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Necrotising periodontal diseases*

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KEY WORDS *necrotising gingivitis, necrotising periodontitis, necrotising stomatitis, primary herpetic gingivostomatitis*

The aetiology of necrotising periodontal diseases is still unknown. However, specific microorganisms and a weakened immune system seem to play a major role in the disease pathogenesis. The clinical pattern for necrotising gingivitis is characterised by painful, necrotic papillae. Initially, the necroses are restricted to the tip of the papillae. However, they then spread rapidly to the apical tissue, causing attachment loss. The condition is then considered to be necrotising periodontitis. A differential diagnosis reveals primary herpetic gingivostomatitis caused by the herpes simplex virus. If possible, the teeth should be brushed carefully during the first appointment and chemical plaque inspection should be carried out using oral rinses. In cases of impaired ingestion or if general health is affected, the use of systemic antibiotics such as metronidazole is indicated. Periodontal treatment with scaling and root planing should initially be carried out once the acute symptoms have abated.

■ Introduction

Necrotising gingivitis, necrotising periodontitis and necrotising stomatitis number among the most serious periodontal diseases. Triggered by bacteria, they almost always go through an acute phase. Therefore the diagnosis is often preceded by the term 'acute'. Literature does not always provide a clear distinction between necrotising gingivitis and necrotising periodontitis; following the non-necrotising form of gingivitis, a diagnosis of 'necrotising gingivitis' should only be made in cases when no attachment loss has occurred[1]. Due to the fact that this disease is mainly combined with attachment loss, in such cases it often concerns a necrotising periodontitis[2]. Further progression of the disease with necrosis beyond the mucogingival junction is characteristic of necrotising stomatitis and distinguishes this from necrotising periodontitis[3]. A much more severe disease, cancrum oris, which is also referred to as noma has some disease characteristics in common with nectrotising periodontitis. This devastating, necrotising and often terminal stomatitis features the same fusospirochetal flora as necrotising periodontitis. It almost exclusively

* Lecture within the framework of the Annual Conference of the New Working Group in Periodontology (NAgP) on 23.09.2006 in Frankfurt/Main

emerges in developing countries and mainly affects children already suffering from systemic diseases and malnutrition[4,5]. Emslie[6] has suggested that noma always develops where there is previously existing necrotising gingivitis. However it has not been possible to confirm this by means of other studies[7-9].

■ Aetiology

The aetiology of necrotising gingivitis and periodontitis is still unknown[10]. However, specific microorganisms[11] and weakened immune defences[12] seem to play a major role in the pathogenesis of necrotising periodontal diseases. Loesche et al[11] indicate that the microorganisms *Treponema* sp, *Selenomonas* sp, *Fusobacterium* sp and *Prevotella intermedia* can always be isolated from lesions in patients with necrotising periodontal diseases, which suggests a possible pathogenetic role of these bacteria[1]. Despite the fact that in some studies it was possible to show the presence of characteristic bacterial flora of spirochetes and fusobacteria in numerous necrotic lesions, their primary aetiological role is unclear[13-15], as this could also relate to a secondary infection.

Studies pertaining to the microbiology in HIV-seropositive patients revealed that the microbiological flora of HIV-associated periodontitis and conventional periodontitis are very similar[16-18]. There has also been discussion of a causal connection between necrotising gingivitis in HIV-seropositive patients and *Candida albicans* infection[19].

Several studies suggest that a weakening of the immune system may be associated with necrotising periodontal diseases. For instance, dysfunctioning of the polymorphonuclear (PMN) leucocytes[12], a defective mitogenic lymphocyte reaction[20] as well as reduced expression of immunoglobulins[21,22] combined with other disorders of the immune system were diagnosed in patients with necrotising periodontal diseases.

If necrotising periodontitis is established, it is possible to assume with 95% probability that the T-lymphocyte count is below 200 cells/mm^3 [23]. In patients infected with HIV and in patients with other disorders that deplete the leukocyte count (e.g. leukaemia), this could be the reason for more frequent occurrences of this form of the disease[24].

In the USA, approximately 2% to 6% of all HIV-seropositive patients suffer from a necrotising periodontal disease[25], where the necrotising periodontal disease of the HIV-seropositive patients is not distinguishable from that of the HIV-seronegative patients[26]. Simultaneously occurring oral candidosis, hairy leukoplakia or Karposi's sarcoma may provide an indication of an HIV infection; however these lesions are by no means always present.

A recent study indicates that changes to the cytokine profile can be verified in Nigerian children with necrotising periodontal diseases. It was also possible to show an increase in the serum level of IL-8, IL-18, IL-6, IL-10, IL-1 as well as in the cortisol level with a slight reduction to the interferon (IFN)-gamma concentration[27]. This could relate to a causal connection between predisposing factors (stress, smoking, malnutrition and alcohol abuse) and necrotising periodontal diseases, due to the fact that these predisposing factors lead to an increase in the serum cortisol concentration as well as to a shift from TH_1- to TH_2-dominated immune response[27].

According to some studies, an additional predisposing factor is poor oral hygiene[26,28,29] and resultant chronic gingivitis[30].

Epidemiological studies indicate frequent occurrences of necrotising periodontal diseases in patients exposed to psychological stress[30–32]. This is specifically the case for soldiers in war situations[30,33], students under exam stress[31,34], patients with depression, or other mental and/or emotional disorders[35,36] that make those affected feel overburdened by particular life situations.

Among other things, smoking is also a predisposing factor for necrotising periodontal diseases[37]. The quantity is particularly important (> 20 cigarettes/day)[32].

■ Clinical pattern

Necrotising gingivitis is an inflammatory, destructive disease that is characterised by ulcerative and necrotic papillae. The ulcers are coated with an off-white to greyish pseudomembrane, which primarily comprises fibrin and necrotic tissue, the latter of which contains leukocytes, erythrocytes and large quantities of bacteria. Spontaneous bleeding occurs

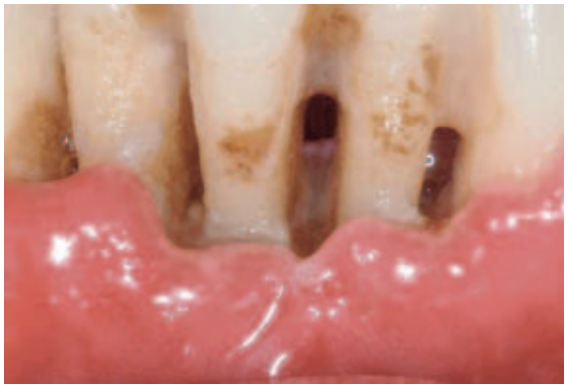


Fig 1 The necrosis divides the papilla into a facial and a lingual section.

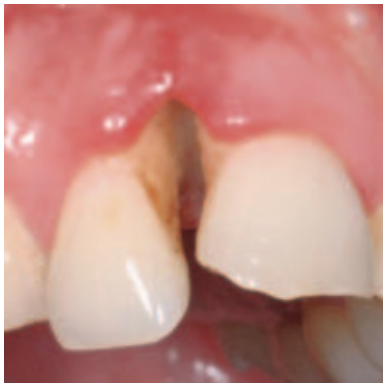


Fig 2 The necrosis between teeth 11 and 12 represents an aesthetic problem for the patient.

when the membrane is removed. Necrotic lesions develop very quickly and are painful. Initially, the necrosis is restricted to the tip of the papillae and is only observed in a small number of interdental areas[38,39]. However, in the majority of cases, the patient's main reason for visiting the dentist is severe pain. At this stage, the necroses have generally already significantly increased in size and number; bleeding occurs either spontaneously or when the area is touched. Characteristic fetor ex ore is often associated with necrotising periodontal diseases[40]. Gingival necroses develop quickly and the affected papillae are generally already separated into a facial and a lingual section and divided by the necrosis (Fig 1) after just a few days. The periodontal ligament and the alveolar bone are often involved in this phase of the disease. This leads to attachment loss[2]. The condition is then considered to be necrotising periodontitis.

The transition from necrotising gingivitis to necrotising periodontitis is generally smooth; it is possible for neighbouring areas to be affected by different stages of the disease. The interdental craters of necrotising periodontitis are significantly larger than those relating to necrotising gingivitis and often lead to cosmetic problems in the anterior tooth region (Fig 2). Together with the destruction of the papillae, the necrosis spreads laterally along the gingival margin and may merge with the neighbouring lesions. In this condition, the pain is felt deep within the bone.

The disease can progress very aggressively and may lead to small or larger necroses of the bone. This is usually the case in severely immunosuppressed patients, such as HIV-seropositive patients.

If the necroses extend beyond the mucogingival junction, the disease pattern for necrotising stomatitis is formed[3]. This severe tissue destruction is typically associated with the HIV infection and malnutrition and may lead to oroantral fistula and/or ostitis as well becoming life-threatening[3,41]. Contact necroses are often observed (necroses of the gingiva often spread to the neighbouring structures that they come into contact with, e.g. lip and cheek mucosa); the necrotic lesions can spread very rapidly and may lead to bone denudation and bone sequestration.

With necrotising periodontitis, swelling of the submandibular lymph nodes generally only occurs in very advanced stages of the disease. Some studies reveal that necrotising gingivitis is not generally accompanied by a fever. However, if this does occur then the body temperature frequently only increases slightly[32,42–44]. The oral hygiene of the affected patient is usually poor as every contact with the gingiva is painful.

■ Diagnosis

Diagnosis of necrotising gingivitis, periodontitis or stomatitis is based on the clinical pattern described above. Necrotising gingivitis and necrotising periodontitis are often mistaken for primary herpetic gingivostomatitis, which is caused by the herpes simplex virus[45].

Table 1 Differences between necrotising periodontal disease and primary herpetic gingivostomatitis.

	Necrotising periodontal disease	Primary herpetic gingivostomatitis
Aetiology	Bacteria	Herpes simplex virus
Age	15 to 30 years old	Primarily during childhood
Site	Interdental papilla	Gingiva and entire oral mucosa
Symptoms	Ulcerations and necrotic tissue coated with an off-white pseudomembrane; fetor ex ore; slight fever may occur	Multiple small blisters which burst and leave behind a round, fibrin-coated ulceration; fetor ex ore; fever
Duration of disorder	One to two days, with treatment	One to two weeks
Infectivity	–	+
Immunity	–	Sometimes
Cure	Irreversible periodontal destruction	Reversible gingival lesions

In industrialised countries, necrotising periodontal diseases primarily occur in young adults. They very rarely develop prior to adolescence[46], whereas primary herpetic gingivostomatitis is most commonly diagnosed during childhood. An additional differential diagnostic criterion is the fact that the clinical pattern for primary herpetic gingivostomatitis comprises multiple small blisters that burst to reveal round, fibrin-coated ulcerations. The lesions are distributed throughout the gingiva and throughout the entire oral mucosa (Table 1). With necrotising gingivitis and periodontitis, the lesions are generally restricted to the interdental papilla and coated with an off-white pseudomembrane.

With some forms of leukaemia, in particular acute leukaemia, necrotising ulcers, which are commonly also associated with the marginal gingiva, may present in the oral mucosa, which constitutes another differential diagnosis.

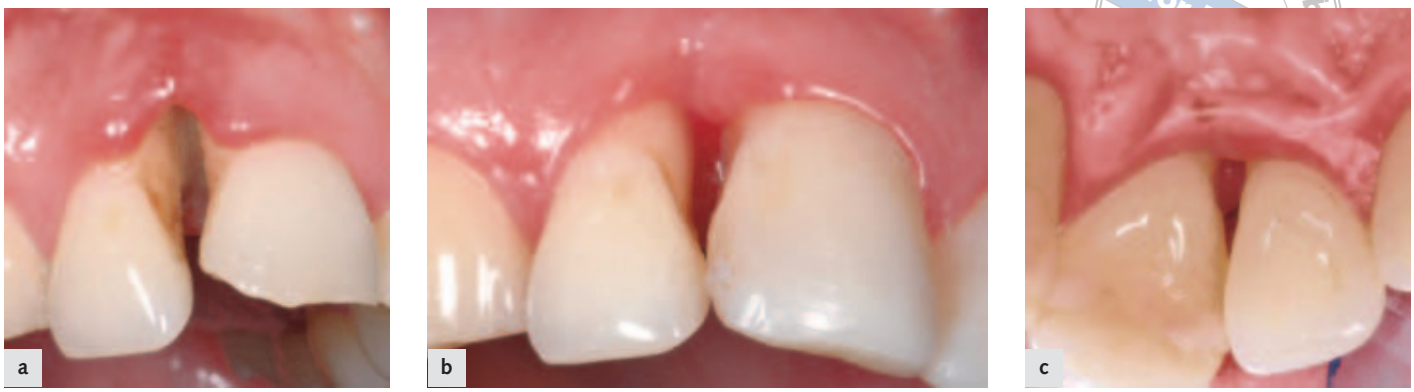
■ Treatment

Treatment for necrotising periodontal diseases can be subdivided into treatment of the acute phase and maintenance treatment. The treatment objective in the acute phase involves the rapid elimination of the disease activity and with it fast pain relief so that the patient can once again ingest food without experiencing pain.

If possible, the teeth should be brushed carefully during the first appointment, taking care not to touch the severely inflamed gums. Due to the fact that brushing the teeth in regions with open wounds is not conducive to healing, the patient should be provided with chemical plaque inspection using oral rinses in addition to mechanical oral hygiene[47]. Hydrogen peroxide and other oxygen-releasing agents have long been used in the treatment of necrotising periodontal diseases. Hydrogen peroxide (3%) is still used to clean necrotic areas or as an oral rinse (equal proportions of 3% H_2O_2 and hot water). The effect of the released oxygen on the anaerobic bacteria is credited for the successful treatment results of H_2O_2[48]. Gaggl et al[49] suggest that oxygen therapy (5 litre oxygen/minute for 15 minutes three times a day for 10 days) administered as a supplement to systemic treatment with antibiotics (metronidazole 500 mg 3 x 1 and 1 g co-amoxiclav 2 x 1 for 10 days) leads to improved treatment results in patients with pronounced necrotising periodontitis. Prior to treatment, all patients harboured *T. denticola, T. forsythensis* and *P. intermedia* bacteria. After treatment using supplementary oxygen therapy, the patients exhibited shallower residual probing depths and less attachment loss; in addition, periodontal pathogenic bacteria are more frequently eliminated than with antibiotic treatment alone.

A further, very effective measure for chemical plaque inspection comprises oral rinsing with 0.2% chlorhexidine gluconate twice a day, especially if no other domestic oral hygiene measures are feasible. Due to the fact that the chlorhexidine solution does not penetrate subgingivally and is relatively quickly inactivated by exudate, necrotic tissue and bacteria, its effectiveness is highly dependant on thorough professional tooth cleaning[50].

In some cases, for example if the patient has a bad reaction to the treatment or if their general state



Figs 3a to c The previously necrotic area has been cured and the size of the gingival crater has been reduced.

of health is reduced, systemic treatment with antibiotics should be prescribed. According to Proctor and Baker[51] as well as Loesche et al[11], treatment with metronidazole (250 mg three times a day) is the medication of choice in this regard and it leads to a significant improvement to the symptoms in the first few days of treatment[28,39,52]. In the treatment of necrotising periodontal diseases that are associated with an HIV infection, metronidazole is conducive to the speedy elimination of pain and to accelerated healing[53]. Acute pain is eliminated within a few hours. The risk of a spreading opportunistic infection in HIV-seropositive patients must not be underestimated. If necessary, antibiotic treatment should be carried out after consultation with the treating physician.

The condition of patients with necrotising periodontal diseases should be monitored on a daily basis until the acute symptoms have abated. With adequate treatment, this should take only a few days. Subgingival scaling and planing of the protruding filling or crown margin should be carried out once the ulcerations have healed.

Maintenance treatment starts once treatment of the acute phase has finished. However it is not often easy to motivate patients suffering from necrotic periodontal diseases and they often do not attend further treatments once the acute discomfort has been eliminated.

Once the previously necrotic areas have healed and the size of the gingival craters has reduced (Fig 3), there will still be some large gingival craters that the patient is unable to clean easily, and therefore present a risk of a relapse of the disease[28,32,54]. Shallow craters can be eliminated simply by means of a gingivectomy, larger ones may necessitate a flap operation. Treatment of nectrotising gingivitis and periodontitis is not complete until all of the gingival defects have been eliminated and optimum conditions for domestic oral hygiene measures have been created[26]. Extensive surgical interventions are to be avoided in patients infected with HIV due to poor healing.

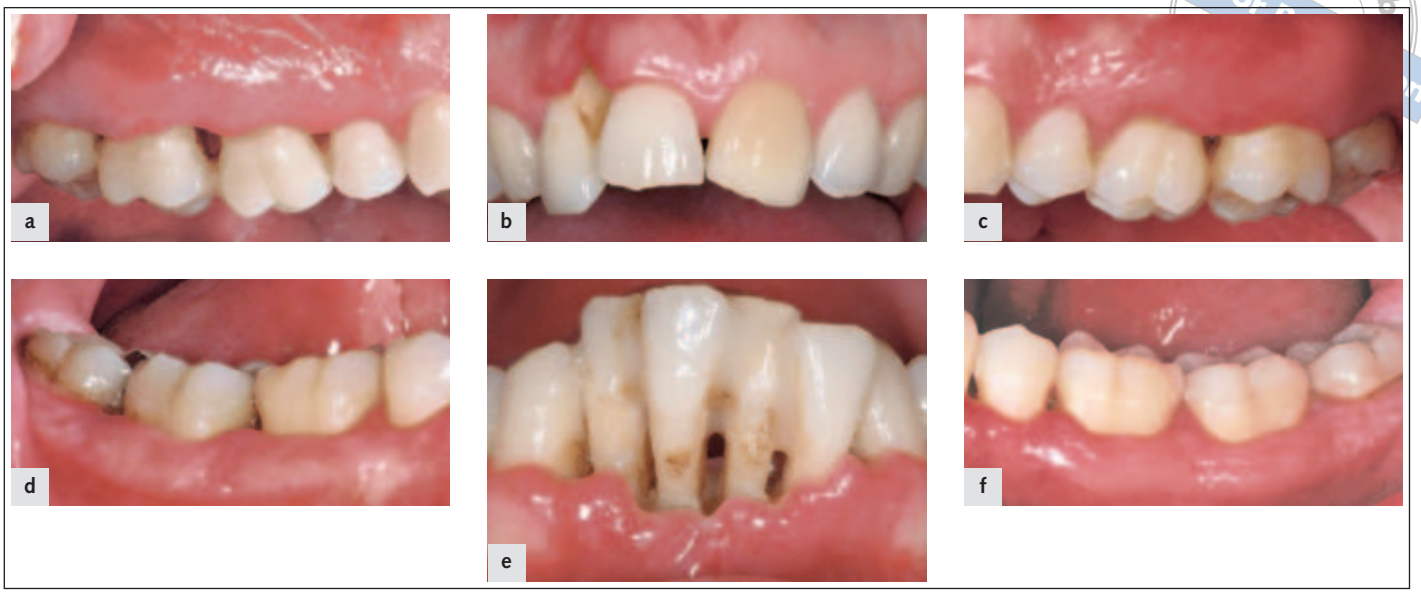
■ Case history

■ Case I

On 23 April 2005, a 31-year-old soldier presented to the Department of Periodontology at the University of Nijmegen with severe pain. He reported that he was presently stationed in Iraq as a helicopter pilot and that he currently smoked approximately 20 cigarettes per day. Due to severe pain, the patient reported not being able to eat properly. Furthermore, he complained of a poor aesthetic situation, which was caused by necrosis between teeth 11 and 12 as well as by severe loosening of the anterior mandibular teeth. The patient was accepted onto the MSc programme for periodontology and implantology.

General medical history

The general medical history was without pathological findings.



Figs 4a to f Clinical situation when the patient was accepted onto the MSc programme.

Dental medical history

In Spring 2003, endodontic treatment was carried out on tooth 37.

Clinical findings

Numerous necroses of the interdental papillae were observed, which were particularly strongly pronounced in regions 11/12 and in the lower jaw anterior teeth region (Fig 2). The plaque and bleeding indices were 92% and 68% respectively. The anterior mandibular teeth were blocked due to their severe loosening (degree III) elsewhere with composite. Teeth 11 and 21 harboured uncomplicated crown fractures and it was possible to measure probing depths of up to 11 mm (Figs 4 and 5).

Radiographic findings

The radiographic findings revealed generalised horizontal bone loss with particularly severely affected regions 16–18, 11 and 12 as well as unpreservable teeth 32–42. Furthermore, it was possible to diagnose a carious lesion on tooth 27 distally and tooth 28 mesially. In addition, it was discovered that the root canal of tooth 37 was insufficiently filled. However, the tooth did not feature chronic apical periodontitis (Fig 6).

Microbiological findings

The microbiological findings revealed a sharp increase in *T. forsythensis* and *F. nucleatum* as well as an increase in *T. denticola, C. rectus, Eubacterium nodatum* and *Eikenella corrodens* bacteria (Fig 7).

Diagnosis

The diagnosis was made of acute necrotising periodontitis that had formed on the base of previously existing chronic periodontitis.

Aetiology

The psychological stress caused to the patient by his deployment in Iraq, in combination with presumable previous poor oral hygiene and smoking 20 cigarettes a day can be considered to be aetiological factors in this case. It was possible to rule out an HIV infection after diagnostic clarification by the military doctor.

Provisional treatment plan

The provisional treatment plan made arrangements for the following points:

- Attempt to stop smoking.
- Instructions regarding oral hygiene.

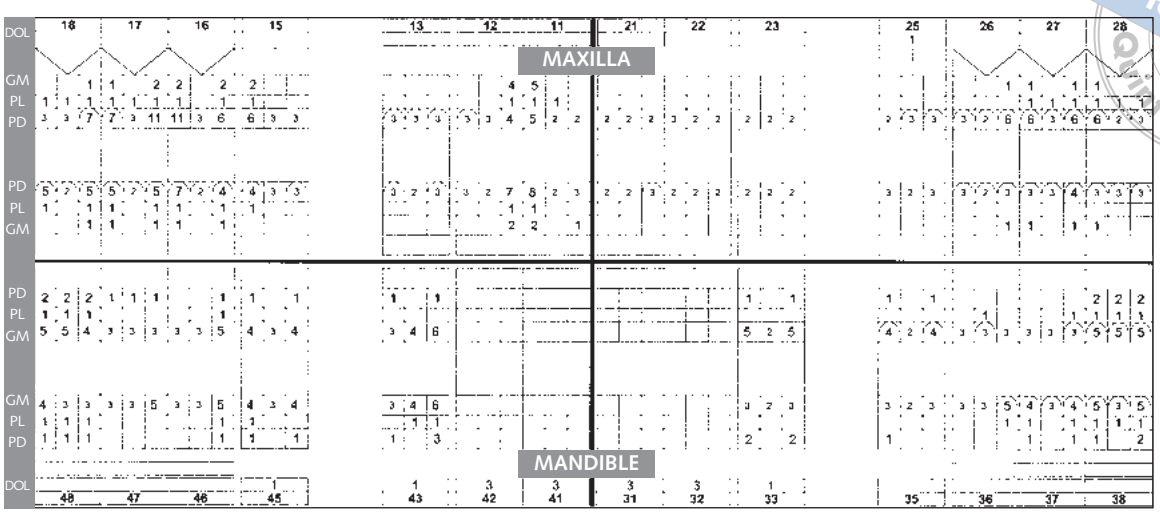


Fig 5 Periodontal status prior to treatment (DOL, degree of loosening; GM, gingival margin; PL, plaque; PD, probing depth, ∩, bleeding on probing).

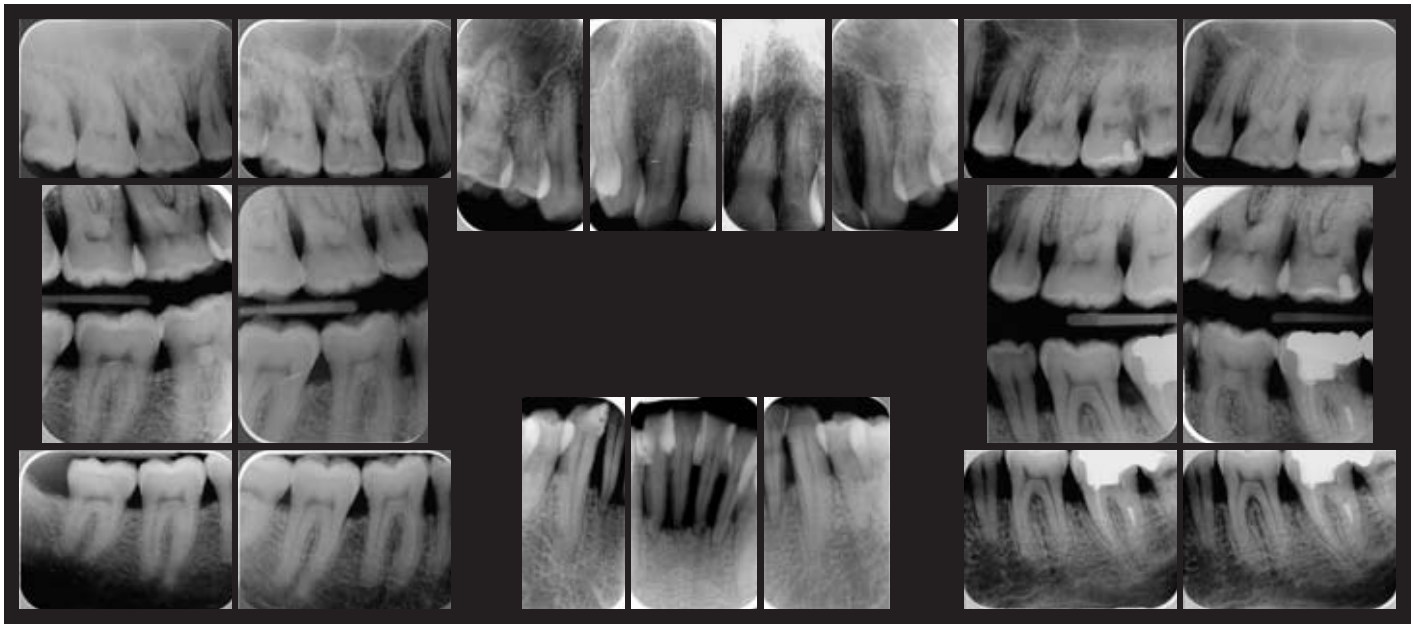


Fig 6 Oral film status with vertical bitewing images.

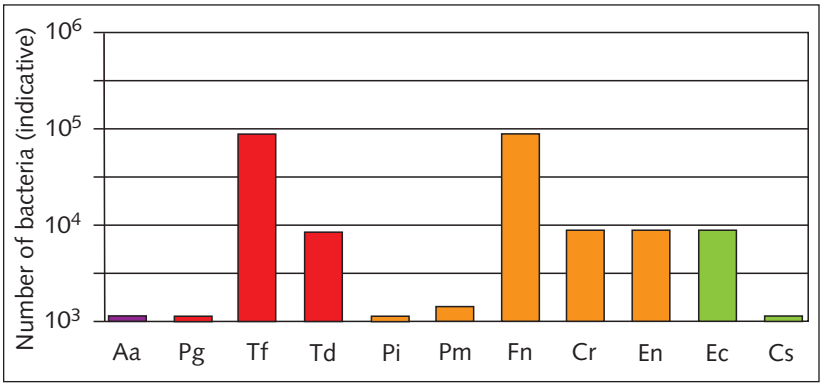


Fig 7 Microbiological findings prior to treatment.

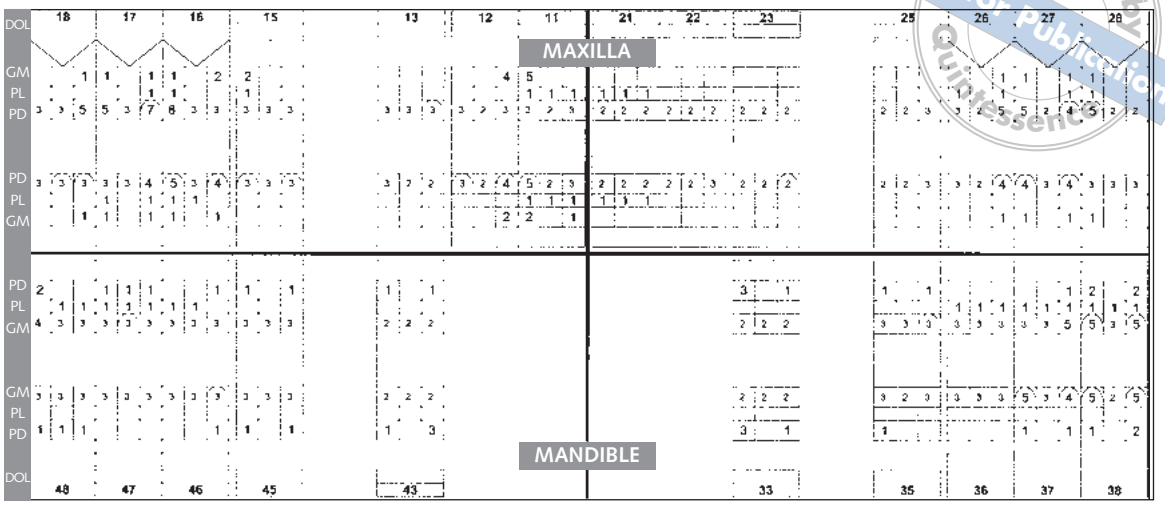


Fig 8 Periodontal status after non-surgical periodontal treatment (DOL, degree of loosening; GM, gingival margin; PL, plaque; PD, probing depth, ∩, bleeding on probing).

- During the acute phase:
  - careful supragingival cleaning;
  - systemic administration of metronidazole (500 mg 3 x 1 for 10 days).
- After pain relief, appointment for initial periodontal treatment.
- Extraction of the anterior mandibular teeth (32–42) with temporary provision.
- Extraction of tooth 28.
- Inspection appointment 6 weeks after the initial periodontal treatment.
- Restoration of teeth 21 and 11.
- Re-evaluation 3 months after the initial periodontal treatment.
- Possible surgical periodontal treatment based on the situation at re-evaluation.
- Maintenance treatment.
- Prosthetic provision.

Clinical findings at the time of re-evaluation

At re-evaluation, three months after the initial periodontal treatment, the patient presented with significantly improved, although not optimum, oral hygiene [plaque index (PI) 27%, sulcus bleeding index (SBI) 18%] and had given up smoking. Increased probing depths requiring further treatment were only apparent in regions 16/17 [probing depth (PD) up to 8 mm] (Fig 8). Severe flattening of the interdental craters was observed, which led to complete regeneration of the papillae in areas that were only slightly affected (Fig 9).

Definitive treatment plan

The definitive treatment plan made arrangements for the following measures:

- Supplementary instructions regarding oral hygiene.
- Surgical periodontal treatment in the
  - first sextant: apically positioned flap
  - second sextant: plastic reconstructive periodontal surgery according to Nemcovsky[55]
  - fifth sextant: insertion of the implants.
- Post-operative treatment.
- Restoration of the fifth sextant with an implant bridge.

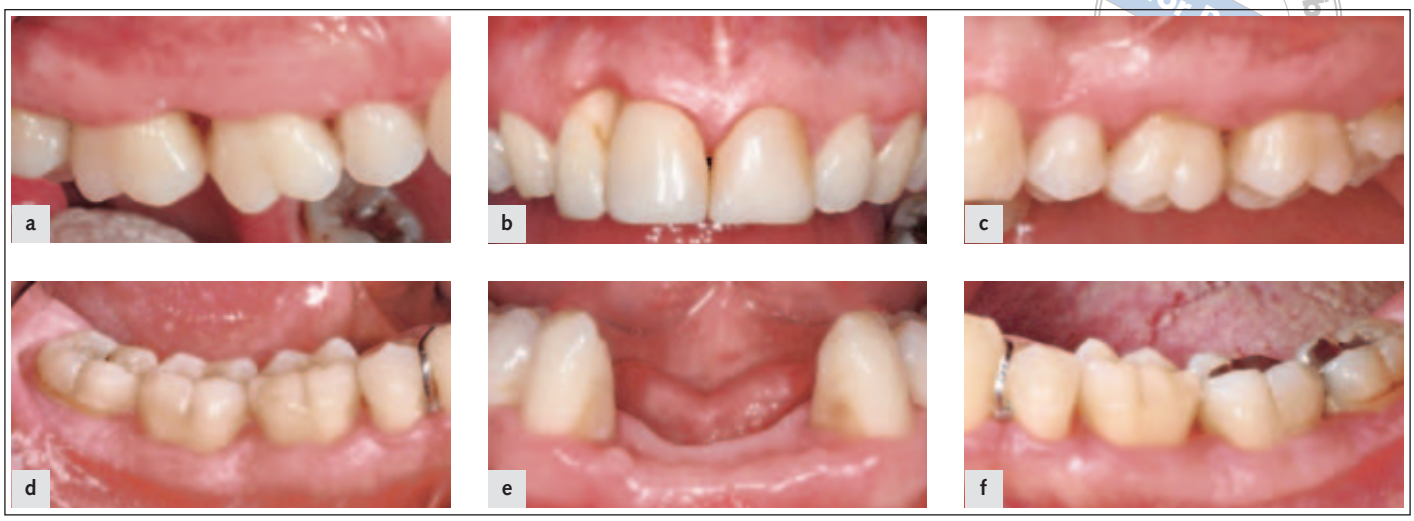
Course of treatment

• Surgical periodontal treatment

Bone sounding revealed horizontal bone loss of 9 mm (Fig 10). A paramarginal, garland-shaped cross-section was selected with a vertical relief cut mesial of tooth 16. After flap elevation, thorough scaling of the defect was carried out, whereby the entire granulation tissue was removed (Fig 11). Subsequently, the bone was re-contoured to achieve positive bone architecture (Fig 12). The wound was closed using 4.0 Vicryl simple interrupted sutures (Fig 13).

• Implantation

After elevation of the flap, the implant was inserted with the help of a drilling template (Replace® Select NP 3.5 x 11.5 mm) (Fig 14). The implants were



Figs 9a to f Clinical situation after non-surgical periodontal treatment and extraction of the anterior mandbular teeth.

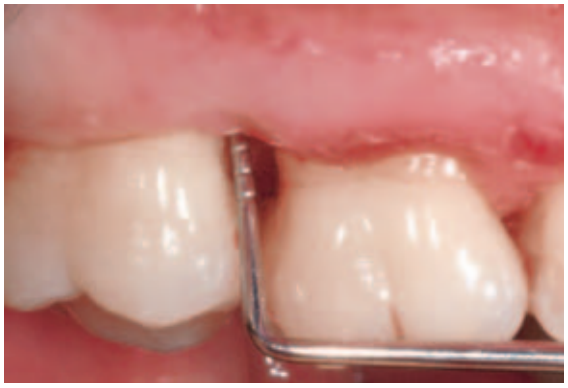


Fig 10 Bone sounding revealed horizontal bone loss of 9 mm.

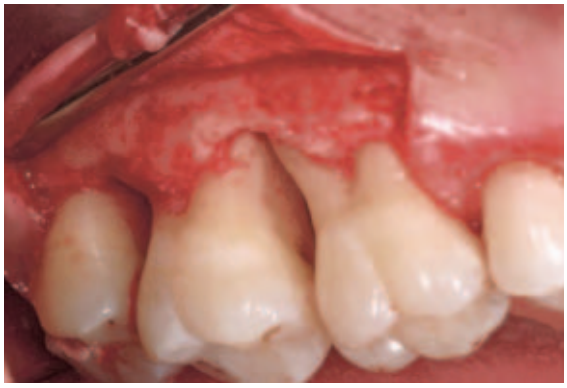


Fig 11 A paramarginal, garland-shaped cross-section was selected with a vertical relief cut mesial of tooth 16. After flap elevation, thorough scaling of the defect was carried out whereby the entire granulation tissue was removed.

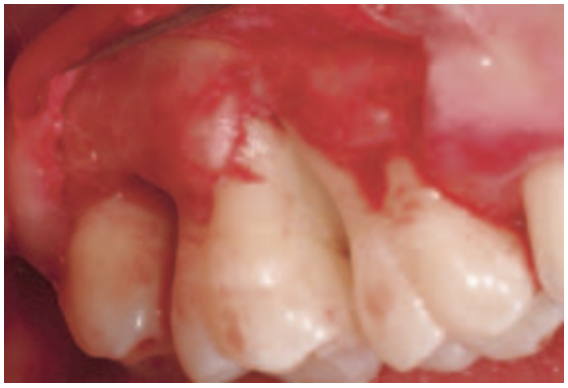


Fig 12 The bone was re-contoured to achieve positive bone architecture.

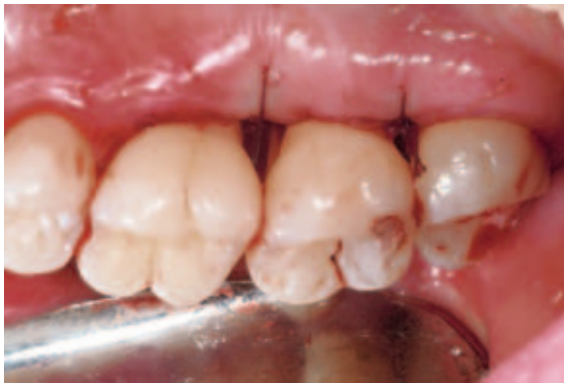


Fig 13 The wound was closed using 4.0 Vicryl simple interrupted sutures.

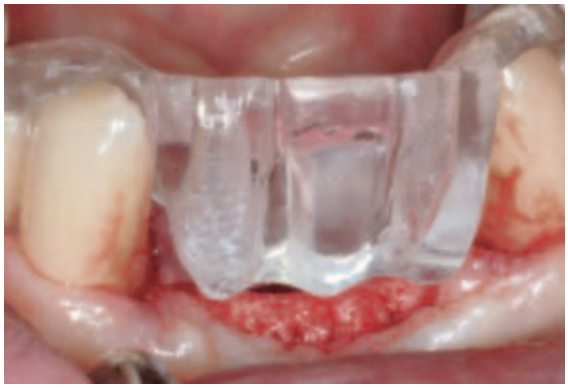


Fig 14 After elevation of the flap, the implant was inserted with the help of a drilling template (Replace® Select NP 3.5 x 11.5 mm).

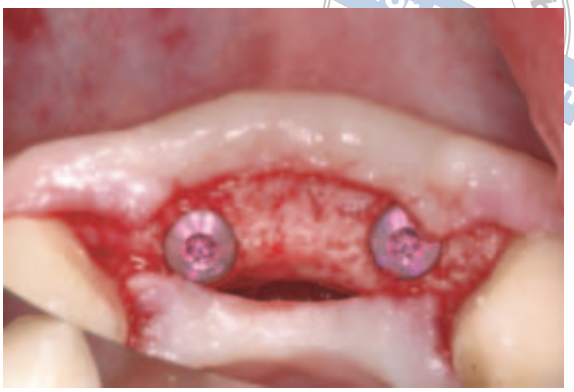


Fig 15 The implants were inserted into regions 32 and 42. For this, it was necessary to ensure a sufficient gap between neighbouring teeth as well as sufficient bone strength buccally and lingually of the implants.

inserted into regions 32 and 42 (Fig 15). For this, it was necessary to ensure a sufficient gap between neighbouring teeth as well as sufficient bone strength buccal and lingual of the implants (Fig 15). Prosthetic provision was carried out after a healing phase of 3 months.

Epicrisis and further treatment planning

Due to impaired ingestion, the patient was prescribed systemic treatment with metronidazole. In this case, the treatment record at the Department of Periodontology at the University of Nijmegen stipulates 500 mg of metronidazole three times a day during the acute phase, but for a minimum of 7 days, which is adapted from the recommendation to administer 20 mg of metronidazole/kg body weight/day. With a daily dose of 750 mg it would be possible to effectively treat an adult with a body weight of only 40 kg. Therefore, an adult should be treated with a daily dose of 1500 mg of metronidazole[56].

Surgical treatment of the residual probing depths of 5 mm is not indicated as such areas can be kept free from inflammation by good oral hygiene and regular recall appointments (quarterly intervals)[57].

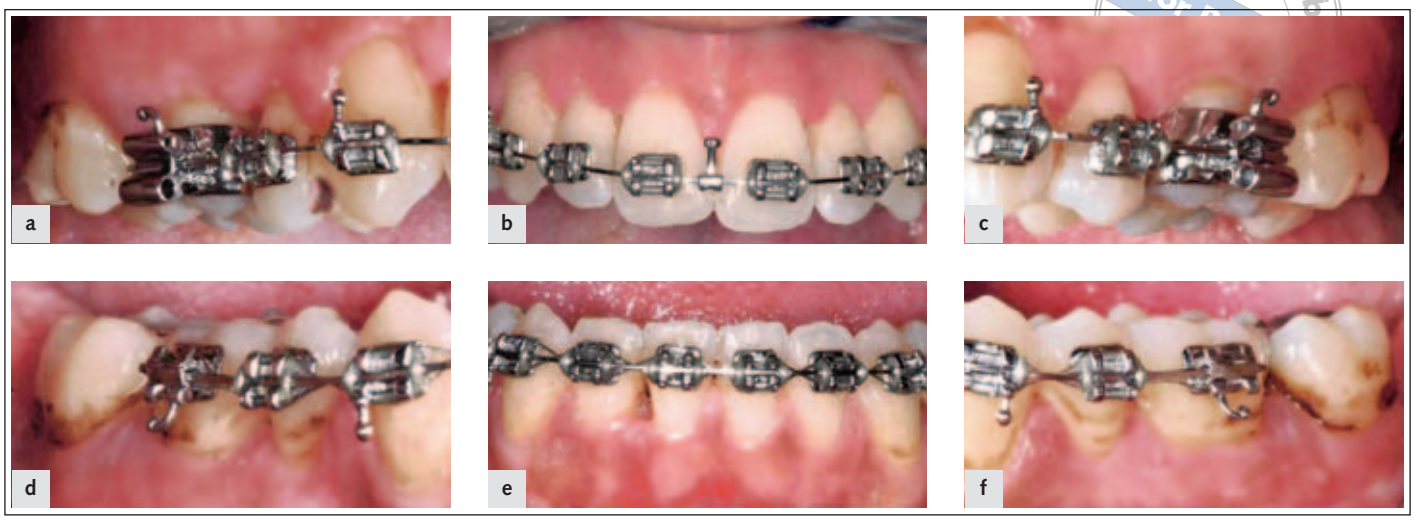
In line with Nemcovsky[55], it has not yet been possible to carry out the planned papilla augmentation as the patient had to return to his unit in Iraq. Papilla augmentation should give the papilla a convex shape in order to create the optimum conditions for the patient with regard to domestic oral hygiene without worsening the aesthetic situation further (Fig 3). The patient was informed that it is not possible to completely reconstruct the papilla.

According to Nemcovsky[55], during papilla augmentation a wedge of connective tissue is removed from the palate and is drawn through a tunnel prepared in the palate buccally. This therefore leads to coronal displacement of the interdental tissue. This method cannot predictably lead to successful treatment. However, Nemcovsky[55] suggests that in the case of non-traumatic procedures and a sound infection prophylaxis, the situation in the case of a failure after treatment is no worse than the initial situation.

Furthermore, re-evaluation of the operation is planned in the first sextant. Once the treatment is completed, the patient is accepted onto a recall programme in which maintenance therapy is carried out at quarterly intervals.

Case II

On 09 March 2005, a 31-year-old female patient was referred from the Department of Orthodontics to the Department of Periodontology at the University of Nijmegen. The patient was single, had a child and was studying on a teacher-training course. The patient smoked more than 20 cigarettes a day. She complained of painful and bleeding gums when brushing her teeth, as well as aesthetically displeasing black triangles, particularly in the anterior maxil-



Figs 16a to f Clinical situation when the patient was accepted onto the MSc programme.

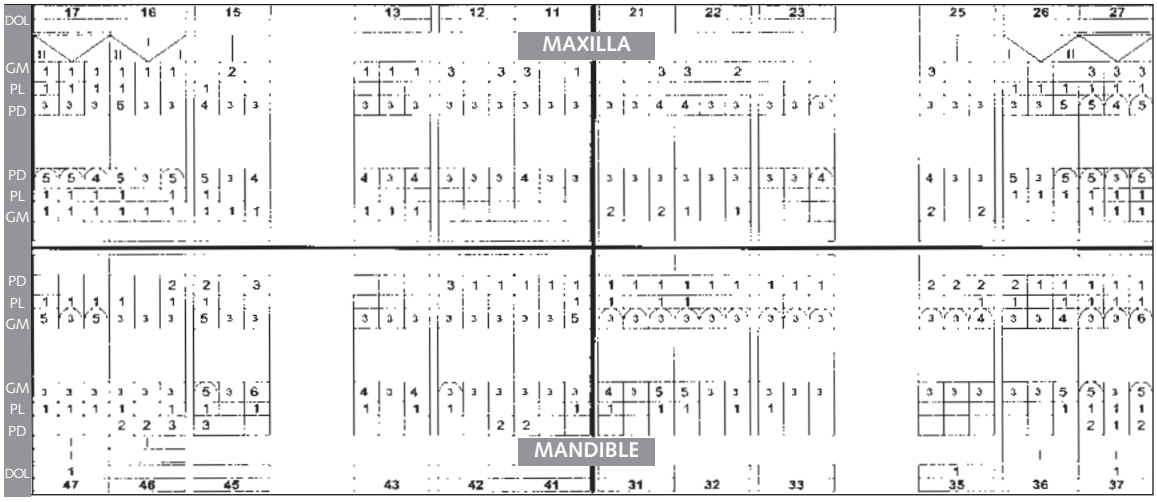


Fig 17 Periodontal status prior to treatment (DOL, degree of loosening; GM, gingival margin; PL, plaque; PD, probing depth, ∩, bleeding on probing).

lary region. The patient was accepted onto the MSc programme for periodontology and implantology.

General medical history

Due to previous chronic renal abscesses, the patient had been treated with antibiotics for a long period. At the time she was introduced to the Department of Periodontology, the patient often felt tired and reported chronic sleep deprivation brought on by exams and looking after her child.

Clinical findings

Sporadic interdental necroses were observed, which were mainly restricted to the tip of the papillae (Fig 16). The plaque and bleeding indices were 40% and 23% respectively. Using the methods of Hamp et al[58], it was possible to measure probing depths of up to 6 mm as well as up to degree II furcation involvements. Teeth 47, 35 and 37 exhibited the degree of loosening to be 1 (Fig 17).

Radiographic findings

The orthopantomogram revealed slight horizontal bone loss, primarily in the molar region (Fig 18).

Microbiological findings

The microbiological findings revealed a sharp increase in *T. forsythensis* and *F. nucleatum* as well as an

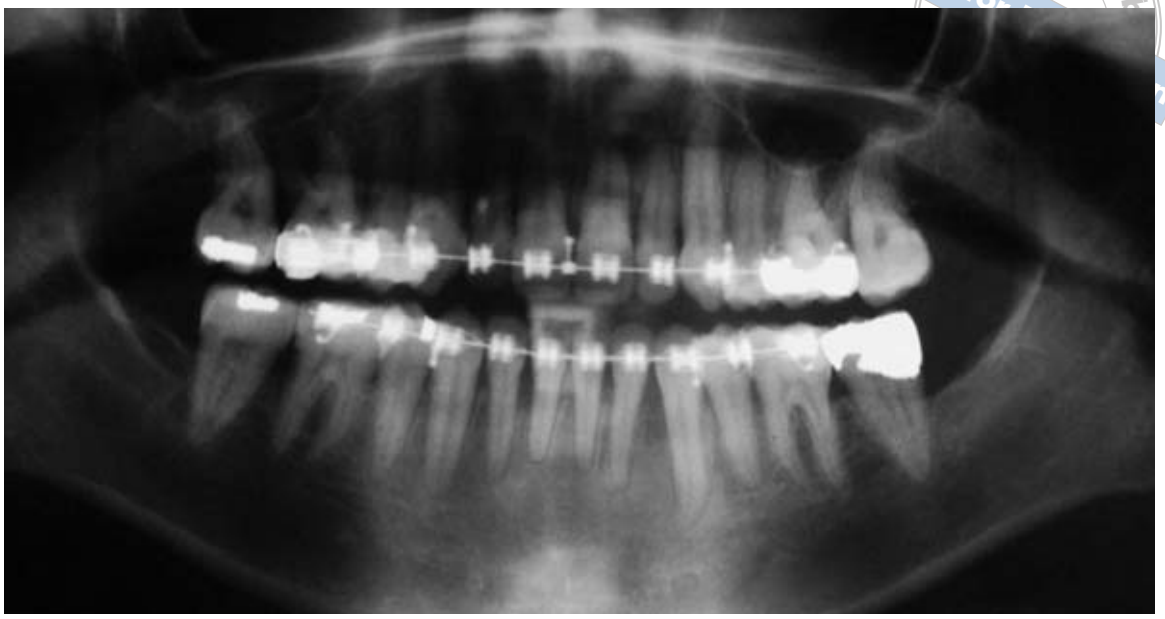


Fig 18 The OPG reveals slight horizontal bone loss, primarily in the molar region.

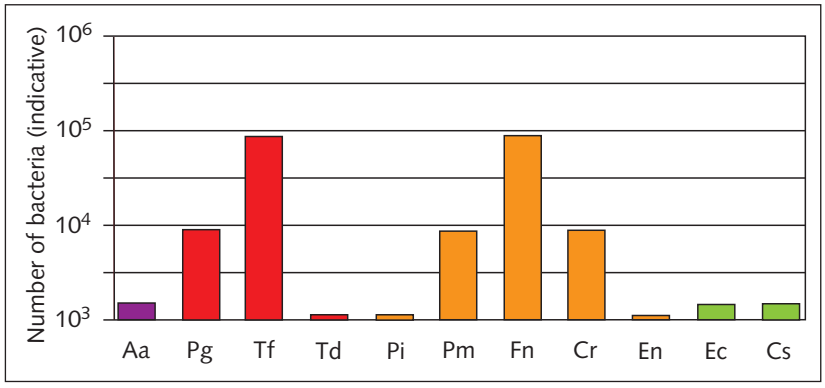


Fig 19 Microbiological findings prior to treatment.

increase in *P. gingivalis, C. rectus* and *Peptostreptococcus micros bacteria. A. actinomycetemcomitans* was present, although at very low levels (Fig 19).

Diagnosis

A diagnosis was made of acute necrotising gingivitis that had established on the base of previously existing chronic periodontitis.

Aetiology

Psychological stress (exam stress) in combination with chronic sleep deprivation and smoking more than 20 cigarettes a day can be considered to be aetiological factors. The GP was able to rule out an HIV infection in this case.

Provisional treatment plan

The provisional treatment plan made arrangements for the following points:

- Attempt to stop smoking.
- Instructions regarding oral hygiene.
- During the acute phase:
  – careful supragingival cleaning.
- Periodontal treatment, carried out in an appointment once the acute symptoms have abated.
- Inspection appointment 6 weeks after the initial periodontal treatment.
- Re-evaluation 3 months after the initial periodontal treatment.
- Possible surgical periodontal treatment based on the situation at re-evaluation.
- Maintenance treatment.

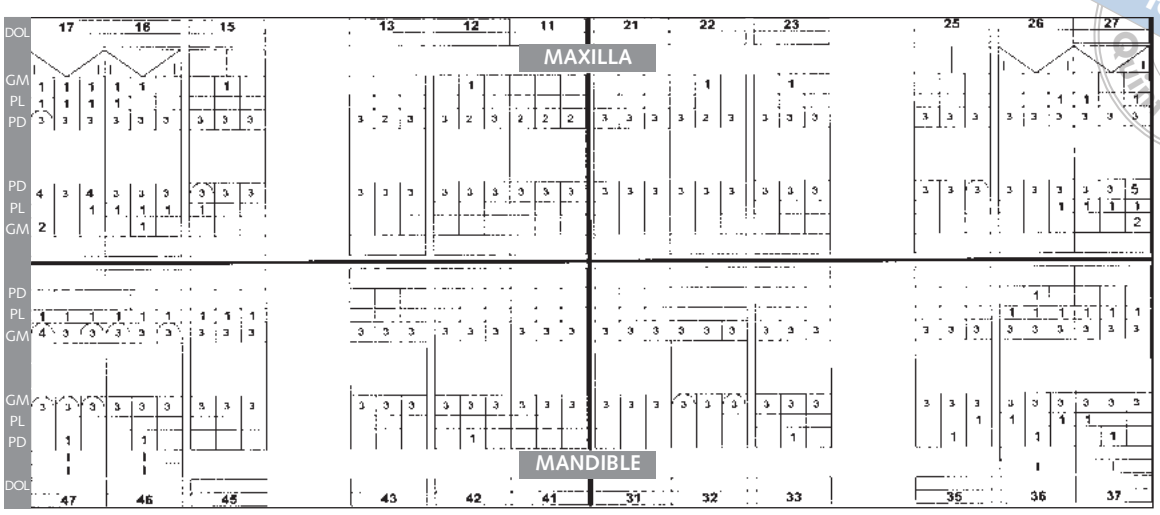
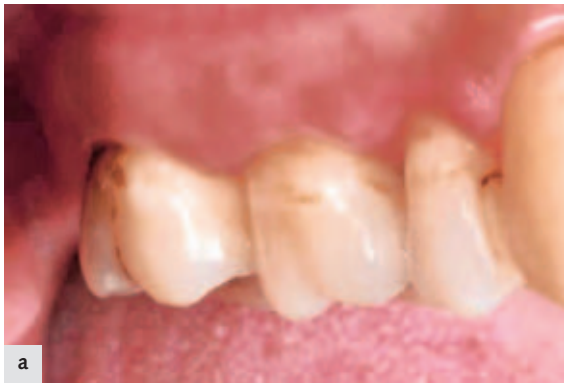
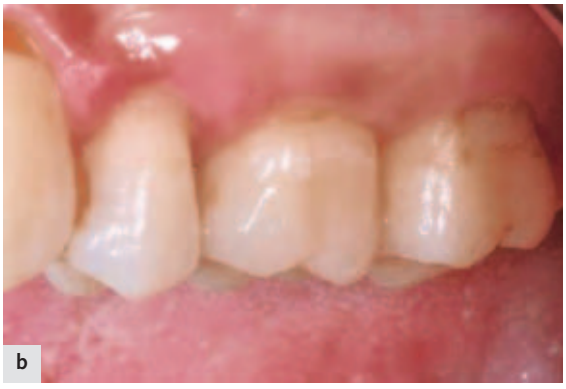
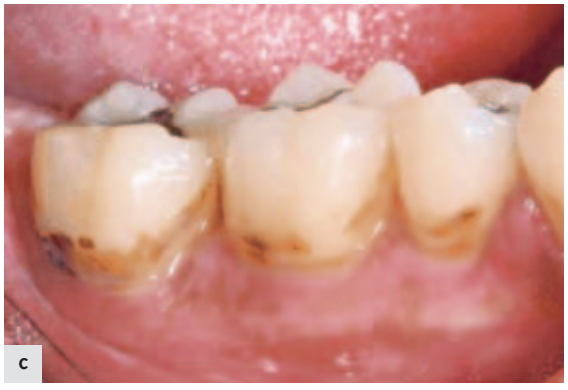
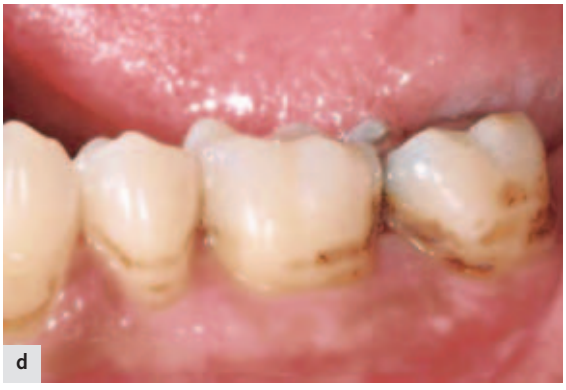


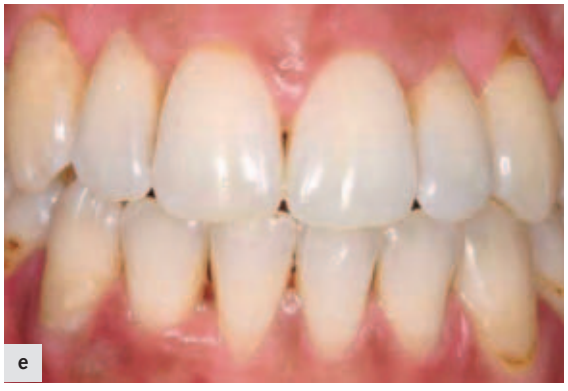
Fig 20 Periodontal status after non-surgical periodontal treatment (DOL, degree of loosening; GM, gingival margin; PL, plaque; PD, probing depth, ∩, bleeding on probing).











Figs 21a to e Clinical situation after non-surgical periodontal treatment.

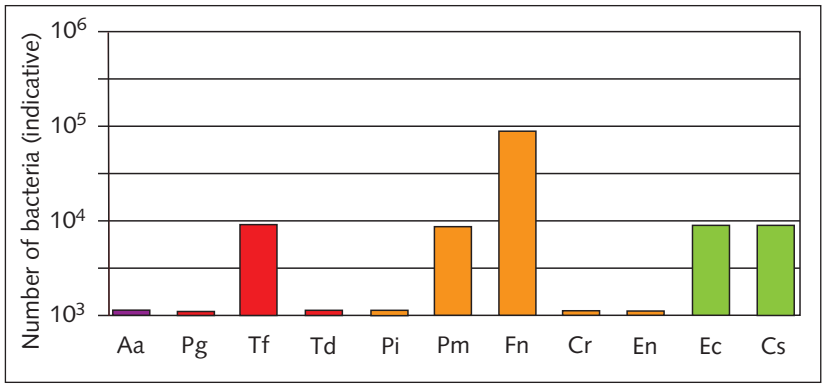


Fig 22 Microbiological findings after non-surgical periodontal treatment.

Clinical findings at the time of re-evaluation

At the point of re-evaluation, the patient exhibited significantly improved plaque and bleeding indices (PI 24%, SBI 8%) and had reduced her smoking to less than 20 cigarettes a day. Residual probing depths of up to 5 mm occurred sporadically in the molar region (Fig 20). The interdental papillae had largely rejuvenated after the non-surgical periodontal treatment (Fig 21).

Microbiological findings at the time of re-evaluation

The microbiological findings after the non-surgical periodontal treatment revealed a sharp increase in *F. nucleatum* as well as an increase in *T. forsythensis, Peptostreptococcus micros, Eikenella corrodens* and *Capnocytophaga* species of bacteria. *A. actinomycetemcomitans, P. gingivalis* and *C. rectus* were no longer present (Fig 22).

Epicrisis

Due to the fact that in this case the patient's ingestion was not impaired and her general heath was not affected, systemic treatment with antibiotics was not deemed necessary. The patient was also still in the position to implement limited domestic oral hygiene. After the non-surgical periodontal treatment was implemented, it was still only possible to measure sporadic residual probing depths of 5 mm. Microbiological testing revealed that improved oral hygiene together with subgingival scaling had resulted in the elimination of *C. rectus, P. gingivalis* and even *A. actinomycetemcomitans* bacteria. Due to the fact that the interdental papillae had largely rejuvenated after the non-surgical periodontal treatment, the patient was satisfied with the appearance of her teeth. Since that time, the patient has been undergoing maintenance treatment (quarterly intervals).

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Originally published (in German) in Parodontologie 2006;17(4):333-348.