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Research Article

Dental Calculus – Mere Ash Heap or Still A Mystery: A Review of Literature on Etiologic Significance of Dental Calculus

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Abstract

Chronic inflammatory periodontal diseases i.e., gingivitis and periodontitis are one of the most common afflictions faced by the human beings. They are considered to be infectious in nature having polymicrobial etiology. Dental plaque, which is a pool of these disease causing microorganisms has been the focus of researchers for the long time, and is considered to be the primary etiologic agent in disease etiopathogenesis. However, dental calculus which is a mineralized product of this plaque remains ignored and is considered merely as an ash heap of minor significance in etiopathogenesis of disease. Even though, there literature evidences of presence of intact and viable bacteria within the non- mineralized channels and islands in supragingival calculus from various studies. However, viability of bacteria in dental calculus and their role in disease pathogenesis still remained obscure. Therefore, this review intends to collate all the available literature and present the studies related to etiologic significance of dental calculus and investigation of the viability of bacteria in dental calculus along with their identification at one place and provide a way forward towards better understanding on etiopathogenesis and treatment of periodontal diseases.

Keywords: Dental Plaque; Dental Calculus; Etiopathogenesis; Gingivitis; Periodontitis Periodontal Diseases; Viable Bacteria

Abbreviations

SEM: Scanning Electron Microscopy

Introduction

Bacterial plaque that coats the teeth is considered to be the main etiologic factor in the development of periodontal diseases. Calculus however, is also considered to play an important role in maintaining and accentuating periodontal diseases by keeping plaque in close contact with the gingival tissues and creating the areas where its removal is difficult [1-4].

Although, a positive correlation between the presence of calculus and the prevalence of gingivitis exists [2,3] but this correlation is not as great as that between plaque and gingivitis [1-4]. In young persons, periodontal conditions are more closely related to plaque accumulation than to calculus, but the situation is reversed with age [1-4]. The incidence of calculus, gingivitis, and periodontal disease increases with age. It is extremely rare to find periodontal pockets in adults without supragingival calculus, although subgingival calculus may be of microscopic proportion in some cases [1-4].

Ironically, it is quite difficult to distinguish between the effects of calculus and plaque on gingiva as mineralized calculus is always covered with a nonmineralized layer of plaque [1,3,5-7]. Many investigators hitherto have attempted to distinguish between their effects however seem to be less satisfied with the currently available knowledge about etiologic significance of dental calculus [1-4]. Knowledge gaps about the topic therefore, remain a matter of concern for most of the academicians and researchers in the branch of periodontics.

There were certain indications of presence of intact and viable bacteria within the non- mineralized channels and islands in supragingival calculus from various studies and reported successful bacterial cultures in their microbiological studies on calculus [8-10]. However, viability of bacteria in dental calculus and their role in disease pathogenesis still remained obscure. Thus, the present review was undertaken to understand the available literature and present the studies related to etiologic significance of dental calculus and investigation of the viability of bacteria in dental calculus along with their identification.

Materials and Methods

For this review, articles in English language were identified by searches on electronic data bases such as Google, Google scholar, Ebscohost, and Pubmed from 1960 through July 2020. The following search terms were used: "Dental Calculus", "Dental Plaque", "Viability", "Viable", "bacteria", "Mineralized Plaque", "un-mineralized plaque and related terminologies".

Results and Discussion

Epidemiologic Studies regarding etiologic significance of dental calculus

Skougard MR., *et al*. (1969), reported on an epidemiological survey conducted in Uganda on 1394 persons aged 2-40 using simplified oral hygiene index (scoring debris and calculus separately) and Russell's periodontal index (PI) [2]. The authors pointed out that the Calculus index – Periodontal Index correlation did not necessarily imply a direct casual relationship since calculus as well as periodontal disease could be the result of the cumulative effect of bacterial plaque. Calculus could also be the result of the increased Periodontal Index and the accompanying increase in inflammatory exudates.

Ainamo J (1970) [2], examined 154 army recruits between the ages of 19 and 22 and found a high positive correlation between calculus (both supra and subgingival) and gingivitis. He found a higher correlation between gingivitis and calculus related plaque than with cariogenic plaque. There was more gingivitis and more calculus deposits on lingual than on facial surfaces of lower second pre-molars and first and second molars. He suggested that the calculus could be the result rather than the cause since the inflammatory exudate could contribute to the mineralization of the plaque. He stated that the pathogenicity of calculus along with overlying plaque may be greater than that of plaque alone.

Alexander AG (1971), examined the distribution pattern of supra and subgingival calculus, bacterial plaque and gingival inflammation in 200 dental students and 200 dental clinic patients [2]. He found that the papillae exhibited the highest prevalence of gingival inflammation and the buccal margins the lowest. This pattern coincided with the highest prevalence of subgingival calculus on the interproximal surface and the lowest on the buccal. However, it was also noted that the number of individual papillae and margins that were inflamed was substantially higher than the number of surfaces with subgingival calculus. In general there was a much closer match in distribution patterns for gingival index and plaque than for gingival index and subgingival calculus.

Buckley LA (1980), examined 300 teenagers, aged 15-17, evenly distributed by age and sex. He found that subgingival calculus was more prevalent than supragingival [2]. There was a strong correlation between the buccal and lingual gingival indices and their respective plaque and supra and subgingival calculus indices, Pearson correlation analysis indicated a higher degree of correlation for gingival indices versus plaque than for gingival indices versus calculus. The correlation was somewhat higher for subgingival calculus than for supragingival calculus.

Douglas CW., *et al*. (1983), provided an interesting insight into the relationship of calculus to periodontal health and disease by examination of trends in the United States in the prevalence and severity of the periodontal diseases [11]. They compared data on 6,675 people who received an oral examination in 1960-1962 as part of a national Health Examination Survey with the results of a Health and Nutrition Examination Survey in 1971-1974. In this

latter survey 20,218 people received an oral examination. In 1960-1962 it was found that 26,1% of the subjects were free of gingival disease; by 1971-1974 this number had increased to 51,4%, This improvement was associated with significant change in the oral hygiene index. When the debris and calculus components were separated, it was found that the debris score had improved significantly, not the calculus score.

Although, numerous epidemiological studies regarding the etiological significance of dental calculus were done; still its etiological significance could not be established because these studies employed indices attempting to correlate mean values for deposits and disease, while in fact the nature of periodontal disease is site specific [1]. Furthermore, the studies did not include plaque adherent to dental calculus in the evaluation [1].

Clinical studies regarding etiologic significance of dental calculus

The clinical data relating to calculus and periodontal disease are derived from either the studies of mechanical methods for removal of deposits, or from studies on the impact on gingivitis of chemical control using anticalculus agents.

Studies using mechanical methods for removal of deposits

The goal of mechanical debridement is to remove calculus and plaque (both tightly and loosely adherent) and toxic agents in the deposits and underlying cementum. But the question which remained unanswered was the pathogenecity of dental calculus. Several studies have addressed such questions.

Suomi JD., *et al*. (1971), examined the effect of oral hygiene instruction and frequent oral prophylaxis (3-4 times per year) over a 3-year period in groups of adults [1]. He noted a three and a half fold difference in rate of attachment loss. The enhanced oral hygiene status in the treatment group included a very significant impact on calculus deposition. During the course of the 3-year study, the treatment group (who had received oral hygiene instruction and frequent prophylaxes) exhibited an increase in supragingival calculus over base line that was half that of the control group (an initial prophylaxis followed by customary care). The difference in subgingival calculus was even more profound; the increase in subgingival calculus in the treatment group was only one quarter of that in the control subjects.

Lightner LM., *et al*. (1971), in their study of preventive periodontic procedures found that with preventive office visits at three month intervals there was no difference in gingival inflammation among subjects who had received plaque control instructions versus non instructed subjects [1]. Careful preventive periodontal treatments, repeated at three month intervals, appeared to be in themselves effective for improving gingival health.

Tagge DL., *et al*. (1975), evaluated both clinically and microscopically, the soft tissue response (in 22 patients) of periodontal pockets two months after treatment by root planing and oral hygiene versus personal oral hygiene alone [12]. Each of the therapies reduced pocket depth and the incidence and severity of gingivitis. Root planing accompanied by oral hygiene measures, however, resulted in a statistically greater improvement than did oral hygiene alone. The toothbrushing was limited in its effectiveness by the presence of subgingival deposits on the non-root-planed surfaces; and as a result there was no significant change in attachment level and less pocket reduction than in those pockets treated by root planing and oral hygiene.

Chawla TN., *et al*. (1975), examined the effect of different dental prophylaxis regimens on periodontal disease in 1600 subjects aged 12-26 over a 2-year period [13]. They found that scaling along with instructed oral hygiene at 6-month intervals provided the maximum benefit. They concluded that the removal of calculus was directly correlated with the improvement in periodontal health. The removal of bacterial plaque alone, however, did not indicate such a correlation.

Hughes TP., *et al*. (1978) studied 15 patients aged 23- 77 for a biometric evaluation of the gingival changes following scaling, root planing and oral hygiene, Subjects were scored at base line, prior to instruction in brushing and flossing, one week and then one month after scaling and root planning [1]. They found that the degree of plaque control had minimal influence on changes in gingival position and attachment levels, at least up to the one month period of observation. Instrumentation of the root surface appeared to be the primary cause of positional gingival changes.

Knowles JW., *et al*. (1979) compared the various modalities of periodontal therapy. Although in some situations surgical procedures offer some advantages, including accessibility to the depos-

its, these studies established the centrality of removal of the subgingival tooth deposits in the treatment of periodontal disease [1].

Hellden LB., *et al*. (1979) studied 12 patients with advanced periodontal disease. The study design provided a comparison of four different treatment modalities following detailed oral hygiene instruction: (1) no treatment, (2) scaling and root planing alone, (3) administration of tetracycline alone, and (4) scaling and root planing combined with the administration of tetracycline [14]. There was a significant decrease in plaque index, gingival index and gingival probing depths in all groups, attesting to the value of the oral hygiene instruction, a significantly greater decrease in probing depth, however, was found in those areas which had received scaling and root planing. The authors suggested that the most likely explanation for this difference is the elimination of plaque-retaining factors including calculus and restoration overhangs in the scaled quadrants.

Morrison EC., *et al*. (1980) examined the effects of initial, non-surgical periodontal treatment (the hygienic phase) on the clinical severity of periodontitis in pockets varying from l->7 mm. They found that the removal of the plaque and calculus deposits resulted in significant reduction in inflammation [15]. Improvement was great enough to call for re-assessment of the need for surgery in some instances. In evaluating the various factors involved in the hygienic phase it was noted that changes in plaque scores could not be correlated with gain in attachment level and reduction in pocket depth.

Axelsson P., *et al*. (1981) did 6-year studying which a dental prophylaxis was given every 2 months during the first 2 years, and every 3 months thereafter. Each of these recall visits included a complete scaling and root planing, combined with oral hygiene instructions to improve the patient's self-performed plaque control program [1]. By the study they established the value of plaque control in preventing caries and maintaining periodontal health.

Philstrom B L., *et al*. (1981) compared the effectiveness of scaling and root planing alone to scaling and root planing followed by periodontal surgery [16]. They noted that the level of plaque control in their patients was disappointing. In spite of this poor performance they noted that pocket depth was reduced, attachment level maintained (or increased) and inflammation reduced over the 4 years of observation. It seemed that the prophylaxis procedure every 3 months was able to help insure the success of the therapy even in the face of the relatively poor control of supragingival deposits. They attributed much of this positive result to the periodic removal of calculus which helped prevent a re-establishment of a pathogenic microflora in the subgingival area.

Ramfjord SP., *et al*. (1982) assessed the effect of individual variations in plaque control on pocket depth and attachment levels in patients they had been following in their long term longitudinal study [17]. This group had been on a 3-month recall schedule for prophylaxis over an 8-year period. The investigators found that with professional tooth cleaning every 3 months, the level of plaque oral hygiene (as measured by plaque scores) was not critical for maintenance of post treatment pocket depth and attachment levels. Patients with good oral hygiene evidenced greater improvement in periodontal status during the initial post treatment period, but these differences were no longer significant after 3 to 4 years of maintenance care.

Morrison EC., *et al*. (1982) noted that severity of recurrent gingivitis during maintenance therapy with a 3-month recall system did not appear to have any significant impact on recurrence of pocket depth or maintenance of clinical attachment level [1]. Apparently the personal oral hygiene is more important in the control of gingivitis than in the progression of periodontitis, provided a 3-month recall with scaling and root planing is available.

Cercek JK., *et al*. (1983) monitored the healing events of 7 patients with generalized chronic periodontitis during three consecutive phases of treatment: (1) brushing and flossing, (2) the use of a Perio-Aid subgingivally, and (3) supra and subgingival instrumentation [18]. They found limited improvement in the bleeding scores and pocket depths on probing with brushing and flossing alone. The use of the Perio-Aid to remove plaque subgingivally provided no additional improvement. With instrumentation there was "further and more pronounced improvement in bleeding scores and probing pocket depths, as well as a reversal of the probing attachment loss," The authors stated that with the presence of subgingival calculus; the subgingival plaque cannot be adequately removed, or if it is removed the mineralized deposits itself are capable of perpetuating periodontal disease.

Studies on the impact on gingivitis/periodontitis of chemical control using anticalculus agents

Stallard RE., *et al*. (1969) evaluated the effect of the macrolide antibiotic, CC 10232 (clindamycin), used as a mouthrinse, on dental plaque, calculus and gingivitis [1]. They found that in short term studies the reductions ranged from 11-23% for dental plaque, 70-91% for calculus and from 55-72% for gingivitis. These findings would suggest that the calculus effect was at least contributing to the reduction in gingivitis.

Suomi JD., *et al*. (1974) examined the long term effects (18 months) on 200 adults of normal, uninstructed use of a dentifrice containing 3% sodium etidronate and 0,22% sodium fluoride [1]. At the end of the 1 month period there was a 42% difference in the amount of supragingival calculus and a 27% difference in subgingival calculus when experimental and control groups were compared. There was no significant difference in gingivitis. The only conclusion at this point was that a 40% decrease in calculus is in itself not enough to effect gingivitis scores. The impact of a greater degree of reduction or of the combination of plaque and calculus reduction remains to be established.

In contrast to the study on chemical control of supragingival calculus, the clinical evidence for mechanical control is strongly suggestive that in addition to the traditional indirect effects, there is a direct role for subgingival calculus in the progression of periodontitis. By what mechanism does these mineralized plaque deposits exert such an influence still remained unanswered.

Morphologic Studies regarding etiologic significance of dental calculus

A series of morphologic studies indicate possible directions and several experimental studies provide support for the concept that subgingival calculus serves as a retentive site for toxic bacterial products. Retention of these foci prevents resolution of the disease process.

Lustmann J., *et al*. (1976)77 treated the deposits with sodium hypochlorite to remove plaque and other organic debris prior to examination with SEM [1]. They noted the presence of calcified masses having a spongy appearance and containing empty spaces representing the former sites of entombed and degenerated organisms.

Friskopp J., *et al*. (1980) used both transmission and scanning electron microscopy in their study of supra and subgingival calculus [20]. They found differences in the nature of the microbial coverings. On supragingival calculus filamentous organisms, oriented at right angles to the surface dominated, Subgingival calculus was covered by cocci, rods and filaments with no distinct pattern of orientation. After treatment with sodium hypochlorite the deposits lost their soft covering and channels of the same dimension as the filamentous organisms were found penetrating into the calculus. These channels were perpendicular to the surface in supragingival deposits and had no specific orientation in the subgingival.

Shirato M., *et al*. (1981) noted the presence of tubular holes in calculus. These holes appeared to be areas of uncalcified bacteria surrounded by calcified matrix [1]. Areas were also noted where the bacteria were calcified but were surrounded by a non calcified space. All of the morphologic studies attest to the porous nature of the calculus deposits.

Friskopp J (1983) noted cavities of non calcified material in a further study of the ultrastructure of supragingival calculus, Subgingival deposits tended to be more homogeneous [21].

Eide B., *et al*. (1983) in their scanning electron microscopic study of the root surfaces of extracted human teeth from patients with advanced periodontal disease found the presence of a mineralized surface coating other than calculus [22]. It was suggested that this coating originates from the inflammatory exudate and may include exogenous cytotoxic substances derived from plaque. Morphologically it corresponds to the dental cuticle and often increases in thickness in a coronal direction and blends with calculus.

These morphologic studies revealed the presence of uncalcified channels and tubular holes within dental calculus. However, the evidences provided by the morphologic studies were indirect. More direct evidences were required to consider dental calculus as a toxic waste dump site and in a sense a slow release device delivering pathogenic products.

Experimental Studies regarding etiologic significance of dental calculus

Baumhammers A., *et al.* (1970) studied the permeability of human and rat dental calculus. Using a series of dyes as well as tritiated endotoxin and tritiated glycine, they showed that human calculus was partially permeated in one hour and completely permeated by the dyes in 24 hours [23]. Radioautographs of the tritiated glycine and endotoxin showed progressive penetration with time. The authors hypothesized that dental calculus can act as a reservoir for irritating substances from microbial plaque and tissue lysis.

Patters MR., *et al*. (1982) assayed the bone resorbing activity using an organ culture system and the presence of antigens of Bacteroides gingivalis in plaque, calculus, cementum and dentin obtained from roots of extracted teeth from patients with severe periodontitis [1]. Significant stimulation of bone resorption was found in the preparations from periodontally involved cementum and in all samples of calculus. The levels of bone resorbing activity were higher in the calculus than in the cementum. Measurement of the relative amounts of antigenic material reactive with an antiserum to B. gingivalis disclosed the calculus contained over twice as much reactive material as cementum. Calculus was only slightly lower in reactivity than subgingival plaque from the root surfaces. Treatment with citric acid was able to remove these bacterial antigens from the cementum of periodontally involved teeth, but not from the subgingival calculus. This study provides the strongest evidence to date of the pathogenic potential of subgingival calculus.

The experimental studies on dental calculus revealed the permeable nature of dental calculus. It may be possible that dental calculus can act as a reservoir for irritating substances from microbial plaque and may have the pathogenic potential. However, these evidences are not sufficient to consider dental calculus as pathogenic.

Thus, from the above epidemiological, clinical, morphological and experimental studies, it is clear that the microbial plaque is considered to be the main etiologic agent for periodontal diseases since it contains pathogenic bacteria [1-4], whereas the dental calculus is not considered to be the etiologic agent. Since, dental calculus is a mineralized plaque and does not contain microorganisms within it, and if it does, than they are only the mineralized ones; and also, it is always covered by a layer of unmineralized plaque [1-4] containing pathogenic microorganisms, it is considered as only a predisposing factor and primary retentive factor.

According to the few authors, calculus may act as reservoir of organisms that may play a crucial role in etiology and progression of periodontal diseases [1,5,9,10]. If the bacteria in calculus are vital they may release toxic metabolic by products that may leach from calculus, initiating inflammatory responses in the oral soft tissues. And, when the bacteria become non vital, the by - products from their degradation, such as lipopolysaccharide cell membrane remnants, might be leached from the calculus into the tissues [1,5,9,10].

This opinion has led the various investigators to investigate the viability of bacteria within dental calculus.

Studies related to bacterial viability within dental calculus

Yardeni J (1948) examined the Gram stained smears of crushed dental calculus and found the presence of epithelial cells, Gram positive threads identical to leptotrichia, Borrelia and scarce Gram negative cocci [24]. He also cultured the calculus samples and obtained the growth of Gram negative cocci, Gram negative rods and filaments and occasional streptococci, lactobaccili, Gram positive threads, diptheroids, fusiforms and borrelia.

Gonzales F., *et al*. (1960), have done an electron microscopic study on ultra thin osmium fixed sections of dental calculus following their decalcification [25]. It revealed densely mineralized areas entrapping many degenerating microorganisms within which similar electron dense crystals were deposited.

Takazoe I., *et al*. (1963) obtained the pure cultures of oral aerobic filamentous microorganisms which were identified as Bacterionema matruchotti [26]. They electron microscopically observed intracellular calcification of these microorganisms upon treatment with calcifying solution *in vitro*. They stated that there is presence of filamentous microorganisms in dental calculus which are calcifiable during later stage of calculus formation.

Little MF., *et al*. (1966) analyzed supragingival calculus both chemically and chromatographically for amino acids and saccharide concluded that calculus matrix is reflection of plaque origin and residues of microflora [27].

Takazoe I., *et al*. (1970) suggested role of proteolipids in mineralization of dental calculus and demonstrated *in vitro* nucleation of hydroxyapatite by a membrane associated proteolipid extract from calcifiable Bacterionema matruchotti isolated from dental calculus [28]. They also observed that there was increase in amount of proteolipids with increase in culture times of Bacterionema matruchotti.

Slack JM., *et al*. (1970) examined various calculus specimens by direct fluorescent antibody techniques and cultural procedures for the presence of gram-positive filamentous or diphtheroid bacteria [29]. *Actinomyces israelii, A viscosus*, *A naeslundii, Arachnia propionica, Rothia dentocariosa, Bacterionema matruchotii,* and *Corynebacterium acnes* were observed in and cultured from the majority of specimens, with more than one species present in every specimen.

Jones SJ (1972) studied the mineralization front and structure of calculus on tooth surface by scanning electron microscopy [30]. He observed a continuous bacteria free mineralized layer on calculus surface facing enamel. The calculus abutting cementum was also bacteria free. The results indicated that bacteria when mineralized are not continuous with mineralized intermicrobial matrix or tooth surface.

Osuoji CI., *et al*. (1974) studied the composition of dental calculus and found Rhamanose as major, neutral sugar in dental calculus [31]. Neither ribose nor deoxyribose which is constituents of bacterial nucleic acids was detected in carbohydrate analysis supporting the suggestion that degradation of plaque organisms accompany mineralization.

Lustmann J., *et al*. (1976) In their scanning electron microscopic study on dental calculus identified calcified microorganisms with minute crystal of 500-400 Ao long deposited over them [19]. Coignoul E., *et al*. (1984) observed microorganisms in deeper layers (towards tooth) of dental calculi adjacent to tooth surfaces in dogs [32]. The bacterial populations were similar to that on surface layer (dental plaque) except the spirocheates were more common. They also stated that bacterial cultures of ground calculus material contain large no. of Streptococci and Actinomyces. Other bacterial populations included *Actinobacter coloacetum, Cornybacterium xerosis, Eikenella corrodens, Morexella sp*., *Pseudomonas sp*. and *Staphylococcus sp*. Many other bacteria are found in calculi but none of these constitute a significant percentage of total bacterial population.

Ruzicka F (1984) observed the ultrathin sections of supra and subgingival dental calculus samples electron microscopically and observed extracellular and intracellular calcifications [33]. Although, the cell walls of bacteria appeared to be calcified, but partly degenerated bacteria were also observed.

Robert JC., *et al*. (1990) used scanning electron microscopy of dental calculus samples in dogs, and found uninhabited bacterial recesses on inner surface of calculus towards tooth [34].

Torok K., *et al*. (1999) sudied dental calculus of 20 mummies with bright light, polarised and scanning electron microscopy [35]. They detected presence of gram positive in all preparates, gram negative bacteria in twelve samples and fungi only in three samples of dental calculus.

Tan B.., *et al*. (2004) investigated the ultra structure of young and mature supragingival calculus and found them similar with various large and small crystal types [10]. Non mineralized channels were observed extending into the calculus, often joining extensive lacunae, both containing intact non mineralized coccoid and rod shaped micro organisms. They concluded that supra gingival calculus contains non mineralized areas which contain bacteria and other debris. The viability of the bacteria and their identification could not be determined in this preliminary investigation. But the viable bacteria within these lacunae may provide source of re-infection.

Tan BT., *et al*. (2004) studied the bacterial viability within human supragingival dental calculus after eliminating the contamination due to overlying plaque by placing calculus samples over night under ultraviolet light on a shaker [9]. Their results indicated the possibility of presence of pathogens within calculus.

Calabrese N., *et al*. (2007) identified the presence of A*ctinobacillus actinomycetumcomitans, Treponema denticola* and *Porphyromonas gingivalis* within samples of dental calculus under transmission electron microscopy using immunogold stanning with polyclonal antibodies [5].

Moolya NN., *et al*. (2010) observed the samples of supragingival calculus under dark field microscopy for motility, under light microscopy for morphotypes (using gram stain), under fluorescent microscopy for viability (using acridine dye) and by culturing for growth [36]. From the results it appeared that viable bacteria were present within calculus, especially within internal channels and lacunae.

Kaur H., *et al*. (2013) examined samples of supra and subgingival calculus using grams stain, fluorescent microscopy, using acridine orange and dark field microscopy [37]. The observations revealed presence of viable bacteria inside dental calculus within the channels and lacunae. Thus, they stated that calculus may serve as reservoir of viable micro organisms and play a crucial role in the etiology and recurrence of oral infections even after treatment.

Gupta S., *et al*. (2016) conducted a study to investigate the viability of bacteria within dental calculus along with their identification [38]. A total of 60 samples of supragingival calculus were harvested from 30 patients having chronic inflammatory periodontal disease. These samples were divided into two groups (Group A and Group B). Samples of Group A were kept non-irradiated and samples of Group B were exposed to UV radiation. These were used for dark-field microscopy, gram staining and bacterial cultures. Bacterial identification of the cultures obtained was also carried out by performing various biochemical assays. The results of the study revealed the presence of motile spirochaetes within the samples under dark-field microscope. Gram staining revealed presence of numerous gram positive cocci and gram negative bacilli. Bacterial cultures showed growth of variety of aerobic and capnophilic microorganisms.

Conclusion

The nonmineralized plaque on calculus surface is the principal irritant. However, the current concepts in periodontics consider the underlying calcified portion only as a significant contributing factor. It does not irritate gingival directly but provides a fixed nidus for the continued accumulation of plaque and retains it close to gingival [1-4].

It is considered that the subgingival calculus may be the product rather than the cause of periodontal pockets. Plaque initiates gingival inflammation, which starts pocket formation and the pocket in turn provides a sheltered area for plaque and bacterial accumulation. The increased flow of gingival fluid associated with gingival inflammation provides the minerals that convert the continually accumulating plaque into subgingival calculus [1-4]. Dental calculus therefore is considered merely as an ash heap of minor significance in etiopathogenesis of disease.

In contrast, based on the results of studies presented in this paper it can be inferred that there is presence of viable aerobic and capnophilic bacteria inside dental calculus which may reside within lacunae and channels in the calculus.

Scientific Rationale of the Review

The current view is that dental calculus is not pathogenic by itself but the dental plaque with which it is covered contains unmineralized, viable and metabolically active bacterias [38]. The present study provides a strong evidence of viability of bacteria in calculus and thus its pathogenic nature. This would help viewing the role of calculus in etiopathogenesis of periodontal diseases from a different angle.

Research Implications of the Review

The findings of the study may be important and valuable and may open doors to further studies on the etiopathogenesis of the periodontal diseases. This study may be a roadway for various studies such as virulence and behaviour of bacteria in dental calculus, their pathogenicity, effect of age, sex, habits on microbiota of dental calculus etc.

Clinical Implications of the Review

Although, the traditional line of treatment for inflammatory periodontal diseases is the complete mechanical debridement of calculus. However, the findings of present study suggest that dental calculus is a reservoir of microorganisms and some of these bacteria are capable of growth when placed in suitable environment. Therefore, certain circumstances such as incomplete removal of calculus/ fracture of calculus deposits while brushing etc., may lead to exposure of these microorganisms which may lead to pathogenesis of diseases.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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