Comparison between Different Classifications of Periodontal Diseases and Their Advantages

Ilma Robo, Saimir Heta, Merilda Tarja, Sonila Kapaj, Eduart Kapaj, Geriona Lasku

Abstract—The classification of periodontal diseases has changed significantly in favor of simplifying the protocol of diagnosis and periodontal treatment. This review study aims to highlight the latest publications in the new periodontal disease classification, talking about the most significant differences versus the old classification with the tendency to express the advantages or disadvantages of clinical application. The aim of the study also includes the growing tendency to link the way of classification of periodontal diseases with predetermined protocols of periodontal treatment of the diagnoses included in the classification. The new classification of periodontal diseases is rather comprehensive in its subdivisions, as the disease is viewed in its entirety, with the biological dimensions of the disease, the degree of aggravation and progression of the disease, in relation to risk factors, predisposition to patient susceptibility and impact of periodontal disease to the general health status of the patient.

Keywords—Periodontal diseases, clinical application, periodontal treatment, oral diagnosis.

I. INTRODUCTION

PERIODONTAL pathologies have been considered as inflammatory diseases of the tooth-bearing structures caused by a bacterial attack, leading to progressive destruction of the periodontal ligament and alveolar bone structure. The progression of this pathology may lead to the appearance of pocket effusion or supra-osseous intra-osseous [1]-[3]. This continuity is also in the hands of the patient, as everything depends very much on the degree of personal oral hygiene of the patient. In the condition of absence of oral hygiene and in the presence of gingival inflammation, plaque-induced gingivitis progresses to apparent inflammatory hypertrophy even in the appearance of pseudo-pocket or supragingival pockets [4]-[6]. The other clinical case is the case of a patient with good oral hygiene, but the presence of a single bacterium A. actinomycetemcomitans causes periodontal disease in the phase of activation and inactivation depending on the individual immunity of the patient, often passes into the appearance of gingival recessions with no tendency of gingival bleeding [6]-[10].

The presence of persistent spontaneous bleeding during probing is an important indicator of the presence of inflammation and the potential for subsequent attachment loss in the bleeding area.

According to the agreement of the North American Conference in 1989 and in Europe 1993, the ways of classifying periodontal disease were identified. The old classification made the division of gingival diseases as gingivitis caused by bacterial plaques and gingivitis caused by non-bacterial plaques, chronic periodontitis, aggressive periodontitis, periodontitis as a manifestation of systemic diseases, periodontal necrotizing periodontitis, periodontal-endodontic lesions, developmental or acquired deformities, and predisposing conditions. This classification was presented in 1999 at the International Conference on Classification of Periodontal Diseases organized by the American Academy of Periodontology [1], [4].

The new classification of periodontal diseases was drafted in 2017 to present and summarize periodontal diseases in different groups. The classification of periodontal diseases is made depending on its three general manifestations: periodontal health gingival diseases and conditions, periodontitis and other conditions affecting the periodontium [4], [11].

Marginal gingivitis affects the structure of the marginal gingiva. Marginal gingiva is distinguished from the fixed gingival through a minimal sulcus, visible by a clinical trained eye of a professional in the field of dentistry. This sulcus tends to increase or deepen physiologically, mainly against sex hormone fluctuations, which is again distinguishable from the watchful eye of a professional [12]-[15].

The marginal gingiva is covered by the oral epithelium on the oral surface and by the sulcular epithelium toward the inside of the sulcus. Beneath the epithelium are gingival fibers blistered into the extracellular matrix. Gingiva has resilience explained this by the presence of elastic fibers which in percentage are much less than collagenous fibers.

Collagen fibers are bundled and each has its own main functions. The gingiva finds its anatomical end or structural limit up to the transseptal fibers, fibers that have maximum regeneration ability even after structural degeneration. This ability is also explained by the anatomical limit, the presence of transseptal fibers that explains the reversibility of the initial gingival disease, which can then progress to periodontitis. The transseptal fibers intersect with the dento-gingival fibers that radiate from the tooth surface in transverse directions to the gingival surface, around the tooth [11], [15].

Chronic periodontitis, the structure that affects the dental structures, is the periodontal ligament. The periodontal ligament consists of collagen bundles organized into six types, depending on their positioning and function. The fibers from the coronal direction to the apical direction are 6 groups:
dentogingival, transpalatal, oblique, vertical, apical and interradicular. Among these fibers, regardless of the type, there are Sharpey fibers which are 5 bundles that are distributed, re-connected and then inserted into the structure in front of the tooth-supporting structures. All bundles of periodontal ligament swim in the intercellular fluid [1]-[3], [16]-[18]. The periodontal ligament functions on basis of the theory of elasticity, due to the possibility of compression of the periodontal ligament bundles, losing possible ripples and reducing the space between the cementum and the bone surface. But the opposite also happens, the ripples of the periodontal ligament can also be directed in cases of pulling of the tooth surface against the bone surface. Both these cases of interaction may be associated with clinical cases of tooth intervention. In the case when the tooth is compressed in the alveoli, the ripples of the periodontal ligament bundles depending on the direction of the force exerted on the tooth surface, increase resulting in the reduction of the periodontal ligament space. The opposite happens if for example we try to extract the tooth, the ripples of the periodontal ligament are directed thus increasing the space of the periodontal ligament depending on where these forces acted [14], [15], [17], [18].

The other theory of periodontal ligament function is the theory of the viscosity and mobility of the matrix from the periodontal ligament space to the bone surface in the case of compression of the tooth against the alveolus [12]-[15]. These movements are not textually sensitive to a single tooth because the entire dentoalveolar apparatus functions as single one and these micro-movements cannot be felt if the amount of moving material does not exceed the norm. In it, against excessive compression or excessive elongation of the periodontal ligament, sensitive forces are exerted on the bony surface. In cases when compression of the periodontal ligament is exercised, water moves towards the bone and the periodontal ligament with collagen bundles compresses, force is exerted on the bone surface, exerting compression on the structures of blood vessels, accompanied by lack of oxygenation of the affected structures bone, if the pressure on the tooth surface persists [19], [20]. Lack of oxygen supply to bone structures indicates the possibility of stimulation of osteoclasts through osteoblast baroreceptors, which enable osteoclast activation. Osteoclasts promote bone resorption. In cases of tension of the ligament fibers the latter exerts an attractive force against the bone, dilating the existing blood vessels in the bone. The dilation of the blood vessel inside the affected bone increases the flow of oxygen-rich blood, which is felt by osteoblasts which are activated to produce new bone with the tendency to narrow the space of the periodontal ligament expanded with time [21]-[24].

Chronic periodontitis occurs with the right moment correlation of complex interactions of infectious agents and host factors.

The onset of the disease, the individual susceptibility to the disease, the progression or aggravation of the disease are significantly influenced by genetic, environmental factors, as risk factors for susceptibility to chronic periodontitis. Among the environmental risk factors, smoking has been found to be associated with increased prevalence and severity of periodontal disease, also a disproportionately high number of people with severe periodontal disease are smokers. It is an expression that there is a strong link between smoking and the unusual form of periodontitis that is resistant to treatment [25], [26].

Based on the literature, in a study evaluating the effect of non-surgical treatment on smokers and non-smokers, the pocket reduction rate was significantly lower in smokers than in non-smokers. The most noticeable difference was observed for the anterior pockets in the maxilla. Moreover, this local effect is also confirmed by the observation that smokers, in general, have more periodontal pockets in the anterior segments, than in those who have not smoked. The studies conducted at MNR Dental College and Hospital, Sangareddy, Hyderabad, India, included the assessment in 150 individuals, with 75 smokers and 75 non-smokers, in the 35-60 age group [2], [5], [29], [30]. Dividing into equal groups of patients, depending on the two subgroups, makes the evaluation of the results of the study somewhat simpler [2], [5], [29], [30].

Periodontal assessment, including periodontal pocket depths and bleeding index, was performed in all four quadrants and in six dental areas, using the Williams periodontal probe. Patients who were systemically healthy, with chronic periodontitis, and smokers who consumed ≥ 10 cigarettes per day for 10 years were included in the study [29]. Patients who underwent periodontal therapy and who were on antibiotic therapy were excluded. For both parameters (pocket depth, bleeding index) the average results in the oral cavity were calculated, as well as specific results separately for the maxilla, mandible, or buccal, lingual surfaces, or depending on the affected teeth, as specifically for the premolars and anterior molars. During the analysis of the collected data sub-categories based on pocket depth, 0-3 mm, 5 mm, 6-7 mm, and ≥ 8 mm. All these sub-categories according to certain categories based on pocket depth, 0-3 mm, 5 mm, 6-7 mm, and ≥ 8 mm. All these sub-categories according to certain subgroups yielded results among smokers and non-smokers, using the z test as a statistical analysis [32], [33].

The effect of vasoconstrictors and the reduction of oxygenation tension in tissues create compatible subgingival and well-organized environment for the colonization of anaerobic bacteria. Smoking causes higher local temperature in the oral cavity, especially in the palatine and buccal surfaces. Compression of the tongue over the lingual surfaces of the mandible incisors protects these teeth from the effect of local temperature rise. So, the concentration of this temperature change in certain areas of the oral cavity expressing the effect of smoking, is different.

Smoking beyond the local temperature, depending on the type of tobacco consumed, also causes intraoral smoke with mini tobacco residues, which have the ability to be fixed on the surface of the tooth causing specific smoking staining. These staining starts from the lingual surfaces to emerge on the interproximal surfaces and then depending on the severity of smoking, this kind of staining appears even more on the vestibular surfaces of the teeth. This is the moment when the patient becomes aware and requests a dental appointment to remove this staining [34]-[37]. Smoking on the other hand
also has "positive" effects. Among these effects we can mention the addition of gingival fluid. The fluid added to the sulcus prevents the adhesion of bacteria to the oral cavity, especially over the surfaces of the affected teeth. Smokers consequently attach to the picture of the chronic periodontium. A smoker with good oral hygiene tends to have the appearance of chronic periodontitis in the oral cavity, where the only concern of the patient will be the superficial vestibular staining [31]-[33].

The difference in the shapes and types of gingival pockets in smokers and non-smokers has been suggested for the local effect of smoking on tooth-bearing structures. The study [37] showed that the percentage of pockets ≥ 5 mm varied in almost all cases, these data were divided for areas in the maxilla and mandible, for buccal and lingual surfaces and mainly depending on the type of teeth. The positioning of the tooth in the oral cavity indicates or rather signals the vulnerability to the local effect of smoking. In the maxilla, anterior teeth, and premolars, the greatest differences in values were found between smokers and non-smokers. Smokers have more areas or teeth with pocket depth ≥ 5 mm, especially on the lingual surface of these teeth. The depth categories of periodontal pockets showed that smokers generally had fewer shallow pockets and more pockets between 4 mm and 7 mm, values that indicate the design of a conservative periodontal plan supported by surgical periodontology. The difference between smokers and non-smokers was most pronounced in the anterior maxillary region [37], [38].

Finding the right keywords, their application in the relevant internet search network, makes it possible to find published articles with the right information on the classifications of periodontal diseases.

The unification of denominations and the application of newer denominations is also a professional challenge on the part of dentists, as it requires the right concentration and desire to assimilate and then apply the new classification system.

II. MATERIALS AND METHODS

The study is of the review type, with the aim of finding the latest publications on the classification of periodontal diseases.

The electronic search was conducted to find the items using mainly MEDLINE, PubMed in the period 2017 to 2020.

The first stage: It involved finding the key terms, derived from articles that talked about the types of periodontal disease. These terms, in the MESH database on the PubMed Site, were:

- Periodontal disease (91282)
- Periodontal classification (4309)
- Review periodontal classification (516)

The combination of periodontal disease, periodontal classification, review, 2018 had the right number of articles that presented and covered the purpose of the study.

In the time interval of predefined years, there are about 41 published articles that meet the described criteria. From these 41 items, based on the keyword combination, 11 selected references in this item were detached. In these articles, it is clearly described either for the way of reorganization according to the new classification or for throwing ideas about the application of this reorganization [7], [27], [32], [39]-[43].

The discussion will be passed to one subgroup which will pass to the other subgroup. What was strongly demanded was the establishment of periodically healthy status as a subclassification, part of the new schematic classification. The combination of keywords also makes it possible to reduce and detach those items with the most important information on how the new classification of periodontal diseases can be applied.

III. RESULTS

The results of this study summarize the findings about the facts published that meet the inclusion criteria. The findings were made on the basis of dealing with the variety of periodontal diseases and the way in which these diseases are classified in the old order of periodontal disease classifications, compared to the new order of periodontal disease classification. These divisions and redistributions are carried out mainly respecting the clinical characteristics of the disease, followed by the logical course of aggravation of the disease in question.

Listed below are the results with expressed differences noticed between the two types of classification [5], [8], [12], [43]-[47]:

- The new classification includes the subdivision for inactive periods of the periodontium or the preliminary stages of reactivation of periodontitis not found in the old classification [5], [48]-[50].
- Bacterial plaque-induced gingivitis includes in the new classification only bacterial plaque, systemic or local risk factors for drug-induced gingivitis and hypertrophy [11], [17], [51]-[55]
- Gingivitis caused not by bacterial plaques: There are 2 separate subgroups: neoplasms and gingival pigmentation, which in the old classification were included gingival manifestations of systemic conditions.
- The new classification includes: genetic gingivitis, specific infections, inflammatory and immune condition, reactive processes (neoplasm, endocrine, nutritional and metabolic), traumatic lesions, gingival pigment. There is a lot of information compression in the field of inclusion [27]-[29], [56]-[59].
- In the old classification, periodontitis was divided mainly on basis of age when it presented as a disease. In the new classification, the group of ulcerative necrotizing periodontitis is separated. This is advantageous, as their treatment is more specific and very different from the routine treatment. The name necrotizing stomatitis is also introduced. This designation in the old classification was more due to the advancement of the localized necrotizing periodontium, and then appeared in some areas [3], [7], [18], [34], [60]-[62].
- The classification here is much more specific and more orienting to the working protocol of periodontal treatment, as patients are divided according to the severity of the disease: mild, moderate and aggravated,
according to the progression of the disease: rapid progression, moderate progression, slow progression of the disease.

- Periodontitis as a manifestation of systemic diseases: The new classification refers to the International Statistical Classification of Diseases and Related Health Problems [53], [54], [56], [62]-[66].

- The concept of aggressive periodontitis and chronic periodontitis has been anymore. The removal of these two concepts also excludes the classification according to the age of the patient as a basic element used in the classification of periodontitis.

- Necrotic Ulcerative Stomatitis is not a consequence of the aggravation of periodontitis and ulcerative gingivitis, but stands as a separate diagnosis in the new classification. The clinical signs remain the same, but the ranking in the new classification distinguishes it from other diseases, as a disease itself, and not as consequence of the aggravation or aggravation of the existing disease [12], [17], [67]-[69].

- Abscesses and endo-periodontal lesions are under a single classification not in two separate subgroups.

- Occlusion trauma has emerged as a separate subgroup detached from muco-gingival deformities during development or acquired. Muco-gingival deformities are well divided based on whether they are congenital or acquired.

- Mucogingival and developmental deformities in the new classification are much more specific with specific designations: gingival phenotype, gingival recession, absence of gingiva, reduced vestibular depth, lack of frenulum, gingival excess. These conditions were much more general in the old classification.

- The classification of trauma from occlusion to the new list is the classification of occlusive trauma by orthodontic forces. Trauma from occlusion in the new classification as a subgroup with itself gives the appropriate clinical importance to this pathology, making it possible to separate this pathology from dental prosthetics to what it really is, in periodontal pathology. The clinical signs of this periodontal disease are similar to the clinical signs caused by endodontic pathologies, but their importance at the occlusion level and the radiographic signs encountered by this pathology spread over time, are specific and categorically well distinguishable [23], [27], [36], [69].

- Prosthetic works affect the periodontal status of abutment teeth, both for fixed and movable prosthetics. The new classification divides clinical cases depending on factors related to teeth and prosthesis. This division is much more facilitative in classification diagnosis and treatment protocol. This subdivision is also related to the iatrogenic factors causing the formation of periodontal tart.

- The new classification includes the classification of periodontal disease around implants [9], [11], [36], [38]. This part is missing in the old classification. The inclusion of peri-implants in the classification is an important input of this classification in the conditions when the cases of implant treatment are increased and consequently the care of patients for oral hygiene is not at the right level [34], [37], [43], [64]-[66].

The new classification of periodontal diseases makes the dividing line between gingivitis and periodontitis more distinct, as a disease itself and systemic reasons that may cause changes in periodontal structures [67], [68].

A very desirable and understandable division is made between the consequences of the existing periodontium in the oral cavity and the appearance of systemic diseases, and vice versa between the existing systemic disease and the appearance of the periodontium in the oral cavity [1], [2], [6], [39]-[43]. This correlation expressed according to the new classification system of periodontal diseases makes the difference in the scope and application of the periodontist, internist and dentist specialized in oral medicine more obvious [67], [69]. The same thing happens with the term trauma from occlusion, as it is the new classification that further gives a previously thought but not expressed division of the scope and field of application of the periododontist and dental prosthetist.

Establishing a healthy status from a periodontal point of view gives you the right to reflect it on maintenance therapy, considering in the protocol of periodontal treatment also the presentation to the dentist according to the calendar of maintenance therapy.

There is a mentality problem among dentists who will apply this new classification order [47]-[50]. This type of periodontal disease classification facilitates the schematic presentation of different types of disease. The schematic presentation of the classification is much simpler than the old one. The only possible problem is to find the logic interconnection of the real clinical signs of existing periodontal disease with the new name or the new classification of the same periodontal disease.

IV. CONCLUSIONS

Clinical practice and the application of treatment protocols necessitate changes in the classification of periodontal disease.

The new classification has been necessary for clinical application, this is supported in the clinical work of many clinicians as it is precisely this experience that requires and needs the changes expressed in the new classification.

Implantation is the direction of periodontology that evolves into applicable materials, working methodology and treatment protocol.

It will take time for periodontological clinicians to assimilate the changed information and apply it in the diagnosis and cartelization of the patient and then closely link it with further treatment protocols according to the new format of periodontal disease classification.

Perhaps, no element has changed in the protocol of treatment of periodontal diseases, but the removal of one disease label and the placement of another label will take time to assimilate and then be applied to routine periodontal dental treatments.
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