Journal of the International Academy of Periodontology (2023) **25/1**: 1-12

DOI: https://doi.org/10.14436/1466-2094.25.1.001-012.oar

Necrotizing gingivitis – diagnosis and treatment: a narrative review and case report

Lília Aguiar Maia[1], Daniele Ferreira Da Cruz[1], Letícia De Lorenzo Abreu[1], Miguel Rojas Caceres[1], César José Da Silva[1], Celeste Urdinaga Hung[1], Magda Feres[1,2], Belén Retamal-Valdes[1,3]

[1]Department of Periodontology, Dental Research Division, Guarulhos University, Guarulhos, São Paulo, Brazil. [2]Department of Oral Medicine, Infection and Immunity, Harvard School of Dental Medicine, Boston, MA, USA. [3]Department of Periodontology, Faculty of Dentistry, Universitas Gadjah Mada, Indonesia.

Abstract

Introduction: Necrotizing periodontal diseases (NPD) are acute lesions with a rapid onset that may cause considerable periodontal tissue destruction. Among these conditions, necrotizing gingivitis (NG) is caused by microorganisms from the oral biofilm, and is associated with predisposing factors such as psychological stress, HIV/AIDS, alcohol and tobacco consumption, malnutrition, inadequate oral hygiene, and other systemic conditions that may compromise the host immune response.

Objective: The aim of this narrative literature review was to describe the available evidence on the diagnosis and treatment of NG, and present a successfully treated case of NG in a 22-year-old woman.

Case report: The patient was smoker, had poor oral hygiene and was experiencing psychological stress. Her complaints were abundant gingival bleeding and halitosis during the past two weeks preceding the first appointment. Clinical findings included considerable biofilm accumulation, generalized gingival bleeding, pseudomembrane and necrotic tissue. Treatment consisted of supragingival scaling, professional prophylaxis, 0.12% chlorhexidine rinsing for two weeks, prescription of analgesics and oral hygiene instructions.

Results: The patient's response to initial therapy was satisfactory. Successful clinical outcomes were observed at seven days after initial therapy, and the patient has remained stable without signs of disease recurrence for six months.

Conclusion: Periodontal health and function were successfully reestablished.

Keywords: Necrotizing gingivitis. Necrotizing periodontal diseases. Periodontal therapy. Periodontal treatment. Case report. Review.

Introduction

Necrotizing periodontal diseases (NPD) are described as acute periodontal lesions, since they have a rapid onset and may lead to substantial destruction of periodontal tissues (American Academy of Periodontology, 2000). NPD are considered the most severe inflammatory periodontal diseases associated with oral biofilm (Herrera *et al*., 2018; Martos *et al*., 2019). The microbial species most commonly associated with these conditions are *Prevotella intermedia*, *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Treponema* and *Selenomonas species* (Novak, 1999; Herrera, *et al*. 2018).

Although these microorganisms play a determinant role in the onset of necrotizing conditions, these are normally associated with predisposing factors. The most relevant predisposing factors are HIV/AIDS, malnutrition, psychological stress, insufficient sleep, inadequate oral hygiene, alcohol consumption, smoking habits and other systemic conditions (Herrera *et al*., 2014). These factors compromise host immune responses and can influence the development and progression of the disease (Herrera *et al*., 2018; Dufty, 2014). A high prevalence of NPD was observed during the Second World War, reinforcing the important correlation between occurrence of the disease and malnutrition, psychological stress and inadequate oral hygiene (Pindborg, 1951; Dufty, 2014).

Some authors have suggested that NPD may have different stages and would start with an initial process of Necrotizing Gingivitis (NG) and then progress to Necrotizing Periodontitis (NP) or to a more severe condition known as Necrotizing stomatitis (Novak, 1999; Herrera *et al*., 2018). NG is commonly described as a rare condition, with the prevalence ranging from < 0.03% to 9.4% (Dufty, Gkranias and Donos, 2017). Since the First and Second World Wars, there has been an overall decline in the prevalence of NG (Dufty, 2014).

Correspondence to: Belén Retamal-Valdes
E-mail: belen.valdes@prof.ung.br

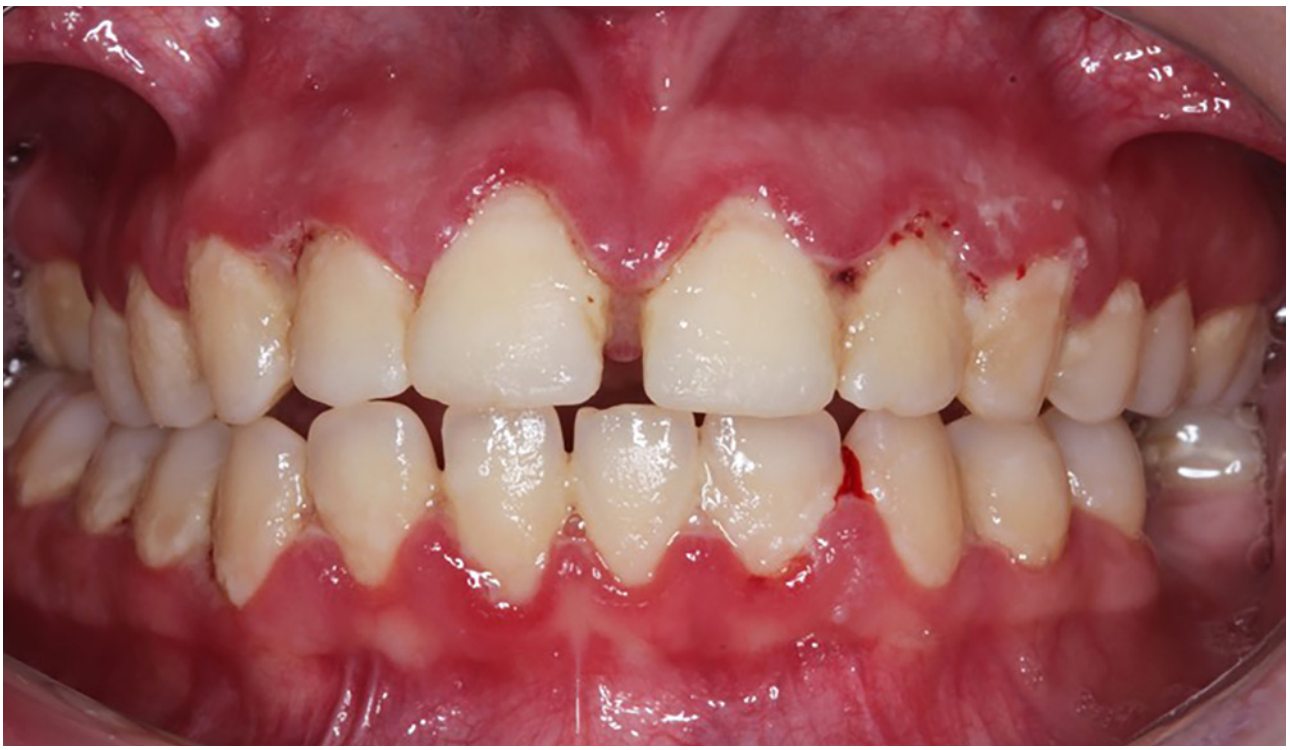

Figure 1. Initial clinical appearance at first evaluation: frontal view.

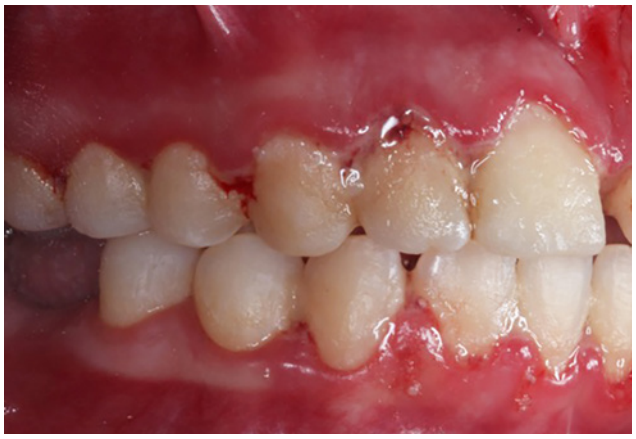

Figure 2. Initial clinical appearance at first evaluation: right lateral view.

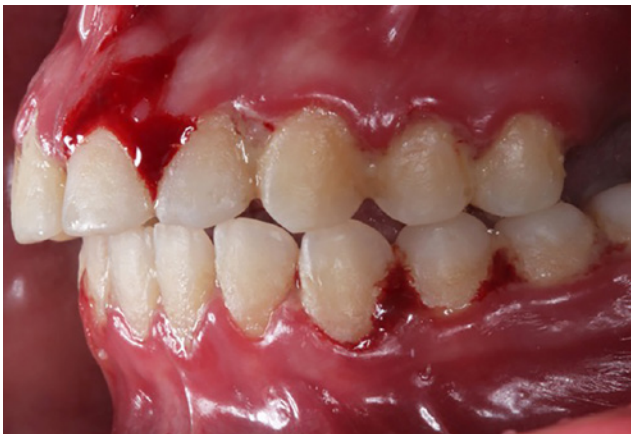

Figure 3. Initial clinical appearance at first evaluation: left lateral view.

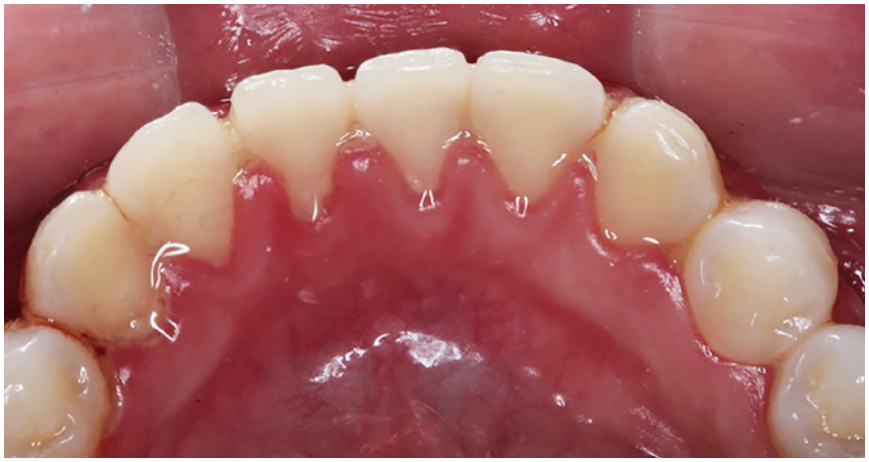

Figure 4. Initial clinical appearance at first evaluation: mandibular occlusal view.

Increasing knowledge about the etiology, diagnosis and treatment of NG is most important in order to provide help with the proper management of this condition in daily clinical practice. Thus, the aim of this narrative literature review was to present the available scientific evidence on the diagnosis and treatment of NG. Furthermore, a successfully treated case of a patient diagnosed with NG will be described.

Literature review

Clinical presentation of NG

A combination of various clinical signs/symptoms may be detected in a NG case. The most relevant are necrosis/ulcer of the interdental papilla, gingival bleeding with little or no provocation, pain or discomfort, pseudomembrane formation and halitosis (Figures 1 - 4) (American Academy of Periodontology, 2000; Herrera *et al*., 2018). Fever, regional lymphadenopathy and malaise are extraoral signs that may or may not be present. Patients suffering from NG usually seek urgent treatment due to intense pain (Dufty, Gkranias and Donos, 2017).

Necrotic and ulcer lesions normally start at the interdental papilla and have a "punched-out" appearance, which can progress to the marginal gingiva of the adjacent teeth. However, the destruction remains limited to the gingival tissues. Therefore, at this stage, no attachment or bone loss are present, since the disease does not affect alveolar bone or periodontal ligament (Herrera *et al*., 2014). Table 1 shows the most relevant clinical findings associated with NG.

Etiopathogenesis of NG

Spirochetes and fusiform bacteria were initially associated with the etiology of NG as far back as 1894 (Plaut, 1894), and 1896 (Rowland, 1999). In 1982, Loesche *et al*. determined that two groups of bacteria could be involved in the etiopathogenesis of this condition: (I) a group of species belonging to a "constant flora", such as *Prevotella intermedia* and species from the genera *Treponema, Fusobacterium* and *Selenomonas*; and (II) species described as the "variable flora", formed by a heterogeneous group of bacteria.

An important aspect of the pathogenesis of NG is the capacity of some microorganisms to invade the host's periodontal tissues, causing necrotic lesions (Rowland *et al*., 1993). Fusiform-spirochete bacteria found in the flora of NG have the capacity to invade the gingival epithelium and viable connective tissue, as well as to release endotoxins that may cause periodontal tissue destruction through activation or modification of the host immune response, confirming the highly pathogenic profile of these microorganisms (Heylings, 1967; Listgarten, 1965; Herrera *et al*., 2014). Due to toxic effects, endotoxins may cause direct destruction of cells and blood vessels. Furthermore, endotoxins can also cause indirect effects by modifying the host inflammatory response, which contributes to more tissue damage in different ways: (I) by acting as antigens and stimulating the host immune response, (II) directly activating the complement system through an alternative pathway and causing release of toxins, or (III) stimulating macrophages, T and B lymphocytes, which stimulates the host immune response and increases the production of cytokines by these cells (Mergenhagen *et al*., 1961; Holmstrup and Westergaard, 2008).

In 1965, while conducting an electronic microscopic investigation on NG damaged tissue, Listgarten found that the elementary histological lesion presented as an ulcer in the stratified squamous epithelium, and that the following four different zones were associated with the gingival lesion: (I) Bacterial zone: composed of a large mass of varying morphotypes of bacteria, including spirochetes; (II) Neutrophil rich zone: underlying the bacterial zone, containing many leukocytes with neutrophils predominating. Bacteria, including many spirochetes, located between the cells; (III) Necrotic zone: presence of disintegrating cells and many spirochetes and other bacteria that appeared to be fusiform; (IV) Spirochetal infiltration zone: tissue elements appeared to be well preserved, but were infiltrated by spirochetes. No other bacteria were observed (Listgarten, 1965; Rowland, 1999).

In the connective tissue, enlarged blood vessels proliferated to form granulation tissue in an abundant leukocytes inflammatory infiltrated area (Holmstrup and Westergaard, 2008). The white-yellow or gray layer observed clinically, appeared to be a fibrin network structure composed of degenerated epithelial cells, leukocytes, erythrocytes, bacteria and cell debris, in the electronic microscopy view (Holmstrup and Westergaard, 2008). Furthermore, the ulcer within the stratified squamous epithelium and the superficial layer of the gingival connective tissue were surrounded by a nonspecific acute inflammatory reaction (Herrera *et al*., 2014).

Table 1. Most relevant clinical findings in NG.

Main clinical findings	Secondary possible clinical findings	Possible extraoral signs
Necrosis/ulcer of the interdental papilla	Pseudomembrane formation	Lymphadenopathy
Gingival bleeding	Halitosis	Fever
Pain or discomfort		Malaise

Predisposing factors

Although the primary etiology of NG is bacterial (Corbet, 2004), predisposing factors might influence the development of the condition by affecting host immune defense mechanisms (Herrera *et al*., 2014). Severe chronic life-threatening diseases (e.g. HIV/AIDS), severe conditions of malnutrition or infections, and other less serious chronic conditions (e.g. smoking habits, emotional stress, inadequate oral hygiene and preexisting gingivitis) have been cited as examples of predisposing factors (Papapanou *et al*., 2018).

In a study conducted in the USA, the most important risk factor for developing NG was found to be HIV infection. Conversely, in non-HIV patients, the most prevalent predisposing factors were previous history of NPD, inadequate oral hygiene, insufficient sleep, emotional stress, poor diet, recent illness, alcohol use, smoking, Caucasian race and age under 21 (Horning and Cohen,1995). In summary, apparently, the most important predisposing factors for NG are those capable of affecting the host immune response, and normally more than one factor is necessary to trigger the onset of disease (Herrera *et al*., 2018).

Classification system

In 1778, Hunter first described specific clinical findings associated with NG as the gum being "swollen and spongy, with ulceration, tenderness and bleeding". He was the first professional to propose a distinction between NG and scurvy. In 1892, however, Vincent was widely accredited with identifying and describing the condition later known as NG (Dufty, Gkranias and Donos, 2017).

As time passed, NG was referred to by various names, such as Vincent's disease, trench-mouth disease, fusospirochete stomatitis, ulcerative membranous gingivitis, acute necrotizing ulcerative gingivitis and necrotizing ulcerative gingivitis (Johnson and Engel, 1986; Armitage, 2002; Herrera *et al*., 2014). The term "acute" was removed from the official nomenclature, since patients were frequently susceptible to future recurrence of disease, which could potentially become a "chronic condition", with a slower rate of destruction (Herrera *et al*., 2018).

In the 1989 World Workshop, the American Academy of Periodontology defined and introduced the terminology Necrotizing Ulcerative Periodontitis and the category known as NPD, which included Necrotizing Ulcerative Gingivitis and Necrotizing Ulcerative Periodontitis (Armitage, 1999; Herrera *et al*., 2014). The current Classification of Periodontal Diseases and Conditions (Caton *et al*., 2018; Papapanou *et al*., 2018), eliminated the term "ulcerative", because ulceration was considered to be secondary to the necrosis, and also proposed specific changes in the classification of NPD, as described in the Table 2 (Herrera *et al*., 2018).

Table 2. Current proposal of classification for NPD (Herrera *et al*., 2018).

Category	Patients	Predisposing conditions	Clinical condition
Necrotizing periodontal diseases in chronically, severely compromised patients	In adults	HIV+/AIDS with CD4 counts < 200 and detectable viral load	NG, NP, NS, Noma. Possible progression
		Other severe systemic conditions (immunosuppression)	
	In children	Severe malnourishment[a]	Not described
		Extreme living conditions[b]	
		Severe (viral) infections[c]	
Necrotizing periodontal diseases in temporarily and/or moderately compromised patients	In gingivitis patients	Uncontrolled factors: stress, nutrition, smoking, habits	Generalized NG. Possible progression to NP
		Previous NPD: residual craters Local factors: root proximity, tooth malposition	Localized NG. Possible progression to NP
	In periodontitis patients	Common predisposing factors for NPD (see paper)	NG. Infrequent progression
			NP. Infrequent progression

NG= necrotizing gingivitis; NP= necrotizing periodontitis; NS= necrotizing stomatitis.
[a] Mean plasma and serum concentrations of retinol, total ascorbic acid, zinc, and albumin markedly reduced, or very marked depletion of plasma retinol, zinc, and ascorbate; and saliva levels of albumin and cortisol, as well as plasma cortisol concentrations, significantly increased.
[b] Living in substandard accommodations, exposure to debilitating childhood diseases, living near livestock, poor oral hygiene, limited access to potable water and poor sanitary disposal of human and animal fecal waste.
[c] Measles, herpes viruses (cytomegalovirus, Epstein-Barr virus-1, herpes simplex virus) chicken pox, malaria, febrile illness.

Treatment

The main objective of NG therapy is to interrupt disease progression and tissue destruction, and eliminating, or at least minimizing the patient's discomfort and pain. The treatment protocol is normally divided into four stages: treatment of the acute phase, treatment of any preexisting periodontal condition, corrective treatment for sequelae of the disease, and the supportive or maintenance phase (Holmstrup and Westergaard, 2008; Herrera *et al*., 2014).

The acute phase of the treatment includes careful superficial debridement, use of chemical formulations such as chlorhexidine-based mouthrinses (0.12-0.2%, twice daily), and re-establishment of effective oral hygiene methods for oral biofilm control (Herrera *et al*., 2014).

Prescription of analgesics may be necessary to help control symptoms of the acute phase, in addition to orally administrated antibiotics, when symptoms suggest systemic involvement or for patients who do not respond well to conventional therapy (Herrera *et al*., 2014; Özberk *et al*., 2018; Martos *et al*., 2019). Among all of the systemic antibiotics, metronidazole seems to be the first choice, due to its effectiveness against strict anaerobes (Martos *et al*., 2019). While signs and symptoms of the acute phase persists, patients must be seen at intervals of 24-48 hours for a thorough mechanical debridement, which may be more effectively performed as the patient's tolerance to pain improves (Klokkevold and Carranza, 2016). Once the acute phase has been controlled, especially considering that many cases may be associated with a preexisting periodontal condition, this is the time point when conventional periodontal therapy must be performed. This phase of treatment includes full-mouth professional prophylaxis and supragingival scaling, associated with reduction or elimination of local and systemic predisposing factors. Continuous motivation and reinforcement of adequate oral hygiene instructions are necessary steps to prevent recurrence of the disease (Horning and Cohen, 1995; Herrera *et al*., 2014).

If necessary, gingival surgical procedures may be performed to correct possible sequelae of the disease. Subsequently, the maintenance phase must begin, with the main objective of controlling predisposing factors and assuring patient's compliance with effective oral hygiene practices (Albandar, 2014). Because this condition is frequently associated with host susceptibility, rapid elimination of factors that could affect development and progression of the disease is essential, and multiprofessional treatment together with the physician is usually required (Marty *et al*., 2016).

Case report

Patient information and timeline

The patient, a 22-year-old woman, school supervisor, resident in Guarulhos-SP (Brazil), was referred for treatment at the Department of Periodontology, School of Dentistry, Dental Research Division, at Guarulhos University (Guarulhos, SP, Brazil). Her main complaints were marked gingival bleeding and halitosis, during the two weeks preceding her first appointment at the University.

During anamnesis, it was detected that the patient was under a high workload and subjected to pressure at work, which was causing psychological stress problems. No systemic conditions were reported, however, the patient informed that she had developed smoking habits during the past four years, and smoked about five cigarettes per day. With regard to her family medical history, she reported that her mother had type 2 diabetes mellitus. Relative to oral self-care, the patient informed that she brushed her teeth twice a day and did not use any other complementary oral hygiene approaches, such as dental floss.

Clinical findings

During extraoral examination, no clinical problems were found. Whereas intraoral examination revealed poor oral hygiene characterized by generalized oral biofilm accumulation and gingival bleeding, accompanied by a grayish pseudomembrane formation, necrosis and ulceration of the interdental papilla and marginal gingiva. These conditions, observed especially in the buccal areas of mandibular and maxillary teeth (Figures 1-3), were already being propagated to the lingual areas of the lower dental arch (Figure 4). The complete periodontal examination was not performed at the first appointment because the patient was feeling generalized discomfort and presented gingival bleeding.

Diagnostic assessment

The patient presented a recent panoramic radiograph that revealed the absence of teeth #16 and #36 (Figure 5). Complementary laboratory exams were requested (coagulation time, complete blood count, fasting glycemia, glycated hemoglobin and anti-HIV antibodies); however, no changes were detected (Table 3). Therefore, based on the clinical findings, emotional stress that the patient was undergoing, and her smoking habit, the diagnosis of NG was defined.

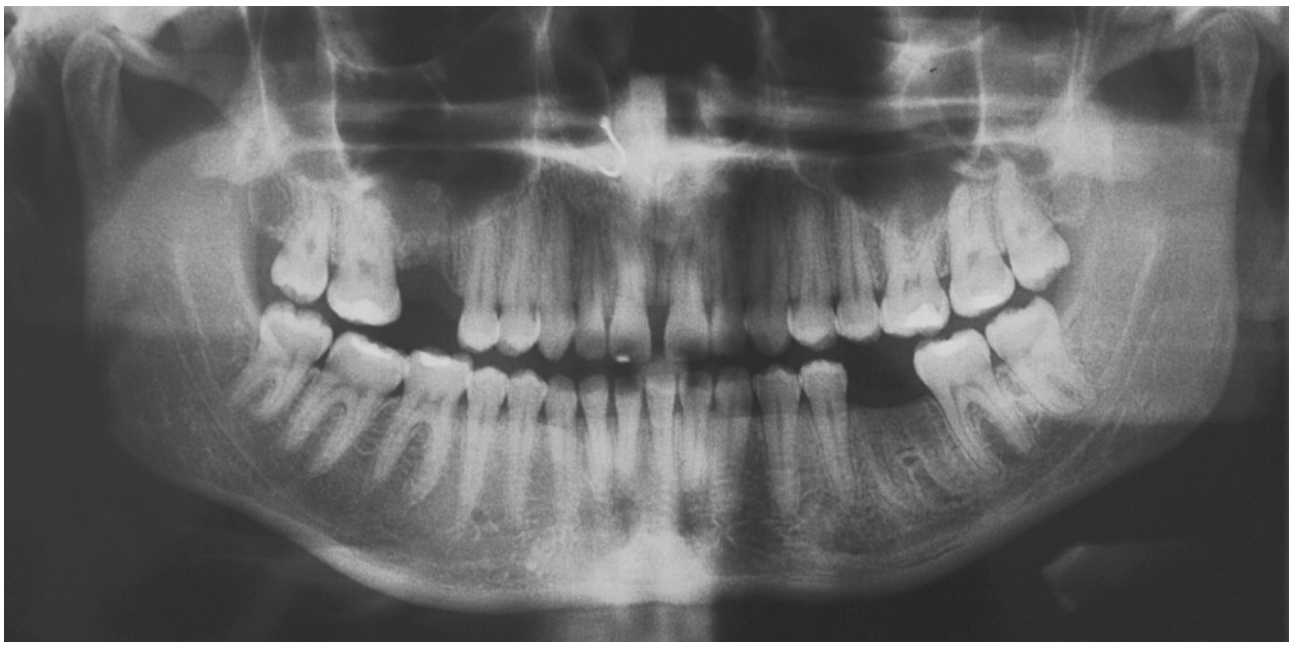

Figure 5. Panoramic radiograph.

Table 3. Complementary laboratory exams.

Erythrogram	Results
Erythrocytes	4,33 millions/mm³
Hemoglobin	13,7 g/dL
Hematocrit	38,7 %
MCV	89,51 fL
MCH	31,67 pg
MCHC	35,38 g/dL
RDW	11,4 %
Leukogram	
Leukocytes	9300 / mm³
Promyelocytes	0
Myelocytes	0
Metamyelocytes	0
Eosinophils	2 – 186 %
Basophils	1 – 93 %
Lymphocytes	44 – 4092 %
Monocytes	6 – 558 %
Platelets	316000 / mm³
MPV	5,2 fL
Coagulation exams	
Activity	83%
INR	1,09 seconds
KPTT	37,8 seconds
Fasting glycemia exam	91 mg/dL
Glycated hemoglobin	5,4%
Anti-HIV antibodies	Non-reactive

MCV= Mean corpuscular volume; MCH= Mean corpuscular hemoglobin; MCHC= Mean corpuscular hemoglobin concentration; RDW= Red cell distribution width; MPV= Mean platelet volume; INR= International normalized ratio; KPTT= Kaolin partial thromboplastin time.

Therapeutic intervention

Initial therapy was divided into two appointments, at seven-day intervals. At the first appointment, after anamnesis, radiographic and clinical examinations, treatment of the acute phase began. Initially, the patient was instructed about the features of the disease and different steps involved in the treatment.

Afterward, careful full-mouth supragingival scaling and professional prophylaxis were performed, associated with 0.12% chlorhexidine gluconate irrigation (Figure 6). Moreover, the patient was instructed about the importance of efficient daily oral hygiene, followed by demonstration of an adequate mechanical oral hygiene technique, using a soft toothbrush, dental floss, conical interdental brushes and 0.12% chlorhexidine gluconate swabs. The patient was also advised to rinse with 0.12% chlorhexidine-based mouthrinse, twice daily for 14 days, and was prescribed 750 mg paracetamol, every 6 h, for 3-5 days to help pain control.

At the second appointment, 7 days after the first intervention, professional full-mouth prophylaxis and complete periodontal examination were performed (Figure 7). Marginal gingival bleeding index, visible plaque index, probing depth, clinical attachment level and bleeding or suppuration on probing were recorded. No tooth mobility or furcation lesions were detected. At this stage, it was confirmed that the disease was limited to the marginal gingival tissues, since no attachment or bone loss were observed. These findings confirmed that it was not a case of necrotizing periodontitis. The patient was informed about the relationship between cigarette smoking and periodontal diseases, as well as the substantial influence that psychological stress may have on the development of NG, along with reinforcement of the oral hygiene instructions.

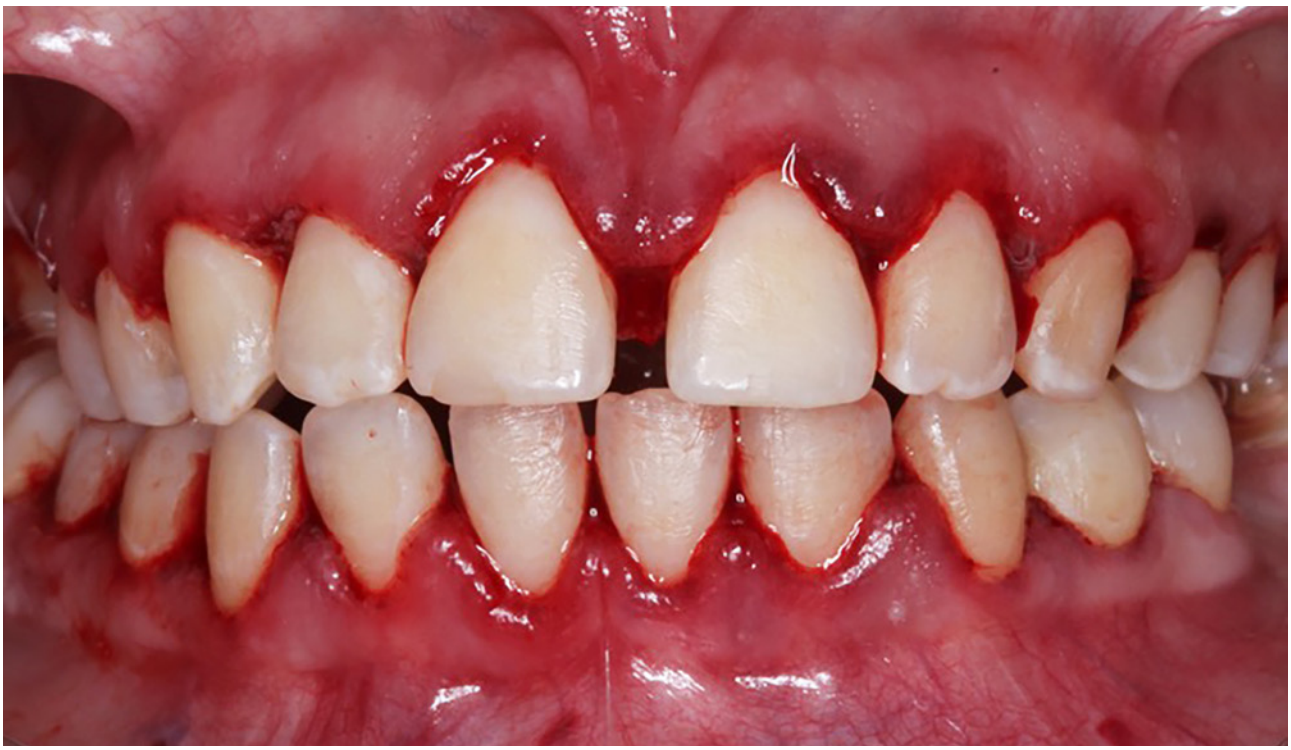

Figure 6. Clinical appearance immediately after initial therapy during first appointment.

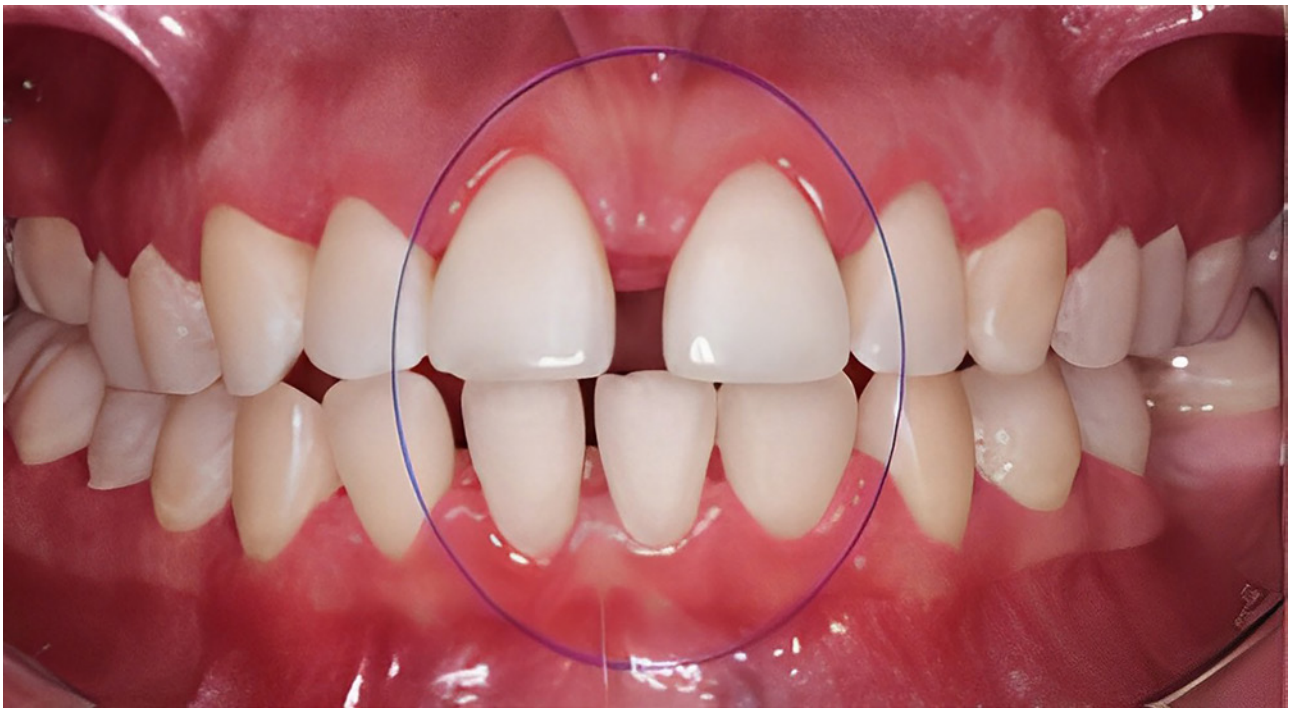

Figure 7. Clinical appearance 7 days after initial therapy.

Follow-up and outcomes

Follow-up appointments were scheduled at four weeks (Figure 8), three months (Figure 9) and six months (Figure 10) after the initial therapy. The main aims of these appointments were: to avoid recurrence of the disease, evaluate patient's adherence to treatment and reinforce the importance of maintaining adequate oral hygiene. Complete periodontal examinations were performed at each visit, and the data are described in Table 4. The patient's periodontal health and function were successfully maintained over time.

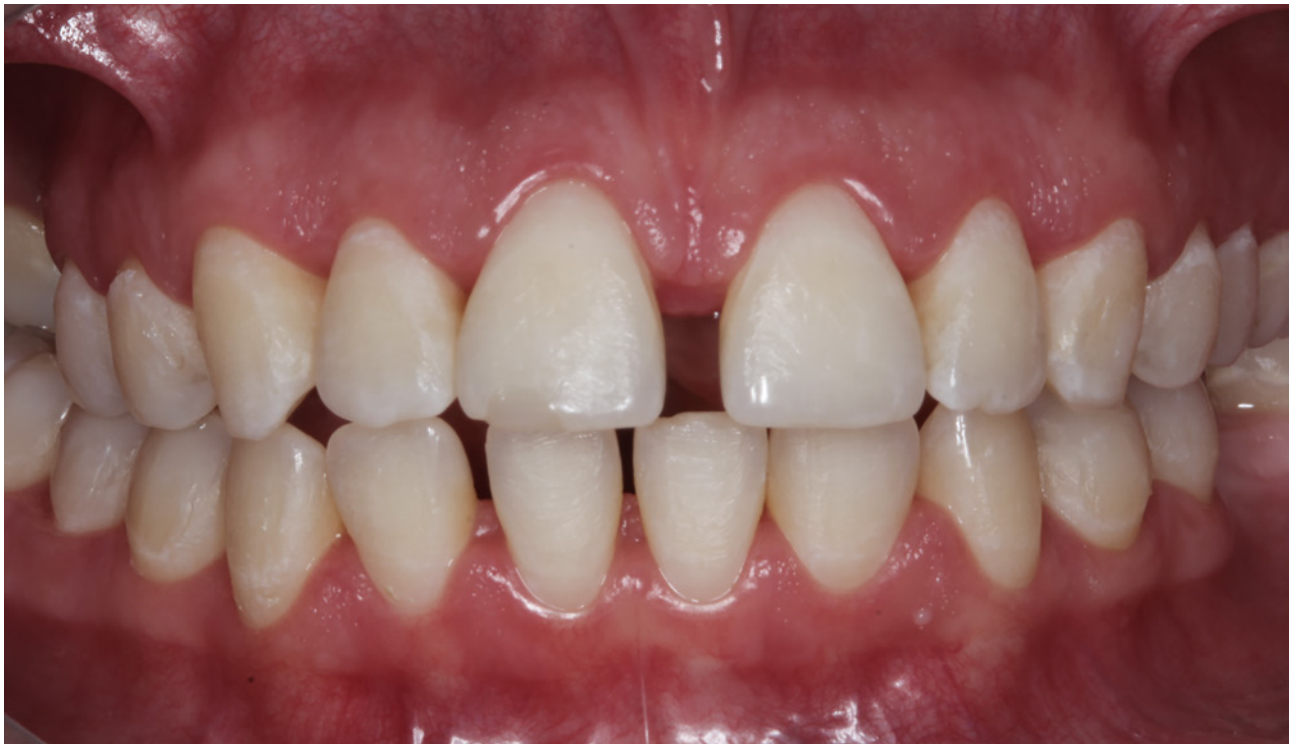

Figure 8. Clinical appearance four weeks after initial therapy.

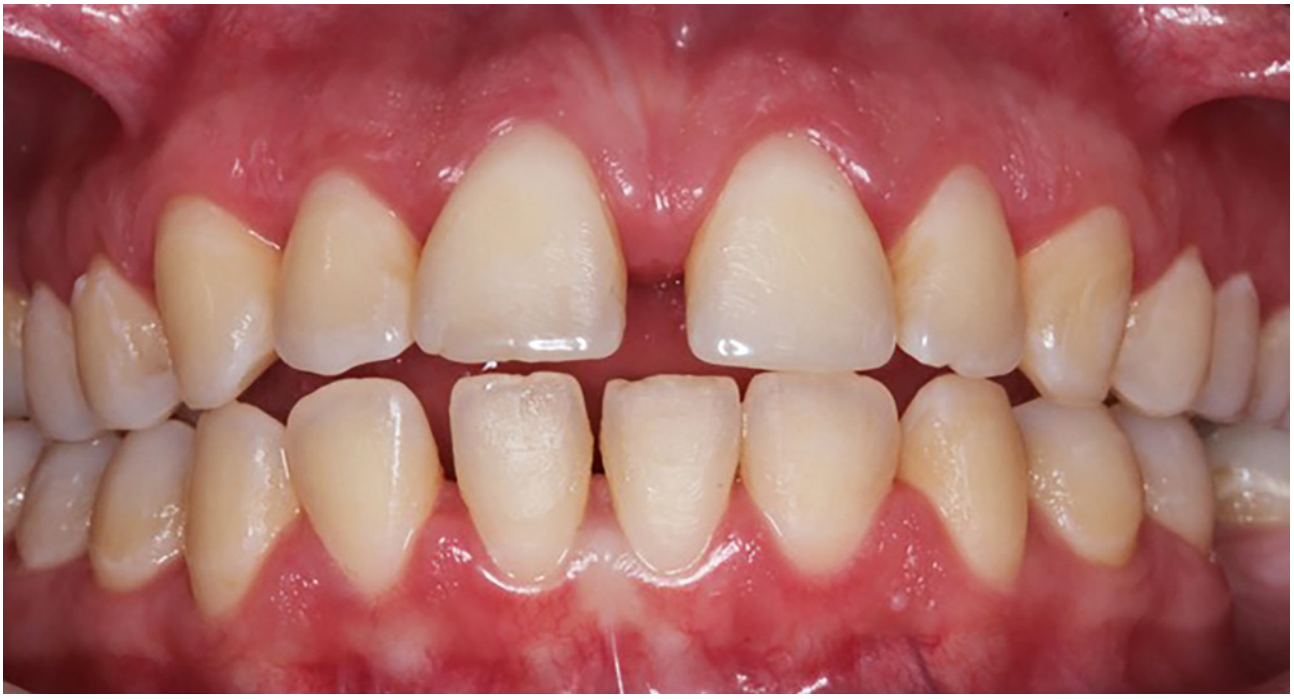

Figure 9. Clinical appearance three months after initial therapy.

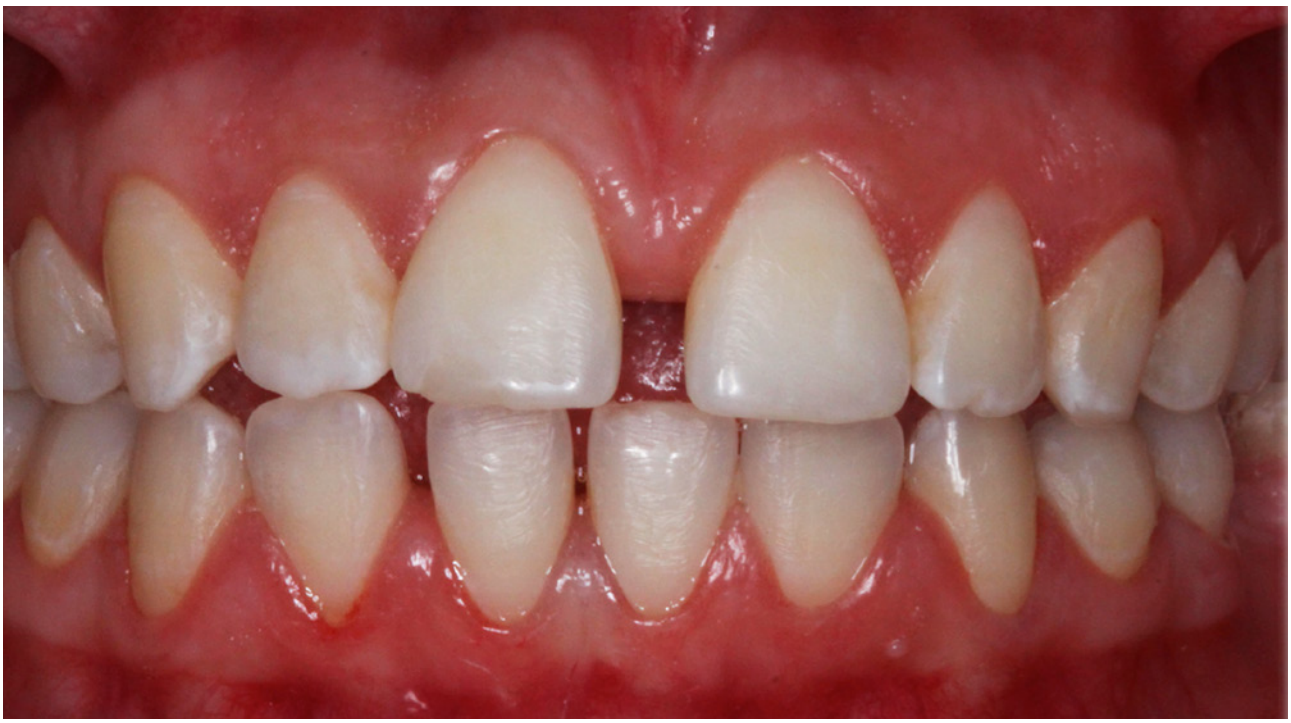

Figure 10. Clinical appearance six months after initial therapy.

Table 4. Means of full-mouth clinical parameters at 7 days and 1, 3 and 6 months post-treatment.

Time-point	Periodontal parameters					
	VPI	GBI	Mean PD	Mean CAL	BOP	SUP
7 days	31,41%	68,59%	2,44 mm	0,00 mm	78,21%	0%
1 month	19,41%	10,48%	2,20 mm	0,00 mm	18,15%	0%
3 months	22,41%	12,49%	2,33 mm	0,00 mm	20,15%	0%
6 months	25,61%	17,33%	2,40 mm	0,00 mm	21,15%	0%

VPI= Visible plaque index; GBI= Gingival bleeding index; PD= Probing depth; CAL= Clinical attachment level; BOP= Bleeding on probing; SUP= Suppuration on probing.

Discussion

NG is ranked among the most severe inflammatory conditions associated with oral biofilm microorganisms (Herrera *et al*., 2014). Although its primary etiological factor is the presence of dental biofilm, predisposing factors such as smoking and compromised host immune responses are critical factors in the pathogenesis of this condition (Corbet, 2004; Herrera *et al*., 2018). The patient of this case report presented several conditions considered to be risk factors for NG (Horning and Cohen, 1995; Herrera *et al*., 2018; Papapanou *et al*., 2018). She had poor oral hygiene, was under psychological stress and had smoking habits.

The patient was warned about the importance of improving oral hygiene and the harmful effects of cigarette smoking and stress on her oral and systemic health. However, controlling smoking and stress is a very difficult task. The persistence of these factors increases patient susceptibility to future disease recurrence, and the risk is increased even further if patient reverts to poor oral hygiene habits (Herrera *et al*., 2014). Tobacco consumption interferes with the functions of both lymphocytes and polymorphonuclear leukocytes, while nicotine induces vasoconstriction in gingival blood vessels, hampering tissue revascularization and wound healing (Rowland *et al*., 1999; Herrera *et al*., 2014). Furthermore, psychological stress tends to affect subject's immune response by increasing corticosteroids and periodontal pathogen levels in the organism, reducing salivary flow and gingival microcirculation, and altering the functions of lymphocytes and polymorphonuclear leukocytes (Loesche and Laughon, 1982; Herrera *et al*., 2018).

The choice of treatment for this case combined mechanical and chemical procedures, including supra and subgingival scaling, use of chlorhexidine 0.12% mouth rinses, implementing oral hygiene instructions and prescription of analgesics. A similar treatment protocol was used in a case reported by Martos *et al.* (2019), in which

periodontal health was maintained during a period of follow-up of up to 10 years. At-home use of 0.12% chlorhexidine gluconate mouth rinse was recommended twice a day for 30 days. Malek *et al.* (2017) also achieved clinical success in the management of NG using a protocol that included a combination of treatments. The authors prescribed systemic antibiotics (250 mg metronidazole every 8h for 7 days) combined with irrigation of the necrotic pseudomembranous lesions with hydrogen peroxide and supragingival debridement, altogether at the first visit. In this particular case, two months after this initial approach, gingivectomy was necessary in order to obtain a more symmetrical and homogeneous architecture of the gingival tissue. On the other hand, Özberk *et al.* (2018) reported a case in which they chose to first prescribe oral antibiotics (500 mg amoxicillin and 400 mg metronidazole 3 times a day for 5 days) and chlorhexidine 0,2% mouthwash twice a day; and to perform mechanical treatment only in the second visit. On these last two cases reported, the patients were experiencing severe pain before the first appointment.

An interesting case-control study was carried out by Dufty *et al.* (2017) with the military population from the British Armed Forces that presented necrotizing ulcerative gingivitis (NUG). Most of the cases received the following treatment: oral hygiene instruction (66.5%), systemic antibiotics (64.4%) and mouthwash (58.1%). Of the selected cases, 48.7% received debridement, analgesics were prescribed in 8.4% of the cases and 10.7% of the patients were instructed to stop smoking. This study described a NUG prevalence of 0.11% in the British Armed Forces. A strong association between NUG and smoking was demonstrated, and it was determined that both oral hygiene and smoking habits must be carefully assessed and controlled during treatment.

The fact that the patient from the present report had no chronic disease and showed no systemic symptoms, such as fever or malaise, at the first appointment, supported the decision of not prescribing antibiotics. There is consensus in literature that orally administrated antibiotics should only be prescribed as an adjunctive treatment of NG in cases associated with evident systemic involvement or acute pain (Herrera *et al*., 2014; Özberk *et al.*, 2018; Martos *et al*., 2019). Maintenance therapy was also implemented, and no recurrence of the disease was observed up to six months post-treatment. This is a very important step of NG treatment, because during these maintenance appointments the dentist has the opportunity to evaluate the patient's oral hygiene procedures, and to talk about the influence of patient's behaviors — especially with regards to predisposing factors, such as stress and smoking. Professional supportive maintenance therapy is an essential element for achieving long-term periodontal health (Martos *et al*., 2019).

Strengths and limitations associated with this case report

Considering that NG is an acute lesion that normally has a very rapid onset, it may lead to a rapid destruction of periodontal tissues and is often associated with pain. Thus, NG requires urgent attention from dental-care providers (Albandar, 2014; Sangani *et al*., 2013). Based on the foregoing premise, one strength of the case presented in this manuscript was the prompt treatment implemented at the first appointment. This is in agreement with other authors in the literature, who highlighted the importance of an early intervention in cases of NG (Corbet, 2004; Herrera *et al*., 2014; Martos *et al*., 2019). The main limitation of the case presented here was the difficulty in controlling the predisposing factors such as smoking habits and emotional stress. In fact, this limitation is applicable to the treatment of many of NG cases in daily clinical practice.

Discussion of additional relevant literature

Diagnosis of NG must be based on three typical clinical features: necrosis/ulcer of the interdental papilla, gingival bleeding and pain (Papapanou *et al*., 2018). Possible secondary clinical findings might also support a correct diagnostic of NG cases, such as halitosis, presence of pseudomembrane, metallic taste, increased and viscous salivary flow, mobility of teeth, regional lymphadenopathy, fever and malaise (Barnes, Bowles and Carter, 1973).

In a study by Horning and Cohen (1995), 65 HIV-positive patients were evaluated, and the percentage of the secondary clinical features found were as follows: pseudomembrane formation (88%), halitosis (87%), lymphadenopathy (44%) and fever (24%). Herrera *et al*. (2018) reported that the most relevant clinical characteristics found in studies assessing NG were: necrosis and ulcer in the interdental papilla (94-100%), gingival bleeding (95-100%), pain (86-100%), pseudomembrane formation (73-88%), halitosis (84-97%), adenopathy (44-61%) and fever (20-39%). Pseudomembrane and halitosis were observed in the present case report.

The relatively constant set of bacteria most commonly found in NG are *Prevotella intermedia* and species from the genera *Selenomonas, Treponema and Fusobacterium* (Horning and Cohen, 1995). Regarding histological features, NG lesions observed under light microscopy showed the presence of an ulcer within the stratified squamous epithelium and the superficial layer of the gingival connective tissue, surrounded by a nonspecific acute inflammatory reaction. Because tools for rapid microbiological or histological diagnosis are not pathognomonic of the disease and are not always available at dental practices, the diagnosis of NG should be based on clinical findings. Microbiological or biopsy evaluation should only be assessed in atypical presentations or non-responding cases (Hooper and Seymour, 1979; Herrera *et al*., 2018).

A variety of conditions may resemble NG and require complementary measures for differential diagnoses, such as: primary herpetic gingivostomatitis, aphthous stomatitis, erythema multiforme, traumatic ulcer, desquamative gingivitis, infectious mononucleosis, acute leukemia, agranulocytosis, secondary syphilis and allergic stomatitis (Horning and Cohen, 1995). In the present case report, no microbiological or biopsy assessments were necessary.

Lessons of this case report

The main lessons learned with this case report refer to the treatment protocol chosen, in particular: (i) prompt implementation of mechanical treatment, (ii) the choice of using chlorhexidine rinsing, (iii) the choice of not using systemic antibiotics, (iv) implementation of a maintenance phase, with follow-up appointments up to six months after active treatment.

Final thoughts and conclusion

Despite being considered a condition with low-prevalence, NG can lead to rapid and severe gingival tissue destruction, and may progress to more destructive stages of the disease if it is not promptly treated. Furthermore, NG is ranked among the most severe biofilm-related periodontal conditions. There are three typical clinical features associated with NG, namely: necrosis/ulcer of the interdental papilla, gingival bleeding and pain. Its primary etiological factor is bacteria present in the oral biofilm. However, predisposing factors that can affect host immune defense mechanisms might play a very important role in the development of NG. The aim of the treatment is to interrupt the rapid tissue destruction and to reverse the inflammatory process associated with the disease. Moreover, constant motivation and reinforcement of adequate oral hygiene instructions are key points to avoid disease recurrence.

In general, if promptly and correctly treated, NG may have a good prognosis, as occurred in the case reported in this study. The therapy performed in the case reported here was successful and effective for maintaining the patient's periodontal health during the six-month period of follow-up.

Acknowledgments and Conflict of Interest

The authors acknowledge the Brazilian Coordination for the Improvement of Higher Education Personnel (CAPES), the National Council for Scientific and Technological Development (CNPq, Brazil) and Latin American Oral Health Association (LAOHA), for supporting some researchers involved in this study. The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

References

American Academy of Periodontology. Parameter on acute periodontal diseases. *Journal of Periodontology 2000*; **71**:863-866.

Albandar JM. Aggressive and acute periodontal diseases. *Periodontology 2000* 2014; **65**:7-12.

Armitage GC. Development of a classification system for periodontal diseases and conditions. *Annals Periodontology* 1999: **4**:1-6.

Armitage GC. Classifying periodontal diseases - a long-standing dilemma. *Periodontology 2000* 2002; **30**: 9-23.

Barnes GP, Bowles WF 3rd and Carter HG. Acute necrotizing ulcerative gingivitis: a survey of 218 cases. *Journal of Periodontology* 1973; **44**:35-42.

Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, *et al*. A new classification scheme for periodontal and peri-implant diseases and conditions - Introduction and key changes from the 1999 classification. *J Clin Periodontol* 2018;**45** Suppl 20:S1-S8.

Corbet EF. Diagnosis of acute periodontal lesions. *Periodontology 2000* 2004; **34**:204-216.

Dufty JR. Report for the pathological committee of the war office of an inquiry into gingivitis and Vincent's disease occurring in the Army. *Journal of the Royal Army Medical Corps 2014*; **160** Suppl 1:i7-i8.

Dufty J, Gkranias N and Donos N. Necrotising ulcerative gingivitis: a literature review. *Oral Health Preventive Dentistry* 2017; **15**:321-327.

Dufty J, Gkranias N, Petrie A, McCormick R, Elmer T, Donos N. Prevalence and treatment of necrotizing ulcerative gingivitis (NUG) in the British Armed Forces: a case-control study. *Clin Oral Investig* 2017; **21**:1935-1944.

Herrera D, Alonso B, de Arriba L, Santa Cruz I, Serrano C and Sanz M. Acute periodontal lesions. *Periodontology 2000* 2014; **65**:149-177.

Herrera D, Retamal-Valdes B, Alonso B and Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *Journal of Periodontology* 2018; **1**:85-102.

Heylings RT. Electron microscopy of acute ulcerative gingivitis (Vincent's type). Demonstration of the fusospirochaetal complex of bacteria within pre-necrotic gingival epithelium. *British Dental Journal* 1967; **122**:51-56.

Holmstrup P and Westergaard J. Necrotizing periodontal disease. In: Lindhe J, Lang NP and Karring T (Eds): *Clinical Periodontology and Implant Dentistry. Oxford*. Blackwell Munksgaard, 2008.

Hooper PA and Seymour GJ. The histopathogenesis of acute ulcerative gingivitis. *Journal of Periodontology* 1979; **50**:419-423.

Horning G and Cohen M. Necrotizing ulcerative gingivitis, periodontitis, and stomatitis: clinical staging and predisposing factors. *Journal of Periodontology* 1995; **66**:990-998.

Johnson BD and Engel D. Acute necrotizing ulcerative gingivitis. A review of diagnosis, etiology and treatment. *Journal of Periodontology* 1986; **57**:141-150.

Klokkevold PR and Carranza FA. Treatment of acute gingival disease. In Newman MG, Takei HH, Klokkevold PR and Carranza FA (Eds): *Carranza's Clinical Periodontology*. Philadelphia. Elsevier, 2016.

Listgarten MA. Electron microscopic observations on the bacterial flora of acute necrotizing ulcerative gingivitis. *Journal of Periodontology* 1965; **36**:328-339.

Loesche WJ, Syed SA, Laughon BE and Stoll J. The bacteriology of acute necrotizing ulcerative gingivitis. *Journal of Periodontology* 1982; **53**:223-230.

Loesche WJ and Laughon BE. Role of spirochetes in periodontal disease. In: Genco RJ, Mergenhagen SE. (Eds): *Host-parasite interactions in periodontal diseases*. Washington, DC. American Society for Microbiology, 1982.

Malek R, Gharibi A, Khlil N and Kissa J. Necrotizing ulcerative gingivitis. *Contemporary Clinical Dentistry* 2017; **8**:496-500.

Martos J, Ahn Pinto KV, Feijó T, *et al*. Clinical treatment of necrotizing ulcerative gingivitis: a case report with 10-year follow-up. *General Dentistry* 2019; **67**:62-65.

Marty M, Palmieri J, Noirrit-Esclassan E, Vaysse F and Bailleul-Forestier I. Necrotizing periodontal diseases in children: a literature review and adjustment of treatment. *Journal of Tropical Pediatrics* 2016; **62**:331-337.

Mergenhagen SE, Hampp EG and Scherp HW. Preparation and biological activities of endotoxins from oral bacteria. *The Journal of Infectious Diseases* 1961; **108**:304-310.

Novak MJ. Necrotizing ulcerative periodontitis. *Annals of Periodontology* 1999; **4**:74-78.

Özberk SS, Gündoğar H, Şenyurt SZ and Erciyas K. Adjunct use of low-level laser therapy on the treatment of necrotizing ulcerative gingivitis: a case report. *Journal of Lasers in Medical Sciences* 2018 **9**:73-75.

Pindborg JJ. Gingivitis in military personnel with special reference to ulceromembranous gingivitis. *Odontol Tidskr* 1951; **59**:403-499.

Plaut HC. Bacterial diagnostic studies of diphtheria and oral diseases. (Studien zur bakteriellen Diagnostik der Diphtherie und der Anginen). *Deutsch Med Wochnschr* 1894; **20**:920-923.

Rowland RW, Mestecky J, Gunsolley JC and Cogen RB. Serum IgG and IgM levels to bacterial antigens in necrotizing ulcerative gingivitis. *Journal of Periodontology* 1993; **64**:195-201.

Rowland RW. Necrotizing ulcerative gingivitis. *Annals of Periodontology* 1999; **4**:65-73.

Sangani I, Watt E and Cross D. Necrotizing ulcerative gingivitis and the orthodontic patient: a case series. *Journal of Orthodontics* 2013; **40**:77-80.

The American Academy of Periodontology. Proceedings of the World Workshop in Clinical Periodontics. Chicago: *The American Academy of Periodontology*; 1989: I/23-I/24.

Papapanou PN, Sanz M, Buduneli N, *et al*. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *Journal of Periodontology* 2018; **89**:173-182.