changes, various medications causing changes in extracellular matrix physiology (such as anticonvulsants, immunosuppressants and antihypertensive drugs) or genetic disorders[3,4]. Medications have been reported as the most common cause of generalized gingival enlargement[5]. Localized gingival enlargement on the other hand may be further classified as "Conditioned Enlargements" and Neoplastic Enlargements[6]. Conditioned gingival enlargement occurs when the patient's systemic condition aggravates the gingival response to bacterial plaque, which is usually necessary for its initiation. The systemic conditions may be specific, hormonal (pregnancy, puberty), nutritional (vitamin C deficiency) and allergic or Non-specific (e.g. Pyogenic granuloma). Neoplastic enlargements account for a comparatively smaller proportion of gingival enlargements and may be referred to as Epulide[6]. Although, a generic term used clinically to designate all discrete tumours and tumourlike masses of the gingiva, most lesions referred to as "epulides" are inflammatory rather than neoplastic[6]. Examples of Neoplastic gingival enlargements are fibroma, peripheral giant cell granuloma and papilloma. Idiopathic gingival enlargement is a rare condition of unknown cause, hence the designation "idiopathic". Some cases have a hereditary basis, though the genetic mechanisms are not well understood[6]. In general, gingival enlargements may impede adequate plaque control therapy causing chronic irritation from accumulated plaque and calculus with possible progression of the enlargement[7]. It also regularly traps plaque or food, producing halitosis or suppuration[8]. Generally, poor plaque control or plaque retentive sites are more likely to be associated with gingival enlargement [7,8]
Some gingival enlargements, whether generalized or localized, may pose diagnostic and therapeutic challenges. This paper reports three recent cases of gingival enlargement treated at the Periodontology clinic of the Lagos University Teaching Hospital, Lagos Nigeria.

Case Reports

Case 1 A 20 year old female student complained of a localized painless swelling in the right cuspid region of the mandible. She reported that the lesion had been excised about six months previously but recurred. No biopsy of this previously excised tissue was conducted. The current intra-oral examination revealed a, firm, round, lobulated mass 5 mm in diameter, on the buccal gingiva between the mandibular right lateral incisor and canine. A periapical radiograph of the mandible right anterior region showed no bony or tooth involvement. The mandibular anterior teeth were crowded and the right mandibular canine was rotated. No tooth was mobile or tender to percussion. The maxillary left central incisor was fractured. The maxillary right first and second molars were carious but there was neither pulpal involvement nor an observable periodontal lesion.
Differential diagnosis for the localized gingival enlargement included fibroma, peripheral giant cell

granuloma, peripheral ossifying fibroma and pyogenic granuloma. Surgical excision of the soft tissue lesion was carried out in conjunction with osteoplasty of the tiny bony protuberance which was located on the adjacent alveolar ridge. A periodontal dressing was placed and at evaluation in one week, healing had proceeded uneventfully.

Microscopic evaluation of the tissue specimen showed fibrocellular connective tissue containing spindle shaped multinucleated fibroblasts as well as few multi-nucleate giant cells. It was surfaced by a parakeratinised hyperplastic stratified squamous epithelium with prominent rete pegs. A diagnosis of peripheral giant cell fibroma was made **(Fig Ia-e).**

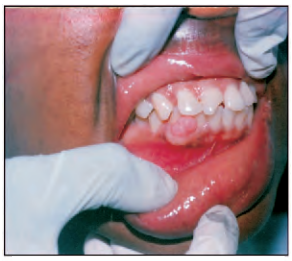

Fig Ia. Localised swelling of the lower arch before surgery

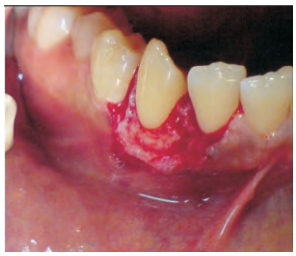

Fig Ib. Site after excision of the swelling and bony protuberance at surgery

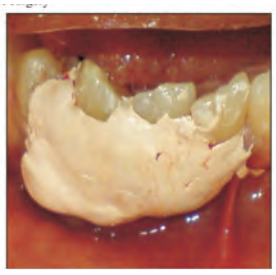

Fig Ic. Periodontal dressing over the site of surgery

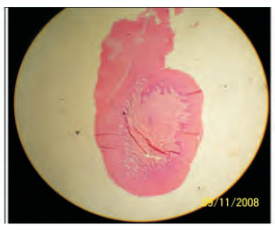

Fig Id. Peripheral giant cell fibruma histologic section showing soft tissue mass surfaced by hyperplastic stratified squamous epithelim overlying a dense fibrous connective tissue x 10

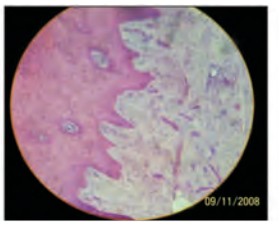

Fig Ie. Histologic section showing part of hyperplastic epithelium overlying a dense fibrous connective tissue stroma, within which are strands of odontogenic epithelium, large stellate and multinucleated giant fibroblasts x 10

Case 2

A 25-year-old male agriculture graduate student presented with bilaterally, swollen gums in both lingual quadrants of the posterior region of the mandible. He complained of previous occasional pain and halitosis of 8 years duration. A history of remission following scaling and polishing by a dentist in Edo State was also provided. There was no positive family history of similar gingival swelling. The patient was a diagnosed asthmatic but without any major episodes in the past 6 years. He used a salbutamol inhaler, was a non-smoker and brushed his teeth once daily.

Intra-oral examination revealed swollen and inflamed gingiva lingual to the mandibular left molars and extending from the attached gingiva to the occlusal level. There was a similar swelling not inflamed though more firm to bony-hard upon palpation around the molars in the other three quadrants of his mouth. Bleeding on probing was elicited in false pockets around the left mandibular molars. Radiographic evaluations yielded nothing unusual. The patient had a Class II malocclusion with an overbite which impinged upon the gingival enlargement, particularly in the mandibular left quadrant. Haematological investigations (FBC, RBC and WBC differentials) showed all parameters to be within normal limits. A clinical diagnosis of chronic hyperplastic gingivitis was made. Scaling and polishing were conducted and oral hygiene instructions given.

Two weeks after this initial conservative periodontal treatment, there was reduction of bleeding on probing. A gingivectomy/gingivoplasty with supra- and subgingival debridement was performed but with difficulty using scalpel due to the dense nature of the swelling. A periodontal dressing was placed for a week with satisfactory healing. A histopathological examination revealed dense fibrous avascular tissue and a diagnosis of idiopathic gingival fibromatosis was made **(Fig IIa & IIb).**

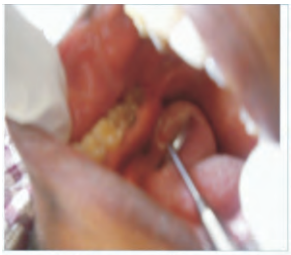

Fig IIa. Diffuse firm swelling on lingual aspect of posterior teeth before surgery

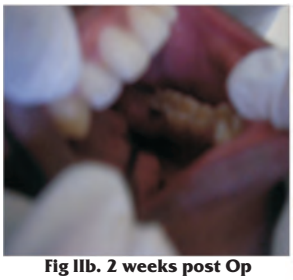

Fig IIb. 2 weeks post Op

Case 3

A 65 year old female retired civil servant complained of swollen gums of three years duration in the mandibular right region. She brushed her teeth twice daily and reported occasional bleeding on brushing and mild pain on mastication. The patient was hypertensive and had previously been on nifedipine which was changed to atenolol six months prior to the visit to the dental clinic. The patient had previously had uneventful extractions of the maxillary first molars, scaling and polishing, implants to replace the right and left maxillary lateral incisors and the maxillary right central incisor. The mandibular left molar was endodontically treated. The oral hygiene was poor with marked swelling and inflammed gingivae around the mandibular right cuspid and bicuspids. The swollen areas bled on probing with minimal true pocketing. Oral hygiene was fair in the mandibular left and maxillary left regions and true pockets with bleeding upon probing existed around the maxillary anterior implants.

Radiological investigations (periapical and orthopantomogram) revealed no unusual bony pathology

in relation to the mandibular right gingival lesions. Haematological investigations were within normal limits as was her blood pressure at the time of examination. The clinical diagnosos made were drug-induced gingival enlargement which was more severe around the mandibular right cuspid and bicuspids regions, peri-implantitis around the maxillary anterior implants, as well as generalized chronic marginal gingivitis.

An initial full mouth scaling and polishing with oral hygiene instruction was carried out, followed by subgingival scaling around the implants. Two weeks after the scaling, a gingivectomy was performed extending from the mandibular right second molar to the lateral incisor. A periodontal dressing was applied for 2 weeks, and a 0.12% chlorhexidine gluconate oral rinse was prescribed twice daily for 2 weeks. Post-surgery evaluations at one and two weeks showed the operative site to be healing uneventfully. A histopathological report of the biopsy specimen showed a hyperplastic proliferating stratified squamous epithelium with intraepithelial buds and dense collagenised fibrous connective tissue. Within the connective tissue were lymphocytes and plasma cells. A diagnosis of mild gingival hyperplasia possibly secondary to nifedipine use was made **(Fig IIIa &IIIb).**

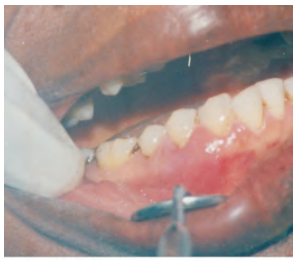

Fig IIIa. Diffuse oedematous/ hyperplastic gingiva before surgery

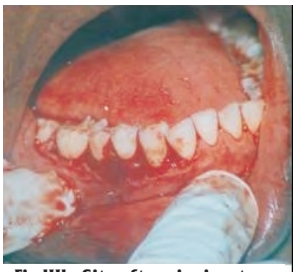

Fig IIIb. Site after gingivectomy at surgery

Discussion

These three cases demonstrate some of the different clinical manifestations and diagnoses for gingival enlargements. The gingiva is a common site for either neoplastic or non-neoplastic lesions[9]. The literature shows that the great majority of localized overgrowths of gingival tissue are considered to be reactive and non-neoplastic lesions[10] as with the case one [3].

In a report by Torres-Domingo et al[11], females are twice as likely as males to develop a giant cell fibroma, the lesion in our Case 1. Such female predilection is also in consonance with the findings of Tamashiro et al[12]. Of the 172 cases of reactive hyperplasia in the oral cavity seen by Zarei et al[13], 64% involved the gingiva. Traumatic fibroma and peripheral fibromas with calcification were seen more commonly in the mandibular region as the case of peripheral giant cell granuloma in this report. In this case, a tiny bony hard protuberance was observed in relation to the overlying fibroma. By this observation, it is to be emphasized that careful examination should be done before surgery and such inconspicuous lesions be considered during surgery. This protuberance contrasts with other reports where bone resorption was the typical finding[1,2].

Generalized gingival enlargements in one or both jaws can occasionally be Idiopathic gingival fibromatosis (IGF) or drug induced. IGF is reported to be a rare disease[14]featuring asymptomatic, nonhaemorrhagic, nonexudative and proliferative lesion of the gingiva[15] which commonly arises during eruption of permanent incisors, though some reports have also mentioned involvement of the primary dentition[15-17]. It is also possible that IGF can be aggravated by secondary inflammation in patients with poor plaque control[18]. The second case was diagnosed as idiopathic as there was no family history of similar lesions. Some patients with IGF may present with isolated lesions or as part of a syndrome[16,17]. There may be variation in the presentation of lesion location. Hereditary GF tends to occur more frequently as a generalized type than IGF[19]. In a previous report by Zachin et al[15] the lesion was on the labial aspect of the posterior region of the jaws[15] while in our case it was on the lingual aspect of the posterior teeth.

The gingiva is generally pink and stippled in health but may become inflamed because of plaque accumulation[20] as in **Case 2** of this report. This contrasts with other presentations of gingival enlargement particularly those that are drug-induced (which are fibrous and vascularised) as demonstrated in **Case 3** of this report. The prevalence of gingival overgrowth has been reported to range between 14.7% and 83%[21,22] among patients taking nifedipine. Currently, the mechanism of drug-induced enlargement is not well understood but is clearly multifactorial[21]. It has been suggested that inhibition of apoptosis by nifedipine may cause epithelial hyperplasia[23]. Evidence suggests that nifedipine inhibits both adherence and lipopolysaccharide-stimulated macrophage-induced death of fibroblasts resulting in gingival overgrowth[23].

Treatment of gingival overgrowth depends on its diagnosis. Clinicians disagree about the timing and number of procedures to be performed surgically in IGF cases[14]. Generally, the treatment protocol includes scaling, chlorhexidene mouthrinse and surgical excision with placement of periodontal dressings[14]as performed in **Case 2** of this report. The treatment of drug-induced gingival enlargement consists of drug substitution where possible and effective plaque control[24,25]. When this regimen fails to achieve the desired result, surgical intervention becomes inevitable to provide a satisfactory outcome. These lesions may recur and patients should be appropriately informed about such a possibility. It is also pertinent that clinicians especially dentists have a clear knowledge and understanding of the drugs that cause this phenomenon and must also consult the physicians in the management of such cases.

Conclusion

Gingival enlargements may present problems in diagnosis and treatment as demonstrated in these three cases. A diagnosis must be established before treatment, and this often requires a biopsy. A biopsy also assists in educating the patient about etiopathogenesis and prognosis of such a lesion. Often, haematological tests are necessary to ensure that the gingival enlargement is not secondary to a systemic disease such as leukemia. Thorough history and clinical examination are necessary to identify aetiological factors in order to prevent recurrence and facilitate optimal oral hygiene care. Non-surgical periodontal therapy by both patient and clinician with frequent recalls must be emphasized particularly in the drug-induced and gingival fibromatosis cases.

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