

CASE REPORT

A case of acute necrotizing periodontitis with not known origin

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Abstract

In cases of aggressive periodontal bone destruction, subgingival microbial analysis should be done, tooth extractions should be planned to control disease progression if non-surgical periodontal treatment is ineffective.

KEYWORDS

aggressive periodontal bone loss, necrotizing periodontitis, tooth extraction

1 | INTRODUCTION

A 30-year-old Asian male suffered from aggressive periodontal bone destruction. The buccal alveolar bones of his mandibular anterior teeth were completely lost within a month. Gingiva biopsies indicated severe acute inflammation. Tooth extractions and adjunctive systemic antibiotics were effective to cease disease progression although etiologies remain unclear.

Necrotizing periodontal diseases (NPD), including necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis, are the most severe inflammatory periodontal disorders caused by plaque bacteria. Necrotizing periodontal diseases are characterized by three typical clinical features (papilla necrosis, bleeding, and pain) and are associated with

host immune response impairments.¹ Necrotizing periodontal diseases can be observed in all age groups but there are geographic differences in the age distribution. Host predisposing factors, including HIV+/AIDS, or other severe systemic conditions, such as malnutrition, psychological stress and insufficient sleep, inadequate oral hygiene, pre-existing gingivitis, and previous history of NPD, tobacco and alcohol consumption, are also favoring NPD. Severe pain is often the chief reason for patient seeking treatment. In necrotizing periodontitis, periodontal attachment and bone destruction were observed, together with more frequent extraoral signs. NPD progression, extent, and severity mainly depended on host-related factors predisposing to these diseases.² Diagnosis of NPD should be primarily based on clinical findings. Microbiological and biopsy assessment may

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be recommended in cases of atypical presentations or nonresponding cases. The following case is a necrotizing periodontitis with atypical clinical manifestations.

2 | CASE REPORT

A 30-year-old Asian male presented with a chief complaint of: “gingiva swelling and increasing mobility of mandibular incisors for 2 weeks.” The patient’s general dentist had observed gingival swelling of tooth 31, 32, and 41, accompanied with Grade III mobility 1 week before. Initial examination by the dentist revealed no facial swelling, no restriction of mouth open and no fever. Cone-beam computed tomography (CBCT) revealed mild horizontal buccal bone loss in mandibular incisors, no cyst or periapical radiolucency was found (Figure 5A1-A4). An initial diagnosis “periodontal abscesses of teeth 31, 32 and 41” was made by the dentist based on gingival swelling. A regime of cephalosporin 0.25 g (tid for 7 days) and metronidazole 0.2 g (tid for 7 days) was prescribed. The patient was then referred to Department of Periodontology, Peking University School and Hospital for further periodontal treatment.

Intraoral examination revealed poor oral hygiene, heavy plaque deposits, and calculi was found on the lingual side of lower incisors because of inability to brush. A gingival examination revealed severe swelling on the whole buccal gingiva of tooth 31, 32, and 41, several fistulas were found, the gingiva swelling was to a less severe extent in the lingual side (Figure 1). Grade III mobility was observed in teeth 31, 32 and 41, probing depth was more than 10 mm in the buccal side, and 5 mm to 7 mm in the lingual sides, the pulp vitality test was dull. While the other teeth exhibited probing depths in the range of 2-4 mm. The full mouth percentage of

bleeding on probing is 53%. There were no occlusal discrepancies or interferences. The patient denied having systemic disease, and he did not report any allergies to food or to drugs. The patient was a never smoker and reported that he did not consume alcohol. He had oral examination and teeth cleaning annually, he denied any gingival pain or toothache before. Two weeks before, he had fever prior to gingival swelling.

Subgingival plaque of mandibular anterior teeth was collected with curettage and analyzed by toluidine blue staining, but no spirochetes or fusiform was found. Patient had been taking antibiotics during sampling that can affect microbiology results. Leukemia or HIV infection was excluded by fasting blood biochemical examination. Gingiva biopsy was collected from the buccal gingiva of tooth 42 and 33. The histopathological examination with H&E staining demonstrated multiple inflammatory cells infiltration, with local abscesses formation (Figure 2A). Immunohistochemical staining with CD 20, CD 38, CD 45pro, CD 68, and LCA analysis did not support the diagnosis of B-cell lymphoma, T-cell lymphoma or macrophage-derived tumors, and Ki67 staining did not support rapid cell proliferation (Figure 3). The diagnosis of granulomatosis or carcinoma of the patient was excluded by gingiva biopsy.

Meanwhile, the patient was referred to Department of Rheumatology, Peking Union Medical College, for further blood biochemical and immunoassay tests to diagnose possible systemic or autoimmune diseases. All blood indexes, including the counts of CD4+ T cells, CD8+ T cells, CD4+ CD8+, CD8+ DR+ and CD8+ CD38+, CD4/CD8 ratio, the levels of TNF, IL-6, IL-8, IL-10, ANCA-IgG, PR3-ANCA, MPO-AVCA, IgG, IgA, and IgM, which were all within normal limits, and no positive outcome was found. Systemic or autoimmune diseases were excluded by systemic blood tests.

Another CBCT examination was taken, which revealed severe progressive bone loss compared with that taken 28 days

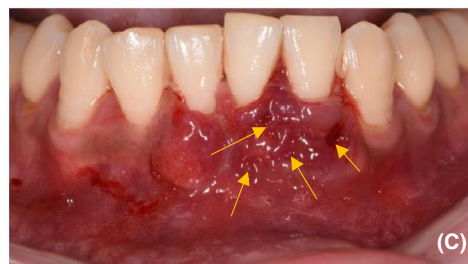


FIGURE 1 Clinical pictures at first visit of periodontics. Severe gingival swelling in the mandibular anterior region (A). Several fistulas were found on the buccal gingiva (arrows) (B). Calculus and moderate gingival swelling were found on the lingual side (C)

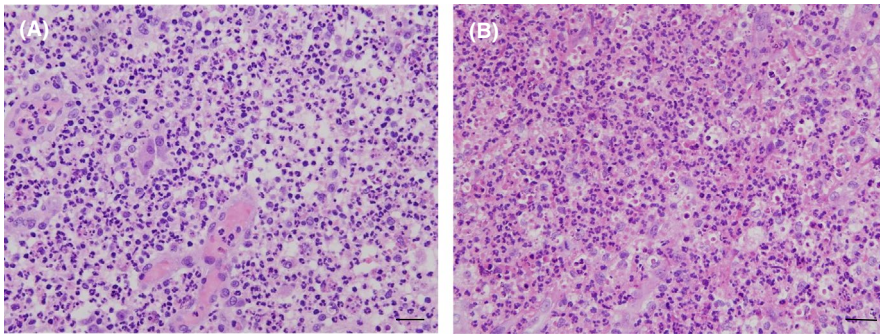


FIGURE 2 Histological appearance of gingiva tissue sampling from the buccal gingiva of tooth 33 (A) and granulation tissue collected during socket curettage after tooth extractions (B). Scale bars, 50 µm

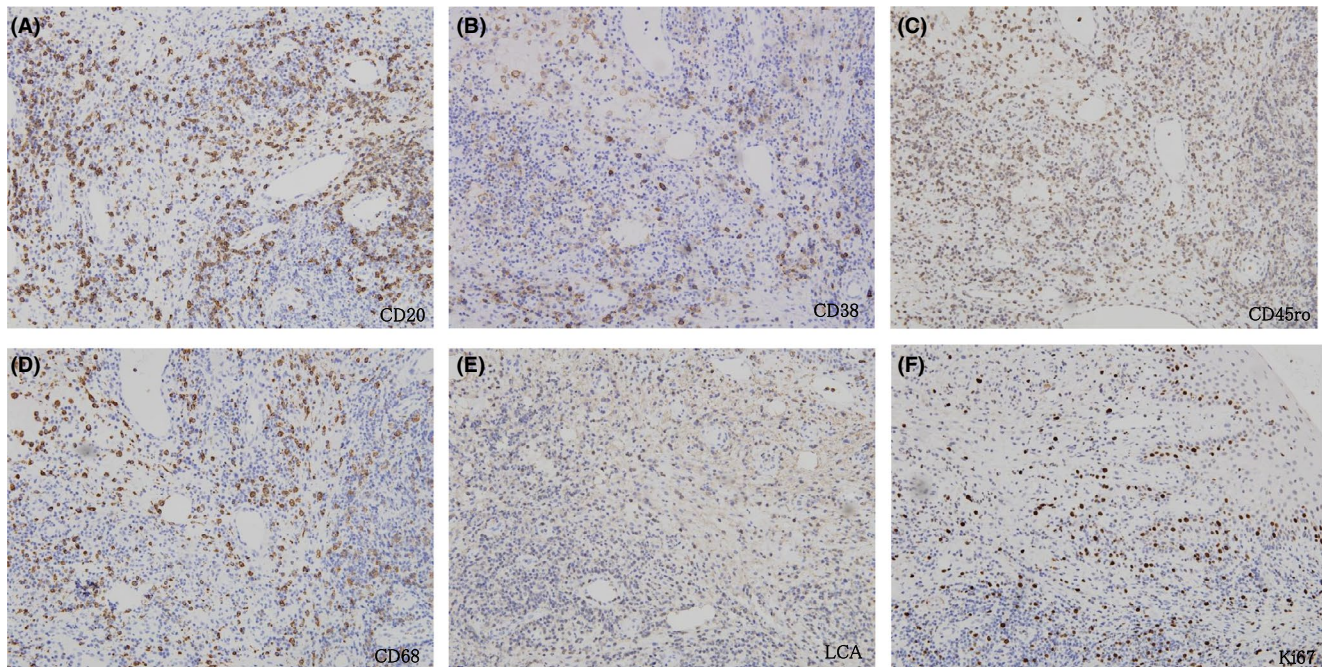


FIGURE 3 Immunohistochemical appearance of CD20 (A), CD38(B), CD45pro(C), CD68(D), LCA (E), and Ki67 (F) in gingival biopsy from buccal gingiva of tooth 33 or tooth 42

before. Buccal bones of tooth 31, 32, and 41 were lost beyond the apex and extending to the neighbor teeth, the buccal bone of teeth 33 and 42 was lost to the apex. The lingual bone of the above teeth seemed relatively intact (Figure 5B1-B4). A new periodontal abscess was formed on the buccal gingiva of tooth 33, which indicated aggressive acute periodontal inflammation.

According to the clinical and radiographic examination, as well as gingiva biopsy results, a diagnosis of acute necrotizing periodontitis was made, based on the American Academy of Periodontology classification.

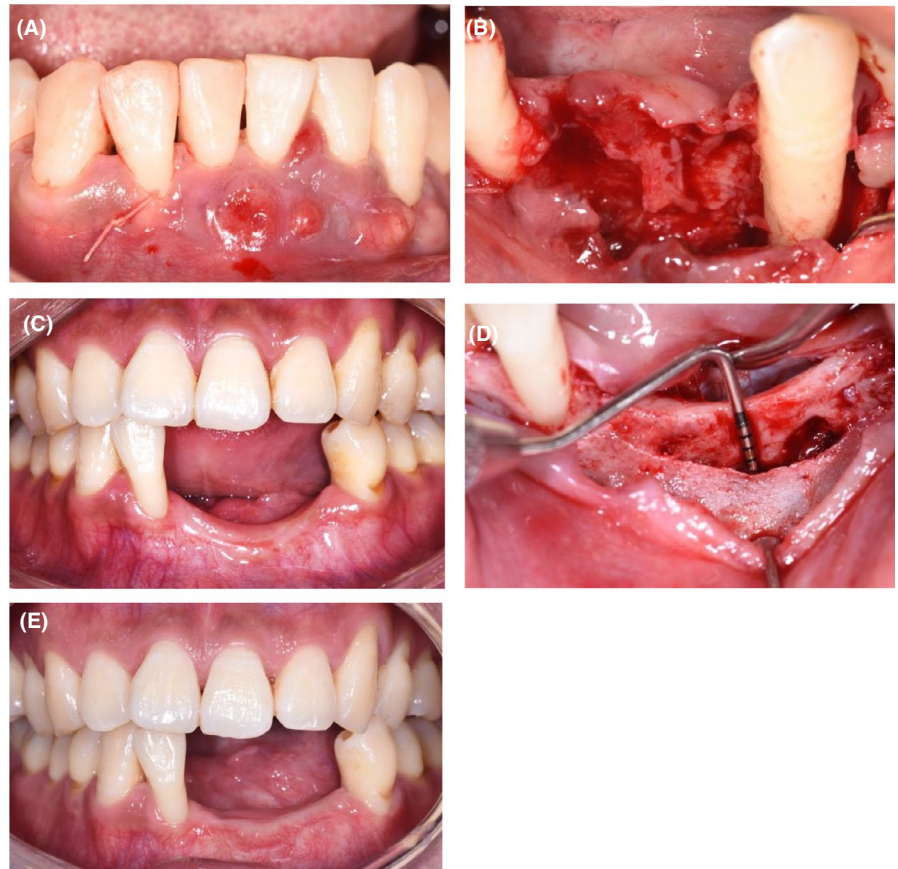
2.1 | Clinical management

On the patient's first visit, ultrasonic scaling was performed and mouth rinse with 0.2% chlorhexidine gluconate twice per day was advised for the patient. Amoxicillin clavulanate

potassium 0.457 g, (bid, for 7 days) and Metronidazole 0.2 g, (tid, for 7 days) were prescribed. Then, gingival biopsy examination was performed, and the patient was referred for blood biochemical and immunoassay tests.

At 3 weeks later, no systemic disease was reported by the pathologist or physician. Clinical re-evaluation (Figure 4A) and CBCT examination (Figure 5B1-B4) revealed that non-surgical periodontal treatment did not control periodontal bone destruction in the mandibular anterior region, teeth extraction was planned. After local anesthesia, teeth 31, 32, and 41 were extracted with no difficulty. Multiple granulation tissue and some bone sequestrum were found during socket curettage. Gingival flap was elevated from tooth 34 to tooth 42, buccal bone loss beyond root apex in tooth 33 but the lingual bone was intact (Figure 4B). Alveolar bone in the distal and lingual side was unaffected although the whole medial and buccal side was lost in tooth 44 and 42. It was decided to extract tooth 33, and remain tooth 44 and 42 after debridement.

FIGURE 4 Clinical situation before tooth extractions(A). Multiple granulation tissue and alveolar bone loss were found during tooth extraction (B). Clinical situation at 4 mo after teeth extraction (C). After flap elevation, a thin buccal bone was found (D). Clinical situation at 6 mo after guided bone regeneration (E)



Absorbable collagen was put in the sockets to keep blood clot before suture. Amoxicillin clavulanate potassium 0.457 g (bid for 7 days) and 0.2% chlorhexidine gluconate twice per day were prescribed after tooth extractions. The sockets healed normally, and the patient felt a relief of discomfort in the mandible. The granulation tissue from sockets while curettage was sent for histopathological examination, demonstrated severe inflammation infiltrated with multiple neutrophils and edema (Figure 2B).

The reevaluation at 4 months after tooth extractions showed that periodontal status of tooth 34 and 42 was stable, with no pus or deep pocket depths (Figure 4C). Another CBCT was taken, which revealed that alveolar bone healed with no further bone destruction (Figure 5C1-C4). Guided bone regeneration was performed, after flap elevation, thin buccal bone was found in the mandible anterior region, the sockets were partially healed (Figure 4D). Postoperative follow-up did not show any complication, the patient healed uneventfully (Figure 4E).

3 | DISCUSSION

Necrotizing periodontal diseases categorized as a separate class of periodontal disease in the American Academy of Periodontology classification is unique and distinct from the other kind of periodontitis.¹ The characteristic clinical

features allow the diagnosis the condition without much difficulty. However, neither severe gingiva pain nor characteristic fetor odor was reported by the patient, and bone sequestrum was only found during tooth extraction, and thus the dentist should pay special attention to this case of necrotizing periodontitis with atypical symptoms.

The etiology of aggressive periodontal bone destruction in this young Asian never-smoker male still remains unclear. The necrotizing periodontitis progression may be rapid and result in necrosis of small or large parts of the alveolar bone. The diagnosis of this patient was based on that the whole buccal alveolar bone lost within 1 month in mandibular anterior region, bone sequestrum were found during tooth extractions, and histological features of gingiva biopsy which demonstrated severe acute inflammatory infection. The clinical manifestations of the patient did not support the diagnosis of aggressive periodontitis³ or periodontal abscesses,⁴ as the rapid periodontal bone destruction located only in mandibular anterior region, did not involve molars, progression of disease was much more rapid than that in periodontal abscesses. The patient receives oral examination annually, no caries or deep pocket was reported before, perio-endo combined lesions were excluded. Granulomatosis, carcinoma, and autoimmune diseases were excluded by gingiva biopsy analysis, blood biochemical, and immunoassay tests. As the patient had been taking antibiotics during plaque sampling, which may negatively influence the results of spirochetes and

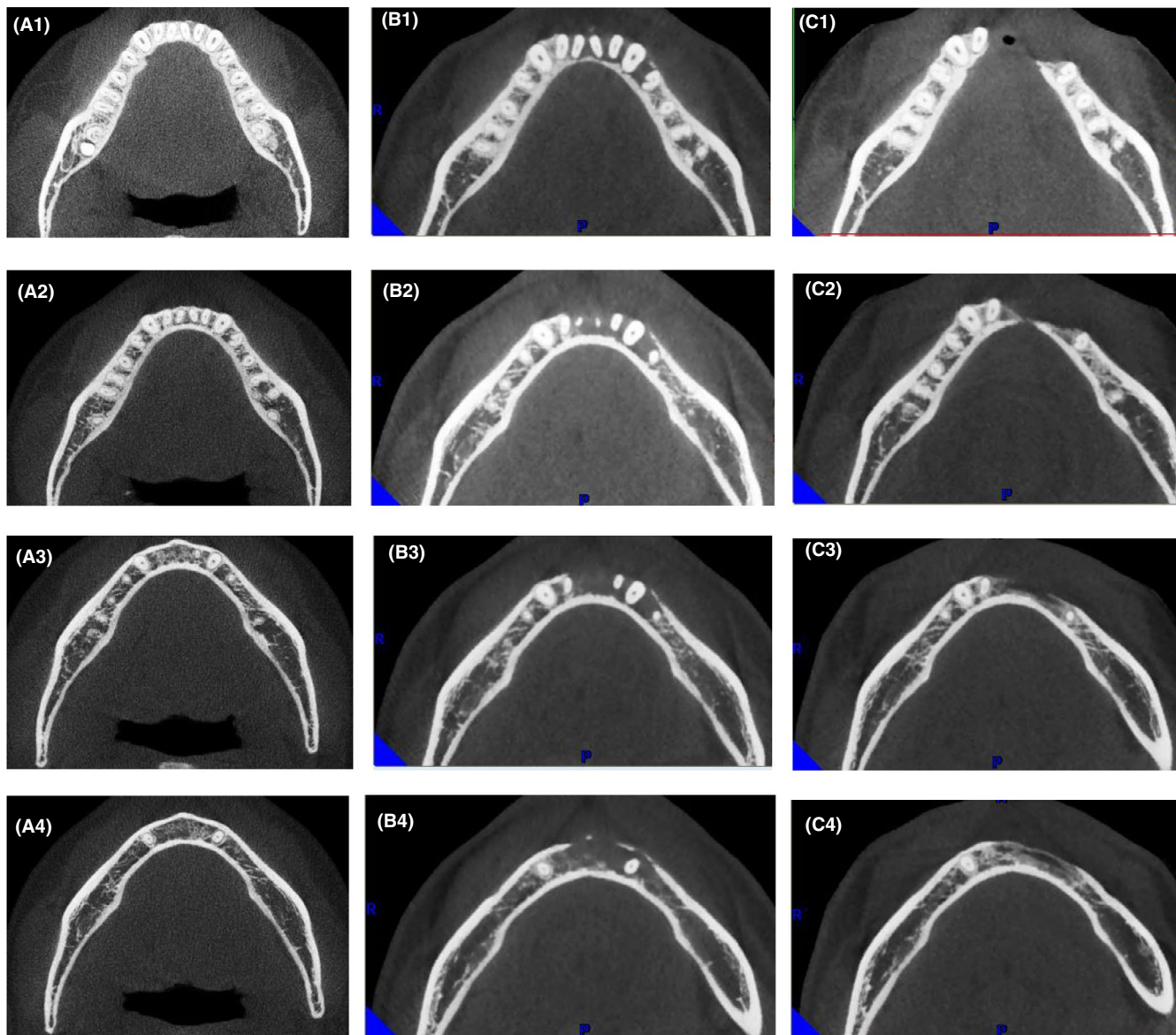


FIGURE 5 CBCT comparison of the initial examination (A), 28 d later (B) and 4 mo after teeth extraction (C). Footnote: Cross-section at (1) cervical root ; (2) middle root ; (3) apical root ; (4) beyond the root apex of incisors

fusiform detection, a diagnosis of necrotizing periodontitis was made although the putative pathogen was negative.

Predisposing factors of necrotizing periodontal diseases including poor oral hygiene, smoking,⁵ severe stress, malnutrition, alcohol abuse, and compromised host immune response.⁶ However, no obvious predisposing factors were found in the patient. The limitations of the case report are that more detailed analysis of subgingival plaque infection should be done, such as herpes virus or Epstein-Barr virus detection,⁷ which may be related to the episodic progressive of human periodontitis, and antibiotics were ineffective for the virus infection.

With respect to mandibular anterior teeth, teeth extraction other than systemic antibiotics maybe more effective to control disease progression. The patient has been taking

antibiotics for 1 month before teeth extraction. However, recurrent gingiva swelling and periodontal abscesses still persisted, and bone destruction progressed rapidly. After tooth extractions, discomfort of the patients relieved, gingiva, and bone healed as normal, the questionable teeth 42 and 34 were preserved, guided bone regeneration at 4 months post-tooth extractions was also successful. Thorough curettage after tooth extractions helps to eliminate the local infection and finally ceased disease progression. It is probably that patient will lose more teeth if we hesitate to perform tooth extractions.

The successful management of the present case of necrotizing periodontitis with atypical symptoms may be helpful for the dentist and physicians. The patient is in close maintenance to prevent possible recurrence of the disease.

4 | CONCLUSION

Although the diagnosis of necrotizing periodontitis in most cases is straightforward, in some cases the etiologies are not very clear and may have different clinical manifestations. Tooth extractions can be planned to cease disease progression when nonsurgical periodontal treatment is not effective.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

RR: contribute to manuscript preparation. HM: contribute to definition of the case. RL: contribute to manuscript editing and review.

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