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Review Article

Acute Necrotizing Ulcerative Gingivitis: A Comprehensive Review of Etiopathogenesis, Clinical Features and Management

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Abstract

Acute Necrotizing Ulcerative Gingivitis (ANUG) is a rapidly progressive periodontal infection characterized by gingival necrosis, ulceration, spontaneous bleeding, pain, and fetid oral odor. Historically referred to as "trench mouth," the disease gained prominence during World War I due to its high prevalence among soldiers exposed to poor oral hygiene, malnutrition, stress, and inadequate living conditions. ANUG is now recognized as an opportunistic polymicrobial infection associated with an imbalance of the normal oral flora, particularly fusiform bacilli and spirochetes, in susceptible individuals. Multiple local and systemic predisposing factors including smoking, emotional stress, malnutrition, immunosuppression, hematological disorders, and human immunodeficiency virus infection contribute to disease development. Clinically, the condition presents with characteristic punched-out interdental papillae covered by a gray pseudomembrane, spontaneous gingival bleeding, halitosis, excessive salivation, and severe pain. If untreated, the disease may progress to necrotizing ulcerative periodontitis, necrotizing stomatitis, or noma. Early recognition and prompt management are crucial for preventing complications and preserving periodontal health. This review discusses the epidemiology, etiopathogenesis, risk factors, microbiology, clinical manifestations, diagnosis, differential diagnosis, complications, treatment strategies, and preventive measures associated with ANUG.

Keywords: Acute Necrotizing Ulcerative Gingivitis; Trench Mouth; Necrotizing Periodontal Disease; Fusospirochetal Infection; Noma; Periodontal Infection

Abbreviations

ANUG: Acute Necrotizing Ulcerative Gingivitis; NUG: Necrotizing Ulcerative Gingivitis; NUP: Necrotizing Ulcerative Periodontitis; HIV: Human Immunodeficiency Virus; AIDS: Acquired Immunodeficiency Syndrome; WHO: World Health Organization.

Introduction

Acute Necrotizing Ulcerative Gingivitis (ANUG) is one of the most distinctive and painful periodontal diseases encountered in clinical practice. It is characterized by rapid destruction of gingival tissues resulting in ulceration, necrosis, bleeding, and severe

discomfort. The disease belongs to the spectrum of necrotizing periodontal diseases, which also includes necrotizing ulcerative periodontitis and necrotizing stomatitis [1].

The condition became widely recognized during World War I when large numbers of soldiers developed painful gingival lesions while serving in overcrowded trenches. Consequently, ANUG became commonly known as "trench mouth". Subsequent investigations demonstrated that the disease was not contagious but rather an endogenous opportunistic infection resulting from the interaction of pathogenic microorganisms with compromised host defenses [3].

The prevalence of ANUG has decreased substantially in developed countries because of improvements in oral hygiene practices, nutrition, and healthcare access. Nevertheless, the disease continues to occur among individuals exposed to stress, tobacco use, poor oral hygiene, malnutrition, and immunosuppressive conditions. In developing regions and among socioeconomically disadvantaged populations, ANUG remains an important oral health concern [2].

The pathogenesis of ANUG is multifactorial and involves a complex interaction between bacterial colonization, host immune response, and environmental risk factors. Understanding these factors is essential for accurate diagnosis, effective treatment, and prevention of recurrence [1].

Historical Background

The earliest descriptions of ulcerative gingival disease date back to ancient Greek and Roman medical literature. However, ANUG gained widespread medical attention during the First World War because of its frequent occurrence among soldiers stationed in trenches. The combination of poor oral hygiene, emotional stress, nutritional deficiencies, sleep deprivation, and tobacco use created ideal conditions for disease development [4].

The term "Vincent's infection" was later introduced because of the association of fusiform bacilli and spirochetes with the disease process. Over time, advances in microbiology and immunology clarified that ANUG is a polymicrobial opportunistic infection rather than a contagious disease [4].

Epidemiology

ANUG primarily affects adolescents and young adults, particularly individuals between 15 and 35 years of age. The disease has a higher prevalence among smokers, military personnel, university students during examination periods, and patients experiencing significant psychological stress [7].

The prevalence is considerably higher among immunocompromised individuals, particularly those infected with HIV. In developing countries, malnutrition and inadequate oral hygiene contribute significantly to disease occurrence. The condition may also affect children suffering from severe nutritional deficiencies and systemic illnesses [8].

Etiology and risk factors

The development of ANUG requires the coexistence of pathogenic microorganisms and host susceptibility factors [5].

Local factors

- **Poor Oral Hygiene:** Accumulation of dental plaque provides a favorable environment for anaerobic bacterial proliferation and tissue destruction [6].
- **Existing Gingivitis:** Pre-existing gingival inflammation increases susceptibility to necrotizing lesions [11].
- **Faulty Dental Restorations:** Overhanging restorations and plaque-retentive surfaces promote bacterial colonization [10].
- **Tobacco Smoking:** Smoking impairs neutrophil function, reduces gingival blood flow, and compromises host immune responses, significantly increasing the risk of ANUG [9].

Systemic factors

- **Psychological Stress:** Stress-induced alterations in cortisol levels and immune function contribute significantly to disease initiation and progression [13].
- **Malnutrition:** Protein-energy malnutrition and micronutrient deficiencies impair host defense mechanisms and tissue repair [15].
- **Hematological Disorders:** Leukemia, neutropenia, and aplastic anemia are frequently associated with severe necrotizing gingival lesions [14].
- **Human Immunodeficiency Virus Infection:** HIV-associated immunosuppression predisposes patients to severe necrotizing periodontal diseases, including NUG and NUP [17].

- **Debilitating Systemic Diseases:** Any systemic condition causing immune dysfunction may increase susceptibility to ANUG [12].

Microbiology of acute necrotizing ulcerative gingivitis

ANUG is considered a polymicrobial anaerobic infection. The microbial flora associated with the disease differs significantly from that found in healthy gingival tissues. The microorganisms most consistently isolated from ANUG lesions include:

- Treponema species (spirochetes)
- Fusobacterium species
- Selenomonas species
- Prevotella intermedia
- Bacteroides species
- Porphyromonas species

Microscopic examination of affected tissues demonstrates extensive infiltration by spirochetes extending into deeper connective tissues. Fusiform bacilli and other anaerobic organisms contribute to tissue destruction through the production of endotoxins, proteolytic enzymes, and inflammatory mediators [20].

The disease develops when ecological changes within the oral cavity favor the proliferation of these pathogenic microorganisms. Host immune suppression further enhances bacterial invasion and accelerates tissue necrosis [21].

Pathogenesis

The pathogenesis of ANUG is multifactorial and involves an interaction between pathogenic microorganisms, host immune responses, and environmental factors [19].

Bacterial colonization

Accumulation of dental plaque creates an anaerobic environment that supports the growth of fusiform bacilli and spirochetes. These organisms produce toxins and enzymes capable of damaging epithelial and connective tissues [18].

Host immune dysfunction

Neutrophils constitute the primary defense against periodontal pathogens. Conditions that impair neutrophil function or reduce immune competence increase susceptibility to ANUG [16].

Tissue destruction

Bacterial endotoxins stimulate inflammatory cytokine production, resulting in:

- Vascular damage
- Epithelial ulceration
- Connective tissue destruction
- Gingival necrosis

The resulting tissue damage creates an environment that further favors anaerobic bacterial growth, producing a self-perpetuating cycle of infection and necrosis [22].

Role of stress

Psychological stress alters immune function through increased cortisol secretion. Elevated cortisol levels suppress cellular immunity, impair neutrophil activity, and enhance susceptibility to periodontal infections [24].

Role of smoking

Smoking contributes to disease progression through:

- Reduced gingival blood flow
- Impaired neutrophil chemotaxis
- Altered cytokine production
- Delayed wound healing

These effects significantly increase the risk and severity of ANUG [26].

Classification of necrotizing periodontal diseases

Necrotizing periodontal diseases are generally classified into three major categories [25].

Necrotizing ulcerative gingivitis (NUG)

The disease is confined to gingival tissues without loss of periodontal attachment [24].

Necrotizing ulcerative periodontitis (NUP)

The disease extends beyond the gingiva and involves destruction of periodontal ligament and alveolar bone [23].

Necrotizing stomatitis

The necrotizing process extends beyond periodontal tissues into adjacent oral mucosa and deeper structures.

Severe untreated cases may progress further into noma [8].

Clinical features

Symptoms

Patients commonly complain of:

- Severe gingival pain
- Spontaneous gingival bleeding
- Excessive salivation
- Metallic taste
- Difficulty in eating
- Difficulty in oral hygiene maintenance
- Halitosis
- General malaise

Some patients report a feeling that the teeth are elongated or slightly loose [22].

Signs

Punched-out interdental papillae

The most characteristic feature is necrosis of interdental papillae, producing a punched-out appearance [21].

Pseudomembrane formation

A grayish-white pseudomembrane covers ulcerated gingival tissues. Removal of the membrane exposes a painful bleeding surface [20].

Gingival bleeding

Spontaneous bleeding occurs frequently and may be the chief complaint [14].

Fetid oral odor

The extensive necrosis and anaerobic bacterial activity produce a characteristic foul odor [12].

Regional lymphadenopathy

Submandibular and cervical lymph nodes may become enlarged and tender [4].

Systemic manifestations

Severe cases may present with:

- Fever
- Malaise
- Fatigue
- Loss of appetite [5]

Advanced disease

Without treatment, lesions may spread to:

- Buccal mucosa
- Lips
- Tongue
- Palate
- Oropharynx

Advanced disease may result in extensive soft tissue destruction and periodontal attachment loss [6].

Histopathological features

Histopathological examination demonstrates four distinct zones [9].

Bacterial zone

The superficial layer consists of a dense collection of bacteria including spirochetes and fusiform organisms [12].

Neutrophil-rich zone

Beneath the bacterial layer lies a region rich in neutrophils mixed with bacterial colonies [1].

Necrotic zone

This zone contains:

- Necrotic tissue debris
- Degenerated collagen fibers
- Numerous microorganisms [26]

Spirochetal infiltration zone

The deepest layer demonstrates invasion of viable connective tissue by spirochetes.

The extensive bacterial penetration explains the rapid progression and aggressive nature of the disease [20].

Diagnosis

Diagnosis is primarily clinical [4].

Diagnostic triad

The accepted diagnostic triad includes:

- Interdental papillary necrosis
- Gingival bleeding
- Pain

These three features are considered essential for diagnosis.

Clinical examination

Clinical evaluation should assess:

- Extent of gingival involvement
- Presence of pseudomembrane
- Oral hygiene status
- Tobacco habits
- Stress levels
- Nutritional status [3]

Laboratory investigations

Routine laboratory testing is not required for uncomplicated cases. However, investigations may be indicated when underlying systemic disease is suspected.

Recommended investigations include:

- Complete blood count
- Peripheral smear
- Blood glucose estimation
- HIV testing
- Nutritional assessment [4].

Radiographic examination

Radiographs are generally normal in early disease.

Bone loss may be evident when progression to necrotizing ulcerative periodontitis has occurred [9].

Differential diagnosis

Several oral diseases may mimic ANUG [2].

Primary herpetic gingivostomatitis

Characterized by diffuse gingival inflammation associated with multiple vesicles and ulcers [3].

Desquamative gingivitis

Usually associated with autoimmune mucocutaneous disorders and lacks punched-out papillae [2].

Agranulocytosis

May present with extensive gingival ulceration but is accompanied by profound hematologic abnormalities [2].

Leukemia

Leukemic gingival lesions may resemble ANUG and require hematological evaluation [7].

Pemphigus vulgaris

Demonstrates widespread mucosal ulceration and positive Nikolsky sign [6].

Mucous membrane pemphigoid

Typically affects older adults and presents with chronic desquamative lesions [15].

Aphthous ulcers

Usually occur as isolated ulcers without gingival necrosis.

Accurate differentiation is essential because management strategies differ considerably among these conditions [20].

Management and Treatment

Successful management of Acute Necrotizing Ulcerative Gingivitis requires immediate control of the acute infection, elimination of predisposing factors, and long-term maintenance of periodontal health. Treatment is generally divided into acute, corrective, and maintenance phases [22].

Acute phase management

The primary objectives during the acute phase are:

- Relief of pain
- Control of infection
- Removal of necrotic tissue
- Prevention of disease progression [22]

Gentle debridement

The initial treatment consists of careful removal of pseudomembranes, necrotic debris, plaque, and calculus. Debridement can be performed using ultrasonic scalers or hand instruments depending on patient comfort [21].

Because the gingival tissues are extremely painful, complete debridement may not always be possible during the first visit. Multiple appointments may be required [21].

Irrigation

Local irrigation helps reduce the microbial load and remove superficial debris.

Commonly used irrigating solutions include:

- Hydrogen peroxide (1.5–3%)
- Warm saline
- Chlorhexidine gluconate

Hydrogen peroxide releases oxygen that suppresses anaerobic microorganisms and assists in cleansing ulcerated surfaces [24].

Oral hygiene instructions

Patients should be instructed regarding:

- Gentle tooth brushing with a soft toothbrush
- Regular plaque control
- Avoidance of traumatic brushing
- Use of antimicrobial mouth rinses

Maintenance of oral hygiene is critical for successful treatment and prevention of recurrence [26].

Antimicrobial therapy

Antibiotics are not required in every patient. Local treatment alone is often sufficient for mild and localized disease [18].

Indications for antibiotic therapy

Systemic antibiotics are recommended when:

- Fever is present
- Regional lymphadenopathy exists
- Lesions extend beyond the gingiva
- Severe pain prevents adequate oral hygiene
- Immunocompromised status is present
- Rapid disease progression occurs [18]

Metronidazole

Metronidazole is considered the drug of choice because of its effectiveness against anaerobic bacteria associated with ANUG.

Typical regimen:

- 250–500 mg three times daily for 5–7 days

Advantages include:

- Excellent anaerobic coverage
- Rapid reduction in pain
- Improved healing [21]

Penicillin

Penicillin remains effective against many ANUG-associated organisms and may be used alone or in combination with metronidazole [22].

Amoxicillin

Amoxicillin is frequently prescribed when broader antimicrobial coverage is desired [24].

Alternative antibiotics

In penicillin-allergic patients:

- Clindamycin
- Azithromycin
- Doxycycline

May be considered depending on clinical circumstances [26].

Correction of predisposing factors

Successful long-term treatment requires identification and elimination of underlying causes [26].

Smoking cessation

Patients should be strongly encouraged to discontinue tobacco use. Smoking cessation improves gingival vascularity, immune response, and periodontal healing [23].

Stress reduction

Psychological stress is an important contributing factor.

Recommended measures include:

- Adequate sleep
- Physical exercise
- Relaxation techniques
- Psychological counseling when required [20]

Nutritional improvement

Patients should receive advice regarding:

- Balanced diet
- Adequate protein intake
- Vitamin supplementation when indicated
- Correction of nutritional deficiencies [18]

Management of systemic disease

Underlying systemic conditions such as diabetes mellitus, leukemia, HIV infection, and neutropenia must be appropriately investigated and managed [11].

Periodontal phase of treatment

Following resolution of acute symptoms, comprehensive periodontal therapy should be performed.

This includes:

Scaling and root planing

Complete removal of plaque and calculus deposits from supra- and subgingival surfaces [17].

Elimination of local irritants

Correction of:

- Overhanging restorations
- Defective crowns
- Plaque-retentive prostheses [16]

Periodontal re-evaluation

Patients should be reassessed after healing to determine:

- Residual pocket depth
- Attachment loss
- Need for further periodontal therapy [23]

Acute Necrotizing Ulcerative Gingivitis in HIV Infection

Necrotizing periodontal diseases are among the most significant oral manifestations of HIV infection [10].

Clinical characteristics

Compared with immunocompetent patients, HIV-positive individuals often demonstrate:

- More extensive tissue destruction
- Severe pain
- Rapid progression
- Frequent recurrence [5]

Necrotizing ulcerative periodontitis

In HIV-infected individuals, ANUG may rapidly progress to necrotizing ulcerative periodontitis with:

- Loss of periodontal attachment
- Alveolar bone destruction
- Tooth mobility
- Tooth loss [4]

Management considerations

Treatment principles remain similar but often require:

- More frequent debridement
- Systemic antibiotics

- Chlorhexidine rinses
- Close monitoring
- Medical consultation [9]

Complications

Although many cases respond favorably to treatment, untreated disease may result in serious complications [21].

Necrotizing ulcerative periodontitis

The infection extends into deeper periodontal structures causing:

- Attachment loss
- Alveolar bone destruction
- Tooth mobility [21].

Necrotizing stomatitis

The necrotizing process spreads beyond the gingiva to involve oral mucosal tissues [17].

Osteomyelitis

Severe infections may involve underlying bone leading to osteomyelitis [6].

Tooth loss

Advanced periodontal destruction can result in loss of affected teeth [6].

Noma (Cancrum Oris)

Noma represents the most devastating complication of ANUG [4].

Definition

Noma is a rapidly progressive gangrenous infection causing massive destruction of oral and facial tissues [3].

Predisposing factors

- Severe malnutrition
- Poverty
- Poor oral hygiene
- Immunodeficiency
- Childhood infections [5]

Clinical features

Patients develop:

- Extensive tissue necrosis
- Facial perforation
- Osteomyelitis
- Severe facial disfigurement [4]

Prognosis

Without treatment, mortality rates are extremely high. Survivors often require complex reconstructive surgery [9].

Prognosis

The prognosis of ANUG is generally excellent when diagnosed early and treated appropriately.

Most patients experience:

- Significant pain reduction within 24–48 hours
- Resolution of acute inflammation within one week
- Complete healing within several weeks

However, recurrence is common if etiologic factors remain uncorrected.

Factors associated with poor prognosis include:

- Continued smoking
- Persistent poor oral hygiene
- Immunosuppression
- Malnutrition
- Failure to seek treatment [22]

Prevention

Prevention focuses on reducing risk factors and maintaining periodontal health [26].

Oral hygiene measures

- Twice-daily tooth brushing
- Interdental cleaning
- Regular professional dental care [20].

Lifestyle modifications

- Smoking cessation
- Balanced nutrition
- Adequate hydration
- Stress management [5]

Regular dental visits

Periodic dental examinations allow early detection and management of gingival inflammation before progression to necrotizing disease [4].

Management of systemic conditions

Control of systemic illnesses and immunodeficiency states significantly reduces the risk of ANUG [1].

Conclusion

Acute Necrotizing Ulcerative Gingivitis is a distinctive necrotizing periodontal disease characterized by rapid gingival destruction, pain, spontaneous bleeding, and fetid oral odor. The condition results from a complex interaction between anaerobic microorganisms and host susceptibility factors such as poor oral hygiene, smoking, psychological stress, malnutrition, and immunosuppression. Although the disease can be alarming in appearance, prompt diagnosis and appropriate treatment usually result in rapid clinical improvement and favorable outcomes. Early debridement, meticulous plaque control, elimination of predisposing factors, and selective use of systemic antibiotics form the cornerstone of management. Recognition of high-risk individuals, particularly immunocompromised patients, is essential to prevent progression to more destructive forms of necrotizing periodontal disease and severe complications such as noma. Continued emphasis on preventive dentistry, oral hygiene education, and risk factor modification remains fundamental in reducing the burden of this potentially debilitating condition

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