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Acute Necrotizing Ulcerative Gingivitis

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I. INTRODUCTION

The American Academy of Periodontology categorized(1,10),"Necrotizing Periodontal Diseases(19)in 1999 "Necrotizing ulcerative gingivitis'' and "Necrotizing ulcerative periodontitis''(3,7).Although known since ancient times by a multitude of names. ANUG is first described by Plaut in 1894 and Vincent in 1896(2).For centuries, military historians have documented a condition of painful, bleeding gingival proposed tissues with accompanying necrosis and fetor oris(1,10).In 2002,Holmstrup and Westergaurd another that include three classification distinct illness under Necrotizing Periodontal disease: Necrotizing gingivitis, Necrotizing Periodontitis, Necrotizing Stomatitis(10).

Necrotizing ulcerative gingivitis 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(4).In past it was also referred to as Vincent infection, Acute ulceromembraneous gingivitis ,Trench mouth(8),Ulcerative gingivitis, Vincent stomatitis, Plaut –Vincent stomatitis, Fedid stomatitis, Stomatitis ulcerosa, Fusospirillary gingivitis and Putrid stomatitis(4,16).Infact, current theories of causative mechanism differ little from those proposed by Vincent : that of an endogenous opportunistic fusospirochetal infection(2,8).The primary cause of necrotizing periodontal disease is infection by microbes including *treponema. spp, selemonas .spp, fusobacterium.spp, prevotella intermedia(13).*Secondary factors are human immunodeficiency virus infection, malnutrition, lack of sleep, stress and smoking(3,7).It occurs most typically among 21 to 24 years old in both developed and developing countries(3,7).

II. HISTORICAL BACKGROUND

Acute necrotizing ulcerative gingivitis was recognized during 4th century BC, Xenophon(20).He 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. NUG occurred in epidemic form in the French army in 19th century, and in 1886 Hersch discussed some features associated with the disease, such as lymph node enlargement, fever, malaise and increase salivation(4,13).In the 1890s , Plaut and Vincent described the disease and attributed its origin to fusiform bacilli and spirochetes(4,9).

III. EPIDEMIOLOGY

The incident of ANUG varies widely depending on the population studied and the clinical criteria used (1).

The prevalence of NUG appears to have rather low in United States and Europe before 1914(4). A 1951 study by Pendborg of the Danish armed forces noted an incident of ANUG of 69% in newly conscripted sailors. In 1963, Giddon et al studied ANUG in college students and reported an incidence of 0.9%. In 1973 Barnes et al studied the incidence of ANUG in a U.S military population and found an incidence of 0.19%.Barnes and coworkers also found 94.5% of individuals with ANUG were white , 81.7% were less than 21 years of age and 96.3% were male. Horning et al in 1990 studied another military population and found an incidence of ANUG of 0.5%(1).

Stevens et al studied the incidence of ANUG in a general clinical population. They found 98% of ANUG patients were white and 2% were black. The average age of patients was 23.9 years.94% patients smoked(1).

A study at a dental clinic in Peague, Czech Republic reported the incidence of NUG as 0.08% among patient between ages of 15 and 19 years old, 0.05% among those 20 and 25 years old and 0.02% among those 25-29 years old(4).

NUG occurs in individual of all ages with incidence higher between ages of 15-30 years. In India , 54-58% of patient in two studies with less than 10 years old .In random school population in Nigeria,11.3% of children between 2 and 6 years old .In Nigerian hospital population it was present in 23% of children who were less than 10 year old (4).

Mel nick et al estimated the prevalence of ANUG to be 0.6% in general population .Their studies of ANUG indicated that mean age to be 23 years with mean percent of 50% males to 50% of females and predominance of whites(4), NUG occurs with low socio-economic growing (16),common among children with Down syndrome of children (16)with mental deficiencies of more than 90% of ANUG patients are smokers(1).

IV. ETIOPATHOGENESIS

The precise etiology of ANUG is not known, however, it is believed to be a polymicrobial infection with the implicated organisms being normal commensals of the oral cavity(5).

a. ROLE OF BACTERIA

Delay in 1927, Plaut in 1084 and Vincent in 1896 postulated that NUG was caused by specific bacteria: Fusiform bacilli and a Spirochetal organism (4, 8, 15, 16).

Hampp and Mergenhagen found small treponemas, B. vincentii and B .bucalis can cause abscess formation in rabbits and guinea pigs (1, 5).

Rosebury et al described a fusospirochetal complex(20) that consists of T. microdentium, intermediate spirochetal vibrios, fusiform bacilli and filamentous organisms(4).Leeshe et al in 1982 found prevotella intermedia(10) in constant flora and consistently identified fusobacterium spp and selemonas spp (10) in ANUG plaque samples (1, 5). He also identified treponema spp in ANUG lesions(1, 5).

Chung et al (1983) found higher IgG and IgM antibody titre to intermediate sized spirochetes and higher IgG titre to B. melaninogencius spp(1,5) and also higher antibodies titers to P. Intermedia in ANUG. Rowland et al (1993) found lower IgG and IgM serum levels to B. intermedia and elevated IgM titers to 3 treponema spp. Cutler et al (1994) found depressed phagocytosis and killing of P. intermedia and also found fusobacterium in subgingival plaque. Loesche et al cultured and microscopically examined the plaque samples and found treponema spp, fusobacterium spp and bacterioids intermedius(16).Listgarten and Lewis identified zone of spirochetal invasion in ANUG lesions, noted that large and intermediate sized spirochetes (15) predominated. Courtois et al (1983) found spirochetal invasion in ANUG (1).Synergistees bacteria are also seen in NUG in association with spirochates(17).Both spirochetes and other bacterias are capable of infiltrating viable connective tissue(14).

Small oral spirochetes with strict depence on glucuronic acid or galacturonic acid were isolated from patients with periodontitis and ANUG (18).Actively proliferatin cultures of beta-glucuronidase active strains are dominated by thin and very short cells (18).Immunohistochemical analysis of the bacterial population has indicated an increased prevalence of T.denticola and pathogen related spirochetes(18).

Riviere et al (1991) found pathogen related oral spirochetes in epithelium and connective tissue adjacent to ANUG lesion (1).

b. ROLE OF HOST RESPONSE

The role of an impaired host response in NUG has long been recognized. Even in the early description of the disease, NUG was associated with physical and emotional stress(11) as well as decreased resistance to infection (4).

Cogen et al reported a study in which the total leukocyte count for both control and ANUG patients were found to be the same, but the patients showed marked depression of polymorphonuclear leukocytes responsiveness in both chemotaxis(10) and phagocytosis(5,4,13).He found a predominance of plasma cells and lymphocytes in the same lesions rather than PMN(5). Alteration in function of leukocytes and lymphocytes(13).Listgaren's study showed predominance of PMNs in ANUG lesions. The Rowland et al study on serum IgG and IgM level in ANUG patients also suggested impaired immune functions in these patients(5).Histologic features show lymphoid cell infiltrate in most cases(14).Protien quantification show increased inflammatory markers(9).High incidence in HIV positive patients(5, 10, 13).Furthermore, ANUG is a prominent feature of AIDS patients(13) with ANUG being 20.8 times more likely to have CD4 count less than 200 cells/mm3(5).

c. LOCAL PREDISPOSING FACTORS

Predisposing factors include; poor oral hygiene(16, 20), emotional stress(20), physical stress(11, 16), smoking(16, 20) and increased resistance to infection, preexisting gingivitis, injury to gingiva (4, 1, 10, 13, 14, 16, 19, 20).Deep periodontal pocket and pericoronal flaps, are particularly vulnerable areas, they offer favorable environment for proliferation of anaerobe fusiform bacilli and spirochetes(4).

Stress(10) causes the release of epinephrine(20)and substance P; a peptide hormone which suppresses both specific and nonspecific immunity(5) .It is common among military cadets, in harsh physical condition (5, 13, 20).

Melnick et al noted that more than 90% of ANUG patient were smokers' .Smoking(10)reduce the levels of circulating antibody and depress chemotactic and phagocytic activities. Kardachi and Clarke noted that nicotine from cigarette smoke activated the release of epinephrine in or near vessel walls. Nicotine and epinephrine together cause severe pressor effect than epinephrine alone (1).Pindborg reported that 98% of his patient with NUG were smokers and the frequency of this disease increases (4) with increase in exposure to tobacco smoke (10, 4)

Schluger who described ANUG as a disease of "filth" believes that allow standard of oral hygiene is the most single factor contributing to ANUG. However he does not state the presence of disease is always a result of lack of oral hygiene (10)on the path of patient but the plaque and debris accumulation occur due to discomfort with oral hygiene practices (1;5).

d. SYTEMIC PREDISPOSING FACTORS

NUG is not found in a well – nourished individual with a fully functional immune system. Immune deficiency may be related to varying levels of nutritional deficiency(10, 13), fatigue caused by chronic sleep deficiency(10), other health habits [alcohol/drug abuse] and systemic disease [eg: diabetes, debilitating infection] (4).

*NUTRITIONAL DEFICIENCY

Malnutriton(13, 16, 19) impairs innate and adaptive defense mechanism in host with associated dysfunction of the cytokine system(5).Nutrional deficiencies such as vitamin B and vitamin C accentuate the response of gingival tissue produced by increased pathogenic flora (4,6). Several researchers have found an increase in the fusospirochetal flora in patient with nutritionally- deficient diets(6).

*DEBILITATING DISEASE(4)

Systemic disturbances (4)include chronic diseases such as syphilis, cancer, metallic intoxication, severe gastrointestinal disorder, blood dyscrasias such as leukemia, anemia and acquired immunodeficiency syndrome.

e. PSYCHOSOMATIC FACTORS

A significant correlation between disease incidence and two personality traits – dominance and abasement – suggest the presence of a NUG –prone personality(4). Cohen and coworkers have suggested that s psychiatric disturbance may lead to activation of the hypothalamic pituitary adrenal axis. This result in elevation of serum and urine cortisol depression of lymphocyte and polymorphonuclear leukocytes function that predispose to ANUG(6).

V. CLINICAL FEATURES

NUG is usually identified as an “acute” disease. NUG often undergoes a diminution in severity without treatment; thereby lead to “subacute stage”(4).NUG can cause tissue destruction that involves the periodontal attachment apparatus, especially in patients with long standing disease or severe immunosuppression. When bone loss occurs, the condition is called necrotizing ulcerative periodontitis [NUP](4).

a. ORAL SIGNS

Lesions are characterized by punched out(13, 12), crater- like depression at the crest of interdental papillae, subsequently involving marginal gingiva and rarely attached gingiva(10, 4, 6).

These crates are covered by grayish pseudomembranous slough, which is demarcated from the remaining of mucosa by a pronounced linear erythema(4, 6).

Gingival hemorrhage and pronounced bleeding(4, 6, 9) on slightest stimulation. Other signs are pain, interdental ulceration, bad taste, fetid odor(16), and increased salivation(4, 6, 16).The maxilla and mandible anterior facial gingivae were most often affected(14, 20).

b. ORAL SYMPTOMS

Lesions are extremely sensitive to touch. Metallic foul taste and the patient is conscious of an excessive amount of “pasty” saliva(4, 6)

c. EXTRAORAL AND SYSTEMIC SIGNS AND SYMPTOMS

Local lymphadenopathy and slight elevation in temperature are common in mild to moderate stages of disease. In severe cases, high fever, increased pulse rate, leukocytosis, loss of appetite and general lassitude are common(4, 6, 10).

d. CLINICAL COURSE

It can vary. Stages in progression of NUG are described by Pindborg and coworkers, the lesion starts as:

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

Horning and Cohen extend the staging as following:

Stage 1: Necrosis of the tip of the interdental papilla (93%)

Stage 2: Necrosis of the entire papilla [19%]

State 3: Necrosis extending to the marginal gingiva [21%]

State 4: Necrosis of the attached gingiva [1%]

State 5: Necrosis involving the buccal and labial mucosa [6%]

Stage 6: Necrosis exposing alveolar bone [1%]

Stage 7: Necrosis performing skin of the cheek [0%]

e. HISTOPATHOLOGY

Microscopically, the NUG lesion is a nonspecific acute necrotizing inflammation of the gingival margin that involves both the stratified squamous epithelium and the underlying connective tissue(4).

VI. RELATION OF BACTERIA TO NECROTIZING ULCERATIVE GINGIVITIS LESION(4, 6, 10, 13, 15)

Zone I: Bacterial zone: it is the most superficial zone, consist of varied bacteria, including a few spirochetes of small, medium- sized and large types.

Zone II: Neutrophil rich zone: Contains numerous leukocytes predominantly neutrophil with bacteria including spirochetes of various types.

Zone III: Necrotic zone: Consist of a dead tissue cell, remnants of connective tissue fragments and numerous spirochetes.

Zone IV: Zone of spirochetal infiltration: Consist of a well preserved tissue infiltration with spirochetes of intermediate and large – sized without other organism.

VII. DIAGNOSIS

Diagnosis is based on clinical finding of gingival pain, ulceration and bleeding. Bacterial studies are useful for the differential diagnosis of NUG and specific infections of the oral cavity(4).

Some bacterial species like *Porphyromonas gingivalis*, *Actinomyces gerencseriae*, *Prevotella intermedia, Prevotella nigrescens* have been detected using "fluorescence" insitu hybridization [FISH] or immune fluorescence[IF]. Image acquisition and analysis of stained bacteria and statistical analysis between quantitative protein assay and FISH staining(9).

-DIFFERENTIAL DIAGNOSIS(6)

a. Gonococcal stomatitis

b. Agranulocytosis

c. Vincent's angina

d. Desquamative gingivitis

e. Acute necroting ulcerative gingivitis in leukemia

f. Acute necrotizing ulcerative gingivitis in AIDS

g. Streptococcal gingivostomatitis

VIII. TREATMENT

During 1st three decades of 20th century vaccines were used systemically or locally in periodontal tissues. These includes pure culture of streptococci and other oral organism, autogenously vaccines and stock vaccine such as van cott's vaccine, Goldenberg's vaccines or Inava endocorps vaccine(8).

Treatment can be given by the non-ambulatory and ambulatory condition of patient(6).Other techniques include the use of UV light, electrochemical techniques(8).Control of systemic factors such as smoking, adequate sleep, reduction of stress(12).

Instruction in proper oral hygiene measures(10, 19) including frequent oral rinses with saline or dilute hydrogen peroxide, should be stressed(1, 6).Scaling and root planning(13), and has an obvious advantage in the esthetic result(2, 7). Hand scaling(13)of teeth have been augmented with ultrasonic instrumentation. Repeated curettage are established for regeneration of papilla(1, 2, 6, 7).

For the treatment include gingival surgery, gingivoplasty(1, 2, 6, 10).Chemotherapeutic agents used are chlorhexidine gluconate(2,7), analgesics(6), pencillins and metronidazole(11, 1, 7, 10, 19).Nutrional supplements such as vitamin B, vitamin C are also given(6).Newer method include "local oxygen therapy"(10).

IX. COMMUNICABILITY

The occurrence of NUG in epidemic like out breaks does not necessarily mean that it is contagious. The affected groups may be afflicted by the disease as a result of common predisposing factors rather than because of its spread from person to person. In all likelihood, both a predispose immunocompromised host and the presence of appropriate bacteria are necessary for the production of this disease(4).

X. SUMMARY

Specific acute periodontal disease(10). Affects primarily young adult(11, 16, 17)whites.Sex distribution is equal. Poor oral hygiene increases the bacterial population, especially large and intermediate spirochetes. Mental and physical stress increases susceptibility to infection and increases the production of corticosteroids, depressed chemotaxis and altered IgG and IgM serum levels to the flora of ANUG. Smoking reduces blood flow to gingival tissues, reduces circulating antibody(1).

Chemotherapeutics such as metronidazole, penicillin are effective. Eradicating malnutrition, improving oral hygiene status(10)are ways of preventing this disease(5).Use of minimally invasive tools, such as digital dentistry can help to avoid microbial infections(9).

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