Journal of Oral Health & Dentistry
JOHD, 1(1): 15-24
www.scitcentral.com

Original Review: Open Access

Periodontal Medicine: Bridging the Gap between the Dental and Medical Professions: Part I

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Received May 7, 2018; Accepted June 7, 2018; Published June 27, 2018

ABSTRACT

The influence of systemic conditions on the oral environment and especially the periodontium has long been recognized and supported by scientific evidence. However, an evidence base for the influence of periodontal diseases on overall systemic health has only recently begun to be established. There is growing evidence that a number of complex human diseases are associated with opportunistic infections in periodontal medicine. As a consequence, there has been a resurgence of interest in oral microbial ecology, mucosal immunity, and associations with systemic conditions, such as prematurity and low birth weight, pulmonary diseases, cardiovascular diseases, and cerebrovascular diseases. This review will highlight the many advances and opportunities for improved health care in the 21st century.

INTRODUCTION

Medical emergencies can occur in any patient; however, they are most prevalent in geriatric or medically compromised patients. There is a rapidly growing segment of the population who's physical or psychosocial problems may complicate dental treatment. The elderly or medically compromised patient who is frequently taking one or more medications such as steroids, anticoagulants, cardiac drugs, or immunosuppressive agents may require special consideration before undergoing dental treatment. As ever-increasing numbers of such individuals seek dental care, it becomes the responsibility of the dentist to avoid adverse therapeutic interactions and to deal with medical emergencies when they occur [1-4] Periodontal diseases, now recognized as bacterial infections, are among the most common, chronic diseases of humans, affecting 5 to 30% of the adult population in the age group of 25 to 75+ years. Periodontal diseases are also among the most important causes of pain, discomfort, and tooth loss in adults[5-6] with an increasing likelihood of medical emergencies in this population, the practising dentist and auxiliary staffs are responsible for identifying patients with a potential for medical risk by obtaining a comprehensive pre-treatment physical evaluation [7-9].

Patient Evaluation at Dental office

The patient evaluation at dental office is performed to determine patients' physical and emotional status and how well they will tolerate a specific dental procedure. Little and King, in 1971, presented the reasons for an evaluation of general health in the dental office. They concluded that it should be done

a) To identify patients with undetected systemic disease that could be a serious threat to the life of the patient or whose condition could be complicated by dental treatment.

b) To identify patients who are taking drugs or medications that could adversely interact with drugs prescribed, that would complicate dental therapy, or that may serve as a clue to an underlying systemic disease the patient has failed to mention

c) To provide information for the dentist to modify the treatment plan for the patient in light of any systemic disease or potential drug interactions.

d) To enable the dentist to select and communicate with a medical consultant concerning the patient's possible systemic problems

e) To help establish a good patient-doctor relationship by showing patients the clinician's interest in them as individuals and concern for their overall well-being

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Citation: Patil P S, Chandanwale A, Pakhmode V & Tatiya H. (2018) Periodontal Medicine: Bridging the Gap between the Dental and Medical Professions: Part I. J Oral Health Dent, 1(1): 15-24.

Information obtained from the medical evaluation may prevent a medical emergency. A well-conceived evaluation of the patient includes, first the medical history which can be obtain in form of questionnaire and the personal interview, second is Present health status, that means The patient should be asked the date and results of the last complete physical examination. (eg. Diabetes, Blood pressure). Third is past medical history which includes whether patient is having any significant past history that affects the present treatment plan, fourth is allergies means The patient should be asked about allergies or reactions to any foods, medications, or environmental factors. Specifically, aspirin, local anaesthetics, antibiotics, and any other potential allergens that may be used in dental therapy should be mentioned. Fifth is Medication, it is imperative to determine the brand and/or generic name of the drug, why and by whom it was prescribed, the dosage, and the length of time the medication has been taken. Patients may not include medications used for allaying anxiety or for inducing sleep, such as tranquilizers and sedative-hypnotic drugs. Sixth is family history, which is taken to determine if there is a familial predisposition to diseases or if there are diseases in which inheritance is an important factor.

Finally, Positive findings should be summarized and recommendations recorded. This will enable the dentist and the dental staff to quickly review a patient‘s medical status at each visit and facilitate the diagnosis and treatment of any medical emergency that may arise. A comprehensive medical history is an important procedure that dentists must adopt and routinely use to ensure that their patients are receiving the optimum benefit from all available health resources.

Genetics and Clinical perspective

It has long been observed that unusual forms of periodontitis, such as disease affecting young individuals (early onset periodontitis), “run in families.” The evidence for a genetic influence on early onset periodontitis has been well reviewed in recent year [10]. Genetic diseases can be divided into three major categories: chromosomal disorders, Mendelian disorders, and non-Mendelian disorders.

Congenital chromosomal disorders are caused by an abnormal dose of normal genes. Because of a deficiency or excess of chromosomal material. Down syndrome (trisomy 21), which is caused by the presence of an extra chromosome 21, is a classic example of a chromosome abnormality.

Mendelian disorders are caused by a mutation in a single gene and, therefore, are also referred to as single-gene (or major gene effect) disorders. The inheritance patterns of Mendelian disorders may be described in terms of the classic patterns of how certain traits, such as autosomal dominant or autosomal recessive disorders, are transmitted through successive generations.

In Non-Mendelian Disorders Most common adult-onset diseases have a genetic component that cannot be explained by either a chromosomal abnormality or a major gene effect. In particular, the genetic influence of non- Mendelian disorders does not fit the typical inheritance patterns within families. These disorders are undoubtedly multifactorial; they are caused by a combination of genetic and environmental factors.

The practical use of genetic information offers the potential to change periodontitis treatment. Genetic predisposition to the onset of periodontitis means that some patients can be identified even before disease begins. This improves the chances of successful prevention. Genetic heterogeneity associated with disease also extends to treatment responsiveness. Distinguishing patients who are of good responders from those who are poor responders will allow more precise chemotherapeutic interventions because drug targets will be more precise.

The main barrier to widespread use of current technology is its poor record in predicting the patient’s future periodontal status. Clinical and biologic evaluations can tell the clinician about the current status of the patient’s periodontium but these signs, symptoms, and clinical judgments have relatively weak prognostic value. By focusing attention on both the etiology and modifiers of periodontitis, rather than on “furing” the results of disease, practitioners can anticipate, manage, and prevent disease much more effectively.

Cardiovascular Diseases and Oral Infections

The relationship between oral infections and cardiovascular disease is well known, particularly with respect to orally derived bacteraemia as a source of organisms that infect damaged heart valves causing bacterial endocarditis. Recently, evidence has emerged relating periodontal infections to coronary artery disease and stroke.

Infective endocarditis (IE) is a microbial infection of a native or prosthetic cardiac valve or surrounding cardiac tissue. It may be caused by a variety of microorganisms, including bacteria, fungi, rickettsia, or chlamydia. Recent evidence linking severe, generalized periodontitis with coronary artery disease, suggests that the periodontics must be prepared to provide safe yet effective therapy to patients with various types of heart condition. [11-12] Dentists may provide dental care for patients with any of these disorders, most often they are called upon to manage patients at risk of IE.[13] Dental procedures that involve manipulation of soft tissue and result in bleeding can produce transient bacteraemia. For example, *43%* of patients with periodontitis experienced transient bacteraemia following routine periodontal probing [14]. Transient bacteraemia may be induced by some surgical or nonsurgical periodontal treatment procedures. However, these bacteraemia rarely persist longer than 15 minutes and the majority dissipate

within 3 to *5* minute [15-16]. The risk of IE derived from transient bacteraemia associated with manipulation of dental tissues must be weighed against the cost and risk of complications associated with administration of systemic antibiotics.

There is some evidence that oral irrigation or use of air-abrasive polishing devices may induce bacteraemia when used inappropriately or in patients with poor periodontal health, and these devices are not recommended [17-20]. Rinsing with antimicrobial agents containing chlorhexidine gluconate or providence iodine prior to manipulation of dental tissues may reduce the overall bacterial bio load. This may be especially important in high risk patients and in those with poor oral hygiene.

The AHA recommendations for specific prophylactic antibiotic regimens for dental procedures are widely published. For most adults, oral administration of *2* g of amoxicillin I hour before the dental procedure is recommended. Clindamycin (600 mg I hour before the dental procedure), cephalexin/cefadroxil or azithromycin/clarithromycin are recommended as alternatives in patients that are allergic to penicillin. Intramuscular or intravascular antibiotic regimens are prescribed for patients that cannot take oral medications. The recommendations are considered adequate for patients that are at high risk from IE, including those with cardiac valve prostheses [21].

Prior to cardiac surgery, dental procedures associated with a high risk of significant bacteraemia should be accompanied by appropriate prophylactic antibiotic support. When possible, dental extractions should be accomplished at least 2 weeks prior to the heart surgery to allow adequate wound healing. Patients those are on anticoagulant regimen i.e. Aspirin which is often used as an antithrombotic agent because of its inhibition of platelet aggregation, most cardiologists prescribe very small daily dosages (80 to 325 mg). At these dose levels, the medication will not significantly alter bleeding time [22]. On occasion, however, patients on higher aspirin levels are at a slight risk for prolonged postoperative haemorrhage following periodontal therapy. For these individuals, the medication should be discontinued for *4* to *7* days prior to the scheduled procedure with the concurrence of the cardiologist [23].

DE Stefano and colleagues [24] found that periodontal disease and poor oral hygiene are stronger indicators of risk of total mortality and of coronary heart disease. They suggest that oral hygiene may be an indicator or a surrogate for lifestyle affecting personal hygiene and health care and might explain the relationship between periodontal disease and heart disease. Multiple studies showing the relationship between periodontal disease and heart disease, after adjusting for many factors associated with lifestyle, such as smoking and weight, suggest that the relationship is not simply explained by lifestyle. Also, the finding that the graded exposure of periodontal disease leads to an increased cumulative index of coronary heart disease argues against lifestyle as a simple explanation for this association [25].

The association between periodontal disease and cardiovascular disease or stroke could be due to residual confounders or incomplete control of confounders. As with most studies that adjust for possible confounders, the adjustments may not be complete, so associations of this magnitude may be due to residual confounders.

Further research will be needed to determine which, and to what extent, factors act singly or in concert to contribute to the formation of athermanous plaques. It is important to know the mechanisms, however, since they add evidence to support the association between periodontal infection and atherosclerosis. In addition, knowing the mechanisms may well lead to simple, cost-effective interventions that would moderate, in part, the contribution of infection to atherosclerosis.

Relationship between Periodontal and Respiratory Diseases

Respiratory diseases are responsible for a significant number of deaths and considerable suffering in humans. Accumulating evidence suggests that oral disorders, particularly periodontal disease, may influence the course of respiratory infection. It is possible that the teeth can serve as a reservoir for respiratory infection. Indeed, the notion that the oral cavity may influence the bacterial flora of the lower bronchi is not new. Oral bacteria can be released from the dental plaque into the salivary secretions, which are then aspirated into the lower respiratory tract to cause pneumonia**.** It has long been known that severe anaerobic lung infections can occur following aspiration of salivary secretions, especially in patients with periodontal disease [26].

Oral bacteria may also have a role in the exacerbations of Chronic Obstructive Pulmonary Disease (COPD). Laboratory studies suggest that oral anaerobes such as *p. gingivulis* can cause marked inflammation when instilled into the lungs of laboratory animals [27]. Lack of attention to oral hygiene results in an increase in the mass and complexity of dental plaque, which may foster bacterial interactions between indigenous plaque bacteria and acknowledged respiratory pathogens such as *P.aueruginosu* and enteric bacilli [28]. Dental plaque may therefore provide a reservoir for colonization of respiratory pathogens that can be shed into saliva. More recently, a prospective study of *57* consecutive patients admitted to medical ICU during a 3-month period assessed the colonization of dental plaque by respiratory pathogens [29]. The amount of dental plaque on the teeth of inpatients increased over time, as did the proportion of respiratory pathogens in their dental plaque.

Mechanisms of Action of Oral Bacteria in the Pathogenesis of Respiratory Infection

Several mechanisms can be envisioned to help explain how oral bacteria can participate in the pathogenesis of respiratory infection:

1. oral pathogens (such as *P. gingivulis, A. actinomycetemcomituns)* may be aspirated into the lung to cause infection
2. periodontal disease-associated enzymes in saliva may modify mucosal surfaces to promote adhesion and colonization by respiratory pathogens
3. periodontal disease-associated enzymes may destroy salivary pellicles on pathogenic bacteria
4. Cytokines originating from periodontal tissues may alter respiratory epithelium to promote infection by respiratory pathogens.

Several reports have documented a strong association between periodontal disease and an increased frequency of oral infections in nursing home resident [30]. However, there are no studies that have identified an association between poor oral hygiene and the increased incidence of pneumonia in such subjects. A possible link between poor oral hygiene and the increased incidence of pneumonia in nursing home residents has been suggested but no supporting evidence was provided [31].

The key role that is orpharyngeal bacterial colonization plays important role in the pathogenesis of bacterial pneumonia, several methods have been proposed to reduce or eliminate colonization in susceptible patients, such as those on mechanical ventilation. It is hypothesize that improved oral hygiene in the hospital setting may decrease the occurrence of oropharyngeal colonization by respiratory pathogens and thus decrease the risk of nosocomial pneumonia. One method, called selective digestive decontamination (SDD), uses antibiotics topically applied to the surfaces of the gastrointestinal tract (including the oral cavity) to reduce the carriage of pathogenic bacteria and thus to prevent respiratory infection [32-33]. However, while diminishing the colonization rate of pathogenic bacteria in the hospital setting, SDD does not appear to have an effect on the mortality rate and seems to foster the selection of antibiotic-resistant bacteria and cross-infection [34-35]. These findings have raised doubts about the widespread use of SDD. Other approaches to reduce colonization of these pathogens certainly deserve more study.

Chlorhexidine appears to be a reasonable choice for this as it has been shown to reduce plaque and salivary levels of bacteria by up to 85% [36]. Interestingly, chlorhexidine gluconate has been shown to reduce transfer of group B streptococci from mother to infant during parturition [37]. An interesting report by DeRiso and colleagues [38] suggests that a 0.12% chlorhexidine gluconate oral rinse reduced the overall nosocomial infection rate by 65% in *353* patients admitted to a cardiovascular ICU, and the incidence of total respiratory tract infections by 69%. These investigators also noted a *43%* reduction in the use of no prophylactic antibiotics in chlorhexidine-treated patients. Finally, overall mortality was reduced to I. 16% in the chlorhexidine-treated group versus 5.56% in the placebo group.

Variety of recommendations has been made to reduce the incidence of nosocomial pneumonia. Surveillance of potential pathogens, identification of high-risk patients, staff education, hand washing, and the proper use of gloves and gowns, all have a positive impact on reducing nosocomial pneumonia. Additional attention paid to oral hygiene may even further reduce the risk of nosocomial pneumonia. Unfortunately, little information is available concerning the effect of improved oral hygiene on infection rates in the hospital or nursing home setting. It would, therefore, seem reasonable to perform appropriate studies to evaluate the effect of improved oral hygiene on respiratory pathogen colonization in high-risk subjects.

Periodontal Disease and Diabetes

Oral complications of diabetes may include alterations in salivary flow and constituents, increased incidence of infection, burning mouth, altered wound healing, and increased prevalence and severity of periodontal disease. Xerostomia and parotid gland enlargement may occur in the diabetic individual. Diabetes patients may complain of burning mouth syndrome associated with decreased salivary flow. Dry mucosal surfaces are easily irritated and often provide a favourable substrate for the growth of fungal organisms. The incidence of candidiasis may be increased in patients with diabetes [39].

Diabetes is often associated with increased gingival inflammation in response to bacterial plaque [40]. This response may be related to the level of glycaemic control, with subjects with well-controlled diabetes having a similar degree of gingivitis as no diabetic individuals and poorly controlled diabetic subjects having significantly increased inflammation Increased gingival inflammation [41-42] may be seen in diabetic subjects even though plaque levels are similar to no diabetic controls.

Diabetic patients must be examined individually to assess their potential response to periodontal therapy. The mere presence of diabetes does not condemn the person to a less favourable periodontal outcome. A diabetic patient with good glycaemic control can be expected to respond in a fashion similar to the no diabetic subject. The presence of poor glycaemic control may place the patient at risk of a less favourable response. In addition, other factors such as smoking or poor plaque control may adversely affect the response to periodontal therapy in diabetic individuals, just as they may in a no diabetic person.

Patients who present to the dental office with intraoral findings suggestive of a previously undiagnosed diabetic condition should be questioned closely. Questions should be

targeted toward eliciting a clear history of polydipsia, polyuria, polyphagia, or recent unexplained weight loss. Patients should also be asked about family history of diabetes.

In patients with suspected poorly controlled diabetes, dental treatment should be limited initially to provision of emergency care [43]. Referral to the patient's physician should include a description of intraoral findings and a brief outline of the patient's dental treatment needs. The dental practitioner should request evaluation of the patient's glycaemic control and appropriate medical management prior to elective dental treatment. In known diabetic patients, it is important to establish the level of glycaemic control early in the examination process. This can be done through physician referral or review of medical records. Key considerations related to dental treatment of the diabetic patient include stress reduction, diet modification, inpatient versus outpatient care, antibiotic use, changes in medication regimens, and appointment timing [44-45].

At one time, a general recommendation was made for diabetic patients to have their dental appointments in the morning. This recommendation was also made for many other medically compromised patients. While morning appointments may be preferable for some diabetic patients, others may be better treated in the afternoon. Appointment timing often depends on the particular medication regimen used by each individual patient. When possible, it is best to plan dental treatment either before or after periods of peak insulin activity because hypoglycaemic reactions are more likely to occur when insulin levels are high [45].

Ensuring oral health in patients with diabetes requires an expanded scope of medical and dental knowledge. There is undoubtedly a close relationship between diabetes and periodontal disease, a relationship requiring further study and exploration. Diabetes increases the risk of periodontal destruction, especially in patients whose glycaemic control is poor. These same patients are most likely to report to the dental office with significant periodontal treatment needs. All diabetic patients should have routine dental evaluation and preventive therapy. The practitioner who understands the role of diabetes in the etiology of oral diseases, the potential for oral infections to influence glycaemic control, the current medical therapeutic approaches to diabetes, and the implications of diabetes on dental care provides the patient with the best chances of successful treatment outcomes.

Periodontal Medicine and Female Patients

Women's life cycle changes present unique challenges to the oral health care profession. Hormonal influences associated with the reproductive process alter periodontal and oral-tissue responses to local factors creating diagnostic and therapeutic dilemmas. It is imperative, therefore, that the clinician recognize, customize, and vary periodontal therapy according to the individual female and the stage of her life cycle.

Puberty

During puberty, the female experiences an increase in the production of sex hormones (estrogen and progesterone) that remains relatively constant following puberty throughout the normal female lifetime reproductive phase. There is also an increase in the prevalence of gingivitis without an increase in the amount of plaque [46]. Recent studies associated with puberty gingivitis indicate proportionately elevated motile rods, spirochetes, and *Prevotellu intermediu.* Statistically significant increases in gingival inflammation and in the proportion of *Prevotella intermediu* and *Prevotella nigrescens* were seen in puberty gingivitis [47].

Preventive care, including a vigorous program of oral hygiene, is vital. Milder gingivitis cases respond well to scaling and root planning with frequent oral hygiene instruction. Severe cases of gingivitis may require microbial culturing, antimicrobial mouthwashes and local site delivery, or antibiotic therapy. Supportive periodontal therapy visits may need increased frequency. Whenever possible, involvement of a parent or caregiver with home care procedures is recommended.

Menses

During the reproductive years, there are on-going changes in the concentration of the gonadotrophins and ovarian hormones during the monthly menstrual cycle. Estrogen and progesterone are steroid hormones produced by the ovaries during the menstrual cycle. The gonadotrophins follicle-stimulating hormone (FSH) and luteinizing hormone (LH) influence estrogen and progesterone to prepare the uterus for implantation of the egg. The concept that ovarian hormones may increase inflammation in gingival tissues and exaggerate the response to local irritants has been postulated by several studies. Gingival inflammation seems to be aggravated by an imbalance and/or increase in sex hormones [48].

Progesterone has been associated with increased permeability of the microvasculature, altering the rate and pattern of collagen production in the gingiva, increasing folate metabolism [49-50], altering the immune response. Gingival tissues have been reported to be more edematous and erythematous preceding the onset of menses in some individuals. In addition, an increase of gingival exudate has been observed during the menstrual period and is sometimes associated with a minor increase in tooth mobility [51]. Intraoral recurrent aphthous ulcers [52], herpes labialis lesions, and Candidae infections occur in some women as a cyclic pattern associated with the luteal phase of their cycle when progesterone is the highest.

During the peak level of progesterone (about 7 to 10 days prior to menstruation), premenstrual syndrome (PMS) also

occurs. There appears to be no significant differences in estrogen and progesterone levels between women who suffer from PMS and women who do not. Yet, women with PMS seem to have lower levels of certain neurotransmitters such as encephalin, endorphins, gamma amino butyric acid (GABA) and serotonin. Depression, irritability, mood swings, and difficulty with memory and concentration may be symptoms of neurotransmitter reduction.

For the women who have increased gingival bleeding and tenderness associated with the menstrual cycle, adherence to 3 to 4-month supportive periodontal therapy appointments is recommended. Antimicrobial mouth rinses prior to cyclic inflammation may be indicated. Particular emphasis should be placed on oral hygiene. Care should be taken during dental treatment to prevent stimulating the more sensitive gag reflex. The clinician should be aware that nonsteroidal anti-inflammatory medication, infection, and acidic foods exacerbate GERD. Fluoride rinses and/or trays, frequent periodontal debridement, and avoidance of mouthwashes with high alcohol content may reduce the associated gingival and caries sequelae.

The PMS patient may be difficult to treat due to emotional and physiologic sensitivity. Treat the gingival and oral mucosal tissues gently. Moisten gauzes or cotton rolls with a lubricant, chlorhexidine rinse, or water before placing them in the aphthous prone patient. Careful retraction of the oral mucosa, cheeks, and lips will be necessary in both the aphthous and herpetic prone patient. Since the hypoglycemic threshold is elevated, advise the patient to have a light snack prior to her appointment.

PREGNANCY

Pregnancy provides unique diagnostic and treatment challenges to the periodontal clinician. It is an opportunity to individualize care at a time when the patient may experience the most profound physiologic and psychologic changes in her life. Awareness exists regarding pregnancy and its effect on periodontal disease; however, recent evidence indicates an inverse relationship to systemic disease. Current research implies that periodontal disease may alter the systemic health of the patient as well as adversely affect the well-being of the foetus by elevating the risk of low-birth-weight, preterm infants.

In 1877, Pinard recorded the first case of “pregnancy gingivitis [53]. Pregnancy gingivitis is extremely common, occurring in approximately 30 to 75 percent of all pregnant women [54-55]. It is characterized by erythema, edema, hyperplasia, and increased bleeding. Histologically, the description is the same as gingivitis. The etiologic factors, however, are different despite clinical and histologic similarities. Pyogenic granulomas occur during pregnancy at a prevalence of 0.2 to 9.6 percent. The “pregnancy tumor” or “pregnancy epulis” are clinically and histologically indistinguishable from pyogenic granulomas occurring in women who are not pregnant or in men. They appear most commonly during the second or third month of pregnancy. The gingiva is the most common site involved (approximately *70%* of all cases), followed by tongue and lips, buccal mucosa, and palate [56]. They usually grow rapidly, bleed easily, and become hyperplastic and nodular. They may be sessile or pedunculated and may be ulcerated. Colour ranges from purplish red to deep blue, depending on the vascularity of the lesion and the degree of venous stasis. The lesion classically occurs in an area of gingivitis and is associated with poor oral hygiene. Often calculus is present. Osseous destruction is not usually associated with pyogenic granulomas of pregnancy.

Alterations in immunocompetency during pregnancy may create an exaggerated response in periodontal supporting structures. Periodontal status prior to pregnancy may influence the progression or severity as the circulating hormones fluctuate. The anterior region of the mouth is more commonly affected, and interproximal sites tend to be most involved [57]. Increased tissue edema may lead to increased pocket depths and relate to a transient tooth mobility [58] Anterior site inflammation may be exacerbated by increased mouth breathing, primarily in the third trimester from “pregnancy rhinitis.”

Periodontal Disease And Preterm Low-Birth weight Births

Due to the pioneering research of Offenbach and co-workers, evidence exists that untreated periodontal disease in pregnant women may be a significant risk factor for preterm (< *37* weeks) low-birth-weight (< 2,500 g) babies [59]. The relationship with genito-urinary tract infection and preterm low birth weight (PLBW) is well documented in human and animal studies. Periodontal researchers suspecting periodontal disease as another source of infection found that mothers of low-birth-weight infants, otherwise having low risk, had significantly more periodontal attachment loss than control mothers having normal-weight infants at birth. The current opinion is that PLBW occurs as a result of infection and is mediated indirectly, principally by the translocation of bacterial products such as endotoxin (lipopolysaccharide [LPS]) and by the action of maternally produced inflammatory mediators [60].

Perimylolysis or acid erosion of teeth may occur if “morning sickness” or esophageal reflux is severe and involves repeated vomiting of gastric contents. Severe reflux may cause scarring of the esophageal sphincter, and the patient may become a more likely candidate for GERD later in life.

Xerostomia is a frequent complaint among pregnant women. One study found this persistent dryness in *44* percent of pregnant participant [61].

A rare finding in pregnancy is ptyalism, or sialorrhea. This excessive secretion of saliva usually begins at *2* to *3* weeks of gestation and may abate at the end of the first trimester.

While its etiology has not been identified, ptyalism may result from the inability of nauseated gravid women to swallow normal amounts of saliva rather than from a true increase in the production of saliva [62].

Because pregnancy places the woman in an immunocompromised state, the clinician must be aware of the total health of the patient. Gestational diabetes, leukemia, and other medical conditions may appear during pregnancy.

The periodontal evaluation of the pregnant patient begins with a thorough medical history. This history should note any complications the patient has encountered in the pregnancy and record any previous previous miscarriages, recent cramping, spotting, or pernicious vomiting. If possible, the next step is to contact the obstetrician to discuss the patient's medical status, dental needs, and proposed treatment plan. The most important objectives in planning dental treatment for the pregnant patient are to establish a healthy oral environment and to obtain optimum oral hygiene levels. These are achieved by means of a good preventive dental program, consisting of nutritional counselling and rigorous plaque control measures in the dental office and at home.

It is prudent to avoid elective dental care other than good plaque control during the first trimester and the last half of the third trimester if possible. The first trimester is the period of organogenesis, when the foetus is highly susceptible to environmental influences. In the last half of the third trimester, there is a hazard of premature delivery because the uterus is very sensitive to external stimuli. Prolonged chair time may need to be avoided because the woman is most uncomfortable at this time. Further, there is a possibility that supine hypotensive syndrome may occur. In a semi-reclining or supine position, the great vessels, particularly the inferior vena cava, are compressed by the gravid uterus. By interfering with venous return, this compression will cause maternal hypotension, decreased cardiac output, and eventual loss of consciousness. Supine hypotensive syndrome can usually be reversed by turning the patient on her left side, thereby removing pressure on the vena cava and allowing blood to return from the lower extremities and pelvic area.

The second trimester is the safest period for providing routine dental care. The emphasis at this time is on controlling active disease and eliminating potential problems that could arise in late pregnancy. Extensive reconstruction procedures and major oral or periodontal surgery should be postponed until after delivery. Pregnancy tumors that are painful, interfere with mastication, or continue to bleed or suppurate after mechanical debridement may require excision and biopsy prior to delivery.

Dental radiography is one of the more controversial areas in the management of a pregnant patient. It is most desirable not to have any irradiation during pregnancy, especially during the first trimester, because the developing fetus is particularly susceptible to radiation damage [63]. However, the safety of dental radiography has been well established, provided features such as high-speed film, filtration, collimation, and lead aprons are used. Of all aids, the most important for the patient is the protective lead apron. Studies have shown that when an apron is used during contemporary dental radiography, gonadal and fetal radiation is virtually unmeasurable [64].

Another area of controversy involves drug therapy because drugs given to a pregnant woman can affect the fetus by diffusion across the placenta. A conservative approach is prudent, the dentist prescribing only the minimum effective dose and duration absolutely essential for the pregnant patient's well-being and only after careful consideration of potential side effects. The dentist may need to be familiar with the classification system established by the Food and Drug Administration (FDA) in 1979 to rate fetal risk levels associated with many prescription drugs. In periodontal therapy, the use of antimicrobial agents is common. During pregnancy, the clinician must weigh the benefits and the risks to both mother and fetus. Antibiotics with systemic effects cross the placenta and reach the fetus. The effect of a particular medication on the fetus depends on the type of antimicrobial, the dosage, the trimester, and the duration of the course of therapy [65]. At this date, there is inadequate research in relation to subgingival irrigation and local site delivery in relation to the developing fetus.

Oral contraceptives

Gingival tissues may have an exaggerated response to local irritants. Inflammation ranges from mild edema and erythema to severe inflammation with haemorrhagic or hyperplastic gingival tissues. It has been reported that there is more exudates in inflamed gingival tissues of OC users than in those of pregnant women [66]. Investigators have reported several mechanisms for the heightened response in gingival tissues. Kalkwarf reported that the response may be due to alteration of the microvasculature, increased gingival permeability, and increasing synthesis of prostaglandin [67]. Prostaglandin E is a potent mediator of inflammation. Jensen and colleagues found dramatic microbial changes in pregnant and OC user groups as compared with a no pregnant group [68].

A comprehensive medical history and an assessment of vital signs (particularly blood pressure) are extremely important in this group of patients. Treatment of gingival inflammation exaggerated by oral contraceptives should include establishing an oral hygiene program and eliminating local predisposing factors. It is also imperative that the patient be informed of their heightened risks and the need for meticulous home care and compliance with supportive periodontal therapy visits. Periodontal surgery may be indicated if there is inadequate resolution after initial therapy (scaling and root planning). Antimicrobial mouthwashes

may be indicated as part of the home care regimen. It may be advisable to perform extraction of teeth (especially of third molars) on non-estrogenic days (days 23 to 28) of the pill cycle, to reduce the risk of a postoperative localized ostitis [69].

CONCLUSION

Transmissible and opportunistic microorganisms are responsible for dental caries. Transmissible and opportunistic microorganisms are also responsible for periodontal diseases. In the case of periodontal diseases, the microbial-induced infection presents a substantial infectious burden to the entire body. Further, specific microorganisms within the microbial ecology associated with the disease process release toxins that invoke an inflammatory response. Bacteria, bacterial toxins, localized tissue response cytokines, and other inflammatory mediators enter the vascular circulation and may activate a systemic response. The subsequent pathogenesis of the disease process reflects gene-gene and gene-environment interactions. Nested in a complex interaction of host susceptibility, external exposures, and life-style behaviours, the management of health and disease will require interdisciplinary education, strategies, and health-care delivery.

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