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CASE REPORT

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An uncommon presentation of an inflammatory gingival enlargement – responding to non-surgical periodontal therapy

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Abstract: *Background:* The various clinical manifestations of inflammatory gingival enlargement reported are more or less similar regardless of the underlying aetiological factors. Unusual presentation and unknown aetiology pose a diagnostic challenge for a periodontist. *Methods:* A 34-year-old Indian woman presented with the complaint of gum swelling that was sessile, lobulated, soft in consistency and bluish red in colour with ulcerated surface in some region, which was covered by the necrotic slough. This type of enlargement was unusual and some underlying systemic pathology was suspected. But a written consultation from her physician confirmed her systemic health, which was based on clinical, radiological and haematological investigations. Histopathological examination confirmed the diagnosis of inflammatory gingival enlargement. Patient was treated with oral hygiene instructions, scaling and root planning. *Result:* Within a month of conventional periodontal therapy, gum enlargement reduced markedly and patient was put on oral hygiene maintenance programme. *Conclusion:* Periodontal therapy is diagnosis-driven and, to the extent possible, should address all the possible factors that impact development and progression of diseases that may affect periodontal tissue. In plaque-induced periodontal diseases, non-surgical periodontal therapy is still a gold standard among all the therapies available.

Key words: gingival hyperplasia; gingivitis; non-surgical periodontal therapy

Introduction

Various aetiological factors have been listed in the literature, causing gingival enlargement and the treatment is based on an understanding of the cause and the underlying pathological changes (1). In most cases, prolonged exposure to bacterial plaque was shown to initiate the condition, and gingival enlargement may result from chronic inflammatory changes. The chronic gingival enlargement starts with papillary gingival enlargement followed by marginal gingival enlargement and gradually producing a life preserver-shaped bulge around the tooth with increasing severity of the condition. Occasionally, it occurs as a discrete sessile or pedunculated mass resembling a tumour (1).

The other aetiological factors causing gingival enlargement are selective drug therapy, (1, 2) familial or genetic conditions, (1, 3) and

pregnancy-induced enlargement (4). Sometimes, it may be the sequelae of other pathological conditions of the oral cavity, such as soft tissue tumours, (1, 5) squamous cell carcinomas (6) and malignant lesions. (1, 5) In these instances, bacterial plaque is usually not directly associated with the condition, and periodontal therapy may be secondary to the control of the malignant condition. Conditions not yet identified could also affect the normal size of the gingiva.

Medline search revealed many cases of localized, exuberant gingival proliferation, but none of them was generalized. This manuscript presents an unusual and severe case of generalized inflammatory gingival enlargement, which responded well to the non-surgical periodontal therapy.

Case description

A 35-year-old woman came to the Department of Periodontics with the chief complaint of gum swelling, which was present since previous 6 months. On general examination, the patient was looking lean and thin with no associated drug and family history of any kind. Her vital signs were within normal limits.

According to the patient, the gingival enlargement was progressive since its onset 6 months earlier, and was causing obvious cosmetic and functional problems. Any associated dental pain was negative. Intra-oral examination of the periodontium revealed a generalized gingival enlargement with a greater severity in the right maxillary posterior palatal tooth region as well as mandibular anterior tooth region. Enlargement was sessile, lobulated, soft in consistency, and bluish red in colour with erythematous ulcerated surface in some region, which was covered by the necrotic slough. The enlarged gingival tissue had covered the entire labial and palatal aspects of teeth #16, 17 and 18 like an apron, which was retractable with almost similar clinical appearance in mandibular anterior teeth (Fig. 1). Considering that the poor maintenance of the oral hygiene by the patient or gingival enlargement could have created impedance for proper oral hygiene, good amount of supragingival accretions were noted on the exposed surfaces of the teeth. Her mean plaque index was 2.3 and mean gingival index was 3. The complete periodontal charting could not be done because of the excessive bleeding during probing and calculus, but she had obvious generalized clinical attachment loss. In addition, multiple root stumps, carious teeth and flaring of front teeth were noted. Her upper central incisor was extracted in her childhood for an unknown reason. A clinical differential diagnosis of inflammatory hyperplasia, acquired haemangioma, Kaposi's sarcoma, metastatic tumours, benign mesenchymal tumours, leukaemic enlargement granulomatous enlargement and squamous cell carcinoma was made.

On radiological examination, orthopantomogram showed generalized horizontal crestal alveolar bone loss without any obvious bony pathology. In addition, tooth #24 showed periapical radiolucency.

Her medical history was non-contributory and according to the patient, this was her first visit to any dentist. She has been investigated with routine haematology, which was normal except leucocytosis with normal electrolytes and renal function. She was referred to the physician to rule out any systemic abnormality, where she had undergone screening for diabetes mellitus, HIV and tuberculosis, but no underlying systemic abnormality was found.

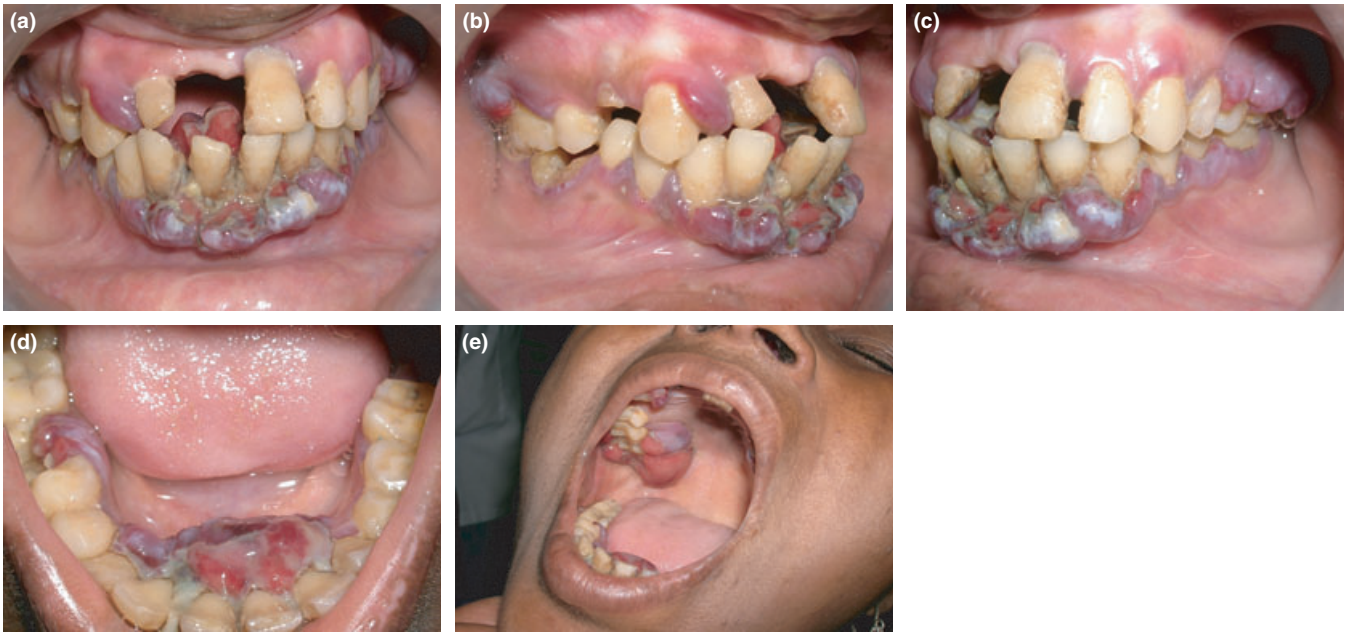

Fig. 1. Initial photographs of gingival enlargement. (a) frontal view, (b) right lateral view, (c) left lateral view, (d) lower occlusal view, (e) upper occlusal view.

Incisional biopsy of the enlarged tissue was performed from the right posterior palatal side under local anaesthesia. During incision, minimal bleeding was noted and after removing a small piece of representative tissue area, a suture was placed. The gingival tissues were collected and stored in 10% formalin and submitted for histopathological analysis. Chlorhexidine (0.2%) mouth wash with an analgesic was prescribed to the patient and the suture was removed after 5 days. Uneventful healing was noted.

On histopathological examination (Fig. 2), the microscopic examination revealed tissue surfaced by parakeratinized gingival epithelium with hyperplasia and denuded areas at places (Fig. 2). The papillary epithelial projections contained connective tissue stroma, which revealed features of granulation tissue. Marked capillary proliferations along with few dilated blood spaces were noted. Fibrillar delicate stromal tissue with inflammatory infiltrate of neutrophils, lymphocytes and plasma cells were seen. Areas of liquefaction necrosis and repair were also noted. These histopathological features are most consistent with non-specific inflammatory hyperplasia, and the pathology report confirmed a diagnosis of non-specific inflammatory gingival enlargement.

Treatment plan formulated for the patient was motivation and oral hygiene instructions along with full mouth scaling and root planning under local anaesthesia in two sittings. After the completion of non-surgical periodontal therapy, the patient was advised to rinse with 10 ml chlorhexidine (0.2%) mouthwash 12 h for 2 weeks. The patient was already taking multivitamin capsules as advised by her physician. She was recalled after 3 weeks for follow-up. On examination, the growth was significantly reduced in size, gingival colour was changed from bluish red to pink, and consistency was also firm (Fig. 3). Her mean plaque index was 1.1 and gingival index was 0.9. Periodontal probing pocket depth was also within normal limits. Her oral hygiene instructions were reinforced and the patient was sent to the respective departments for treatment of other dental problems.

Discussion

Categorization of diseases affecting the gingiva requires evaluation of patient signs and symptoms, medical and dental histories, and a clinical examination that includes the extent, distribution, duration and physical description of lesions affecting the gingiva, clinical or relative attachment levels and radiographs (7). Gingival enlargements are a common clinical finding and most represent a reactive hyperplasia as a direct result of plaque-related inflammatory gingival disease. The usual clinical presentation of plaque-induced gingival hyperplasia includes enlarged gingival contours due to oedema or fibrosis (8), colour transition to a red and/or bluish red hue (9), bleeding upon probing (9) and increased gingival exudates (10). However, seldom it presents itself as it presented in our case.

After looking carefully into the patient's medical, dental and family history, we ruled out drug-induced GE and hereditary GE. Hereditary GE usually manifests much earlier in life and appears more firm and fibrotic, clinically (3). Other differential diagnosis, which were made clinically e.g. acquired haemangioma, Kaposi's sarcoma, metastatic tumours, benign mesenchymal tumours and squamous cell carcinoma were also ruled out through a thorough medical examination, blood investigation and histopathological examination. Kaposi's sarcoma is a common condition in AIDS and sometimes in other immunodeficient conditions and may look similar to the condition presented here (11), but ELISA test for HIV was negative, which ruled out the possibility of Kaposi's sarcoma. Sometimes, metastatic tumours may also mimic the same condition, (11) but no primary lesion was noted. Gingival hyperplasia as a result of leukaemic infiltrate is an unusual situation (12). Moreover, no abnormality in morphology and number of cells was seen in the patient's blood investigation as well as in microscopic picture of biopsy specimen. Benign mesenchymal tumours were also a possibility, but ruled out through microscopic examination. Granulomatous gingivitis can indicate a foreign body reaction or systemic condition, such as Crohn's disease or Wegener's granulomatosis. Collections of histiocytes are present in the submucosa, along with lymphocytes and scattered multinucleated giant cells (13). No such abnormality was found in microscopic examination.

The microscopic features were most consistent with non-specific inflammatory gingival enlargement, and the pathology report confirmed a diagnosis. However, a medication-induced gingival enlargement could not be ruled out based on the microscopic examination alone, but after taking medical and drug history carefully, this was also ruled out.

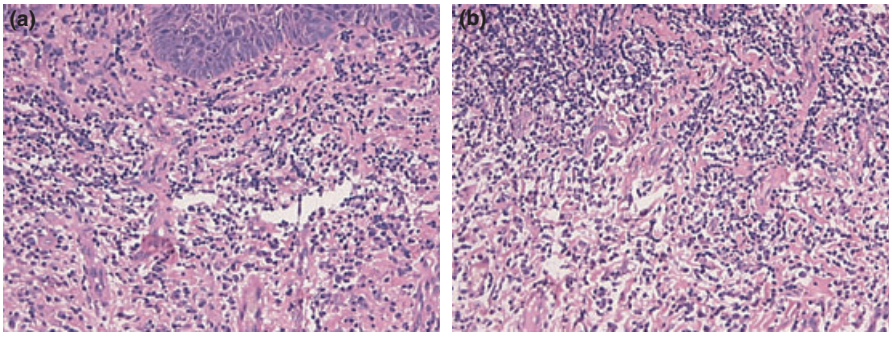

Fig. 2. Microscopic picture. (a) epithelium showed basilar hyperplasia at places with juxta epithelial intense inflammatory infiltrate (10x), (b) the inflammatory infiltrate consisted of macrophages, polymorphs and predominant lymphocytes with multiple dilated capillaries (40x).

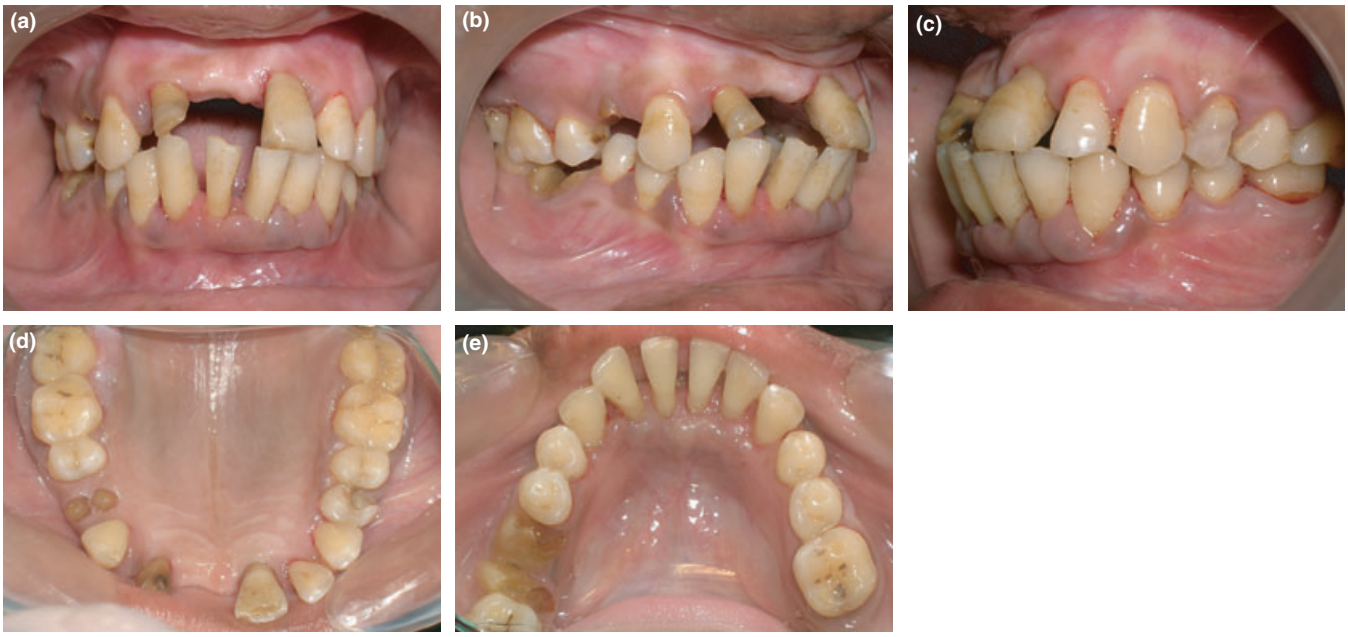

Fig. 3. Post-treatment reduction in gingival enlargement. (a) frontal view, (b) right lateral view, (c) left lateral view, (d) upper occlusal view, (e) lower occlusal view.

Based on clinical examination, it was difficult to ascertain a direct cause and effect relationship. Prolonged exposure to the dental plaque as well as low host resistance may be the cause of exuberant reactive proliferation of the gingiva (14). Hormonal changes during menstruation may also be one of the causes of exuberant proliferation of the gingiva (4). Oral inflammatory hyperplastic lesions are a local response of tissue to injury (15) and it can be considered an over-exuberant reparative response. Calculi, overhanging margin of restorations, foreign bodies, chronic biting, margin of caries, sharp spicules of bone and overextended borders of appliances are the possible sources of traumatic irritants (11). In the present case, none of them was present except calculi, which may be the possible cause of irritation. Dental plaque, which contains a number of microorganisms, also produces an inflammation, which in turn stimulates the formation of granulation tissue that consists of proliferating endothelial cells, chronic inflammatory cells and few fibroblasts (14). Clinically, the lesion is asymptomatic and smoothly contoured or lobulated with a very red appearance because of rich vascularity and transparency of the non-keratinized epithelial covering. It is moderately soft and spongy and blanches on careful digital pressure (14).

Treatments of such types of inflammatory hyperplasias are removal of such irritants and maintenance of oral hygiene by the patient. As the recurring insult is eliminated, the lesion shrinks markedly as the inflammation subsides, and the vascularity is reduced. The decrease in the size of the lesion is directly proportional to the amount of inflammation present (11). If the lesion is composed mostly of fibrous tissue, there is little shrinkage, but if considerable granulation tissue and inflammation exist, there is marked shrinkage (11). In the present case also because of marked redness and inflammation, after removal of local irritant factor, shrinkage of the lesions was noted.

In conclusion, development of a logical and properly sequenced treatment plan is a derivative of the periodontal assessment and diagnosis. Periodontal therapy is diagnosis- driven and, to the extent possible, should address all modifying factors and risk factors that impact development and progression of plaque-induced periodontal disease. Thorough scaling and root planing (SRP) is still considered the gold standard in periodontal therapy. Beyond SRP, no one treatment modality is the answer in every case. However, the clinician must have specific endpoints or goals that therapy should achieve.

Conflict of interest and source of funding

The authors declare that they have no conflict of interest. This study was self-funded by the authors.

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