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Rapidly Progressive Periodontal Disease Associated with Human Immunodeficiency Virus

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ABSTRACT

Severe periodontal inflammation with generalized dental plaque accumulation, spontaneous and severe gingival bleeding, fungal infection, and interdental papillae necrosis are presented in a patient infected with human immunodeficiency virus (HIV). Bite-wing radiographs revealed a generalized horizontal alveolar bone loss of 7-8 millimetres in both arches. Erythematous patches were noted on the gingival mucosa in both jaws. DNA testing was performed to identify the periodontopathogens. The patient had no signs or symptoms of acquired immunodeficiency syndrome. This case-report presents the massive periodontal destruction that occurred in a patient infected with HIV. Therefore, it is highly recommended that patients infected with HIV should be regularly monitored to aid in early detection and to provide proper management of periodontal inflammatory conditions to minimize its destruction.

Key words: Periodontitis. HIV-AIDS. Interdental papillae necrosis. Periodontal bone loss.

INTRODUCTION

Periodontal inflammatory disorder may either be a slowly progressive or rapidly progressive disease. If left undiagnosed and untreated in its early stages, rapidly progressive periodontal disease may cause alveolar bone destruction and ultimately tooth loss at an alarming rate.

Rapidly progressive periodontal disease is a common manifestation in immunocompromised patients particularly those infected with Human Immuno-deficiency Virus (HIV) in comparison to periodontal disease in HIV-negative individuals.¹⁻³ The group of disorders affecting individuals infected with HIV is predominantly notable since oral manifestations are usually the first clinical expressions of the viral infection.¹ There are many established oral manifestations of HIV including oral candidiasis, oral leukoplakia and periodontal disease. In a recent study, Paster *et al.* reported a diverse variety of microbes associated with the development of periodontal diseases in HIV-positive patients which differ from the typical periodontal pathogens such as *Porphyromonas gingivalis* (*P. gingivalis*).⁴ Mechanical

plaque removal and oral hygiene maintenance are important steps in healing periodontal inflammatory conditions.⁵

In the present report, we describe a case of an unusual rapidly progressive periodontal disease and necrotizing ulcerative periodontitis in a HIV-positive individual.

CASE REPORT

A 49-year-old male reported with a 10-year history of infection with the HIV. The patient also reported a three-year history of loss of vision, renal failure, and hypertension.

The patient presented with severe pain, ulcerated gingival papillae, mobile teeth and fetor oris. A comprehensive full-mouth periodontal examination revealed the presence of severe periodontal inflammatory conditions with a generalized dental plaque accumulation and spontaneous gingival bleeding on gentle probing. Necrotizing ulcerative periodontitis manifests as a rapid necrosis and destruction of the gingiva and periodontal attachment apparatus. White and erythematous patches were observed on the gingival mucosa in both jaws (Figure 1). These white patches were gently scraped off and a light microscopic investigation revealed the presence of fungal hyphae. At the first-visit full-mouth peri-apical and bite-wing radiographs were taken (Figure 2). The radiographs showed the bone level to be at the cemento-enamel junction. However, at the one year follow-up, the radiographs displayed an aggressive and generalized horizontal alveolar bone loss of 7-8 millimetres in both arches (Figure 3).

Subgingival plaque was collected using absorbent paper points and a polymerase chain reaction-based test (Micro-IDent HAIN Lifescience GmbH, Nehren, Germany)

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DISCUSSION

To our knowledge from indexed literature, this is one of the few case-reports that showed extensive periodontal bone loss (PBL) in a HIV-positive patient. In a recent study, the association between periodontal bone loss and HIV-infection was investigated. PBL was measured on radiographs obtained from HIV-positive patients.⁴ The results showed no significant differences in periodontal bone loss between HIV-positive patients and controls (HIV-negative individuals).⁴ The present report is in disagreement with these results since the patient presented with extensive periodontal bone loss. Although the prevalence of periodontal pathogens in HIV-positive and negative individuals has been reported to be similar; however, it may be hypothesized that the microbial load may be significantly higher in immunocompromised individuals as compared HIV-negative subjects with periodontal disease. Murray *et al.* reported that the prevalence of periodontal pathogens (*P. gingivalis* and *P. intermedia*, *E. corrodens*, and *C. rectus*) was significantly higher in sub-gingival plaques removed from HIV-positive individuals than those removed from HIV-negative individuals.⁶ Hence, the increased bacterial counts may be associated with an overproduction of endotoxins by these pathogens thereby amplifying the magnitude of periodontal bone loss; nevertheless, controversy persists in this regard. Cross and Smith used culturing, followed by a colony lift method and DNA probes, to compare plaques from HIV-positive and negative patients with periodontal disease.⁷ Periodontal pathogens, including *C. rectus*, *Capnocytophaga ochracea*, *P. gingivalis* and *P. intermedia* were highly prevalent in plaques removed from both groups, i.e., 75 to 100% prevalence correspondingly. The only difference between the groups was a slight but significantly higher proportion of *P. gingivalis* in the HIV-positive subjects, which was attributed to a subgroup of HIV-positive subjects with widespread attachment loss. These findings suggest that there is no unique plaque flora which accounts for the aggressive nature of periodontal disease in HIV-positive subjects.

A direct relationship between periodontal disease and systemic conditions such as cardiovascular disease, diabetes and renal disorders has been reported.^{8,9} Renvert *et al.* showed significantly higher counts of putative pathogens in patients with CVD as compared to controls (individuals without cardiovascular disease).⁸ It is noteworthy that the HIV-positive patient presented in the current report also had a history of cardiovascular disease and renal disorder. Therefore, it seems that HIV-infection coupled with hypertension and RF may have amplified the severity of periodontal bone loss in the present case.

Although *Candida* (*C.*) species (majorly *C. albicans*) are commonly found in the normal oral flora; however, under immunocompromised conditions, they may also



Figure 1: White and erythematous patches on the gingival mucosa in both jaws.

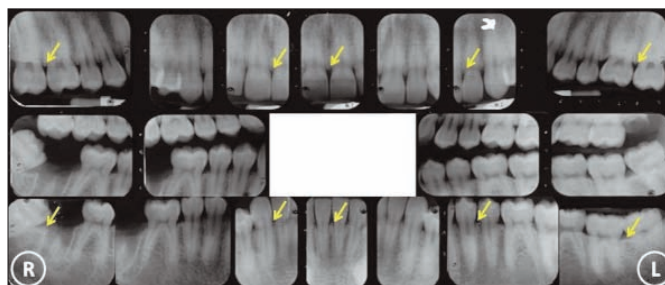


Figure 2: Intra-oral periapical and bitewing radiographs performed at the first visit showing alveolar bone at the cemento-enamel junction (yellow arrows) in both arches.

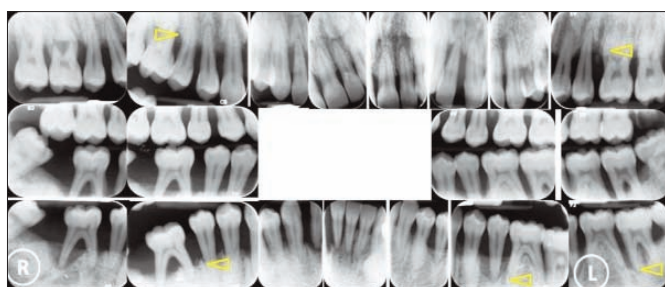


Figure 3: Intra-oral periapical and bitewing radiographs taken at one year follow-up showing an aggressive and generalized horizontal alveolar bone loss of 7 to 8 (yellow arrows) millimetres in both arches.

was performed for identification and quantification of putative periodontopathogens. The microbial results showed the presence of high amounts of anaerobic bacteria including *P. gingivalis*, *Prevotella intermedia* (*P. intermedia*), *Campylobacter rectus* (*C. rectus*) and *Eikenella corrodens* (*E. corrodens*).

become opportunistic pathogens.¹⁰ It has been reported that periodontal inflammatory conditions are aggravated in immunocompromised patients with *C. albicans* colonization compared to patients without *C. albicans* in the same group.¹⁰ It may, therefore, be hypothesized that oral fungi may contribute in aggravating periodontal inflammation in HIV-positive individuals. In the present case-report, fungal hyphae were identified on the oral mucosa; however, their contribution in exacerbating periodontal inflammation remains unclear. It has also been reported that periodontal inflammation is worse in immunocompromised females with oral *Candida* colonization as compared to males in the same group.¹⁰

It is mandatory for healthcare providers to be aware of the characteristics and presentation of oral manifestations of HIV infection; however, rapidly progressive periodontal disease may not necessarily be the first sign of HIV infection in the affected patients. It is recommended that in patients harbouring high amounts of anaerobic bacteria in the dental plaque, antibiotic treatment (such as Amoxicillin and Metronidazole) and antifungal therapy (such as Miconazole) should be used as adjunct therapies with conventional scaling and root planning. The selection of therapy must take into account, the periodontal status, the patient's medical status and possible adverse reactions to antibiotics.

This case-report presents the massive periodontal destruction that occurred in a patient infected with human immunodeficiency virus. Therefore, it is highly recommended that patients infected with the human immunodeficiency virus should 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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