Review of Dental Diseases and Homoeopathic Therapeutics

(Quick Review for Dental/Homoeopathy Students and Practitioners)

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Preface

Dear Doctors,

Greetings. Dental & Homoeopathy, though difficult, is one of the crucial subjects for post graduate, Ph.D. entrance examinations.

This edition of review book has been completely revamped and updated. This e- book provided with colour pictures to make understanding of the text, lot easier. This will be quite handy for final year students. It will be adequate for the final year medicine theory examination and at the same time it will orient you, exactly towards entrance examinations so that getting a P.G, Ph.D. of choice immediately after completing internship become a reality!

This review book of dental and homoeopathy is to link recent scientific advances to disease pathogenesis and therapeutic innovations. After studying this book, student will be able to answer all the questions and have a through explanation for each of the answers.

This review book is dedicated to all dental/homoeopathy students and practitioners.

We have tried to make this review book error free but sincerely apologize for any mistake that may have escaped my notice.

We will highly appreciate the suggestions and criticisms are most welcome from our readers for the improvement of the book.

Dr. Siva Rami Reddy E

Contents

Chapter - 1

Dental Caries

Dental caries is a pathologic process depending on several etiologic factors, which cause the destruction of the dental tissues and produces local and general complications. It is one of the most widespread diseases in the civilized populations with a prevalence of 40% at the age of seven years and 85% in seventeen year-old boys. However there is some evidence that that incidence in children aged five seventeen years has decreased about 36% in the last decades and approximately 50% of children can be considered caries-free in the permanent dentition [1-4].

Dental caries are responsible for a high rate of morbidity among the population and are associated with a reduced quality of life. It is known that the prevalence of dental caries among the general population has been linked to socio economic and demographic conditions, as well as behavioral aspects [5-7]. Therefore, in most developed countries, the prevalence of dental caries show a clear tendency to decline in the last three decades of the twentieth century and early twenty first century. Low socioeconomic status, low monthly household income and low educational level are associated with less access to dental services and oral hygiene products, poorer knowledge regarding oral health and oral hygiene and, consequently, a greater frequency and severity of dental caries [8]. The disease develops in both the crowns and roots of teeth, and it can arise in early childhood as an aggressive tooth decay that affects the primary teeth of infants and toddlers. Risk for caries includes physical, biological, environmental, behavioral, and lifestyle related factors such as high numbers of cariogenic bacteria, inadequate salivary flow, insufficient fluoride exposure, poor oral hygiene, inappropriate methods of feeding infants, and poverty.

For decades, the sugar fermenting, acidogenic species Streptococcus mutans has been considered the main causative agent of dental caries and most diagnostic and therapeutic strategies have been targeted toward this microorganism. However, recent DNA and RNA based studies from carious lesions have uncovered an extraordinarily diverse ecosystem where S. mutans accounts only a tiny fraction of the bacterial community. This

supports the concept that consortia formed by multiple microorganisms act collectively, probably synergistically, to initiate and expand the cavity [9].

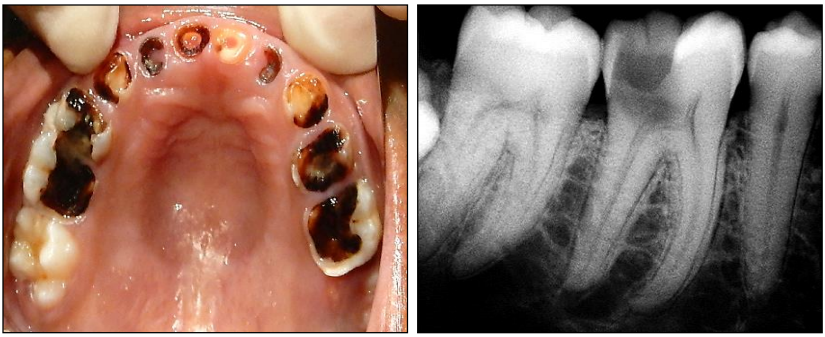

Fig 1: Dental caries

The evidence supports a concept of caries as a dietary carbohydrate-modified bacterial infectious disease. Its key feature is a dietary carbohydrate-induced enrichment of the plaque microbiota with organisms such as the mutans streptococci and lactobacilli which causes an increase of plaque's pH-lowering and cariogenic potential. The shift in the plaque proportions of these organisms appears to be related to their relatively high acid tolerance. A large body of evidence also supports a major effect of saliva on caries development. Integration of salivary effects with the concept of caries as a dietary carbohydrate-modified bacterial infectious disease suggests a broader concept which includes a major role of saliva in the regulation of the exposure of tooth surfaces to carbohydrate and of plaque acidity and, hence, the microbial composition and the pH-lowering and cariogenic potential of dental plaque [10].

Dental caries refers to the localized destruction of susceptible dental hard tissues by acidic by products from the bacterial fermentation of dietary carbohydrates. It is a chronic disease that progresses slowly in most of the people. Which results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms (plaque). The biofilm is characterised by microbial activity, resulting in fluctuations in plaque pH. This is a result of both bacterial acid production and buffering action from saliva and the surrounding tooth structure. The tooth surface is therefore in a dynamic equilibrium with its surrounding environment. As the pH falls below a critical value, the demineralisation of enamel, dentine or cementum occurs, while a gain of mineral (remineralisation) occurs as the pH increases.

Primary caries can occur on different tooth surfaces. On an approximal surface, the lesion starts and forms beneath the contact area between teeth.

Caries on an occlusal surface is also a localised phenomenon in pit and fissure. On both occlusal and approximal surfaces, enamel caries is a three-dimensional subsurface demineralisation that spreads along the enamel prisms. Secondary caries is a lesion located at the margin of a dental restoration. It represents a caries lesion adjacent to the margin and there may be signs of demineralisation (wall lesions) along the cavity wall which could be a consequence of microleakage. However, clinical and microbiological studies indicate that this leakage does not lead to active demineralisation beneath the restoration.

Historically, researchers have focused on biological and dietary effects on children's oral health to explain caries development. In recent years, children's oral health outcomes using a broader framework, which incorporates psychosocial and environmental predictors as well as the biological and dietary effects. These frameworks generally classify conditions associated with disease into five broad domains: genetics and biology, social environment, physical environment, health influencing behaviors and medical care. These relevant variables explain why some children, despite use of fluoride and abundant information about caries prevention, develop carious lesions. The caries model by Fisher Owens and co-workers includes different levels of the environment that can affect caries development: child level; family level; and community level.

Caries may be characterized by the experience of pain, problem with eating, chewing, smiling and communication due to missing, discolored or damaged teeth. Visible holes or pits in teeth, Brown, black or white staining on any surface of a tooth, Bad breath and foul tastes, fever, chills, abscess, and trismus. Worldwide, approximately 2.43 billion people (36% of the population) have dental caries in their permanent teeth. In baby teeth it affects about 620 million people or 9% of the population. The disease is most prevalent in Latin American countries, countries in the Middle East, and South Asia, and least prevalent in China. In the United States, dental caries is the most common chronic childhood disease, being at least five times more common than asthma [11-15].

Child level Visible plaque, early colonisation by caries-related bacteria, the presence of mutans streptococci, frequent intake of sweetened drinks, infrequent tooth brushing, illness and use of antibiotics have all been associated with caries developments in preschool children. Family level Family level characteristics associated with caries risk in children included are demographic factors of the family, parental oral health behaviors and attitudes, dental anxiety and dental attendance, maternal health and lifestyle

in pregnancy and early childhood. Community-level Children's oral health is likely to be better in a community that values good oral health. Cultural aspects and the neighbourhood may have implications for caries development. The dental care system and amount of dental care available may affect or alhealthand the development of caries preschool children [16].

Frostell and others assessed the potential of various sugars and starch products to favor dental caries in occlusal sulci and on smooth surfaces of rats that had been infected with a bacterial flora conducive to multi surface caries. Although crevicular lesions were found in all groups, their prevalence and severity were not consistently associated with comparable activity on smooth surfaces. The most active sulcal lesions occurred in animals on a diet containing sucrose and mixtures of starch with sucrose or sorbitol. Plaque and smooth surface lesions were most prevalent in groups that were fed sucrose. The least smooth surface activity occurred in animals fed maltose, starch, and hydrogenated starch. Hydrogenated starch, which contains sorbitol and hydrogenated dextrins, is not rapidly fermented by oral bacteria. As mentioned before, it does not favor multi surface caries, and it is used as a substitute for highly fermentable sugars in some sweets now manufactured in Sweden. Far more attention has been focused on caries activity in the enamel than on lesions that affect the cementum and root surfaces of teeth. Root surface caries probably will be of greater concern as more people are treated for periodontal pathosis by means of gingival resection and interdental splinting. The postulation that dietobacterial interactions conducive to root surface caries may differ from those which produce enamel lesions can be supported by observations in man and in animals. Root surface caries may occur in humans without coronal lesions. This has often been observed clinically but only occasionally reported in epidemiological studies.

The potential of certain strains of streptococci to induce plaque and multi surface cavitations has been conclusively established in animals and strongly implicated in humans. The virulence of the many streptococcal strains prevalent in the mouth ranges from none or negligible to that which is extremely high. Although some of the strains studied appear to resemble Streptococcus mutans-S mitis, S bovis, S salivarius, S Sanguis-a number of the strains isolated do not resemble exactly any of the species described in reference texts. It will not be surprising if the situation is somewhat comparable to that of pneumococci, which have been divided into 75 to 80 serological types, of which 4 are considered to be serious pathogens. The more than 70 other types account for fewer infections, although they can be

isolated from the throats of normal persons. Thus, different types of pneumococci predominate in health and disease. Since many types of acidogenic bacteria do not tend to adhere to the teeth, it is important to determine which types of microorganisms have the potential to form plaques associated with multi surface caries. De Stoppelaar and others isolated dextran producing streptococci from human dental plaque. Some of their isolates fermented mannitol and sorbitol and had other biochemical characteristics that suggested they might be isolates o f S bovis. Isolates that did not ferment mannitol and sorbitol were classified as S sanguis. Fitzgerald and Fitzgerald found that highly active smooth surface caries developed in gerbils that were fed sucrose and that had been challenged by an inoculation of a streptococcus (human) that was slightly resistant to streptomycin. This mutant strain was consistently recovered and identified in the inoculated animals and never recovered from the controls that remained caries free. Larson and Fitzgerald found identical results in another species, Mystromys albicaudatus. This animal is commonly known as the white tailed rat but is phylogenetically closer to hamsters than to rats. Since the molar teeth of both of these animals have few or no deep occlusal crevices, caries activity would have been negligible unless adherent plaques had formed on the teeth [17, 18].

Chapter - 2

Oral Cancer

Oral cancer is a malignant neoplasia which arises on the lip or oral cavity. Is traditionally defined as a squamous cell carcinoma (OSCC), because in the dental area, 90% of cancers are histologically originated in the squamous cells. Oral cancer is two to three times more prevalent in men than women in most ethnic groups.

The risk for developing oral cancer is 3 times higher in smokers compared with nonsmokers. Besides, the risk for oral cancer is 35% lower in people who quit smoking four years ago than those who continue smoking and not higher in persons with no smoking antecedents for over 20 years when compared with people who have never smoked. Cigarette smoke weakens immunity in the oral cavity by promoting gingivitis, periodontitis and oral cancer. This smoke contains several elements that promote cancer and they basically can be grouped into three distinct groups: nitrosamines, benzopyrenes and aromatic amines. These chemicals are called pre carcinogens, which must suffer coordinated alterations by oxidative enzymes, so that the final product becomes poor in electrons and into an agent to be covalently bound to the DNA, generating an adduct mutated region [19-23].

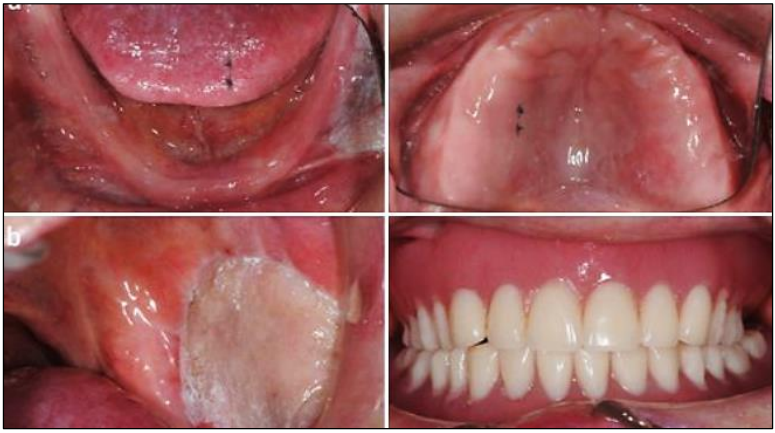

Fig 2: Oral cancer

Alcohol (ethanol) can act as a both locally and systemically risk factor: increased permeability of oral mucosa, dissolving lipids components of the epithelium, causing epithelial atrophy and interference in DNA synthesis and repair; it also has genotoxicity and mutagenic effects, causing decreased salivary flow, affects the liver's ability to deal with toxic or potentially carcinogenic compounds, and their chronic use is associated with an impairment of innate and acquired immunity, resulting in increased susceptibility to infections and neoplasms. The most-common sites of HPV-related head and neck squamous cell carcinoma (HNSCC) are the tonsils and base of tongue within the oropharynx, with a prevalence rate of 75%; HPV-related HNSCC is rare in non oropharyngeal sites [24].

Diagnosis

Computed tomography (CT) effectively demonstrates bony changes such as erosive lesions at the skull base or mandible. Magnetic resonance imaging (MRI) has multiplanar imaging capacity and can show subtle variations in soft tissue distinguishing inflammatory changes from fibrosis or recurrent tumor. In patients with unknown etiology, fine needle aspiration biopsy (FNAB) can save time and expense of an extensive work up for malignant disease and the potential complications of open biopsy. Several studies have shown that FNAB has an excellent diagnostic accuracy. The incorporation findings of molecular biology into the clinical practice of head and neck oncology are yet to become a reality. However a new tumor antigen, A9/α6β4 integrin, has recently been shown to have prognostic value. Assays for mutations in the *p53* gene have also shown promise in screening cancer. Application of molecular biology in future will add up to refinement in oral cancer screening technique [25].

Chapter - 3

Cleft Lip and Palate

Orofacial clefts include a range of congenital deformities most commonly presenting as cleft lip with or without cleft palate (CLP) or isolated cleft palate (CP). CLP is the second most common congenital birth defect in the U.S. trailing only Down syndrome. There are roughly 7,000 infants born with orofacial clefts in the U.S. annually. Beyond the physical effects on the patient, CLP also has significant psychological and socioeconomic effects on both patient and family, including disruption of psychosocial functioning and decreased quality of life (QOL). It is associated with increased mortality from many causes, including suicide as well as substantial healthcare costs. Cleft lips can be unilateral or bilateral, and may involve the alveolus or palate. Affected individuals may present with other congenital anomalies and may be part of a genetic syndrome [27].

Normal lip development occurs between weeks 4 and 8 of gestation. By the end of week 4, the frontonasal prominence forms from migrating neural crest cells of the first pharyngeal arch. Nasal placodes, representing ectodermal thickening, develop at the caudal end of this structure and divide the paired medial and lateral nasal processes. The primary palate forms from the fusion of the paired medial nasal processes by week 6, giving rise to the premaxilla: central upper lip, maxillary alveolar arch and four incisor teeth, and hard palate anterior to the incisive foramen. The secondary palate develops after the primary palate during weeks 6-12.

The medial projections of the maxillary processes form palatal shelves which rise above the tongue, fusing medially at the midline, anteriorly with the primary palate, and superiorly with the septum. The incisive foramen marks the anterior extent of the secondary palate. Formation of the primary and secondary palates completes the separation of nasal and oral cavities, permitting simultaneous respiration, and mastication [28].

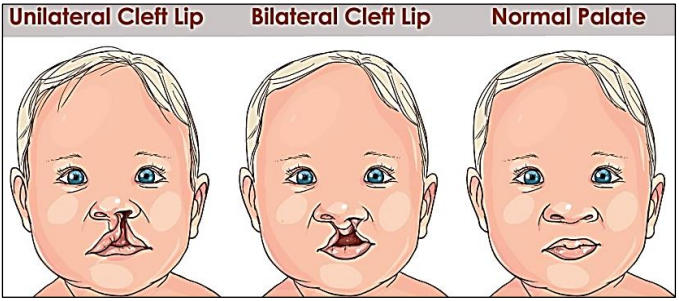

Fig 3: Cleft lip and palate

Cleft lip is consistently more common in males at a 2:1 ratio, in contrast to cleft palate which has a similar ratio in favor of females. Some have postulated that common maternal hormones may be involved in both sex determination and orofacial clefting. Genetic susceptibility has long been identified as a major component of CLP. Monozygotic twin studies suggest that genetics account for 40-60% of orofacial clefting. The interferon regulatory factor 6 *(IRF6)* genes is consistently associated with non-syndromic CLP in multiple studies and is also the causative agent of van der Woude syndrome, the most common syndromic cause of cleft lip. Recent availability of genome wide association studies (GWAS) has identified several new genetic loci, including a "gene desert" region on chromosome 8q24. Over 500 Mendelian syndromes are listed in the Online Mendelian Inheritance in Man (OMIM) database. The most common and well known syndrome associated with cleft lip is van der Woude syndrome. It is caused by a defect in the *IRF6 gene* on chromosome 1 and is inherited in an autosomal dominant fashion. Typical clinical features are cleft lip and/or palate, lower lip pit or fistula, and dental anomalies.

The most common syndrome associated with isolated cleft palate is velocardiofacial syndrome (VCFS). Several authors classified the cleft lip and palate. Veau classified the clefts into four main groups:

- Clefts of soft palate
- Clefts of hard palate
- Unilateral clefts of the lip, alveolus and palate
- Bilateral clefts of the lip, alveolus and palate

According to Koch *et al*., (1995), Kernahan (1971) suggested a new classification of cleft lip and palate and it gives the shape of Y letter and includes:

- 1 and 4 represent the right and left side of the nasal floor, respectively
- 2 and 5 represent the right and left side of the lip, respectively
- 3 and 6 represent the right and left side of the paired alveolar segment, respectively
- 7 represent the primary palate
- 8 and 9 represent the secondary palate

Clinical features

One of the first manifestations associated with cleft palate is difficulty with feeding. Breast feeding can be challenging and specialized feeding strategies including the use of a Habermann feeder, Montgomery nipple, bulb syringe, or pigeon feeder are frequently required. The infant should be fed in a slightly upright position. Aerophagia is a problem in these infants and requires more frequent burping and slower feeding. In rare cases, a nasogastric tube or surgical gastric tube may be necessary as the infant learns to feed from a bottle [29].

Management

Early treatment and evaluation Initial assessment of a child with cleft lip is undertaken to know the extent of the cleft. The parents are informed of the cleft by a specialist nurse. The team often discusses with parents the treatment plan of their child. Parent feels worry of this defect and it is very important to show them previous cases of clefts which were treated to improve the psychological side. The most important thing is the feeding of the infant. A variety of feeding devices are available and this depends on the type of clefts. For example, for infants with isolated cleft lip, a bottle or breast can be used.

On the other hand, infants with cleft lip and palate face challenges when feeding they are incapable of sucking either their mother's nipple or from a bottle. For that reason, feeding devices such as nipples, cross curt nipples and longer nipple can be successfully assist the infant when feeding. Repair of the lip, nose, and anterior palate Repair of the lip this surgery is undertaken about 3 months of child life and it involves the mobilization of the tissue in the defect side of the lip and dissection of orbicularis oris muscle. This permits the closure of the lip. Repair of anterior palate anterior palate repair involves the use of vomerine flap which is taken from nasal septum to close the defect. Nasal repair involves the separation the adhesion between alar cartilage and overlying skin and underling mucosa and then these structures are repositioned again using different methods of fixation.

Alveolar bone grafting this surgery involves filling a bony defect with cancellous bone (from the iliac crest or tibial plateau) and it is performed at the time of mixed dentition (7-14 years). Cleft palate surgery is performed about 6 month of child life and involves a mobilization of mucoperiosteal flaps of the hard palate. Soft palate muscles are then dissected from the bony edge of the hard palate and repositioned. An incision is then made in the lateral part of the palate to reduce the tension in the midline [30-32]. In some cases palatal repair did not solve the speech problem. For this reason, the surgery is undertaken to provide velopharyngeal closure between soft palate and pharyngeal wall. The surgery involves the elevation of myomucosal flap (Pharyngeal flap) from the posterior pharyngeal wall and then this flap is attached to the posterior soft palate to achieve the closure between soft palate and pharyngeal wall.

Chapter - 4

Gingivitis

The gingiva is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth. The shallow crevice or space around the tooth bounded by the surface of the tooth on one side and the epithelium lining the free margin of the gingival. The depth of the gingival sulcus is or is an important diagnostic parameter under absolute normal or ideal conditions, the depth of the 1 gingival sulcus is 0 mm or close to 0 mm. These strict conditions of normalcy can be produced experimentally only in germfree animals or 2, 3 after intense, prolonged plaque control.

An early definition of gingivitis simply stated that gingivitis was inflammation of the 4 gingiva and gingivitis is inflammation of the gingiva in which the junctional epithelium remains attached to the tooth at its original level. The association between periodontitis and systemic disease may be due to both increased systemic inflammation and to translocation of bacteria into the bloodstream. Despite its importance, the microbial ecology of periodontal disease in different oral habitats remains incompletely understood. Studies of the oral microbiome in periodontal disease typically focus on small populations in developed countries with advanced dental health care systems, which may not be representative of the natural history of periodontal disease in the absence of treatment.

The most common viral infections are herpes simplex virus type1 (HSV-1) and 2 (HSV-2) and varicella-zoster virus. HSV is the most common viral infection of the oral/facial area. It has two subtypes: type 1, which affects the oral cavity; and type 2, which affects the genitals. Primary herpetic gingivostomatitis is most commonly observed in children from 7 months to 4 years of age but can also be found in adolescents or young adults. Children are often infected with HSV by their own parents if these have recurrent herpes lesions [34-40].

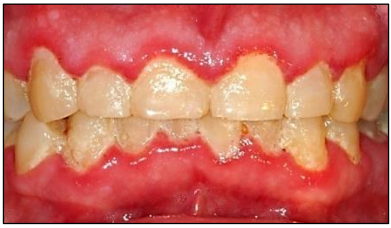

Fig 4: Gingivitis

The incubation period of the virus is 1 week, and healing occurs after approximately 10 to 14 days. Following infection and local replication at mucosal surfaces, HSV 1 enters sensory nerve endings and is transported by retrograde axonal transport to neuronal cell bodies, where a more restricted replication cycle takes place, usually culminating in the latent infection of these neurons.

Latency allows maintenance of the viral genome in nonpathogenic and non-replicate form, serving as a reservoir for a later viral attack on the host. Reactivation of the virus in sensory ganglia causes cutaneous and mucocutaneous infection of the face, usually on the lips. Candida albicans is a diploid fungus (a form of yeast) that causes opportunistic oral and genital infections in humans. This commensal species populates the gastrointestinal tract. It affects 80% of the human population with no harmful effects but its overgrowth results in candidosis. Infection by candida albicans is the most common mycotic infection of the oral mucosa and is commonly associated with impaired host immune responses such as immunodeficiency, reduced salivary flow rate, smoking, corticosteroid treatment, or the use of antibiotics Candidosis is not usually observed in the gingival tissue of healthy individuals but, when affected, the most frequent clinical feature is gingival redness associated with a granular surface. This infection appears in four main forms: acute pseudomembranous candidosis, acute atrophic candidosis, chronic hyperplastic candidosis, and chronic atrophic candidosis. Linear gingival erythema is a non-plaque-induced gingival lesion exhibiting a distinct erythematous band of the marginal gingiva, with either diffuse or punctate erythema of the attached gingiva. Lichen planus is the most common mucocutaneous disease involving the gingiva, with a prevalence of 0.5-2.5%.

Type of gingivitis

Gingivitis is two types that are Acute and Chronic Inflammation. Acute Inflammation: Gingival abscess is a localized, painful, rapidly expanding lesion that is usually of sudden onset. It appears as a red swelling with a

smooth, shiny surface within 24 to 48 hours. Chronic Inflammation: A gingival enlargement originates as a slight ballooning of the interdental papilla and marginal gingival. It is complicated by acute infection or trauma.

Stages of gingivitis

Stage I gingivitis

The initial lesion: The first manifestations of gingival inflammation are vascular changes consisting essentially of dilation of capillaries and increased blood flow. These initial inflammatory changes occur in response to microbial activation of resident leukocytes and the subsequent stimulation of endothelial cells. Clinically, this initial response of the gingiva to bacterial plaque 6 (subclinical gingivitis) is not apparent.

Stage II gingivitis

The early lesion: The early lesion evolves from the initial lesion with about one week after the beginning of plaque accumulation.

State III gingivitis

The established lesion: It is evolves, characterized by a predominance of plasma cells and B lymphocytes and probably in conjunction with a pocket epithelium. Clinical findings are changes of colour, size and texture. State IV Gingivitis: The Advanced Lesion: the lesion in to alveolar bone characterizes a fourth stage known as the advanced lesion or phase of periodontal breakdown.

Clinical features

Gingivitis is of sudden onset and short duration and can be painful and bleeding. Recurrent gingivitis reappears after having been eliminated by treatment or disappearing spontaneously. Chronic gingivitis is slow in onset and of long duration, and is painless, unless complicated by acute or subacute exacerbations. Chronic gingivitis is the type most commonly encountered. Chronic gingivitis is a fluctuating disease in which inflammation persists or resolves and normal areas become inflamed. Etiologic factors are gingivitis that vitamin C and K deficiency, platelet disorders, Multiple myeloma and post rubella purpura. The effects of hormonal replacement therapy, oral contraceptives, pregnancy and the menstrual cycle are also reported to affect gingival bleeding and endocrine changes. The gingival coral pink colour becomes pale when vascularisation is reduced. The gingival enlargements do to inflammation. Complication of gingivitis is periodontitis [41-45].

Chapter - 5

Periodontitis

Periodontitis is one of the most ubiquitous diseases and is characterized by the destruction of connective tissue and dental bone support following an inflammatory host response secondary to infection by periodontal bacteria. Severe periodontitis, which may result in tooth loss, is found in 5-20% of most adult populations worldwide. Children and adolescents can have any of the several forms of periodontitis such as aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases [46].

The oral bacterial microbiome includes over 700 different phylotypes, with approximately 400 species found in subgingival plaque. The subgingival microflora in periodontitis can harbor hundreds of bacterial species but only a small number has been associated with the progression of disease and considered etiologically important. Subgingival plaque from deepened periodontal pockets is dominated by gram-negative anaerobic rods and spirochetes.

Strong evidence has implicated *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* to the pathogenesis of adult periodontitis. In addition, Bacteroides forsythus, *Prevotella intermedia*, *Peptostreptococcus micros*, and *Fusobacterium nucleatum* have been strongly linked with the progression of adult periodontitis.

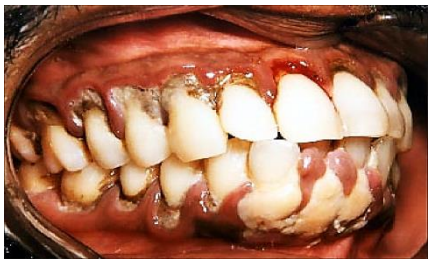

Fig 5: Periodontitis

There is accumulating evidence for a higher level of periodontal disease among smokers. Tobacco smoking exerts a substantial destructive effect on the periodontal tissues and increases the rate of periodontal disease

progression. Risk factors including tobacco smoking modify the host response to the challenge of bacteria in microbial dental plaque. Smokers with periodontal disease seem to show less signs of clinical inflammation and gingival bleeding compared to nonsmokers. That could be explained by the fact that nicotine exerts local vasoconstriction, reducing blood flow, edema, and clinical signs of inflammation. Nicotine acetylcholine receptor has been found to play an important role in the development of nicotine related periodontitis. Patients with undiagnosed or poorly controlled diabetes mellitus type 1 or type 2 are at higher risk for periodontal disease [47].

There are many studies that demonstrate an association between diabetes and an increased susceptibility to oral infections including periodontal disease. Periodontal disease is capable of predisposing to vascular disease due to the rich source of subgingival microbial species and host's response. Furthermore, we must be aware that these diseases share many risk factors and there are evident similarities to the basic pathogenic mechanisms. Patients with inadequate stress behavior strategies (defensive coping) are at greater risk for severe periodontal disease. Stress is associated with poor oral hygiene, increased glucocorticoid secretion that can depress immune function, increased insulin resistance, and potentially increased risk of periodontitis.

Obesity has been reported to be an important risk factor for periodontal disease. Several explanations for the association between obesity and periodontal disease. Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia. Offenbacher *et al*. found significantly more periodontal attachment loss among mothers of PLBW infants compared with mothers of normal term infants [48-50].

McDevitt *et al*. demonstrate that the composite IL-1 genotype is significantly associated with the severity of adult periodontitis. They also confirmed that both IL-1 genotyping and smoking history provide objective risk factors for periodontal disease in a private practice environment. Cholesterol has long been known to play a crucial role in predicting risk for heart attack in seemingly healthy people. But half of all heart attacks occur among people who do not have high cholesterol. Also, the classical risk factors of CVD cannot account for all the variation in the incidence of CVD cases.

Clinical features

Plaque accumulation, calculus formation, gingival redness and swelling, gingival bleeding and suppuration which may occur either spontaneously or

when subjected to probing, halitosis (bad breath), and loss of alveolar bone. Others include: deepening of the gingival crevice resulting in the formation of a pathological periodontal pocket, root exposure due to gingival recession, and increased tooth mobility. Severe forms of the disease may lead to tooth migration, compromised esthetics, impaired masticatory function, and tooth loss ultimately [51].

Complications

Respiratory system

Scannapieco *et al*. showed that lung function decreased with increasing periodontal attachment loss. Therefore, they concluded that a potential association between periodontitis and chronic pulmonary diseases like chronic obstructive pulmonary disease (COPD) may exist.

Endocrine system

While it has been established that people with diabetes are more prone to developing periodontal disease, new research is suggesting that periodontal disease may, in turn, be a risk factor for diabetes.

Carcinoma

Earlier literature review showed that chronic periodontitis is an independent clinical high-risk profile for head and neck squamous cell carcinoma (HNSCC), especially in the oral cavity, followed by the oropharynx and larynx.

Chapter - 6

Periodontal Abscess

The periodontal abscess is a frequent periodontal condition in which periodontal tissues may be rapidly destroyed. Its importance is based on the possible need of urgent care, the affectation of tooth prognosis, and the possibility of infection spreading.

An abscess is a localized collection of pus that has built up within the tissue of the body. An abscess is so called because there is an abscessus (Latin word means “a going away or departure”) of portions of the tissue from each other to make room for the suppurated matter lodged between them. Empyema is the accumulation of pus in a preexisting rather than a newly formed cavity. The process of abscess formation is known as suppuration and the bacteria which cause suppuration is known as pyogenic bacteria. Pus is a purulent exudate, creamy or opaque in appearance, composed of dead and living neutrophils, red cells, fragments of tissue debris and fibrin. In old pus, macrophages and cholesterol crystals are also present. An abscess is a defensive reaction of the body tissue to prevent the spread of infectious materials to other parts of the body. Abscess is lined by pyogenic membrane. The pyogenic membranes formed by the neutrophils and macrophages in an attempt to keep the pus from infecting neighboring structures. However, such membrane tends to prevent immune cells from attacking bacteria in the pus, or from reaching the causative organism or foreign body [52-55].

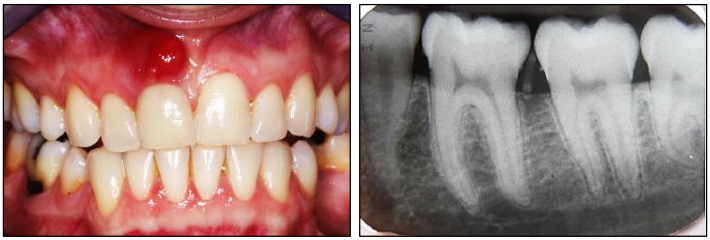

Fig 6: Periodontal abscess

Abscess of the periodontium is a localized collection of pus (i.e. an abscess) within the tissues of the periodontium.

Abscesses of the periodontium have been classified primarily, based on their anatomical locations into three types: gingival abscess, pericoronal abscess and periodontal abscess. Gingival abscess is a localized, purulent infection involves only the soft gum tissue near the marginal gingiva or the interdental papilla. Pericoronal abscess is a localized, purulent infection within the gum tissue surrounding the crown of a partially or fully erupted tooth. A periodontal abscess is a localized, purulent infection involving a greater dimension of the gum tissue, extending apically and adjacent to a periodontal pocket. A periodontal abscess is also known as lateral abscess or parietal abscess; however, when marginal soft tissues are affected in isolation, it is called a gingival abscess. Periodontal abscess and gingival abscess are identical histologically and differ only in location.

The causes of the periodontal abscess associated with periodontitis are extension of infection or inflammation from pocket, presence of tortuous pocket with cul-de-sac, after scaling and or after routine oral prophylaxis, inadequate scaling, marginal closure of pocket, pocket lumen insufficient to drain, treatment with systemic antibiotics without subgingival debridement, nifedipine therapy and treatment with GTR membrane both resorbable and non-resorbable. The periodontal abscess in periodontitis may occur at various stages: acute exacerbation of untreated periodontitis during periodontal treatment, refractory periodontitis and during periodontal maintenance [56].

Entry of bacteria into pocket wall could be the first event. Inflammatory cells are then attracted by chemotactic factors released by bacteria and the inflammatory reaction leads to tissue destruction. There is subsequent encapsulation of bacterial infection and production of pus. The rate of destruction in abscesses will depend on growth of bacteria inside the foci and its virulence as well as local pH, since acidic environment will favour activity of lysosomal enzyme. Intra oral features in case of acute periodontal abscess include ovoid elevation of gingiva along lateral aspect of root, redness, mobility, elevation of tooth in socket and tenderness to percussion or mastication. Regarding mobility 56.5% to 79% of teeth showed mobility.10-40% patients show regional lymphadenopathy. Symptoms may vary from slight discomfort to severe pain and swelling, 55% of acute periodontal abscesses in upper jaw, 48% on buccal aspect, 24% on distal aspect, 13.8% on lingual/palatal aspect and 62% on mesial aspect complained severe pain.

Diagnosis

Intra oral or dental radiographs as periapical, bitewings and OPG are used to assess marginal bone loss and periapical condition of involved tooth. Gutta percha point placed through sinus might locate the source of the abscess.

Chapter - 7

Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis (NUG) is a distinct and specific form of periodontal diseases. It has an acute clinical presentation with the distinctive characteristics of rapid onset of gingival pain, interdental gingival necrosis, and bleeding. It has been given many names: Vincent's disease, fusospirochetal gingivitis, trench mouth, acute ulcerative gingivitis, necrotizing gingivitis, and acute necrotizing ulcerative gingivitis. Actually and according to the recent data, the prevalence rate of Necrotizing ulcerative gingivitis varies over a wide range from 6.7% in Chilean students between 12 and 21 years. It has an acute clinical presentation with the distinctive characteristics of rapid onset of interdental gingival necrosis, gingival pain, bleeding, and halitosis. Systemic symptoms such as lymphadenopathy and malaise could be also found.

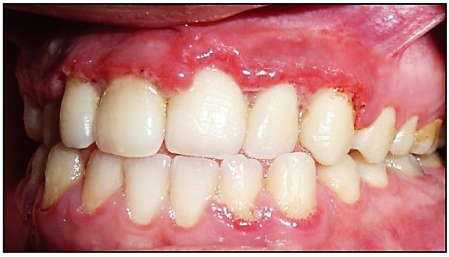

Fig 7: Necrotizing ulcerative gingivitis

There are various predisposing factors such as stress, nutritional deficiencies, and immune system dysfunctions, especially, HIV infection that seems to play a major role in the pathogenesis of Necrotizing ulcerative gingivitis. In human immunodeficiency virus and acquired immune deficiency syndrome (HIV/AIDS) patients, oral lesions are important because they are easily observed with the naked eye and can represent the first clinical signs of diseases. The prevalence of necrotizing gingivitis varies, but previous studies have suggested rates from 0% to 6.3% in HIV/AIDS patients. Although ANUG is not particularly rare, it occurs less frequently than other oral diseases. Furthermore, ANUG has been correlated with CD4+ T-lymphocyte counts below 200 cells/μL.

This indicates that physicians can speculate on CD4+ T-lymphocyte counts from the presence of ANUG, and vice versa. Thus, it is important for physicians to look for oral lesions including ANUG in HIV/AIDS patients [57-60].

Chapter - 8

Homoeopathic Therapeutics

Phosphorous: Bleeding will stop with this medicine. The common acid debility is very marked in this remedy.

Toothache chamomilla: This may help those individuals with lowered pain threshold, three doses of 30 C over several hours prior to dental visit.

Antimonium crudum: It is use for a toothache that is worse at night and is aggravated by heat.

Aranea diadema: Use for sudden, severe pain in the teeth after lying down.

Calcarea carbonica: Use for a toothache that worsens when exposed to cold air or hot.

Abscesses belladonna: This remedy indicated that red, inflammation, heat, throbbing and burning. Great children's remedy. It acts up on every part of the nervous system producing active congestion. No thirst, anxiety or fear. Early dental abscess accompanied by redness. This is aggravated by slightest touch.

Hepar sulph: In chronic abscess this drug every 6 hours is prescribed up to one day. Where abscess is accompanied by pus formation, increased salivation and where gums are sour to touch and bleed easily.

Silicea: Twice daily issued (6C) when the abscess starts to drain.

Calendulas dilute the tincture one part to 20 parts of water. Use as a gentle but effective mouth rinse.

Delayed eruption of teeth

Use Calc carb 6C twice a week.

In gingivitis homoeopathic medicines are below following:

Ignatia: Use for headache as if a nail were driven through the side of the head. Aranea diadema: use for radiating pain in the right side of the face that is aggravated by cold. Also use for sudden, severe pain in the teeth at night after lying down.

Lachesis: Left sided complaints.

Lycopodium: Right side complaints.

Sanguinaria: Right side neuralgia, facial migraine. Emphasis on Conditions Involving Tooth Socket Post.

Extraction

Belladonna is indicated that red, inflammation, heat, throbbing and burning. No thirst with fear.

Pulsatilla: Use for diminished saliva with no thirst.

Disease related to

Aconite (potency 12C) and Gelsemium (potency 12C) are the applications that worked wonderfully in reducing the fear of anxious patient who reported for dental treatment. These does wonder especially for children, a dose taken morning of the procedure, is sufficient to reduce the anxiety level of patient and we know that a relaxed patient contributes to easy, gentle work for the dentist.

Arnica (Potency 30C) works with the inflammation and trauma from routine dental procedures. All patients who reported with chronic gingivitis and underwent scaling for the same were given and post-scaling the colour, contour, consistency of gingival tissue were noted, and the results showed the remarkable healing of gingival tissue. This application can be given to reduce postoperative swelling from any oral surgical procedure, and soreness following insertion of new complete or partial dentures.

Nerve pain

Hypericum (Potency 9C) works wonderfully for nerve pain due to post-operative surgery (tooth extraction or mucogingival surgery) and accidental facial trauma.

Toothache with abscess

Belladonna (Potency 6C) was given to patient who reported with sudden onset of gum or tooth abscess that was accompanied by redness, inflammation and throbbing pain. One thing to note here is that this application work in early abscess while Hepar sulphuris (Potency 6C) use for abscess accompanied by pus formation, Pyrogenium is use when pus is present without drainage, Silicea is use to hasten the discharge when pus is draining.

Painful injection

Ledum (Potency 30C) is an excellent for reducing post trauma effects of dental injections especially for palatal injections and inferior alveolar nerve blocks and it was given routinely to patients who were given injections.

Local anaesthesia antidote

Chamomilla (Potency 12C) is an excellent antidote when local anaesthetic are used for any dental treatment. Patients can begin taking it every fifteen minutes after dental treatment is completed to reduce numbness. This can also minimizes the lip bite seen in children after inferior alveolar nerve block which is caused due to numbness for long duration.

Tooth cavity

Plantago tincture is a remedy that we found most beneficial when rubbed onto or around a tooth or teeth that are sensitive to hot or cold, or applied into cavity.

Oral surgery

Silica (Potency 6C) works well by causing any root fragments or splinters of bone to exfoliate following any oral surgery and was routinely given to patients after extraction. Staphysagria (Potency 30C) was given when incisions of soft tissues was required such as periodontal flap procedures, third molar surgery where access flaps is required and during periodontal graft procedure.

Bleeding

Initially Arnica (Potency 200C) every 15 minutes for Post-surgical bleeding that was not controlled by pressure with moist gauze was given, but when it was bright red colour then ferrum phos (Potency 6C) every 15 minutes, and when it was dark colour then lachesis (Potency 30C) every hourly was given. Both worked wonderfully by their haemostatic actions as well as patient's compliance to this homeopathic application.

Orthodontic treatment

Ruta is valuable in patients undergoing orthodontic treatment. It was given after any adjustment of braces or wires and if the wires have cut into cheeks or lips which are very common during fixed and removable orthodontic treatment.

Teething problem

Chamomilla (Potency 15C) was ideal for delayed and difficult tooth eruptions in children and in those patients who reported nerve pain in

association with tooth eruption then Hypericum (Potency 9C) was prescribed.

Post cavity filling

Hypericum because of its nerve injury healing property were routinely given to patient who reported for tooth cavity filling since any preparation of the tooth can involve minute nerve tubules present in tooth. TRISMUS: Magnesium phosphorus or Causticum were given to patients for relieving cramping of the jaw or inability to open the mouth. The long appointments have become part of today modern dentistry for achieving "Hollywood smiles".

Dry socket

Mouthwash of salvia infusions was given several times a day to diminish pain of "dry socket" in 5 patients and a few drops of hypercal (Hypericum and Clandedula tinctures) was also given, in a small glass of warm water to relieve dryness and make mouth much more comfortable. With throbbing pain belladonna (potency 30C) every hour and with unbearable pain coffea (potency 30C) every 15 minutes was given.

Oral ulcers

Feverfew was prescribed for relieving discomfort caused by mouth ulcers in 10 patients. This should be continued for a few days after ulcers have gone to break the cycle of repeated attacks.

Amalgam filling

Merc sol (Potency 30C) is a wonderful remedy for eliminating mercury from the body. The patients who were anxious about amalgam fillings were given this remedy: also whenever an amalgam filling was removed, it was also given to patients to minimize the ill effects of mercury in body.

Temporomandibular joint disorder

Arum triphyllum was given to patient who complaint of pain in temporomandibular joint on swallowing. I would also like to add that Calcarea fluorica is useful in hypermobile joints, and Calcarea phosphorica can be given when mouth cannot be opened without pain.

Toothache

Belladonna was given to patient who reported with the toothache that was accompanied by throbbing pain and was worse from motion, touch or chewing, and Staphysagria was given to patients who complaint of pain from

major decay of a number of teeth. Two other homeopathic remedy that are useful in toothache are, Coffea cruda when toothache that get worsens with heat and is relieved by ice while Magnesia phosphorica is useful in toothache that worsens with cold and relived from warmth. In allopathic normally for any kind of toothache, analgesic is prescribed generally but when homeopathic medicine is to be given then we have to see the characteristic of that toothache then only the remedies could do wonder.

Oral herpes lesions

Patients with oral herpes lesions could be given Graphites (Potency 7C), Petroleum (Potency 9C), Rhus toxicodendron (Potency 7C) and Arsenicum album (Potency 7C) and the lesions resolve early with minimum discomfort to patients.

Severe gingivitis

Arsenicum album and Ferrum phosphoricum were given to the patient who reported with severe gingivitis.

Pain: Chamomilla (potency 200C), one hour prior to surgery, and repeated every 15 minutes as necessary for no more than 6 doses.

Halitosis

Carbo veg (potency 30C) with Merc sol (potency 30C) twice daily were given to patients followed by the desired dental treatment.

Bruxism

Patients were given cina (potency 30C) one hour before sleep and while awake nux vomica (potency 30C) twice daily till the symptoms subside.

Abrasions

Hypericum and calendula tinctures (hypercal) should be applied to the area three times daily till the symptoms subside.

References

1. Selwitz RH, Ismail AI, Pitts NB. Dental caries. The Lancet. 2007; 369:51–59.
2. Brunelle JA, Carlos JP. Recent trends in dental caries in U.S. children and the effect of water fluoridation. J Dent Res. 1990; 69:723–7.
3. Orland FJ. An evaluation of bacteria and antibiotics in the dental caries process. Mil Surg. 1950; 106:345–7.
4. Azarpazhooh A, Main PA. Pit and fissure sealants in the prevention of dental caries in children and adolescents: a systematic review. J Can Dent Ass. 2008; 74:171–7.
5. Mickenautsch S, Yengopal V. Anticariogenic effect of xylitol versus fluoride - a quantitative systematic review of clinical trials. Int. Dent J. 2012; 62:6–20.
6. Weyant RJ, Tracy SL, Anselmo TT, Beltrán-Aguilar ED, Donly KJ, Frese WA. Topical fluoride for caries prevention: executive summary of the updated clinical recommendations and supporting systematic review. J Am Dent Assoc. 2013; 144:1279–91.
7. Chou R, Cantor A, Zakher B, Mitchell JP, Pappas M. Preventing dental caries in children <5 years: systematic review updating USPSTF recommendation. Pediatrics. 2013; 132:332–50.
8. Santos AP, Oliveira BH, Nadanovsky P. Effects of low and standard fluoride toothpastes on caries and fluorosis: systematic review and meta-analysis. Caries Res. 2013; 47:382–90.
9. Cagetti MG, Campus G, Milia E, Lingström P. A systematic review on fluoridated food in caries prevention. Acta. Odontol Scand.2013; 71:381-7.
10. Mickenautsch S, Yengopal V. Anticariogenic effect of xylitol versus fluoride-a quantitative systematic review of clinical trials. Int. Dent J. 2012; 62:6-20.
11. Fisher-Owens SA, Gansky SA, Platt LJ, Weintraub JA, Soobader MJ, Bramlett MD*et al*. Influences on children's oral health: a conceptual model. Pediatrics. 2007; 120: 510-520.
12. Petersen PE, Bourgeois D, Ogwa H, Estupinan-Day S, and Ndiaye C. The global burden of disease and risks to oral health. Bull world health organ. 2005; 83: 661-669.

13. Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. Caries Res. 2004; 38: 182-191.

14. Toffenetti F and Mjör IA. Secondary caries: a literature review with case reports. Quintessence Int. 2000; 31: 165-179.

15. Marsch PD and Martin MV. Oral Microbiology. 5th edition. Elsevier, 2009; pp 220-222.

16. Declerck D, Leroy R, Martens L, Lesaffre E, Garcia-Zattera MJ, Vanden BS, *et al*. Factors associated with prevalence and severity of caries experience in preschool children. Community Dent Oral Epidemiol. 2008; 36:168-178.

17. Fisher-Owens SA, Gansky SA, Platt LJ, Weintraub JA, Soobader MJ, Bramlett MD, *et al*. Influences on children's oral health: a conceptual model. Pediatrics. 2007; 120: 510-520.

18. Islam B, Khan SN and Khan AU. Dental caries: from infection to prevention. Med Sci. Monit. 2007; 13: 196-203.

19. Soben P. 2nd Ed. New Delhi: Arya Publications; 2000. Essentials of Preventive and Community Dentistry; pp. 468–505.

20. Harrison LB, Sessions RB, Hong WB. New York: Lippincott-Raven Publishers; 2001. Head and Neck Cancer a Multidisciplinary Approach; p. 253.

21. Sankaranarayanan R, Nair MK, Mathew B, Balaram P, Sebastian P, Dutt SC. Recent results of oral cancer research in Kerala, India. Head Neck. 1992; 14:107–12.

22. Moore SR, Johnson NW, Pierce AM, Wilson DF. The epidemiology of tongue cancer: A review of global incidence. Oral Dis. 2000; 6:75–84.

23. Chiang CP, Chang MC, Lee JJ, Chang JY, Lee PH, Hahn LJ, *et al*. Hamsters chewing betel quid or areca nut directly show a decrease in body weight and survival rates with concomitant epithelial hyperplasia of cheek pouch. Oral Oncol. 2004; 40:720–7.

24. Suen M. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2003. Cancer of the Head and Neck: Evaluation, Classification, and Staging; pp. 33-49.

25. Gupta PC, Ray CS. Smokeless tobacco and health in India and South Asia. Respirology. 2003; 8:419-31.

26. Lorente C, Cordier S, Goujard J. *et al*. Occupational Exposure and Congenital Malformation Working Group. Tobacco and alcohol use

during pregnancy and risk of oral clefts. Am J Public Health. 2000; 90(3):415–419.

27. Matthews M S, Cohen M, Viglione M, Brown AS. Prenatal counseling for cleft lip and palate. Plast Reconstr Surg. 1998; 101(1):1–5.

28. Hopper R A, Cutting C, Grayson B. Philadelphia, PA: Lippincott, Williams & Wilkins; 2007. Cleft lip and palate; pp. 201-225.

29. Matsune S, Sando I, Takahashi H. Insertion of the tensor veli palatini muscle into the eustachian tube cartilage in cleft palate cases. Ann Otol. Rhinol Laryngol. 1991; 100(6):439–446.

30. Mahdi A. Shkoukani M, Chen M, Vong A. Cleft lip-a comprehensive review Frontiers in pediatric, 2013; 1(53): 1-10.

31. Goodacre T, Swan M. Cleft lip and palate: current management. Paediaterics and Child Health, 2008; 18(6): 283-292.

32. Muhamad A, Azzaldeen A, Watted N. Cleft Lip and Palate; A Comprehensive Review. International Journal of Basic and Applied Medical Sciences ISSN: 2277-2103 (Online), 2014; 4(1):338-355.

33. Petersen PE, Bourgeois D, Ogawa H, Estupinan-Day S, Ndiaye C. 2005. The global burden of oral diseases and risks to oral health. Bull World Health Organ 83:661–669.

34. Li X, Kolltveit KM, Tronstad L, Olsen I. 2000. Systemic diseases caused by oral infection. Clin Microbiol Rev 13:547–558.

35. Becerik S, Ozçaka O, Nalbantsoy A, Atilla G, Celec P, Behuliak M, Emingil G. (2010). Effects of menstrual cycle on periodontal health and gingival crevicular fluid markers. J Periodontol. 2010 May; 81(5):673-81.

36. Ide M, Papapanou PN. 2013. Epidemiology of association between maternal periodontal disease and adverse pregnancy outcomes-systematic review. J Periodontol 84:S181–S194.

37. Bermejo-Fenoll A, Sánchez-Pérez A. (2004) Necrotising periodontal diseases. Med Oral Patol Oral Cir Bucal. 2004; 9 Suppl: 114-9; 108-14.

38. Van Dyke TE. 2008. The management of inflammation in periodontal disease. J Periodontol 79:1601–1608.

39. Canto AM, Müller H, Freitas RR, Santos PS. (2010) Oral lichen planus (OLP): clinical and complementary diagnosis. An Bras Dermatol. 2010 Oct; 85(5):669-75.

40. Jeffcoat MK, Reddy MS. 1991. Progression of probing attachment loss in adult periodontitis. J Periodontol 62:185–189.

41. Demirer S, Ozdemir H, Sencan M, Marakoglu I. (2007) gingival hyperplasia as an early diagnostic oral manifestation in acute monocytic leukemia: a case report. Eur J Dent. 2007 Apr; 1(2):111-4.

42. Mdala I, Olsen I, Haffajee AD, Socransky SS, Thoresen M, de Blasio BF. 2014. Comparing clinical attachment level and pocket depth for predicting periodontal disease progression in healthy sites of patients with chronic periodontitis using multi-state Markov models. J Clin Periodontol 41:837–845.

43. Mariotti A. (2005) Estrogen and extracellular matrix influence human gingival fibroblast proliferation and protein production. J Periodontol. 2005 Aug; 76(8):1391-7.

44. Huttenhower C, Gevers D, Knight R, Abubucker S, Badger JH, Chinwalla AT, Creasy HH, Earl AM, FitzGerald MG, Fulton RS, Giglio MG, Hallsworth-Pepin K, Lobos EA, Madupu R, Magrini V, Martin JC, Mitreva M, Muzny DM, Sodergren EJ, Versalovic J, Wollam AM, Worley KC, Wortman JR, Young SK, Zeng Q, Aagaard KM, Abolude OO, Allen-Vercoe E, Alm EJ, Alvarado L, Andersen GL, Anderson S, Appelbaum E, Arachchi HM, Armitage G, Arze CA, Ayvaz T, Baker CC, Begg L, Belachew T, Bhonagiri V, Bihan M, Blaser MJ, Bloom T, Bonazzi V, Brooks J, Buck GA, Buhay CJ, Busam DA, Campbell JL, *et al*. 2012. Structure, function and diversity of the healthy human microbiome. Nature 486:207–214.

45. Parizi JL, Nai GA. (2010) Amalgam tattoo: a cause of sinusitis? J Appl Oral Sci. 2010 Jan-Feb; 18(1):100-4.

46. Bascones-Martínez A, Muñoz-Corcuera M, Noronha S, Mota P, Bascones-Ilundain C, Campo-Trapero J. Host defence mechanisms against bacterial aggression in periodontal disease: basic mechanisms. Medicina Oral, Patologia Oraly Cirugia Bucal. 2009; 14(12):e680–e685.

47. Zhang L, Henson BS, Camargo PM, Wong DT. The clinical value of salivary biomarkers for periodontal disease. Periodontology 2000. 2009; 51(1):25–37.

48. Khalili J. Periodontal disease: an overview for medical practitioners. Liker'ka sprava. 2008; (3-4):10–21.

49. Armitage GC. Development of a classification system for periodontal diseases and conditions. Annals of Periodontology. 1999; 4(1):1–6.

50. Armitage GC, Cullinan MP. Comparison of the clinical features of chronic and aggressive periodontitis. *Periodontology 2000*. 2010; 53(1):12–27.

51. Petersen PE. The World Oral Health Report 2003: Continuous improvement of oral health in the 21st century--the approach of the WHO Global Oral Health Programme. Community Dent Oral Epidemiol. 2003; 31:3–23.

52. A.J. Singer, and D.A. Talan, Management of skin abscesses in the era of methicillin-resistant staphylococcus aureus, N Eng. J Med370, 2014, 1039-1047.

53. H. Mohan, Inflammation-acute and chronic, in: H. Mohan (Ed.), Essential Pathology for dental students, 1(New Delhi: Jaypee Brothers Medical Publishers (P) Ltd, 1996)68.

54. B. Shalu, Periodontal abscess, in: B. Shalu, B. Manish (Ed.), Periodontics revisited, 1 (New Delhi: Jaypee Brothers Medical Publishers (P) Ltd, 2011) 173.

55. F.A. Carranza, P.M. Camargo, The periodontal pocket, in: M.G. Newman MG, H.H. Takei, F.A. Carranza (Ed.), Carranza's clinical periodontology, 9 (Philadelphia: Saunder's Elsevier, 2003) 349-448.

56. M.M. Dello Russso, The post-prophylaxis periodontal abscess: etiology and management, Int. J Periodont Rest Dent 1, 1985, 29-37.

57. Gonçalves LS, Gonçalves BML, Fontes TV. Periodontal disease in HIV-infected adults in the HAART era: Clinical, immunological, and microbiological aspects. Arch Oral Biol 58: 1385-1396, 2013.

58. Wood NH, Blignaut E, Lemmer J, Meyerov R, Feller L. Necrotizing periodontal diseases in a semirural district of South Africa. AIDS Res Treat 2011: 638584, 2011.

59. Lamster IB, Grbic JT, Bucklan RS, Mitchell-Lewis D, Reynolds HS, Zambon JJ. Epidemiology and diagnosis of HIV-associated periodontal diseases. Oral Dis 3 (Suppl 1): S141-S148, 1997.

60. Glick M, Muzyka BC, Salkin LM, Lurie D. Necrotizing ulcerative periodontitis: a marker for immune deterioration and a predictor for the diagnosis of AIDS. J Periodontol 65: 393-397, 1994.

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