

Necrotizing Gingivitis and Periodontics an Outlook

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Introduction

Acute necrotizing ulcerative gingivitis (ANUG) is characterized by painful ulceration of the gum between the teeth, a pronounced tendency to gingival bleeding and halitosis [1]. ANUG is caused by an imbalance in the normal flora of the gingival sulcus [2]. ANUG is a rare infectious disease of the gum tissue, affecting <1% of the population [3]. Although its prevalence of this disease is not high, its clinical importance is clear as it represents the most severe of conditions associated with the dental biofilm. If left untreated, ANUG can lead to very rapid tissue destruction, necrotizing ulcerative periodontitis (NUP), and even cancrum oris (noma), which is commonly fatal [4]. Although the acronym ANUG is frequently used, it is a misnomer. NUG often undergoes a diminution in severity without treatment, thereby, leading to a subacute stage with milder clinical symptoms [5].

Definition

Necrotizing ulcerative gingivitis (NUG) is a microbial disease of the gingiva in the context of an impaired host response. It is characterized by necrosis and sloughing of gingival tissue and it presents with characteristic signs and symptoms [5].

Synonyms: [6]

1. Vincent's disease
2. Fusospirochaetal gingivitis
3. Trench mouth
4. Acute ulcerative gingivitis

Historical background

It was recognised as far back as the 4th century B.C by Xenophon, who mentioned that greek soldiers were affected with "sore mouth" and foul smelling breath. John Hunter, in 1778, described the clinical findings and differentiated it from scurvy and chronic destructive periodontal disease [5]. The informal name trench mouth arose during world war I as many soldiers developed the disease, probably because of the poor conditions and extreme psychological stress [6].

Etiology

ANUG is mainly due to bacterial infections and mainly associated

with fusiform and spirochaete bacteria. One study identified spirochetes and majority of gram-negative bacteria, including bacteriodes intermedium and Fusobacterium species as the most important causes [7,8].

These diseases are infectious condition and most critically predisposing factor is immunosuppression individuals most notably found in HIV and other conditions like leukaemia, neutropenia, diabetes mellitus and long term immunosuppressant therapy also been noted [9-13].

Epidemiology

Generally, ANUG has been documented by historians since the 4th century BC ANUG is more common in the young, particularly severely malnourished children and young adults with human immunodeficiency virus infection [3,14].

It is commonly found in improving or developing countries with low social and economic status. The disease has occurred in epidemic like pattern, but it is not contagious [15].

Pathophysiology

Psychological stress, poor diet, insufficient sleep, alcoholic, tobacco, poor oral hygiene, pre-existing gingivitis and HIV infection and these factors shows impairment to host [16].

These bacteriodes in addition to other primarily gram negative species, produce a vast array of metabolites (collagenase, endotoxins, hydrogen sulphide and fibrinolysin responsible for the destruction [13].

Histopathology

The purpose of this study was to provide additional evidence that in the lesions of acute necrotizing ulcerative gingivitis, spirochaetes and other microorganisms are capable of invading the non-necrotic lamina propria of affected gingiva.

The result indicated that spirochaetes and other bacteria are capable of penetrating the viable connective tissue of lamina propria [17].

A few case series have demonstrated the usefulness of Gram staining in supporting the diagnosis, which would otherwise rest solely on clinical grounds [8]. The clinical appearance of ANUG reflects its histopathology [16].

An electron microscopic study of ANUG was first undertaken by Listgarten, who identified 4 zones containing spirochaetes from tissue section of ANUG lesion:

- Bacterial zone
- Neutrophil rich zone
- Necrotic zone
- Zone of spirochaetal infiltration upto 250 micrometer beneath the ulcerated surface.

Intermediate sized spirochaetes were predominant in ANUG lesions [18].

Light microscopy shows ulceration of the stratified squamous epithelium with the fibrinous pseudo membrane. Epithelium and connective tissue adjacent to the area of ulceration is heavily infiltrated by polymorphonuclear leukocytes (PMNs) [19].

Classification [5]

Stages in progression of necrotizing ulcerative gingivitis (NUG) are described by Pindborg and co-workers.

The lesion starts as

- Erosion of the tip of the interdental papilla
- The lesion involving all the papilla and also involving the marginal gingiva
- The attached gingiva also gets involved
- Exposure of the bone with complete loss of interdental papilla, marginal gingiva and attached gingiva.

Horning and Cohen extended the staging as follows:

- Stage 1: necrosis of the tip of the interdental papilla (93%)
- Stage 2: necrosis of the entire papilla (19%)
- Stage 3: necrosis extending to marginal gingiva (21%)
- Stage 4: necrosis of the attached gingiva (1%)
- Stage 5: necrosis involving the buccal and labial mucosa (6%)
- Stage 6: necrosis exposing alveolar bone (1%)
- Stage 7: necrosis perforating skin of the cheek (0%)

According to Horning and Cohen:

- Stage 1 is NUG
- Stage 2 may be either NUG or NUP because attachment loss may have occurred.
- Stage 3 and 4 correspond to NUP
- Stage 5 and 6 correspond to necrotizing stomatitis
- Stage 7 would be noma.

Clinical Features

- Punched out and crater like depression [5].
- Severe gum pain [20]
- Profuse gum bleeding that requires little or no provocation [15].
- Interdental papilla are ulcerated with dead tissue [20]
- The papillary necrosis of ANUG has been described as punched out [15]
- Foul breath [15]
- Bad taste (metallic taste) [20]
- Malaise, fever and/or cervical lymph nodes enlargement are rare [20]
- Sudden onset [21]
- Excessive amount of pasty saliva [5]

Complications

Acute necrotizing ulcerative gingivitis may lead to devastating tissue damage in the form of necrotizing periodontitis, necrotizing stomatitis, and cancrum oris (noma), which is frequently fatal. Its identification and treatment is a challenge, but a necessity, for adult and paediatric practitioners alike [22].

Diagnosis

Diagnosis is usually clinical. Smear for Fusospirochaetal bacteria and leukocytes, blood picture occasionally [20].

IgG and IgM antibody titres to 8 bacterial isolates were measured by indirect immunofluorescence and ELISA in sera from ANUG patients during the acute phase, from ANUG patient during the convalescent phase, from patients with gingivitis and from subjects with normal gingiva. Compared to gingivitis and healthy groups, the ANUG groups exhibit significantly higher IgG and IgM titres to intermediate sized spirochaetes [23].

The diagnosis of ANUG must be made fundamentally according to the presence or absence of primary clinical symptoms; the interproximal gingival necrosis often described by “punched out” gingival bleeding with little or no provocation and intensive pain which is a hallmark of the gingival lesion [24].

Differential Diagnosis [25]

- Primary herpetic gingivostomatitis
- Desquamative gingivitis
- Agranulocytosis
- Cyclic neutropenia
- Leukemia
- Ascorbic acid deficiency and gingivitis

Treatment

Treatment of necrotizing ulcerative gingivitis consists of:

1. Alleviation of the acute inflammation plus treatment of chronic disease either underlying the acute involvement or elsewhere in the oral cavity.
2. Alleviation of generalized toxic symptoms such as fever or malaise.
3. Correction of systemic conditions that contribute to the initiation or progress of the gingival changes.

Treatment should follow an orderly sequence and is divided into treatment for

1. Non-ambulatory patient: With symptoms of generalized systemic complications.
2. Ambulatory patient: With no serious systemic complications.

Treatment for Non-Ambulatory Patients

Day 1:

- Local treatment limited to gently removing the necrotic pseudo membrane with a pellet of cotton saturated with hydrogen peroxide (H₂O₂)
- Advised bed rest and rinse the mouth every 2 hours with a diluted 3 percent hydrogen peroxide.
- Systemic antibiotics like penicillin or metronidazole can be prescribed.

Day 2:

If condition is improved, proceed to the treatment described for ambulatory patients. If there is no improvement at the end of the 24 hours, a bedside visit should be made. The treatment again includes gently swab the area with hydrogen peroxide, instructions

of the previous day are repeated.

Day 3:

Most cases, the condition will be improved, start the treatment for ambulatory patients.

Treatment for Ambulatory Patients

First visit:

- ✓ Clinician should obtain a general impression of the patient's background, including information regarding recent illness, living conditions, dietary background, type of employment, hours of rest and mental stress.
- ✓ Patient's general appearance should be observed, apparent nutritional status and responsiveness or lassitude, and temperature noted.
- ✓ Sub Maxillary and Sub Mental lymph nodes palpated.
- ✓ Oral cavity examined for characteristic lesion of NUG, its distribution and possible involvement of the oropharyngeal region.
- ✓ Oral hygiene evaluated, presence of pericoronal flaps, periodontal pockets and local irritants determined.
- ✓ Patient questioned regarding history of the acute disease and its onset and duration, recurrence; previous treatment - how long, type of treatment etc.
- ✓ Initial treatment confined to acutely involved areas, which are isolated with cotton rolls and dried.
- ✓ Topical anesthesia applied and area gently swabbed with a cotton pellet to remove the pseudo membrane and non-attached surface debris.
- ✓ Area cleansed with warm water, superficial calculus is removed. (Ultrasonic)
- ✓ Subgingival scaling and curettage contraindicated to prevent extension of infection to deeper tissues and also bacteremia.
- ✓ Unless an emergency exists, extractions or periodontal surgery are postponed until the patient has been symptom free for a period of 4 weeks to minimize likelihood of exacerbation.
- ✓ Patients with moderate or severe NUG and local lymphadenopathy or other systemic symptoms are placed on an antibiotic regimen of Penicillin (500mg 6th hourly); Metronidazole 500mg b.i.d 7 days is also effective.
- ✓ Antibiotics continued until systemic complications subside

Instructions after First Visit:

- Avoid tobacco, alcohol and condiments.
- Rinse with a glassful of an equal mixture of 3% H₂O₂ and warm water every 2 hours and/or twice daily with 0.12% chlorhexidine solution.
- Avoid excessive physical exertion or prolonged exposure to the sun.
- Confine tooth brushing to the removal of surface debris with a bland dentifrice; over zealous brushing and the use of dental floss or interdental cleaners will be painful.

Second visit:

- ✓ 1 to 2 days later, the patient's condition is usually improved; pain diminished or no longer present.
- ✓ Gingival margins of the involved areas are erythematous, but without a superficial pseudo membrane.
- ✓ Scaling performed if sensitivity permits.
- ✓ Instructions as same as above.

Third visit:

- ✓ 1 to 2 days after the second visit, patient should be essentially symptom free.
- ✓ Scaling and root planning repeated.
- ✓ Instructed in plaque control procedures.

- ✓ H₂O₂ rinses discontinued; chlorhexidine rinses maintained for 2-3 weeks.

Fourth visit:

- ✓ Oral hygiene instructions are reinforced and thorough scaling and root planning are performed.

Fifth visit:

- ✓ Appointments are fixed for treatment of chronic gingivitis, periodontal pockets and pericoronal flaps, and for the elimination of all local irritants. Patient is placed on maintenance programme.

Gingival Changes with Healing

- ✓ Removal of surface pseudo membrane exposes the underlying red, hemorrhagic, craterlike depressions in the gingival.
- ✓ In the next stage, the bulk and redness of the crater margins are reduced, but the surface remains shiny.
- ✓ Early signs of restoration of normal gingival contour and color follow this.
- ✓ Final stage: normal gingival color, consistency, surface texture and contour are restored. Portions of the root exposed by the acute disease are covered by healthy gingiva.

Prognosis

Adequate treatment usually prevents the progression of the disease, and ulcer healing can be expected in a few days utilizing this treatment approach. Nevertheless, lack of treatment can lead to deterioration in the form of potentially fatal conditions such as necrotizing ulcerative periodontitis (NUP) and even cancrum oris (noma) [3].

Patient Education

Healthy gums are extremely important to a patient's overall health. Maintaining good oral hygiene is the most effective way to avoid developing destructive gum disease such as gingivitis. There are many steps to take to keep gums healthy, including twice-daily brushing, daily flossing or interdental cleaning, and visiting a dentist regularly. Health conditions like HIV infection, diabetes, and cancer can reduce a person's ability to fight infection, which can increase the risk of developing gum disease. Other factors that can affect the gums are medications, such as anti-seizure medications and some blood pressure medications, hormonal changes, and the use of tobacco. Thus, it is very important for pregnant patients and patients meeting any of these criteria to see a dentist regularly. Keeping gums healthy and preventing disease is possible with daily care and regular visits to a dentist [26].

Conclusion

ANUG is a specific acute periodontal disease. The diagnosis seems evident according to the three typical clinical features as papilla necrosis, bleeding, and pain on the one hand and the identification of risk factors that alter the host response on the other hand. Treatment should be organized on successive steps, and the acute phase treatment should be provided immediately to prevent sequelae and craters in soft tissues that will lead to new relapses. Finally, a good compliance with the oral hygiene practices and maintenance do guarantee better and stable outcomes [27].

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