CASE REPORT

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Effective Management of Acute Necrotizing Ulcerative Gingivitis with Proper Diagnosis and Immediate Treatment

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Necrotizing periodontal diseases, especially acute necrotizing ulcerative gingivitis (ANUG), it should be noted, occur abruptly and progress rapidly, eventually causing severe soft-tissue and alveolar bone loss. This report presents the cases of two ANUG patients and provides a brief treatment protocol for easy and effective clinical management. After proper diagnosis, sequential treatment with cessation of mechanical brushing, along with a prescription of systemic antibiotics and chlorhexidine as a mouth rinse, scaling, root planing, and supportive periodontal therapy, was utilized. In all cases discussed in this report, there was marked improvement in a few days. ANUG, though an uncommon disease, can be efficiently managed with proper diagnosis and immediate treatment.

Key Words: Anti-bacterial agents; Chlorhexidine; Gingivitis

Introduction

In an international workshop for the categorization of periodontal diseases and conditions in 1999, a new category, "necrotizing periodontal diseases," which includes necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis, was introduced[1]. Recently, the term "necrotizing gingivitis and necrotizing periodontitis" has replaced "necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis"[2]. Additionally, according to the location of the area involved, necrotizing periodontal diseases are now subdivided into necrotizing gingivitis, necrotizing periodontitis and necrotizing stomatitis[3,4].

The primary cause of necrotizing periodontal

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diseases is infection by microbes including *Treponema* species (spp.), *Selemonas* spp., *Fusobacterium* spp., and *Prevotella intermedia*; secondary factors are human immunodeficiency virus (HIV) infection, malnutrition, lack of sleep, stress, and smoking[2]. Any combination of these factors causes down-regulation of host immune response and triggers pathologic changes of the periodontium. Moreover, the rapid increase of such microbes leads to distinct clinical signs and symptoms of necrotizing periodontal diseases[5,6].

According to several studies, the three major clinical signs and symptoms necessary for reliable diagnosis of necrotizing periodontal diseases include (1) acute necrosis and ulcer at interdental papilla, (2) pain, and (3) bleeding[7]. Acute necrosis and ulcers result in crater-like inverted papilla. Pain and gingival bleeding around one or two teeth appear abruptly, the most common sites of which are the maxillary and mandibular anterior gingival areas[5]. Necrotizing periodontal diseases occur most typically among 21 to 24 years old in both developed and developing countries[2,8].

Although the prevalence rates of necrotizing periodontal diseases, especially acute necrotizing ulcerative gingivitis (ANUG), are not high, it should be emphasized that they can effect the most severe periodontal condition and, in doing so, can lead to soft-tissue destruction or alveolar bone loss within a short period of time[2,4]. Therefore, proper initial diagnosis and immediate treatment are most important. This report presents the cases of two ANUG patients, based on which a brief treatment protocol for easy and effective management of ANUG is provided.

Case Report

This study protocol was approved by the Pusan National University Hospital Institutional Review Board (#PNUH-E-2016079).

1. Case 1

A 23-year-old male whose chief complaint was spontaneous bleeding on the upper and lower right side was referred from a local dental clinic. He had no contributory medical history but was a heavy smoker, and stated that due to severe stress, he had not been able to sleep well during the past two weeks. An intraoral examination demonstrated spontaneous bleeding and pain on the right side accompanied by a severe foul odor and ulcerated, punched-out papilla with pseudomembrane (Fig. 1). A radiographic examination revealed no specific alveolar bone loss (Fig. 2). To check for any possible relationship with systemic diseases or infections such as HIV or other microbial infections, a complete blood count (CBC) with differential

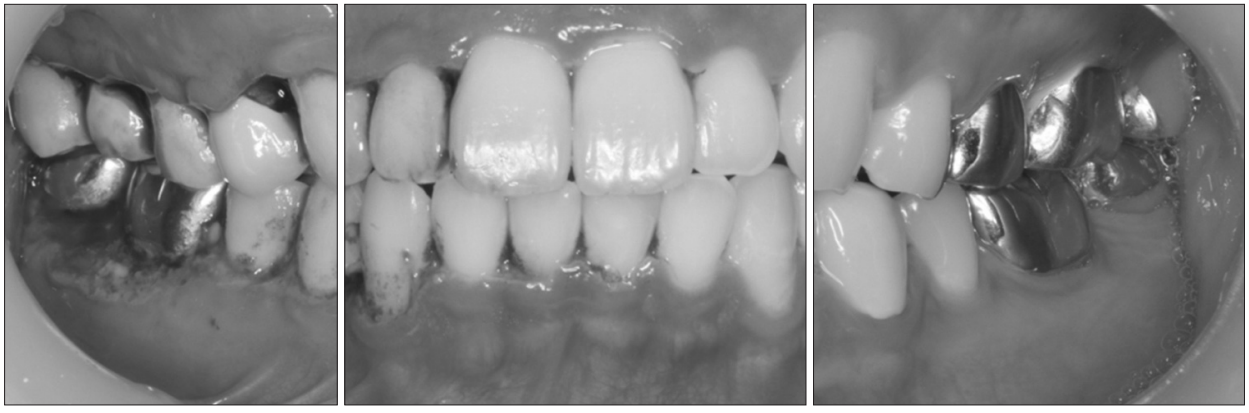

Fig. 1. Pretreatment clinical view of Case 1. Note the gingival bleeding and ulcerated, punched out papilla with pseudomembrane on the right side.

analysis was performed. The test results revealed an elevated neutrophil percentage (80.3%) coupled with a slightly reduced lymphocyte count (15.5%), a finding which indicated an acute infection state.

On the basis of the clinical and laboratory findings, the patient was diagnosed with ANUG and prescribed metronidazole 500 mg (Flasinyl; CJ Healthcare, Seoul, Korea) and amoxicillin and clavulanate potassium 625 mg (Augmentin; Ilsung, Seoul, Korea) as systemic antibiotics and chlorhexidine (Hexamedine; Bukwang Pharm., Seoul, Korea) as a mouth rinse. He was instructed to cease mechanical tooth brushing in order to avoid bleeding and pain. Two days later, spontaneous bleeding and pain were completely resolved, and the necrosis and ulceration of the papilla had almost completely disappeared (Fig. 3). Supragingival scaling and root planing were performed, and the patient was instructed to resume oral hygiene care and to quit smoking. After seven days, the appearance of the gingival outline was almost recovered but papillary loss remained in the maxillary right first premolar area; therefore, additional subgingival root planing was performed (Fig. 4). At the 1-month follow-up, the patient demonstrated a good oral hygiene state, and the papilla crater at the maxillary right first premolar had improved (Fig. 5). It was therefore determined that the maxillary right first premolar would not require periodontal surgery but rather would be monitored consistently in follow-up examinations, as there was no deep pocket depth or alveolar bone loss.

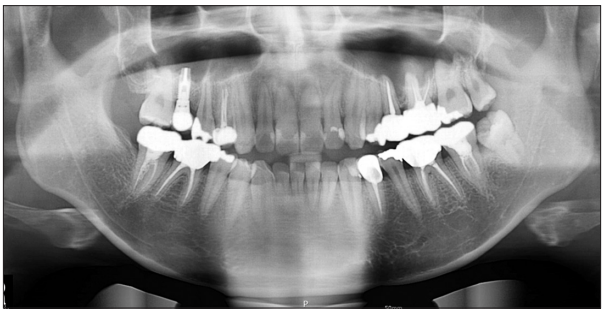

Fig. 2. Pretreatment radiograph of Case 1. The radiographic examination revealed no specific alveolar bone loss.

2. Case 2

A 22-year-old male presented with spontaneous bleeding and severe pain with ulceration. He had no remarkable medical history but was a heavy smoker. Two days previously, he had visited a local dental clinic due to the painful ulceration, and was prescribed steroid ointment. However, the ulcer became worse, and spontaneous bleeding initiated. An intraoral clinical examination demonstrated

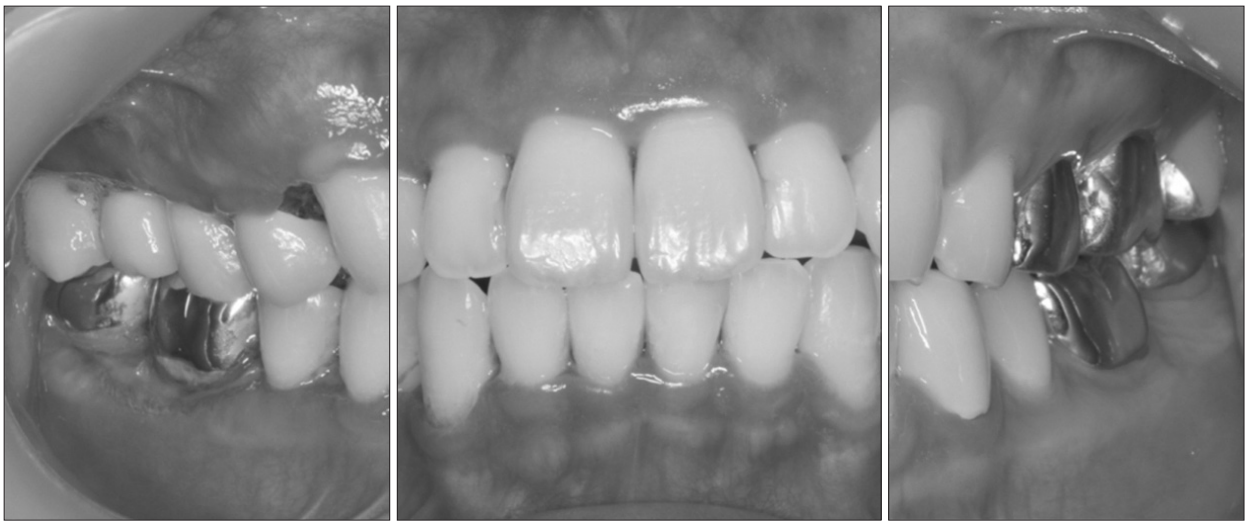

Fig. 3. Posttreatment at day 2 of Case 1. Note the remission of gingival bleeding.

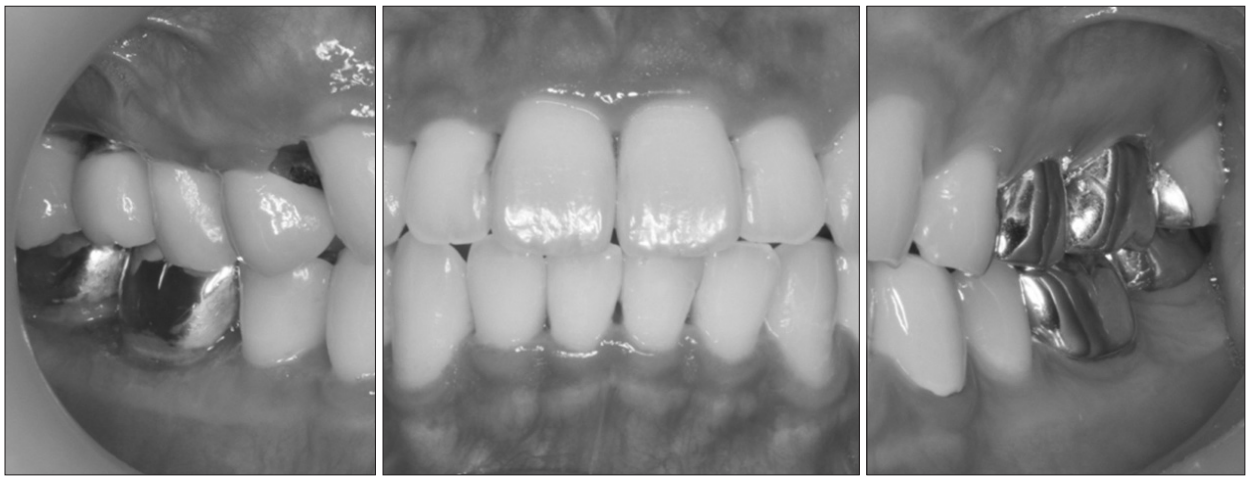

Fig. 4. Posttreatment at day 7 of Case 1. Note the disappearance of the ulceration.

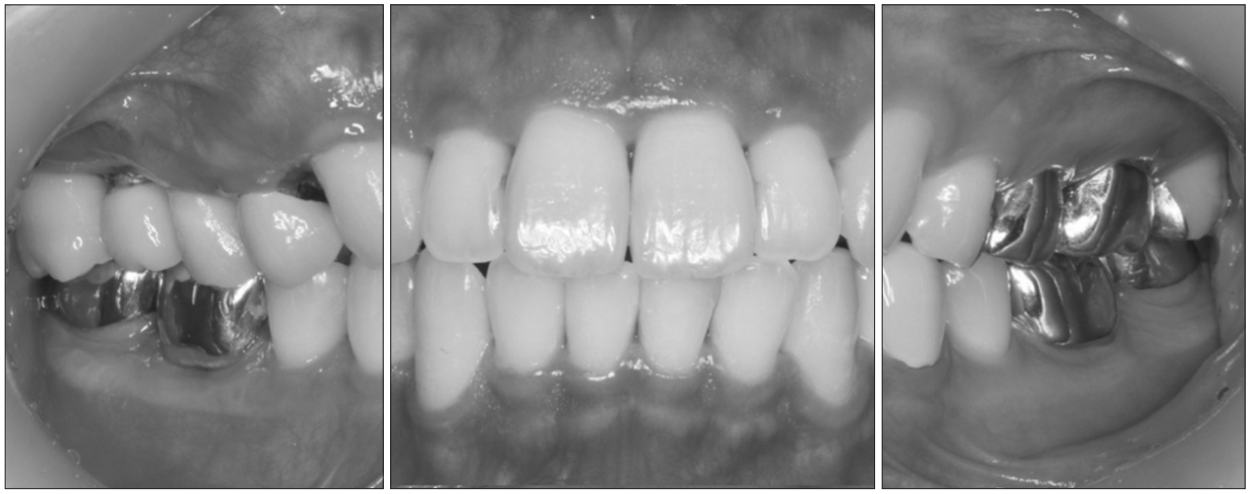

Fig. 5. Posttreatment at 1 month of Case 1. Note the complete healing of the right gingiva and maintenance of gingival health.

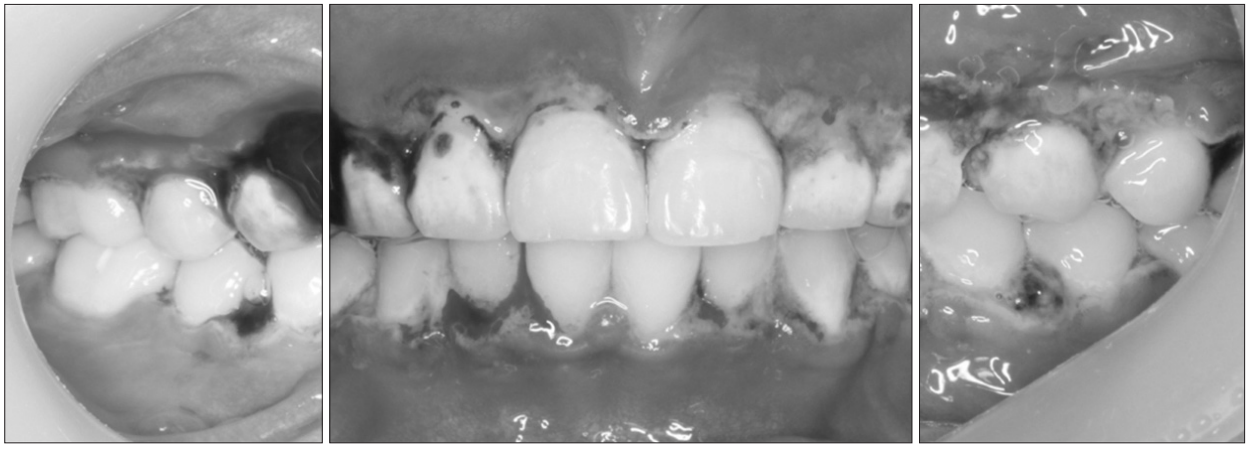

Fig. 6. Pretreatment clinical view of Case 2. Note the gingival bleeding with blood clot and ulcerated, punched out papilla with pseudomembrane on the right and left sides.

spontaneous gingival bleeding with a large blood clot and an ulcerated, punched-out papilla with pseudomembrane (Fig. 6). A panoramic X-ray revealed no alveolar bone destruction but multiple severe dental caries (Fig. 7). The patient reported that due to stress, he had skipped meals and did not brush his teeth at all during the previous two weeks. Additionally, he was found to be running a fever (38.9°C). In order to uncover any other systemic diseases or infections, a CBC with differential analysis was performed, the results of which revealed an elevated neutrophil percentage (77.7%) with a slightly reduced lymphocyte count (12.6%). Based on the overall clinical and laboratory findings, the patient was diagnosed with ANUG.

Subsequently, the blood clot was carefully removed by a periodontist, and the patient was prescribed systemic antibiotics with chlorhexidine as a mouth rinse. He was instructed to cease mechanical tooth brushing so as to avoid bleeding and pain. Two days later, the bleeding and pain were completely resolved, and the necrosis and ulceration of the papilla had almost entirely disappeared (Fig. 8). Supragingival scaling and root planing were then performed, and the patient was instructed to resume oral hygiene care and to quit smoking. After seven days, the appearance of the gingival outline was almost recovered, and dental caries treatment was initiated (Fig. 9). At the 1-month follow-up, the patient demonstrated good oral hygiene care, and dental caries treatment was continued. Long-term follow-up was scheduled (Fig. 10).

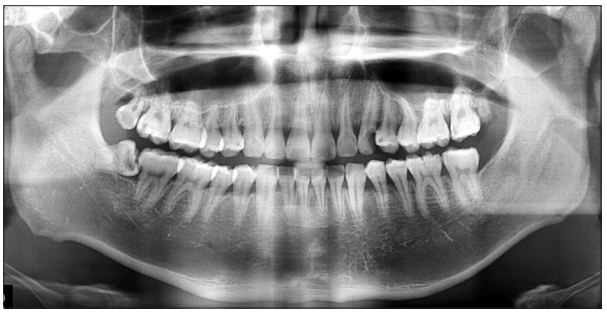

Fig. 7. Pretreatment radiograph of Case 2. The radiographic examination revealed no specific alveolar bone loss, but multiple dental caries.

Discussion

Microbes are believed to be the primary cause of ANUG; *Treponema* spp. and *P. intermedia*, for example, play important roles in its occurrence[9]. Spirochetes, fusiform microbes found in necrotic lesions, have an ability to invade the epithelium and connective tissue and release endotoxins, which can lead to periodontal tissue destruction through activation or modification of host immune

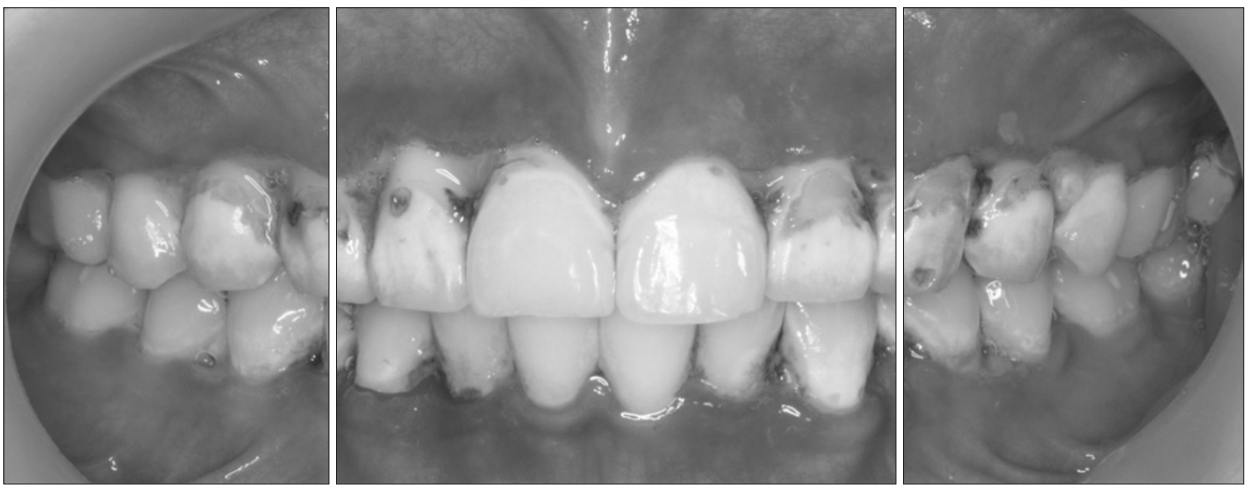

Fig. 8. Posttreatment at day 2 of Case 2. Note the remission of gingival bleeding but with persistent ulceration in some regions.

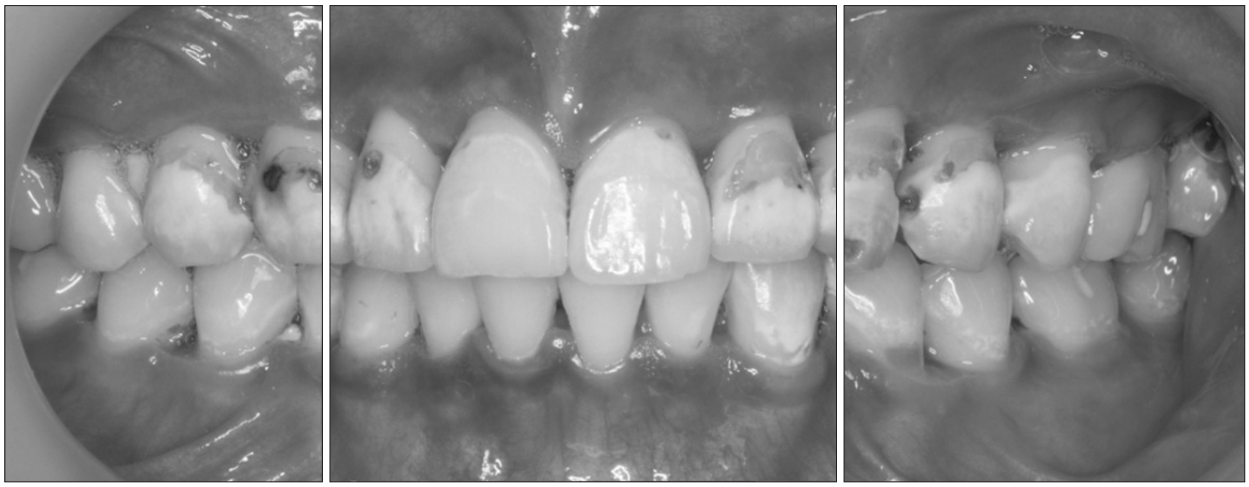

Fig. 9. Posttreatment at day 7 of Case 2. Note the disappearance of the ulceration.

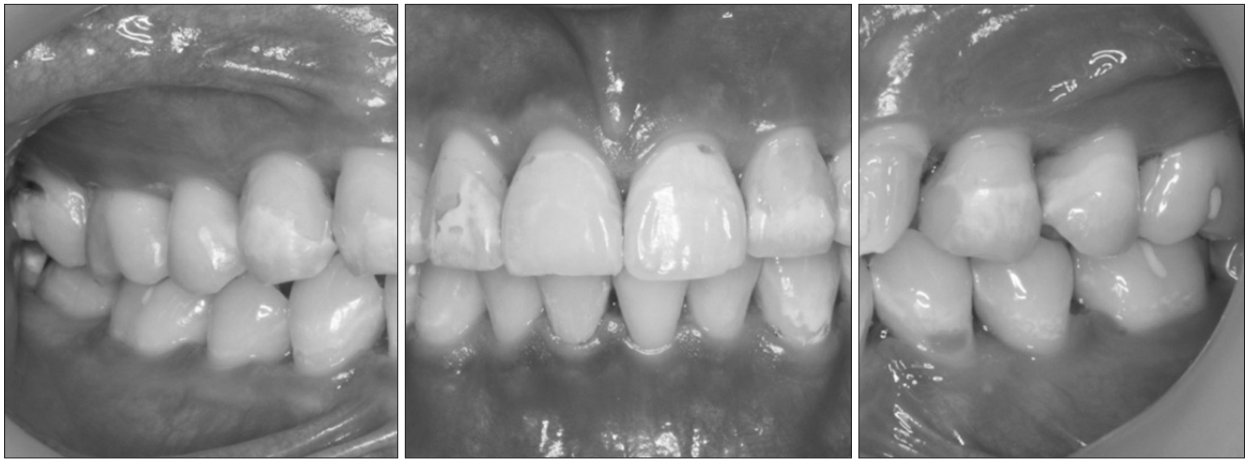

Fig. 10. Posttreatment at 1 month of Case 2. Note the complete healing of the gingiva and the initiation of caries treatment.

response[10].

Pindborg[11] and Goldhaber[12] reported that nicotine released in smoking activates the secretion of noradrenaline and adrenaline, which cause the reduction of gingival circulation. Similarly, psychological stress can also activate sympathetic nerves and reduce blood flow to the gingiva by systemic secretion of adrenaline and production of peripheral noradrenaline in gingival blood vessel walls. Such vasoconstrictors combine with the endotoxins of gram-negative microbes that cause ANUG[5]. Malnutrition is a frequent and important contributory factor to ANUG in developing countries, as it reduces the synthesis of acute-phase proteins by incomplete acute-phase response, and causes the depletion of major antioxidant nutrients, especially in children and young adults[13]. The two young patients whose cases are discussed in these pages were smokers suffering from malnutrition and severe stress over the course of the preceding two weeks.

The diagnosis of ANUG is based on clinical findings; lesions usually initiate at the interdental papilla and demonstrate a typical "punched-out" appearance with inverted papilla[2,9]. The interdental papillary tip and col area is often the starting point of ANUG due to aseptic necrosis of the gingival epithelium when there is insufficient

blood supply. MacPhee and Beagrie[14] deemed this area the initiating area of acute necrotic gingival lesion. In cases of ANUG, spontaneous gingival bleeding and pain occur abruptly according to the severity and range of the lesion. Occasionally, halitosis, adenopathies and fever are accompanying syndromes. The pseudomembrane forms over the necrotic area at the interdental papilla and, once removed, exposes the connective tissue with its nerve endings, leading to bleeding and severe pain[5]. The present cases demonstrated not only necrosis, ulceration with pseudomembane, spontaneous bleeding and severe pain at the interdental papilla, but also halitosis and fever.

At the beginning of the 20th century, many scholars persisted in the belief that bateriologic smear and microscopic examination from oral lesions were essential to accurate ANUG diagnosis[15]. However, in 1945, a research commission of the American Dental Association suggested that one could not diagnose ANUG simply by smear test without confirmation of clinical findings and Barnes et al.[16] posited that because ANUG's characteristic signs distinguish it from other diseases, it could be definitively diagnosed based only on clinical findings. This argument is supported by the fact that the microbes associated with ANUG can be found not only in healthy people but also in patients with gingivitis and periodontitis; therefore, microbiological testing alone would not provide definitive diagnostic information[9,17]. Taken together, a diagnosis of ANUG could be based on characteristic clinical findings with additional microbiological testing or biopsy[2]. For the diagnosis of the present cases, clinical examination to find characteristic signs and laboratory tests to exclude the possibility of other systemic infections were performed before immediate treatment. However, additional microscopic examination for identification of microbial type would be useful to provide a more definitive diagnosis.

Acute herpetic gingivostomatitis (AHG) should be considered for differential diagnosis with ANUG, though it usually has a preceding, prodromal stage marked by fever and malaise. Above all, AHG is not a bacterial-infection-based disease but a viral-infection-based one[18]. Within days, multiple vesicles appear at the gingiva, buccal mucosa, palate, tongue and posterior pharynx, becoming ulcers after their rupture. Nevertheless, these lesions can be healed spontaneously without scar formation after five or seven days. The patients discussed in this case report presented with distinct clinical ANUG findings such as ulceration (limited to the free gingiva) and spontaneous bleeding, and did not previously show any prodromal symptoms nor any vesicles within several days. Therefore, the possibility of AHG was excluded.

ANUG can lead to severe tissue destruction and attachment loss, and can develop into necrotizing periodontitis if not properly controlled at the early stage. Nevertheless, it can be managed easily with adequate diagnosis and sequential-phase treatment[2]. The first phase is acute treatment, the purpose of which is to prevent progression of tissue loss by infection and to provide relief for pain. During the first visit, superficial debridement should be performed to remove soft and mineralized deposits; use of a power-driven debridement device such as an ultrasonic scaler, with minimal pressure, is recommended. As complete debridement is difficult to achieve, and also because infection-based symptoms such as fever and malaise are usually present, systemic antibiotics should be considered. Mechanical oral hygiene care should be restricted for this phase, as tooth brushing would delay healing and cause pain. Instead, the use of chemical plaque-control formulations, for example 0.12% to 0.2% chlorhexidine-based mouth rinse or mouthwash such as warm water mixed with 3% hydrogen peroxide solution, is recommended[2].

In the initial treatment phase, the use of chlorhexidine until two weeks is recommended. After a few days, pain in the gingival margin and

Table 1. Schematic diagram of proposed treatment approach

Treatment phase	Oral hygiene care	Mechanical debridement	Chemical rinsing	Systemic antibiotics
Acute phase	X	X	Chlorhexidline	O
Pre-existing condition phase	O	Scaling, root planing	Chlorhexidline	X
Disease sequelae correction phase	O	Gingivectomy periodontal flap	X	X
Maintenance phase	O	Supportive periodontal therapy	X	X

papilla would be reduced, by which time proper tooth cleaning would be possible[19]. A mouthwash with hydrogen peroxide solution is useful in the initial treatment phase because it effectively prevents the colonization of anaerobes[20]. However, it has been demonstrated that mouthwash therapy can improve the clinical picture only slightly, and is not very effective in reducing the number of microbes that cause actual illness[20].

The second treatment phase is the melioration of pre-existing conditions. In this phase, symptoms would be improved after the initial acute phase; accordingly, scaling and root planing along with intense oral hygiene instruction should be initiated. Furthermore, control of systemic factors by, for example, stopping smoking, maintaining sufficient sleep and reducing stress, should be undertaken. The third phase entails the treatment of disease sequelae; specifically, gingivectomy or periodontal flap surgery is performed to correct the remaining superficial or deep craters. Surgical treatment should be considered only if deemed necessary after proper oral hygiene establishment. The fourth and final treatment phase proceeds by supportive periodontal therapy to maintain oral hygiene and control of systemic factors[2].

The two patients discussed in this report rejected superficial debridement and mouthwashes with hydrogen peroxide solution on the initial visit due to the resultant severe pain. Alternatively, the restriction of mechanical brushing and chlorhexidine for mouth rinsing, along with systemic antibiotics, were prescribed. Two days later, after pain and bleeding disappeared and the size of the ulcers was reduced, supragingival scaling and root planing could be performed. Seven days later, additional subgingival root planing was performed, and continuous supportive periodontal therapy was scheduled (Table 1).

In both of the cases discussed in this report, proper diagnosis was followed by marked improvement of their condition, which was achieved by simple sequential treatment. In modern society, ANUG is not a common disease; still, where it arises, it can be easily managed with proper diagnosis and immediate, sequential treatment.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Acknowledgement

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