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Necrotizing Periodontal Diseases: Multidisciplinary Perspectives on Pathogenesis, Diagnosis, and Management

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Abstract

Background: Necrotizing periodontal diseases (NPDs) are acute, destructive infections of periodontal tissues characterized by rapid onset, severe pain, and tissue necrosis. They encompass necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis, forming a continuum that can progress to noma in extreme cases.

Aim: To review the pathogenesis, clinical features, diagnostic approach, and management strategies for NPDs, emphasizing multidisciplinary perspectives and preventive measures.

Methods: This narrative review synthesizes current evidence from clinical studies, histopathologic analyses, and epidemiologic data to outline etiologic factors, microbial profiles, and therapeutic protocols.

Results: NPDs primarily affect immunocompromised individuals, those with malnutrition, or under severe psychosocial stress. Key risk factors include HIV infection, hematologic disorders, tobacco use, and poor oral hygiene. Microbial dysbiosis involving Fusobacterium, Prevotella, and spirochetes drives tissue destruction through synergistic virulence and host immune failure. Management centers on early diagnosis, local debridement, chlorhexidine rinses, analgesia, and systemic antibiotics when systemic involvement occurs. Long-term prognosis depends on controlling systemic and behavioral risk factors.

Conclusion: NPDs represent preventable yet aggressive conditions requiring prompt intervention and interprofessional collaboration. Early recognition, risk factor modification, and structured maintenance programs are essential to prevent recurrence and severe complications such as osteonecrosis and noma.

Keywords: Necrotizing periodontal disease, gingivitis, periodontitis, immunosuppression, oral infection, multidisciplinary management

Introduction

Necrotizing periodontal diseases (NPDs) constitute a distinct group of acute inflammatory conditions of the periodontal tissues characterized by rapid tissue destruction, necrosis, and significant patient morbidity. This disease spectrum includes necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis, with noma (cancrum oris) representing the most devastating and advanced manifestation. Rather than being separate disease entities, these conditions are widely regarded as progressive stages along a single pathological continuum, unified by common etiologic factors, overlapping clinical features, and similar underlying biological mechanisms.[1][2] The hallmark clinical presentation of NPDs includes severe pain, gingival necrosis, spontaneous bleeding, and ulceration of the interdental papillae, with progression leading to periodontal attachment loss and, in advanced stages, destruction of alveolar bone. Among the necrotizing periodontal diseases, necrotizing gingivitis represents the earliest and most frequently encountered form. It

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is characterized by necrosis limited primarily to the interdental papillae, which develop a distinctive "punched-out" appearance accompanied by erythema, bleeding, and a grayish pseudomembrane. This stage is most commonly observed in individuals with compromised immune function, including those affected by systemic illness, malnutrition, psychosocial stress, or immunosuppressive conditions. Importantly, necrotizing gingivitis does not initially involve loss of periodontal attachment or alveolar bone, rendering the condition potentially reversible when promptly recognized and appropriately managed. Early intervention can arrest disease progression, restore gingival integrity, and prevent long-term periodontal sequelae. In the absence of timely treatment, however, necrotizing gingivitis may rapidly progress to necrotizing periodontitis, a more destructive condition marked by the extension of necrosis beyond the gingival tissues into the periodontal attachment apparatus. At this stage, patients develop attachment loss, gingival recession, and exposure of underlying bone, often accompanied by the formation of a necrotic pseudomembrane. Systemic manifestations become more prominent and may include fever, malaise, lymphadenopathy, and pronounced halitosis, reflecting the acute inflammatory and infectious nature of the disease.[3] The rapidity with which necrotizing periodontitis can evolve underscores the aggressive biological behavior of NPDs and the critical importance of early diagnosis and intervention.

Further progression into the underlying alveolar bone results in necrotizing stomatitis, a severe condition characterized by osteonecrosis and sequestration of bone. This stage represents a transition from periodontal disease to a more extensive orofacial necrotizing process, in which both soft and hard tissues are affected. Necrotizing stomatitis is associated with profound tissue destruction, severe pain, and significant functional impairment, often in individuals with profound immunosuppression or severe systemic compromise. The disease reflects a breakdown in local and systemic defense mechanisms, allowing unchecked microbial invasion and tissue necrosis. At the extreme end of the disease spectrum lies noma, also known as cancrum oris, a fulminant, noncontagious gangrenous condition of the orofacial region. Noma predominantly affects severely malnourished children and young adults living in low-resource settings, where poverty, poor sanitation, limited access to healthcare, and underlying infectious diseases converge.[4] The disease is characterized by rapid and extensive destruction of facial soft tissues and bone, often leading to catastrophic disfigurement and a high risk of mortality if untreated. Even among survivors, noma results in profound functional, aesthetic, and psychosocial consequences. The progression from necrotizing periodontal disease to noma highlights the shared pathophysiological basis of these conditions and illustrates how local periodontal infection, when compounded by systemic vulnerability, can escalate into life-threatening disease. Collectively, necrotizing periodontal diseases represent a severe but preventable group of conditions whose progression reflects the interplay between microbial challenge, host immune response, and environmental or systemic risk factors. Understanding NPDs as stages of a single disease process reinforces the need for early recognition, comprehensive management, and public health interventions aimed at addressing underlying risk factors to prevent progression to advanced and devastating forms such as necrotizing stomatitis and noma.[1][2][3][4]

Etiology

Necrotizing periodontal diseases (NPDs) are best understood as opportunistic infections that emerge when a pathogenic microbial challenge converges with compromised host defenses and unfavorable local environmental conditions.

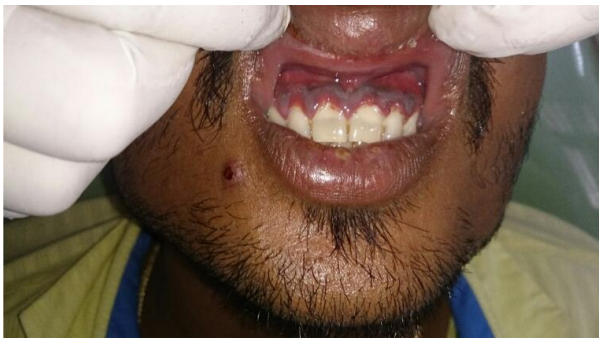

Fig. 1: Necrotizing Gingivitis with Pseudomembrane Formation.

Although these disorders are infectious in nature, the dominant determinant of susceptibility is not exposure to an external pathogen, but rather an impaired capacity of the host to maintain mucosal and periodontal immune surveillance. Accordingly, immunosuppression is consistently identified as the most significant predisposing factor across the NPD spectrum. In clinical practice, cases of necrotizing gingivitis and necrotizing periodontitis occur predominantly—often almost exclusively—among individuals with underlying immunodeficiency, with human immunodeficiency virus (HIV) infection representing the most prominent association. Importantly, the degree of immune compromise appears to correlate with the probability and severity of disease expression. A CD4 count below 200 cells/μL in a patient with HIV has been described as more strongly associated with necrotizing periodontitis than any other recognized risk factor, underscoring the centrality of cell-mediated immune dysfunction in enabling rapid periodontal breakdown.[5] Beyond HIV, multiple systemic conditions that impair neutrophil function, alter inflammatory regulation, or compromise tissue repair have been implicated in the pathogenesis of NPDs.

Hematologic malignancies such as leukemia and states of neutropenia are biologically plausible contributors, as they reduce the cellular arm of innate defense required to contain polymicrobial biofilms. Similarly, diabetes mellitus may increase susceptibility by impairing neutrophil chemotaxis and phagocytosis, increasing inflammatory burden, and compromising microvascular perfusion, thereby diminishing the periodontal tissues' ability to resist microbial invasion and to recover from injury.[6][7] Prolonged exposure to immunosuppressive therapies, including corticosteroids and other immunomodulatory agents, further amplifies vulnerability by dampening protective immune responses and facilitating opportunistic microbial overgrowth. Collectively, these observations reinforce the concept that NPDs frequently represent a clinical endpoint of systemic immune dysregulation manifesting in the periodontium.

While immunosuppression is the dominant substrate, a series of behavioral and environmental factors can substantially influence disease initiation and clinical severity. Tobacco smoking is repeatedly associated with NPDs and is prevalent in many affected cohorts, likely reflecting its adverse effects on gingival microcirculation, immune cell function, and local inflammatory signaling. Severe psychological stress, inadequate sleep, and poor oral hygiene are also recognized contributors, potentially through their combined effects on immune competence, health behaviors, salivary composition, and biofilm accumulation. Notably, NPDs are not limited to immunocompromised individuals. Disease can occur in immunocompetent hosts under sufficiently permissive conditions, highlighting that the etiologic model is multifactorial rather than exclusively immunologic. Malnutrition is especially important in this regard. In young children living in poverty and experiencing protein deficiency, impaired immune development and reduced tissue repair capacity create a biological milieu favorable to necrotizing infection. Malnutrition may also be relevant in populations where dietary quality is poor despite resource availability, such as college students, in whom irregular meals, micronutrient insufficiency, sleep deprivation, and stress may combine to weaken host resistance and promote disease expression.[8] Within this continuum, a prior history of necrotizing gingivitis is often viewed as a risk indicator for progression to necrotizing periodontitis, suggesting either persistent underlying host susceptibility, incomplete resolution of biofilm dysbiosis, or recurrent exposure to the same triggering factors. At the microbial level, necrotizing lesions in NPDs are associated with a characteristic polymicrobial consortium. Organisms frequently identified include Fusobacterium species, Prevotella intermedia, Porphyromonas gingivalis, and Treponema species, all of which are well recognized within the broader ecology of periodontal disease and are capable of contributing to tissue destruction through proteolytic enzymes, pro-inflammatory mediators, and immune evasion strategies.[9] The clinical observation that antibiotic therapy can reduce symptoms during the acute phase lends support to the role of bacterial involvement in the disease process. However, whether these microorganisms function as primary etiologic agents or represent secondary overgrowth in a niche created by immune suppression and tissue breakdown remains uncertain. This uncertainty is reinforced by the fact that NPDs are not considered transmissible in the conventional sense, implying that disease emergence is driven predominantly by endogenous microbial shifts and host vulnerability rather than by acquisition of an external pathogen.[10] In other words, NPDs appear to reflect a failure of equilibrium between resident biofilm communities and host defense mechanisms rather than a classic communicable infection.

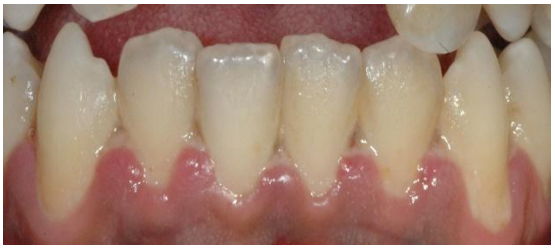

Fig. 2: Necrotizing Gingivitis.

Recent evidence supports an etiologic model based on dysbiosis and synergistic microbial behavior. A study comparing microbial and inflammatory profiles between necrotizing gingivitis and chronic gingivitis identified a predominance of Bacteroidetes—particularly Prevotella species—together with reduced microbial diversity in necrotizing gingivitis. In parallel, affected patients demonstrated elevated serum cytokine levels, suggesting that local infection is accompanied by a measurable systemic inflammatory response. These findings align with the concept that necrotizing gingivitis represents an opportunistic mixed microbial infection in which a dysbiotic community, acting cooperatively, drives rapid tissue necrosis when host defenses are compromised.[11] Reduced microbial diversity may reflect ecological collapse within the periodontal biofilm, allowing expansion of proteolytic, inflammation-promoting taxa that intensify tissue damage and further impair host containment. Despite these advances, the etiology of NPDs remains incompletely defined. Current evidence suggests that preexisting periodontal conditions may undergo an abrupt, destructive exacerbation when permissive host or environmental factors are present.[12] This framing is clinically important because it emphasizes that NPDs may not arise de novo in otherwise healthy periodontal tissues; rather, they may represent a sudden transition from stable gingivitis or subclinical periodontal

inflammation to fulminant necrotizing disease under conditions such as immunosuppression, malnutrition, stress, or smoking. Consequently, etiologic understanding must integrate systemic immunologic status, local periodontal ecology, and contextual behavioral exposures to fully explain disease initiation, progression, and recurrence.

Epidemiology

Necrotizing periodontal diseases (NPDs) are relatively uncommon conditions worldwide, with the global prevalence estimated to be less than 1% of the general population. Despite their rarity, NPDs carry significant clinical importance due to their aggressive course, potential for rapid tissue destruction, and association with systemic vulnerability. The epidemiologic distribution of these diseases is not uniform; rather, it reflects marked disparities related to age, nutritional status, immune competence, and socioeconomic context. Populations most frequently affected include young adults between 18 and 30 years of age, severely malnourished children, and individuals with conditions that compromise immune function, such as HIV infection, hematologic malignancies, or prolonged immunosuppressive therapy. In young adults, the occurrence of NPDs is often linked to a convergence of behavioral and environmental stressors. This age group may experience increased psychological stress, irregular sleep patterns, suboptimal nutrition, tobacco use, and inconsistent oral hygiene practices, all of which can impair local and systemic immune responses. When combined with existing gingival inflammation, these factors may facilitate the transition from relatively benign periodontal disease to necrotizing forms. In contrast, pediatric cases are most frequently observed among malnourished children living in low-resource settings, where poverty, food insecurity, and limited access to healthcare create conditions conducive to disease development and progression. In these environments, inadequate protein intake and micronutrient deficiencies impair immune development and tissue repair, markedly increasing susceptibility to necrotizing infections of the oral cavity.[13]

Geographically, NPDs are more prevalent in regions characterized by lower socioeconomic status and limited healthcare infrastructure. In such settings, barriers to preventive dental care, lack of early intervention, and a higher burden of systemic illness contribute to delayed diagnosis and more severe disease presentations. Conversely, NPDs are relatively uncommon in developed countries, where improved nutrition, widespread access to dental services, and effective management of systemic diseases have reduced both incidence and severity. When cases do occur in high-income nations, they are often associated with identifiable risk factors, such as immunosuppression, substance use, or extreme psychosocial stress, rather than representing sporadic disease in otherwise healthy individuals. Malnutrition remains one of the most important epidemiologic determinants of NPDs, particularly in children. The disease frequently affects those with severe protein-energy malnutrition, underscoring the critical role of nutritional status in maintaining periodontal health and immune competence.[13] Additional epidemiologic contributors include tobacco smoking, which is consistently associated with increased risk due to its adverse effects on gingival blood flow and host immune response, and poor oral hygiene, which promotes biofilm accumulation and microbial dysbiosis.[14][15] Collectively, these epidemiologic patterns highlight that NPDs are diseases of vulnerability, emerging at the intersection of biological susceptibility and adverse social and environmental conditions.

Pathophysiology

The pathophysiology of necrotizing periodontal diseases (NPDs) is best conceptualized as an acute, destructive breakdown of periodontal tissue homeostasis driven by a dysbiotic microbial burden in the setting of compromised host defenses. Although the precise pathogenic sequence remains incompletely elucidated, necrotizing gingivitis is widely considered an opportunistic process in which microorganisms that are ordinarily commensal within the oral biofilm acquire pathogenic potential when local and systemic protective mechanisms are impaired.[16] This framework emphasizes that the initiating event is not necessarily acquisition of a novel pathogen, but rather a shift in the equilibrium between resident microbial communities and the immune and barrier functions of the gingival tissues. Within necrotizing lesions, Fusobacterium species and spirochetes are frequently detected, often in association with other predominantly gram-negative anaerobic organisms.[17] Their recurrent identification has supported their inclusion in the putative microbial signature of NPDs. Nevertheless, the interpretation of these findings remains nuanced. It is not definitively established whether Fusobacterium and spirochetes function as primary etiologic agents that initiate the necrotizing process or whether they expand secondarily within tissue already rendered vulnerable by immunosuppression, malnutrition, psychosocial stress, or other host-level permissive conditions. This ambiguity is clinically relevant because it reinforces the view that microbial presence alone is insufficient to explain disease onset; rather, the host's capacity to regulate biofilm ecology and mount an effective inflammatory response appears to be the critical determinant that governs whether a stable gingival state deteriorates into acute necrosis. From a mechanistic perspective, the rapid progression of tissue destruction in NPDs is consistent with the combined effects of microbial virulence factors and dysregulated host inflammation. Fusobacteria, spirochetes, and related anaerobes can generate a variety of tissue-damaging metabolites and enzymes, including collagenases and other

proteolytic factors that degrade extracellular matrix components. Endotoxins derived from gram-negative bacterial cell walls amplify inflammatory signaling, increasing vascular permeability, leukocyte recruitment, and local tissue edema, changes that can further impair microcirculation and oxygen delivery. In addition, metabolites such as hydrogen sulfide contribute to cytotoxicity and disruption of epithelial integrity, while fibrinolysin can alter local clot stability and tissue repair dynamics.[18] The cumulative effect of these products is accelerated destruction across multiple periodontal compartments, including the gingiva, periodontal ligament, and alveolar bone, accounting for the clinically observed rapid onset of necrosis, ulceration, and, in advanced cases, attachment and bone loss. Host factors are integral to this pathophysiologic cascade. When immune function is compromised, neutrophil activity, phagocytosis, and cytokine regulation may be insufficient to contain bacterial proliferation and invasion. Simultaneously, stress-related neuroendocrine responses and nutritional deficiencies may impair tissue repair and alter salivary and mucosal defenses, facilitating microbial adherence and deeper penetration. Thus, NPDs can be understood as a failure of containment: microbial metabolites and invasion exceed the threshold of host defensive capacity, leading to necrosis and rapid tissue sloughing. This model also explains why NPDs are more common in immunocompromised and malnourished populations and why recurrence may occur unless systemic and behavioral drivers are addressed alongside local periodontal therapy [18].

Histopathology

Histopathologic studies have provided a structured view of the microanatomical progression of necrotizing periodontal diseases and have helped clarify the relationship between microbial colonization and tissue destruction. A landmark contribution was made in 1965 by Listgarten, who utilized electron microscopy to demonstrate spirochete invasion in the condition historically referred to as “necrotizing ulcerative gingivitis.” In this work, four distinct histological zones were described, arranged in order of increasing depth: the bacterial zone, the neutrophil-rich zone, the necrotic zone, and the zone of spirochete infiltration.[19] This zonal framework remains influential because it integrates microbial distribution with host inflammatory response and the degree of tissue structural integrity. The most superficial layer, the bacterial zone, contains a dense and diverse microbial population composed of multiple bacterial morphotypes. This layer reflects heavy plaque-related colonization of the ulcerated gingival surface, where epithelial disruption permits direct microbial contact with underlying tissues. Immediately beneath this lies the neutrophil-rich zone, which is characterized by an intense inflammatory infiltrate dominated by neutrophils, though other leukocyte populations may also be present. This zone represents the host’s acute innate immune attempt to contain infection at the interface between colonized surface debris and viable connective tissue. The density of neutrophils and associated inflammatory exudate in this region aligns with the clinical observation of spontaneous bleeding and purulent or pseudomembranous surface material [19].

Deeper still is the necrotic zone, a region marked by extensive tissue breakdown. Histologically, it contains large numbers of dead cells, disintegrating leukocytes, and fragmented connective tissue elements. The extracellular matrix architecture is severely disrupted, and the tissue microenvironment reflects a culmination of microbial enzymatic activity, inflammatory mediator release, ischemic injury, and impaired repair. Importantly, spirochetes and other bacterial species can be observed not only in the superficial bacterial zone but also within the neutrophil-rich and necrotic zones, indicating that multiple organisms participate in the destructive milieu of the superficial and intermediate tissue planes. The deepest region described is the zone of spirochete infiltration, which is distinctive because it contains structurally intact tissue into which spirochetes penetrate. In Listgarten’s observations, while spirochetes could be identified across the first three zones, they were uniquely present as the predominant organism within this deepest infiltrative layer.[19] This finding supports the concept that spirochetes may possess enhanced invasive capacity relative to other organisms in the lesion, enabling them to traverse tissue planes and potentially contribute to progression into deeper periodontal structures. Clinically, the histologic pattern aligns with the aggressive behavior of NPDs, where tissue necrosis and ulceration can expand rapidly if host defenses are insufficient. The zonal model therefore provides a coherent microscopic correlate to the clinical phenotype of necrotizing periodontal disease and remains a useful conceptual tool for understanding how microbial invasion, acute inflammation, and tissue necrosis coexist within a single evolving lesion.[19]

History and Physical

Necrotizing periodontal diseases (NPDs) characteristically present in individuals with systemic or local conditions that impair immune competence or substantially alter host response. A history of immunosuppression—whether related to systemic disease, medical therapy, malnutrition, or chronic stress—is frequently elicited during clinical assessment. Significant psychological stress has long been recognized as a contributory factor, likely through neuroendocrine-mediated suppression of immune function and neglect of oral self-care. Tobacco smoking represents one of the most consistent and important behavioral risk factors

associated with disease onset and progression, owing to its detrimental effects on gingival microcirculation, inflammatory regulation, and tissue healing capacity. Careful exploration of these risk factors during history-taking is essential, as they provide critical context for both diagnosis and subsequent management planning. Patients with NPDs typically describe an acute and dramatic onset of symptoms, often developing over the course of hours to days. Severe pain is a defining complaint and serves as a key distinguishing feature when compared with other forms of periodontal disease, which are frequently painless or only mildly uncomfortable. The pain is often described as sharp, throbbing, or burning and may be exacerbated by eating, speaking, or tooth brushing. Fetid breath is another prominent symptom and reflects the presence of necrotic tissue and anaerobic bacterial metabolism. In cases of necrotizing periodontitis, rapid destruction of the periodontal attachment apparatus can occur within days, leading to noticeable changes in tooth support and, in severe cases, tooth mobility. Some patients also report a metallic taste or spontaneous bleeding, further contributing to distress and functional impairment [19][20].

Systemic manifestations may accompany local findings, particularly in more advanced stages of disease. Regional lymphadenopathy is not uncommon and reflects the acute inflammatory and infectious nature of the condition. Fever, malaise, and general weakness may be present, especially when tissue necrosis is extensive or when the host's immune response is significantly compromised. Pain severity often leads patients to reduce or entirely abandon oral hygiene practices, which paradoxically exacerbates plaque accumulation, worsens halitosis, and accelerates disease progression. Additionally, oral intake may be significantly reduced due to pain, contributing to dehydration, nutritional compromise, and overall malaise, thereby reinforcing a cycle of systemic and local deterioration.[20] On physical examination, tissue necrosis is the hallmark clinical feature of NPDs and is most readily identified within the gingival tissues. The interdental papillae are typically affected first and display a characteristic punched-out or cratered morphology. The gingiva appears ulcerated, friable, and necrotic, often covered by a grayish-white or yellow pseudomembrane composed of plasma proteins, fibrin, necrotic debris, and inflammatory cells. This pseudomembrane can often be gently removed, revealing an underlying bleeding and highly painful surface. As disease progresses from necrotizing gingivitis to necrotizing periodontitis, destruction extends beyond the gingival margin into the periodontal attachment apparatus, resulting in loss of connective tissue attachment and resorption of alveolar bone [20].

A distinctive linear erythematous band commonly surrounds the necrotic area, sharply demarcating the ulcerated tissue from adjacent clinically normal attached gingiva, free gingiva, and alveolar mucosa. Bleeding may occur spontaneously or with minimal provocation, reflecting both vascular fragility and intense inflammation. Although attachment loss is the defining feature of necrotizing periodontitis, deep periodontal pockets are surprisingly uncommon. This apparent paradox is explained by the rapid necrosis and sloughing of the junctional epithelium, which limits the time available for pocket formation before tissue destruction occurs. In the most advanced forms, particularly necrotizing stomatitis, the destructive process extends beyond the confines of the gingiva and periodontal tissues. Mucosal involvement surpasses the mucogingival junction, leading to exposure of underlying bone. Alveolar necrosis may ensue, sometimes accompanied by sequestration of devitalized bone fragments. These findings signal a severe breakdown of local defense mechanisms and are often associated with profound systemic compromise. Recognition of these clinical features during physical examination is crucial, as they indicate both disease severity and the need for urgent, comprehensive intervention to prevent further tissue loss and systemic complications.[20]

Evaluation

The diagnosis of necrotizing periodontal diseases (NPDs) is primarily clinical and relies on the integration of characteristic intraoral findings with relevant patient history and risk factors. Unlike many chronic periodontal conditions that require extensive adjunctive testing, NPDs exhibit distinctive features that are often sufficient to establish the diagnosis at the bedside or dental chair. A thorough intraoral examination is essential and should focus on identifying gingival necrosis, punched-out interdental papillae, ulceration, spontaneous bleeding, pseudomembrane formation, and the extent of periodontal attachment loss. Because disease severity may progress rapidly, careful documentation of the distribution and depth of tissue involvement is important for both baseline assessment and subsequent monitoring of therapeutic response. Intraoral evaluation should always be complemented by a detailed extraoral examination. Assessment of regional lymph nodes, particularly the submandibular and cervical chains, is necessary to identify lymphadenopathy, which may indicate an acute inflammatory or infectious response. Facial inspection should be performed to detect asymmetry, swelling, or skin involvement, findings that may suggest extension beyond the periodontal tissues, particularly in advanced cases such as necrotizing stomatitis. Evaluation of facial contours, jaw mobility, and signs of trismus can provide additional clues to the presence of deeper tissue involvement or secondary complications. Histopathologic examination via biopsy generally has limited diagnostic utility in NPDs and is not routinely indicated. When performed, biopsy specimens

typically reveal nonspecific inflammatory changes, tissue necrosis, and microbial colonization rather than features that definitively distinguish NPDs from other ulcerative or necrotic oral conditions. As a result, biopsy is usually reserved for atypical presentations, failure to respond to appropriate therapy, or situations in which malignancy, autoimmune disease, or other unusual pathology is suspected. In most cases, clinical findings and disease course provide sufficient information to guide diagnosis and management [21].

Radiographic imaging plays an important adjunctive role, particularly in assessing the extent of alveolar bone involvement and in differentiating necrotizing periodontitis from necrotizing stomatitis or other osseous pathologies. Conventional intraoral or panoramic radiographs may demonstrate a spectrum of findings ranging from minimal or subtle crestal bone loss to extensive areas of destruction. In cases involving bone necrosis, radiographic features may include irregular, patchy, or "moth-eaten" radiolucencies, reflecting areas of osteolysis and devitalized bone. These appearances can resemble those seen in medication-related osteonecrosis of the jaw, osteoradionecrosis, or chronic osteomyelitis, necessitating careful clinical correlation. Radiopaque sequestra, representing fragments of necrotic bone, may also be visible in more advanced disease. Computed tomography (CT) offers superior spatial resolution and detailed visualization of osseous and soft tissue structures and is particularly valuable when the extent of bone involvement is unclear or when differentiating among various causes of jaw necrosis. CT imaging can delineate cortical disruption, sequestration, and soft tissue extension, thereby assisting in diagnostic clarification and treatment planning. However, imaging findings alone are insufficient to establish a definitive diagnosis, which must ultimately be based on characteristic clinical features and a compatible patient history. Laboratory investigations are an important component of the evaluation, especially in patients with severe, recurrent, or atypical presentations. Blood tests may be indicated to assess for underlying systemic conditions that predispose to NPDs, such as leukemia, neutropenia, agranulocytosis, or other hematologic abnormalities. In some cases, unexplained or recurrent episodes of necrotizing periodontal disease may serve as an early clinical marker of an undiagnosed systemic disorder, prompting more comprehensive medical evaluation. Identifying and addressing such underlying conditions is essential, as successful management of NPDs often depends not only on local therapy but also on correction or stabilization of systemic risk factors [21].

Treatment / Management

The management of necrotizing periodontal diseases (NPDs), including necrotizing gingivitis and necrotizing periodontitis, is centered on prompt local therapy aimed at controlling infection, alleviating pain, and halting rapid tissue destruction. The therapeutic approach is tailored to the severity and extent of disease, as well as the patient's systemic condition and ability to tolerate intervention. In most cases, initial treatment focuses on conservative, nonsurgical measures designed to stabilize the acute phase before definitive periodontal rehabilitation is undertaken. Early intervention is critical, as timely management can prevent progression to more destructive forms and reduce systemic morbidity. Local therapy constitutes the cornerstone of treatment and begins with gentle mechanical debridement of necrotic and plaque-laden tissues. This is typically performed under local anesthesia to minimize patient discomfort, given the intense pain associated with these conditions. Debridement aims to remove bacterial biofilm, necrotic debris, and inflamed tissue, thereby reducing microbial load and facilitating healing. In conjunction with mechanical cleaning, removal of the superficial pseudomembrane is an important step. This is commonly achieved using a cotton pellet moistened with a 0.12% chlorhexidine solution, allowing for atraumatic cleansing while simultaneously delivering an antimicrobial agent to the affected tissues. The elimination of this pseudomembrane not only reduces bacterial burden but also improves tissue oxygenation and patient comfort. Adjunctive chemical plaque control plays a key role during the acute phase, particularly when pain limits effective tooth brushing. Patients are typically advised to use a 0.12% chlorhexidine mouthwash or, in some cases, a diluted hydrogen peroxide rinse. These agents provide antimicrobial activity, reduce plaque accumulation, and assist in controlling halitosis. Reinforcement of oral hygiene practices is essential, but instructions must be adapted to the patient's tolerance level, emphasizing gentle techniques and gradual resumption of routine care as pain subsides. Analgesia is often required, and nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly prescribed to relieve pain and reduce inflammation, improving the patient's ability to eat, drink, and maintain oral hygiene [21].

Systemic antibiotics are not routinely required in all cases of NPDs but are indicated as an adjunct to local therapy when systemic involvement is evident. Clinical signs such as fever, malaise, regional lymphadenopathy, or failure to respond adequately to local measures suggest bacterial dissemination or an exaggerated inflammatory response, warranting antimicrobial support.[21] Metronidazole is generally considered the antibiotic of choice due to its effectiveness against anaerobic organisms commonly implicated in necrotizing periodontal lesions. Amoxicillin may be used as an alternative or adjunctive agent, particularly when metronidazole is contraindicated or poorly tolerated. Antibiotic therapy should always be complemented,

rather than replaced, thorough local debridement, as pharmacologic treatment alone is insufficient to control disease progression. An essential component of management involves identification and modification of underlying risk factors that predispose disease onset and recurrence. Tobacco smoking cessation should be strongly encouraged, as continued smoking impairs healing and increases the likelihood of relapse. Psychological stress, which may contribute to immune dysregulation and neglect of oral hygiene, should be addressed through counseling, stress-reduction strategies, or referral to appropriate support services when indicated. Nutritional status must also be assessed, particularly in patients with signs of malnutrition or inadequate dietary intake. Improving nutritional balance enhances immune function and tissue repair, both of which are critical for recovery. Patients diagnosed with necrotizing periodontitis should undergo medical evaluation to identify potential systemic conditions associated with immunosuppression. Disorders such as HIV infection, hematologic disease, or metabolic conditions may underlie the periodontal presentation and must be managed concurrently to achieve sustained disease control. Coordination between dental and medical providers is therefore essential, ensuring that systemic therapy and periodontal treatment proceed in parallel [21].

Supportive care recommendations play a valuable role in symptom control and recovery. Patients are often advised to maintain adequate hydration, avoid spicy or irritating foods that may exacerbate pain, and ensure sufficient rest and sleep to support immune function. Vitamin supplementation, particularly in individuals with nutritional deficiencies, may further aid tissue healing. Close follow-up is critical, both to monitor resolution of the acute phase and to initiate definitive periodontal therapy once inflammation has subsided. Following resolution of active necrosis, residual soft and hard tissue defects may remain, particularly in cases of necrotizing periodontitis. These defects can complicate plaque control and predispose to recurrent disease. Surgical intervention may therefore be indicated in selected patients. Minor soft tissue irregularities can be addressed with gingivoplasty to recontour gingival margins and facilitate hygiene. More extensive bony defects may require osteoplasty to restore physiologic architecture and improve long-term periodontal stability. Such procedures are performed only after disease stabilization and emphasize the importance of phased management in optimizing both functional and esthetic outcomes [21].

Differential Diagnosis

Accurate diagnosis of necrotizing periodontal diseases (NPDs) requires careful differentiation from a range of oral and systemic conditions that can present with gingival ulceration, pain, erythema, bleeding, or tissue sloughing. Because NPDs are diagnosed clinically and often occur in patients with systemic vulnerability, the evaluation should combine a meticulous oral examination with targeted history-taking and appropriate ancillary testing, particularly when the presentation is atypical, unusually severe, recurrent, or unresponsive to standard therapy.[22] The diagnostic challenge arises from the fact that several disorders can mimic individual components of the NPD phenotype—such as necrosis, pseudomembrane formation, or gingival bleeding—yet differ substantially in etiology, prognosis, and required treatment. Oral mucositis, for example, may produce painful erythema, ulceration, and pseudomembrane-like sloughing, particularly in patients undergoing chemotherapy or radiotherapy. However, mucositis typically follows a treatment-related timeline and often involves diffuse mucosal surfaces rather than the interdental papilla–predominant pattern that is characteristic of NPDs. Linear gingival erythema may occur in immunocompromised individuals, especially those with HIV, and can be confused with early necrotizing disease due to erythema and bleeding. Nonetheless, it is usually distinguished by a linear band of erythema along the gingival margin without the punched-out papillary necrosis typical of NPDs. Viral conditions, particularly herpes simplex virus infection, can cause painful ulcerations and systemic symptoms, but lesions often occur on keratinized mucosa with vesicular precursors and do not typically produce the papillary cratered necrosis that defines necrotizing gingivitis [22].

Nutritional deficiencies, such as scurvy, may present with gingival swelling, bleeding, and impaired wound healing, sometimes leading to ulceration. The broader clinical context—dietary history, systemic signs, and laboratory confirmation of vitamin C deficiency—helps differentiate scurvy from NPDs. Gingivostomatitis may also resemble NPDs when severe inflammation and ulceration are present, but the distribution is often more generalized across oral mucosa rather than focused necrosis of interdental papillae. Desquamative gingivitis, often associated with mucocutaneous diseases such as lichen planus or pemphigoid, can present with erythema, epithelial sloughing, and pain; however, it typically exhibits chronicity and diffuse mucosal fragility rather than the acute necrotizing pattern of NPDs. Invasive fungal disease is a critical differential consideration in profoundly immunosuppressed individuals because it may cause rapidly progressive necrosis of oral tissues. Differentiation is essential because invasive fungal infections can be life-threatening and require urgent systemic antifungal therapy and often surgical debridement. Illicit-drug-related gingival disease may present with ulceration, necrosis, and poor oral hygiene, but history of substance use and lesion distribution assist in distinguishing it from classic NPDs. Systemic hematologic disorders such as agranulocytosis and

leukemia can produce gingival ulceration, necrosis, and spontaneous bleeding due to impaired immune defense and thrombocytopenia. These conditions may either mimic NPDs or coexist with it, making complete blood count and clinical correlation indispensable in suspicious cases.[22] Finally, chronic periodontitis remains an important comparator because it can involve attachment loss and bone destruction; however, chronic periodontitis usually progresses slowly, is often painless, and lacks the acute severe pain and necrotic interdental crater formation that typify NPDs. Taken together, rigorous clinical correlation and judicious testing are necessary to ensure accurate diagnosis and to avoid missing systemic illness or invasive infection.[22]

Staging

Contemporary classification frameworks recognize necrotizing periodontal diseases as a unified category characterized by ulceration and tissue necrosis within the periodontium, with clinical subsets defined by the anatomic scope and depth of tissue destruction. According to the 2017 classification system proposed by the American Academy of Periodontology, NPDs comprise three principal subsets: necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis. This staging approach reflects the concept of a disease continuum, in which increasing severity corresponds to progressively deeper and more extensive tissue involvement.[23] The clinical utility of this framework lies in its ability to guide diagnostic precision, therapeutic intensity, and prognostic counseling. Necrotizing gingivitis represents the most localized subset, in which necrosis and ulceration are confined to the gingival tissues. Clinically, this stage is defined by painful interdental papillary necrosis, punched-out crater-like lesions, spontaneous bleeding, and pseudomembrane formation, yet without confirmed attachment loss or destruction of supporting periodontal structures. In this subset, intervention is typically directed at controlling the acute infection, restoring hygiene, and addressing precipitating risk factors, and the potential for reversibility is relatively high when treatment is prompt. Progression to necrotizing periodontitis is marked by extension of the necrotizing process beyond the marginal gingiva into the periodontal attachment apparatus. In this stage, destruction affects the periodontium adjacent to each tooth and includes involvement of gingival tissues, periodontal ligament, and alveolar bone.[23] Clinically, attachment loss and gingival recession may occur rapidly, sometimes within days, differentiating this entity from chronic periodontitis, which typically evolves over months to years. The rapidity of destruction also explains why deep periodontal pockets may be less prominent than expected despite significant attachment loss, as necrosis and sloughing can precede stable pocket formation. Necrotizing periodontitis often signals substantial host compromise and frequently warrants medical evaluation for immunosuppressive conditions and intensified follow-up. Necrotizing stomatitis represents the most extensive periodontal subset, characterized by destruction that exceeds the confines of the gingiva and periodontium and extends beyond the mucogingival junction. Involvement may spread into the oral mucous membranes of adjacent sites such as the cheek, tongue, or palate, reflecting a broader necrotizing process with high risk of bone exposure, osteonecrosis, and sequestration.[23] This stage typically occurs in individuals with profound systemic compromise and requires urgent multidisciplinary management. The staging framework therefore operationalizes severity based on anatomical extent, allowing clinicians to align intervention strategies with disease burden and systemic risk [23].

Prognosis

The prognosis of necrotizing periodontal diseases is variable and is influenced less by local lesion appearance alone than by the host context in which the disease occurs. A defining feature of NPDs is their tendency toward recurrence, making regular recall visits and structured maintenance programs essential for early detection of reemergence and prompt retreatment. Prognostic outcomes depend heavily on the effectiveness of risk factor control, as the same vulnerabilities that facilitate disease onset—immunosuppression, malnutrition, tobacco exposure, poor oral hygiene, and psychosocial stress—also promote relapse when unaddressed. Consequently, durable remission often requires a dual focus: local periodontal stabilization and systematic modification of precipitating factors. Improved outcomes are consistently associated with restoration and maintenance of effective oral hygiene, as reduction of biofilm burden decreases the likelihood of dysbiosis and recurrent necrosis. Adequate nutrition, particularly sufficient protein and micronutrient intake, supports immune competence and tissue repair and is especially important in pediatric and socioeconomically vulnerable populations. Stress reduction and sleep optimization may improve host response and facilitate adherence to daily care. Smoking cessation is another strong prognostic determinant, as continued tobacco use is associated with impaired healing and persistent susceptibility. The prognosis is also closely tied to management of underlying systemic disease. In patients with HIV, for example, appropriate antiretroviral therapy may restore immune function and significantly reduce recurrence risk and disease severity. Similar principles apply to other immunocompromising states, in which medical stabilization can shift the host environment away from permissive conditions for necrotizing infection. Thus, prognosis is favorable when both periodontal therapy and systemic risk

mitigation are achieved, but guarded when immune dysfunction, malnutrition, or harmful behaviors persist [23].

Complications

Complications of necrotizing periodontal diseases arise from the aggressive tempo and destructive nature of these conditions, particularly when diagnosis is delayed, treatment is incomplete, or underlying systemic factors remain uncontrolled. The most direct sequelae involve progressive loss of periodontal attachment, which can culminate in tooth mobility and eventual tooth loss. Because NPDs may advance rapidly, the window for preventing irreversible structural damage can be narrow, and even short delays may result in substantial tissue compromise. Extensive soft tissue necrosis is another major complication and may produce persistent gingival deformity, interdental cratering, and mucosal defects that complicate plaque control and predispose to recurrence. When disease extends into supporting bone, alveolar bone exposure may occur, sometimes accompanied by osteonecrosis and sequestration of devitalized bone fragments. Sequestra can perpetuate inflammation, cause persistent pain, and may require surgical removal after acute infection resolves. Systemic complications are also possible. Bacteremia may occur in severe cases, particularly in individuals with poor host defense, and may contribute to systemic inflammatory symptoms or complicated comorbid conditions. Functional consequences, including reduced oral intake due to pain, can precipitate weight loss and dehydration, further weaken immune competence and perpetuate a cycle of vulnerability.[24] These risks underscore the need for clinician vigilance, timely escalation when disease is extensive, and strong emphasis on adherence to both acute therapy and maintenance care. Preventive strategies and interprofessional collaboration are central to reducing the overall disease burden, particularly in patients with systemic compromise or socioeconomic barriers to care.

Patient Education

Prevention of necrotizing periodontal diseases is fundamentally oriented toward early recognition and modification of risk factors that render the host susceptible to opportunistic periodontal necrosis. Patient education should prioritize the role of meticulous oral hygiene, as consistent biofilm control reduces the microbial burden and helps prevent dysbiosis that can precipitate acute necrotizing episodes. Counseling should also address nutrition, emphasizing a balanced diet with adequate protein intake and sufficient micronutrients to support immune function and tissue repair. This point is particularly critical in children and individuals facing food insecurity, where nutritional intervention may be as important as local periodontal therapy in preventing recurrence. Effective stress management and improved sleep hygiene should be included in preventive counseling, as psychological stress and sleep deprivation may impair immune regulation and reduce adherence to daily oral care. Smoking cessation is essential given the strong association between tobacco exposure and NPD risk; patients should be informed that smoking compromises gingival perfusion, impairs healing, and increases recurrence likelihood. Importantly, immunocompromised individuals—including those with HIV, leukemia, or on immunosuppressive therapy—should be explicitly counseled regarding their heightened susceptibility and the need for earlier evaluation when symptoms arise. Education should emphasize recognition of early warning signs such as acute gingival pain, spontaneous bleeding, malodor, and the characteristic punched-out interdental papillae, as prompt presentation at the onset of symptoms can prevent progression to necrotizing periodontitis and more destructive disease stages. The prior history of NPD is a clinically meaningful risk marker for recurrence; therefore, regular monitoring and maintenance visits should be framed as a preventive necessity rather than optional follow-up. Timely dental care at the first indication of relapse substantially improves outcomes and reduces complication rates [24].

Enhancing Healthcare Team Outcomes

Optimizing outcomes in necrotizing periodontal diseases requires coordinated care that extends beyond the oral cavity and addresses the systemic and behavioral drivers of disease susceptibility. NPDs are clinical diagnoses characterized by acute onset, severe pain, gingival necrosis, interdental ulceration, and, in advanced stages, osteonecrosis. General dentists and primary oral healthcare providers must therefore be proficient in recognizing the classic phenotype and distinguishing it from mimicking conditions. They also play a pivotal role in initiating early local therapy, controlling acute infection, and delivering effective counseling regarding modifiable risk factors, including tobacco use, oral hygiene practices, nutrition, and stress. Because recurrence is common, the clinical team must establish structured follow-up and maintenance pathways to detect early relapse and reinforce preventive behaviors. Complex cases frequently require referral to periodontists or oral and maxillofacial surgeons, particularly when necrosis is extensive, when bony involvement is suspected, or when reconstructive procedures are needed after acute disease resolution. Long-term rehabilitation may involve periodontal surgery to correct soft tissue defects or osteoplasty to manage bony irregularities, which necessitates specialist oversight. Crucially, because NPDs often occur in the setting of systemic immunocompromise, dental management must be integrated with medical evaluation. Coordination with physicians and relevant medical specialists enables investigation for underlying conditions such as HIV infection, hematologic disease, or metabolic dysfunction and ensures that systemic therapy is

optimized alongside periodontal treatment. In vulnerable populations, collaboration with nutritionists, social workers, and primary care teams can address malnutrition, socioeconomic barriers, and adherence challenges. Through such interprofessional coordination—linking early diagnosis, effective local therapy, systemic stabilization, and long-term maintenance—patient outcomes improve, recurrence decreases, and the risk of severe complications is minimized.

Role of Dentists, Radiologists, Pathologists, Nursing, and Health Assistant:

Effective management of necrotizing periodontal diseases (NPDs) requires a coordinated, multidisciplinary approach. Dentists serve as the primary point of care, responsible for early recognition of hallmark signs such as gingival necrosis, pseudomembrane formation, and interdental papillary ulceration. They initiate local therapy through mechanical debridement, antimicrobial rinses, and pain control, while educating patients on oral hygiene and risk factor modification. Dentists also play a critical role in identifying systemic associations—such as HIV or hematologic disorders—and ensuring timely referral for medical evaluation. Radiologists contribute by assessing the extent of osseous involvement, particularly in advanced cases like necrotizing stomatitis. Imaging modalities, including panoramic radiographs and CT scans, help differentiate NPDs from other jaw pathologies and detect complications such as osteonecrosis or sequestration of bone fragments. Accurate imaging interpretation guides surgical planning and prevents misdiagnosis of conditions mimicking NPDs. Nursing professionals provide essential supportive care, including pain management, hydration monitoring, and reinforcement of oral hygiene instructions. They assist in implementing infection control measures, facilitate patient education on nutrition and stress reduction, and monitor systemic symptoms such as fever or lymphadenopathy that may indicate disease progression. Health assistants play a vital role in patient follow-up and continuity of care. They help schedule recall visits, track adherence to prescribed therapies, and support community-based preventive programs targeting malnutrition and tobacco cessation. Their involvement ensures that patients maintain long-term oral health and reduces recurrence risk. Collectively, this interprofessional collaboration enhances diagnostic accuracy, accelerates treatment, and addresses systemic vulnerabilities, ultimately improving patient outcomes and minimizing severe complications such as noma and osteonecrosis.

Conclusion:

Necrotizing periodontal diseases remain a significant clinical challenge due to their rapid progression, severe morbidity, and strong association with systemic vulnerability. Although uncommon globally, their impact is profound, particularly in immunocompromised or malnourished populations. The continuum from necrotizing gingivitis to necrotizing stomatitis underscores the importance of early detection and timely intervention. Successful management hinges on a dual approach: immediate local therapy to control infection and comprehensive strategies to address underlying systemic and behavioral risk factors. Tobacco cessation, nutritional optimization, stress reduction, and medical stabilization of immunosuppressive conditions are critical for durable remission. Interprofessional collaboration between dental and medical teams enhances diagnostic accuracy, facilitates systemic evaluation, and ensures holistic care. Preventive education plays a pivotal role, empowering patients to recognize early symptoms and maintain effective oral hygiene. Ultimately, prognosis improves when clinicians adopt a proactive, multidisciplinary model that integrates acute treatment with long-term maintenance and risk mitigation. By prioritizing early recognition and systemic health, healthcare providers can significantly reduce recurrence, prevent complications such as osteonecrosis and noma, and improve overall patient outcomes.

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