


Epidemiology of Periodontal Diseases			Healthcare
Arjola Metushaj		'Eurodent' Clinics ,Vlore, Albania.	
Abstract			
<p>Periodontium is a complex organ consisting of gingiva, periodontal ligament, cement, alveolar bone and neurovascular wisps. Alveolar process consists of three structures: the lamina dura, trabekulare bone, and compact bone. Lamina dura is covered by compact bone and serves for fixing the periodontal ligament fibers. Vascularization: it is provided with blood from alveolaris superior artery for the upper jaw and from alveolaris inferior artery for the lower jaw. Upper jaw: from the terminal branches of dental artery, posterior alveolar artery, and the infraorbital artery and that Palatine Major. Lower jaw: from the branches of the facial artery, a. mentalb.buccal, and c. sublingual.</p>			

Introduction

Periodontal Diseases are dividend into two groups: 1. Gingivitis; 2. Periodontitis.

Periodontal Diseases are caused by the presence of bacterial plaque that is localized only in gingivar tissue which means it is a reversible inflammation.

These diseases consist of Gram-positive microorganisms, such as: Actinomycete, Lactobacillus and Streptococcus and gram-negative microorganisms, such as: Porphyromans, Prevotella, and Aggregatibacter and Bacteroides. In the inflamed gums it is ascertained the presence of Treponema. Bleeding of gums is caused by actinomycetes. Clinical symptoms of inflammation of the gum tissue (gingiva) are the following: 1. Rubor (color) 2. Tumor (swelling) 3. Calorie (local temperature) 4. Dolor (pain) 5. Spontaneous bleeding

Etiology of Periodontal Diseases

Periodontal disease involves not only inflammation of the gums but also the periodontal ligament and alveolar bone.

There are two main factors:

1. Local factors: affects in the progress of inflammation the microbial mass quantity and their quality, three types of bacteria have been identified and that affect periodontal diseases and are called also as 'red complex', such as: 1.Porphyromonas gingivalis 2.Bacteriodes Forsythus 3. A.Actinomycetemcomitans (before Aggregatibacter-actinomycetemcomitans)

Aggregatibacter-actinomycetemcomitans: causes Juvenil periodontitis (which appears on the ages of adolescence).

Accumulation of plaque in the level of gingival sulcus¹⁶ favors bacterial products to cause inflammation of the gums where there are locally observed changes in Gingival crevicular fluid (GCF)¹⁷.

¹⁶ The gingival sulcus is an area of potential space between a tooth and the surrounding gingival tissue and is lined by sulcular epithelium. (Source: en.wikipedia.org/wiki/Gingival_sulcus)

This fluid is *eksudat serosa* that accumulates in gingival sulcus level and affects the formation of crevicular fluid that can produce mediators of inflammation. This mechanism is an inflammatory response with neutrophil predominance of macrophages and lymphocytes that migrate and destroy enzymes and mediators of inflammation.

Mediators such as cytokines, metalloproteinases favor tissue destruction of periodontitis. Immune response that is expressed with cytotoxic protein production will influence to the loss of periodontal structures. Dental stones: operate in marginal gingiva board disrupting the structure of connective attachment.

- Environmental factors: tobacco use is a risk factor for periodontal diseases and increases for 4 times more the prevalence of periodontal disease.

- General factors:

- Age - with age increasing, the prevalence of periodontal disease increases, too.

- Sex - men are more affected than women

- Genetics - in monozygotic twins vulnerability is the same while in heterozygote twins vulnerability to periodontal disease is not the same.

- Gingivitis of pregnancy: affects 30-100% of females in the second month of pregnancy. It is accompanied by redness and bleeding gums - The presence of granular tumors (swelling of the gums). It is apparent a rise in the microbial flora of subgingiva dental plaque – Gum problems normalize after birth.

Gravidar Gingivitis: localizes in subgingival level and consists of aerobic, anaerobic, and *Bacteroides melaninogenicus* microorganisms (*Prevotella melaninogica*) and *Prevotella intermedia*.

- Stress - affects in the hormonal disorder that normally leads to increased risk for periodontal disease.

- Divorce - financial problems and depression affect periodontal diseases.

- Diet and way of nutrition - effect in the development of dental plaque. Some vitamins have anti-inflammatory action and positively affect in the condition of periodontitis.

- People with overweight – are risk factors for periodontal disease.

- Immunodeficiency diseases: AIDS - is a risk factor for periodontal disease and have a high prevalence.

- To the patients with HIV seropositive it is often noticed the presence of ulcero-necrotic periodontitis.

- The impact of systemic diseases: Diabetes mellitus type 1 and 2 are risk factors for periodontal diseases.

- Socio-economic factor: is a risk factor for periodontal diseases however it is much more combined with other factors.

Assessment of risk for periodontal disease is based on microbiological results of subgingival plaque. Analysis of subgingival plaque: - It has to do with examining the type and amount of plaque - the presence of periodontal pathogens hydrolyze the BANA enzyme (that influences the birth of periodontopathic bacteria) - organisms that are able to colonize subgingival plaque are: *Treptonema denticola*, *Bacteroides Porphyromonas gingivalis* and *forsythus*.

- Temperature in the subgingival region: - is indicative of the condition of gum inflammation – The increase of temperature is an inflammatory response periodontitis - Temperature is measured in six places for a tooth.

¹⁷ Gingival crevicular fluid (GCF) is an inflammatory exudate that can be collected at the gingival margin or within the gingival crevice. (Source: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3453730/>)

- Genetic analysis of the risk for periodontal disease: - It is used the PST test, Boston, MA - which determines the presence of two genes, interleukin 1, which according to the data is a periodontal risk factors for chronic diseases. To determine the polymorphisms of the gene it is used the blood test analysis taken in finger.

- Gingival crevicular fluid (GCF) –It is sweat liquid, which with an active mechanism affects in preserving homeostasis in the gum's region. During gingival inflammation it turns into a juice liberating immune cells and cytokine. Examination of the gingival fluid is a very important element for early diagnosis of periodontal diseases.

- Mouth Cancer: Diagnosis of saliva is the main method of early detection of mouth cancer. In mouth cancer we have a large number of proteins as well as nucleic acid.

- General Diseases: Many chronic disease their first manifestations exhibit in front of the oral cavity. - It is believed that the microbial flora of dental plaque can enter into the blood and present risk for cardiovascular diseases, diabeitit mellitus.

- Diabetes Mellitus: increases the risk for periodontal disease. It is emphasized that the glycosylated hemoglobin (HbA1c) is considered as risk factor for periodontal disease as well as prolonged drug treatment in years for these diseases, such as diabetes, hypertension, heart disease, renal diseases, pulmonary diseases all affect directly or indirectly in the oral cavity.

Measuring Systems of Periodontal Disease

Gingivitis is caused by gums that bleed during and after tooth brushing, red, swollen, or tender gums, persistent bad breath or bad taste in the mouth, receding gums, formation of deep pockets between teeth and gums, loose or shifting teeth, changes in the way teeth fit together upon biting down, or in the fit of partial dentures.¹⁸

Gingival index (GI) – Is estimated from 0-3

- 0 is the normal state

- 3 severe inflammation is characterized by edema, redness, softening and spontaneous bleeding – It is based during a light probing.

The Eastman interdental bleeding (EIB)- Also is used for measuring gingival bleeding gingivare and their inflammation. It is evaluated by means of an interdental right stake which enters into the interproximal space parallel to the plan of occlusion: 1-2mm under the gingiva.

Modified Gingival Index (MGI): - Rating is based on color, edema and spontaneous bleeding of gums in 4 scales (0-4)

- plaque index (PLL): - It is estimated the subgingival plaque on its thickness during the gingival edge. Periodontal proces are used. It is estimated by scales from 0-3.

- periodontal index (PI): - Defines the scope and scale of advancing periodontal disease, carried by a periodontal probe in particular teeth such as:

1. Incisive Central maxillar left ICML
2. Incisive Central maxillar right ICMR

¹⁸ <http://www.webmd.com/oral-health/guide/gingivitis-periodontal-disease>

3. Molar 1 maxillar right M1MR
4. Molar 1 maxillar left M1ML
5. Premolar 1 maxillar left P1ML
6. Premolar 1 mandibular right P1MR

- Clinical Level of Fixation (CLF): - It is estimated by measuring the depth of the pocket and gingival move – in the smalt level - cement with periodontal probe. - Depth of sonding is done for every tooth in 6 dots: Mezio-buccal, disdo-buccal, mezio-lingual, disto-lingual, lingual and buco-facial.

Preventive Strategies for Periodontal Diseases

- Dental plates: it is the main etiological factor of periodontal disease consisting of bacerial flora complex located on the surface of teeth.

Initially, it is formed a pelicula which enables adhesion of bacteria to the oral cavity where later the plates gradually mature, bacteria are added and bacterial colonies are formed, gram-negative microbes are placed and aerobes which are responsible for the onset of periodontopathies.

To manage periodontity is necessary elimination of subgingival plaque, which is the primary object for the prevention of periodontal disease as well as and teeth washing, oral washings and dental use of disinfectants have a great importance in the prevention of periodontal diseases. A particular importance is also the time of teeth brushing which should not be less than 1 minute for each quarter of the jaw.

- ADA (Association of American dentistry) has approved two antiseptics for the mouth rinse: chlorexidine (CHX) and essential oils (EO)

– Chlorexidina(CHX):

- Acts against fungus and viruses
- Affects to plaque reduce from 50% to 60% for 12-14 hours, impacting on reducing gum inflammation up to 45%
- It is used in concentration of 0.12% twice a day before bedtime for 30sec for 14 days in a row. It should be used for 30min before and after "teeth brushing"
- It reduces the leve of Streptococcus mutans in the human's shliva.

Chlorexidine (CHX) 0.05% with sodium fluoride (NaF) 0.05% impacting on the improvement of bleeding gums and their inflammation.

Side effects: 1. Irritation of oral mucosa 2. Superficial desquamation of these tissues 3. Hypersensitivity of teeth 4. Formation of stains on the teeth surface

– Essential Oils (EO) - Affects in the elimination of microbial flora in subgingival plaque and gum inflammation.

- Listerine: rinsing with it is as effective as flossing in fighting tooth and gum decay. It prevents from dental supa and subgingival plaques formation. It is used twice a day with an amount of 20ml per 30sec. Contains fluoride to prevent tooth decay.

Toothpastes should contain: Triclosan, Stanium Fluoride

- Triclosan: It is considered as an anti-plaque agent. Toothpastes containing 0.3% triclosan and 2% co-polymer are considered pastes with high antibacterial action that affect in the inflammation of gums.
- Stanium Fluoride: It has a high antibacterial action. Toothpastes containing pirophosphate inhibit the formation of dental stones. It is worth mentioning that prevention strategies should be based on risk factors for periodontal diseases, such as: genetic factors, the way of nutrition, diabetes and smoking.

References

1. Berglundh T, Lindhe J, Ericsson I, Marinello Cp, Liljenberg B, Thomsen P The soft tissues barrier at implants and teeth, 1991 (81-90)
2. Davarpanah M, Martinez H, Kebir M, Etienne D, Tecucianu J.F. Wide –diameter implants new concepts, 2001 (149-159)
3. Bahato Handelsman M Use of wide implants and double implants in the posterior jaw: A clinical report, 1996 (379-386)
4. Saadoun Andre P Des micro-inserts ultrasoniques dans le traitement parodontal et en maintenance implantaire Federation des Medecins de France, 2002 <http://fmf.affinitesante.com>
5. Friberg B, Jemt T Lekholm U Early failures in 4641 consecutively placed dental implants; a study from stage 1 surgery to the connection of the completed prosthesis, 1991 (141-146)
6. Jaffin R.A. Berman C.I. The excessive loss of Branemark fixtures in type IV bone: a 5 year analysis, 1991
7. Wennerberg A. Implant design and surface factors, 2003
8. Khayat P, Chiche F, Exbrayat P Interet et indications d'un implant vise a hexagone interne: le Screw-Vent, 1994 (309-321)
9. Exbrayat Patrick Le titane et ses alliages, 2003
10. Langer B, Langer I, Herrmann I, Jorneus I The wide fixture: a solution for special bone situations and a rescue for the compromised implant. Part 1, 1993 (400-408)
11. Bert Marc, Missika Patrick Implantologie chirurgicale et prothetique, 1996
12. Niznick G.A. A multimodal approach to implant prosthodontics, 1989 (869-878)
13. Aparicio C, Orozco P Use of 5mm diameter implants, 1998, (398-406)
14. Attard Nj, Zarb Ga Implant prosthodontic management of partially edentulous patients missing posterior teeth, 2003 (352-359)