Dental caries, gingivitis, periodontitis: a review

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Abstract
Microbial populations colonizing the teeth and periodontal tissues are a major source of pathogens responsible for oral and dental infections including dental caries, gingivitis, periodontitis etc. Dental caries is a multifactor and infectious disease resulting due to interaction of three different aspects like dietary sugar, susceptible tooth enamel and oral microbial colonization. Plaques from caries active sites have significantly higher proportion of Streptococcus mutans (principle acid producer) with pH levels of 5.0 or lower. Dental decay occurs when normal demineralization remineralization is disturbed. On the other hand the most common form of gingivitis is chronic or long standing plaque induced gingivitis while acute necrotizing ulcerative gingivitis is most aggressive, developing gingivitis is associated with increasing numbers of Actinomyces israeliwhereas gingivitis with bleeding is associated with A. viscosus and pigmented Bacteroides. Periodontitis is defined as loss of alveolar support to the tooth and can be differentiated microbiologically and clinically into adult, localised juvenile and pre-pubertal periodontitis. Various species of Bacteroides, Actinomyces, Fusobacterium etc. have been isolated from cases of active periodontitis. Thus wherever possible both aerobic and anaerobic culture should be performed and appropriate antibiotic therapy should be prescribed instead of empirical treatment.

Keywords: *Dental caries – Gingivitis – Periodontitis.*

INTRODUCTION

Orodental infections are largely endogenous in nature[1]*. Microbial populations colonizing the teeth and periodontal tissues are a major source of pathogens responsible for oral and dental infections including dental caries, gingivitis, periodontal diseases; pericoronitis, endodontic infections, peri-implantitis and post extraction infections. These infections may develop in and around natural teeth or may even follow tooth extraction*[2]*.*

DENTAL CARIES

Dental caries is a multifactorial and infectious disease. The three different aspects which interact and result in dental caries are: (a) Dietary sugar especially fermentable sucrose; (b) Susceptible tooth enamel and (c) Oral microbial colonization.

Microbial acid production in plaque

The nonspecific plaque hypothesis has been assigned to the microbes which play a role in acid production

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when exposed to dietary carbohydrates. Stephan[3] studied the kinetics of acid production in patients who were caries-free (CF) and in those who exhibited various degrees of caries activity. pH readings were obtained prior to rinsing for 2 minutes with a 10% glucose solution and at intervals thereafter until the pH returned to its original value. There was a rapid pH drop, indicating that the glucose was instantaneously converted to acid products, mainly lactic acid. It was reported that the pH levels persisted at or below 5.0 in the plaque of caries-active (CA) subjects for a period of 20 to 50 minutes, whereas in the CF subjects the plaque pH dropped to about 6.0 and returned to resting values within 40 minutes. Thus comparable glucose exposures resulted in different pH intensities at the plaque-enamel interface that could be related to the current caries status of the individual. This indicated that either: (a) Salivary buffers of the CA subjects were inadequate relative to the CF subjects, OR (b) The plaques in CA subjects were producing more acid and at a much faster rate than the plaque in CF subjects, OR (c) Both of the above.

Protective role of saliva

Saliva is remarkably protective against decay. It is evident from the rampant decay that ensues when salivary flow is very low, Le. in xerostomia patients. The following factors contribute to the protective role of saliva[4]:

a) when food is masticated, the salivary flow increases; this not only serves to prepare the bolus for swallowing, but also provides a large liquid volume for plaque acids to diffuse into, as well as an increased concentration of bicarbonate buffer to neutralize these acids;

b) saliva contains pH raising factors such as urea and a tetrapeptide called sialin which contains lysine and arginine; the hydrolysis of these basic compounds by certain members of the plaque flora liberates ammonia hence causing the pH of saliva to rise;

c) saliva and the fluid phase of plaque are supersaturating with calcium and phosphate ions; the maintenance of the supersaturated state of these ions provides a constant and a powerful remineralization mechanism on those surfaces of the tooth that are bathed by saliva;

d) salivary proteins or glycoproteins such as lysozyme, lactoperoxidase, lactoferrin and high molecular weight agglutinins possess antibacterial activities.

Salivary antibacterial proteins are found in similar levels in CF and' caries-susceptible individuals. Analysis of salivary electrolytes, flow, and buffering capacity in CF or caries-susceptible individuals has failed to show any differences in these measured parameters. Thus suggesting that caries susceptibility is not related to any apparent abnormality of the saliva and that the low pH following glucose ingestion in CA subjects reflects greater microbial acid production in the plaques.

Critical pH

Critical pH is defined as the pH at which the tooth begins to lose some of its mineral contents i.e. demineralisation[4]. Its value is in the vicinity of pH 5.0 to 5.5. At critical pH, the hydroxyapatite G the tooth acts as a buffer. Following the initial rapid fall in pH there is a plateau section in which the pH remains relatively stable for 10 to 40 minutes. It is during this plateau that the tooth mineral dissolves to buffer further microbial acid production. If the pH drops further to 3 or 4, the surface layer of mineral would be irreversibly lost. At pH 5.0, the minerals are lost from the subsurface layers in such a way that the repair (in the form of remineralization) can proceed once the pH returns to values above the critical pH.

Demineralization – remineralization

The surface of enamel has a remarkable conservative pattern of dissolution, with regard to the development of dental decay[5]. Whenever a fermentable dietary substrate diffuses into the plaque and is converted to acid end products, some degree of subsurface demineralization occurs. Then, in between meals and snacks, the pH returns to neutrality and calcium and phosphate in the plaque, driven by the supersaturated concentration gradient, diffuse into the lesion and promote remineralisation. Demineralization which progresses to cavitations occurs if the frequency and magnitude of acid production overwhelm the repair process.

Demineralization predominates if: (a) there is frequent eating, or (b) the repair process is compromised by a reduction in salivary flow.

Remineralization predominates if: (a) the plaque acid production is restricted, as occurs with

the ingestion of low sucrose diets, or (b) sugar substitutes are used for between - meal snacks, or (c) fluoride is used, which promotes remineralization.

Although there are differences as to how and which microorganisms produce carious lesions, it is uniformly agreed that caries cannot occur without microorganisms. Indirect evidences implicating microorganisms in the etiology of caries are as follows:

1) germ free animals do not develop caries;
2) antibiotics fed to animals are effective in reducing the incidence and severity of caries;
3) interrupted teeth do not develop caries, though the same teeth when exposed to the oral environment and microflora can become carious;
4) oral bacteria can demineralize enamel and dentine in vitro and produce carious lesions;
5) microorganisms have been histologically demonstrated invading carious enamel and dentine and can be isolated and cultivated from carious lesions[6].

Plaques from CA sites have significantly higher proportions of S. mutens than do plaques from CF sites, especially when the CF sites are in CF individuals[7]. pH levels of 5.0 or lower persist in plaques from CA sites for upto several hours. Harper and Loesche[8] conducted a survey of the ability of certain plaque species to convert sucrose to lactic acid at pH 5.0. A. viscosus, S. sanguis, S. salivarius and S. mitis were found to be virtually unresponsive, whereas S. mutans and to a lesser extent L. casei were quite active. It suggests that S. mutans is the principal acid producer in vivo when plaque pH is buffered on the acid plateau. Frequent ingestion of sucrose, could lead to the selection of a microbe that, because of its acidity, is most competitive at pH levels that both solubilize the tooth mineral and discriminate against other plaque species. Acidity is also a trait possessed by the Lactobacilli, a group of organisms otherwise not noted for their role in acid production, especially from sucrose. The delayed appearance of Lactobacilli in the carious lesion is compatible with the pH reaching such acidic values that these the most acidity of plaque species can thrive.

Dental decay occurs when the normal demineralization remineralization cycle is disturbed by either an increase in the acid challenge or a decrease in the salivary repair functions. A change in dietary pattern to one of frequent ingestion of sucrose leads to the selection of S. mutans and Lactobacilli which, because of their acidity at the critical pH for enamel demineralization, exploit this sucrose bioavailability to expand their niche in the plaque. Under less severe sucrose exposure the metabolic activity of the S. mutans can potentialize the postprandial pH drops at the plaque-enamel interface, thereby interfering with the normal salivary remineralizing system and leading eventually to tooth decay[4].

There are four types of lesions in dental caries and different organisms display some selectivity as to which tooth surface they can attract[6]. These sites are protected from the cleansing action of saliva, tongue and oral musculature. Therefore food, bacteria, salivary proteins and other oral debris readily collect here.

Pit and fissure caries

Pit and fissure caries is the commonest carious lesion found in man. Many organisms can colonize in fissures, which provide mechanical retention for bacteria. Gnotobiotic rats mono-infected with either S. mutans, S. salivarious, S. sanguis, L. acidophilus, L. casei, A. viscosus, A. naeslaundii or A. israelii develop fissure lesions. Preliminary findings indicate that cocci constitute about 80% of the viable organisms in the first· two days. Rods and filaments increase to 12% in five days old fissures.

Smooth surface caries

A limited number of organisms have proved ability to colonise smooth surfaces. S. mutans is very significant in this respect.

Root caries

Gram-positive filamentous rods, including Actinomyces species, have been associated with this type of lesion. Strains of Nocardia, S. mutans, S. sanguis besides causing enamel caries may at times also cause root caries.

Deep dentinal caries

The environment in deep dentinal lesions is different from that at other locations; therefore, the flora here is also different. The predominant organism is Laclobacillus which accounts for approximately one third of all bacteria. Frequently

isolated Gram-positive anaerobic rods and filaments are Bifidobacterium, Propionibacterium and Actinomyces. The incidence of Gram-positive facultative cocci is low.

Plaque contains Gram-positive cocci of genus Streptococcus as the predominant organisms, irrespective of age and previous die. These streptococci have been divided into various groups on the basis on their colony morphology and physiological characteristics[9].

a) Streptococcus mutans

They ferment mannitol and sorbitol, and produce extra cellular glucose from sucrose. With 'the exception of S. ferus, these microorganisms are cariogenic in animal models[10]. Clarke[11] isolated such organisms from human carious lesions and called them S. mutans because on Gram stain they were more oval than round and thus appeared to be a mutant form of a streptococcus.

When S. mutans strains were collected from different sources it became apparent that considerable serological[12] and genetic heterogeneity existed[13]. The similarity in pathogenesis has led most investigators to call all of them by the specific epithet S. mutans. Eight serotypes could be recognized on the basis of carbohydrate antigens and deoxyribonucleic acid (DNA) hybridization studies revealed the existence of four genetic groups. S. mutans was assigned to those human isolates that resembled Clarke's original description and the representative strain of S. mutans that was present in the National Collection of Type Cultures under the number NCTC 10499. S. mutans contains which posses c, e and f antigens. Serotype 'c' accounts for about 70 to 100% of the human isolates of mutans streptococci (MS), therefore, it is appropriate that S. mutans be the specific epithet for the human type of MS. Most of the remaining human isolates of MS, posses d, g, h carbohydrate antigens and are called S. sobrinus, S. rattus (serotype b), S. cricetus and S. ferus were the epithets assigned to MS isolated from laboratory - bred rats, laboratory - bred hamsters and wild rats respectively.. S. mutans; S. sobrinus, S. rattus and S. cricetus are cariogenic in animal models[4].

Studies of plaque from humans indicate that S. mutans is pandemic in many parts of the world[14]. S. mutans is found in large numbers in the plaque isolated from CA populations, and more frequently in plaque overlying carious lesions than in plaque from sound tooth surfaces[15].

Human salivary concentrations of S. mutans ranges from undetectable to 10^6 to 10^7 CFU (colony forming units)/mL[16]. Salivary contamination of cups, glasses and eating utensils such as spoons may account for the transmission of S. mutans from parent to child. Mothers with salivary concentrations of S. mutans more than 10^7 CFU/mL are more likely to infect their infants than mothers with lower salivary levels. S. mutans exhibits several important properties[6]:

1) it is not fastidious in its growth requirements as are most streptococci;

2) it has the ability to use ammonia as the sole source of nitrogen, which gives it an ecological advantage and it can adapt itself for growth in the deepest part of microbial aggregations on the teeth, where the anaerobic environment and ammonia may be sufficient to permit survival without exogenous amino acids;

3) it synthesizes insoluble polysaccharides from sucrose;

4) it is homofermentative lactic acid former;

5) it colonizes on tooth surfaces;

6) it is the most acidogenic and acidity of the oral streptococcal species;

7) cariogenic strains of S. mutanscontain lysogenic bacteriophage which is lacking in non-cariogenic strains;

8) sucrose greatly favors the colonization by S. mutans; it is now recognized that the organisms can adhere independently of sucrose and it has been postulated that this is mediated either by interactions of components on the surface of organisms with blood group reactive salivary glycoproteins in acquired pellicle or by electrostatic interaction between the teichoic acids on the bacterial cell surface, calcium ions and salivary glycoproteins[17].

Considerable evidence from historic, epidemiologic observations and animal experiments indicates that shortly after sucrose is introduced into the diet, there is a notably higher incidence of dental decay. When human volunteers switched from their usual diets to ones high in sucrose to plaque proportions of S. mutans, Lactobacilli, Veillonella spp. and yeasts increased, while those of S. sanguis decreased[18].

Fitzgerald and Keyes[19] provided the first definitive evidence for bacterial specificity and that

the cariogenicity of a microorganism depended upon adhesive properties of a microorganism in addition to production of adds from fermentable carbohydrates.

Sucrose metabolism by S. mutans is complex and when sucrose is in excess, it results in lactic acid production[20]. Before sucrose enters the cells, a certain small percentage ↔10%) is transformed by a variety of hexose transferases into glucose or fructose that either diffuse into the surrounding environment or remain associated with the cell. Various studies indicate that in animal models glucan formation is a virulence factor that is important primarily for smooth surface decay involving an infection 'with S. sobrinus.

McCabe, Keyes and Howell[21], noted that in the presence of sucrose, S. mutans formed adhesive colonies which stuck to the surface of culture vessels or to any wire or object suspended in the culture media. Such colonies were not formed in glucose broth or by the non-cariogenic species when they were grown in sucrose broth. This suggested that the ability of S. mutans to form adhesive plaque was related to its odontopatology activity.

Chemical analysis indicated that the adhesive material was a glucose homopolymer or glucan which contained dextran. At least two different glucotransferases (GTFs) are needed to synthesize these glucans. One enzyme called GTF-S, which synthesizes a soluble alpha-(1-6)-branched dextran, whereas, the other enzyme called GTF-I, synthesizes an insoluble alpha-(1-3) D-mutan. S. mutans appears to form primarily GTF-S whereas S. sobrinus has both GTF-S and GTF-I activities. The pH in plaque can drop below 5.0 in 30 to 120 minutes following exposure to a fermentable carbohydrate. Most plaque bacteria are not metabolically active at these pH values but S. mutans and, to a lesser extent. S. sobrinus have a pH optirrla at about 5.0 to 5.5 and may be selected in the plaque at these pH values.

Acidity is related to the ability of the cell membrane to maintain a relatively alkaline cytoplasm in an acid environment. The frequent ingestion of sucrose results in lowering of pH to critical level. At this pH the tooth mineral solubilizes and discrimination occurs between plaque species. Only those species are selected which are acidity e.g. S. mutans and Lactobacillus.

Colonization by S. mutans occurs after tooth eruption, and if the fissures become colonized in their depths, then decay may be inevitable. However, if this colonization is delayed until the fissure depths are occupied by other bacteria, there is a possibility that decay will not occur or its occurrence will be greatly reduced. Loesche[4] reviewed the role of S. mutans in dental decay and summarised that there is convincing circumstantial evidence and S. mutans, possibly S. sobrinus and Lactobacilli are human odontopathologies. Acidity appears to be the most consistent attribute of S. mutans that can be associated with both its selection in stagnant areas and its cariogenicity.

b) S. sanguis

This is one of the predominant groups of streptococci colonising on the teeth. By using a sensitive immunoflourescent technique, investigators have found S. sanguis in all plaques tested and on some tongues, but not in any throat specimens. Certain strains within this group are minimally cariogenics in animals but most are not. Caries from this strain occurs primarily in sulci and is 'significantly less extensive on tooth than that of S. mutans.

c) S. salivarious

They have been found in the plaque, throat, nasopharynx and oral mucosa, but their natural habitat is dorsum of the tongue. In humans they have only a minor degree of cariogenic potential.

d) S. mitis

The proportion of this group varies among subjects, however, it is found most regularly on the non-keratinized mucosa, particularly the cheek, lip and ventral surface of the tongue.

Lactobacilli are Gram-positive, non-spore forming rods which generally grow best under microaerophilic conditions. Lactobacilli are found mostly as transients in the mouths of infants. Lactobacilli represent about 1 % of the oral flora, L. casei and L. fermentum being the most common species. A favourite habitat of Lactobacilli is in the dentine of deep carious lesions[22].

Actinomycoses is a Gram-positive, non-motile, non-sporing organism occurring as rods and filaments which vary considerably in length.

Filaments are usually long and slender and may be branched. Five species have been found in oral flora[23]:

- Anaerobic: A. israelli, A. bovis;

- Facultative anaerobic: A. viscosus, A. naeslundii, A. odontolyticus.

All species of Actinomycoses ferment glucose, producing mostly lactic acid and lesser amounts of acetic and succinic acid. Actinomycoses is a good plaque former, capable of adhering to wires and forming tenacious deposits on the teeth of infected animals. It is the most common group of organisms isolated from the sub gingival microflora and from plaque of human root surface caries[24]. A. naeslundii predominates in the plaque of young children, while plaque from teenagers and adults has a higher proportion of A. viscosus. High numbers of A. viscosus have been associated with gingivitis[23].

Syed et al.[25] and Sumney and Jordan[26] have identified S. mutans, S. sanguis, enterococci, Bifidobacterium, Veillonella in root surface caries. Little mention was made of Lactobacilli as components of the flora; this probably relates to the methods used in sampling and cultivation of the organisms.

Gingivitis is defined as inflammation in soft tissues (gingivae) around teeth without loss of periodontal support. Several types of gingivitis are distinct clinically and microbiologically. The most common form is chronic or long standing plaque induced gingivitis, while the most aggressive form is the acute necrotizing ulcerative gingivitis (ANUG). Other forms of gingivitis Developing gingivitis was associated with increasing numbers of A. israelli, whereas gingivitis with bleeding was associated with A. viscosus and certain species of pigmented Bacteroides, probably P. gingivalis[27].

White and Mayrand[28] observed that species of Bacteroides were the cause of gingivitis. Sites with a gingival index score of 3, had more Gram-negative anaerobic rods than did the less affected sites. 31.8 percent of these bacteria were Bacteroides assacharolyticus, a species absent from healthy sulcus.

Moore et al.[29] reported the results of their extensive studies of the microflora associated with human experimental gingivitis. They also observed that although the composition of the flora from one subject to another was reasonably similar for first 4 days of plaque accumulation, the composition subsequently became more diverse, with a great deal of variation from subject to subject and related in part to inflammatory status of the site sampled. They found colonization and increases in proportions of species of Actinomycoses, Streptococcus, Fusobacterium, Veillonella and Treponema to be associated reproducibly with the development of gingivitis. As the lesion became more severe, additional species appeared, some of which have been associated with periodontitis. Kormman and Loesche[30] studied the microflora of gingivitis in pregnant women. They observed B. intermedius was associated with the appearance and severity of gingivitis. Proportions of B. intermedius correlated with the levels of plasma estrogen and progesterone.

Periodontitis is defined as loss of alveolar support to the tooth. It can be differentiated microbiologically and clinically into adult periodontitis, localized juvenile periodontitis and pre-pubertal periodontitis.

Adult periodontitis

A wide range of species has been isolated in cases of periodontal disease. Dzink, Socransky and Haffajee[31] compared sites of active and inactive periodontal disease and identified a set of putative pathogens associated with active sites. The species isolated included P. gingivalis. P. intermedia, Campy/ obacter rectus, B. forsyth us and Actinobacillus ctinomycetemccomitans. Additional species (S. ora lis, Actinomyces spp. and V. parvu/a) were associated with inactive sites and were proposed as possibly protective or beneficial organisms[32].

Localized juvenile periodontitis

Classic localized juvenile periodonhtitis (LJP) affects adolescents and is aggressively destructive. Lesions ap localised to selected teeth, usually first permanent molars and central incisor Unlike most forms of periodontal disease, LJP is characterised by minin tooth associated plaque and inflammation. A genetic component for 1 disease has been described and may include a defect in polymorphonuc leucocytes[33].

Prepubertal periodontitis

Prepubertal periodontitis can preser localised or generalised defects affecting deciduous teeth. Simi' between prepubertal and LJP include minimal

inflammation, infection c sites with A. actinomycetemcomitansand a strong association with a systemic leucocyte defect[34]. Other species. cultured from sites of prepubertal periodontitis included P. intermedia, P. gingiva lis, Capnocytophaga sputigena and Eikenella corrodens. Successful treatment of prepubertal periodontitis after the elimination of A. actinomycetemcomitans reduces the polymorphonuclear neutrophil defect. This observation suggests that this defect may be trigered by the infection with A. actinomycetemcomitans.

MICROBIOLOGY OF PERIODONTITIS

A long series of previously reviewed and recent studies show a close relationship between cultivable P. gingiva lis. P. intermedia and severe adult periodontitis[35]. In investigations from Virginia by Moore et al.[36], small amounts of B. gingiva lis and relatively low proportions of B. intermedius were detected in deep periodontal pockets. The reason for the low recovery of black-pigmented Bacteroides in these studies is not known but may be due to varying criteria of patient selection, the microbiological sampling and culture methods used, or true geographically-based differences.

Slot[37] studied the importance of B. gingiva lis, B. intermedius and A. actinomycetemcomitans in progressive adult periodontitis. A total of 146 adults with a history of advanced periodontitis presented with 130 periodontitis lesions which in the preceding 5 years had experienced a marked loss of radiographically detectable alveolar bone height and 105 periodontal sites which for 2 to 17 years had exhibited no further alveolar bone loss. B. gingival/is was found in 54 progressing periodontal sites and 6 non-progressing sites. B. intermedius occurred in 76 progressing and 38 non-progressing sites. Association of B. intermedius with gingivitis may explain its high prevalence in non progressing sites. A. actinomycetemcomitans was demonstrated in 65 progressing lesions but only in 5 non progressing lesions.

Chakraborti et al.[38] studied the role of anaerobes in advanced adult periodontitis (AAP). 36 normal gingival ahd 100 AAP samples were studied. Anaerobes were found in 38.8 percent and 42 percent of normal gingiva and patients of AAP respectively. Aerobic organisms were more in AAP (100%) than in normal gingiva (94%). The anaerobes in normal gingiva showed scanty growth, while they.showed moderate to heavy growth in 19 percent of cases of AAP. The number of cultures with respect to the anaerobic isolate was more in AAP (1:0.5) than normal gingiva (1:0.4). S. aureus, S. pneumoniae and Klebsiella were detected in AAP but not in normal gingiva. Of the anaerobes Gram-positive bacilli were more in AAP, whereas Gram-positive cocci predominated in normal gingiva. The incidence of Gram-negative bacilli did not differ appreciably in the two groups.

Beena et al.[1] studied the bacterial flora in periodontal infections. They isolated and compared the isolates from patients of active· periodontitis, inactive periodontitis, gingivitis and normal healthy individuals. More anaerobes were isolated from the cases of active periodontitis (69.42%), followed by inactive periodontitis (62.42%), gingivitis (57.53%) and in normal oral flora (45.65%). Anaerobes were isolated from all cases of periodontal infection and these infections were polymicrobial. Anaerobic Gram-negative bacilli like B. melaninogenicus and Fusobacterium were isolated in large numbers from active periodontitis. B. melaninogenicus was not isolated from the flora of healthy individuals. The rate/lesion in active periodontitis was 4.4, inactive periodontitis 3.73, gingivitis 3.65 and normal flora 2.3 respectively. All the anaerobes isolated were sensitive to metronidazole.

Doiphode et al.[39] in their study on management of advanced periodontitis with initial antimicrobial therapy followed by conventional periodontal treatment, subjected 50 cases of periodontitis for microbiological analysis. These were classified as juvenile periodontitis (9 cases), rapidly progressive periodontitis (28 cases) and adult periodontitis (13 cases). Black pigmented Bacteroides were the commonest isolates (80%) followed by A. actinomycetemcomitans (56%) Capnocytophaga in 8 percent of the cases.

Moore et al.[40]studied patients with severe periodontitis, moderate periodontitis and individuals undergoing experimental gingivitis with an inflammation score of 2.0. The subgingival pocket flora was not statistically different among the various individuals, but the combined floras did differ from the found at normal disease free

sites. F. nuclea-tum, Eubacterium and Peptostreptococcus micros were the most prominent component at diseased sites. B. gingivalis was found, but its proportion was less than B. intermedius.

ANTIMICROBIAL THERAPY

The concept of dental infections as being bacteriologically nonspecific offers no rationale for antibacterial treatment. A standardised dilution technique as well as a disc diffusion technique can be used to measure specific bactericidal and minimum inhibitory concentrations of anaerobes.

Thadepalli and Chuah[41] stated that penicillin, ampicillin, cephalothin, cefazole and all other first generation cephalosporins are ineffective because of beta lactamase produced by a large variety of anaerobes. Broad spectrum penicillins like carbenicillin, ticarcillin, mezlocillin and piperacillin are highly effective even against beta lactamase producing strains because beta lactamase is ineffective against these drugs. Most of the third generation cephalosporins like cefoperazone, ceftizoxime, cefotaxime are all effective against anaerobic bacteria. Chloramphenicol has been the time honoured remedy for anaerobic infections but several failures have occurred with this drug. Macrolide group of antibiotics like erythromycin are effective in vitro but in vivo failures are common and therefore not recommended. Clindamycin and lincomycin-are very effective. Metronidazole without question is very active against anaerobes.

Walker et al.[42] determined the in vitro antibiotic susceptibility of periodontal bacteria, both aerobic and anaerobic. They found that although most bacteria were relatively susceptible to the penicillins, greater activity was generally noted with amoxycillin than with either ampicillin or penicillin.

Clindamycin and metronidazole both demonstrated excellent activity against anaerobic Gram-negative rods. Erythromycin, a commonly used antibiotic in periodontal infections, was considerably less active than the other antibiotics against the majority of the periodontal bacteria. Thus it is important to know the antibiotic sensitivity of periodontal bacteria instead of empirical therapy.

Predominant culture studies are too laborius for routine culture in oral lesions. Several more practical rapid methods have been developed like gas liquid chromatography, immunologic ass3.Y and DNA probe methods[2].

Cárie dentária, gengivite, periodontite: uma revisão

Resumo

As populações microbianas que colonizam os dentes e os tecidos periodontais são uma fonte importante de patógenos responsáveis por infecções dentárias, incluindo infecções orais, cárie, gengivite, periodontite, etc. A cárie dentária é uma doença infecciosa e multifatorial, resultante da interação de três diferentes aspectos, tais como: dieta com açúcar, esmalte dentário e cavidade oral sensível à colonização microbiana. Placas onde há cárie ativa têm uma proporção significativamente mais elevada de ter *Streptococcus mutans* (princípio: produtor de ácido), com pH de 5,0 ou menor. O enfraquecimento dentário ocorre quando há um distúrbio na desmineralização e remineralização do dente. Por outro lado, a forma mais comum da gengivite é a crônica ou a longa permanência da placa, enquanto a gengivite aguda, ulcerativa necrotizante, é a mais agressiva. O desenvolvimento da gengivite está associado a um número crescente de *Actinomyces gengivite israeli*, enquanto que o sangramento está associado com A. viscosus e bacterióides. A periodontite é definida como a perda do apoio alveolar dos dentes e pode ser diferenciada clinicamente e microbiologicamente no adulto, no jovem e no pré-púbere. Várias espécies de bacterióides, *Actinomyces*, *Fusobacterium*, etc, foram isoladas de casos de periodontite ativa. Assim, sempre que possível, tanto a cultura aeróbia quanto anaeróbia deve ser realizada e uma terapia antibiótica adequada deve ser prescrita em vez de tratamento empírico.

Palavras-chave: Cárie dentária – Periodontite – Gengivite.

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