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Relation of periodontal pathogens in placenta with low birth weight babies

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Abstract
Aim: To evaluate the possible correlation between periodontal pathogens in placenta with low birth weight babies.
Materials and Method: The study included 30 pregnant females distributed equally into study (delivered preterm low birth weight babies) and control group (delivered at normal term with normal weight babies). At the time of delivery, sample of placental extract was collected. Within two days postpartum clinical periodontal parameters of mothers i.e. subgingival plaque and calculus sample was collected. Placental extract and subgingival plaque and calculus samples were subjected to anaerobic culture, and various microorganisms present were identified.
Results: A significant difference ($p<0.05$) was found between no. of weeks of pregnancy, weight of baby at birth, no. of teeth with probing depth (PD) ≥4mm, no. of teeth with clinical attachment loss (CAL) ≥4mm, percentage (%) of sites with bleeding on probing (BOP), between study and control groups.
Conclusion: Positive correlation was seen between placental and plaque microorganisms in preterm group, indicating periodontitis to be a potential risk factor for preterm low birth weight babies.

Keywords: periodontitis, preterm low birth weight, risk factor

Introduction

Preterm infants (born with a low birth weight) represent major social and economic public health problem in developing/developed nations [1, 2]. The risk factors for PLBW include older (> 34 years) and younger (<17 years) maternal age; African-American ancestry; low socioeconomic status; inadequate prenatal care; drug, alcohol and tobacco abuse; hypertension; genitourinary tract infection; diabetes mellitus; multiple pregnancies and smoking [3]. Some adverse pregnancy outcomes are thought to be associated with subclinical infections including periodontitis. Interventional studies of Lopez *et al*. in 2002 [4]. Concluded that periodontal treatment reduces significantly the incidence of PB or LBW in women with periodontitis, **w**hereas Michalowicz *et al*., [5] found no statistical significant difference and periodontal treatment did not significantly alter the rates of PB. Offenbacher *et al* [6, 7] showed significant differences and they concluded that periodontal treatment reduces the incidence of PB.

Animal studies have supported the hypothesis of a significant relationship between an increase in serum PGE_2 induced by experimental *Porphyromonas gingivalis* and *Escherichia coli* infections in pregnant hamsters resulting in growth deficit and fetal mortality [8]. Many cases of histologically confirmed chorioamnionitis are not associated with active infection of the genitourinary tract and the results of culture are negative, both of which indicate that local infection is not the sole cause of this condition [9, 10]. Hematogenous spread of infection from oral cavity to amniotic fluid was suggested by Hill [11]. Placenta is the only source of nutrition from mother to the fetus, but can also act as source of infection in the same way.

Using immunocytochemistry, the presence of *Porphyromonas gingivalis* antigens in placental tissues can be detected [12]. The biological effects of P. gingivalis on intrauterine tissues remain unclear. It can be detected in chorionic tissues of hospitalized high-risk pregnant women, and may induce interleukin-6 and interleukin-8 production via TLR-2 in chorion-derived cells causing preterm rupture of the membrane [13].

Patients with preterm delivery and generalized periodontal disease had a higher frequency of chorioamnionitis and funisitis [14]. Although some recent studies failed to demonstrate an association [15], other reports—including animal, and human epidemiological and interventional studies—have suggested that poor periodontal status may result in preterm delivery and low birth weight. [16] The presence of periodontal pathogens in placenta/placental extract has never been clearly elucidated. So the purpose of the present study was to evaluate the possible correlation between microorganisms present in placental extract and in subgingival plaque and calculus, with the pregnancy outcomes.

Material and Method

The study included 30 pregnant females (15 each in study and control group), aged 18 -35 years who had given birth at GMC, Jammu between July 2017 to June 2019. Consent form was signed by all the participants. The study group included women who had delivered preterm low birth weight babies (pregnancy< 37 weeks and weight < 2500gm), and females who had delivered at normal term and normal weight babies were included in the control group.

Inclusion Criteria

All women who had ≥20 teeth; had periodontitis defined as the presence of at least 4 teeth with probing depth ≥4mm and clinical attachment loss ≥ 2mm; and had bleeding on probing at ≥35% of tooth sites, were included in the study group. And women with healthy periodontium were included in control group.

Exclusion Criteria

Patients having severe infection of genital or urinary system (including bacterial vaginosis), pre-eclampsia, prophylactic antibiotics for invasive procedures, obstetric abnormalities (including placenta previa, hydramnion, gestosis or deformities of uterus), systemic problems such as diabetes, hypertension and hypothyroidism, history of smoking, drinking and other tobacco related habit and history of drug abuse were excluded from the study.

Clinical Examination

Within two days postpartum, detailed medical history was taken with the help of preformed questionnaire. A full mouth periodontal examination was carried out for every subject and the data was recorded for no of teeth present, bleeding on probing, probing depth and clinical attachment loss by a William graduated probe at four sites per tooth according to WHO Oral health surveys; 2013 [17].

Placental Extract Samples

Placental extract samples were collected at the time of delivery by the Obstetrician performing the procedure. Samples were transferred into vial containing transport media (Robertson's cooked meat broth) and stored at 4˚C before transferring to the laboratory for processing [18].

Plaque and Calculus Sample

In each subject, 2 periodontal sites with the deepest periodontal pockets were selected for microbial sampling. Supragingival plaque was removed and the sample sites were isolated from saliva. All the samples were obtained by means of sterile Gracey curettes. The curette was inserted into the pocket and subgingival plaque and calculus was collected by a single scaling stroke. The collected plaque and calculus mass was immediately transferred into vials with transport media (Robertson's cooked meat broth). All samples were thereafter transported to the laboratory of the division of Microbiology, and kept at 4˚C. Routine anaerobic bacteriological cultures were studied from placental extract & periodontal plaque and calculus samples. Processing was completed within 4 weeks from sample collection [19].

Statistical Analysis

The descriptive and inferential analysis of the data was done by using IBM SPSS. Statistics Windows, Version 20.0. (Armonk, NY: IBM Corp). Chi-square test was used for inter group comparison. Spearman Rank Correlation test was used for seeing correlation between plaque and placental microbial count.

Results

Table 1 show values expressed in terms of mean±SD, except for education and gestational diabetes which was expressed in terms of number (percentage). By applying the chi-square test no significant difference was found between age and the total no. of teeth between study and control groups ($p<0.05$). A significant difference was found between no. of weeks of pregnancy, weight of baby at birth, no. of teeth with probing depth (PD) ≥4mm, no. of teeth with clinical attachment loss (CAL) ≥4mm, percentage (%) of sites with bleeding on probing (BOP), between study and control groups. In microbiological profile a significant difference was present between study and control groups for no. of *A. actinomycetecomitans* (Aa), *P. gingivalis* (Pg) and *micrococcus* colonies in both plaque and placenta samples. A significant difference was also present between study and control groups for total no. of colonies in plaque and placenta samples. Some of the placenta extract samples were found to be sterile in control group.

In table 2, plaque and placenta microbial count were found to be correlated for all 5 groups of microorganisms ($p<0.01$). So, results of our study clearly indicate that in the study group females, Aa, Pg and micrococcus have a high count in both plaque and placenta samples, which were not seen in the control group.

Table 1: Comparison among the groups for demographic, periodontal and microbial parameters

Parameter	Study Group (n=15)	Control Group (n=15)	Statistical Significance	
			z/χ^2	p
Demographic Profile				
Age (Mean±SD)	26.07±2.69	27.27±2.55	1.210	0.233
Education, above 12 (No., %)	11 (73.3%)	12 (80%)	0.186	0.666
Medical and Gestational Profile				
Gestational Diabetes (No., %)	1 (6.7%)	2 (13.3%)	0.370	0.543
No. of Weeks of pregnancy (Mean±SD)	34.00±1.51	38.87±0.83	4.715	<0.01*
Weight of baby at birth (in kg) (Mean±SD)	2.23±0.30	3.08±0.16	4.675	<0.01*
Periodontal Health Profile				
No. of teeth with PPD ≥4 mm (Mean±SD)	5.53±0.99	1.13±0.64	4.763	<0.01*
No. of teeth with CAL ≥4 mm (Mean±SD)	5.60±0.83	1.27±0.59	4.780	<0.01*
Percentage of sites with BOP (Mean±SD)	47.60±9.58	4.55±1.17	4.671	<0.01*
Total No. of teeth (Mean±SD)	27.87±1.36	27.27±1.83	0.253	0.806
Microbiological Profile (represented in exponential form *i.e.* 10^n – all values represent *n.*				
a) Plaque				
No. of AA colonies (Mean±SD)	2.87±2.48	0.53±0.92	2.556	0.02*
No. of PG colonies (Mean±SD)	3.47±2.23	0.80±1.01	3.080	0.003*
No. of Fn colonies (Mean±SD)	0.93±1.62	0.27±0.70	1.129	0.436
No. of Pseudo colonies (Mean±SD)	1.07±1.87	0.40±0.83	0.756	0.595
No. of Micrococcus colonies (Mean±SD)	2.27±1.94	0.83±0.21	2.764	0.015*
Total No. of colonies(Mean±SD)	10.60±3.27	2.40±0.83	4.859	<0.01*
b) Placenta				
No. of AA colonies (Mean±SD)	2.73±2.34	0.13±0.52	3.203	0.007*
No. of PG colonies (Mean±SD)	3.27±2.09	0.13±0.52	3.800	<0.01*

No. of Fn colonies (Mean±SD)	1.00±1.73	0.13±0.52	1.567	0.117
No. of Pseudo colonies (Mean±SD)	1.27±0.83	0.40±0.83	1.200	0.367
No. of Micrococcus colonies (Mean±SD)	2.33±2.02	0.47±0.99	2.708	0.016*
Total No. of colonies (Mean±SD)	10.60±3.09	1.27±1.10	4.746	<0.001*

*: statistically significant

Table 2: Correlation between Plaque and Placental Microbial Count

Parameter	r	P value
No. of Aa colonies	0.913	<0.001*
No. of Pg colonies	0.901	<0.001*
No. of Fn colonies	0.921	<0.001*
No. of Pseudo colonies	0.791	<0.001*
No. of Micrococcus colonies	0.984	<0.001*
Total No.	0.936	<0.001*

R: Spearman Rank Correlation test, *: statistically significant

Discussion

A significant difference was found for probing depth, clinical attachment loss and bleeding on probing between case and control group, showing that case group females had periodontitis, while control group had healthy periodontium. Maternal age, total number of teeth, education level and gestational diabetes status were controlled factors. The above results were in comparison with the Moreu G *et al* 2005 [20].

A significant difference was present between the number of colony forming unit per millimetre in preterm and term group in plaque and placental sample. *P. gingivalis, A. actinomycetecomitans* and *micrococcus* were most commonly encountered microorganisms in case group for both plaque and placental extract samples. This shows that periodontal pathogens can affect placenta and can later affect pregnancy outcomes. Spread of infection may have a hematogenous route from oral site to placenta, this have been supported by findings of Bearfield *et al*., who have found that bacteria from infected gums may spread to uterus and fetus [21]. Ebersole *et al*., found that women who delivered preterm had significantly lower antibody level of P. gingivalis during second trimester than women who delivered at term [22].

Engebretson *et al*. in 2000 [23] determined from a study of 164 women, mothers of preterm had significantly higher levels of periodontal pathogens. Furthermore, they suggested that periodontal treatment in pregnant women may substantially reduce the risk of having premature babies with LBW.

Limitation of the present study was its small sample size, nutritional level, socioeconomic status, oral hygiene practices of patients not evaluated and the culture technique used because of the limited resources available. So, further studies with large sample size and better microbiological analysis are required to draw definite conclusion.

Conclusion

It can be concluded that positive correlation was seen in our study between placental and plaque microorganisms in preterm group (study group). This may be indicative of periodontitis being a potential causative factor for preterm low birth weight babies. Further more studies are required with better microbiological analysis techniques and larger sample size to validate the same.

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