Tikrit Journal Of Dental Sciences 1(2011) 71-76

Effects of Gingivitis During Pregnancy on Incidence of Low Birth Weight Baby in Al-Dour City, October 2009 - March 2010

Nahla K. Asaad BSc.,MSc.[(1)]

Key words
Gingivitis, pregnancy, low birth weight baby.

Abstract

It has been suggested that gingivitis during pregnancy could have a causal relationship with low weights for newborns. The objective of this study is to estimate the effect of gingivitis during pregnancy on the birth weight of newborns.

Pregnant women were randomly selected and whom their gum had inflamed were contained in Group 1 (or G1); and this sample size was 50 subjects. This group was compared with a control group of 50 pregnant women whom presented with healthy gums and contained in Group 2 (or G2). Mothers were examined by a dentist who was not informed of the group classified. A probe was used to measure extent and severity of gingivitis. The extension index (EI) and severity index (SI) of gingivitis were determined.Both groups of mothers were near in terms of maternal age, parity, weight, height, skin color, obesity, socioeconomic status, parity, antenatal care, abortion, bacteriuria, nutritional status, diabetes mellitus, previous hypertension, and preeclampsia. The characteristics of the newborns were: birth weight: G1 = 1854±590 gram and G2 = 3175±610 gram; gestational age: G1 = 34±6 weeks and G2 = 38±3 weeks; duration of stay in the neonatal intensive care unit: G1 = 1.8±0.6 days and G2 = 0.7±0.4 days. Average EI: G1 = 84.663±11.634 and G2 = 69.010±18.122 ($p<0.05$). Average SI: G1 = 1.217±0.623 and G2 = 0.705±0.404 ($p<0.05$). The study analysis indicated a marked relation between gingivitis and low birth weight. Our data suggested that gingivitis during pregnancy may be a risk factor for low weight at birth.

Introduction

In dentistry, it is known that chronic infection of the teeth and gums, even if it does not cause discomfort for the patient, may result in distant insidious lesions that may be fatal. In this sense, it has been described that the oral condition of patients should not be considered as separate problems, but rather in relation to the human body as a whole.[1]

In the beginning of the 20th century (1910), William Hunter stated that he had treated several patients with obscure complaints that disappeared after he requested the removal of their dental prothesis.[2] Recently, in the United States, there has been an increasing attention towards studies regarding the relationship of tooth extraction and cardiovascular diseases. In 1993, De Stefano et al, after an average 14-year follow-up study with 20,000 patients, observed that "dental disease is associated with an increased risk

(1) lecturer, Department of Basic Science, College of Dentistry, University of Tikrit.

of coronary heart disease".[3] In 1994, Collins et al. in a study on the effects of Escherichia coli and Porphyromonas gingivalis lipopolysaccharide on pregnancy outcome, suggested that maternal exposure to P. gingivalis prior to and during pregnancy can induce deleterious effects on the developing fetus.[4] The same authors also studied inflammatory mediator response and pregnancy outcome infected with Porphyromonas gingivalis. Results in that study indicated statistically significant increasing levels of both PGE2 and TNF-alpha, and fetal growth retardation of 24%.[5] More recently, Offenbacher et al. suggested that periodontal infection in pregnant women is a risk factor for preterm, low weight births.[6]

Gingivitis

Gingivitis can be defined as inflammation of the gingival tissue without loss of tooth attachment (i.e. periodontal ligament). Gingivitis is an irritation of the gums. It is usually caused by bacterial plaque that accumulates in the small gaps between the gums and the teeth. These accumulations may be tiny, even microscopic, but the bacteria in them produce foreign chemicals and toxins that cause inflammation of the gums around the teeth. This inflammation can, over the years, cause deep pockets between the teeth and gums and loss of bone around teeth, an effect otherwise known as periodontitis.[7]

Proper maintenance disrupts and removes this plaque. Once cleaned, plaque will begin to grow on the teeth within hours. However, it takes approximately 3 months for the pathogenic type of bacteria (typically gram negative anaerobes and spirochetes) to grow back into deep pockets and restart the inflammatory process. Calculus may start to reform within 24 hours. Ideally, scientific studies show that all people with deep periodontal pockets (greater than 5 mm) should have the pockets between their teeth and gums cleaned by a dental hygienist or dentist every 3–4 months.[8]Many dental professionals only recommend cleanings every 6 months. If the inflammation in the gums becomes especially well-developed, it can invade the gums and allow tiny amounts of bacteria and bacterial toxins to enter the bloodstream. The patient may not be able to notice this, but studies suggest this can result in a generalized increase in inflammation in the body and/or cause possible long term heart problems. Periodontitis has also been linked to diabetes, arteriosclerosis, osteoporosis , pancreatic cancer and pre-term low birth weight babies.[9]

When the teeth are not cleaned properly by regular brushing and flossing, bacterial plaque accumulates, and becomes mineralized by calcium and other minerals in the saliva transforming it into a hard material called calculus which harbors bacteria and irritates the gingiva. Also, as the bacterial plaque biofilm becomes thicker this creates an anoxygenic environment which allows more pathogenic bacteria to flourish and release toxins and cause gingival inflammation. Pregnancy, uncontrolled diabetes mellitus and the onset of puberty increase the risk of gingivitis, due to hormonal changes that may increase the susceptibility of the gums or alter the composition of the dentogingival microflora. The risk of gingivitis is increased by misaligned teeth, the rough edges of fillings, and ill fitting or unclean dentures, bridges, and crowns. This is due to their plaque retentive properties.[7, 9]

The symptoms of gingivitis are as follows: swollen gums, mouth sores, bright-red, or purple gums, shiny gums, swollen gums that emit pus, severe oral odor, gums that are tender, or painful to the touch, gums that bleed easily, even with gentle brushing, and especially when flossing, gum pockets, and bad breath.

Researchers analyzed government data on calcium consumption and periodontal disease indicators in nearly 13,000 U.S. adults. They found that men and women who had calcium intakes of fewer than 500 milligrams, or about half the recommended dietary allowance, were almost twice as likely to have gum disease, as measured by the loss of

attachment of the gums from the teeth. The association was particularly evident for people in their 20s and 30s.[9] Research says the connection between calcium and gum disease is likely due to calcium's role in building density in the alveolar bone that supports the teeth. Preventing gum disease may also benefit a healthy heart. According to physicians with The Institute for Good Medicine at the Pennsylvania Medical Society, good oral health can reduce risk of cardiac events. Poor oral health can lead to infections that can travel within the bloodstream.[7]Complications include: Tooth loss, Recurrence of gingivitis, Periodontitis, Infection or abscess of the gingiva or the jaw bones, Trench mouth (bacterial infection and ulceration of the gums).[7, 9]

Patients and Methods

A 100 Mothers (50 with gingivitis and 50 healthy gum) were selected from October 2009 to March 2010 in Al-Dour city attending Primary Health Care Centers (PHCCs) as part of their antenatal care. Group 1 was the sample that include pregnant women and gingivitis was diagnosed when they set for dental examinations. Group 2, (control group), was include pregnant women presented with no gingivitis. The selected women in this study were given information regarding our research for follow up to complete data collection and health education. Additional information were collected using a questionnaire designed with the objective of confirming data obtained from medical charts. Data related to clinical condition of the gum of pregnant women were always collected by the same dentist. Consequently, the probability of error was constant. The dentist was not given information as to the group of the patient being examined. Dental examination checked for presence of gingival recession; congestion, and active inflammation. In order to measure gingival retraction, depth of groove we employed a high-precision, millimeter probe, University of North Carolina (UNC-15). The extension and severity index of gingivitis were determined according to Carlos *et al*.[10] These indices are used as a simple method for epidemiological studies and, according to the authors, can be applied to various epidemiological data providing high comparability and low loss of information. The extension of the disease is determined by the number of teeth with gingival problems. The extension index is calculated simply by the percentage of areas examined that were affected. The severity index of the disease indicates the average criteria of inflammation of the gum, that is, the areas in a tooth in which exceeded 1 mm. Collected data were statistically analyzed by SPSS v.13.0 with Anova and T-test were used as measures of significance.[11]

Results

Table 1 summarizes the characteristics of pregnant women in groups 1 and 2. Both groups were near in relation to possible risk factors for low birth weight with no statistically significant differences between both of the two groups.Both groups in this study were similar for age, height, weight, skin color, and incidence of nutritional alterations. The social profile of both groups was also similar. Both groups presented similar data regarding primiparity, mothers who had insufficient or no prenatal exams.

Positive cases of low birth weight (LBW) was 66.0% in study group and 32.0% in control group. Birth weight was 1854±590 grams in first group, and 3175±610 grams in second group. Gestational age of newborns was 34±6 weeks for group 1 and 38±3 weeks for group 2. The mean duration of hospital stay was 1.8±0.6 days for group1 and 0.7±0.4 days for group 2 (Table 2). In group 1, the extension index of gingivitis of mothers with LBW was 84.663±11.634, while those with normal birth weight was 69.010±18.122. The severity indices of gingivitis for mothers with LBW was 1.217±0.623; while for those of normal birth weight was 0.705±0.404 (Table 3A).

Discussion

The near criteria in study and control groups was convenient to reduce the error resulted from multifactorial overlap regarding aetiology of LBW (Table 1). Mothers with gingivitis presented more than 2-fold incidence of LBW compared with mothers with healthy gingiva. Though this result presented a weak statistical significance (T-test = 0.9055 at $p<0.05$), it was agreed with Slots, 1998.[12] Most low birth weight (LBW) newborns in the first group were admitted to the Neonatal Intensive Care Unit (ICU). Their weight presented near half of that of the second group and this presented a significant relationship (T-test = 1.8541 at $p<0.05$) and also agreed with Slots, 1998.[12] There was no significant difference between gestational ages between study and control groups. The mean duration of hospital stay was about 2.5-fold higher for study group than for control group (Table 2). None of the obstetrical risk variables examined for this population presented a statistically significant association with low weight at birth of newborns. Conversely, however, in the comparison between the two groups, differences between indicators of gingivitis were statistically significant with a higher extension index presented for mothers with LBW more than those with no LBW of group 1. Similar result is presented comparing severity index (Table 3B). These differences suggest the possibility of a causal relation between maternal gingivitis and low weight. Our data presented the same trend found by Offembacher et al, 1998.[13] Currently, it is understood that the onset and continuation of periodontal disease is caused by a small group of bacteria, which is predominantly gram negative, anaerobic or microaeroph-ilic, and that colonizes the sub-gingival area. In a recent workshop on clinical periodontics, it was concluded that most cases of human periodontitis are caused by Porphyromonas gingivalis, Bacteroides forsythus, and Actinobacillus ctinomy-cetemcomitans [14].Infections caused by the referred microorganisms can turn into chronic reserves of lipopolysaccharide and stimulate the production of prostaglandin E2 (PGE2) and interleukin-1 beta, thus affecting the placental membrane through blood circulation. Recently, Offembacher et al have found significantly higher levels of prostaglandin E (PGE) on gingival crevicular fluid of mothers of premature, low-birth-weight newborns. In addition, they also found a significant inverse association between weight at birth and levels of PGE.[13]

It is possible that the results of Offembacher et al present an explanation for the association between periodontal disease and low weight at birth: the levels of PGE2 and TNF would increase progressively during gestation until a critical threshold is reached and labor is induced.[6] In this sense, the molecules produced in the periodontium can reach the circulation, cross the placenta, and increase levels of PGE and TNF in the amniotic fluid. Cytokines can also participate in the process of rupture of membranes. It is understood that the TNF and Interleukin-1 can induce the liberation of protease (collagenase, elastase, and others) from macrophages that, in turn, digest the fetal membrane, leading to its rupture. It is also possible that a set of underlying genetic and/or environmental factors act together towards putting the patient at risk for having both periodontal disease and premature, low-birth-weight newborns. Page, 2007, while analyzing the theories on focal infection and many diseases that have been related to periodontal pathological processes, emphasizes that gingivitis and other clinical diseases can frequently occur at the same time without any relation of cause and effect.[14, 15]

Conclusions and Recommendations

The implications of a possible causal relation between periodontal disease and low weight at birth cannot, however, be ignored. Low birth weight significantly increases risk for death, for neurological sequelae, and for unsatisfactory neurological development. It also increases healthcare costs since a large percentage of low-birth-weight newborns require intensive or intermediate care. If

periodontal diseases increase the incidence of low weight at birth, it seems clear that public health policies should also be directed towards periodontal healthcare of pregnantwomen.Consequently, perinatol-ogists and prenatal healthcare professionals should be aware of the importance of dental healthcare of pregnant women for both the health of the mother and, possibly, of the baby.

The data regarding association between gingivitis, alterations, and premature, low-birth-weight newborns indicate the necessity for broader, randomized studies comparing the pregnancy outcome of mothers with and without gingivitis and, moreover, comparing the results obtained with and without treatment of periodontal disease (in general) during pregnancy.

Table (1):- Characteristics of pregnant women of gingivitis (Group 1) compared to those of controls (Group 2); differences were not statistically significant. (G1 n=50, G2 n=50)

Characteristics	Group 1	Group 2
Age (years) (median, range)	26 (18-36)	25 (20-33)
Weight (kg)	65.4±13.0	66.7±13.2
Height (cm)	160.4±10.9	162.1±11.6
White skin (subjects)	46	48
Obesity	15	19
Low family income	17	16
Primiparous	30	28
Insufficient prenatal care	7	10
No prenatal care	4	4
Previous abortion	7	9
Previous LBW	8	11
Premature rupture of membrane	28	26
Placenta previa	2	4
Bacteriuria	35	30
Malnutrition	1	0
Diabetes	0	2
Previous hypertension	4	6
Preeclampsia	0	1

Table (2):- Characteristics of (Group 1) and control (Group 2).

Characteristics of newborns	Group 1	Group 2
LBWB*† (<2500g)	66.0%	32.0%
Weight at birth‡ (mean±SD)	1854±590g	3175±610g
Gestational age according to DLMP** (weeks)	34±6	38±3
Duration of stay at Neonatal ICU (mean±SD)	1.8±0.6	0.7±0.4

* DLMP = date of last menstrual period; ** LBWB = low birth weight baby; † T-test = 0.9055 ($p<0.05$); ‡ T-test = 1.8541 ($p<0.05$)

Table (3A):- Mean and standard deviation of extension and severity indexes of gingivitis in mothers with LBW babies compared to that of mothers of normal birth weight babies in group 1 (Analysis of Variance (ANOVA) for differences in index averages).

	LBWB		No LBW		Anova	
	Mean	SD*	Mean	SD*	F-ratio	P**
Extension index	84.663	11.634	69.010	18.122	4.996	0.023
Severity index	1.217	0.623	0.705	0.404	8.692	0.004

* SD = Standard Deviation; ** P = statistical significance

Table (3B):- Mean and standard deviation of extension and severity indexes of gingivitis in mothers of LBW newborns in Group 1 and of mothers in Group 2; Analysis of Variance (ANOVA) for differences in index averages.

	Group 1		Group 2		Anova	
	Average	SD*	Average	SD*	F-ratio	P**
Extension index	89.788	18.355	72.420	20.717	5.119	0.033
Severity index	1.377	0.626	0.754	0.413	8.963	0.006

* SD = Standard Deviation; ** P= statistical significance

References

1-Appleton JLT. Bacterial infection in dental practice. Philadelphia: Lea & Febiger; 1950. p.517-21.

2-Ring ME. Dentistry - an illustrated history. Toronto: CV Mosby Co; 1985. p.283.

3-DeStefano, *et al*. Dental disease and risk of coronary heart disease and mortality. BMJ 1993; 306:688-91.

4-Collins JG, Smith MA, and Arnold RR. The effects of *E. coli* and *P. gingivalis* LPS on pregnancy outcome in golden hamster. Infect Immun 1994; 62:4652-5.

5-Collins JG, Windley HW, and Arnold RR. Effects of a *Porphyromonas gingivalis* infection on inflammatory mediator response and pregnancy outcome in the hamster. Infect Immum 1994;62:4356-61.

6-Offenbacher S, Katz V, and Fertik G. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontology 1996; 67:1103-13.

7-The American Academy of Periodontology. Proceedings of the World Workshop in Clinical Periodontics. Chicago: The American Academy of Periodontology; 2009: I/23-I/24.

8-American Dental Hygienists' Association Position Paper on the Oral Prophylaxis.

9-Institute for Good Medicine at the Pennsylvania Medical Society, http://www.myfamilywellness.org/MainMenuCategories/FamilyHealthCenter/Heart/Oralhealth.aspx, 2009.

10-Carlos J, Wolf M, and Kingman G. The extend and severity index: as simple method for use in epidemiologic studies of periodontal disease. J Clin Periodontol 1986;13:500-4.

11-Saflol BS. Your guide to the statistical program SPSS. The Arabic institute for training and statistical researches: 90-4.

12-Slots J. Casual or causal relationship between periodontal infection and non-oral disease. J Dent Res l998;77:1764-65.

13-Ofembacher S, Jared HL, O´Reilly PG et al. Potential pathogenic mechanism of periodontitis associated pregnancy complications. Ann Periodontl 1998;3:230-50.

14-Page R, Kornman K. The pathogenesis of human periodontitis: an introduction. Periodontology 2007;14:9-11.

15-Consensus Report for Periodontal Disease: pathogenesis and microbial factors. Ann Periodontol 1996;1:926-32.