Role of anaerobic bacteria in dental infection

K.Pavithra
Undergraduate Student,
Saveetha dental college hospitals, Saveetha University,
162,Poonamelle high road,
Velapanchavadi,
Chennai-600095, India.

Dr.Gopinath
Department of Microbiology,
Saveetha Dental College and hospitals,Saveetha University,
Chennai, India.

Corresponding Author
Dr.Nivethitha
Department of Conservative and Endodontics,
Saveetha dental college hospitals, Saveetha University,
162, Poonamelle high road,
Velapanchavadi,
Chennai-600095, India.

Abstract
Anaerobic bacteria are a common cause of infections, some of which can be serious and life-threatening. Because anaerobes are the predominant components of the normal flora of skin and mucous membranes, they are a common cause infections of endogenous origin. Anaerobes make up a significant part of the oral and dental indigenous and pathogenic flora. Their role in periodontal disease, root canal infections, and infections of the hard and soft oral tissue, as well as their importance as foci for disseminated infectious disease is well established. Isolates include *Fusobacterium*, *Bacteroides*, *Actinomyces*, *Peptococcus*, *Peptostreptococcus*, *Selenomonas*, *Eubacterium*, *Propionibacterium*, and *Treponema*. Recently, several significant advances in our knowledge have set the stage for future research. First, circulating levels of hormones in pregnant women were shown to be stimulatory to *Bacteroides* species, which were associated with increased levels of gingival infection. Second, bacterial invasion of the soft and hard periodontal tissues has been documented in gingivitis, advanced periodontitis, and localized juvenile periodontitis. The frequency and identity of invading bacteria will determine the implications for diagnosis and treatment.Anaerobes involve almost all dental infections,These include dental abscess, endodontal pulpitis and periodontal infection, and perimandibular space infection. Pulpitis can lead to abscess formation and eventually spread to the mandible and other neck spaces.

The fate of the infection depends on the virulence of the bacteria, host resistance factors, and regional anatomy. Serious consequences arising from the spread of a dental abscess lead to significant morbidity and mortality. Acute dental abscess is polymicrobial, comprising of strict anaerobes, such as anaerobic cocci, *Prevotella*, *Fusobacterium* species, and facultative anaerobes, such as viridans group *streptococci* and the *Streptococcus anginosus* group. Numerous novel, uncultivable and fastidious organisms have been identified as potential pathogens with the use of non-culture techniques. The majority of localized dental abscesses respond to surgical treatment while the use of antimicrobials is limited to severe spreading infections.

Keywords: dentalinfection, Pulpitis, Dentoalveolarabscess, Periodontitis, Gingivitis, Pericoronitis.

Introduction:-
Anaerobes make up a significant part of oral and dental infection and pathogenic flora. Their role in periodontal disease , root canal disease, infection of hard and soft oral tissue(1). complexity of the oral and dental microbiota has prevented the clear elucidation of specific etiologic agents in most forms of oral and dental infections. These infections are the most common infections in humans(2). There are 264 morphologically and biochemically distinct bacterial groups or species that colonise oral and dental ecologic sites.Common anaerobic isolates include Fusobacterium, Bacteroides, Actinomyces, Peptococcus, Peptostreptococcus, Selenomonas, Eubacterium, Propionibacterium, and Treponema.The accumulation of bacteria on or around the tooth is referred to as plaque(3).Once pathogenic bacteria within the plaque become established, they can cause localized and disseminated (systemic) complications: bacterial endocarditis, infection of orthopedic or other prosthesis, pleuropulmonary infection, cavernous sinus infection, septicemia, maxillary sinus infection, mediastinal infection, and brain abscess(4).

Most common type of anaerobes in dental infection are:-
Pulpitis
Dentoalveolar abscess
Periodontitis
Gingivitis

Pericoronitis

Pulpitis

Pulpitis is an inflammation of the dental pulp that can result from thermal, chemical, traumatic, or bacterial irritation. The most frequent inducer of pulpitis is dental caries that leads to destruction of enamel and dentin resulting in bacterial invasion. Secondary infection of the pulp by supragingival anaerobes occurs frequently in teeth with longstanding caries. Invasion of the pulp and spread of infection to the periapical areas can promote spreading of infection to other anatomical areas(5). The symptoms of acute suppurative pulpitis include low-grade fever, pain, soreness of the tooth, and facial swelling. Pain is usual induced by hot liquids, a reaction believed to be caused by expansion of gases produced by gas-forming bacteria trapped inside the root canal(6).

Sampling from the root canal for recovery of organisms, before treatment, during treatment and at the end of therapy to insure eradication of the infection is useful, and can differentiate between infectious and non-infectious pulpitis. The patient may experience intense pain that may be difficult to localise. It may be referred to the opposite mandible or maxilla or to areas supplied by common branches of the fifth cranial nerve(7).X-rays, pulp testers, percussion, and palpation are helpful aids in confirming the diagnosis. Cleansing of the cavity to remove debris and packing the cavity with zinc oxide-eugenol cement usually will afford relief in early pulpitis.

Once pulpitis developed the infected pulpal tissue should be removed and root canal therapy instituted, or the tooth should be extracted.It is proved beyond doubt that presence of microbiota is a major deterrent in endodontic infection by the classical study by Kakehashi *et* . There are so many ways by which the microorganisms reach the pulp and it is of prime importance that we know the same for our treatment planning. The various routes by which the microorganisms reach the pulp are as follows.(8)

Dentinal tubules: After a carious lesion or during dental procedures, microorganisms may use the pathway in a centripetal direction to reach the pulp. Bacteria gain access to the pulp when the dentin distance between the border of carious lesion and the pulp is 0.2 mm.

Open cavity: Direct pulp exposure of traumatic origin such as in coronal fracture, or that of iatrogenic nature due to operative procedures, breaks the physical barrier imposed by dental structures and leaves pulp in contact with the septic oral environment.

Periodontal membrane: Microorganisms from gingival sulcus may reach the pulp chamber through the periodontal membrane, using a lateral channel or the apical foramen as a pathway. This pathway becomes available to microorganisms during a dental prophylaxis, due to dental luxation, and more significantly, as a result of the migration of epithelial insertion to the establishment of periodontal pockets.(9)

Blood stream: A transient bacteremia may occur for any number of reasons during the normal day of a healthy individual. The bacteria present in the blood would be attracted to the dental pulp following trauma or operative procedure that produced inflammation without causing pulp exposure. This attraction through blood or lymph is known as anachoresis, which serves as a path for endodontic infection.

Faulty restoration: Studies have proven that salivary contamination from the occlusal aspect can reach the periapical area in less than 6 weeks in canals obturated with guttapercha and sealer.(9)If the temporary seal is broken or if the tooth structure fractures before final restoration, or if the final restoration is inadequate, bacteria may gain access to the periapical tissue and result in infection.

Extent: Microorganisms might reach the principal and/or lateral canals migrating from an infected tooth to a healthy pulp as a consequence of the contiguousness of the tissues, thereby spreading the infection to an adjacent tooth.(10)

Endodontic biofilms

Endodontic microbiota is established to be less diverse compared to oral microbiota. Progression of infection alters the nutritional and environmental status within the root canal, making it more anaerobic with depleted nutritional levels. These changes offer a tough ecological niche for the surviving microorganisms(11). But complete disinfection of root canal is very difficult to achieve because of persistent microbes in anatomical complexities and apical portion of root canal. Because biofilm is the manner of bacterial growth which survives unfavorable environmental and nutritional conditions, the root canal environment will favor biofilm formation(12).

Endodontic bacterial biofilms can be categorized as
*intracanal biofilms,
*extraradicular biofilms,
*periapical biofilms and
*biomaterial-centered infections.

Intracanal microbial biofilms

They are microbial biofilms formed on the root canal dentin of an endodontically infected tooth.

Extraradicular microbial biofilms
They are also termed as root surface biofilms which are formed on the root (cementum) surface adjacent to the root apex of endodontically infected teeth.

Extraradicular biofilms are reported with asymptomatic periapical periodontitis and in chronic apical abscesses with sinus tracts. Sometimes, the extraradicular biofilm becomes calcified and gets associated with periapical inflammation and delayed periapical healing in spite of adequate orthograde root canal treatment(13).

Periapical microbial biofilms
They are isolated biofilms found in the periapical region of endodontically infected teeth. Periapical biofilms may or may not be dependent on the root canal. These microorganisms have the ability to overcome host defense mechanisms, thrive in the inflamed periapical tissue and subsequently induce a periapical infection(14).

Biomaterial-centered infection
Biomaterial centered infection is caused when bacteria adhere to an artificial biomaterial surface and form biofilm structures(15). Presence of biomaterials in close proximity to the host immune system can increase the susceptibility to biofilm. In endodontics, biomaterial-centered biofilms form on root canal obturating materials. These biofilms can be intraradicular or extraradicular depending on whether the obturating material is within the root canal or has extruded beyond the root apex

Dentoalveolar abscess:-
An alveolar or apical abscess may be either acute or chronic. The acute alveolar abscess is an extension of necrotic or putrescent pulp into the periapical area, which induces bone and tissue necrosis and accumulation of pus. It may also occur after trauma to the teeth or from periapical localisation of organisms. As the abscess growth, more tissue may be involved, including adjacent teeth, and the pressure within the abscess may produce a fistula to the gingival surface or to the oral or nasal cavities.An abscess can be focal or diffuse and present as red tender fluctuant gingival swelling.

Pain from an acute abscess usually is intense and continuous. The involved tooth is painful when percussed. Hot or cold foods may increase the pain.A chronic periapical abscess presents few clinical signs, since it is essentially a circumscribed area of mild infection that spreads slowly(16).In time, the infection may become granulomatous. Radiographic studies of the involved tooth can be helpful, and free air eventually can be observed in the tissues. Complications can occur by direct extension or homogenous spread. If treatment is delayed, the infection may spread directly through adjacent tissues, causing cellulitis (phlegmona), varying degrees of facial edema, and fever. The infection may extend into osseous tissues or into the soft tissues of the floor of the mouth.

Local swelling and gingival fistulas may develop opposite the apex of the tooth, especially with deciduous teeth(17).Serious complications from periapical infections are relatively. The infection can spread to tissues in other portions of the oral cavity, causing submandibular or superficial sublingual abscesses; abscesses may be produced also in the submaxillary triangle or in the parapharyngeal or submasseteric space(18).

In the maxilla, periapical infection may affect only the soft tissues of the face, where it is less serious. It may extend, however, to the intratemporal space including the sinuses and then to the nervous system, where it can cause serious complications such as subdural empyema, brain abscess, or meningitis. Treatment is Extraction or root canal therapy and drainage of pus usually are indicated. Antibiotic prophylaxis is recommended if extraction or drainage is contemplated in patients at risk of developing endocarditis. Penicillin and erythromycin have been used(19,20).

Gingivitis:-
Purulent gingival pockets or gingival abscesses may complicate periodontal disease. Gingivitis results from accumulation of plaque and bacteria in the gingival crevice. Gingivitis is an inflammation of the gingiva, characterized by swelling, redness, change of normal contours, and bleeding(21,22). Swelling deepens the crevice between the gingivae and the teeth, forming gingival pockets. Although the patient usually experiences no pain, a mild foul smell may be noticed(23).Gingivitis may be acute or may be chronic with remissions and exacerbations(24).

The plaque accumulates in the small gaps between teeth, in the gingival grooves and in areas known as plaque traps: locations that serve to accumulate and maintain plaque. Examples of plaque traps include bulky and overhanging restorative margins, claps of removable partial dentures and calculus (tartar) that forms on teeth. Although these accumulations may be tiny, the bacteria in them produce chemicals, such as degradative enzymes, and toxins, such as lipopolysaccharide (LPS, otherwise known as endotoxin) or lipoteichoic acid (LTA), that promote an inflammatory response in the gum tissue.(25) This inflammation can cause an enlargement of the gingiva and subsequent formation.(26) Early plaque in health consists of a relatively simple bacterial community dominated by Gram-positive cocci and rods.

As plaque matures and gingivitis develops, the communities become increasingly complex with higher proportions of Gram-negative rods, fusiforms, filaments, spirilla and spirochetes. Later experimental gingivitis studies, using culture, provided more information regarding the specific bacterial species present in plaque(27,28)

Periodontitis

Subgingival plaque is associated with periodontal diseases. The bacteria that colonize the area are primarily anaerobic. Both Gram-negative and Gram-positive species are regularly isolated(16).Most of these bacteria utilize protein and other nutrients provided in the subgingival environment by the gingival fluid. Once established in the subgingival areas, periodontal infections usually drain into the oral cavity via a periodontal pocket(30). If the drainage of the periodontal pocket is obstructed, an acute process results. Abscess formation is usually limited to the alveolar process.

In some cases, spread to adjacent spaces may be noted(31). Focal or diffuse periodontal abscesses can develop. They appear as red fluctuant swelling of the gingiva or mucosa, which are tender. As the underlying tissues are affected, a complete destruction of the periodontium occurs, with subsequent loss of teeth(32,33).

Heterogenic subgingival flora has been found in chronic periodontitis but the bacteria most cultivated in elevated levels are P. gingivalis, T. forsythia, P. intermedia, C. rectus, Eikenella corrodens, F. nucleatum, A. actinomycetemcomitans, P. micros, Treponema denticola, and Eubacterium spp. Gramnegative anaerobes and capnophiles are dominant; spirochetes may be present. In the course of initiation and progression of the inflammatory process, the subgingival bacteria increase in numbers and invade the pocket epithelial cells and, subsequently, the underlying tissues(34). It has been proven that A. actinomycetemcomitans and P. gingivalis can invade the gingival tissues and this fact is distinctive for the more severe chronic periodontitis and aggressive periodontitis. Some recent data demonstrate some herpes viruses present in the periodontal pockets, e.g. Epstein-Barr virus-1 (EBV-1) and human cytomegalovirus (HCMV) (35,36).

The microbiota in aggressive periodontitis is dominated by Gram-negative capnophiles and anaerobic rods. In localized aggressive periodontitis, A. actinomycetemcomitans is constantly present; this microorganism may constitute up to 90 % of the cultivable microflora but essential levels of other microorganisms (Capnocytophaga, E. corrodens, P. gingivalis) have been found in periodontal pockets(37). In other forms of aggressive periodontitis, A. actinomycetemcomitans, P. gingivalis, P. intermedia, C. rectus are dominant. Herpesviruses, including Epstein-Barr virus-1 (EBV-1) and human cytomegalovirus (HCMV), can also be encountered. A. actinomycetemcomitans is defined as a species typical for localized juvenile periodontitis(38).

Pericoronitis

Pericoronitis is an infection of the pericoronal soft tissue associated with gum flaps (opercula) that partially overlie the crown of the tooth. Pericoronal infection is normally caused by a mixture of bacterial species present in the mouth, such as Streptococci and particularly various anaerobic species(39,40). This can result in abscess formation. Left untreated, the abscess can spontaneously drain into the mouth from beneath the operculum. In chronic pericoronitis, drainage may happen through an approximal sinus tract(41).The chronically inflamed soft tissues around the tooth may give few if any symptoms. The teeth most often involved are the third mandibular molars.

The infection is caused by microorganisms and debris that become entrapped in the gingival pocket between the tooth and the overlying soft tissue. If the overlying soft tissue becomes swollen, the drainage is obstructed and inflammatory exudate is entrapped and will spread to other anatomical sites.Pericoronitis is usually accompanied by swelling of the soft tissues and marked trismus. However, the underlying alveolar bone is not usually involved(42). In most cases, antibiotic treatment is necessary to avoid spread of the infection(42). The microorganisms most often isolated from acute pericoronitis are anaerobic cocci, Fusobacteria spp. and anaerobic gram negative bacilli(42).Treatment of pericoronitis also includes gentle debridement and irrigation under the tissue flap. Excision of the gum flap may be considered(43). Antibiotics and incision and drainage may be needed if fascial plains cellulitis develops(43).

Conclusion:-

An anaerobic infection depends on adequate and rapid management. Microorganisms that establish in the untreated root canal experience an environment of nutritional diversity. In contrast, well-filled root canal offers the microbial flora a small, dry, nutritionally limited space. Thus, we should obtain a better understanding of the characteristics and properties of bacteria and their biofilms along with the environmental changes, to enhance success.The main principles of managing anaerobic infections are neutralising the toxins produced by anaerobic bacteria, preventing the local proliferation of these organisms by altering the environment and preventing their dissemination and spread to healthy tissue.Toxin can be neutralised by specific antitoxins. Controlling the environment can be attained by draining the pus, surgical debriding of necrotic tissue, improving blood circulation, alleviating any obstruction and by improving tissue oxygenation. Therapy with hyperbaric oxygen (HBO) may also be useful. The main goal of antimicrobials is in restricting the local and systemic spread of the microorganisms.

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