PERIODONTAL STATUS OF POSTPARTUM WOMEN IN RELATION TO PRETERM BIRTH AND LOW BIRTH WEIGHT AT KENYATTA NATIONAL HOSPITAL

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DECLARATION

I declare that this thesis is my original work and has not been presented for the award of a degree in any other university.

Signed: _____ Date: _____

This thesis has been submitted for examination with the approval of my supervisors from University of Nairobi.

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DEDICATION

This work is dedicated to my daughter Lisa Kaittany.

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LIST OF ACRONYMS

AAP	American Academy of Periodontology
IL- 1β	Interleukin 1- beta
KNH	Kenyatta National Hospital
LPS	Lipopolysaccharide
WHO	World Health Organization
PDL	Periodontal ligament
PGE_1	Prostaglandin E_1
PGE_2	Prostaglandin E_2
TNF-α	Tissue necrosis factor alpha
PLBW	Preterm birth and low birth weight
PPROM	Preterm premature rupture of membranes
ROM	Rupture of membranes
LBW	Low birth weight
NBW	Normal birth weight
PT	Preterm birth
GI	Gingival index
PS	Plaque score
CAL	Clinical attachment loss
SD	Standard deviation
SPSS	Statistical Package for Social Sciences

UWC	University of the Western Cape
MPH	Masters in Public Health
MSc.Dent	Master of Science in Dentistry
MClin Dent	Masters in Clinical Dentistry
PG-DIP RM	Post graduate Diploma in Research Methods

DEFINITION OF TERMS

Periodontitis – This is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both. (CDC / AAP 1997)

Preterm birth – This is the delivery of an infant after 20 weeks gestation but less than 37 weeks of gestation[1].

Low birth weight – This is an infant weighing less than 2500g at birth[1].

ABSTRACT

Background: Periodontal diseases in Kenya are highly prevalent. Gingivitis has been reported to affect up to 90% while chronic periodontitis affects 1-10% of the population. Preterm birth and low birth weight (PLBW) are leading causes of perinatal complications worldwide and contribute upto 70% of perinatal mortality. About 50% of the causes of preterm birth are idiopathic and a large proportion of PLBWS are of unknown aetiology (70%), therefore identification of the risk factors is paramount. While some studies have associated periodontitis with preterm birth and low birth weight pregnancy outcomes, others have found contradictory results.

There is no available literature in Kenya on the association of preterm birth and low birth weight among post partum women with periodontal disease. Premature birth, low birth weight and periodontal diseases continue to be fundamental health problems despite efforts to control for all known risk factors.

Aim: The aim of this study was to investigate the periodontal status of post partum women and its relationship with preterm birth and low birth weight pregnancy outcomes.

Study design: This was a descriptive cross-sectional study which was carried out among post partum women at the labour ward in Kenyatta National Hospital.

Methods: Two hundred and forty six participants were recruited into the study. A validated semi-structured, interviewer-administered questionnaire was used to collect sociodemographic data, past medical history; self reported gingival symptoms as well as the oral health practices of the study group. Information of the weight of the infant

at birth was retrieved from the medical records. The gestational age which was calculated from the last menstrual period and via ultrasound where available was retrieved from the medical records. Oral hygiene status and periodontal status were recorded on the clinical examination form (Appendix IV). The Silness and Löe 1964 plaque index and the Löe and Silness 1963 gingival index were used to assess the oral hygiene status and gingival inflammation respectively. Periodontitis was assessed using periodontal probing depth (PPD), recession and clinical attachment loss (CAL).

Data analysis and presentation: Statistical Package for Social Sciences (SPSS) version 20.0 for Windows was used for data analysis. Chi-square, Fishers tests, Independent t-tests and ANOVA tests were used for analysis. Pearson Correlation was also done to find the relationship between variables. Data was presented in the form of frequency diagrams, tables and pie charts.

Results: Two hundred and forty six study participants were examined. Majority (43%) of the participants were aged between 26-30 years with an age range of 18-42 years. The mean gestation age was 37 months with a range of 20-42 months. The average birth weight was 3015g with a range of 500-4690grams. Slightly more than half of the participants (57.3%) brushed their teeth once daily. There was 100% prevalence of gingivitis and no mother suffered from gingival overgrowth. The prevalence of severe periodontitis was 3.7 %(9), 27.2 %(67) or moderate periodontitis and 69.1 %(170) for mild periodontitis. The average probing depth was 2.8mm (S.D±0.37) and the average CAL was 2.34mm (S.D±0.42). The prevalence of preterm birth was 19.9% (49) and low birth weight was 12.6 % (31) while prevalence of combined PLBW was 6.5% (16). There was a positive correlation between the

mean gingival score and mean plaque score (r=0.534, p= 0.0001). There was a negative correlation between the mean gingival score and gestation age(r=-0.029, p=0.647) and birth weight(r=-0.100, p=0.118). There was no statistically significant relationship between periodontitis and preterm birth and low birth weight. (X^2= 2.72, df=2, p=0.250; X2= 1.45, df = 2, p= 0.483). None of thee mothers that had preterm birth or low birth weight infant suffered from severe periodontitis.

Conclusion: There was no association between periodontal disease and preterm birth and low birth weight.

Recommendations: Large prospective cohort studies are required in this area to investigate any associations between periodontitis and preterm and low birth weight and identify other risk factors.

CHAPTER ONE: INTRODUCTION AND LITERATURE REVIEW

Periodontal diseases are the second commonest oral diseases and affect the tooth supporting structures[2]. In Kenya, up to 90% of the population suffer from gingivitis while 1-10% suffers from chronic periodontitis[3, 4]. Other studies have reported that the prevalence of periodontitis is estimated to be about 80% among Kenyan adults with severe periodontitis affecting 50% of adults aged 45- 54 years[5]. Prevalence of periodontitis increases with age with 95% of adults over 50 years having severe disease[5]. Pregnancy gingivitis is initiated by plaque and exacerbated by sex hormones[6]. The incidence of pregnancy gingivitis has been reported at varying degrees ranging from 30 to 100% with an incidence during the first trimester of 67.49%, 74.19% in the second and 79.17% during the third trimester[7].

Several studies done over the last decade have associated periodontal disease with preterm birth and low birth weight (PLBWS) [8, 9, 10]. Preterm birth and low birth weight have been reported as leading causes of perinatal problems worldwide and accounts for a significant percentage of infant mortality. Preterm birth and low birth weight contributes to 70% of perinatal mortality[11] and 50% long term neurologic morbidity[10,12] with increased chances of developing neuronal, respiratory, gastrointestinal, cardiovascular and congenital diseases[13]. Preterm birth and low birth weight are the leading causes of infant mortality among black infants[14]. In the United States an incidence of 6-15% has been reported[15] while regionally prevalence of PLBW is about 16%–19%[16]. In Kenya, the prevalence of low birth weight infants was reported in 2005-2009 at 8%[17]. The prevalence of preterm birth and low birth weight

has not reduced significantly worldwide and regionally despite efforts to control the known risk factors.

About 70% of preterm and low birth weights are idiopathic while 50% of the causes of preterm birth are of unknown aetiology and therefore identification of the risk factors is paramount[15].

There is no available literature in Kenya on the association of periodontal disease and preterm birth and low birth weight pregnancy outcomes. Therefore, the aim of this study will be to determine the periodontal status of post partum women and its association with preterm birth and low birth weight.

1.1 LITERATURE REVIEW

Neonatal morbidity continues to be recognized internationally as a public health priority. More than 15 years since the launch of the safe motherhood initiative, the neonatal mortality in Africa continues to rise. Neonatal mortality is estimated at 31 deaths per 100 live births. Globally 13 million babies are born before 37 weeks and the rates are highest in low and middle income countries[17]. The top three causes of newborn death in Africa are infections (29%), prematurity (25%), and asphyxia (24%) [18].

1.2 Periodontal disease

Periodontitis is a chronic bacterial infection that affects the tooth supporting structures. Periodontal disease is the second commonest cause of tooth loss[2]. In Africa, gingivitis affects 50% of the population while 35% are affected by varying degrees of periodontitis[3]. In Kenya, up to 90% of the population suffer from gingivitis while 1-10% suffers from chronic periodontitis[4]. Prevalence of 53% has been reported in US adults with at least one periodontal site with attachment loss of 3mm or more[4].

Periodontal disease is caused by microorganisms within the dental plaque, which elicit an inflammatory host response, and ultimately result in destruction of the periodontium. Dental plaque is the most important causative factor in the development of dental caries and periodontal diseases. The tissue destruction results in clinical attachment loss due to the migration of the junctional epithelium apically, bone loss and pocket formation. The prevalence of attachment loss has been reported to increase steadily with age from 35.7% for the 30-39 year old age group to a high of 89.2% for the 80-90 year old age group in the United States[20]. Predisposing factors to

periodontal disease include age, race, smoking, economic status, education status, number of pregnancy, tooth brushing frequency, previous dental visits among others[21].

Gingivitis is modified by endocrine changes as seen in puberty, pregnancy, diabetes mellitus, use of medications like oral contraceptive pills and malnutrition. The exacerbation of disease activity during puberty, menstruation, and pregnancy appears to be caused by hormonal fluctuations on the cellular and immunological functions of the host. In pregnancy, these changes are highly prevalent even in the presence of low levels of plaque[20] and the signs of gingivitis and the amount of plaque are closely related after parturition than during pregnancy [5,21].

In the last decade, periodontal infections have been associated with different systemic diseases e.g. osteoporosis, diabetes mellitus, respiratory diseases, preeclampsia, cardiovascular diseases, infections and preterm birth and low birth weight [22].

1.3 Pregnancy and periodontal disease

Pregnancy is a physiological state characterized by an increase in oestrogen and progesterone hormones which are responsible for the changes that occur in women at specific phases of their life starting from puberty[20].

Several gingival changes occur in pregnancy which includes increased gingival probing depths, increased gingival inflammation especially on the marginal gingival and interdental papilla, oedema, pitting, increased gingival crevicular fluid flow, increased bleeding on probing, and increased tooth mobility. The progression or severity of gingivitis may advance during pregnancy due to the influence of fluctuating hormones[5]. Hormone levels in pregnancy rise dramatically with

progesterone levels rising to 100ng/ml, ten times the concentration at peak luteal phase of menses while estradiol reaches 30 times the normal. Estrogens regulates cellular proliferation, differentiation and keratinization whereas progesterone influences microvasculature permeability, alters the rate and pattern of collagen production and increases the metabolic breakdown of folate which is necessary for tissue maintenance. These hormones regulate cellular processes via intracellular receptors with the resultant effects are as a result of increased concentration of the unbound hormone which diffuses through the cell membrane. Gingival tissues have progesterone and oestrogen receptors thus are target organs for sex hormones. This could explain why probing depths, increasing number of gingival sites and erythema have been shown to increase upto 1month postpartum after which they decrease[20].

Similarly, the maternal immune response is suppressed in pregnancy with decreased neutrophil chemotaxis, depression of cell mediated immunity, phagocytosis and decreased T-cell response due to elevated progesterone levels. Ovarian hormones stimulate the production of prostaglandins PGE_1 and PGE_2 which are potent mediators of inflammation thus gingival inflammation increases markedly in pregnancy after which they decrease [20].

The incidence of pregnancy gingivitis has been reported at varying degrees ranging from 73.57%, of which the incidence during the first trimester is 67.49%, the second 74.19% and the third, 79.17%[7]. During the second trimester of pregnancy, the proportion of Gram-negative anaerobic bacteria in dental plaque increases with respect to aerobic bacteria[19]. *Porphyromonas gingivalis* increases progressively during pregnancy with a peak in the third trimester which can be explained by the high levels

of progesterone, a growth factor for this pathogen followed by an abrupt decrease after delivery[23]. Similarly increased gingival crevicular fluid, PGE_2 levels have been suggested as a marker of current periodontal disease activity and decreasing birth weight[23].

Pregnancy is associated with modifications in the subgingival biofilm with an increased prevalence of *Actinomyces Actinomycetecomitans* during pregnancy to 16.4% - 20.5% as opposed to 6.3% in gingivitis [24].

1.4 Pregnancy outcomes: Preterm birth, Low birth weight, premature preterm rupture of membranes and risk factors

Approximately 12% of pregnancies result in preterm birth[1]. Prematurity contributes to 85% of neonatal morbidity and mortality[25] and prematurity and low birth weight contributes to 30% of neonatal morbidity and mortality [26]. Preterm birth and low birth weight are leading perinatal problems worldwide and they account for an important percentage of perinatal morbidity and mortality[10, 25]. They have evident public health implications and their incidence has not decreased despite of the many attempts at their prevention. While there is a reduction in under-5 mortality, neonatal mortality has shown less progress and accounts for 42% of under-5 deaths[17]. The exact mechanism(s) of preterm labour is largely unknown but some causes include decidual hemorrhage, cervical incompetence, uterine distortion e.g. fibroids, cervical inflammation (e.g. resulting from bacterial vaginosis [BV], trichomonas), maternal inflammation/fever (e.g. urinary tract infection), hormonal changes (e.g. mediated by maternal or fetal stress), and uteroplacental insufficiency (e.g. in hypertension, insulin-dependent diabetes, drug abuse, smoking, alcohol consumption) [1].

Several risk factors for preterm birth have been reported which include demographic characteristics, behavioral factors like alcohol, smoking or drug use during pregnancy, and aspects of obstetric history such as previous preterm birth, race, extremes of maternal age (< 17 years or >35 years), low socioeconomic status, and low pre pregnancy weight, African-American ancestry, inadequate prenatal care, low maternal body mass index (BMI), chronic diseases like hypertension, generalized infections, genitourinary tract infections, cervical incompetence, diabetes, nutritional status, stress and multiple pregnancies[13.]

Nutritional status during pregnancy can be described by indicators of body size such as body-mass index (BMI), nutritional intake, and serum assessments for various analytes. A low pre pregnancy BMI is associated with a high risk of spontaneous preterm birth whereas obesity can be protective. Women with low serum concentrations of iron, folate, or zinc have more preterm births than those with measurements within the normal range. There is a raised risk of preterm birth in pregnancies arising within close temporal proximity to a previous delivery. An inter pregnancy interval of less than 6 months confers a greater than two-fold increased risk of preterm birth after adjustment for confounding variables. Maternal depletion might be another cause because pregnancy consumes maternal stores of essential vitamins, minerals, and amino acids. A short interval between two pregnancies decreases the opportunity to replenish these nutrients[15]. There are many potential mechanisms by which maternal nutritional status might affect preterm birth e.g. spontaneous preterm birth can be caused by maternal thinness associated with decreased blood volume and reduced uterine blood flow. Thin women might also consume fewer vitamins and

minerals, low concentrations of which are associated with decreased blood flow and increased maternal infections. Obese women are more likely to have infants with congenital anomalies, such as neural-tube defects, and these infants are more likely to be delivered preterm. Obese women are also more likely to develop pre-eclampsia and diabetes which has been associated with preterm births.

Tobacco use increases the risk of preterm birth (<2–fold) after adjustment for other factors. The mechanism(s) by which smoking is related to preterm birth is unclear. However, nicotine and carbon monoxide are powerful vasoconstrictors, and are associated with placental damage and decreased uteroplacental blood flow. Both pathways lead to fetal growth restriction and indicated preterm births. Smoking is also associated with a systemic inflammatory response and can increase spontaneous preterm birth. Although heavy alcohol consumption has been associated with preterm birth, neither mild nor moderate alcohol use is generally regarded as a risk factor for preterm birth. Cocaine and heroin use have been associated with preterm birth in several studies[52].

Vaginal bleeding caused by placental abruption or placenta previa is associated with a very high risk of preterm delivery. Extremes in the volume of amniotic fluid—polyhydramnios or oligohydramnios—are associated with preterm labour and PPROM. Maternal medical disorders, such as thyroid disease, asthma, diabetes, and hypertension, are associated with increased rates of preterm delivery, many of which are indicated because of maternal complications. History of cervical surgical procedures have also been associated with an increase in spontaneous preterm

delivery, as have various anomalies of the uterus itself—such as the presence of a septum[1].

The perinatal complications associated with low birth weight are most often attributable to fetal prematurity, but may sometimes also arise as the result of intrauterine growth restriction[1].

Rupture of membranes can occur prematurely or at term. Preterm premature rupture of membranes (PPROM) is ROM prior to 37 weeks' gestation. PPROM is associated with 30-40% of preterm deliveries and is the leading identifiable cause of preterm delivery. PPROM complicates 3% of all pregnancies and poses significant risks of morbidity and mortality for the fetus and the mother[25, 20]. At term, programmed cell death and activation of catabolic enzymes such as collagenases and mechanical forces results in ruptured membranes. PPROM occurs probably due to the same mechanisms and premature activation of these pathways.

However, PPROM also appears to be linked to underlying pathologic processes, most likely due to inflammation and/or infection of the membranes. Various risk factors are associated with PPROM which include low socioeconomic status, low body mass index, tobacco use, preterm labor history, urinary tract infection, vaginal bleeding at any time in pregnancy and amniocentesis[25].

1.5 Preterm birth, low birth weight and Periodontitis

Maternal infections have been reported as the most important risk factors for PLBWS. Intrauterine infection is a frequent and important mechanism leading to preterm birth. The mechanisms by which infections lead to preterm labour are related to activation of the innate immune system. Microorganisms are recognised by pattern-recognition receptors—e.g., toll-like receptors, which in turn elicit the release of inflammatory chemokines and cytokines—such as interleukin-8, interleukin 1-β, and tumour necrosis factor (TNF)-α. Microbial endotoxins and pro-inflammatory cytokines stimulate the production of prostaglandins, other inflammatory mediators, and matrix-degrading enzymes.

Prostaglandins stimulate uterine contractility, whereas degradation of extracellular matrix in the fetal membranes leads to PPROM. Microbiological studies suggest that intrauterine infection might account for 25–40% of preterm births; however, 25–40% might be a minimum estimate because intrauterine infection is difficult to detect with conventional culture techniques.

Periodontal infections have been postulated to lead to PLBWS in the same mechanism as maternal infections by production of inflammatory mediators like prostaglandins, interleukins, interferons and lipopolysaccharide (LPS). The prevalence of PLBWS in mothers with periodontal disease has been reported at 12.2% and PPROM at 24.4% by a Brazilian study[27]. The prevalence of mild periodontitis in women with delivery of a PLBW has been reported at 42.7% as opposed to 30% prevalence reported in mothers with normal weight infants. This association has been reported to be stronger among mothers with low education levels[9].

Several theories exist to explain the association of periodontitis with PLBW. One proposed mechanism suggests that intra-amniotic levels of these mediators rise steadily throughout pregnancy until a threshold is reached at which labour is induced. It is therefore postulated that the presence of infection may result in abnormally elevated production of these otherwise normal physiological mediators of parturition and may trigger preterm birth resulting in low birth weight[9].

More recently, it has been suggested that subclinical maternal infections such as periodontal disease contribute to premature birth and low birth weight as a result of pathogenic micro-organisms, or indeed the microbial products, such as lipopolysaccharide (LPS). These reach the uterus via the bloodstream and induce cytokine release in the decidua or the membranes resulting in increased prostaglandin production and uterine muscle contractions[36]. Similarly, some studies have reported an increased incidence of histological chorioamnionitis in preterm delivery. However, given that it may not be associated with symptomatic infections of the genitourinary tract and that culture may produce a negative result, it is proposed that infection remote from the feto-placental complex and genitourinary tract may play a role. Therefore, the gram-negative bacteria associated with progressive disease and subclinical infection produce a variety of bioactive molecules that may directly affect the host [19].

In chronic periodontitis, bacteria and/or their components disseminate from the inflamed areas into the circulation and challenge the immune system[20]. The peripheral blood monocytes challenged by bacterial lipopolysaccharide secrete a wide spectrum of molecules, including cytokines IL-1ß, TNF-α, IL-6 and PGE_2 and matrix metalloproteinases[20]. These trigger elevation of serum TNF-α and decrease in serum

IL-10 in distant organs like the liver, uterus and the increase in the physiological levels of PGE_2 and TNF-α in the amniotic fluid leading to preterm labour[28]. Similarly *Fusobacterium nucleatum* and other subspecies coming from the oral flora have been found in the amniotic fluid of women with preterm births[19]. PGE_2, TNF-α, IL-1β are normally involved in the onset of labour at specific critical concentrations thus can trigger premature labour when abnormally elevated. In addition, inflammatory mediators may leak from inflamed periodontal pocket areas into the circulation[29].

Offenbacher et al. concluded that mothers with periodontal disease had more than seven times the risk of delivering a PLBW infant. The levels of *P. gingivalis*, *A. Actinomycetecomitans*, *Treponema denticola*, and *Tanerrela forsythensis* were significantly higher in the mothers with PLBW[9]. Studies have also reported that the risk for prematurity increases as the levels of immunoglobulin M (IgM) in the umbilical blood increase[32].

On the other hand, some studies have shown no differences in clinical periodontal status between PLBW and term normal birth weight pregnancy outcome. Similarly various studies have reported no significant differences in the amount and occurrence rates of individual periodontal pathogens in the subgingival plaque between women with PLBWS and those without PLBWS exist[30, 31, 32]. Variability among studies in definitions of periodontal disease and adverse pregnancy outcomes as well as widespread inadequate control for confounding factors and possible effect modification make it difficult to base meaningful conclusions on published data. However, while there are indications of an association between periodontal disease and increased risk of adverse pregnancy outcome in some populations, there is no conclusive evidence that treating periodontal disease improves birth outcome[51].

CHAPTER TWO

STATEMENT OF THE RESEARCH PROBLEM AND JUSTIFICATION

2.1 PROBLEM STATEMENT

Periodontal diseases are highly prevalent in Kenya. Prevalence of gingivitis among adult population in Kenya is reported at 90% while 1-10% suffers from chronic periodontitis[4]. Neonatal morbidity continues to be recognized internationally as a public health priority. In Kenya, neonatal mortality is estimated at 31 deaths per 100 live births[18]. Prematurity and low birth weight combined contributes to 30% of neonatal morbidity and mortality[26]. Approximately 70% of causes of PLBWS are not known thus there is need to identify and investigate all possible causative factors[15]. Periodontitis has been linked with a number of systemic illnesses including preterm labour and low birth weight[9].

2.2 JUSTIFICATION

Premature and low birth weight infants are at an increased risk of mortality and morbidity[13]. Despite varying efforts, the prevalence of infant mortality and morbidity, preterm birth and low birth weight in Kenya and worldwide are still high and still increasing. Studies done in the region and globally have found highly conflicting data on the association between periodontal diseases and PLBWS[1]. In Kenya, there is no available data on this topic. This study therefore aims to determine the association between the periodontal status of post partum women and preterm labour and low birth weight pregnancy outcomes. Results obtained from this study could help shape

policies on the management of periodontal diseases for pregnant women and identification of risk factors for PLBW with the aim of reducing incidence.

2.3 OBJECTIVES

2.3.1 Main objectives

- To investigate the periodontal status of post partum women in relation to preterm birth and low birth weight babies.

2.3.2 Specific objectives

1. To determine the prevalence and severity of gingivitis in post partum women within 24 hours of delivery
2. To determine the prevalence and severity of periodontitis in post partum women within 24 hours of delivery
3. To establish the birth weight of the infant at birth
4. To establish the gestational age at birth
5. To determine the relationship between periodontal status and preterm birth
6. To determine the relationship between periodontal status and low birth weight

2.4 NULL HYPOTHESIS

There is no association between periodontal status of post partum women and preterm birth and low birth weight.

2.5 VARIABLES

2.5.1 Socio demographic

Age – number of years

Education level- highest education level

Marital status- married or not married

2.5.2 Confounding factors:

Smoking habit

Alcohol intake

Weight of the mother

Height of the mother

Socioeconomic status

Maternal infections

2.5.3 Independent (exposure) variables:

Gingivitis- gingival index

Periodontitis- Probing depth, Clinical attachment loss

2.5.4 Dependent (outcome) variables:

Infants born less than 37wks old

Infant weighing less than 2500g

CHAPTER THREE

MATERIALS AND METHODS

3.1 STUDY DESIGN

This was a cross sectional descriptive hospital based study.

3.2 STUDY AREA

The study was carried out at the labour ward of Kenyatta National Hospital. Kenyatta National Hospital is a National referral hospital located in Nairobi County and handles referral patients from other hospitals in the Nairobi region, nearby regional hospitals and other referral hospitals in the country.

3.3 STUDY POPULATION

This constituted women aged 18- 35 years who delivered at the labour ward. They were examined within 24 hours of delivery by the principal investigator and the weight of the infant and gestation age at birth was recorded.

3.3.1 Case definition

The definition used in this study was drawn from the World Health Organization on Preterm birth and Low birth weight and the American Academy of Periodontology (CDC/AAP) for periodontitis (Table 1)[47]. However, since the CDC/AAP case definition (2007)8 classify patients with mild periodontitis and no periodontitis together a definition for mild and no diseases was set (see table 1). A preterm birth was defined as the delivery of an infant after 20 weeks gestation but less than 37 weeks. Low birth weight was defined as infant weighing less than 2500g at birth.

Table 1: Definitions of Periodontal Disease- adapted from CDC/AAP 2007

	CAL	PD
Severe periodontitis	>2 interproximal sites with CAL >6mm(not on same tooth) and	>1 interproximal site with PD >5mm
Moderate	>2interproximal sites with CAL >4mm(not on same tooth)	>2 interproximal sites with PD>5MM (not on same tooth)
Mild	Neither moderate or severe periodontitis	

**Third molars excluded*. *PPD* periodontal probing depth, *CAL*clinical attachment loss*

3.3.2 Inclusion criteria

- All post partum women who consented to the study
- Women who underwent normal delivery
- Women who delivered a single baby
- Women above 18 years of age and less than 35 years

3.3.3 Exclusion criteria

- Active infections or conditions other than periodontal diseases such as genitourinary tract infection, malaria , pyelonephritis, influenza
- Antibiotic therapy in the last 2 weeks

- Chronic illnesses- diabetes mellitus, heart disease, renal diseases, hyperthyroidism, HIV/AIDS, anemia, diabetes, cardiovascular disorders, hepatic deficiency.
- Multiple pregnancies (twins, etc.)
- Cervical incompetence
- Maternal trauma, burns
- Uterine factors- müllerian duct abnormalities, fibroid uterus
- Any person less than 18years of age and above 35 years
- All persons who did not consent to the study
- All persons who required emergency treatment

3.4 SAMPLE SIZE DETERMINATION

The prevalence of periodontitis among adult Kenyans has been reported at 80% (Baelum, Manji F 1988). Using Fishers formula for prevalence in cross sectional studies when the study population is 10000 or above, the desired sample size was thus determined as follows:

$$n = \frac{z^2 \times p\,(1-p)}{c^2}$$

n = desired sample size

z = confidence level at 95% (standard value of 1.96)

p = estimated prevalence of periodontitis among Kenyans (80%)

c = margin of error at 5% (standard value of 0.05)

$$n = \frac{1.96^2\,(0.8)\,(0.2)}{0.05^2}$$

= 245.86 = **<u>246</u>**

However, since the total numbers of mothers who delivered within the 3 months of data collection were 4500, the sample size used for this study was calculated with the formula used when total study population is less than 10000:

$$nf = \frac{n}{(1+\frac{n}{N})}$$

Where:

nf = the desired sample size when population is less than 10,000

n = the desired sample size when population is more than 10000.

N = the estimate of population size.

$$nf = \frac{246}{(1+\frac{246}{4500})}$$

=234

3.5 SAMPLE DESIGN AND PROCEDURE

Convenient sampling was done. All patients who delivered at the labour ward of Kenyatta National Hospital, who fit the inclusion criteria and consented to the study, were recruited. The patient details were filled in the screening form **(Appendix II).**

3.6 DATA COLLECTION INSTRUMENTS AND TECHNIQUE

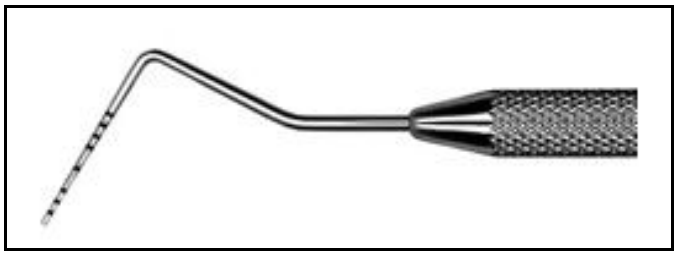
3.6.1 Data collection tools

A screening form was used to select the 246 participants after screening of 350 mothers who delivered. **(Appendix II)**. All who suffered from any of the conditions

in the exclusion criteria were excluded. The participants who fulfilled the inclusion criteria received a thorough explanation of the purpose of the study and those who consented and assented signed an informed consent form. **(Appendix V)**. A semi structured questionnaire **(Appendix III)** was administered to the patient to collect sociodemographic variables.

A clinical examination form (**Appendix IV)** was used to record data on various oral hygiene and periodontal status of the participants. Data was collected from periodontal examination of the post partum women and from medical records for the infants. Periodontal Clinical examination of all teeth except the 3rd molars at six points (mesiobuccal, buccal, distobuccal, lingual, mesiolingual, distolingual) was done under illumination from a head torch using disposable gloves, masks, gauze, a sterile University of Michigan –O- probe with Williams markings periodontal probe and oral dental mirrors. **(Appendix IV)**. The presence or absence of gingival overgrowth was noted.

Fig 1: University of Michigan O probe with Williams markings



The following indices were used to assess the oral hygiene and periodontal status;

Table 2: Indices used for Various Clinical Parameters during Data Collection

Index	Variables
Silness and Loe 1964	Oral hygiene- plaque score.
Loe and Silness gingival index- 1963	Gingival inflammation
CDC and AAP definition (2007)	Clinical attachment loss
CDC and AAP definition (2007)	Periodontal pockets

3.6.2 Preliminary Phase

The preliminary phase of the study was carried out in the study area in order to work out logistics.

3.6.3 Data Collection Phase

Data collection was carried out from November 2011 to January 2012. Sociodemographic variables were collected via interview by the principal investigator and recorded. A trained assistant recorded the details in the forms.

Clinical examination

Examination of the recruited participants was carried out at the post labour wards GFA, GFB,1A with most of the participants seated upright on the hospital bed while the ones who couldn't sit up because of caesarean section were examined while lying supine on the hospital bed. The examination was done under illuminated light from a head torch. Sterile University of Michigan O probe with William's markings periodontal probes and mouth mirrors were used in the examination. Clinical findings

including plaque, gingivitis, periodontal probing depth and recession were obtained and recorded in the clinical examination form.

Measurement of Gingivitis

Gingival inflammation was assessed using the gingival index by Loe and Silness (1963) **(Appendix Ia)**. This assessment was done by sweeping the periodontal probe under light finger pressure at the buccal and lingual gingival sulcus of 'Ramfjord' index teeth i.e. 16, 21, 24, 36, 41 and 44 (FDI nomenclature) and the observations recorded after 15 seconds in the clinical examination form.

Measurement of plaque levels

The plaque levels on the buccal and lingual surfaces of 'Ramfjord' index teeth was assessed using the Silness and Loe Gingival index 1964 **(Appendix Ib)** and recorded in the clinical examination form.

Gingival overgrowth

The gingiva on all the teeth was evaluated for any enlargement and coded as present or absent.

Periodontal Disease Measurements

Periodontal Probing Depth (PPD) - the height of the free gingival margin to the most apical location of the periodontal pocket was determined at six sites per tooth (mesio-facial, mid-facial, disto-facial, disto-lingual/palatal, mid lingual/palatal, and mesio-lingual/palatal) using a University of Michigan -O- probe with Williams markings. The measurements obtained were recorded on the clinical examination form by a trained assistant.

Recession- The distance between the heights of the free gingival margin to the CEJ was also measured at six sites per tooth (mesio-facial, mid-facial, disto-facial, disto-

lingual/palatal, mid lingual/palatal, and mesio-lingual/palatal). Positive values were given when the gingival margin was located apical to CEJ.

Clinical Attachment Loss (CAL) - PPD plus recession yielded the clinical attachment loss (CAL). PPD and REC values were rounded up to the next whole millimetre value. CAL in this study was only determined for teeth with recession.

These periodontal parameters were only measured for teeth with at least one-half of a remaining clinical crown (i.e. at least three contiguous sites in which PPD and REC were measurable).

Pregnancy outcomes

The weight at birth, gestation age at birth and the patient's weight and height was retrieved from the patient's medical records and recorded in the clinical examination forms.

Infection control

Infection control was achieved by the use of disposable face masks, examination gloves and autoclaved periodontal probes. The investigator washed their hands with antiseptic solution after every examination. Oral health instructions and health education was given verbally with the aid of a model and toothbrush to all the study participants.

3.7 DATA QUALITY AND CONTROL

The quality of data was achieved at the data collection point by ensuring completeness of questionnaires, legibility of records and validity of responses. Data entry was carried out twice by the principal investigator in Ms- Excel ®, Ms-Access®, SPSS (Statistical Package for Social Sciences computer package) SPSS

version 20.0 (SPSS Inc, Chicago, Illinois, USA) and R. Data cleaning, validation and correction for missing values was done. All information collected was coded and password protected whereas the questionnaires were properly kept in lockable drawers for confidentiality.

3.8 DATA ANALYSIS PLAN

Data was analysed using the same SPSS version 20.0 and Microsoft Excel. Descriptive and inferential statistics were used. Descriptive statistics were measures of central tendencies and dispersions for continuous variables (age, plaque scores, gingivitis, PPD, CAL, gestation age and birth weight. Pearson correlation test was used to determine the association between key variables. Significance levels were accepted at $\alpha=0.05$. The data was presented in the form of tables and figures.

3.9 RELIABILITY AND VALIDITY

The principal investigator carried out all the measurements on the patients to reduce inter-examiner variation. Intraexaminer variability was assessed by repeating examination of every 15th patient. Kappa values were calculated for plaque score, gingival inflammation, periodontal probing depth (PPD) and clinical attachment loss (CAL). An almost perfect agreement was obtained with Kappa scores range of between 0.8-0.93 (0.9 for plaque score, 0.93 for gingival inflammation, 0.80 for PPD and 0.82 for CAL).

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3.10 ETHICAL CONSIDERATIONS

The authority to carry out the study was sought from the Kenyatta National Hospital and University of Nairobi Ethics and Research Standards Board **(Appendix V)**. Only patients who consented to the study were included. Permission was obtained from the Director, Kenyatta National hospital to conduct the study at the labour ward. Any patient who required dental treatment was advised accordingly and referred to a dental clinic for management. Oral health education was given to all study participants. The information collected was treated with utmost confidentiality and no patient names were included in the questionnaire.

3.11 PERCEIVED BENEFITS

The patients received free dental check-ups and were informed of their dental health status and advised accordingly. Referral to Kenyatta National Hospital (KNH) dental department and the University of Nairobi (UON) School of Dental Sciences for treatment was done appropriately. Prescriptions of antibiotics, analgesics and antiseptics were given where indicated. This study has provided new information on the periodontal status of post partum women in relation to PLBWS which is currently not available in Kenya. The results of the study will be published with the aim of increasing awareness in the periodontal and gynaecological fields. The results of this study will contribute in enlightening the controversial issue of periodontal disease and its association with PLBW. Identification of the risk factors to PLBWS will contribute towards millennium development goals of reducing infant mortality by 2015.

This study is also a partial fulfilment of the Masters of Dental Surgery in Periodontology at the University of Nairobi.

Disclosure

The cost of the study was met by the principal investigator for academic purposes. Assistance was also obtained from University of Nairobi, School of Dental Sciences.

3.12 LIMITATIONS

Convenient sampling was done to select the study population. Examination was done with the patient propped up in bed and this might have been uncomfortable for the patient and making examination difficult.

Index teeth were used in calculation of gingival score and plaque score and this does not give the true picture of the condition of the periodontium.

Information on the mother had to be retrieved from the hospital maternal records and as such there were a number of missing values due to incomplete records.

CHAPTER FOUR

RESULTS

4.1 SOCIODEMOGRAPHIC CHARACTERISTICS

A total of 246 participants were recruited into the study. The average age of the mothers was 27years (SD±5) with a range of 18-35 years. The mean gestation age was 37.6 months (SD±3.8) with a range of 20-42 months. The mean weight of the mothers was 62.7kg (SD±8.9) with a range of 44-97kgs. The average birth weight of the infants was 3015.8grams SD ±627 with a range of 500grams to 4690grams while the weight of the previous child was an average of 2929grams SD± 876 and majority of the women were in their second pregnancy. Other sociodemographic characteristics are described in table 3 below.

Table 3: Sociodemographic characteristics of study participants enrolled in the study

Variables		Proportion of participants
		n (%)
Infant Gender	Male	137(55.7)
	Female	109(44.3)
Maternal age(yrs)	18-25	96(39)
	26-30	108(43)
	31-35	75(31)
Education	Primary & <	54(21.9)
	Secondary	52(48.8)
	Tertiary	72(29.3)
Marital status	Married	221(89.8)
	Single	25(10.2)

4.2 ORAL HEALTH STATUS

Participants' oral health status was assessed and data on gingival score and plaque score was collected. The mean gingival score was 1.07 (SD±0.17) and the mean plaque score was (1.45 SD±0.45). The mean gingival score and mean plaque score had a positive correlation and the relationship was statistically significant(r=0.534 p=0.0001). There was no mother who suffered from gingival overgrowth. While 173(70.3%) participants suffered from mild gingivitis, 73(29.7%) had moderate gingivitis and no respondent suffering from severe gingivitis. Thirty three (13.4%) of the participants had good oral hygiene 190(77.2) had fair oral hygiene while 23(9.3%) had poor oral hygiene. Of the participants who brushed their teeth once per day 98 (39.8%) and 43 (17.5%) had mild and moderate gingivitis respectively. Of the ones

that brushed twice a day, 72 (29.3%) and 27 (11%) had mild and moderate gingivitis respectively. The mean gingival score among mothers who had low birth weight and preterm birth was 1.32 (SD±0.47) and 1.29 (SD±0.45) respectively. There was a positive correlation between the mean gingival score and alcohol consumption and the association was statistically significant(r= 0.206, p= 0.001). The distribution of gingivitis according to the other variables is outlined in table 4.

Table 4: Distribution of gingivitis according to different study variables

Variable **(n (%)**

	Mild	Moderate	Test	df	p=0.05
Age groups (yrs)			X^2=2.83	2	0.24
18-25	64(26)	32(12.9)			
26-30	20(8.1)	67(27.2)			
31-35	42(17)	21(8.5)			
Level of education			X^2=5.7	3	0.133
Primary	31(12.4)	23(9.4)			
Secondary	89(36)	31(12.4)			
Tertiary	53(22)	19(8)			
Marital status			X^2=0.429	1	0.512
Married	154(63)	67(27)			
Single	19(7.7)	6(2)			
Toothbrushing frequency			X^2=2.9	3	0.40
Once	98(40)	44(18)			
Twice	72(29)	27(11)			
Thrice	3(1)	2(1)			
Smoking			X^2=0.229	1	0.632
Yes	4(2)	1(0)			
No	169(69)	72(29)			
Alcohol			X^2=1.374	1	0.241
Yes	6(2)	5(2)			
No	167(68)	68(28)			
Gestation age					
< 37 weeks	33(13)	16(7)	X^2=0.260	1	0.610
≥ 37weeks	140(57)	57(23)			
Birth weight					
< 2500g	21(9)	10(4)	X^2=0.113	1	0.736
≥ 2500g	152(62)	63(26)			
Average CAL			r=-0.149		0.019

See the key below;

Mild gingivitis	*Oedema present with absence of bleeding*
Moderate gingivitis	*Oedema, glazing, bleeding on probing*
Severe gingivitis	*Oedema, ulcerations with spontaneous bleeding*

There were more mothers in the younger age group of 18-25yrs that had poor oral hygiene 12(4.9%) compared to the older mothers 5(2%) in the 31-42years age group. Similarly there were less of the participants who had studied to tertiary level 2(0.8%) that had poor oral hygiene compared to the ones who had studied upto secondary level 21(8.6%). There was a statistically significant association between the mean plaque score and education level (X^2= 12.6, df=4, p= 0.049), smoking (X^2=6.009, df=2, p=0.05). There was a negative correlation between plaque score and maternal age (r=-0.192, p=0.003), average probing depth and clinical attachment loss (r=-0.134, p=0.04) There was no statistically significant relationship between the mean plaque score and low birth weight (X^2= 0.52, df= 2, p=0.746) and preterm birth (X^2= 4.07, df=2, p=0.131). The distribution of plaque score according to other variables is shown in table 5. The key for table 5 is as shown below;

Good oral hygiene	*A film of plaque on the free gingival margin and adjacent area of the tooth. The plaque maybe seen in situ or after application of disclosing tablet or probe on the tooth surface*
Fair oral hygiene	*Moderate accumulation of soft deposits within gingival pocket and/or tooth margin which can be seen with the naked eye*
Poor oral hygiene	*Abundant soft matter within the gingival pocket and/ or on the tooth and gingival margin*

Table 5: Distribution of mean plaque score among sociodemographic characteristics of the participants

Variable	Plaque score			Test	df	P
		n (%)				
	Good	**Fair**	**Poor**			
Gingivitis				X^2=22.74	2	0.0001
Mild	31(13)	134(54)	8(3)			
Moderate	2(1)	56(23)	15(6)			
Maternal age(yrs)				X^2=5.4	2	0.248
18-25	8(3.3)	76(30.9)	12(4.9)			
26-30	13(5.3)	68(27.6)	6(2.4)			
31-35	12(4.9)	46(18.7)	5(2)			
Level of Education				X^2=12.6	6	0.049
Primary &<	2(0.8)	43(17.5)	9(3.7)			
Secondary	20(8.1)	88(35.8)	12(4.9)			
Tertiary	11(4.5)	59(24)	2(0.8)			
Marital Status				X^2= 0.123	2	0.98
Married	30(12)	170(69)	21(9)			
Single	3(1)	20(8)	2(1)			
Toothbrushing Frequency						
Once	15(6)	113(46)	13(5)	X^2= 15.55	2	0.016
Twice	16(7)	75(30)	8(3)			
Thrice	2(1)	2(1)	1(0.08)			
Smoking				X^2=6.009	2	0.051
Yes	0	3(1)	2(1)			
No	33(13)	187(76)	21(9)			
Alcohol						
Yes	0	8(3)	3(1)	X^2= 5.53	2	0.06
No	33(13)	182(74)	20(8)			
Gestation Age				X^2=4.07	2	0.131
< 37 Weeks	10(4.1)	37(15)	2(0.8)			
≥ 37 Weeks	23(9.3)	153(62.2)	21(8.5)			
Birth Weight				X^2=0.529	2	0.768
< 2500grams	4(1.6)	23(9.3)	4(1.6)			
≥ 2500 Grams	29(11.8)	167(67.9)	19(7.7)			
Parity				r=0.055		0.391
Average CAL				r=-.134		0.036

4.3 SELF REPORTED GINGIVAL SYMPTOMS

Data was obtained from the questionnaire on self reported gingival symptoms. Of the patients with mild and moderate gingivitis, 41(24%) and 44 (31%) had reported gingival bleeding. One hundred and fifty two participants (62%) of the mothers with LBW infants had bleeding gums while 140(57%) of those who had PT births had bleeding gums. The rest of the reported symptoms are shown in table 6 below.

Table 6: Self reported symptoms

Variable	n (%)
Gingival swelling	14(5.7)
Mobile teeth	72(29.3)
Pain	50(20.3)
Dental visit last 1 yr	11(4.5)

4.4 ORAL HEALTH PRACTICES AND ORAL HEALTH SEEKING BEHAVIOUR

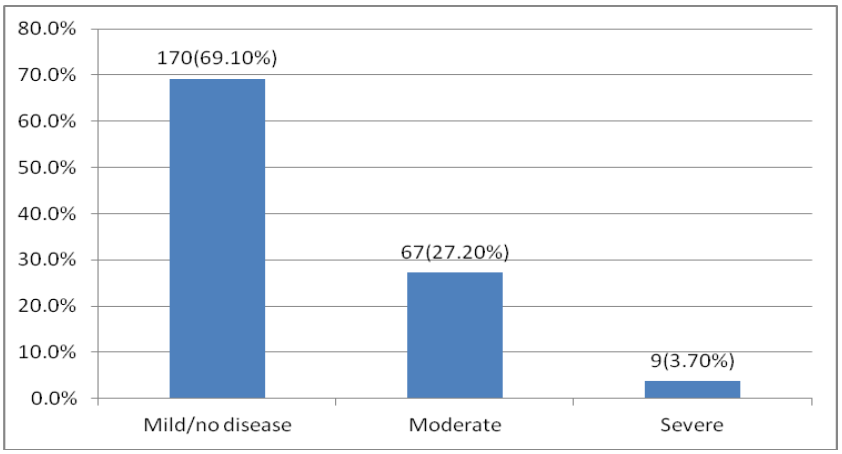
Ninety eight percent (98%) of the respondents reported that they brushed their teeth and of these, (141)57.3 % brushed their teeth once daily ,(99)40.2% brushed twice a day and (5)2% brushed three times a day while one participant reported that she did not brush her teeth at all. Interdental cleaning using toothpick, dental floss or piece of string was reported to be carried out by 14.2% of the study population. There was no association between the toothbrushing frequency and the severity of periodontitis

(X^2=3.31, df =6, p=0.768), education level(X^2= 7.2, df=6, p=0.300) and maternal age(X^2 =10.1, df= 9, p= 0.335).

4.5. PREVALENCE AND SEVERITY OF CHRONIC PERIODONTITIS

Periodontitis was described using the CDC/AAP classification of 2007[47]. The average probing depth was 2.28mm (S.D±0.37) and the average CAL was 2.34mm (S.D±0.42). The table below outlines the prevalence and severity of periodontitis as seen in this study.

Fig 2: Prevalence and severity of chronic periodontitis



Severe periodontitis was noted in 9(3.7%) of the study population. None of the mothers that had preterm birth or low birth weight infant suffered from severe periodontitis. There was a negative correlation between average CAL and maternal age and the association was statistically significant association (r= -0.192, p=0.003). There was no statistically significant association between average probing depth and CAL with education, marital status, preterm birth and low birth weight, toothbrushing frequency.

Table 7: The Distribution of Periodontitis according to Severity among the Participants (CDC/AAP Consensus Definition)

Variable	Periodontitis			Test	df	p
	n (%)					
	Severe	**Moderate**	**Mild**			
Maternal age(yrs)				X^2=3.25	4	0.531
18-25	5(2)	22(8.9)	69(28)			
26-30	3(1.2)	28(11.4)	56(22.8)			
31-35	1(0.4)	17(6.9)	45(18.3)			
Level of Education				X^2=7.18	6	0.275
Primary &<	0	16(6.5)	38(15)			
Secondary	8(3.3)	31(12.6)	81(32.9)			
Tertiary	1(0.4)	20(8.1)	51(20.7)			
Marital Status				X^2= 3.201	2	0.177
Married	9(3.7)	63(25.6)	149(60.6)			
Single	0	4(1.6)	21(8.5)			
Toothbrushing Frequency						
Once	5(2)	37(15)	100(41)	X^2= 3.315	6	0.768
Twice	4(2)	30(12)	65(26)			
Thrice	0	0	5(2)			
Smoking				X^2=0.553	2	0.758
Yes	0	2(1)	3(1)			
No	9(4)	65(26)	167(68)			
Alcohol				X^2= 0.443	2	0.801
Yes	0	3(1)	8(3)			
No	9(4)	64(26)	162(66)			
Gestation Age				X^2=2.77	2	0.252
< 37 Weeks	0	12(5)	37(15)			
≥ 37 Weeks	0	64(26)	133(54)			
Birth Weight				X^2=1.45	2	0.486
< 2500grams	0	8(3)	23(9)			
≥ 2500 Grams	0	68(28)	147(60)			

See the key below;

	CAL	*PD*
Severe periodontitis	*>2 interproximal sites with CAL >6mm(not on same tooth) and*	*>1 interproximal site with PD >5mm*
Moderate periodontitis	*>2interporximal sites with CAL >4mm(not on same tooth)*	*>2 interproximal sites with PD>5MM (not on same tooth)*
Mild periodontitis	*Neither moderate or severe periodontitis*	

4.6 PRETERM AND LOW BIRTH WEIGHT

The prevalence of low birth weight was 12.6 %(31) and the preterm birth was found in 49(19.9%) of the participants. The mothers who delivered both preterm and low birth weight infant were 16(6.5%), the ones who delivered preterm normal birth weight was 33(13.4%), and term low birth weight infant was 15 (6%). Majority of the study participants delivered a term and normal birth weight infant 133(54%), 147(60%) respectively. There was a statistically significant association between gestation age and maternal age (X^2=9.8, df=2, p=0.007) and between gestation age and marital status (X^2=7.03, df=1, p=0.008). The distribution of gestation age and birth weight according to different study variables is shown in table 8 and 9 below. There was a statistically significant relationship between weight of the baby and weight of previous baby (r= 0.171, p=0.036).

Table 8: Distribution of birth weight according to study participant's characteristics

Variable	n (%)		Test	df	P
	Low birth weight	Normal birth weight			
Maternal age(yrs)			X^2=4.04	2	0.133
18-25	17(7)	79(32)			
26-30	7(3)	80(33)			
31-35	7(3)	63(26)			
Level of education					
Primary &<	11(4)	43(17)	X^2=4.62	3	0.201
Secondary	13(5)	107(43)			
Tertiary	7(3)	65(26)			
Marital Status			X^2=1.86	1	0.172
Married	30(12)	191(78)			
Single	1(0)	24(10)			
Toothbrushing Frequency			X^2=1.38	3	0.709
Once	20(8)	1(0.08)			
Twice	11(4)	121(49)			
Thrice	0	88(36)			
Smoking					
Yes	2(1)	3(1)	X^2=3.47	1	0.062
No	29(12)	212(86)			
Alcohol			X^2=0.32	1	0.568
Yes	2(1)	9(4)			
No	29(12)	206(84)			
Parity			r=-.032		0.618
Average PD			r=0.079		0.220

See the key below;

Low birth weight	*2500grams*
Normal birth weight	*>2500grams*

Table 9: Distribution of gestation age according to study participant's characteristics

Variable	n (%)		Test	df	p
	Preterm	Term			
Maternal age(yrs)			X^2=9.8	2	0.007
18-25	28(11)	68(28)			
26-30	15(6)	72(29)			
31-35	6(2)	57(23)			
Level of Education			X^2=2.07	3	0.557
Primary &<	16(6.5)	38(15)			
Secondary	31(12)	81(32.9)			
Tertiary	20(8.1)	51(20.7)			
Marital Status			X^2=7.03	1	0.008
Married	39(16)	182(74)			
Single	10(4)	15(6)			
Toothbrushing Frequency			X^2=1.57	3	0.666
Once	27(11)	142(58)			
Twice	20(8)	99(40)			
Thrice	2(1)	5(2)			
Smoking			X^2=1.29	1	0.256
Yes	2(1)	3(1)			
No	47(19)	194(79)			
Alcohol			X^2=0.84		0.358
Yes	1(0)	10(4)			
No	48(20)	187(76)			
Parity			r=0.059		0.354
Average PD			r=0.025		0.694

See the key below;

Preterm birth	*Birth between 20weeks and before 37 weeks*
Term	*Birth at or after 37 weeks*

Table 10: Distribution of gestation age and birth weight according to severity of periodontitis

Variable	Periodontitis		Test	df	p
	n (%)				
Gestation age	**Moderate & >**	**Mild**			
Preterm birth	5(12)	37(15)	X^2=2.771	2	0.250
Term birth	64(26)	133(54)			
Birth weight					
Low birth weight	8(3)	23(9)	X^2= 1.45	2	0.483
Normal birth weight	68(28)	147(60)			

CHAPTER FIVE

DISCUSSION

Majority of the women were aged between 26-30yrs. Most of the women (98.2%) had formal education which could be explained by the fact that study was carried out in an urban area.

5.1 Oral hygiene practices

One hundred and forty one (57.3%) of the women brushed their teeth once a day in this study and this is similar to a study by Ingrida that reported that 58.8% of the women brushed their teeth once a day, while 40.2% brushed twice daily compared to 27.4%[33]. In this study, 4.5% of the women had sought dental treatment during pregnancy compared to 27.3% in a study by Dinas et al. Twenty percent of participants reported to have experienced dental pain in the course of the pregnancy which is a lower percentage compared to the study by Dinas et al. that reported an incidence of 53.4%[34]. The difference could be explained by the individuals' perceptions of pain and their ability to decipher if they have a symptom indicative of disease. Only 11(4.5%) of the study participants had visited a dentist in the last 1 year which is much lower than in the study by Dinas that reported 19.6% of the population having visited a dentist within a year. The fact that dental treatment in Greece is offered for free could explain the higher attendance as in our setup there is a cost implication.

5.2 Oral health status

The mean gingival score in this study was 1.07 SD±0.17 and the plaque score was 1.45 (SD±0.45). This was lower than a study by Ingrida[33] that reported a mean GI and PS of 1.48 and 1.51 respectively. The association between the mean gingival score and mean plaque score was statistically significant. Studies have reported that plaque is a risk factor for gingivitis[48]. This study reported bleeding gums among 57% of mothers who delivered a preterm infant and among 62% of mothers who delivered a low birth weight infant. This prevalence is much higher than was reported in a study by Agueda et al[39] that reported bleeding gums in 26% and 26.8% of mothers who delivered preterm and low birth weight infants respectively. The higher prevalence in this study could be explained by to differences in socioeconomic status and oral health seeking behaviour which is higher in developed countries compared to developing countries like Kenya. Gingivitis has been reported to increase with increasing gestation due to the increasing hormonal levels[7]. A Tanzanian study by Mumghamba et al reported gingival bleeding in 79.3% of the mothers who delivered preterm and low birth weight infants. The Tanzanian study was carried out among women from rural and urban areas and this mixture could explain the higher prevalence of bleeding gums in the Tanzanian study. The availability of dental services in rural areas is lower than in urban areas and this could impact the access to treatment[21].rural residence has also been shown to be a risk factor for developing periodontal diseases[46]. Similar to findings in this study, the relationship between the gingivitis and preterm and low birth weight was not statistically significant (p = 0.081)[35]. This study did not find an association between the mean gingival score, gestation age and birth weight. This is contrary to a study by Hu et al that reported

that gingivitis increases with increase in gestation[7]. The fact that this study was cross sectional and evaluated gingival status in post-partum women as opposed to the study by Hu et al that evaluated changes from first to third trimester could explain the differences.

In this study, fewer participants (19.3%) had poor oral hygiene compared to the study by Baelum in Kenya that reported 75-95% of the population as having poor oral hygiene[5]. The study by Baelum involved a heterogeneous population of men and women. It can be hypothesized therefore that the fact that men were included in the study by Baelum could have increased the prevalence reported as the male gender is a risk factor in plaque accumulation and periodontal disease[46]. The association between plaque score and education level, maternal age, smoking was statistically significant. This could be explained by the fact that increasing age, low education level and smoking are risk factors for periodontitis[46]. Similarly, a study by Agueda et al[39] also found a significant association between plaque score and age, education and smoking. The similarities could be explained by the similar methodology and characteristics of study participants between the studies. In this study, plaque score was significantly associated with probing depth and clinical attachment loss. Agueda et al reported similar association in the Spanish study. The fact that plaque has been reported to be a risk factor for periodontal disease could explain this relationship[46]. There was a positive correlation such that the plaque scores increased with advancing gestation. It can be hypothesized that increasing fatigue as pregnancy advances could hamper oral hygiene measures by the expectant mothers. However the plaque score reduced as birth weight increased.

5.3 Periodontal diseases

The prevalence of gingivitis in this study was reported at 100%. This tally's closely relates to findings from a study by Ng'ang'a et al that reported a prevalence of up to 90%[4]. However this figure is higher than a study by Hu et al that reported prevalence of gingivitis in pregnant women in 3rd trimester at 79.17%[7] and the differences in races and socioeconomic status could explain the lower prevalence. Gingivitis has been shown to increase in pregnant women due to the high amounts of oestrogen and progesterone[5]. While 29.7% of the women suffered from bleeding on probing in this study, a lower incidence was reported by Ingrida at 15.14% despite almost similar age group[33]. This could be explained by the differences in socioeconomic status and access to oral healthcare. However, a study by Nuamah et al reported a prevalence of gingival bleeding among pregnant women at 89% which is much higher than in this study[36]. The study by Nuamah incorporated women from rural and urban areas and this could explain the higher prevalence.

The prevalence of periodontitis was reported at 27.2% for moderate periodontitis and 3.7% for severe periodontitis in this study. This was much higher than in a study by Kaimenyi that reported a prevalence of between 1-10% among the general population[4]. The methodology Employed in diagnosis of periodontitis was different and this could explain the different results. The prevalence of severe periodontitis in this study was 3.7% which is much higher than a study by Ingrida that reported a prevalence of 0.37%[33]. The differences could be attributed to the socioeconomic status differences in the different study populations. Also, the study by Ingrida did not take into account the attachment loss and this could explain the lower prevalence reported. Low socioeconomic status ad been shown to be a risk factor for periodontitis

and this offers an explanation for the higher prevalence of periodontitis reported in our setting[46]. Almost two thirds (69.1%) of the study participants had mild or no periodontitis and this could be attributed to the younger age groups of the study participants as severity of disease tends to increase with age[5]. Similar to a study by Mumghamba in Tanzania, this study reported a statistically significant association between clinical attachment loss and age and this is understandable as the incidence of periodontitis increased with advancing age[46]. Compared to the study by Mumghamba et al on prevalence of periodontitis among 18-35yr olds, the prevalence of moderate periodontitis in this study was higher at 27.2% and 3.7% for severe periodontitis. In his study no person suffered from severe periodontitis. However, the component of CAL was not included and thus reducing the overall presence of periodontitis. Thus, these differences in methodology could explain this difference[49.]

There was no association between education level, smoking habit, alcohol intake and periodontitis. This is contrary to studies that have reported low education level, smoking and alcohol intake as a risk factor for periodontitis[46]. The plausible explanation could be due to the low sample of members who reported smoking or alcohol habits. Similarly, this study was carried out in an urban setting and most mothers had formal education upto secondary and tertiary levels.

5.4 Preterm birth and low birth weight

The prevalence in this study for preterm birth was 19.9% and 12.6% for low birth weight. This was much higher than was reported by UNICEF 2005-2009 survey where the prevalence of LBW was 8%[17]. The higher prevalence in this study could be due to the fact that this study was carried out on pregnant women only as compared to the survey that was carried out in the general population thus varying the study

methodology[17]. Similarly, it has been reported that populations with low socioeconomic and health care levels have prevalence of preterm birth and/or low birth weight which is higher than 10% and this could explain the differences between these two studies[1].

A study by Agueda et al found a prevalence of preterm birth at 6.6%, low birth weight 6.0%, preterm low birth weight at 3.3%[39]. The prevalence reported by Agueda et al is lower than the current study and could be explained by differences in socioeconomic status and antenatal care between developed and developing countries. Also the mothers in the Agueda et al study had access to free antenatal care which could have increased its uptake. Cruz et al reported a prevalence of preterm birth of 2.94% and low birth weight of 3.53% which is much lower than in this study[9]. The differences in race of the participants could explain the differences in the prevalence as studies have shown that PLBW is more prevalent among the black race[15].This study was carried on in a developed country where the prevalence of PLBW is expected to be lower A study by Watson-Jones et al in 2007 reported prevalence of 8% for LBW and 12% for preterm birth which is comparable to this study[37] and these findings can be explained by the similarities in race, socioeconomic status among populations in Kenya and Tanzania. American College of Obstetricians and Gynaecologists (ACOG) reported a prevalence of preterm birth at 12% in 2001[1] among the American population. However the prevalence in this study was 19.9% and this higher prevalence could attribute to the differences in socioeconomic levels, availability of antenatal care. It has also been reported that the prevalence of preterm birth and low birth weight is higher in developing countries compared to developed countries[15, 31].

There was a statistically significant association between current infant birth weight and birth weight of the previous child. Goldenberg et al and Agueda et al have found strong association between birth weight and weight of the previous child. The weight of the previous child has been reported as a risk factor for low birth weight[20, 39]. There was a statistically significant association between gestation age and maternal age with gestation age increasing as age of the mother increased. There was a statistically significant association between gestation age and marital status in this study. These findings are similar to a study by Agueda et al. The similarities in the methodology of the two studies could be a plausible explanation. Single status and low and high maternal age have been shown to be risk factors for preterm birth[50].There was no statistically significant association between birth weight and mothers age and parity. There was a statistically significant association between weight of the previous baby and weight of current baby in this study which is similar to a study by Ismail et al[45].

5.5 Association of periodontitis and preterm birth and low birth weight

The prevalence of mild periodontitis in women with delivery of a LBW and preterm infant in this study was 9% and 15% respectively. There was no association between periodontitis and low birth weight and preterm birth in this study.

A comparable studies done by Bassani et al and Vettore et al used a definition similar to the one used in this study and found no association between periodontitis and preterm birth and low birth weight[40,41]. The study by Vettore et al is comparable to this study since there was exclusion of all risk factors and confounders. However it included women above 30 years of age which could have raised disease severity while

in this study majority of the participants were below 30 years of age. This agrees with the fact that in this study age was not associated with low birth weight.

Similarly, Vergnes and Sixou in their meta-analysis reported a likely association of periodontitis and adverse pregnancy outcomes[43]. They reported that the heterogeneity of various studies in this area makes it difficult to compare two studies precisely. They concluded that there was no evidence to support the notion that maternal periodontal disease is a significant risk factor for PLBW. Moreu et al[44] in a longitudinal study reported that periodontitis is a risk factor for low birth weight but not for preterm birth which is partly contrary to this study as no association was found between periodontitis and PLBW. This could be explained by the different study methodology. Also different populations may be subject to different risk factors for the development of a specific pathology and this could explain the varying results among studies. Also, Michalowicz et al showed that treatment of mothers with periodontitis did not alter the preterm and low birth weight birth outcomes despite reduction in sites with bleeding on probing[31].

On the contrary, other studies have shown a positive association between periodontitis and PLBW. Radnai et al found a positive association between periodontitis and preterm birth and low birth weight. Similar to this study, any mothers with any of known confounding factors were eliminated from the study. However, the study was carried out among Caucasians and the definition used for periodontitis could have exaggerated the incidence of periodontitis. This could explain the differences in the study outcomes[38]. Studies by Agueda et al in Spain defined periodontitis as PPD >4mm and found a positive association[39]. Despite shared sociodemographic

characteristics of participants with this study, Agueda et al in a prospective cohort study reported a significant association between preterm birth and low birth weight with age, plaque score and periodontitis. The differences in methodology could explain the different results as different methodologies can affect the diagnosis of periodontitis[49]. However, the different case definitions of periodontitis as well as varying definitions of adverse pregnancy outcomes that have been used in this area makes it difficult to compare results from studies[51].

CONCLUSIONS

Within the limitations of this study it can be concluded that;

- There was no association between periodontal disease and preterm birth and low birth weight.

RECOMMENDATIONS

- Large prospective cohort studies are required in this area to investigate any associations between periodontitis and preterm and low birth weight and identify other risk factors.
- A greater emphasis on the need for dental education targeting mothers who deliver in Kenyatta National hospital is necessary to increase awareness.

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APPENDICES

APPENDIX I: INDICES

Plaque index Ia: Silness and Loe plaque index 1964

The Plaque Index System

Scores	Criteria
0	No plaque
1	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen in situ only after application of disclosing solution or by using the probe on the tooth surface.
2	Moderate accumulation of soft deposit s within the gingival pocket, or the tooth and gingival margin which can be seen with the naked eye.
3	Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

Appendix Ib:Gingival index II: Loe and Silness 1963

0- normal, absence of oedema and no bleeding on probing

1- Oedema present with absence of bleeding

2- Oedema, glazing and bleeding on probing

3- Oedema, ulcerations with spontaneous bleeding

APPENDIX II: SCREENING FORM

Date: _____Serial No: _____ _____

Age (yrs): _____ File No: _____

	Yes	No
Infections		
Diabetes		
Hypertension		
Hepatitis		
Antibiotics in the last 2 weeks		
Tooth cleaning in the last 1 year		
Caesarian section before		
History of delivery of twins		
weight of your previous child		
Number of pregnancy		
Cervical surgery		

APPENDIX III: QUESTIONNAIRE

TITLE: PERIODONTAL STATUS OF POSTPARTUM WOMEN IN RELATION TO PRETERM BIRTH AND LOW BIRTH WEIGHT AT KENYATTA NATIONAL HOSPITAL

Date: _____Serial No: _____

Age (yrs): _____ File No: _____

Gestation age: _____Level of Education: _____

Marital status: Single ☐ Married ☐

Breadwinners income per month	Kshs.1000-5000	
	Kshs.5000-10000	
	Kshs.10000-15000	
	Kshs.15000-20000	
	> Kshs.20000	

1. Do you brush your teeth? Yes ☐ No ☐
2. How frequently do you brush your teeth?

   Once a day ☐

   Twice a day ☐

   Three times a day ☐

   Other (specify) ☐
3. What do you use to brush your teeth?

   Commercial toothbrush ☐

   Chewing stick ☐ Fingers ☐ Other (specify) ☐
4. Do you practice interdental cleaning? Yes ☐ No ☐

5. If yes, what do you use?

Toothpick Daily ☐ Weekly ☐ Monthly ☐

Dental floss Daily ☐ Weekly ☐ Monthly ☐

String

Other (specify) ☐

6. Do your gums bleed when you are brushing? Yes ☐ No ☐

7. Have had any gingival growth during the pregnancy? Yes ☐ No ☐

8. Have you experienced mobile teeth? Yes ☐ No ☐

9. Have you suffered from pain in your teeth during this pregnancy?

Yes ☐ No ☐

10. If yes, what treatment was done?

Took painkillers ☐

Removal of the tooth ☐

Tooth cleaning ☐

11. Do you practice any of the following habits?

Smoking ☐

Alcohol ☐

Miraa chewing ☐

12. Have you visited a dentist in the last 1year? Yes ☐ No ☐

13. What treatment was done?

Check up ☐ Cleaning ☐

Surgery ☐ Filling ☐

Removal of a tooth ☐

Prescribed medications (which one)…… .....

APPENDIX IV: CLINICAL EXAMINATION FORMS

Mothers weight (kgs):_____Mother's height: _____

Sex of child: _____ Mother's age (yrs):_____

Weight of the baby at birth: _____

GINGIVAL SCORE: Löe-Silness Index - 1963

Tooth	16		11		24		36		31		44	
Surface	F	P	F	P	F	P	F	L	F	L	F	L
Score												

PLAQUE SCORE: Silness-Löe Index - 1964

Tooth	16		11		24		36		31		44	
Surface	F	P	F	P	F	P	F	L	F	L	F	L
Score												

Periodontal probing depth

Maxillary arch

Tooth	17			16			15			14			13			12			11			21			22			23			24			25			26			27		
Palatal																																									
Recession																																									
CAL																																									
Facial																																									
Recession																																									
CAL																																									
Mobility																																									

Periodontal probing depth

Mandibular Arch

Tooth	47			46			45			44			43			42			41			31			32			33			34			35			36			37		
Lingual																																									
Recession																																									
CAL																																									
Facial																																									
Recession																																									
CAL																																									
Mobility																																									

APPENDIX V: CONSENT FORM

This is to certify that I, _____ hereby agree to participate in this educational and research study on "Periodontal status of postpartum women in relation to preterm birth and low birth weight babies at Kenyatta National Hospital". This will be carried out by Dr. Wangari Veronica Wanjiru, a postgraduate student pursuing a Master's degree in Periodontology at the University of Nairobi, School of Dental Sciences. O. Box 15- 00202 Nairobi. The consent to carry out this study has been given by the University of Nairobi and Kenyatta National Hospital Ethics board. I understand that this study will involve a mouth examination using a dental mirror and periodontal probe where all the teeth will be examined for plaque, gum bleeding, pocket depth and mobility. I understand that no dental treatment will be rendered during this appointment rather my current oral health status will be evaluated and I will be referred for treatment if need be.

Perceived benefits

I understand this will benefit me personally as I will be informed of any abnormal findings in my mouth so that I may voluntarily seek treatment. I understand the results obtained from this study will provide baseline information for the development of a protocol to identify and reduce dental disease and the risk factors in pregnancy.

Risks

There are no anticipated risks for participating in the study. I understand that I will be given a free dental check up and advice on oral hygiene measures and referred if further treatment is required. However, there is a chance of slight discomfort in my gums and bleeding during the examination.

Costs and payments:

I understand that this study is strictly voluntary and no monetary compensation will be given.

Confidentiality:

I understand that all personal information learned about me in this research will be kept strictly confidential

Withdrawal privilege:

I understand that I may refuse to participate or withdraw from the study at any time without penalty or prejudice. If I do this, I will continue to receive health care at KNH as I would normally receive.

Voluntary consent:

I certify that I have read all of this consent form or it has been read to me and that I understand it. Any questions pertaining to the research have been answered to my satisfaction. My signature below means I freely agree to participate in this study.

Signature of participant ……..Date:.....

Investigators statement:

I certify that I have explained to the above individual the nature and purpose of this study, potential benefits and possible risks associated with participation in this study. I have answered any questions that have been raised. I have explained the above to the participant on the date on this consent from.

Investigator……Date:.....

MAELEZO YA KUTAFUTA IDHINI KUTOKA KWA KINA MAMA WALIOJIFUNGUA WANAOSHIRIKI KATIKA UTAFITI

Kiini cha Utafiti

Hii ni kuonyesha ya kwamba mimi_____nimepatiana ruhusa ya kushiriki katika utafiti unaochunguza "HALI YA UFIZI YA AKINA MAMA WALIOJIFUNGUA NA UHUSIANO WAKE NA UZANI WA WATOTO NA UMRI UJAUZITO WAKATI WA KUZALIWA KATIKA HOSPITALI KUU YA KENYATTA, NAIROBI KENYA". Huu utafiti unafanywa na daktari Wangari Veronica Wanjiru ,mwanafunzi katika chuo kiuu cha Nairobi, Sanduku la Posta 15-00202 KNH. Naelewa kwamba huu utafiti utahusika na kuangalia hali ya ufizi ya meno yangu na hakuna matibabu yeyote ambayo itatekelezwa. Kama kuna kasoro nitapewa mawaidha kuhusu jinsi ya kupata matibabu kwingine. Utafiti huu umeidhinishwa na kamati ya Chuo Kikuu cha Nairobi na kamati ya kitafiti inayo simamia sayansi inayohusu tiba ya magonjwa ya Hospitali Kuu ya Kenyatta.

Manufaa na madhara ya Utafiti

Nitajulishwa matokeo ya utafiti baada ya kuangaliwa na nitapewa mawaidha yanayohitajika . Pia nikiwa na mahitaji ya dharura ya kimatibabu nitatumwa kwa mtaalamu katika hospitali kuu. Matokeo ya utafiti huu yatawaisidia wanasayansi wa hapa nchini na wa kimataifa kugundua na kupunguza mambo yanayosababisha watoto kuzaliwa kabla ya muda wakiwa na uzito wa chini.

Nimeelewa kwamba hakuna gharama yeyote kwa kushiriki katika utafiti huu.

Hifadhi ya Nakala ya Habari Utakayotoa

Habari zote zitakazokusanywa kutoka kwako zitahifadhiwa kwa siri na kutumiwa tu katika utafiti huu. Majina yangu hayataandikwa mahali popote wakati wowote.

Nakala zote za habari kukuhusu zitafungiwa katika makabati maalum wakati wote wa utafiti huu. Habari hizi zitawekwa kwenye komputa na mchunguzi peke yake ndiye atakayetumia kitambulisho cha siri ili kufikia habari hizi.

Tunasistiza usiri huu katika kusimamia habari tutakazopewa ili kuzuia kujulikana kwa watakaoshirki katika utafiti huu. Hakuna majina yatakayotumika katika vikao vya sayansi kwa umma na ripoti zitakazochapishwa katia mijarida za sayansi.

Idhini na Sahihi

Nimesoma maelezo yaliyoko hapa juu na nimekubali kwa hiari kushiriki katika utafiti huu.

…… …….

Jina la Mshiriki Sahihi ya Mshiriki na Tarehe

Mimi niliyepewa jukumu la kupeana maelezo kuhusu utafiti huu kwa mshiriki aliyetajwa hapa juu, nimepeana maelezo kamili kulingana na masomo na ujuzi wangu katika kazi hii. Kwa hivyo ninahitimu kufanya jukumu hili.

…… ……

Jina la mtafiti ya aliyetoa maelezo Sahihi ya Mtafiti na Tarehe

……..... ……

Jina la Shahidi Sahihi ya Shahidi na Tarehe

APPENDIX VI: Ethical approval