



A Clinical Review on Dental Calculus and it’s Role in Periodontal Disease Progression

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Abstract

Periodontal diseases stand next to Dental caries among the most prevalent diseases. Individuals often present with bleeding from gums and yellow to black discoloration. This discoloration is often referred to as dental calculus. Dental calculus is not age specific and is present in both males and females. It also has a close association with dental plaque. The main constituent is calcium phosphate with variable compositions in the two sub variants. Its variant subgingival calculus has proven to effect both clinical attachment level and pocket depth. Management includes scaling and Root planing along with professional recalls. Here a short clinical review is presented on the importance and appearance of dental calculus.

Keywords: Calculus; Plaque; Calcium phosphate; Root planing; Pocket depth

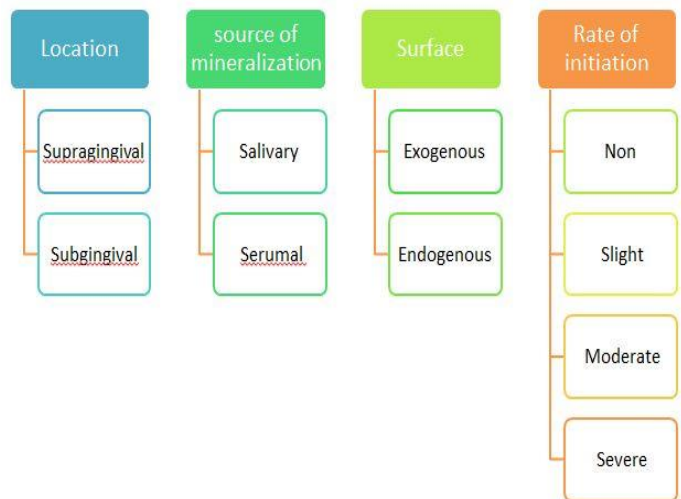
Introduction

Periodontal diseases are characterized by loss of epithelial attachment and bone loss. Individuals often present with yellowish and brownish discoloration which is commonly called as calculus. Calculus can be defined as a hard deposit that is formed by mineralization of dental plaque on the surfaces of natural teeth and dental prosthesis which are usually covered by a layer of unmineralized plaque [1]. Dental calculus is a chief contributing factor in the development of periodontal diseases [1]. The prerequisite for calculus is dental plaque. Calculus is a big problem because it serves many purposes like accumulation of bacterial toxins and prevention of adequate elimination (as it contains lot of surface roughness). For the patient, it becomes difficult to maintain oral hygiene [2]. A series of factors are related to calculus formation. Among them the prominent factors are bacterial plaque retention, biochemical factors (characterized by saliva or crevicular fluid), microorganisms and dietary factors [3-6]. It is often observed that dental calculus formation is not uniform among various groups of population and its deposition varies widely from individual to individual. Some subjects, despite maintaining good plaque control have an inherent tendency to accumulate calculus in an extraordinary way. It simply means that even frequent visits to the dentist could not

help the individuals to maintain oral hygiene. Hence this clinical view has aimed to signify the importance of calculus in progression of periodontal diseases.

Flowchart 1: Classification (Source et -Aghanashini et al, (2016)).

Classification of Dental calculus (Tannenbaum P et al and Melz)



Microbiology of Dental Calculus

Microbes are responsible for any type of infection in the body. Oral cavity is sterile at birth (Carranza 10th edition) [7,8]. Dental plaque also harbors a lot of bacteria (Anerud, 1991) [9,10]. Among them, the major are red complex bacteria namely *Porphyromonas gingivalis*, *Treponema palladium* and *Tannerella Forsythus* (Hafazee and Socransky, 1994) [11]. They are clinically significant because they are present at sites where there is a lot of bleeding. Some enzymes like Lactate dehydrogenase and alkaline or acid phosphatase have been identified in dental plaque and are in association and development of the plaque [1]. Bacteria are not essential for calculus formation, but they are responsible for its development. Hence, high amount of calculus indicates that oral hygiene has been poor for months or even years [6]. Even calculus was observed in germ free animals also (Gustaffson & Norman, 1962) [12].

Flowchart 2: Composition of calculus.

Composition of calculus (Carranza 10th edition)

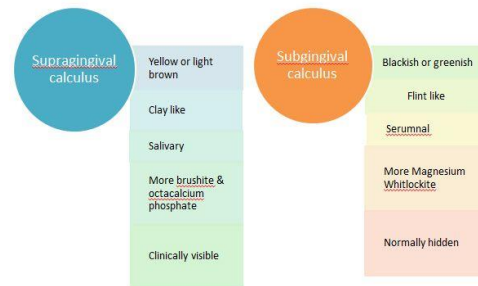


Figure 1 and 2: Clinical difference between supra gingival calculus and subgingival calculus.



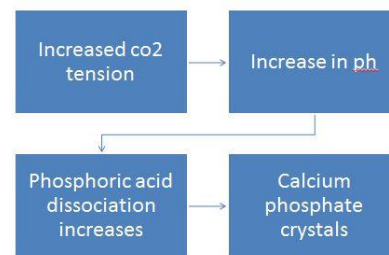
Flowchart 3: Differences between supra gingival vs subgingival calculus.

Types of calculus (Sahithya, RS. Essentials of periodontology. 1st edition).

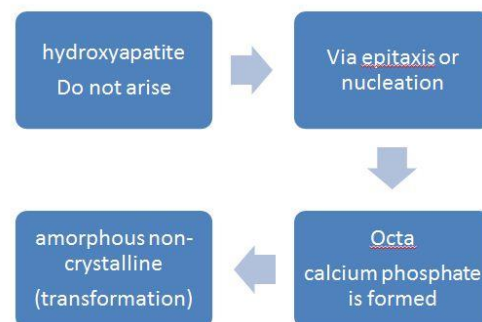


Flowcharts 5-9: Theories of calculus formation.

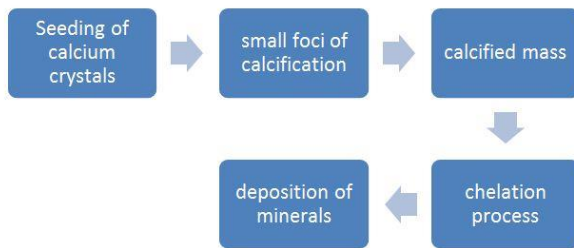
Booster mechanism (Aghanashini et al)



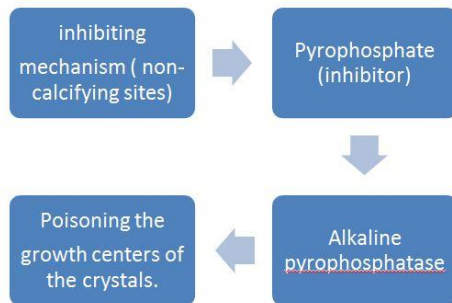
Epitactic mechanism (Aghanashini et al)



Inhibition theory (Aghanashini et al)



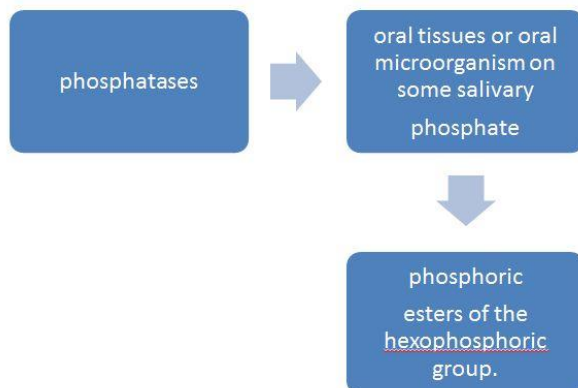
Transformation theory (Aghanashini et al)



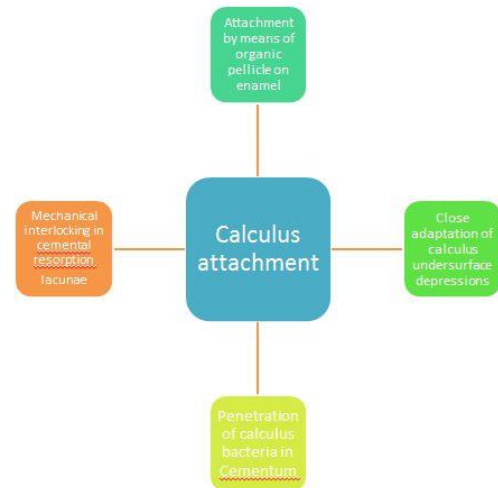
Bacteriological theory (Aghanashini et al)



Enzymatic theory (Aghanashini et al)



Calculus attachment (Carranza 10th edition)



Discussion

It is a really an uphill task to determine the effects of calculus and plaque on gingiva since calculus is always having with plaque on dentition. There is always a correlation between the presence of calculus and prevalence of gingivitis. This association may lead to proliferation of periodontal diseases. The rough calculus was found to initiate inflammation in the adjacent periodontal tissues, and it acts as an ideal substrate for subgingival microbial colonization (thereby acting as a niche harboring bacterial plaque) [13]. It is also acting as an irritant to the periodontal tissues, distends the periodontal pocket wall and is responsible for inhibition of polymorph nuclear leukocytes. So, despite the primary or secondary relationship in pocket formation (refers to periodontal disease progression and continuous irritation), calculus is a significant pathogenic factor in periodontal disease [6].

But various studies have emphasized that calculus do have a major role in progression of calculus. Ainamo (1970) found a high positive correlation between calculus (both supra- and subgingival and gingivitis) in 154 army individuals of age groups (19 and 22) based on retention index (RI) [13,14]. They observed that there was some association between the microflora of gingivitis and calculus (different from caries microflora). Ainamo (1970) also found that there was more prominence of gingivitis as well as calculus deposits on oral than on facial surfaces of premolars and molars [13]. It can be explained based on area where the salivary secretions (supragingival calculus was in higher concentration) are greater, thereby showing the pathogenicity of calculus along with plaque as detrimental when compared to that of plaque alone. Alexander (1971) also observed the same findings [15]. Buckley (1980) examined the prevalence of subgingival and supragingival calculus among 300 individuals (teenagers of age group 15 -17 years). But his findings were

contrary as compared to other individuals [16]. He found greater prevalence of subgingival calculus when compared to supragingival calculus. However, distribution pattern was same. Furthermore, Lennon and Clerehugh (1984) explored (in 2-year longitudinal study) the role of sub-gingival calculus in periodontal disease in teenagers [17]. They concluded that the presence of subgingival calculus was the best predictor of future attachment loss. Axelsson and Lindhe (1981) conducted a 6-year longitudinal study to determine the prevention of caries and periodontal disease by oral hygiene maintenance and repeated prophylaxis [18]. The study concluded that subjects who utilized proper oral hygiene techniques had very less signs of gingivitis and periodontal tissue attachment loss. Even caries was also absent. Similar strategies of frequent recalls (characteristic of all the adult plaque control) were done by Goteborg [1]. All these studies highlighted that the plaque control and professional oral prophylaxis had certainly played an important role in maintaining the gingival and periodontal health. Tagge et al (1975) did a comparative study by assessing both clinically and microscopically, the soft tissue response in suprabony periodontal pockets after treatment by root planning and oral hygiene and oral hygiene measures alone [19]. It was observed that the therapies decreased the incidence and severity of gingivitis along with pocket depth. However, root planning combined with oral hygiene measures resulted in a statistically significant improvement when compared to personal oral hygiene measures alone. The reason could be attributed to the lack of tooth brushing effectiveness on the non-root planed teeth (having subgingival deposits) on the non-root than in those treated by root planning with oral hygiene prophylaxis. Morrison et al (1980) examined the effects of initial and non-surgical periodontal treatment on the periodontitis and its severity [20]. The results showed that there was a significant reduction in inflammation after removal of the plaque and calculus deposits. Also, it was observed in their findings that changes in plaque scores could not be correlated with attachment level gain and pocket depth reduction, but removal of subgingival calculus is the key factor for the results.

Conclusion

Calculus is an important factor for periodontitis. Among the two components, subgingival calculus is proven to effect the clinical attachment level and pocket depth. Removal of calculus is must for adequate gingival health. Hence, every clinician should focus on removal of calculus timely and individuals should maintain oral hygiene with priority.

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