Periodontitis Disease and Systemic Condition: Overview of Systematic Reviews

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Abstract- The periodontal disease are highly prevalent and can affect up to 90% of the worldwide population. Gingivitis the mildest from periodontal disease, is caused by the bacterial bio film (dental plaque) that accumulates on teeth adjacent to the gingival (gum). Periodontitis result in loss of connective tissue and bone support and is a major cause of tooth loss in adult. In addition to pathogenic microorganisms in the bio film, genetic, dermatological, especially tobacco use, contribute to the cause of these diseases. Prevention and treatment are aimed at controlling the bacterial bio film and other risk factor, arresting progressive, disease and restoring lost tooth support.

1. INTRODUCTION

Many people have inflamed gums every now and then. A gum inflammation (gingivitis) usually doesn't cause any major problem at first. But it may spread to other parts of the periodontium (the soft tissue and bone responsible for keeping our teeth firmly anchored) and cause damage there. The medical term for inflammation of the periodontium is periodontitis. Over time periodontitis can cause teeth to loosen. Good oral hygiene can help to prevent gingivitis. Only if you clean your teeth properly can treatment by a dentist stop-or at least slow down-the progression of periodontitis.

Gingivitis is the mildest form of periodontal disease and can be found in up to 90% of the population. It is a term used to describe the inflammation of the gingiva due to the accumulation of bacteria and debris between the gum line and tooth also known as dental plaque. Gingivitis is a reactive condition that is reversible upon the improvement of oral hygiene. The bacteria then can penetrate deeper into the tissue and surrounding periodontium. This trigger a host response in an attempt to defend against the invading bacteria. During the process of protecting against the bacteria, the host defenses also lead to the destruction of the periodontium. Periodontium leads to loss of attachment of the periodontium, which subsequently progresses to alveolar bone loss of the affected tooth.[1][2][3]

The American Academy of periodontology, in collaboration with the European federation of periodontology, devise a new classification of periodontal and peri-implant diseases. In this new classification, periodontitis can be subdivided into three categories:

- Necrotizing periodontal diseases
- Periodontitis
- Periodontitis as a manifestation of systemic diseases

Necrotizing periodontal disease refers to a virulent, rapidly progressing disease that is mostly seen in immune-suppressed patients, such as those with HIV. This form of periodontal disease includes necrosis of the gingival found between the teeth, bleeding, and associated pain.[4][5]

2. ETIOLOGY

The risk factor can be subdivided into modifiable risk factors, including smoking tobacco, poor oral hygiene, diabetes mellitus, and pregnancy, and non –modifiable risk factor, like age and heredity, including genetic disease.

Improper oral hygiene techniques can lead to the build-up of bacteria and plaque on the teeth, initiating gingivitis and potentially progressing to periodontitis. This relationship has been demonstrated in the literature, with the increasing build-up of dental plaque being directly associated with increased severity and prevalence of periodontal disease. With inadequate oral hygiene, anaerobic organisms responsible for the progression of periodontal disease can then colonize in deeper areas of the periodontium where they can then execute their destructive action. The main bacteria found in periodontitis include Aggregatibacter, porphyromonas, and tannerella forsythia. These organisms produce inflammation by triggering the release of inflammatory

mediators, and other defensive products from the host.[6][7][8][9]

The most noteworthy major modifiable risk factor of periodontal disease is tobacco smoking. Additionally, tobacco smoking is associated with greater levels of bone loss, attachment loss, deep periodontal pockets associated with the disease, and tooth loss, as compared to non-smokers[10][11]. In addition to the increase severity of periodontal disease, tobacco smoking is also associated with a significant of treatment.[12][13] [14][15]

Pregnancy is associated with fluctuation in hormone level, change that have been show to promote an inflammatory response that is linked to gingivitis and periodontitis [16][17]. Although not clearly understood, maternal hormones were show to be positivity correlated with levels of porphyromonas gingivalis, a key microbe in the progression of periodontal disease.[18][19]

This aggregation of inflammatory cells puts older individuals at greater risk of experiencing the destruction of the periodontium. Additionally due to aging being associated with a loss of dexterity, older individuals tend of be less proficient with their oral hygiene practice. This result in higher plaque levels which is a known risk factor for the development of periodontal disease. [20][21][22]

The etiology of the development of periodontal disease within these systemic disease has also been documented in the literature. These disorder include down syndrome, Ehlers-Danlos syndrome (type IV and VIII).[23][24][25]

3. EPIDEMIOLOGY

Periodontal disease can be seen in up to 90% of the global population, making it the most common oral disease. In the united states alone, cross-section studies show that approximately 50% of adults currently have some form of gingivitits and up to 80% have experienced some form of periodontal disease in the their life. Lower income and education levels were also associated with severe periodontitis.[26]

4. PATHOPHYSIOLOGY

To understand the pathophysiology of periodontal disease it is essential to know about the complex dental bio film as well as the immune response associated with the disease. Dental plaque is a complex bio film which is the colonization of bacteria enclose by a protective matrix. This matrix is composed of extracellular polysaccharide and glycoproteins providing a proactive environment for microbes in the dental bio film. This component of the dental bio film makers it 1000 to 1500 times more resistance to antimicrobial agents.

The bio film by different p^H concentration and metabolites has various microenvironment within the bio film. These microenvironment makes the ecosystem suitable for a variety of microbes inhibiting in the same dental plaque.[27]

The initial layer deposited on the surface of teeth in the formation of dental plaque is “acquired pellicle”. This layer is formed within second of the exposure of tooth surface, followed by the initial attachment of the early colonizers of the bio film. Streptococcus and actinomyces species are the primary colonizer which are gram positive, facultative bacteria.[28]

The host as a response to the bacterial infection by classic innate immune response. The response includes signs of acute inflammation including increased redness of gingiva bleeding and swollen gums as well migration of neutrophils to the site of inflammation. The innate immunity also triggers the host cell to different into more specific cells in turn increasing the pro inflammatory mediators like interleukin 1-beta,prostaglandins and tumor necrosis factor.[29]

5. HISTORY AND PHYSICAL

Periodontal disease has been described as an inflammatory condition by the Chinese and Egyptians as early as 4000 years ago. He gave various treatment modalities for the treatment of periodontitis like scaling and root planning use of mouthwashes and dentifrices.

Chronic periodontitis is more prevalent in the adult population but can occur in younger patients too. The progression of chronic periodontitis is on a slower pace associated with specific microbial bacteria. Generalized signs of gingival inflammation are present with periodontal pockets ranging from 4 to 12mm.

The first reported symptom of periodontal disease is bleeding during brushing or flossing. More

severe symptoms at the time of presentation include pain and tenderness during chewing of specific substances, sensitive teeth, receding gums, the formation of discoloring plaque, tooth mobility, and loss of teeth.[30]

Routing dental screenings are invaluable in recognizing early disease states and directing early intervention. Dental probes are used to measure dental pocket adjacent to several teeth. A probing depth of greater than 3mm may be indicative of periodontal disease.[31]

6. EVOLUTION

Case of chronic periodontitis and aggressive periodontitis, both clinical as well as radiographic examination, is mandatory. Clinical examination involve estimation of local factor development discrepancies of teeth leading to increased plaque accumulation bleeding on probing periodontal probing pocket depth estimation is done manually or using pressure-sensitive probes, determination of involvement, recession, determination of clinical attachment level. Periodontal pathogens including aggregatibacter, porphyromonas gingivalis and campylobacter rectus are associated with periodontitis in various microbiological, epidemiological studies.[32]

7. TREATMENT

The main aim of periodontal therapy is to improve the gingival health of the patient and preserve the remaining periodontal tissue. Both local factor, as well as the bacteria load of periodontopathogens, require reduction along with the correction of behavioral facto such as cessation of smoking and tobacco consumption is a part of periodontal treatment.[33]

8. NONSURGICAL TREATMENT

- Root planning: Root planning smooth the root surface discouraging further buildup of tartar and bacteria and remove bacteria byproduct that contribute to inflammation and healing reattachment of the gum to the tooth surface.
- Scaling: Scaling remove tartar and bacteria from your tooth surface and beneath your gum. It may be performed using instruments a laser or an ultrasonic device.
- Antibiotic: topical or oral antibiotic can help control bacterial infection. Oral antibiotics may be necessary to completely eliminate infection causing bacteria.

9. SURGICAL TREATMENT

- Soft tissue grafts: lose gum tissue your gum line recedes. You may need to have some of the damaged soft tissue reinforced. This is usually done by removing a small amount of from the roof of your mouth using tissue from another donor source and attaching it to the affected site.
- Bone grafting: this produce is performed when periododntitis has destroyed the bone surrounding you tooth root. The bone graft help prevent tooth loss by holding your tooth in place. It also serves as a platform for the re growth of nature bone.
- Tissue-stimulating proteins: Another technique involves applying a special gel to a disease tooth root. This gel contains the same proteins found in developing tooth enamel and stimulates the growth of healthy bone and tissue.

Although rare, systemic antibiotics are sometimes indicated such as in the case of persistent deep periodontal pockets. The most common antimicrobial agent prescribed include.

- Tetracyclines
- Penicillin
- Macrolides
- Quinolones
- Cephalosporins
- Nitroimidazole compound

This pharmacological agent vary in modes of action and can be prescribed to patients with a range of susceptible microorganisms including those with antibacterial resistance.[34]

10. DETERRENCE AND PATIENT EDUCATION

Provide patients with education about how to modify risk factor such as smoking and oral hygiene. Interdisciplinary healthcare professional should work together to monitor and treat underlying causes, including diabetes mellitus. The

importance of regular dental check-ups and home oral hygiene should be reinforced to the general population.

11. DIFFERENTIAL DIAGNOSIS

Periodontal disease may depict as gingival or periodontal abscess, acute necrotizing ulcerative gingivitis, endodontic lesions. In cases of localized periodontal disease, the origin of the disease requires differentiation between pulpal and periodontal origin. Non-responding cases of preriodontitis categorize as refectory periodontitis.

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