

Review Article

Knowledge and management towards pyorrhea alveolaris

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ABSTRACT:

Periodontal diseases are disease processes involving the periodontium, a term used to describe the supportive apparatus surrounding the tooth, which includes the gingival tissue, alveolar bone, cementum and periodontal ligaments. Periodontal diseases like pyorrhea are mainly the result of infections and inflammation of the gums and bone that surround and support the teeth. It is a reactive condition that is reversible upon the improvement of oral hygiene. Periodontitis is when the periodontal condition has progressed beyond the gingivitis into a chronic destructive irreversible inflammatory disease state. The bacteria then can penetrate deeper into the tissues and surrounding periodontium.

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INTRODUCTION

As distinguished from gingivitis, periodontitis is inflammation that extends to periodontal structures beyond the gingiva, producing a loss of the connective tissue attachment of the teeth. There appear to be multiple forms of periodontitis that have differences among them in etiology, natural history, progression or response to therapy. The term pyorrhea comes from the Greek words meaning discharge of pus, which is a multifactorial disease and directly affects the oral gums and the bone. For my purpose in current situation the term projects the idea of inflammation of gum margin with progressive destruction of underlying bone. Pyorrhea is bacterial in nature and is result of advanced or untreated gingivitis. Pyorrhea affects the periodontium which holds up the gingiva, bone and ligament.

Pyorrhea at initial stage: It usually begins as a small irritated portion of gum tissue and progress slowly.

With the first death of tissue early in disease there is formation of pus in quantity too small to be seen with naked eye. It may progress many months or even many years before pus begins to flow from about the neck of teeth and teeth become loose. Idea of patient not suffering with pyorrhea till pus starts flowing from

gingival margins is not correct. At the beginning of disease gum colour changes just slightly from the normal and develops a small lesion.

Pyorrhea at intermediate stage: As the session deepens due to the irritant which initiated the disease at beginning, gum shows more evidence of irritation until finally tissue reveals presence of venous blood and gingival hypertrophy could be seen. Gums become more puffed and red, erythematous.

Pyorrhea at advance stage: The tissue destruction goes on towards the apex of tooth i.e. root breaking the integration between alveolar process as well as gum tissue and periodontal membrane. After this the tooth gets loosened due to this process of pus formation and tissue death. The session produced around tooth that cause their loosening from socket are called periodontal pockets.

Effects of pyorrhea on general/ overall health: When oral health is compromised by disease or injury, general health is also affected. The pain and discomfort associated with oral diseases leads to social isolation and mental health hazards. Pyorrhea involves pus discharge which is taken down to the

alimentary canal. Pyorrhea also leads to micro organism getting absorbed into circulation directly. Pyorrhea leading to advanced stages because of being undiagnosed and lack of awareness cause loosening of tooth one after the other and lead to destruction of their natural support. Reduces the masticating efficiency and major chunk is swallowed and the churning of food meant to be done by oral cavity is now had to be done by digestive system just affecting the alimentary canal and overall health.

Infective endocarditis: Particularly undetected persisting odontogenic infections and their secondary symptoms may lead to bacterial transfer from oral cavity into the blood stream eventually enabling the bacterial adherence to endocardial epithelium surface and thus promoting infective endocarditis. Oral streptococci possess the major threat for infective endocarditis. Dysbiosis refers to changes of the relative amount of certain bacteria ultimately resulting in the transition from a commensal to a highly pathogenic bacterial community. In terms of dental caries, this shift eventually enhances the acidic potential of the biofilm and thus leads to demineralization of dental hard tissues known as caries. Although the formation of a dental biofilm is known to be essential for the initial development of carious lesions, it must be emphasized that dental caries is based on a complex multifactorial etiology in which, in addition to the biofilm, interactions between components such as salivary flow and composition, structure of dental hard tissues, environmental factors and genetic predisposition are required. Behavioral factors such as dietary habits and "oral hygiene" play a crucial role in the formation of carious lesions.

Epidemiology: Some periodontal diseases are estimated to affect 19% global population. That estimates to 1 billion population world wide. Main risk factor being poor oral hygiene and tobacco use in rural and sub urban population. Prevalence of periodontal disease in India was 51% according to study in 2020 supported by NIH.

Pooled prevalence of gingivitis was 46.6%. Males had a higher prevalence than women. Population above 65 years age had higher tendency to develop periodontal diseases. Urban population had a higher prevalence of gingivitis. Whereas rural population had higher prevalence of periodontal diseases. Role of fibroblasts in periodontal diseases. Human gingival fibroblasts (HGFs) are the most abundant cells in gingival connective tissue [1]. Human periodontal ligament fibroblasts (HPLFs) are located between the teeth and the alveolar bone, namely, the "periodontal ligament", and contribute to the stable embedding of teeth [2]. These cells are typical fibroblasts in periodontal tissues, and they maintain the homeostasis of connective tissue through the secretion and degradation of components of the extracellular matrix, such as collagen. The degradation/destruction of the

extracellular matrix in connective tissue has been detected in periodontal lesions. On the other hand, the accumulation of the extracellular matrix has been reported in gingival overgrowth. Although drug-induced gingival overgrowth may occur with "inflammation" in periodontal connective tissues in clinical settings, periodontitis is a secondary issue induced by the infection of periodontal bacteria into the deep periodontal pockets caused by the enlargement of gingival tissues.

Symptoms that help in making the confirmatory diagnosis:

1. gum inflammation - Early stage of pyorrhea is characterised by red swollen and tender gums
2. bleeding gums - Gums often bleed while flossing/brushing indicating an issue
3. gum recession - As the disease advances gums may pull away from teeth leading to exposed tooth roots
4. tooth mobility - Occurs in disease due to weakened bone support
5. bad breath - Foul smelling breath often accompanies periodontitis due to food enlargement in periodontal pockets and getting degraded in oral cavity by bacterial colonisation
6. pus formation —pockets of pus often appear between teeth and gums

Treatment

1. Scaling and root planning: Egis non surgical procedure involves removing the tartar and bacteria from tooth surfaces and smoothing root surfaces to aid gum attachment
 2. periodontal surgery : In severe cases surgical interventions have to be made where Opg is suggested to confirm the diagnosis of progressive bone loss is made - which is further treated with splinting over 3-4 weeks and then attempting a flap surgery to scrap the de-mineralised bone and granulation tissue
 3. antibiotics: Mostly anaerobic antibiotics such as metronidazole are prescribed considering them the major causative agent
 4. laser therapy: It have proved to be boon for removing diseased tissue and promoting healing
 1. maintaining excellent oral hygiene
 2. regular dental checkups
 3. smoking cessation
 4. Balanced diet
 5. Managing underlying medical conditions such as diabetes which pave a way for opportunistic infections
 6. Giving the history of chronic medication for systemic disease to arrest the disease at present stage
- The expression of gingivitis can be modified by some relatively commonly used medicines, especially certain agents for treatment of convulsive disorders, some cardiovascular drugs and certain immunosuppressants. The modification consists of a hypertrophy of the connective tissue elements of the

gingiva (primarily collagen), so that the gingiva appears swollen or overgrown. The amount of associated inflammation is a function of the accumulation of bacterial plaque. The prototypical gingival hypertrophy-producing central nervous system agent is phenytoin (or diphenylhydantoin). About half of patients who chronically take phenytoin develop the gingival overgrowth. Hypertrophy-producing cardiovascular agents are primarily calcium channel blockers, such as nifedipine and oxodipine. Some other calcium channel blockers also are associated with gingival overgrowth. The immunosuppressant cyclosporin represents the other major class of drug associated with gingival hypertrophy.

Ex: in some patients undergoing cardiac medications are sensitive to amlodipine and lead to pyorrhea

Drugs / ointments with combination of metronidazole, chlorhexidine and lidocaine have been found to be highly contributing in providing relief and arresting the disease at that stage

CONCLUSION

Keeping in view the progressive nature of disease, one should seek dental clinician as early as possible. Considering the non-surgical and surgical approach for treating the disease, maintaining oral hygiene plays a key role in prevention of disease. Attention not paid to oral hygiene after treatment could lead to recurrence.

Ointments prescribed for gingival hypertrophy play a major role in arresting the disease at that stage. Once having history of any other chronic illness like diabetes, should pay attention to overall health including oral health. Not getting rehabilitation done once undergoing serial extractions also paves a way for periodontal

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