NECROTIZING PERIODONTAL DISEASE - PREDISPOSING FACTORS. A REVIEW

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ABSTRACT

Necrotizing gingivitis, necrotizing periodontitis and necrotizing stomatitis are classified together under the common name necrotizing periodontal disease (NPD). The etiology is associated with bacterial infection (fusobacteria and spirochetes), but NPD is also predisposed by various factors: local (poor oral hygiene, pre-existing plaque-induced gingivitis, local trauma, smoking, alcohol abuse) and systemic factors (emotional stress, fatigue and insomnia, social economic status and altered host response, malnutrition or systemic diseases).

Keywords: *necrotizing periodontal disease, predisposing factors, etiology*

INTRODUCTION

Necrotizing gingivitis, necrotizing periodontitis and necrotizing stomatitis are presented with the common name necrotizing periodontal disease (NPD). They usually have an acute course of progression.

Necrotizing gingivitis (NG) affects only the gingival tissues and is defined as a "rapidly destructive non-contagious gingival infection" with complex etiology. Necrotic inflammation in necrotizing periodontitis (NP) affects gingival tissues and also periodontal ligament and alveolar bone, and is associated with loss of clinical attachment level and alveolar bone loss at the affected sites. NP is always preceded by NG that progresses in NP. Necrotizing stomatitis (NS) is a severe life-threatening orofacial gangrenous disease, also called noma.

The etiology of NPD is associated with bacterial infection, but the disease is predisposed by various factors as well – malnutrition, smoking, etc. Therefore, the fusiform-spirochetal infection along with a reduced host immune response seems to play a major role in the pathogenesis of NPD. That is why it is useful to be informed about the possible predisposing factors and epidemiology of these periodontal diseases in order to identify all predisposing factors and to propose proper measures for prevention in every particular case.

AIM

The aim of this review article is to summarize and analyze the most important predisposing factors that are associated with the occurrence of NPD.

MATERIALS AND METHODS

The article is based on various researches including retrospective, randomized trials and case reports articles in PubMed and Google Scholar databases. The survey concerns the etiology of NPD (bacterial infection and predisposing factors) and

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analyzes previous systematic reviews of the problem. All the articles that are included are in English only.

LITERATURE SURVEY

The etiological factors discussed in the occurrence of NPD are the symbiosis between fusobacteria and spirochetes and the impact of predisposing factors that decrease the host-defense response against endogenous bacterial infection (1).

The disease onset is due to invasion of spirochetes, fusiforms and other bacilli in individuals with diminished host resistance. The dominant bacteria (constant flora) that are found in cultures of bacterial plaque taken from individuals with necrotic lesions of NPD are *Treponema vincenti*, *Fusobacterium nucleatum*, *B. fusiformis*, *Campylobacter*, *Peptostreptococcus* spp., *Selenomonas* spp., *P. gingivalis* and *Pr. intermedia* (as the amount of fusobacteria in NG saliva is much higher than in healthy mouth saliva) (2). The variable flora consists of diverse bacterial types. Studies show that 78% of all cultured microorganisms in these necrotic lesions are anaerobic Gram (-) bacteria. Spirochetes and fusiform bacteria invade the epithelium, and spirochetes also invade the underlying connective tissue. These species and probably other bacteria have a pivotal role in the pathogenesis of NPD (3). This bacterial etiology has been first described by Plaut in 1894, and later by Vincent in 1896 in the so-called Vincent's angina in which necrotic areas are localized in the tonsils (4).

Consequently, NPD is a bacterial infection that manifests clinically after a decrease in the host immune response to local and general predisposing factors. Usually, more than one predisposing factor is needed to initiate the disease (5). Possible predisposing factors that play a role in the pathogenesis of necrotic periodontal diseases are poor oral hygiene, pre-existing plaque-induced gingivitis, local trauma, emotional stress, fatigue and insomnia, smoking and alcohol abuse, social economic status (6), and altered host response to malnutrition or systemic diseases (HIV infection, leukemia, measles, tuberculosis) (7) .

Local predisposing factors are: poor oral hygiene (8), pre-existing plaque-induced gingivitis (9), plaque retentive factors (restorations, orthodontic anomalies, root proximity, presence of dental calculus), local trauma, smoking and chewing tobacco (10).

Systemic predisposing factors are: emotional and physiological stress (11), malnutrition (12), fatigue and insomnia (1), hormonal imbalance, impaired function of polymorphonuclear leukocytes (PMNs) (13), systemic diseases affecting the immune response (immunosuppression) – diabetes mellitus, leukemia, anemia, AIDS, syphilis, ulcerative colitis, viral infections. Some seasonality has been reported (mainly in the winter season and rainy periods) (14), age (mainly in young individuals – 15-30 years of age) (15), alcohol and drug abuse (1), Caucasian origin (16,17).

1. Poor Oral Hygiene

In cases of poor oral hygiene, streptococci multiply rapidly, consuming oxygen and creating anaerobic conditions for the development of invasive fusiform bacteria and spirochetes. In relation to that, the inadequate personal oral hygiene procedures lead to an increase in the incidence of NPD (8).

2. Pre-Existing Plaque-Induced Gingivitis

Bacterial plaque accumulated due to poor oral hygiene can lead to induction of gingival inflammation, so the pre-existing plaque-induced gingivitis can also be a predisposing risk factor in the pathogenesis of NG and NP (18).

3. Smoking

Smoking is another local factor that predisposes to the occurrence of NPD. Smokers (≥ 20 cigarettes per day) have a decreased count of T-helper lymphocytes and impaired functions of PMNs (chemotaxis and phagocytosis), as well (10). Adrenaline secretion during smoking leads to vasoconstriction of blood vessels in gingival tissues (19). The results of various researchers present the fact that most of the patients with NPD are heavy smokers. According to one study by the American Academy of Periodontology, 98% of patients with necrotic gingivitis are smokers (19).

4. Emotional and Physiological Stress

Emotional stress has an important role in the etiology of NPD and refers to systemic predisposing factors. NPD is more prevalent in subjects with emotional and mental stress (military personnel, patients with depression and other emotional disorders, students during an exam period) due to impaired immune response (13). Stress reduces tissue resistance,

increasing levels of corticosteroids and catecholamines (20). As a result, the microcirculation in gingiva and saliva is reduced, as well as the functions of neutrophils and lymphocytes, which facilitates bacterial invasion and destructive processes (1). During a period of stress, adrenal activity and urinary cortisol levels increase (11).

5. Malnutrition

Malnutrition results in decreased resistance to infection and can be a key systemic factor in the development of NPD (e.g. vitamin C deficiency, vitamin B2 deficiency, protein deficiency) (12,21). Noma is usually described in severely malnourished children from low-socioeconomic groups (especially in developing countries) (6). It develops after periodontal infection as a result of reduced defense against infections (e.g. smallpox) (22,23). Elevated levels of cortisol and pro-inflammatory cytokines have been found in malnourished individuals (IL-6, IL-8, IL-10, IL-18, and IL-1β). In addition, malnutrition includes impaired T-helper / T-suppressor ratio, histaminemia, hormonal imbalance with increased levels of free cortisol in the blood and saliva (24).

6. Systemic Diseases Affecting the Immune Response (Immunosuppression)

Necrotizing periodontal diseases are common in immunocompromised patients, especially in HIV (+) or AIDS individuals (CD4<200 detectable viral load) (25), as well as in severe viral infections (chickenpox, measles, herpes zoster, herpetic gingivostomatitis, and malaria) (26), and systemic diseases – leukemia, neutropenia, uncontrolled diabetes mellitus (27). The disease activity in these patients sometimes shows limited association with etiological factors (28).

Decreased chemotaxis and phagocytosis of neutrophils also play a role in NPD (13).

Patients with immune dysfunction are susceptible to necrotizing periodontal diseases.

7. Age and Seasonality

Age and seasonality are also predisposing factors for NPD. The disease can occur at any age, but it is more common in young people with poor oral hygiene, predominantly in the ages between 22 and 25 (22).

Disease is more common in the periods September-October or December-January (29).

8. Inadequate Sleep

Inadequate sleep has been mentioned by many patients with NPD (1).

9. Excessive Alcohol Consumption

Excessive alcohol consumption also plays a role in the pathogenesis of NPD by its numerous physiological effects (1).

10. Low Socioeconomic Status

NPD is more prevalent in developing countries, where the socioeconomic status of individuals is lower.

11. Caucasian Origin

Several studies have demonstrated 95% preponderance of Caucasian patients with NPD (1,16,17).

DISCUSSION

Although NPD is rare in developing countries, it is severe and with rapid progression. Fusospirochetal infection and altered host immunity are required to initiate the disease. The bacterial microbiota is essential for the occurrence of the disease, but it is not sufficient to cause NPD.

There is a relationship between the microbial etiology of necrotic lesions and the immunocompromised host (30). A lot of factors facilitate the development of the disease in the individuals, including stress, immunosuppression, malnutrition, smoking, trauma, or pre-existing gingivitis (7). Predisposing factors may interact with the host defense systems and render the patient susceptible to NPD and that is why they are associated with the onset and severity of this disease and have an effect on the pathogenesis. Usually, just one of these factors is not sufficient to establish the disease.

Control or elimination of predisposing factors has a pivotal role in the prevention of the occurrence, progression and relapse of necrotizing diseases (31,32).

NPD is not a transmissible disease (33).

CONCLUSION

In conclusion, specific microorganisms are necessary for the occurrence of NPD, but the onset of the disease is due to the role of predisposing factors that significantly change the resistance of host response and permit bacterial invasion.

That is why it is important to collect detailed information, to assess the etiology of NPD, to identify and analyze the predisposing factors and to establish all possible preventive measures.

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