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Necrotising periodontal diseases: an update on classification and management

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Rachel Ogunleye,[1] **Obioma Ukoha**,[1] **Weronika Nasterska**,[1] **Ewen McColl**,[2] **Fatima Dantata**[3] and **Ifeoluwa Adetula**[4] provide an update on necrotising periodontal diseases for the whole dental team and provide tips on how to manage these conditions.

Abstract

Necrotising periodontal diseases can present with significant morbidity and, whilst unusual, they are not entirely uncommon in primary care. With this in mind, members of the dental team should be aware of relatively recent changes to the classification and management of these diseases to optimise patient outcomes. Similarly, understanding the bacteriology, patterns of tissue breakdown, and management and maintenance of these conditions will allow clinicians to manage these diseases should they present. The progression of necrotising diseases in Sub-Saharan Africa to present as noma is also discussed. The objective is to help the reader understand the classification and management of necrotising periodontal diseases.

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Introduction

Necrotising periodontal diseases (NPDs) are a severe form of dental disease that can be accompanied by systemic illness and involve the destruction of periodontal tissue and connective tissue. They range in extent and severity, with manifestations ranging from minor loss of papillary architecture to the extensive destruction caused by noma. While description and classification of these diseases has ranged from trench mouth to acute necrotising ulcerative gingivitis, this has relatively recently been reclassified to emphasise the range and progression of this condition.

Background/history

Periodontitis is defined as a chronic multifactorial inflammatory disease involving progressive destruction of the tooth-supporting apparatus (peridontium and alveolar bone), which may ultimately result in tooth loss.[1] Diverse communities of oral bacteria and microbes (microbiome), including within periodontal pockets, are essential for periodontal health. However, during necrotising periodontal diseases, bacterial dysbiosis may occur, resulting in the predominance of certain pathogenic strains that induce the inflammatory host response and symptoms associated with this form of gum disease. For example, *Neisseria spp*, *Corynebacterium spp* and *Prevotella spp* are found in necrotic lesions of patients with necrotising periodontitis.[2]

New classification

In 2017, a new classification system was developed in the world workshop on the 'Classification of periodontal and peri-implant diseases and conditions'.[3] The new periodontal classifications led to a shift in the way clinicians classify and diagnose periodontal diseases. In the previous 1999 classification,[4] NPDs were classified as 'necrotising ulcerative gingivitis' and 'necrotising ulcerative periodontitis'. In the 2017 classification system, NPDs are defined as: necrotising gingivitis (NG), necrotising periodontitis (NP), necrotising stomatitis (NS) and noma.[5] Clinically, it can be challenging to differentiate between NG and NP as often the transition is difficult to distinguish as the disease advances. Studies suggest that NG, NP, NS and noma are different stages of the same disease as they share clinical characteristics and, to some extent, treatment.[5]

The case shown in Figure 1 is from a 25-year-old patient who presented to a primary care dental facility at Peninsula Dental School, University of Plymouth. This case demonstrates the presentation of NP and some of the key features of NPD on which we will expand in this paper.

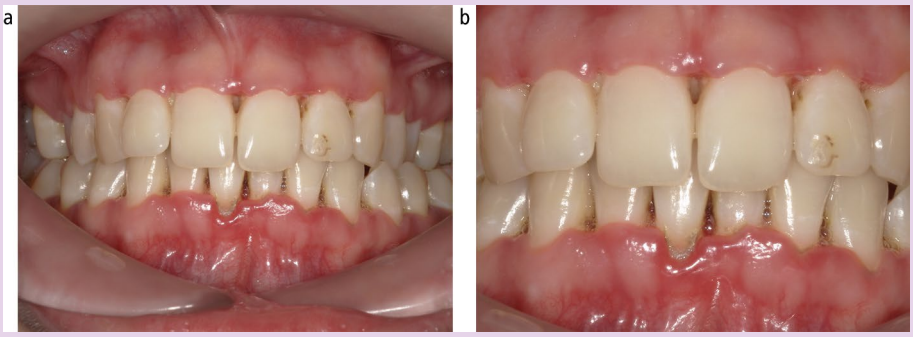

Fig. 1 a, b) Typical presentation of necrotising periodontitis in a 24-year-old

Microbiology

NPDs arise due to microbial infection of a susceptible host. Plaut first described the specific bacteria involved in 1894 and Vincent in 1896, who identified the involvement of the fusospirochetal complex in the disease aetiology.[6] Histologically, four zones of the disease have been described in electron microscopy studies:[7,8,9] a bacterial zone (including spirochetes and other bacteria); a neutrophil-rich zone (containing neutrophils, leucocytes and some bacteria); a necrotic zone (characterised by tissue necrosis and presence of *Spirochetes* and fusiform bacteria); and a spirochetal infiltration zone (where the tissue is still well preserved but *Spirochetes* are already present).

Immune assays[10,11] and polymerase chain reaction studies of isolated bacteria *ex vivo*[12] have confirmed spirochetal involvement. Other bacteria such as *Prevotella intermedia*, *Treponema*, *Selenomonas* and *Fusobacterium* species have been identified as constant flora by culture studies.[13] However, we are also beginning to understand the importance of the interactions between bacteria within the oral microbiome during necrotising peridontits,[2] with the reduction of commensal bacteria in favour of pathogenic strains such as *Prevotella spp*, but further research is still needed in this field. Many of the bacteria involved in NPDs are opportunistic pathogens commonly present in healthy patients, which highlights the importance of a balanced oral microbiome and host susceptibility to pathogenic strains. Host inflammatory responses may be further enhanced by underlying risk factors, such as smoking and diabetes; it is not just the existence of the bacteria themselves.

Risk factors

Multiple predisposing factors are significant in explaining the pathogenesis of NPDs. The most common risk factors relate to poor oral hygiene, smoking, stress and young age, very much as seen in First World War trenches – hence the previous description of trench mouth. Pre-existing gingivitis and a history of NPD are also thought to be important and may be linked to the normal gingival architecture being compromised, due to previous disease episodes and patients not improving their oral hygiene or the gingival architecture making this difficult to clean.

> *'Multiple predisposing factors are significant in explaining the pathogenesis of NPDs.'*

While poor oral hygiene is a key element, many of the other factors described may impact on the host response to this bacterial dysbiosis. Similarly, other predisposing factors altering the host's immune response include, but are not limited to, undiagnosed human immunodeficiency virus (HIV) infection, where the host's immune response is compromised.[14,15] These patients may also not respond so well to treatment and recurrence remains a distinct possibility. Literature reports that malnutrition is also an important predisposing factor for NG/NP, especially in developing countries.[16]

Management

Necrotising gingivitis and necrotising periodontitis

When reaching a diagnosis, it is important to recognise the typical presentation of NPDs as quoted above. This includes bleeding, loss of papillary architecture as in Figure 1, punched out papilla, metallic taste, sloughing and grey appearance of the papilla. Additionally, as disease progresses, exposure of the underlying alveolar bone may occur. As is always the case, a thorough examination and special tests as necessary are indicated in order to develop a differential diagnosis. However, in our experience, the presentation of the disease is so distinct that the diagnosis is relatively straightforward.

The treatment of necrotising diseases should be approached in stages, including treatment of the acute phase, treatment of any pre-existing conditions, addressing risk factors, treatment of disease sequelae and transition to supportive or maintenance phase.[17] Once a diagnosis of NG/NP has been made, it is essential to first begin by addressing the cause of the episode and target treatment to alleviate the risk factors discussed above. Essential supportive measures include advice and demonstrations to improve oral hygiene (done gently at first, due to the markedly inflamed nature of the tissues on presentation) and advising on smoking cessation. In many cases, advising on rest and incorporating fluid intake with analgesics is recommended as required.

In the acute phase, gentle debridement using a hand scaler or ultrasonic device is indicated but this is very dependent on what the patient can tolerate. As the pain may be significant, it may be necessary to perform this debridement over several days or under local anaesthesia. Once the most acute pain has subsided, further reinforcement of oral hygiene is indicated and tailored to what the patient can tolerate (soft toothbrushing and interdental aids as necessary).

Where the pain is significant in the acute phase, mouth rinses may help. Regular rinsing with a teaspoon of salt in hot water or twice a day with 6% hydrogen peroxide mouthwash or 0.2% chlorhexidine may help in the first few days following initial debridement. Likewise, the mouthwashes can be used to manage pain alongside over-the-counter analgesics, such as paracetamol, if there is a wait to be seen.

In instances where the patient is pyrexic on presentation, systemic antibiotics may

be indicated. Metronidazole is selected as the first-line antibiotic treatment, effective against anaerobes, including the *Fusospirochetal* anaerobes associated with this disease. Antibiotics should only be prescribed following national guidelines when systemic involvement in the form of pyrexia, malaise, or lymphadenopathy is present.

In these circumstances, metronidazole is the antibiotic of choice as it is effective against anaerobes as found in the *Fusospirochetal* complex found in NPDs. The prescription as per the British National Formulary should consist of metronidazole 400 mg, eight-hourly, for 3 days. Alternatively, amoxicillin may be used if metronidazole is deemed inappropriate.[18] Figure 2 is the same case as in Figure 1 which was treated initially with gentle debridement, oral hygiene instruction, metronidazole (as presented with pyrexia) and subsequent professional mechanical plaque removal. The reduction in inflammation and reconfiguration of papillary architecture can be noted.

Patients with necrotising diseases may also require the clinician to liaise with their physician and highlighting the unusual nature of the condition will be important. This is because the patient may potentially have an underlying immunocompromising condition (for example, acquired immune deficiency syndrome [AIDS], HIV-positive, leukaemia, cyclic neutropenia).[19]

Once stable, the maintenance phase will involve regular reviews to maintain and reinforce oral hygiene and reassess risk factors as necessary. The loss of papillary architecture may mean oral hygiene regimes have to be tailored accordingly and appropriate sizing of interdental brushes will be essential in this maintenance phase.

Necrotising stomatitis

NS is a further progression of the disease with risk factors as discussed previously. NS, as with the other diseases, is painful and when the gingival architecture is so severely compromised, this can lead to exposure of the underlying alveolar bone which can also necrose.[20,21] The clinical presentation is similar to the other necrotising periodontal diseases with a higher likelihood of pyrexia and cervical lymphadenopathy, with patients often feeling systemically unwell. There are no national guidelines for the treatment of NS; however, case reviews indicate successful treatment following a similar strategy to the other necrotising periodontal diseases.[18,21]

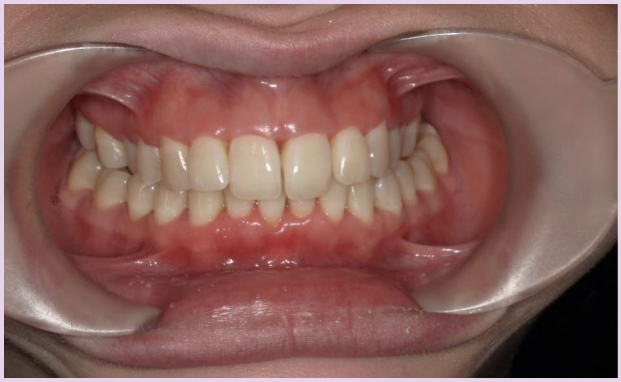

Fig. 2 Treated necrotising periodontitis case

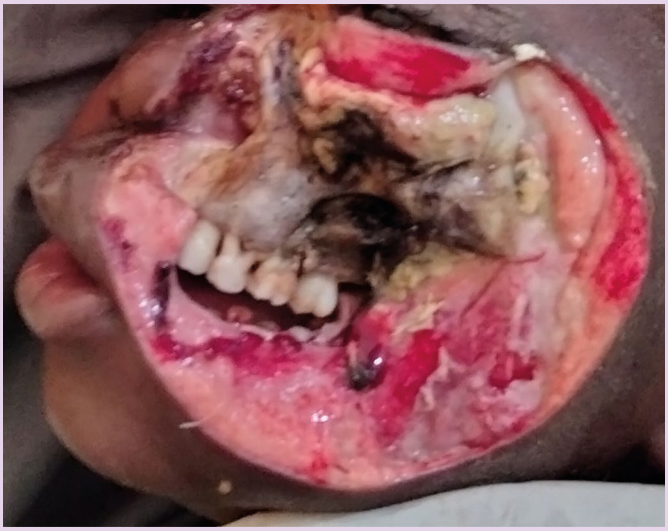

Fig. 3 Noma presenting in Nigeria

'The disease is characterised by necrotising fasciitis, osteonecrosis and myonecrosis, that leads to severe facial disfigurement and disability.'

As always, the key is improving the patient's oral hygiene and home care and where there is pyrexia and systemic involvement, metronidazole prescription, as discussed previously, is indicated. As with the other NPDs, recall visits are essential, particularly given the damage to the periodontal architecture and surgery may be indicated to address this damage as far as possible.[21]

Noma

The World Health Organisation (WHO) describe noma as 'noma (from Greek: to "devour") is a necrotising disease that destroys the mouth and the face'. This disease is characterised by necrotising fasciitis, osteonecrosis and myonecrosis, that leads to severe facial disfigurement and disability.[22] Figure 3 indicates presentation of the disease as it affects the circumoral soft tissue; in many cases, affecting the whole of the face.

Noma mostly affects children aged 2–6 years living in Sub-Saharan Africa, with an estimated incidence ranging from 30,000–140,000 cases annually.[23] However, many cases of noma are believed to be underreported

due to lack of access to adequate healthcare, lack of awareness, neglect and stigma.[24] The WHO also estimated an 80–90% mortality rate of noma cases annually in the absence of treatment.[22] Affected patients may suffer severe dehydration, sepsis, aspiration pneumonia, respiratory insufficiency, or starvation and if not treated it can be life threatening.[24] Although noma is an infectious disease, there are several predisposing factors that have been linked to the occurrence and progression of this disease. These include chronic malnutrition, extreme poverty, poor living conditions, unsafe drinking water, poor oral hygiene, limited access to healthcare facilities, lack of childhood vaccinations and immunosuppression resulting from comorbidities, including measles, anaemia, malaria, kwashiorkor and HIV/AIDS.[22,23,24] Treatment of noma depends on the stage at which patients present; however, treatment usually includes non-surgical periodontal therapy, antibiotics and possibly surgical rehabilitation.[25] As is the case with many other oral diseases, noma is preventable through risk factor control. Unfortunately, many of the risk factors for noma are not due to the personal decisions of the patient but are due to greater political and social issues. Organisations like the ZeroNOMA initiative[26] in Nigeria are currently taking action to the fight against noma by raising awareness, conducting research and acting as advocates for noma patients.

Conclusion

NPDs are a rare but challenging conditions to manage. By understanding disease classification, aetiology and disease progression, clinicians should be equipped to manage conditions within their competence range and similarly the need to refer as appropriate.

Ethics declaration
The authors declare no conflicts of interest.
Written consent to publish was obtained for Figure 3.

Author contributions
Ewen McColl had the idea to write a paper on this subject matter and edited contributions from Rachel Ogunleye, Obioma Ukoha and Weronika Nasterska. Images of NP were provided from an undergraduate clinic at Peninsula Dental School. Fatima Dantata and Adetula Ifeoluwa wrote the section on noma and provided all images of noma.

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References

1. West N, Chapple I, Claydon N *et al.* BSP implementation of European S3 – level evidence-based treatment guidelines for stage I–III periodontitis in UK clinical practice. *J Dent* 2021; **106:** 103562.
2. Jia J, Zhou Y, Wang X, Liu Y. Subgingival microbiome dynamic alteration associated with necrotizing periodontal disease: A case report. *Medicine (Baltimore)* 2021; DOI: 10.1097/MD.0000000000024311.
3. American Academy of Periodontology. 2017 Classification of Periodontal and Peri-implant Diseases and Conditions. 2017. Available at https://www.perio.org/2017wwdc (accessed September 2020).
4. Annals of Periodontology. 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. Papers. Oak Brook, Illinois, October 30 – November 2, 1999. *Ann Periodontol* 1999; **4:** 1–112.
5. Papapanou P, 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. *J Periodontol* 2018; DOI: 10.1002/JPER.17-0721.
6. Rowland R W. Necrotising ulcerative gingivitis. *Ann Periodontol* 1999; **4:** 65–73.
7. Listgarten M A. Electron microscopic observations on the bacterial flora of acute necrotising ulcerative gingivitis. *J Periodontol (1930)* 1965; **36:** 328–339.
8. Heylings R T. Electron microscopy of acute ulcerative gingivitis (Vincent's type). Demonstration of the fusospirochaetal complex of bacteria within pre-necrotic gingival epithelium. *Br Dent J* 1967; **122:** 51–56.
9. Courtois G J 3rd, Cobb C M, Killoy W J. Acute necrotising ulcerative gingivitis. A transmission electron microscope study. *J Periodontol* 1983; **54:** 671–679.
10. Riviere G R, Wagoner M A, Baker-Zander S A *et al.* Identification of spirochetes related to Treponema pallidum in necrotising ulcerative gingivitis and chronic periodontitis. *N Engl J Med* 1991; **325:** 539–543.
11. Riviere G R, Weisz K S, Simonson L G, Lukehart S A. Pathogen-related spirochetes identified within gingival tissue from patients with acute necrotising ulcerative gingivitis. *Infect Immun* 1991; **59:** 2653–2657.
12. Dewhirst F E, Tamer M A, Ericson R E *et al.* The diversity of periodontal spirochetes by 16S rRNA analysis. *Oral Microbiol Immunol* 2000; **15:** 196–202.
13. Loesche W J, Syed S A, Laughon B E, Stoll J. The bacteriology of acute necrotising ulcerative gingivitis. *J Periodontol* 1982; **53:** 223–230.
14. Journal of the Canadian Dental Association. Managing Patients With Necrotizing Ulcerative Periodontitis. 2013. Available at https://jcda.ca/article/d44 (accessed February 2022).
15. Ahlgren M, Funk T, Marimo C, Ndiaye C, Alfvén T. Management of noma: practice competence and knowledge among healthcare workers in a rural district of Zambia. *Glob Health Action* 2017; **10:** 1340253.
16. World Health Organisation. Oral health. 2022. Available at https://www.who.int/news-room/fact-sheets/detail/oral-health (accessed February 2022).
17. Malek R, Gharibi A, Khlil N, Kissa J. Necrotizing Ulcerative Gingivitis. *Contemp Clin Dent* 2017; **8:** 496–500.
18. National Institute for Health and Care Excellence. Scenario: Acute necrotizing ulcerative gingivitis. 2021. Available at https://cks.nice.org.uk/topics/gingivitis-periodontitis/management/acute-necrotizing-ulcerative-gingivitis/ (accessed February 2022).
19. Gasner N S, Schure R S. *Necrotizing Periodontal Diseases*. Florida: StatPearls Publishing, 2022.
20. Falkler W A Jr, Martin S A, Vincent J W, Tall B D, Nauman R K, Suzuki J B. A clinical, demographic and microbiologic study of ANUG patients in an urban dental school. *J Clin Periodontol* 1987; **14:** 307–314.
21. Suenaga H, Saijo H, Chikazu D *et al.* Necrotising Ulcerative Stomatitis in a Neutropenic Patient with Malignant Lymphoma. *Asian J Oral Maxillofac Surg* 2008; **20:** 196–200.
22. Feller L, Khammissa R A G, Altini M, Lemmer J. Noma (cancrum oris): An unresolved global challenge. *Periodontol 2000* 2019; **80:** 189–199.
23. World Health Organisation. Noma is a severe disease: It is treatable if detected and managed early. 2016. Available at https://www.afro.who.int/sites/default/files/2017-07/Information_brochure_EN.pdf (accessed October 2022).
24. Farley E, Oyemakinde M J, Schuurmans J *et al.* The prevalence of noma in northwest Nigeria. *BMJ Glob Health* 2020; DOI: 10.1136/bmjgh-2019-002141.
25. Farley E, Mehta U, Srour M L, Lenglet A. Noma (cancrum oris): A scoping literature review of a neglected disease (1843 to 2021). *PLoS Negl Trop Dis* 2021; DOI: 10.1371/journal.pntd.0009844.
26. ZeroNOMA. Available at https://zeronoma.org/ (accessed March 2022).

https://doi.org/10.1038/s41407-023-1749-x