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The Hidden Danger - Understanding Periodontal Pocket

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ABSTRACT

The conversion of junctional epithelium to pocket epithelium is regarded as a hallmark in the development of periodontitis. Knowledge of factors contributing to the initiation and progression of pocket formation is important. It may result in the development of better preventive measures and improved healing outcomes after therapeutic interventions. destruction of the structural integrity of the junctional epithelium, which includes disruption of cell-to-cell contacts and detachment from the tooth surface, consequently leading to pocket formation, disequilibrates this delicate defense system. One of the main variables used to evaluate the clinical success of periodontal therapy is periodontal pocket depth reduction. Therefore, reflecting on how the periodontal pocket process's role has helped guide how we perform and interpret periodontal research is important. A full understanding of periodontal pocket etiology is required to design a more precise treatment strategy and avoid the advancement of periodontal disorders.

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Introduction

The periodontal pocket is defined as a pathologically deepened gingival sulcus around a tooth at the gingival margin. Accordingly, the space between the pathologically detached gingiva and the tooth is called a pocket. A gingival sulcus depth of up to 0.5 mm may be considered clinically healthy. Different classification types of pockets exist [1,2]. To establish better preventive measures and therapeutic outcomes for the treatment of this disease process, this article gives a comprehensive insight into the development and advancement of periodontal pockets.

Classification

Periodontal pocket classified into true gingival pockets and periodontal pockets the former being related to gingivitis, whereas the latter is found in periodontitis. Detachment of junctional epithelial cells and inflammation is part of both gingival and periodontal pockets. In a gingival pocket there is, in contrast to a pseudo pocket, inflammation and destruction of the underlying periodontal tissues together with coronal detachment of junctional epithelial cells but without bone destruction. In a periodontal pocket, bone destruction by osteoclastic resorption is a characteristic feature beyond inflammation, tissue destruction and detachment of junctional epithelium [2,3]. Two types of periodontal pockets exist: suprabony pockets; and intrabony pockets. In a suprabony (supracrestal or supra-alveolar) pocket, the bottom of the pocket is coronal to the alveolar crest. When the bottom of the pocket is apical to the alveolar crest, which means that bone is present lateral to the pocket wall, the pocket is called an intrabony (infrabony, subcrestal or intra-alveolar) pocket [2-4]. Another type of classification takes into consideration the pocket morphology according to the number of surfaces involved. A simple pocket involves one tooth surface only, a compound pocket involves more than one surface and a complex (or spiral) pocket means that the base of the pocket is not in direct communication with the gingival margin. Finally, depending on the disease activity, there are active and inactive pockets.

Pathogenesis

The conversion of junctional epithelium to pocket epithelium is regarded as a hallmark in the development of periodontitis, the potential factors contributing to the initiation of pocket formation need to be critically analyzed. Microorganisms are the primary etiologic cause of periodontal disease and there is good evidence that pocket formation is related to bacterial colonization of the subgingival tooth surface. Nevertheless, there is a lack of experiments evaluating the mechanisms of pocket formation. Previous discussions on the initiation of pocket development centered around whether: (i) the epithelial cells first recede and later, as a consequence of this, biofilm can migrate apically; or (ii) bacterial products force the epithelial cells to migrate apically. Degenerative changes, such as loss of cellular continuity and detachment from the tooth, are first observed in the coronal-most portion of the junctional epithelium (i.e. at the sulcus bottom) [5-7]. Several possibilities have been proposed to explain intra-epithelial cleavage in the junctional epithelium. With increasing degree of inflammation, an increase in both migration of polymorphonuclear neutrophils and passage of gingival crevicular

fluid through the intercellular spaces occurs [8]. A moderate distension of intercellular spaces is not considered to compromise the structural and functional integrity of the junctional epithelium [9]. An increased number of leukocytes is, however, considered as a contributing factor that eventually leads to focal disintegration of the junctional epithelium. This is in line with the concept that the host itself is the driving force behind decomposition of the junctional epithelium [9]. The cysteine proteinases, referred to as gingipains (namely virulence factors produced by Porphyromonas gingivalis, a species of bacterium implicated as a major etiological agent of chronic periodontitis), have been the focus of intense research [10-12]. As a result, a new effect of gingipains was discovered. Gingipains specifically proteolytically degrade components of cell-to-cell junctional complexes in epithelial cells [13,14]. In addition, gingipains also cleave intercellular adhesion molecule-1 on oral epithelial cells, which consequently leads to disruption of the interaction between polymorphonuclear neutrophils and epithelial cells, a sort of immune evasion by P. gingivalis. The mechanism of action is explained in Figure 1 And Figure 2. Periodontitis has also been associated with longitudinal HbA1c increases in people who do not have diabetes, suggesting that periodontal inflammation may influence the risk of developing diabetes [15].

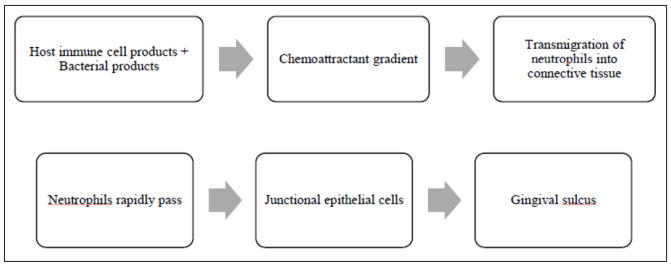

Figure 1: Initial lesion

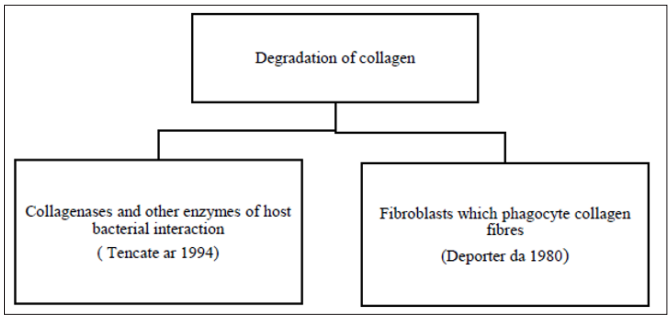

Figure 2: Degradation of Collagen

Histopathology

Histopathologically, a pocket is 'a pathologically altered gingival sulcus, lined to a variable extent with pocket epithelium. Furthermore, the pocket epithelium, which lines the pocket wall facing periodontal tissues, is defined as 'unattached epithelial lining of the pocket, which extends from the sulcular epithelium to the junctional epithelium. It is characterized by marked proliferation of retial ridges around inflamed connective tissue papillae and by a tendency to micro-ulceration [16]. the junctional epithelium and pocket epithelium have some features in common, such as formation of a barrier against microorganisms and their products, passage of gingival fluid and leukocytes (in particular neutrophilic granulocytes) and concomitant infiltration with mononuclear leukocytes [17]. On closer inspection, however, the pocket situation demonstrates characteristic features distinctly different from the healthy conditions in a gingival sulcus environment

Clinical Feature of Periodontal Pocket

On probing - gingival hemorrhage and suppuration, in deep chronic pockets - tooth movement and diastema development, bluish red discoloration of pocket wall extending from gingival margin to alveolar mucosa, flaccid with smooth shiny surface, pain in periodontal pocket on examination with probe and the patient may also report with sensitivity towards cold and hot, and an emergence of deep dull pain which could be localized or deep within the alveolar bone [18].

Content of the Periodontal Pocket

Debris, microorganisms and their products (enzymes, endotoxins, and other metabolic products), gingival fluid, food remnants, salivary mucin, desquamated epithelial cells, leukocytes, plaque covered calculus, purulent exudate [19].

Microtopograpgy of Soft Tissue Wall of the Pocket

Areas of relative quiescence - A relatively flat surface with tiny depressions and mounds, as well as intermittent cell shedding. Areas of bacterial accumulation - depressions on the epithelium surface with a lot of debris and bacterial clumps entering the expanded intercellular gaps. Cocci, rods, filaments, and a few spirochetes are among the organisms found. Areas of leukocyte-bacteria interaction - A large number of leukocytes are present, and they are covered in bacteria in what appears to be a phagocytosis process. Areas of intense epithelial desquamation - Semi-attached and folded epithelial squames, which are occasionally partially covered with microorganisms. Areas of emergence of leukocytes - Leukocytes emerge from the pocket wall through perforations in the intercellular gaps. Areas of ulceration with connective tissue visible. Areas of hemorrhage with a large number of erythrocytes [20].

Diagnosis of Pocket

Careful probing of the gingival border along each tooth surface is the only consistent means of locating and determining the extent of the periodontal pocket. Marquis color-coded probe, UNC-15 probe, Michigan 'O' probes, Plastic probes, World Health Organization (WHO) probes, and William's probe are some of the probes used to locate the periodontal pocket. Pockets are normally not seen on radiographs; however, they can be spotted using gutta percha points or calibrated silver points in conjunction with radiography [21]. The distance from the free gingival margin (FGM) to the bottom of the gingival/ periodontal pocket is termed pocket probing depth (PPD). Like clinical attachment level (CAL), PPD is assessed to the nearest millimeter on all tooth surfaces by walking the probe by means of a standardized and graduated periodontal probe [22].

Treatment Plan

To eliminate pocket lining, pocket wall, and pocket reduction by tissue shrinkage, to achieve a stable and maintainable state, and to stimulate periodontal regeneration [23]. residual PD ≥5 mm, especially when associated with persisting BoP, was claimed as a site-specific, positive predictive factor for further clinical attachment loss during supportive periodontal therapy (SPT) Chapple et al [24]. Non-surgical periodontal therapy (NST) has been suggested as the ideal initial treatment for patients suffering from periodontitis Lindhe et al and stated a critical probing depth of 4.2 described where we can proceed with surgical treatment below the critical probing depth loss of attachment will happen and above the critical probing depth gain in clinical attachment will occur [25].

Non-surgical therapy

NST consists of subgingival debridement, subgingival scaling, and root planning [26]. Theoretically, these are well-differentiated procedures. However, they are performed clinically at the same time, being named "scaling and root planning" (SRP), which is considered the cornerstone of cause-related therapy Graziani et al., [27]. A consistent amount of evidence has indicated that SRP is effective in controlling inflammation, reducing PD, and improving CAL(Van der Weijden & Timmerman) [28]. However, SRP is technically demanding, and complete calculus removal is difficult to achieve [29]. Residual PDs must be recorded at the end of NST and through SPT in order to tailor the treatment plan on specific patient-centered needs. Further treatment may include periodontal surgeries, aiming at PD reduction, or personalized SPT, as shorter recall intervals have been associated with increased periodontal stability along time even in the presence of PD ≥5 mm [30].

Surgical therapy

Accessibility for proper SRP, plaque control, pocket reduction, and new attachment [27]. Periodontal surgery is the indicated management option for residual deep pockets after non-surgical therapy for teeth with a prognosis other than "hopeless" and in cases where periodontal therapy prognosis is likely [31-32]. The indication for specific procedures varies with pocket type, as described in Figure 3 [33].

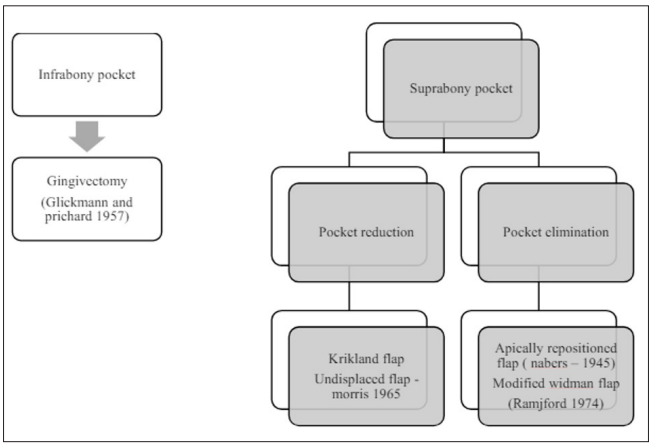

Figure 3: Treatment Strategy for Surgical Periodontal Therapy

Gingivectomy

Residual pocketing with excessively thick, fibrous gingival tissue but no underlying bone defect

Wedge Procedure

- Residual pocketing at a distal or mesial tooth surface that faces excessively thick gingival tissue
- Typically, at the most distal tooth of the arch, adjacent to thick tissue of the maxillary tuberosity or the retromolar pad

Gingival Flap Procedure

- As monotherapy for supra bony pockets: residual pocketing without an underlying bone defect, surgery accesses root surfaces for calculus and dental biofilm removal
- As monotherapy for infra bony pockets associated with shallow bone defects in the anterior maxilla
- As part of regenerative therapy to provide surgical access to bone defects

Osseous Surgery

- As monotherapy for infra bony pockets associated with shallow bone defects or irregular bone contours
- Commonly as part of regenerative therapy to provide surgical access to bone defects and improve bone defect morphology

Bone Grafting, Guided Tissue Regeneration, Biologics or Combinations of these

Infrabony pockets with deep bone defects; bone defects should be favorable to regeneration

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