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### V. Dvrvk

Department of Therapeutic Dentistry FPDO, Dental center of Danylo Halytskyi Lviv National Medical University, Lviv, 79010, Ukraine.

### R. Shkrebnyuk

Department of Therapeutic Dentistry FPDO, Dental center of Danylo Halytskyi Lviv National Medical University, Lviv, 79010, Ukraine.

## Symptomatology of inflammatory parodontal disease

### V. Dyryk, R. Shkrebnyuk

On the basis of own observations and literature analysis of the most frequently prevalence clinical symptoms in cases of inflammatory parodontal disease is given and their pathogenetic fundamentals are disclosed. Clinical symptoms of inflammatory diseases are considered as sublimation of their pathogenetic fundamentals and as clinical manifestations the basic for pathology processes of tissue structure.

**Keywords:** Gingivitis, Parodontal disease, Inflammatory.

### 1. Introduction

The most frequently occurring clinical symptoms of inflammatory parodontal disease and their pathogenetic fundamentals are discussed in this article. The author describes mechanisms of chronic inflammatory process symptoms formation in parodontal tissues. Clinical symptoms of inflammatory diseases are considered as sublimation of their pathogenetic fundamentals [3, 4].

Investigations of inflammatory parodontal disease and its pathological mechanisms were regularly carried out. However diagnostic errors often occur. Theoretical knowledge of the dentist, individual and general clinical experiences are of great importance in diagnostics and parodontal disease treatment.

The symptomatology of inflammatory parodontal disease is characterized by a number of common features [1, 4, 5, 6].

Usually patients seek medical advice of the doctor only when they have chronic inflammatory parodontal disease. This phenomenon can be explained by their underestimation of pathological parodontal changes. Destructive changes prevail in the clinical symptoms of the chronic inflammatory process.

Important inflammatory parodontal symptoms are connected with modifications in blood vessels, such as reddening, swelling and stomatorrhagia.

The purpose of research was to study all symptoms mentioned above have a pathogenetic similarity.

### 2. Results of the investigation and their discussion

Acute gingivitis and its exacerbation are characterized by hyperaemia producing reddening of gingival mucosa. Hyperaemia develops during inflammation as a result of dilatation of capillary blood vessels. Delivery of blood to the affected area increases, and the gum tissue looks as if it contained more vessels than it must have according to standards.

Oedema may appear on the background of inflammatory chronic parodontal disease. Oedema is an enlarging of the gum volume. It is connected with the yield of plasmatic proteins and cell elements beyond the limits of blood wallsides as a result of their permeability violation and increasing of osmotic and oncotic pressure. It increases due to interstitial substance degradation originating under the influence of inflammatory cytokines. The gum structure undergoes changes because of the oedema [8, 7].

Special attention should be paid to the bleeding of the gums under small mechanical effects. This symptom is the earliest indication of a developing inflammatory parodontal disease.

The pathogenesis of this symptom is associated with increasing damage of vessel wallsides as inflammation progresses. During this process the membranes permeability of expanded microblood vessels and their wallside fragility are a deciding factor. Susceptibility to ruptures of blood vessels is increasing. This produces a hemorrhagic effect [2, 10].

Parodontitis pain may be provoked by teeth and inflated gums.

Teeth hyperesthesia at the denudation of their dental cervix / tooth roots is an usual case on the background of an acute inflammatory parodontal process. It is connected with the opening of

**Correspondence:** 

V. Dvrvk

Department of Therapeutic Dentistry FPDO, Dental center of Danylo Halytskyi Lviv National Medical University, Lviv, 79010, Ukraine.

dentinal canaliculus and with the development of hydrodynamic reactions inside them as well as with the changes of osmotic and oncotic pressure in the dentinal liquid [1,2]

Gum aches are not as regular as tooth aches. Complaints about discomfort and painful symptoms relatively occur not very often and become evident only when the chronic disease grows acute and manifests itself.

Complex mechanisms of alarm signals provoking under inflammation the development of stress organism reactions (systematic or local) are the basis of ache as a clinical symptom [9].

There are only a few clinical investigations of pain with inflammatory parodontal disease in our home and foreign stomatology. The pathophysiological fundamentals of this disease are practically not studied. In adjacent medical and clinical fields more attention is paid to the problem of pain [11]. Very often gum inflammation without pain males easier the life of a practicing dentist and prevents him from a crowd of patients with pain. In the same time because of its absence patients appear at the parodontologist's too late.

One of the hidden symptoms of parodontitis is the parodontal pocket. Its formation separates this nasologic type of illness from the preceding chronic gingivitis. The formation of the parodontal pocket starts with the deepening of the dentogingival sulcus. During medical inspection with probing we can not learn whether the parodontal pocket was formed or not [7, 10].

The only criterion for exact diagnostics may be the (un accessable for clinical methods) estimation of the dentogingival junction state.

It is necessary to note that the microbe plaque and the dental calculus (especially the subgingival one) play the most important role in the pathogenesis of parodontal destructive processes, including the parodontal pocket formation.

It is generally recognized that there exists a direct correlation between the parodontal pocket depth and the parodontitis stage. Therefore the indices of the parodontal pocket depth are applied to deter mining the parodontium degree in the clinics, though the existing methods for measuring the parodontal pocket depth are extremely imperfect.

One of the main specific features of parodontitis is the pathological mobility of teeth. This symptom is connected with the damage processes on the background of inflammation in the parodontal ligament, in the surrounding interstitial substance as well as in the dental alveolus tissue [4, 10].

One of the very apparent clinical symptoms of an active inflammatory destructive parodontal process is the tooth root denudation.

During our clinical observations this denudation was found in patients having a light parodontitis and in patients with a medium parodontitis. The development of tooth root denudation is directly associated with destruction of the bearing tooth tissues and with reduction of their volume. We must remember that as a result of an active root denudation clinically discovered parodontal pocket depth decreases.

The indices of hygiene state and mouth cavity are not symptoms, they rather refer to the sphere of quantitative estimation of the etiological microbe factor in relation to parodontal inflammation. Clinical experiments proved that the processes of dental calculus formation (microbe plaque) and parodontal inflammatory dynamics are parallel. Usually this inflammation starts with typical gingivitis symptoms which are transformed in the course of time into parodontitis.

Our investigations have shown that together with the dental deposit and dental calculus the parodontal inflammation starts in the dentogingival junction. It is due to the influence of microorganisms on the epithelial cells of the gum and gingival mucosa, and under the microbe invasion – on the subepithelial conjunctive tissue basis.

The toxic influence of microorganisms vital function products activates cell and humoral inflammatory mediators and modulators, which sharply increase the permeability of dental blood wallsides. An active hyperaemia turning into a vessel stasis is developing [3, 11].

The above mentioned clinical symptoms of inflammatory parodontal disease do not cover all the complexity and variety of the problem, among them: the x-ray method; the laboratory methods (cytological, biochemical, microbiological) in order to determine the parodontal disease features.

### 3. Conclusions

Thus, the parodontal disease clinical symptoms can be divided into two groups: symptoms common for gingivitis and parodontitis and symptoms characteristic only of parodontium. Common symptoms are vascular reactions and disfunctions, as well as a bad smell from the mouth, feeling of discomfort, pains in the gums, teeth hyperesthesia [1,11].

Extremely important for a correct diagnosis of the parodontal disease is the evaluation of the month cavity hygiene with the help of hygienic indices.

The pathological mobility of teeth and the formation of parodontal pocket refer to the second group of clinical symptoms. These clinical symptoms reveal themselves when the destructive component symptoms become apparent (parodontal pocket, pathological mobility of teeth, their dislocation etc.).

The first group of clinical symptoms only shows if the given pathology is a standard or a disease [2, 10].

The second group of clinical symptoms can serve as an objective criterion for differentiating gingivitis from parodontium. However it is possible only in the case of a well developed parodontium with evident parodontal clinical symptomatology.

### 4. References

- 1. Tozum T, Taguchi A. Role of dental panoramic radiographs in assessment of future dental conditions in patients with osteoporosis and periodontitis // NY State Dent. J 2004; 70:32-35.
- Glostalo PV, Ek E, Daitinen J. Optimism and dife Satisfaction as Determinants for Dental and General Health Behavior – Oral Health Habits linked to Cardiovascular Risk Factors, m.g. // Dent Res 2003; 82(3):194-199.
- 3. Rog C. Page Periodontitis and respiratory diseases: discussion, contusions, and recommendations // Ann. Periodontal 2001; (6):91-98.
- Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstruegement and Prevention of Chronic Obstructive Lung Decease // National Institutes of Health. National Heart, Lung and Blood Institute; updated, 2003, 1-96.
- 5. Van der Weijden YA, Timmerman MF, Dancer MM, Nijboer A, Saxton CF, Vander Welden W. Effect of preexperimental maintenance care duration on development of gingivitis in partial mouth experimental gingivitis model. J. Periodont Res 1994; 29:168-173.

- 6. Noguchil K, Ruwanpural SM, Yan M. *et al.* Downregulation of interleukin –induced matrix metalloproteinase 13 expression via EPI receptors by prostaglandin E2 in human periodontal ligament cells // Oral Micriol. Immunol 2005; 20(1):56.
- Waschul B, Herbor HA, Stiller-Winkler R. et al. Effect of plague, physiological stress and gender on crevicular IL-Ibeta secretion // G. Clin. Periodontal 2003; 30(3):238-248.
- 8. Clapp BR, Hirschield YM, Storry C. *et al.* Inflammation and Endothelial Function: Direct Vascular, Effects of Human C-Reactive Protein on Nitric Oxide Bioavailability Circulation 2005; 111(12):1530-1536.
- 9. Duffield GS. The in amatory macrophage: a story of jekill and Hyde // Clinical Science 2003; 104(1):27-38.
- 10. Keles YC, Acikyoz Y, Ayas B. Determation of systemically and locally induced periodontal defects in rats // Indian J. Med. Res 2005; 121:176-184.
- 11. Van Dyke TF., Sheilesh D. Risk factors for periodontitis. // G. Periodontal 2005; 7(1):3-7.