Relationship of Periodontal Health of Mothers with Pre Term Low Birth Weight Child

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Abstract:

Periodontitis is considered as a continuous pathogenic and inflammatory challenge at a systemic level, due to the large epithelium surface that could be ulcerated in the periodontal pockets. Thus the subgingival microbiota in patients with periodontitis provides a significant and persistent gram-negative bacterial challenge to the host. These organisms and their products, such as lipopolysaccharides (LPS), have ready access to the periodontal tissues and the circulation via the sulcular epithelium, which is frequently ulcerated and discontinuous. The World Health Organization defines Preterm Birth as any live birth at less than 37 weeks gestation. Delivery at less than 32 weeks is termed very preterm, and delivery at less than 28 weeks extremely preterm. Birth weights are considered to be low if <2500g, very low if <1500g, and extremely low if <1000g.34 Preterm birth (PTB) that occurs at less than 37 weeks gestation & associated low birth weight of fewer than 2500 grams (about 5 ½ grams) represents the major cause of neonatal morbidity and, among survivors, a major contributor to long-term disability, the present review article is a try to explain the correlation between the periodontal health of mothers with preterm low birth weight child.

Key Words: Periodontal health, Preterm birth, Lipopolysaccharides, Gingival Health.

Introduction:

Periodontal disease comprises several conditions affecting the health of the periodontium. Periodontal disease is among the most common disease of humans, affecting 5 to 30% of the adult population. Clinically, bleeding on probing is the first predictor of the presence of periodontal disease followed by the development of periodontal pockets and loss of clinical attachment level. This occurs because of loss of periodontal ligament

and disruption of its attachment to cementum, by the migration of the epithelial attachment along the root surface and also the resorption of alveolar bone occurs.[1]

Periodontitis can be considered a continuous pathogenic and inflammatory challenge at a systemic level, due to the large epithelium surface that could be ulcerated in the periodontal pockets. The total surface area of the pocket epithelium in contact with subgingival bacteria and their products in a patient with generalized moderate periodontitis has been estimated to be approximately the size of the palm of an adult hand, with even larger areas of exposure in cases of more advanced periodontal destruction. Thus the subgingival microbiota in patients with periodontitis provides a significant and persistent gram-negative bacterial challenge to the host. These organisms and their products, such as lipopolysaccharides (LPS), have ready access to the periodontal tissues and the circulation via the sulcular epithelium, which is frequently ulcerated and discontinuous.[2]

Even with treatment, complete eradication of these organisms is difficult, and their re-emergence is often rapid. Bacteremias are common after mechanical periodontal therapy and also occur frequently during normal daily function and oral hygiene procedures. This fact allows bacteria and their products to reach other parts of the organism, creating lesions at different levels. Even some periodontopathogens, like Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans, can directly invade cells and tissues. This exposition to Gram-negative bacteria and their products can generate an immuno-inflammatory response with potential damages to different organs and systems.[3]

WD Miller (1891) published a classic article entitled "The human mouth as a focus of infection." In 1900, William Hunter wrote an article entitled "Oral sepsis as a cause of disease". In 1911, Frank Billings, Professor of Medicine and head of the focal infection research team at Rush Medical College and Presbyterian Hospital in Chicago, replaced the term oral sepsis with ''focal infection''. He defined a focus of infection as a ''circumscribed area of tissue infected with pathogenic organisms'' and the term focal infection implied.[4]

Haffajee & Socransky[5], several specific subgingival oral bacteria including Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Tannerella forsythia, Campylobacter rectus, Fusobacterium nucleatum, and spirochaetes are associated with severe forms of periodontal disease. A group of pathogens, not normally found in the oral cavity, except as transients, has been associated with periodontal disease, including Enterobacteriaceae, Pseudomonaceae, Klebsiella spp, Actinobacteria, and others, such as Staphylococcus aureus and Candida albicans.[6]

Seymour supports the concept that the immune system responds to plaque microorganisms. He found that the infiltrate in the periodontal lesion consists of lymphocytes and macrophages; whereas T lymphocytes predominate in the stable lesion, the proportion of B cells and plasma cells is increased in the progressive lesion.

Host hyper-responsiveness or reactivity is induced by periodontal infection and includes activation of neutrophils, which migrate to the area of periodontal infection, and induction of antibodies, both of which appear to be protective. Extracellular degradation is usually thought to occur at neutral pH values. Consequently, proteinases of the metallo and serine families will be optimally functional and seem to be most responsible for the initial phases of degradation.

This leads to connective tissue destruction and production of proinflammatory cytokines, such as IL-1, resulting in alveolar bone resorption. These cytokines can cause activation of fibroblasts, which then produce major metalloproteinases that destroy the extracellular matrix. Pro-inflammatory cytokines such as IL-1, IL-6, and TNF-α, lead to activation of osteoclasts, thereby causing bone resorption. Genetic polymorphisms in the pro-inflammatory cytokines IL-1 and TNF α have been associated with adult periodontitis.30 The oral pathogens and inflammatory mediators such as interleukin-1 (IL-1) and tumor necrosis factor-α (TNF-α) from periodontal lesions immediately reach the bloodstream inducing systemic inflammatory reactants such as acute-phase proteins, and immune effectors including systemic antibodies to periodontal bacteria.[7]

The World Health Organization defines Preterm Birth as any live birth at less than 37 weeks gestation. Delivery at less than 32 weeks is termed very preterm, and delivery at less than 28 weeks extremely preterm. Birth weights are considered to be low if <2500g, very low if <1500g, and extremely low if <1000g.34 Preterm birth (PTB) that occurs at less than 37 weeks gestation & associated low birth weight of fewer than 2500 grams (about 5 ½ grams) represents the major cause of neonatal morbidity and, among survivors, a major contributor to long-term disability.[8]

Sharma et al (2009)[9] Low literacy level and low per capita income were found to be significant risk factors of LBW in a study done in India.

Socio-economic factors associated with preterm labor include social class, (usually assessed by earnings and education), working conditions (professional status, ergonomic environment, working hours), physical and traveling activities, daily life activities, lifestyle, family status, and the psychosocial state as related to past and current pregnancy history together with current stress factors. The most stressful events were related to family illness, mortality, disruption, violence, or financial distress.[10]

Low weight and Body mass index (BMI) at conception or delivery, as well as poor weight gain during pregnancy, is associated with LBW, prematurity, and maternal delivery complications.[11]

Abrams and Selvin (1995)[12] Lack of weight gain in the second trimester especially correlates with decreased birth weight.

The growth of the fetus is affected by the nutrients and oxygen it receives from the mother. Maternal diet in pregnancy has little effect on birth weight but may program the infant. A fetus may adapt to undernutrition by

modifying metabolism, this may take the form of changing rates of hormone production, slowing the growth rate.[13]

Gardosi (2005)[14]Maternal and fetal genotypes in the etiology of preterm birth. A higher risk of spontaneous preterm delivery has been associated with genetically driven excessive amniotic fluid IL-I or with a disturbance of bioavailability and/or bio-response of this cytokine, which is central to the pro-inflammatory reaction to infectious stimulants. The fetus also has a role in pre-term birth; the fetus recognizes a hostile intrauterine environment and may precipitate labor by premature activation of the fetal–placental parturition pathway

Small women typically have smaller infants. If a woman begins pregnancy weighing less than 100 pounds, the risk of delivering an SGA (Small-for-gestational-age) infant is increased at least twofold.54 Brooks et al, 1995 concluded that the environment provided by the donor mother was more important than the genetic contribution to birthweight.[15]

Lanning et al (1983)[16] noted that pregnant hamsters challenged with Escherichia coli LPS had a malformation of fetuses, spontaneous abortions, and low fetal weight. The work by Lanning and co-workers demonstrated that infections in pregnant animals could elicit many pregnancy complications including spontaneous abortion, preterm labor, low birth weight, fetal growth restriction, and skeletal abnormalities.

Offenbacher et al (2005)[17] found that maternal C. rectus infection-induced placental inflammation and decidual hyperplasia as well as a concomitant increase in fetal brain IFN-γ. Maternal infection with C. rectus increased mouse pup mortality and also affected the hippocampal region of the neonatal brain, suggesting that maternal infection with C. rectus may also affect perinatal neurological growth and development.

Dasanayake (1998)[18] in a 1:1 matched case-control study (n = 55 pairs) evaluated the hypothesis that poor oral health of the pregnant woman is a risk factor for LBW. The effect of periodontal and dental caries status of the woman at the time of delivery on the birth weight of the infant was evaluated by using conditional logistic regression analyses while controlling for known risk factors for LBW. Mothers of LBW infants were shorter, less educated, married to men of lower occupational class, had less healthy areas of gingiva and more areas with bleeding and calculus, and gained less weight during the pregnancy. Conditional logistic regression analyses indicated that mothers with more healthy areas of gingiva had a lower risk of giving birth to an LBW infant. The risk of LBW was higher in mothers who had no or late prenatal care (OR = 3.9). They concluded that poor periodontal health of the mother is a potential independent risk factor for LBW.

Dasanayake et al (2001)[19] followed a predominantly African American and socioeconomically homogeneous group of 448 women from the second trimester of their first pregnancy and observed the periodontal pathogen-specific maternal serum IgG levels concerning the birth weight of the infant while controlling for known risk factors for LBW. Porphyromonas gingivalis (P.g.)-specific maternal serum IgG levels were higher in the LBW

group compared to the normal birth weight (NBW) group (p=0.004). Women with higher levels of P.g.-specific IgG had higher odds of giving birth to LBW infants (odds ratio [OR] = 4.1; 95%; confidence interval [CI] for odds ratio = 1.3 to 12.8). This association remained significant after controlling for smoking, age, IgG levels against other selected periodontal pathogens, and race. Thus they concluded that low birth weight deliveries were associated with a higher maternal serum antibody level against P. gingivalis at mid-trimester.

Konopka et al (2003)[20] assessed the relationship between periodontal diseases and PLBW in the population of women from the Lower Silesian Region (Poland), and the evaluation of prostaglandin E2 (PGE2), interleukin-1 beta (IL-1 beta) levels in gingival crevicular (GCF) and blood serum in women with PLBW and women giving birth on time as well as secretion of these proinflammatory mediators in whole blood after bacterial lipopolysaccharide stimulation. In the studied population women over 28 years and exposed to medical risk factors had more frequent PLBW occurrence probability. In primiparous over there was a 4 times greater probability of preterm labor, and in case of the severe and generalized periodontitis presence, there is a 3.9 times higher possibility of PLBW compared to women with healthy periodontium. In all women with PLBW there is a significantly higher PGE2 and IL-1 beta concentration in GCF, and in primiparous also PGE2 level in blood serum, compared to controls.

Mokeem et al (2004)[21] examined the prevalence and relationship between periodontal disease and preterm low birth weight (PLBW) among Saudi mothers at King Khalid University Hospital in Riyadh, Saudi Arabia. The prevalence of the PLBW was found to be 11.3%, and the prevalence of periodontal disease was high among the study population. The risk of PLBW remained high with increasing periodontal disease (odds ratio [OR] 4.21, 95% confident interval [CI] 1.99-8.93) despite controlling the other risk factors such as age, smoking, and social class. They concluded that there is a correlation between periodontal disease and PLBW in King Khalid University Hospital Riyadh.

Moliterno et al (2005)[22] examined 150 mothers, The periodontal examination included measurements of probing pocket depth (PPD) and clinical attachment loss (CAL) in six sites from all existing teeth, except for third molars. There were significant associations found with low birth weight (LBW) babies with periodontitis (odds ratio (OR) 3.48) Periodontitis was considered a risk indicator for LBW in this sample, similar to other risk factors already recognized by obstetricians.

Jarjoura et al (2005)[23] in an observational study involving 83 preterm cases (<37 weeks gestation) and 120 term delivery controls found preterm birth to be associated with severe periodontitis, i.e. five or more sites with clinical attachment loss ≥3 mm, adjusted OR = 2.75, (95% CI 1.1–7.54).

Alves et al (2006)[24] assessed the periodontal status of purpura and determined its possible relationship with preterm low birth weight (PLBW) delivery. The sample included 59 women seen at two maternity hospitals The Periodontal Screening and Recording (PSR) were used for periodontal assessment. Nineteen mothers had

premature and low birth weight babies (gestational age below 37 weeks and birth weight below 2,500 g – a group I), and 40 had mature, normal-weight babies (gestational age over 37 weeks and birth weight over 2,500 g – group II). There was a higher rate of periodontal disease in group I (84.21% – 16/19) as compared with group II (37.5% – 15/40) the data also showed a significant association between periodontal disease and PLBW (OR = 8.9 – 95% CI: 2.22 - 35.65; p = 0.001). It was concluded that maternal periodontal disease was an associated factor for prematurity and low birth weight in that sample.

Radnai et al (2006)[25] undertook a case-control study to detect whether initial chronic localized periodontitis could be a risk factor for preterm birth (PB) and fetal growth restriction. A significant association was found between PB and initial chronic localized periodontitis, the criteria being bleeding at ≥50% of the examined teeth and having at least at one site at >4mm probing depth (p = 0.0001). The adjusted odds ratio for initial chronic localized periodontitis was 3.32, 95% CI: 1.64-6.69. The average weight of newborns of mothers with periodontitis was significantly less than that of the women without periodontitis (p = 0.002). The results support the hypothesis that initial chronic localized periodontitis of pregnant women could lead to PB and birth-weight reduction.

Jeffcoat and co-workers (2001)[26, 27] also found a positive association between maternal periodontal disease and preterm birth in a comparable US cohort study involving 1313 pregnant subjects. Complete periodontal, medical, and behavioral assessments were made between 21 and 24 weeks gestation for each subject. Gestational age of the infants were determined following delivery, and logistic regression modeling was performed to assess any relationship between periodontal disease and preterm birth while making adjustments for other known risk factors. Notably, subjects with severe or generalized periodontal disease had an adjusted OR of 4.45 (95% CI: 2.16–9.18) for preterm delivery (<37 weeks) as compared with periodontally healthy subjects. The adjusted OR increased with advancing prematurity to 5.28 (95% CI: 2.05–13.60) before 35 weeks gestational age and to 7.07 (95% CI: 1.70–27.4) before 32 weeks gestational age. Hence, mothers with severe periodontal disease were four to seven times more likely to deliver a preterm infant relative to mothers with periodontal health.

Lopez et al (2002)[28, 29] investigated whether the maintenance of the mothers' periodontal health after 28 weeks' gestation reduces the risk of PLBW. The incidence of PLBW was 2.5% in periodontally healthy women and 8.6% in women with PD (p=0.0004, relative risk = 3.5, 95% CI: 1.7 to 7.3). Risk factors significantly associated with PLBW were previous PLBW, PD, fewer than 6 pre-natal visits, and low maternal weight gain. PD was associated with both preterm birth and low birth weight, independent of other risk factors.

Romero et al conducted a study to determine whether maternal periodontal disease (PD) could be associated with the nutritional condition of newborns. After controlling for traditional risk factors for premature childbirth and low birth weight, 69 mothers were selected: 13 were periodontally healthy and 56 had varying stages of PD. A decrease in the average newborn's weight and gestational age was observed as the mother's level of PD

increased. Correlation analysis demonstrated a highly significant clinical relationship between more severe PD and lower birth weight ($r = -0.49$; $P < 0.01$); a highly significant relationship was also clinically demonstrated between increasing PD severity and decreasing gestational age of the newborn babies ($r = -0.59$; $P < 0.01$). There were significant differences in the weight and gestational age of the newborns of mothers with PD. These data suggest that PD in pregnant women could be a clinically significant risk factor for preterm deliveries and low birth weight.

Marin et al[30] undertook a study to evaluate the proposed association between periodontal disease and infant birth weight. Periodontal disease in normal Caucasian pregnant women, older than 25 years was found to be statistically associated with a reduction in infant birth weight.

Moreu et al (2005)[31] determined the influence of periodontal status on low-birth-weight pre-term delivery. 96 pregnant women were examined in their first, second, and third trimester to record plaque scores, clinically assessed gingival inflammation and probing depth (mean depth and percentage of sites with a depth of > 3 mm). No statistically significant association was found between gestational age and periodontal parameters. No significant relationship was found between low-weight delivery and plaque index measurements, although the association with gingival index was close to significant. A relationship was observed between low-weight birth and probing depth measurements, especially the percentage of sites of > 3 mm depth, which was statistically significant ($p = 0.0038$) even when gestational age was controlled for. According to these results, periodontal disease was found to be a significant risk factor for low birth weight but not for preterm delivery.

Toygar et al (2007)[32] correlated maternal periodontal disease with birth outcomes in a Turkish population and evaluate maternal periodontal health. The study consisted of 3,576 Turkish women who gave birth within 24 hours of the onset of labor. The adjusted odds ratio was generated from various logistic regression models. The mean birth weight and weeks of gestation decreased as the CPITN level increased ($P<0.001$ for both). They concluded that maternal periodontal disease may be a risk factor for an adverse pregnancy outcome.

Agueda et al (2008)[33] determined the association between periodontitis and the incidence of preterm birth (PB), low birth weight (LBW), and preterm low birth weight (PLBW). One thousand and ninety-six women were enrolled. Periodontal data, pregnancy outcome variables, and information on other factors that may influence adverse pregnancy outcomes were collected. Data were analyzed using a logistic regression model. The incidence of PB and LBW was 6.6% and 6.0%, respectively. The incidence of PLBW was 3.3%. PB was related to the mother's age, systemic diseases, the onset of prenatal care, previous PBs, complications of pregnancy, type of delivery, the presence of untreated caries, and the presence of periodontitis (odds ratio 1.77, 95% confidence interval: 1.08–2.88). LBW was related to the mother's smoking habits, ethnicity, systemic diseases, previous LBW babies, complications of pregnancy, and type of delivery. PLBW was related to the mother's age, the onset of prenatal care, systemic diseases, previous LBW babies, complications of pregnancy, and type of delivery. The factors involved in many cases of adverse pregnancy outcomes have still not being

identified, although systemic infections may play a role. This study found a modest association between periodontitis and pre-term birth.

Mitchell-Lewis et[34] al investigated the relationship between periodontal infections and preterm births and/or low birth weight in a cohort of young, minority, pregnant and post-partum women. Periodontal treatment was provided to 74 pregnant women and the incidence of preterm and/or low birth weight as compared with the 90 women studied after the birth of their babies. Although the incidence of adverse pregnancy outcomes was higher in women without periodontal treatment, this difference was not statistically significant (the authors consider that it is could be due to the small sample size.) However, preterm and/or low birth weight mothers had significantly higher levels of Tannerella forsythensis and Campylobacter rectus.

López et al.[35] found a reduction in the rate of preterm births and/or low birth weight in women that have received periodontal treatment before the 28th gestation week when they were compared with women that have not received any treatment. This reduction was significant for healthy periodontal women compared with women with gingivitis87 and with periodontitis.

Jeffcoat et al.[36] in a pilot study, studied 366 women with periodontitis between the 21st and 25th gestation weeks in three intervention groups: 1-dental prophylaxis plus placebo capsule; 2-scaling and root planning plus placebo capsule; and 3-scaling and root planning plus metronidazole capsule. They conclude that performing scaling and root planning in pregnant women with periodontitis may reduce preterm births in that population, but adjunctive metronidazole therapy did not improve pregnancy outcomes.

Michalowicz et al.[37] studied the effect of scaling and root planning before the 21st gestation week, plus monthly tooth polishing in 823 pregnant women. They did not find significant differences between treatment and control groups in birth weight or in the rate of delivery of infants that were small for gestational age, although there were more spontaneous abortions or stillbirths in the control group.

Madianos et al(2002)[38] analyzed the association between periodontitis and an increased risk of coronary heart disease and preterm and/or low birth weight deliveries. Only one cohort study and four case-control studies met the established criteria. Of these four studies, two considered periodontitis clinical indicators and the other two only microbiological data. Of the three studies that clinically evaluated periodontitis, two found a significant association between periodontitis and adverse pregnancy outcomes. However, the multivariate model in both studies was not adjusted adequately for the confounding variables, and both studies were carried out in a predominantly Afro-American population, which interfered with the extrapolation of the results to other racial groups. The study with negative results inadequately measured the exposition (CPITN). The authors concluded that better designed observational and intervention studies were needed.

Scannapieco et al (2003)[39] published a systematic review with 12 studies, three of which were intervention studies, although only one was randomized. The authors concluded that periodontal disease may be a risk

factor for preterm birth and low birth weight but there was no clear evidence that periodontal disease has a causal role in adverse pregnancy outcomes. Hence additional longitudinal, and intervention studies were needed to validate this association and to determine whether it was a causal relationship.

Xiong et al (2006)[40] identified 25 studies (13 case-control, nine cohort, and three controlled trials) that focused on preterm low birth weight, low birth weight, pre-term birth, birth weight by gestational age, miscarriage, or pregnancy loss, and pre-eclampsia. Of these, 18 suggested an association between periodontal disease and increased risk of adverse pregnancy outcome and seven found no evidence of an association. The conclusion was that although there is evidence of an association between periodontal disease and increased risk of preterm birth and low birth weight, especially in economically disadvantaged populations, potential biases and the limited number of randomized controlled trials prevents a clear conclusion.

Vettore et al (2006)[41] published a systematic review based on 36 studies. Twenty-six showed positive associations between periodontal disease and adverse pregnancy outcomes and 10 did not show this association. They noted clear differences between studies concerning the measurement of periodontal disease and adverse pregnancy outcomes. Moreover, they reported that most studies did not control for confounders, thus raising doubts about conclusions that can be made from them. The authors concluded that, although 26 of the 36 studies included in this review consider a positive relationship between periodontal disease and adverse pregnancy outcomes, there is no sound scientific justification to recommend screening of periodontal disease in pregnant women as a means to reduce such outcomes.

Khader and Ta'ani (2005)[42] identified 40 articles but only five met the quality criteria to be included in their analysis. The authors concluded that periodontitis in pregnant women significantly increases the risk of preterm birth or low birth weight, but without convincing evidence that treatment of periodontal disease will reduce the risk of preterm birth. This meta-analysis has important limitations due to the reduced number of articles and their heterogeneity.

Vergnes & Sixou (2007)[43] pooled data from 17 observational studies totaling 7151 women, 1056 (14.8%) of whom delivered a pre-term and/or low birth weight infant. The pooled estimate for the risk of mothers with periodontal disease to have a pre-term and/or low birth weight infant was 2.83 (95% CI: 1.95–4.10, $p < 0.0001$). For the outcome ''pre-term birth'' alone, the overall OR was 2.27 (95% CI: 1.06–4.85, $p < 0.05$), and for the outcome ''low birth weight'' alone the OR was 4.03 (95% CI: 2.05– 7.93, $p < 0.0001$). It was concluded that there was a probable association between periodontal disease and these adverse pregnancy outcomes, but it should be noted that they found significant statistical heterogeneity across studies.

Fogacci et al (2011)[44] collected data from only RCTs on the effect of periodontal therapy on preterm birth and LBW. The search resulted in 14 clinical studies. Ten articles met the inclusion criteria for preterm birth and four for LBW. Five meta-analyses on preterm birth were performed according to different criteria: 1) use

of probing depth and attachment loss for periodontitis definition 2) controlling for multiparity 3) controlling for previous preterm birth, 4) controlling for genitourinary infections and 5) all the previous criteria. In all meta-analyses, the effect of periodontal treatment on preterm birth and LBW was not statistically significant. Results of this meta-analysis did not support the hypothesis that periodontal therapy reduces preterm birth and LBW indices.

Ahmad Haerian-Ardakani et al (2013)[45] 88 ex-pregnant women who had attended to Gynecology Department of Hospitals and birth centers in Yazd for delivery from 2009-2010, with no systemic problems before or during pregnancy recorded in their medical history were chosen and allocated into two equal groups. The case group has consisted of 44 women who gave birth to infants weighing less than 2500 gr, and the control group gave birth to infants weighing more than 2500 gr. The mean age of both groups was 24 years.

4900 sites were examined in the case group and 5168 sites in the control group. The mean number of sextants with CPITN grade IV (or periodontitis) was significantly higher in the case group (p=0.0006 Mann-Whitney test) but the mean number of sextants with CPITN grade zero or healthy gingiva (p=0.042), grade I or mild gingivitis (p=0.002), grade II or established gingivitis (p<0.0001) was significantly higher in the control group, Percentage of the sextants diagnosed with periodontitis (CPITN grade III and IV) in women with LBW infants (case group) was 1.6 times more than the control group. The number of sites that had bleeding on probing was significantly higher in the case group (p<0.0001, student's t-test). The amount of supragingival calculus was also significantly higher in the case group (p=0.007, student's t-test). Among the LBW risk factors, only the previous history of LBW babies was significantly higher in the case group (p=0.0081). There was no significant difference between the two groups in other risk factors: husbands' job, infant's sex, and mother's educational level (p=0.068 Chi-square test). Maternal weight gain was significantly higher in the control group.

MarcelaYang Hui Zi et al (2015)[46] Periodontitis is associated with induction of premature births and other gestational complications, which may compromise the subject throughout life. Periodontal pathogens may be involved in this process, directly inducing fetal abnormalities, or inducing the inflammatory response and the contraction of myocytes, inducing prematurity. However, there are still no data on differences induced by different oral microbial communities. Furthermore, the current data had not provided conclusive answers for the effect of periodontal treatment in preventing PT deliveries and other gestational complications, and definitive intervention protocols were not established.

Mohammad Reza Karimi et al (2016)[47] conducted a study on 264 pregnant mothers; Nine percent of mothers had babies with weights less than 2500gr at birth (LBW). 21% of mothers included in the primiparity subgroup categorized under case group had babies with low weight at birth which reveals a significant difference In the multiparity subgroup/control group, 3% of mothers had babies with low weight at birth, while18% of mothers

included in multiparity subgroup categorized under case group had babies with low weight at birth, and this reveals a significant difference.

Conclusion:

All pioneer studies conclude here that there is an important link between the periodontal health status of mothers with preterm low birth weight child, mothers who maintain comprehensive periodontal health status during their pregnancy, have delivered a child with good health but at the same time mothers who have not maintained their respective periodontal health status during pregnancy, have delivered a child with preterm low birth weight. Proper examination and diagnosis of periodontal health staus of mothers should be made compulsory and at the same time, gynecologists and endocrinologists also should be made aware of this disease correlation.

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