Commentary

# Calculus

## A risk factor for failed periodontal therapy

Stephen K. Harrel, DDS; Charles M. Cobb, DDS, PhD

At present, there is little question that periodontitis results from the interaction between a dysbiotic oral biofilm and the host immune system.[1] This has not always been the case. Before the classic study by Löe and colleagues[2] on the cause of gingival inflammation, there was widespread belief that calculus played a central role in periodontitis.[3] The plaque theory was proposed as the primary etiology for periodontitis, with calculus being relegated to a secondary etiologic factor.

Thus, calculus is commonly viewed as a rough-surfaced mineralized plaque that promotes biofilm retention and makes oral hygiene more difficult.

However, calculus continues to be an issue in the treatment of periodontitis. When a patient responds favorably to periodontal treatment but has persistent isolated areas of inflammation, the problem is frequently residual calculus.[4] Calculus in areas of persistent inflammation may be microscopic, visible only at high magnification or by laser fluorescence. These small residual areas of calculus have been referred to as microislands of calculus or fractured calculus.[5]

Despite the prevailing theory that periodontitis is initiated by a dysbiotic biofilm, there are studies suggesting that calculus may play an independent role in the initiation, progression, and associated cellular dysfunction of periodontitis. In this commentary, we offer a perspective on calculus as an etiologic factor in periodontitis and present relevant research supporting a role for calculus in the initiation, response to therapy, and progression of periodontitis.

### PLAQUE THEORY

In his 1746 book *The Surgeon Dentist*, Fauchard[6] associated periodontitis with soft and hard material on the teeth (accretions). It was not until the 1965 article by Löe and colleagues[2] that the central role of biofilm in the inflammation of periodontal disease was accepted.

Löe and colleagues[2] showed that gingival inflammation commenced within 14 days after cessation of oral hygiene and occurred in the absence of calculus. Furthermore, removal of the biofilm by oral hygiene was adequate to eliminate the gingival inflammation. Waerhaug[7] advanced the role of biofilm by showing a direct correlation between the advancing plaque front and destruction of the periodontal supporting structures. Thus, the question arises: Is the only role for calculus in periodontitis that of an environmental risk factor that harbors biofilm or does calculus play another role in periodontal destruction?

### INDEPENDENT PATHOLOGIC ROLE OF CALCULUS

Studies suggest that calculus may play a role in periodontitis beyond that of biofilm retention. Using an animal model, Allen and Kerr[8] looked at the role of sterile calculus in tissue inflammation and reported that over time there was an inflammatory reaction that resulted in granulation tissue like that seen in human periodontitis. This raised the question: Could calculus cause periodontal inflammation in the absence of a viable bacterial biofilm? Obviously, investigating this question in a human model would be difficult as deliberately leaving calculus in a periodontal pocket after treatment would be unethical.

Wilson and colleagues[4] evaluated the role of calculus in human periodontitis, using endoscopy to evaluate inflammatory patterns on the pocket wall in patients with severe periodontitis. The authors found that the diseased pocket wall was not uniformly inflamed but exhibited isolated islands of inflammation. Rotation of the endoscope to view the adjacent root surface determined that 70% of the inflamed area was associated with calculus covered by biofilm. The remaining 30% of the inflamed area was associated with biofilm only. This study showed that most of the periodontal pocket inflammation was associated with calculus, thereby validating the importance of calculus in the pathophysiology of human periodontitis.

Studies support the importance of calculus as an independent risk factor for periodontal disease.[9-11] Ziauddin and colleagues[12] determined that cultures of epithelial cells will phagocytize sterile calculus and subsequently undergo cell death. Subsequently, Ziauddin and colleagues[11] showed the cytotoxic effects of sterile calculus, using an in vitro model of HSC-2 oral epithelial cells and THP-1 macrophages. The authors described the pathway of cell death as pyroptosis, a form of lytic programmed cell death associated with inflammation. The mechanism of pyroptosis starts with epithelial cells phagocytizing calculus crystals in a manner similar to cellular phagocytosis and destruction of bacteria. However, when crystalline particles of calculus are brought into the epithelial cell, the result is death of the cell. It is possible that a similar pathway may function in humans, with the end result being damage to the epithelial barrier of the periodontal pocket.

These studies indicate that calculus plays a role beyond that of plaque retention. If true, it helps explain why inflammation in the diseased pocket wall is often associated with calculus and why residual calculus is found at sites of inflammation after scaling and root planing. Collectively, these studies emphasize the importance of calculus in periodontitis and the need to completely remove all calculus during treatment.

## CLINICAL IMPLICATIONS

The plaque theory and the role of biofilm in the inflammation associated with the early stages of periodontitis remain unchallenged. There is also no question that calculus plays a role in periodontal disease by plaque retention and thus is a complicating factor in treatment and periodontal maintenance. The question is whether calculus has a more defined role in the progression of periodontitis and whether residual calculus contributes to a poor healing response.

In aggregate, the in vitro[11,12] animal,[8] and human studies[4] show that calculus can be considered an independent risk factor for periodontal inflammation. On the basis of the findings of these studies, the removal of calculus needs to remain a high priority in periodontal therapy.

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The need for calculus removal in periodontal treatment has been de-emphasized. Indeed, some have advocated that it is unnecessary to totally remove calculus, and it is only necessary to decontaminate the pocket.[13] Reducing bacterial loads in the periodontal pocket by decontamination is undoubtedly beneficial in the short term. However, multiple studies have shown that the pocket microbial flora quickly rebounds to pretreatment levels.[14] Furthermore, clinical experience has shown that in areas of persistent inflammation and progressive disease, residual calculus is a frequent finding. The inadequate removal of calculus, even leaving subgingival microislands and fractured remnants of calculus, represents a risk for failed periodontal therapy.[15]

## CONCLUSION

The removal of calculus has historically been an integral part of periodontal therapy. Although the role of biofilm as the initiating factor in the pathogenesis of periodontal disease has been conclusively proven, the role of calculus as a complicating factor remains. The removal of calculus is still a central goal of periodontal therapy, but calculus removal is often de-emphasized at the clinical level. This de-emphasis is often a factor in the failure of periodontal therapy. Removal of calculus during periodontal treatment should be emphasized, and further studies are needed to define the role of calculus in periodontal destruction. ■

https://doi.org/10.1016/j.adaj.2023.08.001

Dr. Harrel is an adjunct professor, Department of Periodontics, School of Dentistry, Texas A&M University, Dallas, TX. Address correspondence to Dr. Harrel, Department of Periodontics, Texas A&M College of Dentistry, 3302 Gaston Ave, Dallas, TX 75246, email skharrel@gmail.com.

Dr. Cobb is a professor emeritus, Department of Periodontics, School of Dentistry, University of Missouri-Kansas City, Kansas City, MO.

**Disclosures.** Drs. Harrel and Cobb did not report any disclosures.

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