Necrotising periodontal diseases

Précis: This article is the first in a new series that aims to provide brief revision of individual conditions that present management challenges in clinical dental practice. This article overviews the aetiology, clinical features and management of necrotising gingivitis. An illustrative case example is used and recommendations for further reading are also provided.

Table 1: Overview of key aspects of NG.

Diagnosis	Usually based on clinical findings alone, specifically the acute onset of symptoms and three key signs: pain, interdental ulceration, and bleeding
Prevalence	<1% of the general population[1,2]
Microbiology	Dominated by anaerobic bacteria; characterised as "fuso-spirochaetal infection"[3] Predominant species: *Fusobacterium* spp., *Treponema* spp., *Selenomonas* spp., *Prevotella intermedia.* Other bacterial species may be variably present. In HIV-affected patients, *Candida albicans* and herpes viruses also noted.
Histopathology	Necrotic lesions can be characterised by several zones,[4] oriented as follows (from superficial to deep): Bacterial area – neutrophil-rich zone – zone of necrosis – spirochaetal infiltration zone
Contagion potential	Increased NG prevalence has been noted in certain groups (e.g., soldiers, students, HIV patients). However, this increased prevalence is thought to be due to shared characteristics and NG is not identified as a contagious disease[5]
Differential diagnosis	▸ Periodontitis ▸ Infectious conditions: (herpetic gingivostomatitis; syphilitic lesions, tuberculosis lesions) ▸ Desquamative gingivitis/lichen planus ▸ Vesiculobullous conditions (e.g., pemphigus) ▸ granulocytosis

Introduction

Necrotising periodontal diseases (NPDs) are a group of infectious diseases that include necrotising gingivitis (NG), necrotising periodontitis (NP) and necrotising stomatitis (NS). These three clinical presentations may represent different stages of a continuum of the same disease process, with shared aetiological factors, and broadly similar initial clinical features and treatments. The term 'ulcerative' is no longer used in classification as ulceration is considered secondary to the gingival necrosis present.

NP may result in destruction of periodontal ligament and supporting bone, while in NS, destruction progresses to deeper tissues such as the lip, cheeks and tongue. These presentations are more frequently seen in patients with HIV/AIDS or other systemic/immune compromise and, in developing countries, among those with severe malnourishment, respectively. In an Irish context, NG will represent the most common clinical form seen in general dental practice and may present as an acute condition requiring urgent management. Consequently, the current review will focus on NG. **Table 1** provides an overview of the key aspects of NG.

Clinical presentation

The clinical presentation of NG incorporates three primary features of near universal presence, and a number of secondary clinical features **(Table 2).** NG is usually localised to one or a few teeth, but can be more widespread. Lesions most commonly involve the tip of the interproximal papilla but may spread to affect the entire papilla. A smaller number of cases (estimated one in five) will also involve marginal gingiva, with a few cases extending to involve attached gingiva or mucosa.[6] A number of non-specific predisposing factors for NG are noted, as outlined in **Table 3**.

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Table 2: **Necrotising periodontal diseases – clinical findings.**

	Primary clinical features
Pain	Usually of rapid onset; varying severity
Gingival necrosis	Interproximal tissue most commonly affected Ulceration may occur secondary to necrosis; if present, crater-like depressions may appear, giving rise to the characteristic 'punched-out' appearance of papillae
Gingival bleeding	Occurs with minimal provocation
	Secondary clinical features
Generally present	Halitosis (foetid in nature) Formation of pseudo-membrane
Sometimes present	Lymphadenopathy Fever Interdental gingival craters Bad taste (often metallic) Increased salivation

Table 3: Predisposing factors for NG.

General	Dental
▸ Young age ▸ Immunosuppression/ systemic disease ▸ Smoking ▸ Alcohol ▸ Stress ▸ General debilitation ▸ Poor diet/malnutrition	▸ Poor oral hygiene (OH) ▸ Pre-existing gingivitis ▸ Previous NPD history ▸ Local factors predisposing to plaque (e.g., tooth malalignment)

Clinical impact

- NPDs may be acute presentations in dental practice, most commonly among a young clientele who are not 'typical' periodontitis patients, and must be identified and managed effectively to address pain.
- Recurrence is common, and since clinical attachment loss may be evidenced even in cases diagnosed as NG,[7] NPD may have a longer-term impact on the patient's treatment needs.

The 2017 World Workshop on Classification recognised that: (i) the extent of disease; and, (ii) risk of progression to more severe conditions, may be

significantly different depending on the predisposing conditions present and the level of systemic compromise experienced by the individual,[8] and consequently proposed modifications to the classification of these diseases. Therefore, patients with NPDs who suffer a longer-term systemic compromise such as severe malnutrition or HIV/AIDS may undergo a more severe disease course, with greater risk of recurrence and/or progression to NP or NS. Conversely, the risk of recurrence and progression may be lower in most patients who present in a general dental practice setting with common predisposing factors. Nevertheless, if these patients have pre-existing periodontitis and/or risk factors cannot be effectively controlled, the possibility of NG progressing to NP may be elevated.

Management

Management of NG includes measures to address both the immediate/short-term and longer-term aspects of the clinical condition.[9]

Immediate/short-term

Aims:

- alter disease course/limit tissue destruction; and,
- address patient discomfort/ acute symptoms.

Includes:

- **manage associated pain;**
- **education:** advise patient about the clinical condition, its clinical course and the need to address predisposing factors;
- **initial instrumentation**: gentle supragingival debridement to remove plaque biofilm and necrotic tissue debris; powered devices (e.g., ultrasonic scaler) are generally recommended as irrigation may facilitate deposit removal; minimise physical trauma to the ulcerated gingival tissues; nevertheless, some minor bleeding is common following instrumentation;
- **personalised oral hygiene instruction (OHI):** the patient may be supplied with an extra-soft toothbrush to facilitate removal of plaque and necrotic tissue with minimal trauma; when brushing, the patient may use water/mouthrinse only to avoid irritation of ulcerated areas; as symptoms improve, mechanical techniques should be adapted and interproximal oral hygiene (OH) reintroduced;
- **chemical OH agent:** prescribe a mouthrinse for use during the period when mechanical OH is difficult/painful; chlorhexidine digluconate (CHX 0.12-0.2%, minimum twice daily) is most commonly prescribed for up to 14 days; it may be practical to supply the patient with a monoject syringe to simplify irrigation of the affected sites;
- **+/- antimicrobials:** prescription of an antimicrobial agent is appropriate where signs suggest systemic involvement (e.g., fever, lymphadenopathy); metronidazole significantly reduces the associated species[3] and is the agent most frequently advocated; dosage regimen (200mg/400mg) and duration (three to seven days) is somewhat equivocal in the literature; these authors most commonly recommend initial dosage of 200mg three times daily for three days; and,
- **regular follow-up:** the patient should be followed up closely in the early days post treatment (e.g., 48 hours and one week), to monitor the disease course and facilitate further removal of biofilm and necrotic debris as patient comfort permits.

Longer-term

Aims:

- address predisposing factors;
- prevent disease recurrence; and,
- provide corrective therapy where relevant.

Includes:

- **treatment of underlying periodontal conditions:** once the acute phase is under control, any required periodontal treatment should be commenced;
- **additional patient education and OHI:** modified as appropriate to achieve secondary prevention of plaque biofilm;
- **address predisposing factors:** general predisposing factors should be modified where feasible and local factors predisposing to plaque accumulation (e.g., restoration overhangs) addressed; advise the patient that NPD may recur if predisposing factors are not addressed;
- **+/- corrective treatment:** NG may alter gingival tissue contours, potentially resulting in cratering of interproximal papillae that may favour plaque accumulation; while corrective surgery is not generally required, gingivectomy and/or gingivoplasty may be used to treat superficial craters, while periodontal flap surgery may be used in severe cases; and,
- **supportive periodontal care:** enrol the patient in regular recall, based on individual risk profile for periodontal disease/NG recurrence.

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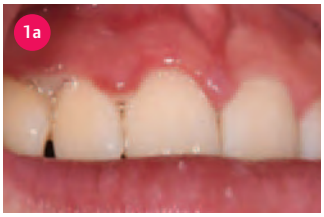
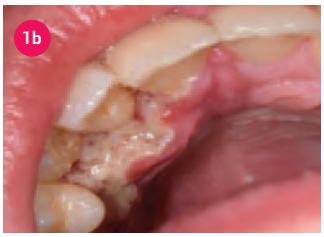
Illustrative clinical case example

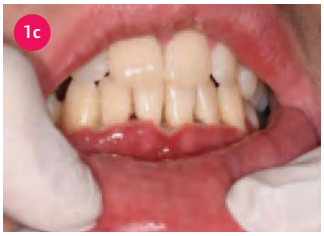
The case below illustrates the management of NG in a healthy 18-year-old male patient presented to the A&E department at Dublin Dental University Hospital complaining of a two-day history of "gum infection" that was painful to touch and described as feeling "like something is eating into my gums".

CLINICAL PRESENTATION

- The gingivae of the maxillary and mandibular anterior sextants were red and tender, with spontaneous bleeding present.
- Principal source of pain was the 1.3-1.2 area, where labial (**Figure 1a**) and palatal (**Figure 1b**) gingival necrosis, gingival bleeding and formation of a pseudo-membrane were noted.
- The mandibular inter-canine region (3.3-4.3) demonstrated labial gingival erythema and loss of tissue tone. Interproximal papillae were ulcerated with 'punched-out' appearance. Papillary tips demonstrated a necrotic surface with formation of a pseudo-membrane (**Figure 1c**).
- Oral hygiene (OH) was fair.
- The patient had a slightly elevated temperature and lymphadenopathy.







FIGURES 1a-1c: Initial clinical presentation demonstrating gingival necrosis and formation of pseudo-membrane at 1.3-1.2 region: (a) labial; and, (b) palatal. A 'punched out' appearance of the interproximal papiallae was evident at 3.3-4.3 region.

PREDISPOSING FACTORS

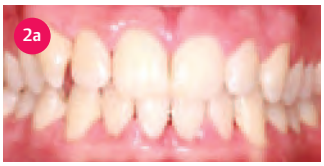
- Stress: recent close family bereavement and upcoming college examinations.
- Smoking: the patient admitted to smoking tobacco and occasionally marijuana to address his stress.
- Diet/nutrition: poor diet since his family bereavement with main daily meals limited predominantly to fast foods.

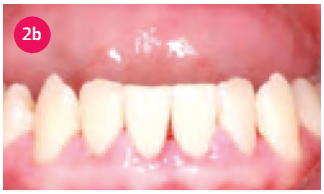
IMMEDIATE MANAGEMENT

- Patient was educated about NG and its predisposing factors.
- Patient was advised regarding appropriate analgesia.
- Detailed, personalised OHI was provided. The patient was supplied with an extra-soft toothbrush and monoject syringe to facilitate gentle OH.
- Affected areas were irrigated with chlorhexidine (CHX) to remove necrotic tissue debris and biofilm. Gentle tooth debridement was performed.
- The patient was prescribed CHX mouthrinse and metronidazole per protocol.

LONGER-TERM MANAGEMENT

- The patient was scheduled for further recall appointments over the following week. Each visit included gentle supragingival instrumentation and adaptation of the OH regimen based on the evolving clinical condition.
- Supportive periodontal care was provided (three-monthly recall) over a subsequent period of one year. As he demonstrated effective OH at each visit and no recurrence of NG, the patient was discharged back to his GDP.
- Healing occurred with relatively favourable gingival tissue tone and contour (**Figure 2a-c**). No surgical tissue recontouring was required.
- Despite counselling on cessation of his tobacco and marijuana smoking habits, and the importance of a balanced and healthy diet, the patient struggled to address these predisposing factors. Consequently, the GDP was advised to monitor the gingival condition regularly and the patient was advised on the potential for NG recurrence.





FIGURES 2a-2c: Clinical presentation of affected areas at start of 12-month supportive periodontal care appointment.