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Case report

Necrotizing periodontitis in a heavy smoker and tobacco chewer – A case report

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ABSTRACT

Necrotizing periodontitis is a distinct and specific disease characterized by rapidly progressing ulceration of the interdental gingiva and then spreading along the gingival margins and leading to acute destruction of periodontal tissues. Necrotizing ulcerative gingival lesions are common in developing countries because of poor nutritional status, poor oral hygiene and debilitating conditions. In the developed world it is mostly seen in patients with the HIV infections and other immune system dysfunctions. The exact etiology of the necrotizing lesions is still unknown; however a fuso-spirochaetal infection along with weakened host immune system seems to play a major role in the pathogenesis of these diseases. Presented is the case of acute necrotizing periodontitis in a 21 year old male patient with no systemic disease but a history of tobacco use (chewing and smoking) since 7 years. The patient was managed by conservative treatment followed by surgery for the correction of gingival defects.

1. Introduction

Necrotizing lesions of the oral cavity generally involve the periodontium. Necrotizing periodontitis (NP) is a destructive periodontal disease mostly associated with the characteristics of necrotizing gingivitis (NG) clinically. These lesions manifest with areas of necrosed gingiva, spontaneous bleeding, intense pain and alveolar bone attachment loss. Factors causing NP are little understood. It may be a consequence of NG lesions or as a result of previous occurring periodontitis. Periodontal pathogens treponema, fusobacterium and prevotella are implicated in the disease. Host immune deficiency as in HIV, diabetes, leukemia, poor oral hygiene, nutritional deficiency, stress and smoking predisposes to this condition [1].

This report describes a very aggressive case of NP in a heavy smoker and tobacco chewer and its successful management.

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2. Case report

2.1. Clinical presentation

A 21 year old male patient reported to the Department of Periodontiology of Dr. Z.A. Dental College, A.M.U. Aligarh, with chief complaint of severe pain and bleeding in the gums along with difficulty in eating since one week. There was history of swelling of gums three months back for which no treatment was undertaken. There was a history of pan chewing along with tobacco since 5 years. Also the patient was a heavy smoker since 7 years (more than 20 cigarettes/day). The patient was systemically healthy and there was no medical history contributory to the dental problem. The patient stopped brushing since the pain started. Earlier the patient used his fingers to clean his teeth. Extraorally, the patient presented with enlarged lymph nodes and slight fever. On intraoral examination, poor oral hygiene was noticed with gross accumulation of dental plaque especially along the gingival margin (Fig. 1). A thin whitish film (pseudomembrane) covered part of the attached gingiva. Examination of the gingiva revealed necrosis of the papillae, causing it to separate into one facial and one lingual portion with an interposed necrotic depression producing considerable tissue destruction leading to the formation of characteristic punched out crater like depressions. Heavy stains were present.

Radiographic examination demonstrated horizontal bone loss in the lower anterior region. (Fig. 2) A diagnosis of necrotizing ulcerative gingivitis with periodontal involvement in lower anterior region was made.

2.2. Case management

After explaining the condition to the patient a written informed consent was taken and a two step treatment plan was designed comprising of conservative and surgical phase.

In the conservative phase, on the very first appointment, after the application of topical anesthesia the acutely inflamed areas were swabbed with a moistened cotton pallet to remove the sloughed tissue and non-attached surface debris along with irrigation with 3% H_2O_2 and sterile warm water. Supragingival scaling was attempted as thoroughly as the condition allowed. Patient was prescribed metronidazole 250 mg QIDS for seven days. A small gingival tissue from the posterior was taken and sections were prepared to evaluate histopathologically. Histopathological report further confirmed our diagnosis. Nonspecific infiltrate comprising of areas of ulcerated squamous epithelium with abundant neutrophils and fibrin pseudomembranes with fragments of necrotic epithelium were seen (Fig. 3a and b). Patient was advised to rinse with 3% H_2O_2 and sterile warm water (1:1 dilution) four times a day and with 0.12% chlorohexidine twice a day. The patient was instructed to avoid tobacco as well as pan chewing, to take adequate rest and take proper diet. Proper oral hygiene instructions were given.

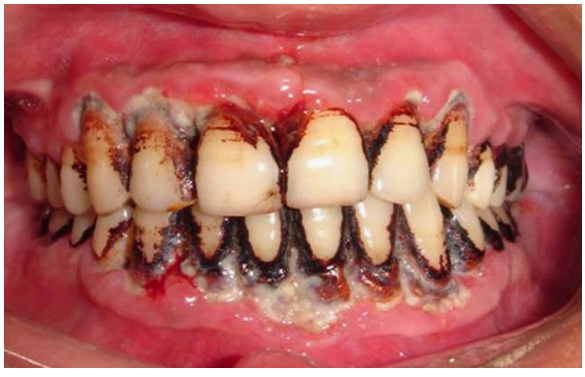

Fig 1 – Clinical view showing the gingival lesions.

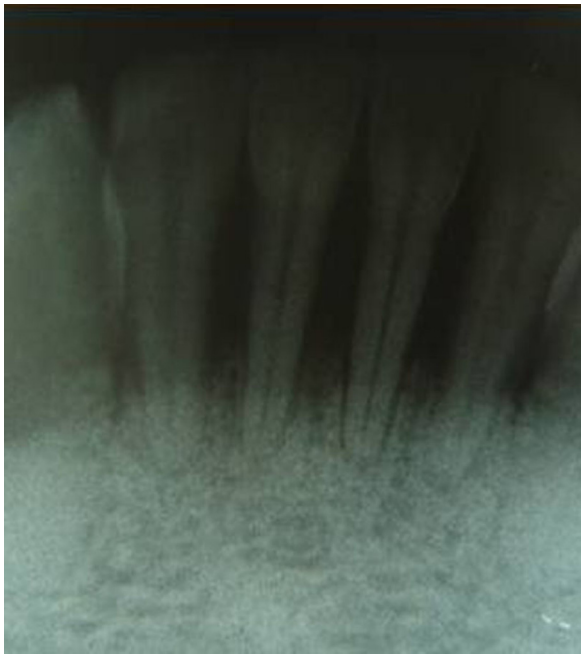

Fig 2 – Intra oral peri-apical radiographic view showing horizontal bone loss in lower anterior region.

After 2–3 days the patient was re-evaluated and again supragingival scaling was performed. At every re-evaluation phase oral hygiene instructions were reinforced.

After five days the patient was almost symptom free, so thorough scaling and root planning was done. At this time 3% H_2O_2 rinses were discontinued but 0.12% chlorohexidine rinses were continued. Patient was again instructed to avoid tobacco as well as pan chewing.

The patient was recalled after four months and gingivoplasty was done in lower anteriors under local anaesthesia.

2.3. Clinical outcomes

The patient was very responsive to the treatment provided. After the very first appointment there was decrease in pain and gingival inflammation. After complete debridement, there was complete remission of inflammation but the gingival contour remained affected. Gingivoplasty corrected this defect but as there was bone loss in the lower anteriors, there was apical shift of gingiva. The patient's condition responded to the therapy given, with a complete resolution of the lesion (Fig. 4).

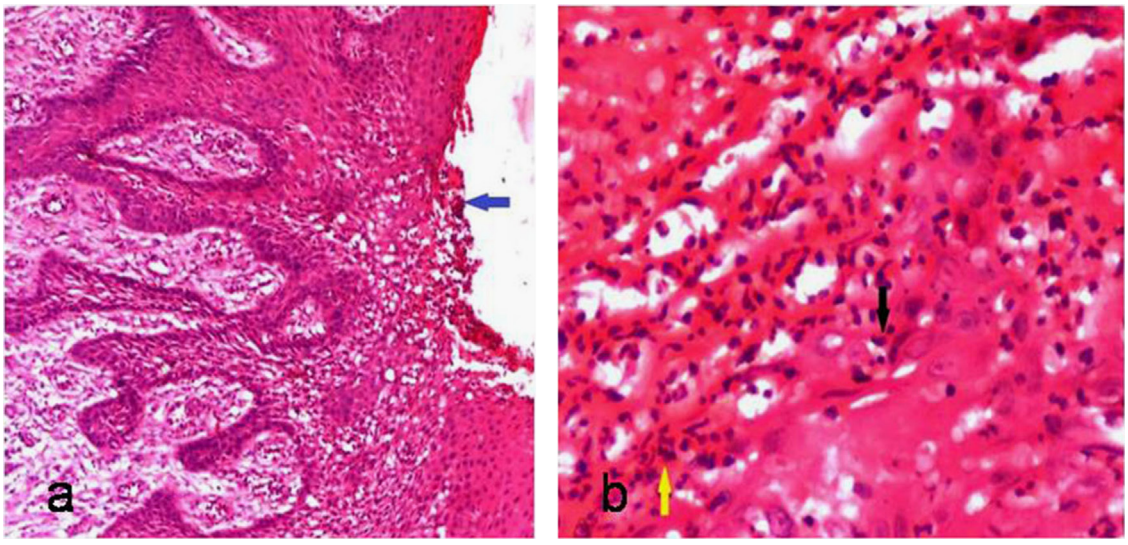

Fig 3 – Histological view under 100X magnification (3a) and 400X (3b).

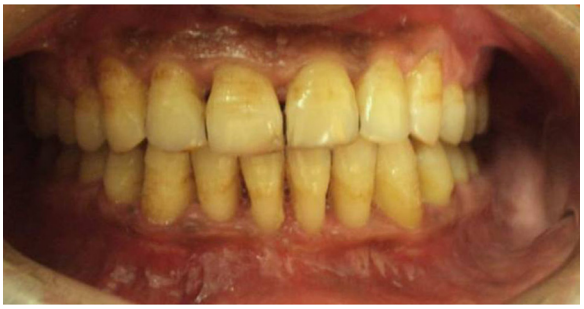

Fig 4 – Post operative view.

3. Discussion

Necrotizing periodontitis is now a rare disease in developed countries but yet found in developing countries due to existing poor nutritional status, stressful living conditions, poor oral hygiene and state of debilitation often resulting from endemic contagious diseases. Diagnosis of this disease is made on clinical signs and symptoms [2]. Early diagnosis and prompt treatment of the disease prevents the progression and cellular destruction and results in resolution of the disease.

Our case presented the classical picture of necrotizing lesions localized to the anteriors and not involving the lingual and posterior regions. Massive destruction may be associated with poor oral hygiene and tobacco use in either chewable form or as cigarettes. It is a known fact that the pathogenic processes involved in any periodontal diseases are modified by environmental factors such as smoking [3]. This may be the result of thousands of chemicals present in cigarette smoke which are powerful inducers of inflammatory responses and are toxic to multiple cell types [4]. Nicotine present in the tobacco causes release of local and systemic catecholamines leading to increase in gingival papillary flow and resulting in papillary necrosis. Also nicotine disrupts the balance of MMP/TIMP ratio thereby resulting in increased collagen and periodontal destruction [5]. Tobacco consumption has direct effect on homeostatic mechanism in the periodontium as well as influences periodontal microflora. Changes in tissue vascularity, alteration in fibroblast attachment and function, suppression of osteoblast proliferation, stimulation of osteoclasts and alteration in PMNL function leading to impaired phagocytosis, superoxide and hydrogen peroxide generation, integrin expression and protease inhibitor production are some of the consequences of tobacco use [6]. Studies demonstrate that smokers have higher plaque levels, more pathogenic flora and less favorable response to periodontal treatment [7]. In majority of the published cases necrotizing lesions have been rarely attributed to tobacco chewing and smoking. Poor oral hygeine may have facilitated the penetration and pathogenecity of the microbes but not necessarily be the predisposing factor to NUP as stated by Taiwo [8] not all with poor oral hygeine may develop necrotizing lesions.

Treatment of necrotizing lesions includes effective removal of local irritating factors that is the plaque and calculus. Hydrogen peroxide mouthwash is advocated to increase the supply of oxygen to the anaerobic microbes thereby inhibiting their growth. Chlorhexidine gluconate mouthwash has antiplaque effects. Prompt periodontal therapy and oral hygiene reinforcement and patient compliance are essential in treatment of such lesions.

In our case the sudden amount of destruction noted is a grave sign of negative consequences of tobacco on oral health. Of importance is that this destruction is in absence of any other immunocomprised conditions like HIV, diabetes and leukemia. This paper highlights the imminent danger faced by the populations where tobacco and its products are not only accessible but quite inexpensive.

4. Conclusion

The case reports the massive periodontal destruction that occurred in a patient consuming tobacco and its successful management. Therefore, it is highly recommended for those with habit of smoking and tobacco chewing should be made

aware of its negative impact on oral health and be regularly monitored to aid in early detection and to provide proper management of periodontal inflammatory conditions to minimize its destruction.

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Conflict of interest

There is no conflict of interest among authors.

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