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Review Article

Role of Oral Microbial Flora in Health and Illness

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ABSTRACT:

Oral microbial flora refers to the population of microorganisms that inhibit skin and mucous membrane of the oral cavity of the normal healthy persons. The microbial cells in the body outnumber the total number of cells in the human body by 10 folds. The mouth harbors a diverse, abundant and complex microbial community on varied surfaces of the oral cavity including both soft and hard tissues of the oral tissues as biofilms. Oral bacteria have evolved mechanisms to sense their environment and invade or modify the host. Bacteria occupy the ecological niche provided by both the tooth surfaces and gingival epithelium. The equilibrium is maintained by the innate host defense system constantly which monitors the bacterial colonization and prevents bacterial in invasion of local tissues. An understanding of the oral environment and microbial interactions leads to understanding the main causes for the onset of oral diseases. The present review highlights the role of microbial flora in health and oral diseases laying emphasis on its pathogenesis.

Key words: Human microbiome, Colonisation, Homeostasis, Adhesions, Anti-bacterial factors.

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INTRODUCTION

Mammals are complex gatherings of mammalian and bacterial cells structured into functional organs, tissues, and cellular communities.[1] Cell rich bacterial communities are more numerous than human cells in each person with a ratio of 10 bacterial cells to each human cell.[2] Human body contains a personalized set of foreign inhabitants essential in maintaining health, yet capable of eliciting a disease. "Human microbiome" is the term coined to describe microbial communities to characterize the extent & diversity of microbiome of human body.[3] Oral cavity being one of the routes of entry in the human body and having favourable physiochemical conditions like warm and moisture makes it an ideal place for growth of microbes.[4]The totality of these microorganisms, their genomes & ecosystems encompasses the microbiome.[5] The resident microflora plays a key role in the development of host and benefits themselves by the phenomenon known as commensalism.[6] These add on in the maintenance of health to the host defense and prevent colonization by exogenous microorganisms.[6] The mouth, similar to other environmentally-exposed sites have autochthotonous and diverse microflora in health.[7] The composition of this microflora includes bacteria, archaea, mycoplasma, virus, protozoa and fungi.[8] The composition of oral microbial flora differs from one person to another as well as one niche of oral cavity to another i.e. mucosal surface lips, tongue, cheek, teeth etc because of their biological features; supporting the growth of the same. The resident microflora is directly influenced by the environmental conditions prevailing in a particular habitat.[7] The key environmental factors affecting the growth of microorganisms in the healthy oral cavity is temperature 35-36^{o} celsius, Oxygen 0-21%, redox potential +200 to <-200Mv, pH -6.75-7.25, nutrients-endogenous and exogenous.[7]

On molecular basis streptococcus, actinomyces, veillonella, granulicatella, neisseria, heamophilus, cornyebacterium, rothia, provotella, capnocytophaga, porphyromonas and fusobacterium can be found.[9-12]

Although saliva lacks the drama of blood, sincerity of sweat and emotions of tears; but still it plays a key role in maintenance of homeostasis of the oral cavity and the growth of many microorganisms. The mouth is continuously bathed with saliva, and this has a profound influence on the ecology of the mouth.[13]The mean salivary pH of 6.75-7.25 favours the growth of micro-organisms and their ionic composition promotes its buffering properties and it's ability to remineralise enamel due to the presence of proteins and glycoproteins such as amylase, mucin, immunoglobulins.

In nutshell, oral health depends on a fine balance of the residential microbial species within biofilms at specific niches of the oral cavity. Factors such as change in diet, insufficient oral hygiene, salivary flow and change in host immune response disrupt homeostasis causing dental caries, periodontal diseases, endodontic infections, tooth loss. Systemic diseases including cardiovascular disease, stroke, preterm birth, diabetes and pneumonia are also believed to occur.[9,11,12,14] The use of culture-independent methods in determining the composition of the oral microbiome, along with next generation DNA sequencing is required to understand the oral-host microbiome interactions relevant to health and disease.[15] The present review highlights the role of microbial flora in health and oral diseases laying emphasis on its pathogenesis.

MOUTH AS A MICROBIAL HABITAT

The characteristics of mouth are ecologically different from all other surfaces of the body and control the types of microbes that are able to persist, so that not all of the microorganisms that enter the mouth are able to inhabit in it. The simple presence of the oral microbiota in the mouth inhibits colonization by pathogens, the phenomenon of colonization resistance.[16] The health of mouth is dependent on the integrity of the mucosa (and enamel) which acts as a physical barrier preventing penetration by microorganisms or antigens. Gingival crevicular fluid (GCF) and saliva act as defense factors in the oral cavity and play an important role in maintaining the integrity of the oral cavity.

Mouth is not a homogenous environment for microbial colonisation.[6] The components of saliva influence the oral micro flora by

a. adsorbing to oral surface, especially teeth, to form a conditioning film (the acquired pellicle) to which microorganisms attach. Adherence involves specific intermolecular interactions between adhesins on the surface of the microorganism and receptors in the acquired pellicle Within the oral cavity, there are two types of surfaces for which bacteria can colonize; the hard surfaces of teeth and the soft tissue of the oral mucosa. Different types of microorganisms prefer distinct niches according to varying surface structures and functions i.e. they exhibit tissue tropism. Each niche provides optimal environment for populating certain microbes. The maxilla, hard palate, soft palate, and even the tongue's lateral sides and dorsal side each have a different bacterial profile.[17,18]
b. Acting as primary sources of nutrients (carbohydrates and proteins) which foster the growth of resident microflora without inducing a damaging pH fall.[19,20]
c. Binding to the surface of bacteria to mask bacterial antigens, thereby making the organism to appear more hostlike.
d. Aggregating microorganisms and thereby facilitating their clearance from the mouth by swallowing; the flow of saliva will also wash away weakly-adherent cells.
e. Inhibiting the attachment and growth of some exogenous microorganisms, via their role as components of the host defenses.
f. Saliva contains several anti-bacterial factors including sIgA which can reduce or prevent microbial colonisation of oral surfaces. Antimicrobial peptides are present, including histidine-rich polypeptides (histatins), and cystatins, which may control the levels of yeasts, and a range of active proteins and glycoproteins (lysozyme, lactoferrin, sialoperoxidase). Serum components can reach the mouth via GCF.[21]

The eruption of teeth also generates another habitat, the gingival crevice (where the tooth emerges from the gums), and an additional major nutrient source for that site (GCF). Teeth allow the accumulation of large masses of microorganisms (predominantly bacteria) and their extracellular products (collectively, this is termed dental plaque), especially at stagnant or retentive sites. In contrast, elsewhere in the body, desquamation ensures that the microbial load is relatively light on most mucosal surfaces.

The flow of GCF is relatively slow at healthy sites, but increases during the inflammatory responses associated with periodontal diseases. IgG is the predominant immunoglobulin in GCF; IgM and IgA are also present, as is complement. GCF contains large numbers of viable neutrophils, as well as a minor number of lymphocytes and monocytes influencing the

ecology of the site by:
(a) removing weakly-adherent microbial cells,
(b) introducing additional components of the host defenses
(c) acting as a novel source of nutrients for the resident microorganisms. The growth of several fastidious obligate anaerobes is dependent on haemin, which can be derived from proteolysis of haemoglobin, haemopexin, and transferrin, etc. **Table 1** shows the oral microbial flora in a healthy oral cavity

Table 1: Principal bacterial genera found in the healthy oral cavity[7]

Gram-positive	Gram-negative
Cocci	**Cocci**
Abiotrophia	Moraxella
Peptostreptococcus	Neisseria
Streptococcus	Veillonella
Stomatococcus	
Rods	**Rods**
Actinomyces	Campylobacter
Bifidobacterium	Capnocytophaga
Cornyebacterium	Desulfobacter
Eubacterium	Desulfovibrio
Lactobacillus	Eiknella
Propionibacterium	Fusobacterium
Pseudoramibacter	Haemophilus
Rothia	Leptotrichia
	Prevotella
	Selenomonas
	Simonsiella
	Treponema

Mechanism by which oral bacteria evade the host defenses during health

- Antigen masking- oral bacteria bind host molecules to their Antigen masking surface ('stealth technology').
- Molecular mimcry -bacterial epitopes resemble those of the host
- Enzyme degradation pioneer streptococci produce IgA1 protease; other bacteria produce general proteases that cleave other Ig's and host defense factors.
- Immune suppression or immune indifference some species are immuno-modulatory, or produce factors that instruct the host defenses to recognize them as 'self' immune ('Commensal communism').
- Antigenic variation: constant subtle changes; may explain clonal turnover
- Unfavourable environment local conditions may be unsuitable for the optimal functioning of the host defenses.

Table 2: Main oral conditions where infective agents are directly implicated [22,23]

Condition	Associated bacterial species
Dental caries	Streptococcus mutans Lactobacillus spp Actinomyces viscosus
Periodontal disease	
Chronic gingivitis	Streptococcus sanguis Streptococcus milleri Actinomyces israelii Actinomyces naeslundii Prevotella intermedia Capnocytophaga spp Fusobacterium nucleatum Veillonella spp
Chronic periodontitis	Porphyromonas gingivalis Prevotella intermedia Fusobacterium nucleatum Tannerella forsythia Aggregatibacter actinomycetemcomitans Selenomonas spp Capnocytophaga spp Spirochaetes
Aggressive periodontitis	Aggregatibacter actinomycetemcomitants Capnocytophaga spp Porphyromonas gingivalis Prevotella intermedia Tannerella forsythia
Necrotizing ulcerative gingivitis	Fusobacterium nucleatum Treponema spp
Dentoalveolar infections Dentoalveolar abscess	Prevotella spp Porphyromonas gingivalis Fusobacterium nucleatum Streptococcus milleri
Ludwig's angina	Porphyromonas spp Prevotella spp, Fusobacterium spp Streptococcus spp
Periodontal abscess	Porphyromonas spp Prevotella spp Fusobacterium spp Streptococcus spp (especially anaerobic) Capnocytophaga spp Actinomyces spp
Osteomyelitis	Tannerella spp Porphyromonas spp Prevotella spp Enterobacteria
Actinomycosis	Actinomyces israelii Actinomyces bovis Actinomyces naeslundii Aggregatibacter actinomycetemcomitans
Salivary gland infections Suppurative parotitis	Alpha haemolytic streptococci Staphylococcus aureus

Once a pathogen possesses virulence factors, exists in abnormal proportions and demonstrates parasitism, all of the following conditions which are required for disease have been satisfied i.e the local environment in which the species can express its virulence properties, the pathogen is in numbers that exceed the threshold for that host; other bacterial species can foster, or at least not inhibit, the diseases manifestation; and the host is susceptible to this pathogen, i.e. currently compromised immune system or specific genetic composition.[24]

The physiological characteristics of the oral flora, from energy production for survival to pathogenicity are determined by the metabolic activity they exhibit. Understanding the range of metabolic characteristics of the microbial ecosystem could be an answer to this question, and would also provide us with an answer to the question of how we could control the microbial ecosystem; in other words, how we could control oral diseases such as dental caries, periodontal diseases and oral malodor.[25] Dental diseases are caused by polymicrobial biofilms and with the progression of the infection, anaerobes become dominant. If prompt and proper treatment is not undertaken, it may lead to tooth loss and systemic infection.[26] Changes in microbial and environmental dynamics in microbial ecosystems may increase the potential pathogenicity within a microbial ecosystem and subsequently initiate and promote oral disease. These successional changes have recently and tentatively been referred to by Marsh as the ecological plaque hypothesis.[27]

DENTAL PLAQUE

Communication is a crucial part in successful organizations. Communication between oral microorganisms is essential for initial colonization and subsequent biofilm formation on the enamel surfaces of teeth and necessitates physical contact between colonizing bacteria and between the bacteria and their host.[28] Retention of bacteria to tooth surface prevents it from being swallowed by saliva. Through retention, these bacteria can form organized, intimate, multispecies communities referred to as dental plaque.[29] Dental plaque is structurally and functionally organized biofilm which adheres resolutely to tooth surfaces as well as restorations and prosthetic appliances. It is a multi-species biofilm comprising of hundreds of bacterial species, salivary polymers, and bacterial extracellular products. The microbial species colonize the teeth, hard palate, tongue, carious lesions, oral mucosa, and periodontal pockets [30.]

The distribution of microbial species in these plaque biofilms varies depending on the anatomical locations and environmental factors.[31,32] Dental plaque is classified into supra-gingival and sub-gingival plaques, and both of them have significant contributions to dental and periodontal diseases.[30]

The predominant microorganisms of supragingival plaque are Gram-positive facultative anaerobic bacteria particularly Actinomyces species, Streptococci and Capnocytophaga species. Gram negative species include Veillonella species, Prevotella species as well as Porphyromonas gingivalis and Tannerella forsythia. Whereas the subgingival plaque comprises the following species, Streptococci, Prevotella denticola, Porphyromonas endodontalis, and Porphyromonas gingivalis.[33] Bacteria in biofilms come across much higher local cell densities than free-floating, planktonic cell populations .[34]

DENTAL CARIES

Dental caries is a chronic disease that progresses slowly in most individuals and is characterized by localized destruction of the tooth following long contact/interaction with acidic products that result from the bacterial fermentation of dietary carbohydrates. [17] Cariogenic bacteria primarily form a biofilm on the tooth surface and an anaerobic environment develops as oxygen is quickly used up. Consequently, bacteria begin anaerobic metabolism and produce low-molecular weight organic acids (of which lactate is the most important) by breaking down carbohydrates that cause tooth decay when pH in the biofilm falls below 5.5, the critical pH. Dental caries is a very good example of how an alteration in the microflora can cause a disease. The normal microflora usually consists of nonmutans streptococci like the salivarius group (e.g. Streptococcus salivarius) on the root surface, mitis group (e.g. S. sanguis) in the pit and fissures and also a small number of microbes of the mutans group (not enough to induce caries). [15] A situation may arise wherein the microbial ecosystem is disturbed and may result in increase in the number of caries inducing organisms like Streptococcus mutans and Lactobacillus species. It is possible that members of the healthy microbial flora have the potential to change the environment through physiological processes such as metabolic activities, subsequently facilitating the introduction of more pathogenic microorganisms like mutans streptococci.[14] Nonmutans Streptococcus and Actinomyces are present in the supragingival area where there is a continuous supply of nutrients by the saliva and also by carbohydrates derived from food. As the supragingival plaque accumulates or if there is an increased supply of carbohydrates, an acidic and anaerobic environment is created. The normal flora adapts to this change in the environment and can become aciduric.[30]

PERIODONTAL DISEASE

Periodontal disease is a pathological inflammatory condition of the gum and bone support (periodontal tissues) surrounding the teeth. The two most common

periodontal diseases are: **gingivitis** and **periodontitis.**

Gingivitis-Most children have signs of some inflammation of the gingival tissue at the necks of the teeth; among adults, the initial stage of gum disease is prevalent. This condition is termed gingivitis and is characterized by redness of the gum margins, swelling and bleeding on brushing. Gingivitis occurs in both chronic and acute forms. Acute gingivitis is usually associated with specific infections, micro-organisms, or trauma. Chronic inflammation of the gum tissue surrounding the teeth is associated with the bacterial biofilm (plaque) that covers the teeth and gums.[1,2] Gingivitis was once seen as the first stage in a chronic degenerative process which resulted in the loss of both gum and bone tissue surrounding the teeth. It is now recognized that gingivitis can be reversed by effective personal oral hygiene practices.

Periodontitis-When periodontal disease affects the bone and supporting tissue, it is termed periodontitis and is characterized by the formation of pockets or spaces between the tooth and gums.[16] This may progress and cause chronic periodontal destruction leading to loosening or loss of teeth. The dynamics of the disease are such that the individual can experience episodes of rapid periodontal disease activity in a relatively short period of time, followed by periods of remission.[19]

Though the majority of adults are affected by gingivitis, gingivitis fortunately does not always develop into periodontal disease. Progression of gum disease is influenced by a number of factors which include oral hygiene and genetic predisposition. One of the challenges for early detection of periodontal disease is its "silent" nature – the disease does not cause pain and can progress unnoticed. In its early stages, bleeding gums during tooth brushing may be the only sign; as the disease advances and the gums deteriorate, the bleeding may stop and there may be no further obvious sign until the teeth start to feel loose.[28]

The subgingival crevice is flooded with gingival crevicular fluid (GCF) which creates a neutral/alkaline environment due to the presence of nitrogenous compounds, such as amino acids, peptides and proteins. As the gingival sulcus deepens, this environment is established and under these conditions, asaccharolytic and anaerobic and/or proteolytic bacteria, such as Fusobacterium, Eubacterium, Campylobacter, Prevotella and Porphyromonas are found. Proteolytic bacteria can degrade nitrogenous compounds into small peptides and amino acids by cell membrane-bound and/ or extracellularly secreted proteases, for subsequent use as metabolic substrates. [11] However, these enzymes secreted by the micro-organisms for degrading the nitrogenous compounds, induce inflammation and immunoreactions.[30]

ENDODONTIC INFECTIONS

The root canal system is, in its healthy and intact state, free from infection. Unlike the oral cavity, the root canal system has no commensal microbiota, and any microorganism detected here can be regarded as a potential pathogen.[14] Microorganisms play an unequivocal role in infecting root canal system. Endodontic infections are different from other oral infections in the fact that they occur in an environment which is closed to begin with since the root canal system is an enclosed one, surrounded by hard tissues all around. Most of the diseases of dental pulp and periradicular tissues are associated with microorganisms. Endodontic infections occur and progress when the root canal system gets exposed to the oral environment by one reason or the other and simultaneously when there is fall in the body's immune response. [16] To begin with, the microbes are confined to the intra-radicular region when the ingress is from a carious lesion or a traumatic injury to the coronal tooth structure. However, the issue if not taken care of, ultimately leads to the egress of pathogens and their by-products from the apical foramen to the periradicular tissues.

An infected root canal system is a prerequisite for the formation of apical periodontitis.[4] The microorganisms that contribute the most to endodontic infections are bacteria, both in biomass and diversity. Although Lactobacillus, Actinomyces, and Streptococcus are among the most abundant genera detected in root canals associated with apical periodontitis, regarding the aseptic nature of the root canal system in its healthy and intact state, any bacteria present can be regarded as an endodontic pathogen.[26] In total, bacteria detected from the oral cavity fall into 13 separate phyla, namely, Firmicutes, Bacteroidetes, Actinobacteria, Proteobacteria, Spirochaetes, Fusobacteria, Synergistes, SR1, TM7, Chloroflexi, Deinococcus, Acidobacteria, and Cyanobacteria.[11]

Primary intraradicular infection refers to the infection of the root canal system from the microbes which have entered the exposed pulp tissue from the oral cavity and further get colonized.[15] This occurs as a result of carious or traumatic exposure of coronal pulp or any other breach in the hard tissue integrity of the tooth structure. Secondary intraradicular infection refers to the infection which occurs inside the root canal system after the treatment of the affected tooth has been initiated .[22] This usually happens due to introduction of microbes into the root canal system during endodontic therapy especially in cases where the tooth is left open for one reason or the other, leakage from temporary fillings during inter-appointment periods, coronal leakage from defective permanent restoration etc.

DENTOALVEOLAR ABSCESS

Dental or dentoalveolar abscess is a denomination used to describe localized collection of pus in the alveolar bone at the root apex of the tooth. It usually occurs secondary to dental caries, trauma, deep fillings or failed root canal treatment. [25] Once the intact pulp chamber is breached, colonization of the root canals occurs with a diverse mix of bacteriological agents. These microorganisms are capable of forming biofilms in root canals, hence making application of the "biofilm concept" plausible in such infections.[19] After entering the periapical tissues via the apical foramen, these bacteriae are capable of inducing acute inflammation leading to pus formation. The pathogenesis of dentoalveolar abscess is polymicrobial in nature, comprising of various facultative anaerobes, such as the viridans group streptococci and Streptococcus anginosus group, and strict anaerobes, especially anaerobic cocci, Prevotella and Fusobacterium species. [25] If not treated at an early stage it may rapidly evolve and spread to adjacent anatomic structures, leading to serious complications such as septicemia, cavernous sinus thrombosis, brain abscess, shock, and occasionally to death. Depending upon the recovery and cultural conditions, strict anaerobes outnumber facultative by a ratio which varies between 1.5 and 3:1 in mixed infections. [21] The most commonly isolated genera include anaerobic streptococci, Fusobacterium species and the black-pigmented anaerobes such as Prevotella and Porphyromonas species. Prevotella species have been reported as the most frequent isolates in numerous studies, found in 10-87% of dentoalveolar abscesses. Prevotella intermedia, Prevotella nigrescen, Prevotella pallens, Porphyromonas endodontalis and Porphyromonas gingivalis are the commonly detected pathogens.

Bacteroides fragilis, a more common isolate from intra-abdominal infections, has only infrequently been reported from acute dentoalveolar infections and is not regarded as an oral commensal.[27] The member of the Bacteroides genus most likely to be recovered from an acute dental abscess is Bacteroides forsythus (now transferred to a new genus as Tannerella forsythia).

Fusobacterium periodonticum and Fusobacterium nucleatum (which includes subsp. nucleatum, subsp. polymorphum, subsp. animalis, subsp. vincentii, and subsp. fusiforme) are frequently detected with F. nucleatum; recovered most frequently from acute dental abscess. [30] Facultative anaerobes belong to the viridans group streptococci and the anginosus group streptococci are commonly implicated in dental abscess. The viridans group streptococci includes mitis group, oralis group, salivarius group, sanguinis group, and the mutans group. The signs and symptoms of the acute dental abscess are pain, swelling, and erythema usually localized to the affected tooth, although the suppuration can frequently spread to the nearby tissues causing fatal complications. Fever, extraoral and intraoral swelling, erythema, tenderness to palpation are notable.[8] Trismus in addition to any changes in the voice such as hoarseness and drooling should prompt the dentist to an emergency situation. The clinical examination should focus on the general status of the patient such as lethargy or extreme sickness. Deep neck and descending necrotizing mediastinal abscesses are a rare complication of the dental abscess and spread of odontogenic infections accounts for a large number of deep neck abscesses.[6]

DRY SOCKET

Alveolar osteitis or dry socket is one of the most common postextraction complication occurring 2 to 4 days postsurgery. A localized fibrinolysis (resulting from conversion of plasminogen to plasmin, which dissolves fibrin crosslinks) occurring within the socket and subsequently leading to loss of blood clot is believed to underlie the pathogenesis of alveolar osteitis.[18] There is an increased incidence of dry socket being reported in patients with poor oral hygiene, higher pre and postoperative microbial counts, pericoronitis and periapical infection. Hence, bacteria are suspected of causing dry socket.28. Nitzan et al (1983) proposed, in particular, the role of anaerobic bacteria, especially Treponemma denticola, which showed plasmin like fibrinolytic activity in vitro.[19] A series of bacteria, which included Enterococcus, Streptococcus viridians, Bacillus coryneform, Proteus vulgaris, Pseudomonas aeruginosa, Citro bacterfreundii, and Escherichia coli were identified in the biologic material within the alveolus in experimental dry socket models.[4] Also, in accordance with the potential role of bacteria in dry socket development, the inoculation of Actinomyces viscosus and Streptococcus mutans in animal sockets was reported to delay the sequence of alveolar repair.

OSTEOMYELITIS

Osteomyelitis can be broken down into "osteo" meaning bone, and "myelitis", meaning inflammation of the fatty tissues within the bone. Osteomyelitis is caused by an infection of the bone or joint, and can be both acute and chronic. It can occur at any age and involve any bone.[3] These infections can be due to one, or many types of bacteria and/or fungi. In order of frequency, osteomyelitis can be the result of a trauma, surgery, or joint insertion or any type of prosthetic material; it can be due to lack of blood flow in diabetes associated foot infections, or it can be the result of an infection that has spread via the blood and has reached the bone (seen mostly in prepubescent children or the elderly).[25]

Osteomyelitis is an infection frequently caused by Staphylococcus bacteria. While some cases of osteomyelitis are of unknown causes, the infection is usually transmitted through the bloodstream from one area of the body to another (Hematogenous osteomyelitis). These blood infections are commonly due to Staphylococcus aureus, Streptococcus species, and aerobic Gram-negative bacilli. If the patient has a compromised immune system, M tuberculosis, Brucella species and fungi should be included as possible causing agents in the disease work-up. [16]

Initially there may be several days of fever, pain at the site of infection, and a generalized feeling of ill health (malaise). This may be followed by an increase in fever (104-105 degrees Fahrenheit), deep localized bone pain, chills, sweating, swelling and painful or limited movement of the nearby joints. The skin near the affected bone may be red (erythema) and there may be a purulent buildup (pus), loss of calcium, destruction of the surrounding tissue (necrosis) and bone deterioration or deformity. However, patients with osteomyelitis involving the hip, vertebrae, and/or pelvis are less likely to present with many signs other than pain. In long bone infections, if the infection spreads from the metaphysis through the bone cortex and within the joint capsular reflection of knee, any discharge of pus into the joint can present as septic arthritis secondary to osteomyelitis.[21] These joints include the knees, wrists hip, ankles, symphysis pubis, and shoulders.

Chronic osteomyelitis usually occurs after an acute episode of osteomyelitis when the infection has not been totally cured, and is sometimes associated with a draining sinus tract. There may be bone pain, swelling, redness and tenderness of the affected area. A discharge of pus from an opening to the infected bone is often the first symptom. [24] There may also be destruction of the bone with pieces of the infected bone separating from the healthy bone. When this occurs, surgery to remove the bone fragments may be necessary.[6]

Spinal infections, referring to vertebral osteomyelitis, are most commonly distributed or spread by way of the bloodstream, or present as post-surgical complications. These spinal infections are often characterized by chronic back pain not relieved by ordinary treatment, including bed rest, heat or pain relievers. There may be fever, localized tenderness, pain, muscle spasms and limited movement. Any patients with known fever, weight loss, bacteremia and/or endocarditis should be sent for spinal imaging if they are experiencing new or worsening back pain. This form of osteomyelitis usually affects people over 50 years of age, and is usually caused by a previous injury, urinary tract infection.[8]

Anaerobic osteomyelitis often affects the lower jawbone (mandible), skull or feet. It is characterized by ulceration and swelling, foul smelling drainage and redness of the affected area.

Diabetic foot infections can progress into osteomyelitis as a result blood vessel insufficiency. These infections usually occur following skin ulcerations in patients with nerve damage (neuropathy).[17] This phenomenon is more common in people with diabetes mellitus or vascular diseases affecting the extremities, especially the toes and small bones of the feet.[1] It is usually seen in people over 50 years old and is characterized by pain and redness of the affected area (erythema), swelling, ulcerations, and drainage of pus.[18] This type of osteomyelitis is difficult to treat because of the underlying vascular disorder that can impair the therapeutic effect of antibiotic treatment.[22]

CONCLUSION

Oral cavity is colonized by a wide range of bacteria. Oral microbial flora, beyond doubt, has a very important role to play in maintenance of homeostasis of the ecosystem in the oral cavity. The bacteria are beneficial to the host but when the composition of the biofilm is changed because of disrupted homeostasis, it may result in caries or periodontal disease. Traditional as well as novel techniques could be used for the detection. New material has been added including advances in molecular biology pertaining to infectious disease, bacterial taxonomy and nomenclature, uncultivable bacteria, biofilms, emerging infections including prion diseases, drug-resistant bacteria and the latest US and UK recommendations for infectious control procedures.

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