DENTAL CARIES: ETIOLOGY, PATHOGENESIS, DIAGNOSIS AND MANAGEMENT

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Abstract

Dental caries is also called as dental decay or cavities in tooth. It occurs from acids produced by bacteria by metabolizing carbohydrates. Initially it appears as white spot lesion which converted into gross cavity if proper intervention is not provided. Dental caries can be diagnosed clinically by visual tactile method of examination. There are various diagnostic aids available for its diagnosis such as radiographic examination using OPG, IOPA, bitewing X-ray, dyes, advanced methods like Fiber-optic transillumination, Wavelength dependent FOTI, Digital imaging FOTI etc. There are various factors contribute for the occurrence of dental caries such as high oral bacterial counts, frequent consumption of sugar-containing foods, inadequate fluoride exposure, low socioeconomic status, xerostomia due to medications or disease and physical disabilities which causes impaired oral hygiene. This paper will discuss about etiology, pathogenesis, diagnosis and management of dental caries.

Keywords: Dental caries, bacteria, microbes, oral cavity, diagnosis.

Introduction

The human body makes residence for trillions of microbes and oral cavity is one of the biggest sources of microbes. Around 700 to 1000 species of microbes colonize the human oral cavity. There is close association between oral microbes and occurrence of oral diseases like dental caries, periodontal and gingival diseases and oral cancers.[1-3]

The term 'Dental Caries' was first time reported in literature around 1634. It came from Latin word 'caries' which means decay. In the starting, it was used to denote holes in teeth. It is observed as one of the oldest and most prevalent disease occurring in humans. It is chronic infectious disease which results from tooth adherent cariogenic bacteria. These bacteria produce acids by metabolizing sugars and these acids results in demineralization of tooth structure.[4]

Caries and periodontal diseases are the main oral health problems in developing countries.[5]Although improved oral health is seen in most of the developed countries, but there are still people with dental problems usually belonging to low socioeconomic status.[6] These oral problems are prevalent in all ages and demographic and socioeconomic groups. Dental caries is most prevalent in Latin America, South Asia, and the Middle East and least common in China.[7] Dental caries occurrence increase with age due to use of denture and poor oral hygiene. Oral diseases such as tooth loss, dental caries, oral mucosal and periodontal diseases, and HIV/AIDS-related oral diseases are the main public health problems in the world. Out of total 291 diseases and injuries evaluated in global burden of disease, untreated tooth decay has the highest rate of prevalence between 70 and 90% of populations, and it is also one of the most common reasons for tooth extraction.[8]

Irrespective of geographic location in the world, both genders are affected from the condition. World health organization observed that 60 to 90% of school children and approximately 100% of adults across the world are affected from dental decay. So the prevention of dental caries plays a prime role in public health management. Federation Dentaire Internationale (FDI) gave the minimal intervention dentistry definition for management of dental caries in year 2002, and emphasized that the existing preventive measure is to maintain a healthy tooth structure as much as possible. Recently it is recommended to detect and monitor caries at early stage, instead of waiting until a cavity is formed. Thus the prevention of dental caries was shifted from the surgical model to a medical model, and the number of individuals getting preventive oral health care has been increasing in recent years. Prevention of dental caries preserves a sound tooth structure, prevents the demineralization of enamel, and promotes natural healing processes.[9]

Etiology of caries

Dental caries is a chronic, multifactorial dental disease which leads to damage and demineralization of hard tooth structure due to acid produced by bacteria.[10]

Theories of dental caries

Early Theories of Caries Etiology

The legend of the worm theory: Ancient Sumerian Text Known as "the legend of the worm" found 5000 BC

Endogenous Theories

Humoral theory - According to this theory, when there is an imbalance between the humors of body, it results in tooth decay.

Vital theory- According to this theory tooth decay originated from within the tooth itself, like a bone gangrene.

Exogenous Theories

Chemical Theory: Parmly proposed that an unidentified "chymical agent" was responsible for caries. According to this theory, teeth are destroyed by the acids formed in the oral cavity by the putrefaction of protein which produced ammonia and was subsequently oxidized

to nitric acid. Robertson also stated that tooth decay is produced by the fermentation of food particles around the teeth.

Parasitic theory: This was the first theory which gives relation ***between micro-*** organisms and caries. It was proposed that even though caries starts purely as a chemical process, but microorganisms continued the disintegration in both enamel and dentin.

Chemicoparasitic theory (1890) (Acidogenic Theory): It explains-microbiological basis of dental caries and proposed in 1890 by W. D. Miller in his book titled "The microorganisms of the human mouth" based upon the work done in Robert Koch's laboratory in Berlin. This theory showed that the degradation of carbohydrate-containing foods resulted in acid formation and was able to demonstrate this process in vitro with isolated oral bacteria and extracted teeth.

Miller summarized his theory as: Dental decay is a chemoparasitic process consisting of two stages that are decalcification or softening of the tissue and dissolution of the softened residue.

Miller advocated an essential role of three factors in the caries process:

- The oral microorganisms
- The carbohydrate substrate
- The acid.

This theory is still considered as the backbone of current knowledge and understanding of the etiology of dental caries.

The proteolytic theory: This theory was given by Gottlieb and Gottlieb 1944.This theory stated that the organic or protein components of tooth are the initial pathways of invasion by microorganisms and, dental caries is essentially a proteolytic process. In this process the microorganisms enter the organic pathways and damage them while advancing through them by forming acids.So certain structures of enamel having high organic material composition, like enamel lamellae and enamel rod sheaths, could serve as a pathway for microorganism invasion through the enamel.

Proteolysis chelation theory: The acidogenic and the proteolytic theory were addressed in this proteolysis chelation theory. This theory was given by Schwartz and his co-workers 1955. Chelation is a process in which there is complexing of the metal ions to form complex substance through coordinate covalent bond which results in formation of poorly dissociated /or weakly ionized compound.[11-14]

Contributing factors for dental caries

1. Host factor

a. Tooth factor

- Morphology and position in the arch
- Chemical nature

b. Saliva

- Composition, pH and antibacterial activity
- Quantity and viscosity of flow

2. Microflora

3. Substrate or diet (physical nature and chemical nature)

4. Time.

Host Factor

a. Tooth Factor - Morphology and position in arch

Deep pits and fissures are more prone to carious attack as compared to smooth surfaces of teeth because of food accumulation and bacterial stagnation occurs more in these areas. The permanent mandibular first molars due to having more complex occlusal morphology with numerous pits and fissures are most prone to dental decay followed by the maxillary first molars and mandibular and maxillary second molars.

The position of the tooth in the dental arch also plays an important role in the incidence of carious lesions. Irregularities in the arch form, crowding and overlapping of the teeth also favor the development of caries as these areas provide an excellent environment for plaque accumulation.

Partially impacted third molars are also more prone to dental caries.

Chemical nature

The chemical elements of enamel such as dicalcium phosphate dihydrate and fluorapatite make the enamel resistant to caries to a certain extent. The presence of mineral ions such as Ca, F, Zn and Fe in more concentrations decreases the enamel solubility, hence decreasing chances of dental decay. Higher the solubility of the enamel surface leads to higher susceptibility to caries development. The mineral content of enamel tends to increase with advancing age, so increased resistance to carious attack.

b. Saliva -Composition

The concentrations of inorganic calcium and phosphorus show alterations within resting and stimulated saliva. Individuals, who are more prone to caries, have low calcium and phosphorus levels. Salivary proteins such as statherin, acidic proline-rich proteins, cystatins, and histatins help in the maintaining the homeostasis of the supersaturated state of saliva. According to Hay and Moreno (1989), statherin is present in stimulated saliva in concentrations sufficient to inhibit the precipitation of calcium and phosphate salts. Studies by Gibbons and Hay (1988) have shown that statherin may contribute to the initial colonization of the tooth surfaces by certain bacteria.

The acidic PRPs bind free calcium, adsorb to hydroxyapatite surfaces, inhibit enamel crystal growth, and regulate hydroxyapatite crystal structure (Hay and Moreno, 1989).

The amount and quality of acidic PRPs and agglutinins are observed to be variable in caries-free and caries-active individuals as shown by the studies of Rosan et al (1982) and Stenudd (1999). The role of cystatins in the caries process is not clear. However, they may play a minor role in the regulation of calcium homeostasis in saliva. Phosphorylated and non-phosphorylated cystatins bind to hydroxyapatite.

Salivary flow rate, pH and buffer capacity

Saliva has the most important function of caries prevention by way of its flushing and neutralizing effects, commonly referred to as 'salivary clearance' or 'oral clearance capacity'. As a thumb rule, the higher the flow rate, the faster the clearance and the higher the buffer capacity. Decreased salivary flow rate leading to reduction of oral defense systems which may cause severe caries and mucosal inflammation.

The pH of saliva at which it ceases to be saturated with calcium and phosphorus is referred to as the 'critical pH'. Normally, the critical pH is 5.5. Below this value, the inorganic content tends to demineralize. The normal pH of resting saliva is 6–7.

Buffering capacity

The buffer capacity of both unstimulated and stimulated saliva consists three main buffer systems: the bicarbonate (HCO−3), the phosphate, and the protein buffer systems. These systems have different pH ranges. The bicarbonate and phosphate systems have pH values of 6.1–6.3 and 6.8–7.0, respectively.

Since most of the salivary buffering capacity operative during food intake and mastication is due to the bicarbonate system, sufficient saliva flow provides the oral cavity with the neutralizing components.The phosphate and protein buffer systems make a minor contribution to the total salivary buffer capacity, relative to the bicarbonate system. The phosphate system is, in principle, analogs to the bicarbonate system but without the important phase-buffering ca-

pacity, and it is relatively independent of the salivary secretion rate.
It is a well-established fact that the buffer capacity of the saliva and the caries experience are inversely related.
The buffer effect of saliva is influenced by the hormonal and metabolic changes, as well as by altered general health. It is generally accepted that the buffer effect is greater in men as compared to women. In women, the buffer effect decreases gradually, independent of flow rate, toward late pregnancy and promptly recovers after delivery. Introduction of either hormone replacement therapy in menopausal women or low-dose oral contraceptives can slightly increase the buffer capacity.

Antibacterial activity

The primary oral innate defense factors are peroxidase systems, lysozyme, lactoferrin, and histatins. In vitro studies have proved that these proteins are known to limit bacterial or fungal growth, interfere with bacterial glucose uptake or glucose metabolism and promote aggregation and, thus eliminate bacteria.
The immunoglobulins, IgG, IgM, IgA, and secretory IgA (sIgA), form the basis of the specific salivary defense against oral microorganisms, including Streptococcus mutans. The most abundantly found immunoglobulin in saliva is dimeric slgA, which is produced by plasma cells present in the salivary glands. Two IgA subclasses are present in saliva; IgAl forms the major component of immunoglobulins, although the relative amount of IgA2 is higher in saliva than in other secretions.Salivary IgA is absent at birth but is generally detectable by the age of 1 week.
In human beings, IgG, mainly of maternal origin, is the only detectable immunoglobulin in the saliva of neonates. Its concentration reduces to non-detectable levels after some months but appears again after tooth eruption.

Quantity and viscosity of flow

The viscosity of the saliva and the amount of saliva produced has a significant impact on the incidence of dental caries.The average person produces at least 500 ml of saliva over a period of 24 hours. The unstimulated flow rate of saliva is 0.3 ml/min, whereas the flow rate during sleep is 0.1 ml/min and during eating or chewing, it increases to 4.0 to 5.0 ml/min. Any reduction in this quantity of saliva as seen in diseases such as Sjögren's syndrome, diabetes, etc. predisposes to dental caries.
Increased viscosity of saliva may prevent its natural cleansing action thereby promoting the deposition of plaque on the tooth surface. Likewise when the salivary viscosity is low, the amount of minerals and bicarbonates are inadequate thereby limiting its anticaries activity.

2. Microflora

The main etiological agent in occlusal and pit and fissure caries is the S. mutans. Deep dentinal caries is commonly associated with lactobacilli, certain gram-positive anaerobes and filaments such as Eubacterium and Actinomyces.Root caries or cemental caries is predominantly associated with Actinomyces viscosus. However other species of Actinomyces such as A. naeslundii and A. nocardia have also been isolated.

3. Substrate and Dietary Factors

The role of diet in the causation of dental caries has been extensively studied.
Various dietary factors have been implicated in the occurrence of dental caries.

Physical nature of diet

It is considered that coarse and fibrous food helps in cleansing the debris from the tooth surface thereby minimizing the incidence of carious lesions. However, refined and sticky starchy food aid in the formation of dental caries.

Chemical nature of diet

It is a well-known fact that food with high refined carbohydrate content has the greatest potential to produce carious lesions.The type of carbohydrate (monosaccharide, disaccharide or polysaccharide), frequency of intake and the time for which the ingested food remains stagnant in the oral cavity or on the tooth surface determine the incidence and severity of the carious lesions.
It is believed that vitamin B deficient individuals have lower incidence of dental caries as this is essential for the growth of oral acidogenic flora and also serves as a component of coenzymes involved in glycolysis.
Vitamin D has an important role in the normal development of teeth. Its deficiency results in hypoplastic teeth which usually have higher incidence of dental caries.
Fluoride content in the diet has no significant role because of its metabolic unavailability. Therefore, the fluoride content in cooking salt and its effect on reducing the incidence of carious lesions is still questionable. However, fluoridated water minimizes the caries incidence.Phosphates, molybdenum and vanadium in the diet helps in minimizing the incidence of carious lesions.

Role of heredity

There are various studies which assessed the genetic modifications in dental enamel, genetic modification of immune response, genetic regulation of salivary function and inherited alterations in sugar metabolism.
It was concluded that heredity plays a subsidiary part in the incidence of caries. It is believed that heredity affects the dental decay only in as much as it controls the shape of a tooth and its pits and fissures and its position in the dental arch.
Senpuku et al (1998) and Acton et al (1999) have correlated specific HLA-DR types with binding S. mutans antigens and S. mutans colonization.
Acton concluded that 'genes within MHC modulate the level of oral cariogenic organisms'.
Mariani et al (1994) in their study of celiac disease, enamel defects and HLA typing observed that HLA-DR3 was associated with increased enamel defects and HLA-DR5, 7 were associated with a reduced frequency of enamel defects. Studies have shown that the genes in the HLA complex are associated with altered enamel development and increased susceptibility to dental caries.

Role of immunity

Salivary IgA and immunoglobulins secreted in the gingival crevicular fluid such as IgG, IgM and IgA along with neutrophil leukocytes and macrophages play an important role in the prevention of dental caries. It is believed that the immune response exerted by the gingival crevicular immune system is more potent compared to the salivary immune mechanism.
Salivary IgA prevents S. mutans from adhering to the tooth surface. The IgG antibodies acting as opsonins, facilitate phagocytosis and the death of S. mutans by the action of macrophages and neutrophil leukocytes.[15]

Pathogenesis of dental caries

Micro organisms that have adhesion ability adhere to salivary pellicle present on tooth surface and they form way for aggregation of other organisms which are unable to adhere to tooth surface initially. For many years, there are two hypothesis considered, one is nonspecific plaque hypothesis—NSPH which considered that all

plaque flora were collectively considered as being pathogenic. Other is specific Plaque hypothesis—SPH means certain specific organisms were pathogenic. Then a new hypothesis was given referred as the 'ecological plaque hypothesis'. According to this pathogenicity to specific species that produce the disease only at specific sites caused by a certain change in the environment of the residential plaque flora. The ecological plaque hypothesis targets the factors that resulted in the environmental change of the plaque.[16-18]

The pathogenesis is discussed under two sections that are:

- Disruption of microbial homeostasis in the 'biofilm'.
- Disruption of mineral homeostasis that is seen between the tooth and the 'oral fluid.[19]

An aggregate of microorganisms in which cells adhere to each other and/or to a tooth surface called as dental biofilm. Fermentable carbohydrates are metabolized by the bacteria present in biofilm and there is production of organic acids, mainly lactic acid. These bacterial products get accumulated in the fluid phase of the biofilm, leading to drop in pH and demineralization of the surface layer of the tooth.[20]

This will lead to increased enamel porosity, the spaces between the crystals become more, and the surface softens, which provides a way for the acids to get deeper into the tooth structure and dematerialize the subsurface.[21]

At this point, the products that are calcium and phosphate, created from demineralization, accumulate in the enamel surface and able to protect it from further mineral loss.[21] The available fluoride can prevent surface demineralization.[22] Sugars are swallowed and cleared by saliva resulting in neutral pH of the biofilm and calcium, phosphate, and fluoride now remineralize the tooth's surface.[21]

If the acidic conditions persist, the pH decrease will continue resulting a condition where the rate of mineral loss in the subsurface is more than the surface, leading to a subsurface lesion. Sufficient amount of mineral loss results in clinically visible white spot. A white spot can be arrested or reversed with behavioral changes and preventive measures implementation. Further progression of caries form micro cavities in the enamel.[21]

Diagnosis of caries

The most commonly dental caires is diagnosed by conventional visual-tactile method. Other methods used are:

- Radiographic examination
- Bitewing
- IOPA
- OPG
- Radiovisiography
- Substraction radiography
- Tuned aperture computerized tomography (TACT)
- Ultrasonic imaging
- Dyes
- Advanced diagnostic aids
- Fiber-optic transillumination (FOTI)
- Wavelength dependent FOTI
- Digital imaging FOTI (DIFOTI)
- Qualitative laser fluorescence (diagonodent)19

Management of dental caries

The prognosis of the dental caries is closely associated to the general condition and oral factors of an individual. There are two methods of caries removal.

First one is nonselective caries removal in which both soft and firm dentine is removed regardless of the approximation of carious lesion to the pulp. This method is also called as complete caries removal or complete caries excavation. The rationale behind this technique is that caries is prevented by this method as it stops spreading of carious lesion as all bacteria and caries are removed. After removal of caries, tooth can be filled effectively with restorative material which is properly retained as available hard sound dentine provides strong basis for it.[23-26]

Second method is selective caries removal in which carious part is selectively removed according to its closeness to the pulp, so soft and/or firm dentine is left and preserved. This method is also called as partial caries removal method. This procedure may be completed in one step or two steps. In one step method, after selective caries removal, cavity is restored with permanent restorative material in a single visit. In two step method, carious dentine is removed in two appointments.[27,28]

White spot lesions can be managed by noninvasive method, which recommends good oral hygiene, the use of fluoride-containing toothpaste, mouthwash, gels, and varnish, casein phosphopeptide amorphous calcium phosphate (CPP-ACP) and casein phosphopeptide-amorphous calcium phosphate fluoride (CPP-AFCP). Management of white spot lesions can also be done by using the resin infiltration technique, which delay or reverse the progression of non cavitated carious lesions.[29,30-32]

Conclusion

Dental caries is a chronic disease that greatly threatens human's health. Dental caries and its related complications can aggravate or induce systemic diseases leading to impaired quality of life. Caries is disease having multifactorial etiology and have high prevalence. It results in destruction of tooth structure if not diagnosed and treated at appropriate time. There is continues research is going on to combat dental caries.

Although there is a relatively well-developed caries management system, the difficulty assessments of dental caries treatment are still needed before making treatment plan. Then caries management plan is conducted to control caries risk factors and manage individual lesions.

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