RELATIONSHIP OF PERIODONTITIS IN PREGNANCY TO PREMATURE AND LOW BIRTH WEIGHT IN A LIBYAN WOMEN SAMPLE

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بسم الله الرحمن الرحيم

DEDICATION

To **soul of;**

My mother &

To **my soul;**

My husband.

ACKNOWLEDGEMENT

First of all, I thank Allah for giving me strength and ability to complete this study, without his blessing, this thesis would not have been possible.

I would like to express my deepest appreciation to all those who have provided me the possibility to complete this thesis.

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LIST OF ABBREVIATIONS

AAP	American Academy of Periodontology.
AB	Antibiotic
ADA	American Dental Association.
AGD	Academy of General Dentistry.
AIDS	Acquired Immune Deficiency Syndrome.
ALRI	Acute Lower Respiratory Infection.
AMPs	Antimicrobial peptides.
AP-1	Activator Protein 1.
APOs	Adverse Pregnancy Outcomes.
BC	Before Christ.
BMI	Body Mass Index.
BV	Bacterial Vaginosis.
BW	Birth Weight.
CAL	Clinical Attachment Level.
CPITN	Community Periodontal Index of Treatment Needs
CRP	C-reactive Protein.
C -rectus	Campylobacter rectus.
DCs	Dendritic Cells.
DNA	Deoxyribonucleic Acid.
FTB	Full Term Birth.
g	Gram.
GCF	Gingival Crevicular Fluid.
GECs	Gingival Epithelium Cells.
GF	Gingival Fibroblast.
GI	Gingival Index.
IF N-γ	Interferon-Gamma.
IgM	Immunoglobulin M.
IL-1	Interleukin 1.
IVF	Intra vitro fertilization.
IUGR	Intrauterine Growth Restriction.

LBW	Low Birth Weight.
LPS	Lipopolysaccharides.
MAK	Mitogen-Activated Protein Kinase.
MIP	Macrophage Inflammatory Protein.
MMPs	Matrix Metalloproteinase.
MOTOR	Maternal Oral Therapy to Reduce Obstetric Risk.
NK	Natural Killer.
NFκB	Nuclear Factor Kappa-B.
OCs	Osteoclasts.
OPT	Obstetrics and Periodontal Therapy.
PAMPs	Pathogen-Associated Molecular Patterns.
PB	Premature Birth.
PD	Periodontal Disease.
PDI	Periodontal Disease Index.
PDLFs	Periodontal Ligament Fibroblasts.
Pg	Porphyromonas Gingivalis.
PGE2	Prostaglandin E2.
PI	Plaque Index.
***P*- Intermedia**	Prevotella intermedia.
PIPS	Periodontal Infection and Prematurity Study.
PLBW	Premature Low Birth Weight.
PPROM	Preterm Premature Rupture of Membranes.
PT	Preterm.
PTB	Preterm Birth.
PTL	Preterm Labour.
RNA	Ribonucleic Acid.
SDF	Stromal-Derived Factor.
SHS	Second Hand Smoke.
SPTB	Spontaneous Preterm Birth.

TGF-β	Transforming Growth Factor.
Th-1	T- helper 1.
Th-2	T-helper 2.
TIMPs	Tissue Inhibitors of Metalloproteinase.
TLRs	Toll-Like Receptors.
TNF	Tumor Necrosis Factor.
TNF- α	Tumor Necrosis Factor-Alpha.
UK	United Kingdom.
USA	United States of America.
UTI	Urinary Tract Infection.
WHO	World Health Organization.
$	Dollar.

ABSTRACT

Introduction: Many studies have been published indicating a positive or negative relationship between periodontal disease and preterm/low birth weight. The inconsistent findings across studies have given the emerging evidence suggesting that associations may be influenced by population characteristics.

Objective: To assess the relation between periodontal disease and the risk of preterm delivery/low birth weight among Libyan women in Benghazi.

Methods: A total of 300 Libyan pregnant women attended to reception of labour ward at gynaecology and obstetrics department of Al- Jomhuriya Hospital in Benghazi for delivery from May to August 2010 included in this study. After recording and excluded traditional risk factors for premature/low birth weight like; age, weight, smoking, prenatal care, medical history, gestational and obstetric history, they were examined for periodontal status using Plaque Index, Gingival Index and Periodontal Disease Index and the data obtained analysed with day of delivery and birth's weight.

Results: The periodontal disease index score showed that 47% of the sample had moderate gingivitis, 29% of the sample had mild gingivitis, 10% had severe gingivitis while 11% had mild periodontitis. According to gingival index score, moderate gingivitis had high percentage (60%) which is related to high percentage of plaque deposition (51%) in plaque index score. And PDI showed insignificant relationship with PLBW at P-value = 0.849.

Conclusion: This study suggests that there is no association between periodontitis and premature/low birth weight among Libyan ladies in Benghazi.

Recommendation: This is only a preliminary study and further research from multicentre from different Libyan cities of large scale samples are needed before this can assume to be a casual relationship among Libyan ladies.

INTRODUCTION

1-INTRODUCTION

The past 20 years have witnessed an increase in research evidence suggesting associations between periodontal disease and increased risk of systemic diseases such as cardiovascular disease, diabetes mellitus, respiratory infections and adverse pregnancy outcomes. Adverse pregnancy outcomes that have been linked to periodontal disease include preterm birth, low birth weight, miscarriage or early pregnancy loss, and pre-eclampsia [1 - 5].

1.1- Historically Recognized Interactions.

The impact of oral infection on systemic health is not a new concept. Ancient Egyptians (2100 BC) mention tooth pain associated with women's reproductive system diseases [6]. In 400 BC, ancient Greece, Hippocrates suggested that arthritis could be cured by the removal of infected teeth [7]. In 1891 Miller originally published his "focal infection theory" suggesting microorganisms or their waste products obtain entrance of parts of the body adjacent to or remote from the mouth [8]. Miller blamed oral foci of infection for a number of regional and systemic diseases, ranging from tonsillitis and middle ear infection to pneumonia, tuberculosis, syphilis, osteomyelitis, endocarditis, meningitis and septicaemia. In the oral cavity, therapeutic edentulation was common as a result of the popularity of focal infection theory.

Since many teeth were extracted without evidence of infection, thereby providing no relief of symptoms, the lack of scientific evidence condemned this theory to dormancy [9,10].

In 1900, British surgeon William Hunter blamed many disease cases on oral sepsis. He concurred that bacteria and their products from local infections could be disseminated throughout the body and cause diseases in other organs [11].

In 1912 Focal infection theory's modern era really began with physician Frank Billing and his cases report of tonsillectomies and tooth extractions claimed to have cured infections of distant organs [(12)].

In 1916, pregnant guinea pigs were inoculated with streptococci harvested from human stillborn foetuses and this inoculation resulted in a 100 percent abortion rate [(13)].

In 1931 Galloway confirming that the focal infection found in teeth, tonsils, sinuses, and kidneys pose a risk to the developing foetus, he recommended that all foci of infection perceived to be a source of danger to any pregnant woman, should be removed early in pregnancy to the advantage of both the mother and the foetus and he first suggested that periodontal disease has more than just an association, but actually contributes to a low birth weight [(13)].

In 1989 Mattila and coworkers in a study from Finland had reported an association between oral diseases, tooth loss, and heart disease. This observation was followed by studies on a variety of other potential associations [(1)]

Collins and colleagues 1994 hypothesized that oral infection, such as periodontitis, could act as a source of bacteria and inflammatory mediators that could disseminate systemically to the foetal–placental unit, via blood circulation, and induce pregnancy complication [(14)].

In 1996 Offenbacher was the first investigator reported a potential association between oral infection and preterm low birth weight infancy [(15)].

In the last two decades, the scientific community has demonstrated a growing interest in determining whether periodontal disease is associated with pregnancy complications as well as research into the biological plausibility of these associations, and investigation of mechanisms that might explain potential causal relationships [(16)].

1.2- Periodontal Disease (PD)

Periodontal disease (PD) is a general term for a series of pathological alterations of the periodontium. The periodontium, in this sense, is the tissue surrounding and supporting a tooth; more importantly, the gingiva, alveolar bone, tooth cementum, and periodontal ligament (connective tissue joining the alveolar bone and the cementum). Fluid that bath the tooth at the gingival margin, known as gingival crevicular fluid, often contains inflammatory mediators and oral pathogens associated with periodontal disease. Although there are numerous periodontal diseases, they can be classified into two large groups: gingivitis and periodontitis. In gingivitis, only the soft gingival tissues are altered. In periodontitis, in addition to the soft tissue the hard tissue (bone, periodontal ligament, and cement) are affected [(17)].

When microorganisms are allowed to attach to the teeth; accumulate and form an organized structure known as a "bacterial biofilm", near the gingival margin, usually what follows is inflammation of the gingiva (gingivitis). In this case, the small space between the gingiva and the teeth, named (gingival sulcus) normally, increases in depth and, consequently, turns into a periodontal pocket (pathological deeping of the sulcus).

If the microbial flora of gingivitis is eliminated, the inflammation will recede and the gingiva will return to its normal status. It is possible that, if not properly treated, the pathological process of gingivitis may reach the hard tissue and, slowly or abruptly, cause alterations and result in periodontitis. In periodontitis, the most important alterations are resorption of the alveolar bone and destruction of the connective tissue between the bone and teeth (periodontal ligament), which result in attachment loss, and ultimately, to excess mobility, infection, and loss of the tooth [(17)].

There is a marked difference between the microbial flora that attaches to the teeth before gingivitis and that related to established periodontitis. In

gingivitis, the microbial flora is predominantly formed of Gam-positive, aerobic, saccharolytic, and immobile bacteria. Where as in periodontitis, the microbial flora is predominantly formed of Gram-negative, anaerobic or microaerophilic, proteolytic, and mobile bacteria **(17)**.

As the disease progresses, the pocket epithelium is the only barrier between the biofilms and connective tissue. The strands of them frequently ulcerated epithelium are easily broached, allowing bacterial access to the connective tissue and blood vessels. In patients with moderate-to-sever periodontitis, the total area of the pocket epithelium in direct contact with the sub gingival biofilm is surprisingly large ($72cm^2$); it may be the size of the palm of the human hand, and in case of advanced disease, it is much larger. Therefore Periodontitis can be considered a continuous pathogenic and inflammatory challenge at a systemic level, due to the large epithelium surface that could be ulcerated in the periodontal pockets this fact allows bacteria and their products to reach other parts of the organism, creating lesions at different levels **(18,19)**.

Periodontal bacteria may get introduced into the blood stream and cause infection after colonizing other sites of the organism (bacterial translocation that cause metastatic infections). Periodontal bacteria may also colonize the lower respiratory tract in individuals with predisposing factors, mainly through direct inhalation, without going to the blood stream, originating pulmonary infections. Periodontal infection can also promote an inflammatory and immune systemic response by releasing inflammatory mediators and liberation of proteins of the acute phase to a distant site, such as the liver, the pancreas, the skeleton or the arteries. Periodontal infection may also cause metastasis lesions due to the effect of microbial circulating toxins. The ability of periodontal pathogens and their virulence factors to disseminate and induce both local and systemic inflammatory responses in the host has led to the hypothesis that periodontal disease may have consequences beyond the periodontal tissue themselves **(20)**.

1.3-Preterm Low Birth Weight (PLBW).

Preterm low birth weight (PLBW) as defined internationally by the 29th world health assembly in 1976, is a birth weight of less than 2500g with a gestational age of less than 37 weeks [21]. Preterm delivery continues to be one of the most significant unsolved problems of public health and perinatology [22,23]. It is the major cause of neonatal mortality/morbidity in the world, accounting for up to 75–85 percent of the early neonatal deaths [24].

Introduction of neonatal intensive care methods during the1960s and the subsequent development of surfactant therapy in the 1980s resulted in improvement in the survival rates of PLBW neonates [25]. However, compared with infants of normal birth weight, PLBW infants are still 40 times more likely to die during the neonatal period, PLBW infants who survive the neonatal period face a higher risk of several neurodevelopment disturbance, health problems such as (asthma, upper and lower respiratory infections, and ear infections) and congenital anomalies [26,27,28].

Although most PLBW children are normal on neurological examination, the rates of neuromotor dysfunction are higher than in control groups [29]. A higher prevalence of behavioural problems is reported for PLBW children, including attention deficit hyperactivity disorder and formal conduct disorder. Learning problems among low birth weight children have been documented through teacher and parent rating of school performance and direct assessments of academic skills in achievement in reading, spelling and math. Studies of intellectual and academic functioning during adolescence of children born in the 1960s and earlier indicated that the adverse consequences of low birth weight were still apparent at that age [30,31].

Preterm birth and low birth weight are important health problem in both developing and industrialized countries. Worldwide, more than 20 million

infants are born with low birth weight with more than 95 percent of them being born in developing countries [32].

It was estimated that 11 percent of all pregnancies end in preterm birth. Globally, about 16 percent of the infants born in the world are low birth weight infants [33,34].

Various factors have been associated with the delivery of preterm and/or low-birth weight infants. Maternal risk factors include age, height, weight, socio-economic status, ethnicity, smoking, alcohol, nutritional status, and stress. In addition, parity, birth interval, previous complications, pre and ante-natal care, maternal hypertension, infections, and cervical incompetence may also be important [35,36].

While various factors have been found to predispose mothers to PLBW deliveries, the inability of medical intervention to resolve such occurrences is probably due to presence of other unidentified contributing factors, one possible contributing factor to this phenomenon is the effect of an infection on PLBW [37,38]. Infection is now considered one of the major causes of PLBW deliveries. The association between preterm labour and bacterial infection is well-established, and it has been reported that as many as 40–50 percent of cases involve an infection [39,40]. Studies have shown that intrauterine infections are highly prevalent among women who give birth prematurely [41].

Four possible mechanisms exist for microbes to spread to the uterus, an otherwise sterile environment: (i) the organisms from the vagina and the cervix ascend to the uterus; (ii) the organisms originate from elsewhere and are transmitted hematogenously; (iii) the organisms from the peritoneal cavity seed retrogradely through the fallopian tube; and (iv) they are inoculated accidentally inside the uterus during invasive procedures. Ascending infection is considered to be by far the most common route of infection. Haematogenous spread of organisms from other body sites to the uterus is a second route [42,43].

Both generalized infections, including viral respiratory infections, diarrhoea and malaria, and more localized infections of the genital and urinary systems can affect the gestational length [44].

1.3.1-Bacterial Vaginosis (BV).

Bacterial Vaginosis (BV) is a vaginal infection and is one of the most common factors associated with PLBW. Which accounts for up to 40 percent [45]. Gram-negative bacteria associated with this condition produce endotoxins and enzymes that stimulate pro-inflammatory cytokine production, which results in increased levels of TNF, IL-1, IL-6, and PGE_2 [46]. During normal pregnancy, the intra-amniotic levels of these mediators rise physiologically until a threshold level is reached, at which point labour, cervical dilatation and delivery are induced. Abnormal production of these mediators in the setting of infection triggers preterm labour and low birth weight [15,38].

However, 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. Also elevated levels of inflammatory mediators had been observed in PLBW deliveries, even in the absence of clinical or subclinical genitourinary tract Infection, it was postulated that the majority of PLBW deliveries are caused by infections of unknown origins. [47].

All these findings lead to the reasoning that an infection might be distant from the placental complex or the genitourinary tract. Thus, it was demonstrated that maternal infections during pregnancy perturb the normal cytokines and hormone regulated gestation resulting in pre-term labour, pre-mature rupture of membrane and pre-term low birth weight [48]. The hypothesis that infection remote from the foetal placental unit may influence PLBW has led to an increased awareness of the potential role of chronic bacterial infections elsewhere in the body [49].

1.4- Link between PD and PLBW.

Biological plausibility of the link between the two conditions; periodontal disease and preterm birth, can be summarized in three potential pathways. One of them refers to the haematogenous dissemination of inflammatory products from a periodontal infection, while the second potential pathway involves the foeto-maternal immune response to oral pathogens. The third pathway proposed to explain the theoretical causal relationship between periodontal disease and preterm birth involves bacteraemia from an oral infection [(49)].

1.5- Importance of the link between PD and PLBW.

A confirmation of whether periodontal disease is a risk factor for adverse pregnancy outcomes would be of great public health importance because periodontal disease is both preventable and curable. Improving periodontal health before or during pregnancy may prevent or reduce the occurrences of these adverse pregnancy outcomes and therefore reduce the maternal and perinatal morbidity and mortality [(50)]. Also, education for patients and health care providers regarding the biological plausibility of the association and the potential risks is indicated [(51)].

LITERATURE REVEIW

2- LITERATURE REVIEW

Although periodontal diseases are well known as an oral problem, in the past decade, there has been a shift in perspective. Research has been focusing on the potential impact of periodontal diseases on systemic health [52 - 55].

This chapter will review the literature on these topics:

1- Periodontal disease (PD).

2- Pre mature low birth weight (PLBW).

3- Association between (PD) and (PLBW).

4- Community perspective.

2.1- Periodontal Disease (PD).

2.1.1- Epidemiology of periodontal disease.

Periodontal disease is one of the most widespread diseases of mankind. No nation and no area of the world are free from it [56]. The most prevalent form of periodontal disease is mild form– gingivitis – affects 75 percent of adults in the United States. More advanced forms of periodontitis are also prevalent, affecting approximately 30 percent (moderate disease) and 10 percent (advanced disease) [57]. Chronic periodontitis affecting approximately 40 percent of the adult population in the United States and 0.13 percent of age between 14-17 had generalized aggressive periodontitis.

In Libya, Study done by Omar and Pitts (1991) on Libyan school children, had shown a high proportion of study participants with gingivitis, about 98.7 percent [58] another study from Benghazi done by Salma Mahfoud (2004) has showed that 96.6 percent of patient had gingivitis, 47.2 percent had chronic periodontitis and was 0.9 percent had localized form of aggressive periodontitis while 1.9 percent had rapid progressive form [59].

2.1.2- Anatomical Structure of Periodontal Tissue.

The periodontium includes those tissues that invest and support the tooth- the gingiva, the cementum covering the root surfaces of each tooth, the periodontal ligament that attaches the tooth root surface to the adjacent alveolar bone process that supports each tooth and the alveolar bone. The gingiva covers the structures that comprise the attachment apparatus (cementum, ligament, and adjacent alveolar bone). The gingiva is divided into free and attached gingiva. The free gingiva extends from the base of the gingival sulcus to the gingival margin. The tissues extending from the bottom of the sulcus to the mucogingival Junction are those that comprise the attached gingiva. Apical to the mucogingival junction, the alveolar mucosa is continuous with the mucous membrane of the lip, cheek, and the floor of the mouth. The adult dentition presents the gingival margin located on the enamel surface approximately 0.5 to 2.5mm coronal to the cervical line of each tooth. The gingival margin is adjacent to the opening of gingival sulcus, which is normally 2-3mm in depth clinically **(60,61)** (Figure 2.1).

Placing of a calibrated instrument, such as a periodontal probe, into the gingival sulcus provides the clinician with a measurement referred to as the probing depth. The term “pocket” is used to describe the histopathology in the soft and possibly the underlying bony tissues, reflecting an inflammatory response to oral infection. The periodontal pocket is the cardinal symptom of periodontitis. It is a pathologic fissure between tooth and sulcular or pocket epithelium, limited at its base by the junction epithelium. It is an abnormal apical extension of the gingival sulcus caused by an extension of the junction epithelium along the root surface and formation of a pocket epithelium as the periodontal ligament is detached and destroyed by the disease process “Pocket” is used to differentiate from the healthy gingival sulcus**(60,61)** (Figure 2.2).

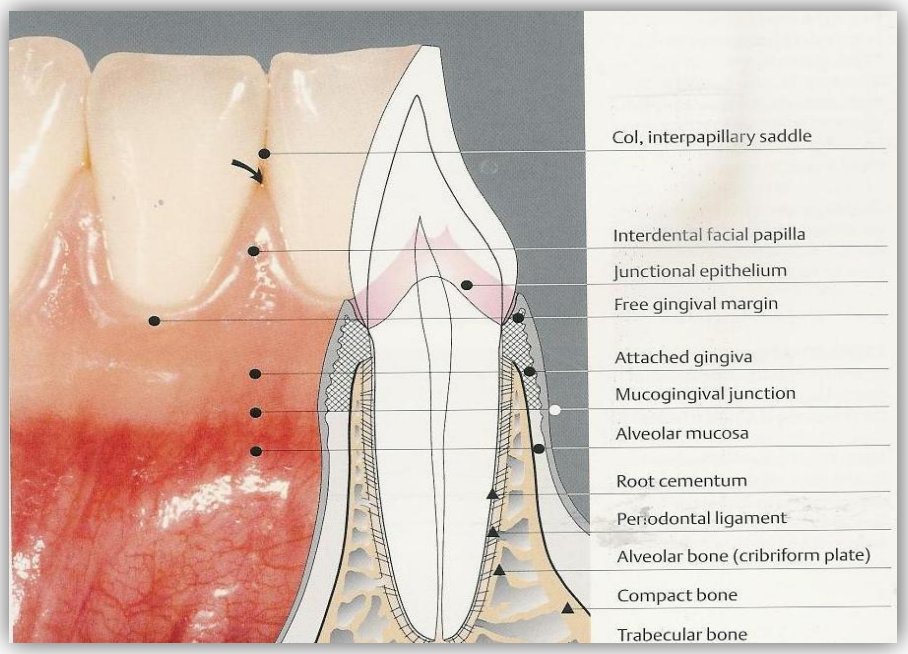

Figure 2.1: Periodontal Structures.
Source: Wolf HE, Rateitschak MH, Hassell TM. Color Atlas of Dental Medicine Periodontology P7. 3rd edition; 2005.

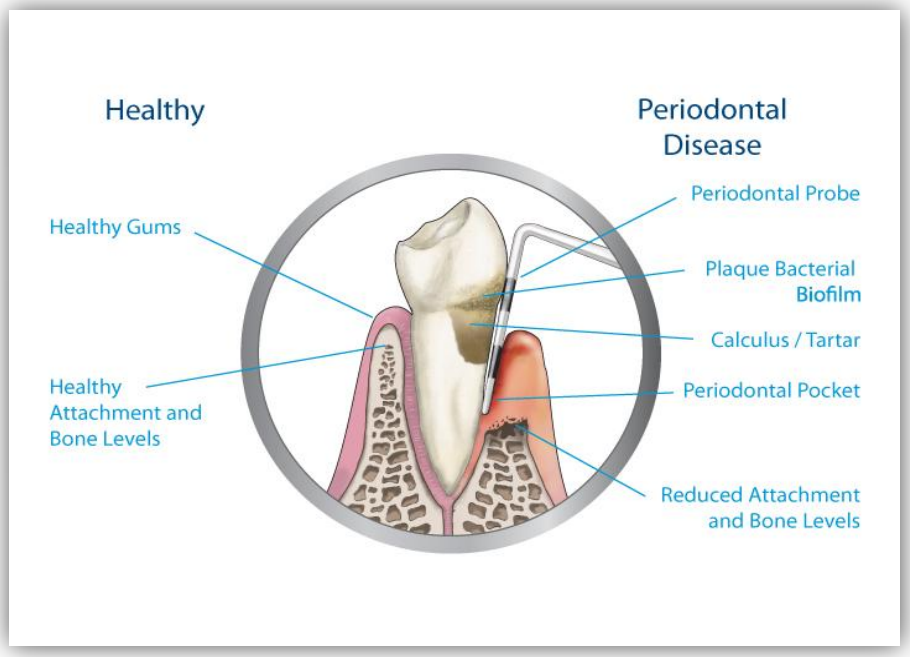

Figure 2.2: Pathological Deeping of Gingival Sulcus.
Source: Clarence Chew. Scaling & Root Planning – The Gold Standard in Treating Gum Disease, Periowave, February 18, 2011.

The gingival sulcus contains fluid. The gingival crevicular fluid (GCF) in disease reflects inflammation as measured by the levels of cytokines and tissue necrosis factor. Pocket depth and pocket levels of cytokine biomarkers can be used to monitor health and disease. Damage to the periodontal tissue is usually detected by means of periodontal probing, which shows loss of attachment of the tooth, or by radiographs that detect alveolar bone loss. These methods evaluate the damage caused by previous destruction episodes resulting in a retrospective diagnosis. GCF provides a quantitative biochemical indicator for the evaluation of the local cellular metabolism that reflects a person's periodontal health status. Since GCF is an inflammatory exudates that reflects ongoing events in the periodontal tissues that produce it [62,63].

2.1.3- Disease of Periodontal Tissue.

There are two major forms of periodontal disease**;** gingivitis (an inflammatory condition of the soft tissues surrounding a tooth or the gingiva) and periodontitis (involving the destruction of such supporting structures as the periodontal ligament, bone, or soft tissues) [64].

Gingivitis characterized by inflammation of the gums, redness, swelling and frequent bleeding (Figure 2.3).

Periodontitis is generally classified into three main categories: chronic disease, aggressive disease and manifestation of non-oral systemic diseases. Chronic and aggressive diseases are subdivided further on the basis of their location (localized or generalized) [65]."Gingivitis may occur simply associated with dental plaque, in which case it is called marginal gingivitis. It may also occur as a result of systemic involvement such as gingivitis in AIDS patients and hyperplasic gingival conditions associated with intake of drugs such as phenytoin, cyclosporine, nifedipine" [66].

Clinical attachment loss, alveolar bone loss, periodontal pocketing and gingival inflammation are the main clinical features of periodontitis [65] (Figure 2.4).

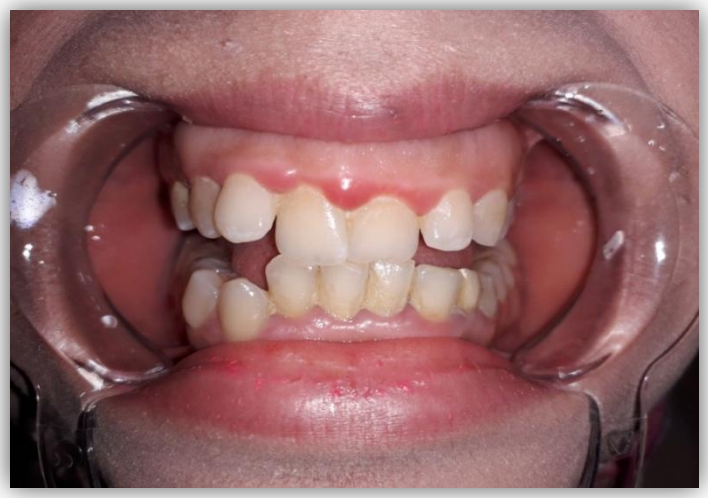

Figure 2.3: Gingivitis
Source: Periodontal Department, Dental Faculty- University of Benghazi**.**

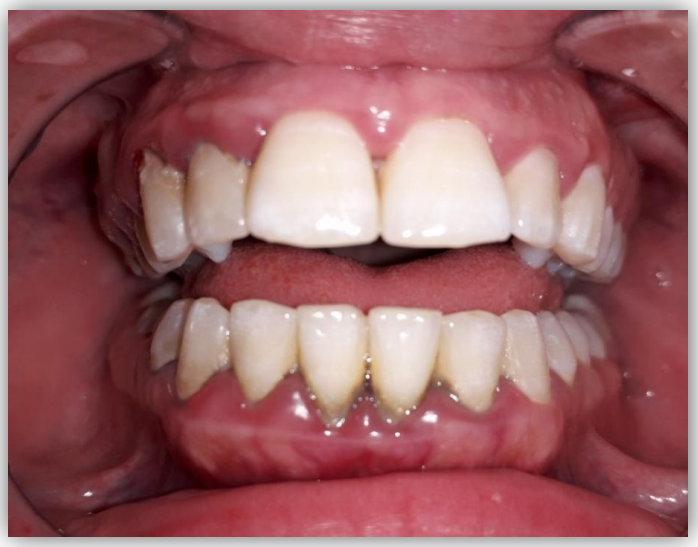

Figure 2.4: Periodontitis.
Source: Periodontal Department, Dental Faculty- University of Benghazi**.**

2.1.4- Aetiology of periodontal disease.

Periodontal disease results from complex interplay between the etiological agents, specific bacteria found in dental plaque, and the host tissue [(9)].

2.1.4.1- Dental Plaque.

Dental plaque (also called biofilm) can be defined as the soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restorations. Which is a sticky, colourless film that constantly forms on teeth in the absence of adequate oral hygiene. The gingival tissue will progress from health to an established or chronic gingivitis over a 3-weeks period [(67,68)].

2.1.4.1.1- Composition of Dental Plaque.

Dental plaque is composed primarily of microorganisms. One gram of plaque (wet weight) contains approximately 2×10^{11} bacteria. Indicate that more than 500 distinct microbial species are found in dental plaque. Nonbacterial microorganisms that are found in plaque include Mycoplasma species, yeasts, protozoa, and viruses.

The microorganisms exist within an intercellular matrix that also contains a few host cells such as epithelial cells, macrophages, and leukocytes. The intercellular matrix, estimated to account for 20 percent to 30 percent of the plaque mass, consists of organic and inorganic materials derived from saliva, gingival crevicular fluid, and bacterial products [(69,70)].

Organic constituents of the matrix include proteins, glycoproteins, polysaccharides and lipid material. Glycoproteins derived from saliva, are an important component of the pellicle and incorporated into the developing plaque biofilm. Polysaccharides produced by bacteria of which dextran is the predominant form, contribute to the organic portion of the matrix. Albumin, (proteins), probably originating from crevicular fluid has been identified as a

component of the plaque matrix. The lipid material consists of debris from the membranes of disrupted bacterial and host cells and possibly food debris.

The inorganic component of plaque is predominately calcium and phosphorus, with trace amounts of other minerals such as sodium, potassium, and fluoride. Plaque is actually heterogeneous in structure, with clear evidence of open fluid-filled channels running through the plaque mass [(71)]. These channels may provide for circulation within plaque to facilitate movement of soluble molecules such as nutrients or waste products. The bacteria exist and proliferate within the intercellular matrix through which the channels course.

This matrix confers a specialized environment, which distinguishes bacteria that exist within the biofilm from those that are free-floating in solutions such as saliva or crevicular fluid. For example, the biofilm matrix functions as a barrier. Substances produced by bacteria within the biofilm are retained and essentially concentrated, which fosters metabolic interactions among the different bacteria [(72,73,74)].

2.1.4.1.2 - Formation of Dental Plaque.

From the moment a baby passes through the birth canal and takes its first breath, microbes begin to reside in its mouth. Later on, as teeth erupt, additional bacteria establish colonies on the tooth surfaces. Dental plaque may be readily visualized on teeth after 1 to 2 days with no oral hygiene measures. In the absence of oral hygiene measures, plaque continues to accumulate until a balance is reached between the forces of plaque removal and those of plaque formation.

The process of plaque formation can be divided into three phases:

1. Formation of the pellicle coating on the tooth surface.
2. Initial colonization by bacteria.
3. Secondary colonization and plaque maturation [(75,76,77)].

2.1.4.1.3 - Classification of dental plaque.

Dental plaque had differentiated into two categories namely: supra- and sub-gingival.

Supra- gingival plaque

Coronal to the dento-gingival junction is most commonly found at:

-Gingival third of the crown of the tooth

-Inter-proximal areas

-Pits and fissures and on other such surface with irregularities.

Sub-gingival plaque

Apical to the dento- gingival junction is usually divided into:

-Tooth adherent zone

-Epithelial adherent zone

-Non adherent zone [(78)].

The different regions of plaque are significant to different processes associated with diseases of the teeth and periodontium. For example, marginal plaque is of prime importance in the development of gingivitis. Supra gingival plaque and tooth-associated sub gingival plaque are critical in calculus formation and root caries, whereas tissue-associated sub gingival plaques are important in the soft tissue destruction that characterizes different forms of periodontitis [(69)].

2.1.5- Association of Plaque Microorganisms with PD.

The oral cavity harbour more than 700 different bacterial species and there are over 500 bacterial species capable of colonizing the subgingival region, but the number of these commonly implicated in the disease process is around 10 or 15 species of gram negative anaerobes and spirochetes [(79,80)].

The initial bacteria colonizing the pellicle-coated tooth surface are predominantly Gram-positive facultative microorganisms such as Actinomyces viscosus and Streptococcus sanguis. These initial colonizers adhere to the pellicle through specific molecules, termed adhesions on the bacterial surface that interact with receptors in the dental pellicle. The plaque mass then matures through the growth of attached species, as well as the colonization and growth **(81,82,83)**.

Secondary colonizers are the microorganisms that do not initially colonize clean tooth surfaces, including; Prevotella intermedia, Prevotella loescheii, Capnocytophagaspp., Fusobacterium nucleatum, and Porphyromonas gingivalis. These microorganisms adhere to cells of bacteria already in the plaque mass. The ability of different species and genera of plaque microorganisms to adhere to one another, by process known as coaggregation in the later stages of plaque formation, coaggregation between different Gram-negative species is likely to predominate **(9,84,85)**.

In the mid-1900s, all bacterial species found in dental plaque were believed to be equally capable of causing disease, and periodontitis was believed to be the result of cumulative exposure to dental plaque. The association of specific bacterial species with disease came about in the early 1960s, when microscopic examination of plaque revealed that different bacterial morph types were found in periodontally healthy versus periodontally diseased sites. In the 1960s and 1970s, technical improvements were made in the procedures used to isolate, cultivate, and identify periodontal microorganisms **(86)**.

Most microorganisms involved in these PD are Gram-negative bacilli, anaerobes (Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Campylobacter rectus or capnophiles (Aggregatibacterium actinomycetemcomitans, Eikenella corrodens, Capnocytophaga ochracea). However, the most notable are the so-called “red-complex“ bacteria: Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola **(87,88)**.

Each type of periodontal disease has a subgingival flora consists of a combination of microorganisms of its own. In aggressive periodontitis were divided into two clinical entities; localized aggressive periodontitis and generalized aggressive periodontitis, each with a different microbiology. The microbiology of generalized aggressive periodontitis is more complex and association of Porphyromonas gingivalis (10 to 15 percent) and other Gram-negative bacilli (Eikenella corrodens, Capnocytophaga sp. Aggregatibacterium actinomycetemcomitans).

The rapidly progressive periodontitis is an aggressive form of periodontitis, the subgingival flora is typically composed of significant proportions of Porphyromonas gingivalis, Prevotella intermedia and other bacteria of the genus Bacteroides but Porphyromonas gingivalis appears to be one of the essential causative microorganisms in rapidly progressing periodontitis **(87)**.

Porphyromonas gingivalis (Pg) specifically has long been identified as "keystone pathogen" as this species is detected infrequently and in low numbers in health, and in greater frequency in destructive forms of the disease. This pathogen has an impressive armamentarium of virulence factors, including fimbrae, degradative enzymes, and exopolysaccharide capsule **(89)**.

PD has been characterized as a microbial-shift disease owing to shift in the subgingival microbial communities that colonize the periodontal pockets from a predominantly Gram-positive aerobic bacteria, to a dominance of Gram-negative anaerobes during the transition from periodontal health to PD Therefore, periodontitis is essentially induced by a dysbiotic microbiota. This concept of periodontal pathogenesis was recently termed "polymicrobial synergy and dysbiosis," or the PSD, model by Hajishengallis et al. (2012) **(90)**.

2.1.6- Pathogenesis of Periodontitis

The pathogenesis of periodontal destruction involves the sequential activation of different components of the host immune and inflammatory response, aimed in the first place at defending the tissues against bacterial aggression, reflecting the essentially protective role of the response, which it also acts as a mediator of this destruction [(91)].

The Gingival epithelia form barriers between bacterial plaque and gingival tissue, providing the first line of defence against plaque bacteria. The epithelium barrier consists of physical, chemical, and immunological barriers. Physical barriers are created by the unique architectural integrity of the stratified gingival epithelia, where epithelial cells are adjoined by tight junction-related structures and adhering junctions.

Chemical barriers are mainly formed by a variety of antimicrobial peptides (AMPs). AMPs, referred to as endogenously produced antibiotics, they have a broad spectrum of antimicrobial activity; thus, they contribute to controlling the bacterial load in the gingival sulcus. Defensins and a cathelicidin are major AMPs detected in the oral cavity [(92)]. The immunological barriers of gingival epithelia are provided by neutrophil, T cells, dendritic cells, macrophages, and mast cells distributed within the epithelia, lamina propria and gingival sulcus [(93,94,95)].

Gingival epithelial cells (GECs) are one of the first host cell types that encounter colonizing bacteria. As a consequence, GECs respond to the presence of bacteria through an elaborate signalling network, producing AMPs and cytokines, leading to host innate immune responses [(96)]. Most of microorganisms can produce tissue destruction in two ways:

(i) Directly, through invasion of the tissues and the production of harmful substances that induce cell death and tissue necrosis; and (ii) indirectly, through activation of inflammatory cells that can produce and release mediators that act on effectors, with potent proinflammatory and catabolic activity that can

interfere with normal host defence mechanisms by deactivating specific antibodies or inhibiting the action of phagocyte cells [97].

Progression of periodontitis occurs due to a combination of factors, including the presence of periodontopathic bacteria, high levels of proinflammatory cytokines; matrix metalloproteinase (MMPs), prostaglandin E2 (PGE2), low levels of anti-inflammatory cytokines including inter-leukin-10 (IL-10), transforming growth factor (TGF-β) and tissue inhibitors of MMPs (TIMPs) [98,99]. Periodontal disease is a consequence of the imbalance between the pathogenic potential of the biofilm and host immune defence properties, resulting in an inflammatory reaction of the periodontium [96].

2.1.7- Risk Factors of Periodontal Disease

Though microbial challenge is a primary initiating factor, there are many other variables that modify disease expression. These risk factors interfere with the way the body responds to bacterial invasion. Without the risk factors, the host may be capable of limiting periodontal tissue destruction.

A risk factor can be defined as an occurrence or characteristic that has been associated with the increased rate of a subsequently occurring disease. Risk factors may be modifiable or non-modifiable. Modifiable risk factors are usually environmental or behavioural in nature whereas non-modifiable risk factors are usually intrinsic to the individual and therefore not easily changed. Non-modifiable risk factors are also known as determinants [100].

2.1.7.1-Modifiable Risk Factors.

Smoking is the best established of the modifiable risk factors for developing periodontal disease. Smoking contributes to increased severity by the release of toxins into the oral cavity. It is the identified environmental risk most strongly associated with periodontal disease. In some studies the impact of smoking outweighs the effect of pathogenic bacteria as a determinant of outcome [101].

Also Diabetes increases the risk of periodontitis through an amplified inflammatory response and depressed wound healing. Diabetics have cytokines that respond to the bacterial challenge at a higher rate than normal. Gingival tissues and crevicular fluid contain elevated concentrations of these cytokines, producing high levels of MMPs that promote tissue destruction and disease severity [(102)]. In addition to smoking and diabetic evidence exists which suggests a relationship between periodontal status and nutrition, alcohol consumption, socioeconomic status and stress levels, although these relationships have not been clearly established [(100)].

2.1.7.2- Non- Modifiable Risk Factors

2.1.7.2.1- Genetic Factors

Risk for periodontitis is not shared equally by the population. It is clear that a high-risk group representing around 10-15 percent of the population, in whom the disease quickly progresses from chronic gingivitis to destructive periodontitis. This differential risk for periodontitis is consistent with heritable elements of susceptibility; periodontal research has greatly expanded to elucidate the role of genetics in periodontal disease states [(103,104,105,106)].

Evidence for a genetic influence on periodontal diseases comes from multiple sources including familial aggregation, formal genetic studies of aggressive periodontitis, the association of periodontitis with certain Mendelian inherited diseases and twin studies of chronic periodontitis [(107)].

Twin studies of adult periodontitis show greater concordance for periodontitis susceptibility between monozygotic twins than between dizygotic twins. It has been estimated that heredity accounts for about 50 percent of the enhanced risk for severe periodontitis. Given the critical role of neutrophil in inflammation, genetic defects in neutrophil function would be expected to affect periodontal disease. Genetic abnormalities in neutrophil function have been demonstrated in 75 percent of patients with aggressive periodontitis [(99)].

Individuals may respond differently. Variations in any number or combination of genes that control the development of the periodontal tissues or the competency of the cellular and humoral immune systems could affect an individual's risk for disease [107]. Consequently, there has been great interest in identifying allelic variants of genes that can be used to assess disease risk for periodontal diseases. Most genetic research in periodontitis has now focused on gene polymorphisms that play a role in immunoregulation or metabolism, such as cytokines, cell-surface receptors, chemokines, enzymes and others that are related to antigen recognition. And this differential response is influenced by the individual's genetic profile [105].

2.1.7.2.2 - Host Response

In response to the aggression, host defence mechanisms activate innate and adaptive immune responses. This action plays a crucial role in the destruction of periodontal tissue. While some bacteria interfere with the normal host defence mechanism by deactivating specific antibodies or inhibiting the action of phagocyte cells. Numerous bacteria can degrade tissue directly [97].

Birkedal- Hansen et al. suggested that host connective tissue is mainly degraded by the host. Thus, the loss of connective tissue is a defence mechanism; the host attempts self-protection by the apical proliferation of junction epithelium, escaping from the toxic root surface to avoid lesion progression [108,109,110].

The innate host response is initiated by toll-like receptors (TLRs); Pathogens can invade gingival epithelial cells by binding β-1 integrin and replicate, avoiding the host surveillance [111].

Toll-like receptors present on gingival epithelial cells can detect microbial structures highly conserved among pathogens, including lipopolysaccharide (LPS), peptidoglycan, bacterial DNA, double-stranded RNA, and lipoprotein, called pathogen-associated molecular patterns (PAMPs) [112]. Once TLRs present

on the surface of resident cells recognize PAMPs, they initiate the activation of several transcription factors including nuclear factor-κB (NFκB) and activator protein- 1 (AP-1) through the mitogen-activated protein kinase (MAK) cascade [113,114].

These in turn activate different innate immunity pathways, including cytokines and chemokines production that recruit non-resident leukocytes to periodontal space. In turn, activated leukocytes, the adaptive immunity cells secrete proinflammatory cytokines and chemokines in the tissues [115]. It is now accepted that the amplification of this initial local host response (lasting approximately 21 days) results in the propagation of the inflammation and leads to the destruction of soft and mineralized periodontal tissues [116].

The resident cells involved in the innate host response are many including epithelial cells, gingival and periodontal ligament, fibroblasts, osteoblast, and dendritic cells [114]. Epithelial cells produce interleukin-8 (IL-8), a neutrophil chemo attractant, which recruits neutrophils migration [117] and increases monocyte adhesion in the blood vessels. Neutrophils that enter the periodontal environment are primed and exhibit increased production of proinflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-α (TNF-α) [118]. These cytokines mediate periodontal tissue destruction by stimulating bone resorption. Monocytes, on the other hand, can differentiate into osteoclasts (OCs) [119].

Dendritic cells (DCs) are encountered once the epithelial barrier is invaded by microorganisms. These cells activate an immune response, either acting as antigen-presenting cells or producing IL-12 and IL-18 that consequently promote interferon-γ (IFN-γ) secretion by NK cells and later by T cells [120].

Periodontal ligament fibroblasts (PDLFs) and gingival fibroblasts (GFs) are the main cells of periodontal soft connective tissue and are accessed as the microorganisms breach the epithelial barrier. They respond through the release of cytokines and degradation molecules. GFs produce TNF-α, (IL)-6, (IL)-8,

macrophage inflammatory protein (MIP)-1 alpha, and stromal-derived factor (SDF)-1, which are important regulators of inflammatory process and bone metabolism [121].

Expression of matrix metalloproteinases (MMPs), laminin-8/9, and laminin-2/4 becomes accentuated in PDLFs; these cells also contribute to periodontal inflammation and bone loss [122]. Microorganisms can go deeper in the periodontal tissue and reach the surface of alveolar bone. All these events, which represent the initial response to the infection, establish a local inflammation proper of the innate immunity and are responsible for the alveolar bone loss.

After this initial response, the infection activates the adaptive immunity process; dendritic cells other than participating to the innate inflammatory response have the ability to capture and present antigens to B and T cells of the acquired immune system [123] Activated CD4 T-helper cells produce subsets of cytokines which will define phenotypically distinguished immune responses; Th-1 and Th-2 cells, respectively, associate with cellular and humoral immunity [124].

B cells are also activated and are transformed into plasma cells, which produce antibodies against bacterial antigens. As a result, tissues affected by periodontitis become colonized with both lymphocytes subtypes, but with a larger proportion of B cells than T cells. This inflammatory scenario drives the destruction of connective tissue and alveolar bone [125].

So Periodontal diseases are inflammatory diseases in which microbial etiologic factors induce a series of host responses that mediate inflammatory events in susceptible individuals, dysregulation of inflammatory and immune pathways leads to chronic inflammation, tissue destruction and disease. That is why periodontitis has been regarded as the result of hyper-immune or hyper-inflammatory responses to plaque bacteria [126,127].

2.2- Premature Low Birth Weight (PLBW).

Pregnancy is normally a healthy physiological process that sometimes has adverse outcomes including low birth weight (<2500g) or very low birth weight (<1500g), pre-term birth (<37weeks) or very pre-term (<32weeks), growth restriction (weight for gestational age), pre-eclampsia (commonly defined as maternal hypertension and proteinuria after the 20th gestational week), miscarriage and/or still birth [(128)].

2.2.1- Definition and Categorization.

2.2.1.1- Birth Weight (BW).

Birth weight is the first weight of the foetus or newborn obtained after birth. For live births, birth weight should preferably be measured within the first hour of life before significant postnatal weight loss has occurred. Birth weight is a strong predictor of infant growth and survival [(129)].

2.2.1.2- Low Birth Weight (LBW).

Low Birth Weight (LBW) is defined as the weight of live born infants less than 2,500g, for a given time period. LBW infants can be classified according to their gestation into term (born after 37 and before 42 completed weeks of gestation) and preterm (born up to 37 completed weeks of gestation) [(130)]. Low birth weight can be caused either by premature delivery (short gestation) or by foetal growth retardation [(131)].

2.2.1.3- Intra-uterine Growth Restriction (IUGR).

Intrauterine Growth Restriction (IUGR) is the birth weight less than 10th percentile for gestational age; birth weight less than 2500g and gestational age greater than 37 weeks; and birth weight less than 2 standard deviations below the mean value for gestational age [(132)].

2.2.1.4- Preterm Birth (PTB).

Preterm Birth is defined as gestational age less than 37 weeks at delivery. There are sub-categories of PTB based on gestational age, namely; extreme prematurity (<28 weeks), severe prematurity or very preterm (28-31 weeks), moderate prematurity (32-33 weeks) and near term or late preterm (34-36 weeks). Prematurity and IUGR are the two main causes of LBW. The majority of LBW in developing countries is due to IUGR, while most LBW in industrialized countries is due to preterm birth [(133)].

2.2.1.5- Premature Low Birth Weight (PLBW).

Premature Low Birth Weight (PLBW) is defined as birth weight less than 2500g at gestational age less than 37 weeks [(134)].

2.2.1.6- Spontaneous Preterm Birth (SPTB).

Spontaneous Preterm Birth (SPTB) is defined as delivery before 37 weeks as a result of spontaneous labour or rupture of membranes [(134)].

2.2.1.7- Preterm Premature Rupture of Membranes (PPROM).

Preterm Premature Rupture of Membranes (PPROM) is defined as spontaneous rupture of the membranes at less than 37 weeks of gestation at least one hour before the onset of contractions [(134)].

2.2.2- Significance of PLBW

Pre-term birth (PTB) is a major cause of infant mortality and morbidity that has considerable societal, medical, and economic repercussions. The rate of PTB appears to be increasing worldwide and efforts to prevent or reduce its prevalence have been largely unsuccessful [(135)].

Infants born with low birth weights begin life immediately disadvantaged and face extremely poor survival rates. In most developing countries it was

approximated that every ten seconds an infant dies from a disease or infection that can be attributed to low birth weight [133].

LBW is an important cause of perinatal, neonatal and post natal mortality and morbidity. Preterm birth accounts for 75 percent of perinatal mortality and more than half of the long-term morbidity. Generally the risk of neonatal mortality for LBW infants is 25 to 30 times greater than for infants with birth weight exceeding 2500g, and it increases sharply as birth weight decreases [135,136]. LBW is closely associated with, inhibited growth, chronic diseases later in life and associated with, impaired immune function, and poor cognitive development for neonates (newborns 1-28 days of age) and infants [137,138].

Infants born LBW are at risk to develop acute diarrhoea or to be hospitalized for diarrhoeal episodes at a rate almost two to four times greater than their normal birth weight counterparts. LBW is an important determinant of diarrhoea, death and of hospitalization from dehydration [138,139]. Although most of the organs of prematurely born infants are immature, the brain and the respiratory system are the systems primarily susceptible to complications arising from PTB. Infants who are LBW risk contracting pneumonia or acute lower respiratory infections (ALRI) at a rate almost twice that of infants with normal birth weight; and more than three times greater if their weight is less than 2000g [140,141].

According to Arifeen (1997) almost half of the infant deaths from pneumonia or ALRI and diarrhoea could be prevented if low birth weight were eliminated [142]. Approximately one in four PTB infants are reported to have substantial neurological morbidity such as cerebral palsy, developmental delay and/or sensory impairments (visual or auditory) the prevalence of cerebral palsy is inversely related to the gestational age of the infant. In the early years of life, preterm infants also tend to have a higher prevalence of minor neuromotor dysfunction and poorer coordination when compared to term infants [140,143].

Adverse birth outcomes can also have a deleterious effect on the teeth. Molar Incisor Hypomineralization and enamel developmental defects in molars and incisors are increased in children who were preterm, had low gestational age or were LBW infants. These children also exhibit higher plaque accumulation and higher associated gingival inflammation; possibly due to the rough surfaces of the teeth and the potential sensitivity associated with these teeth which might interfere with tooth brushing [(144)].

LBW is also implicated as a contributor to impaired immune function which may be sustained throughout childhood. Although most preterm babies in developed countries survive, they are at increased risk of neurodevelopmental impairments and respiratory and gastrointestinal complications [(144)].

In Benghazi the prevalence of LBW is 4 percent comparatively low in comparison to other developing countries but with limited resources mortality rate is 10.6 percent [(145)].

2.2.2.1- Economic implications.

The cost associated with providing care for preterm infants, who may spend numerous months in hospital, has significant implications for the economy these effects exert a heavy burden on families, society and health system [(146)]. A nationwide survey carried out by Russell and colleagues in 2001, regarding the hospital cost of preterm infants showed that in the United States, 4.6 million infants were hospitalized, costing $12.4 billion. Of these, 8 percent of hospitalizations were for preterm or low birth weight infants; this cost is partly explained by the increased cost of caring for preterm infants in hospital.

Additionally, preterm infants and extremely preterm infant have on average an increased length of stay in hospital than uncomplicated newborns: 12.9 days and 42.2 days versus 1.9 days respectively [(147)]. These data suggest that major infant and paediatric cost savings can be achieved by the identification and

prevention of preterm birth in addition, Addressing preterm birth is essential for accelerating progress towards Millennium Development Goal- 4 [148].

2.2.3- PLBW on a Global Scale

An estimated 15 million babies are born too early every year. Complications of preterm birth are the single largest direct cause of neonatal deaths, responsible for 35 percent of the world's 3.1 million deaths a year, and are now the second most common cause of death after pneumonia in children under 5 years old. This means that altogether more than one million babies die each year due to complications of PLBW [146,149,150].

Of all live births worldwide in 2010, 11.1 percent were born preterm. In the USA, the preterm delivery rate is 12-13 percentages, In Libya 8.3 percent, In Europe and other developed countries reported rates are generally 5-9 percentage. In low-income countries, on average, 12 percent of babies are born prematurely [149]. The differences in rates are not so striking, but the differences in outcome are dramatic. More than 90 percent of extremely preterm babies (<28 weeks) born in low-income countries die within the first few days of life, yet less than 10 percent of babies of this gestation die in high-income settings [150,151].

2.2.4- Physiology of Normal Pregnancy

After conception, the placenta that is totally derived from the foetus invades and grows supported completely by the maternal uterine tissue. Through the vessel-rich placenta, there is exchange of nutrients and waste between the mother and the foetus. This transportation occurs via the umbilical cord that connects the foetus with the placenta. Having the necessary resources, the foetus grows in the amniotic fluid which is contained by the amniotic sac. The walls of this cavity consist of the amnion and the chorion and like the placenta are attached to the uterus through the decidua and the myometrium [152] (Figure. 2.5).

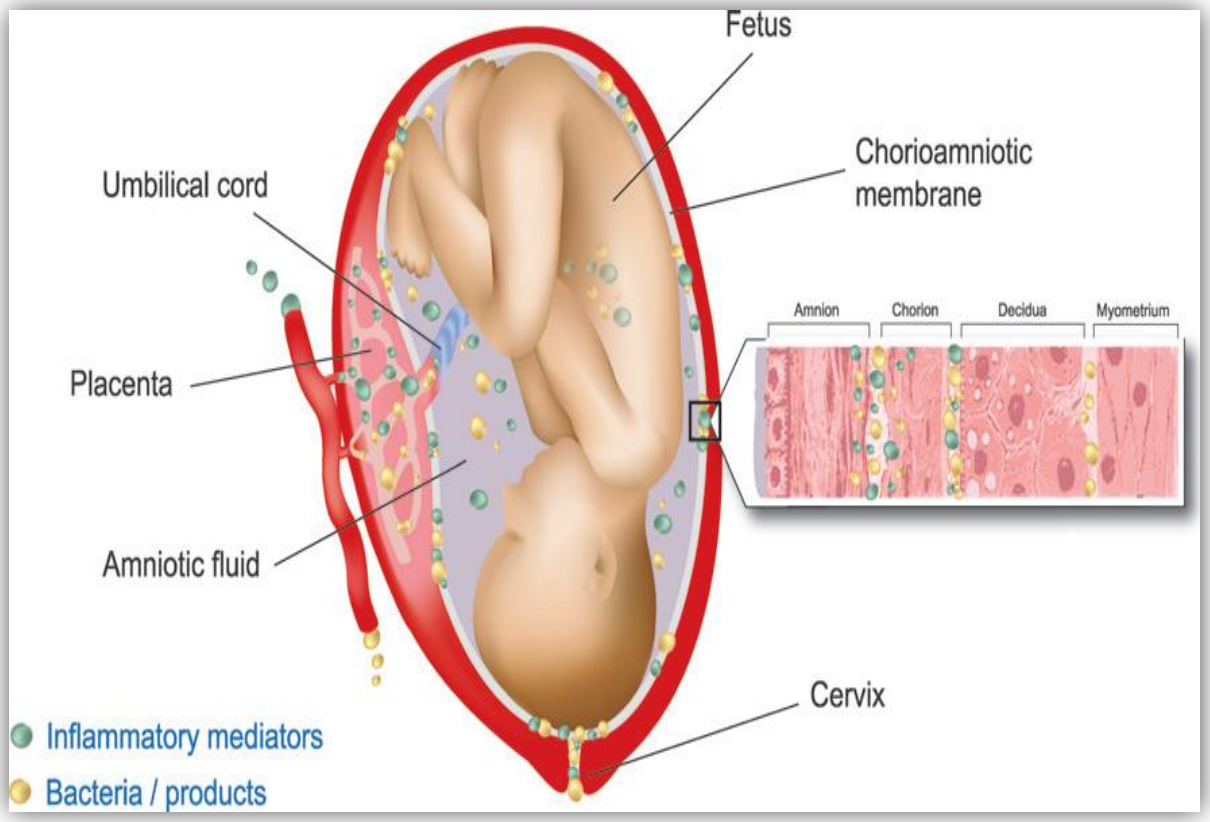

Figure2.5: Anatomical structure of placental unit

Source. Madianos PN, et al. Adverse pregnancy outcomes (APOs) and periodontal disease: pathogenic mechanisms, *J Periodontol*, 2013; 84(4 Suppl.):S170-S180.

As the foetus grows the increasing needs for nutrients and the decreasing space become critical parameters for the survival of both mother and foetus. Hence, as pregnancy progresses, amniotic fluid levels of prostaglandin E2 (PGE2) and inflammatory cytokines, such as TNF-α and IL-1b rise Steadily until a critical threshold level is reached to induce rupture of the amniotic sac membranes, uterine contraction, cervical dilation and delivery. Thus, normal parturition is controlled by inflammatory signalling and this process represents a triggering mechanism that can be modified by external stimuli including infection and inflammatory stressors [(152)].

2.2.5- Pathogenic Mechanisms of Preterm Labour

The pathogenesis of preterm labour and subsequent preterm birth is not well understood but is suspected to be the result of an idiopathic activation of the normal labour process or of a pathologic insult to normal gestational function, including infection (identified or not) [(153)] A large number of studies associate an increase in the levels of local and systemic markers of inflammation with adverse pregnancy outputs (APOs). Hence, elevated levels of IL-1b, IL-6, TNF-α, PGE2, fibronectin and α-foetoprotein in the amniotic fluid have been associated with premature birth (PB) [(154,155)].

Moreover, C-reactive protein (CRP), which is an acute phase reactant synthesized by the liver in response to pro-inflammatory cytokines. Hence a marker of systemic inflammation is also associated with PLBW [(156,157,158)]. As the increased release of inflammatory cytokines and mediators plays critical roles in the pathogenesis of PB/LBW, infections of the genitourinary tract have been evaluated [(159)].

Indeed, intra-uterine infection can be confined to the decidua (deciduitis), extend to the space between the amnion and the chorion (chorioamnionitis), and reach the amniotic fluid (amniotic fluid infection). Moreover, it may involve the

placenta (villitis), the connective tissue of the umbilical cord (funisitis) and the fetus (sepsis).

Microorganisms can gain access to the amniotic cavity by:-

1-Ascending from the vagina and the cervix,

2- Haematogenous dissemination through the placenta,

3-Accidental introduction at the time of invasive procedures (amniocentesis),

4- Retrograde spread through the fallopian tubes [(134)].

2.2.6- Causes of Preterm Labor.

Preterm labour is thought to be a syndrome initiated by multiple mechanisms, including infection or inflammation and other immunologically mediated processes. A precise mechanism cannot be established in most cases, therefore factors associated with preterm birth, but not obviously in the causal pathway, have been sought to explain preterm labour [(134)]. Following are the factors associated with preterm birth**.**

2.2.6.1-Risk Factors Associated With Preterm Birth.

Multiple factors have been associated with PB and/or LBW, some of which are preventable, e.g., alcohol, smoking or drug use during pregnancy, high or low maternal age (≥34 years old or ≤17 years old), African-American ancestry, low socioeconomic status, inadequate prenatal care, low maternal body mass index (BMI), hypertension, generalized infections, genitourinary tract infections, cervical incompetence, diabetes, nutritional status, stress and multiple pregnancies [(160,161)].

Increasing efforts have been made to diminish the effects of these risk factors by preventive interventions during prenatal care. However, these have not reduced the frequency of PB and/or LBW infants partly because these risk factors are not present in approximately 50 percent of cases. Consequently, the

search continues for other causes for PB and/or LBW including the presence of chronic infectious diseases [160,161,162].

2.2.6.2-Infection and PLBW.

Twenty-five percent to 50 percent of PLBW deliveries occur without any known aetiology, and there is increasing evidence that infection may play a significant role in pre-term delivery. Both generalized infections, including viral respiratory infections, diarrhoea and malaria, and more localized infections of the genital and urinary systems can affect the gestational length.

Associations between chorioamnionitis, infection of the amniotic fluid and PLBW have been established [163,164]. The infection hypothesis suggests that during a subclinical infection, the micro-organisms and their lipopolysaccharides enter the uterine cavity during pregnancy by the ascending route from the lower genital tract or by the blood-borne route from a non-genital route, hence causing PTB. It has been suggested that adverse pregnancy outcomes (APO_s) is commonly associated with Bacterial Vaginosis (BV) [165,166,167].

2.2.6.2.1- Bacterial Vaginosis (BV).

Bacterial Vaginosis is a condition in which the normal, lactobacillus predominant vaginal flora is replaced with anaerobic bacteria, Gardnerella vaginalis, and Mycoplasma hominis [46]. BV associated with preterm delivery, premature rupture of membranes, infection of the chorion and amnion, histological chorioamnionitis and infection of amniotic fluid [168 - 174].

Bacterial invasion of the choriodecidual space can activate the foetal membranes or trigger the maternal immune system to produce a wide variety of cytokines and growth factors. This has been shown to elicit an inflammatory burden resulting stimulation of prostaglandin synthesis and the release of matrix metalloproteinase (MMPs), which account for the uterine contractions and

membrane rupture, respectively, leading to the induction of labour [175,176,177]. Intrauterine infection remains asymptomatic until labour begins or the membranes rupture. Sometimes even during labour, most of the women have no symptoms other than preterm labour abdominal pain, or peripheral-blood leukocytosis. Therefore, identifying women with intrauterine infections or abnormal quantities in amniotic fluid is a crucial task [178,179].

The association between infections of the uterine, genital and urinary systems and the risks of PT/ LBW (PLBW) deliveries has been demonstrated by a considerable number of studies. However, it was noted that a consistent and reproducible feature of PLBW cases, which is in increased level of maternal inflammatory mediators and cytokines such as prostaglandin E_2 (PGE_2) and tumour necrosis factor alpha (TNF-α), may occur even in the absence of infections of the amniotic cavity or the genitourinary tract. This has led to a conclusion that PLBW cases are probably caused by extra-uterine infections of unknown origin [174,178,180,181].

This suggests that distant sites of infection (oral cavity) or sepsis may target the placental membranes. For these reasons, researchers have explored the role of other maternal infections, including periodontitis, in the aetiology of preterm birth [38].

2.3 – ASSOCIATION BETWEEN PD and PLBW

2.3.1- Pregnancy and Periodontal Tissue

In a woman's life, there are major physiological and hormonal changes occur in pregnancy. 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. The changes occurring in the women body are the result of adaptation in order to create and maintain the conditions for the development of the foetus and birth. The changes apply to all physiological systems and processes in the body [182,183,184].

During pregnancy, changes in hormone levels promote an inflammatory response that increases the risk of developing gingivitis and periodontitis as a result of varying hormone levels, without any changes in the plaque levels [185,186]. Fifty percent to seventy percent of all women will develop gingivitis during their pregnancy. This type of gingivitis is typically seen between the second and eighth month of pregnancy commonly referred to as "pregnancy gingivitis" [187,188]. The incidence of pregnancy gingivitis has been reported at varying degrees ranging during the first trimester is 67.49 percent, the second 74.19 percent and the third, 79.17 percent [189].

During the course of their pregnancy, women tend to suffer a decline in their periodontal health and suffer from the exacerbation of pre-existing unfavourable periodontal conditions. Several gingival changes occur in pregnancy which includes increased gingival inflammation especially on the gingival margin and interdental papilla, oedema, pitting, increased gingival crevicular fluid flow, increased bleeding on probing, increased gingival probing depths and increased tooth mobility [190,191,192]. Hormonal changes through increased production of oestrogens and progesterone are suspected to be the cause of the increased risk for gingival and periodontal diseases during pregnancy [190].

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 erythemas have been shown to increase up to 1 month postpartum after which they decrease. Increased levels of the hormones progesterone and oestrogen can have an effect on the small blood vessels of the gingiva [193,194]. The progression or severity of gingivitis may advance during pregnancy due to the influence of fluctuating hormones [192].

Multiple mechanisms have been suggested to explain how hormonal changes increase the susceptibility to periodontal diseases. Firstly, gingival inflammation could be due to the increased vascular flow caused by changes in hormone levels, resulting in greater vascular permeability, gingival edema and increased prostaglandin production. Also, during pregnancy there is a change in the immune system and change in connective tissue metabolism. For example, the number of neutrophils increases during pregnancy and their function is altered resulting in a gingival tissue which is less resistant to infection. In addition, there is a decrease in IL-6 production, which will also lower the resistance to infection [195].

During pregnancy, increased levels of progesterone and oestrogen paralleled gingival conditions and proportions of *P*. intermedia (Prevotella intermedia). The shift of microorganisms, represented by an increasing anaerobic-to-aerobic ratio, is a result of change in the subgingival microenvironment caused by an accumulation of active progesterone whose metabolism is reduced during pregnancy and the ability of *P*. intermedia to substitute an essential growth factor, vitamin K, with progesterone and oestrogen. Jansen et al. (1981) demonstrated a 55-fold increase in the proportion of *P*. intermedia in pregnant women compared with the nonpregnant controls. This suggests that progesterone plays a major role in the shift in microorganisms. This increases the mother's susceptibility to oral infections, allowing pathogenic bacteria to proliferate and contribute to inflammation in the

gingiva [193,195]. High concentrations of progesterone alters the rate and pattern of collagen production and increases the metabolic breakdown of folate which is necessary for tissue maintenance, also inhibits the production of matrix metalloproteinase which cause the destruction of the collagen fibres in the course of periodontitis [183]. The increase in oestrogen levels affect cell proliferation and an increase in gingival epithelial glycogen and reduce the effectiveness of the epithelial barrier [196].

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 PGE1 and PGE2 which are potent mediators of inflammation this hyper- Inflammatory state increases the sensitivity of the gingiva to the pathogenic bacteria found in dental biofilm. During labour, when the placenta is withdrawn, a marked fall occurs in both progesterone and estrogens levels. Within 2–3 days of delivery, the hormone concentrations have reached their non-pregnant levels [182].

2.3.2- Biological Hypotheses Link between PD & PLBW

There is a large body of evidence pointing to infection as a key factor in adverse pregnancy out comes [166,197,198,199]. Oral mechanical manipulation (e.g., tooth brushing, dental procedures, and even routine mastication) can cause bacteraemia [200]. Chronic periodontal infections can produce local and systemic host responses leading to transient bacteraemia. Lipopolysaccharides (LPS) endotoxins and other bacterial substances can gain access to gingival tissue, initiate and perpetuate local inflammatory reactions, and consequently produce high levels of proinflammatory cytokines. Such activations of maternal inflammatory cell responses and cytokine cascades play important roles in the pathophysiological processes of preterm labour, low birth weight, and pre-eclampsia [166,200].

In addition, LPS, bacteria from subgingival plaque, and proinflammatory cytokines from inflamed periodontal tissue can enter the bloodstream, reach the maternal-fetal interface, trigger or worsen maternal inflammatory response, and increase plasma levels of prostaglandin and cytokines (e.g., tumour necrosis factor), so physiological levels of PGE_2 and TNF-α in the amniotic fluid may increase and induce a preterm birth [(201,202,203)]. Thus, it appears that periodontal disease may play a nonspecific role in various adverse pregnancy outcomes. In addition; during the second trimester of pregnancy, the proportion of Gram-negative anaerobic bacteria in dental plaque increases respect to aerobic bacteria. Fusobacterium nucleatum and other subspecies coming from the oral flora have been found in the amniotic fluid of women with preterm births [(48)].

Moreover; the risk of prematurity was higher when IgM was detected against at least one periodontal pathogen and even higher when high levels of inflammatory mediators were measured. These results suggest that the global effect of the foetus exposition to oral pathogens and the inflammatory foetus response may be a mechanism by which maternal periodontitis increases the risk of preterm births [(204)]. Considering these evidences, mainly, three biological hypotheses theories have been proposed to link preterm birth and periodontal diseases (Figure 2.5) which are:

(i) Bacterial spreading.

(ii) Inflammatory products dissemination.

(iii)Role of feto-maternal immune response against oral pathogens [(205)].

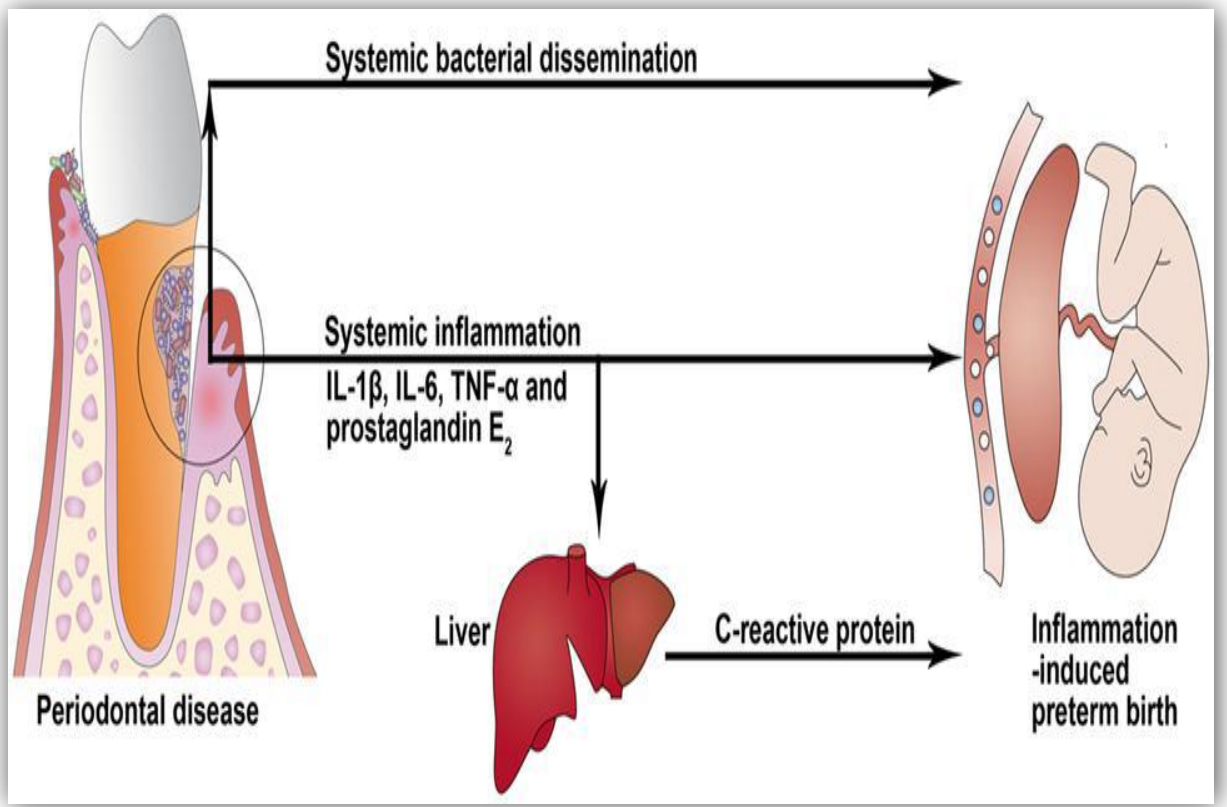

Figure 2.6: Potential biological mechanisms linking PD to PT

Source: Hongyu Ren and Minquan Du. Role of Maternal Periodontitis in Preterm Birth, Front Immunol, 2017; 8: 139.

2.3.3-Epidemiological studies about the link between PD and PLBW

Offenbacher and co-associates (1996) were the first group of investigators to report a link between poor maternal periodontal health and adverse pregnancy outcomes (APO_s) including preterm delivery; they concluded that 18.2 percent of PLBW may result from periodontal disease. Since then, researchers have investigated these possible associations for over a decade [206,207]. Offenbacher's study results suggest that periodontal disease may be a risk factor for PLBW. But what the study literally showed was a very strong association. Subsequent reports have found mixed results; corroborated by some authorities [208,209,210,211,212]. But contradictory by others [213,214,215]. No study had repeated the degree of association originally reported by Offenbacher and co-associates (1996) who suggested that periodontal infection during pregnancy could lead to a seven-fold risk of PTB; Jeffcoat et al. (2001) claimed 4.4-fold risk of PTB, Lopez et al. (2002) suggested a 3.5 for LBW/PT in women with moderate periodontal disease [209,216].

Two relatively large studies in the United Kingdom (UK) failed to find any association between maternal periodontal disease and risk of preterm delivery [214,215]. The reasons for the differences in findings are unclear. In the United States (US), associations between periodontal disease and preterm delivery appear to be stronger and more consistent in studies that included higher proportions of subjects from African-American racial/ethnic groups and subjects who smoke during pregnancy [50,217]. Several investigators have noted that positive associations were more commonly observed in the US studies where the proportion of African-American subjects exceeded 60 percent [207,209].

One difference was found in studies conducted in the USA or in developing countries and those conducted in European countries and Canada the former tended to include African American women and women from

economically disadvantaged families; they consistently reported significant associations between periodontal disease and adverse pregnancy outcomes. In contrast, the studies conducted in European countries or Canada (all of which offer their citizens universal health care) did not find an association between periodontal disease and APO. This suggests that the effects of periodontal disease on APO may be different according to the socio-economic status and access to dental care [(50)].

Important periodontal pathogens have been detected in human placentas of women with preeclampsia [(187)] and in the amniotic fluid of pregnant women with a diagnosis of premature labour [(185).] Foetal exposure to periodontal pathogens from maternal oral biofilm has also been demonstrated in umbilical cord blood samples from preterm births by detecting maternal immunoglobulin G (IgG) as well as fetal immunoglobulin M (IgM) to one or more specific oral pathogens [(218).] Suggesting that P. gingivalis and C. rectus could act as fetal infectious agents eliciting complications during pregnancy [(219)].

Despite isolating periodontal pathogens in dental plaques collected from women who delivered preterm and who had periodontitis, Dortbudak et al. (2005) failed to isolate microorganisms in amniotic fluid [(212)]. These differences in studies may be due to difference in the distribution and virulence of specific periodontal pathogens contributes to heterogeneity across studies. This is supported by studies documenting differences in the periodontal pathogens detected across populations sampled from diverse geographic locations [(220,221)]. Several studies report associations of APO/PLBW with higher gingival crevicular fluid levels of PGE_2 and IL-1b [(48,222,223)], and elevated amniotic fluid concentrations of PGE2, IL-1b and IL-8 [(212)].

Despite a tendency of higher mean gingival crevice fluid IL-1b levels, Noack et al. (2005) found no periodontitis associated increased risk for PLBW and the varying results could be due to the effects of specific environmental or genetic risk factors, which result in variable maternal reactions [(224)].

Results from Interventional studies have also been inconsistent. A three-year retrospective examination of a large insurance company database suggested that receiving preventive dental treatment is associated with a lower incidence of APO compared with instances in which no dental services are delivered [225].

However, multicenter intervention studies such as The Maternal Oral Therapy to Reduce Obstetric Risk (MOTOR) [226], The Periodontal Infection and Prematurity Study (PIPS) [227] and The Obstetrics and Periodontal Therapy (OPT) trials [228,229] did not significantly reduce the rates of adverse pregnancy outcomes although the periodontal health of the patients improved.

These results contradict smaller studies which showed significant differences between PTB (preterm birth) and FTB (full-term birth) following treatment [230,231]. Inconsistencies may arise due to population differences, severity of disease, different clonal types of bacterial species as well as differences in the management of patients [232]. From intervention studies would tend to suggest that periodontitis treatment during pregnancy whilst not causing damage doesn't seems to offers great benefits in terms of reducing pregnancy complications [233]. Results from previous observational and Interventional studies conducted in different populations have also been inconsistent. The reasons for these inconsistencies are unclear [50].

In 2011 Africa CWJ. mentioned that "significant association between periodontal disease and adverse pregnancy outcomes found in, Thailand, Saudi Arabia, Turkey, Brazil , Venezuela, Chile, Senegal, South Africa, Hungary, Croatia, Finland, USA, Austria, Taiwan and Japan. No association and/or contradictory outcomes were reported by studies undertaken in many countries such as Sri Lanka, Pakistan, Turkey, England, Germany, Iceland, Tanzania, Rwanda**,** Brazil, Chile" [234] and Italy [235].

Many of US studies that showed a positive association had involved black and Hispanic Americans from low socio-economic backgrounds whereas those

showed no association were UK based. This suggests that ethnic or environmental factors may play role in PLBW [236]. The Genetic and demographic factors, as well as different inclusion criteria for patients, could account for these different results [237].

2.3.4-Problems with Studies of PD&PLBW Relationship.

The literature is controversial on the role of periodontitis and its influence on PLBW. It was proved hard to make any clear conclusions from these studies because of the many different study designs, sampling methods, definitions of periodontal disease and adverse pregnancy outcomes, confounding factors, and possible effect modification by known or unknown factors [238,239,240].
There are several potential biases among the studies like:

2.3.4.1- Problem with periodontal disease definition.

The most important biases were the variation in periodontal disease definitions in periodontal research. Commonly accepted clinical measures of periodontal disease are clinical attachment level (CAL), the distance between the cemento-enamel junction and clinical pocket base) and probing depth (PD) the distance from the gingival margin to the apical part of the pocket, which were established 45 years ago. Although various indices have been developed since then, most have limited validity, and limited sensitivity for disease detection [241].

Because there is no universally accepted standard for periodontal disease diagnosis in periodontal research, most of the researchers used their own case definitions (mostly based on disease distribution within the study population) that combined PD and CAL. There wasn't same definition used in two or more studies, even by the same author(s) in different studies. Such variation in case definitions has been shown to have an impact on observed relationships between maternal periodontitis and pregnancy outcomes. Obviously, selecting different criteria to define periodontal disease will lead to different results [241,242].

2.3.4.2- Confounding Factors.

For those studies that reported an association, questions remain whether the observed associations represent a causal relationship or are due to the confounding effects of other variables [(241)].

All the studies testing the association between periodontal disease and adverse pregnancy outcomes were inconsistent in controlling confounders. Psychological stress, physical activity, gestational weight gain, violence, and social support the most important risk factors for adverse pregnancy outcomes also previous histories of adverse pregnancy outcomes, infections (e.g., Bacterial Vaginosis and chorioamnionitis), antibiotic use during pregnancies, or maternal disorders (hypertension, diabetes), were not considered.

Even though some of the studies adjusted for race, smoking, socioeconomic status and other important confounding variables, it is possible that some residual confounding effects remain. This is a major shortcoming and raises doubts as to the conclusions of all such studies [(240,241)].

2.3.4.3- Study sample size.

Insufficient sample size seems to be a concern for many of the studies had fewer than 100 patients [(17,161,162,212,224,243,244,245,246,247,248,249)]. Thus increasing the potential for associations observed by chance (random error) or lack of statistical power.

2.3.4.4- Problem with definition of PLBW.

Although the definition of PLBW seems homogeneous among studies, it has to be mentioned that the size of babies at birth vary considerably among populations [(48)]. The mean weight of infants born in India is about 2,900g, while in Sweden is 3,500g [(250)]. In the United States; the mean birth weight of the white infants is 3,446 g, and 3,089 g for the infants of the US-born black women [(251)]. In Benghazi it is about 3,200g [(252)]. This notion should be taken into consideration.

2.4- Community Perspective.

In 2004, the American Academy of Periodontology (AAP) issued a position statement regarding dental care for pregnant women. The AAP recommended that all women who were pregnant or planning a pregnancy should receive preventive dental care, including a periodontal examination, a prophylaxis, and restorative treatment [(253)]: proposed that scaling and root planning should be complete early in the second trimester and that any presence of acute infection or abscess should be treated immediately, irrespective of gestational age. Treating infection as early as possible will remove a potential source of infection that could be harmful to the mother and the baby.

In 2006, after a treatment trial failed to show an effect of scaling and root planning on birth outcomes, the AAP confirmed that treatment of periodontitis in pregnant women is safe and should be performed to improve the oral health of the woman [(228,254)]. This has been supported by The Academy of General Dentistry (AGD) whose recommendations are similar to the AAP but they suggest that pregnant women have a tiered treatment plane to include an examination in the first trimester, a dental cleaning in the second trimester, and then, depending on the patient, another appointment early in the third trimester. They also recommend communication between the dental provider and the obstetrician for any dental emergency that would require anaesthesia or other medication to be prescribed [(255)].

All health care providers should advise women that maintaining good oral health during pregnancy is not only safe but necessary to reduce the risk of infection to the mother and possibly the foetus. While it remains inconclusive whether maternal periodontal treatment improves pregnancy outcome, it is clear that treatment of varying degrees of clinical periodontal disease during pregnancy is safe and improves maternal oral health [(210,228,230)].

AIMS OF STUDY

3-AIMS OF THE STUDY

The study was carried out to:

1- Determine the relationship between periodontitis and premature low birth weight among Libyan ladies in Benghazi.

2- Estimate the prevalence and severity of periodontal disease in Libyan pregnant women sample.

SUBJECTS &METHOD

4-SUBJECTS & METHOD

4.1 -Sitting and Time.

The study took place at Al-Jomhuriya Hospital; The main and the biggest teaching hospital in the east region of Libya. From May 2010 to August 2010 at; Obstetrics and Gynaecology department, and laboratory department of Al-Jomhuriya Hospital, after a granted permission obtained. (Appendix I).

4.2 –Type of the study.

Cross-sectional descriptive study.

4.3 -Sample.

The study was conducted on a 300 pregnant ladies attended to reception department of labour, at gynaecology and obstetrics department of Al-Jomhuriya Hospital in Benghazi for delivery.

They were informed of the purpose and the design of the study before they accepted in the study. Furthermore, they were given full information about the nature of the procedure they were to receive. Consent Permission (Appendix II).

4.4 -Inclusion and Exclusion Criteria.

4.4.1-The inclusion criteria of the subject included:-

1. All women were Libyan
2. Non-smoker,
3. Non-alcoholic drinker
4. Their age $\geq$ 18 and $\leq$ 40 years
5. Their delivery had taken place at Al-Jomhuriya Hospital in labour department.

4.4.2-The exclusion criteria included:-

1. Participants with history of IVF (Intra vitro fertilization).
2. Planned caesarean delivery.
3. Participant women who presented cardiopathy, diabetes, or hypertension during their pregnancies.
4. Indication of prophylactic antibiotics for invasive procedures
5. Participants with multiple pregnancies.
6. Polyhydramnios, Malpresentations, cervical incompetence
7. Any obstetric complication such as antepartum haemorrhage.

4.5 -Data Collection.

4.5.1- Interview.

The women who volunteered to participate in the research were invited to answer questionnaire during an interview. Containing the following demographic sections; identification, socio-demographic data, obstetric history, gestational history, smoking, and general health condition.

All data were obtained through personal interview face to face and from maternal record if needed. All these information was documented using yes or no response. Structured questionnaire in (Appendix III).

Identification data

Like maternal name, age, residence and nationality recorded during interview. Participant who were not Libyan and their age <18 and >40 years were excluded from the study.

Socioeconomic history

The assessment of socio-economic status was characterized by:

1) Educational level (elementary, primary, secondary or higher).
2) Occupation of mother.
3) Father's occupation.
4) Other income to family if present.
5) Family numbers.
6) Family type; nuclear (live alone) or extended (live with parent).
7) Type of house; owner or renting.

Obstetric history

Assessed by history of; number of previous pregnancy, number of live birth, previous spontaneous miscarriage, previous preterm delivery, previous low birth weight, history of urinary tract infections or Bacterial Vaginosis during pregnancy, and prenatal care which assessed by number of prenatal visit (booked or unbooked).

All these informations were documented using yes or no response. Participants with history of IVF, or planned caesarean delivery or multiple pregnancies excluded from the study.

Gestational history

Ultrasound scanning used to establish an accurate gestational age, and to exclude; multiple pregnancies, polyhydramniose, malpresentation, intrauterine death and other possible obstetric cause of preterm labour.

Gestational age was based on the last menstrual period and early ultrasound dating before 20 week (collected from maternal record book). If the two agreed within 14 days, we used the former to assign gestational age. If the two dates different by more than 14 days or no menstrual dates were available, we used ultrasound date.

General health history

Actual previous general medical full history taken, participant women who presented cardiopathy or diabetes or hypertension during their pregnancies, were excluded.

All included women were non-alcoholic drinkers, non-smoker and history of passive smoking recorded using yes or no response.

4.5.2- Mothers weighing.

After the interview, all the participants' weight recorded in kilogram, as the weight is a risk factor of preterm delivery. The thinner the mother, the weaker she would be and thus less able to carry full term. It has been documented that women with a poor nutritional status are at greater risk for preterm birth [256,257].

4.5.3- Vaginal swaps.

After weighing the patients, High Vaginal swaps taken from each participant before the delivery to avoid contamination, all swaps sent to Microbiology Department in Al-Jomhuriya Hospital Lab.

In Microbiology Department, culture of the swaps done on; Blood, Mac Conkey and Chocolate Petri dish media. After incubation for 24 hours in the incubator we note either there is growth or not (negative or positive culture). If there is growth in culture, Gram stain done for identification the type of the microorganism growth; it is either from normal vaginal flora or pathogenic microorganism growth.

4.5.4- Periodontal Status Measurements.

Following the interview, the mother weighting is done and vaginal swaps were taken. All participants underwent a clinical periodontal examination. Periodontal status was determined by using Plaque index (**PI)** – Silness & Loe [258], Gingival index (**GI)** - Loe & Silness [259] and periodontal disease index Ramfjord (**PDI**)[260].

The periodontal examination took place in reception ward with the subject supine on the hospital bed to facilitate a producible examination position. And with external portable light source. Sterile disposable dental kit contains; mirrors, explorer, gauze, cotton roll, gloves and mask were used for each patient to assess plaque accumulation and gingival status. While Michigan periodontal probes with Williams' markings rounded down to whole millimetre at 3 mm, 6 mm and 8 mm used to measure clinical attachment loss. The level of clinical attachment loss was calculated from cemento- enamel junction to the base of the periodontal pocket.

Radiographs not taken for this study for the patient safety. As such the radiographic alveolar bone level was not assessed, because the actual degree of periodontitis is better shown by probing depth[(261)]. In relation to the association of periodontal infection with periodontal pocket, the size of the surface area of the pocket, through which bacterial products can invade the periodontal tissue, was found to be more important than bone levels[(262)].

4.5.4.1- Plaque Index Measurement (PI).

Recorded the amount of plaque by using explorer on a 0-3 scales for Six selected teeth

6 2	4
4	2 6

At four surfaces per tooth (mid-mesial, mid-facial, mid-distal, and mid-lingual), according to plaque index of Silness & Loe criteria (Appendix IV). After the scores for the four surfaces recorded, added together and divided by four to give the plaque index (PI) for only one tooth, these were in turn summed and divided by the number of teeth examined (6) to express the (PI) for individual.

4.5.4.2- Gingival Index Measurement (GI).

Recorded on a 0-3 scales for six selected teeth

6 2	4
4	2 6

According to Gingival index (**GI)** – Loe & Silness criteria (Appendix V). The scores for the four surfaces recorded (mid-mesial, mid-facial, mid-distal, and mid-lingual) added together then divided by four to give the gingival index (GI) for the only one tooth, these were in turn summed and divided by the number of teeth examined to express the (GI) for individual.

4.5.4.3- Periodontal Disease Index (PDI).

Examination done by using the Michigan probe which has markings at 3 mm, 6 mm and 8 mm. on six specific teeth

6	1 4
4 1	6

And four surfaces (mid-mesial, mid-facial, mid-distal, and mid-lingual) for each tooth were each tooth is scored on scale from 0 to 6 according to criteria of periodontal disease index of Ramfjord (Appendix VI). The scores from the four surfaces recorded (mid-mesial, mid-facial, mid-distal, and mid-lingual) added together then divided by four to give the PDI for only one tooth, these were in turn summed and divided by the number of teeth examined to express the PDI for individual.

At the end of the periodontal examination, each participant was given oral hygiene instructions, and instruction regarding dental treatment needs by educating participants; Educational component will focus on the importance of oral health and the impact of oral disease on perinatal health. Following the educational component, referrals for an appropriate dentists were provided for participants to begin improving their oral health. On the day of the delivery newborns weight were collected from the labour register book in labour department.

RESULTS

5-RESULTS

Results were expressed as mean ± standard deviation (SD) or number and percentage. Statistical analysis was performed with the aid of the statistical package for the social sciences (SPSS) computer program (version 18 windows). t- test, x^2 (chi-square test) and Pearson correlation were used when needed, P-value considered significant when $P \leq 0.05$.

5.1- Characteristic of study population.

1- Age.

The highest percentage of the sample is between 26-30 years old, 27.6 percent of the sample is between 31-35 years old as well as 27.7 percent of the sample is between 21-25 years old. That mean the highest percent of the sample is less than 30 years old. Mean= 28.3 years. Std. Deviation =4.78 years. Median=28 years. Mode= 30. Minimum=18 years. Maximum =39 years. As shown in table (5.1) and figure (5.1).

2- Residence.

Table (5.2) and figure (5.2) show that 71.7 percent of the sample is from Benghazi and 28.3 percent of the sample is resident outside Benghazi.

3- Prenatal visit of the subjects.

Table (5.3) and figure (5.3) show that 99 percents of the subjects visited their doctor during prenatal care more than 6 times (booked) and only 1 percent not booked.

Table 5. 1:Categorization of subjects according to the age.

Age group/ Years	No.	%
≤ 20	18	6
21 – 25	74	24.7
26– 30	107	35.7
31 - 35	83	27.6
36 - 40	18	6
Total	300	100

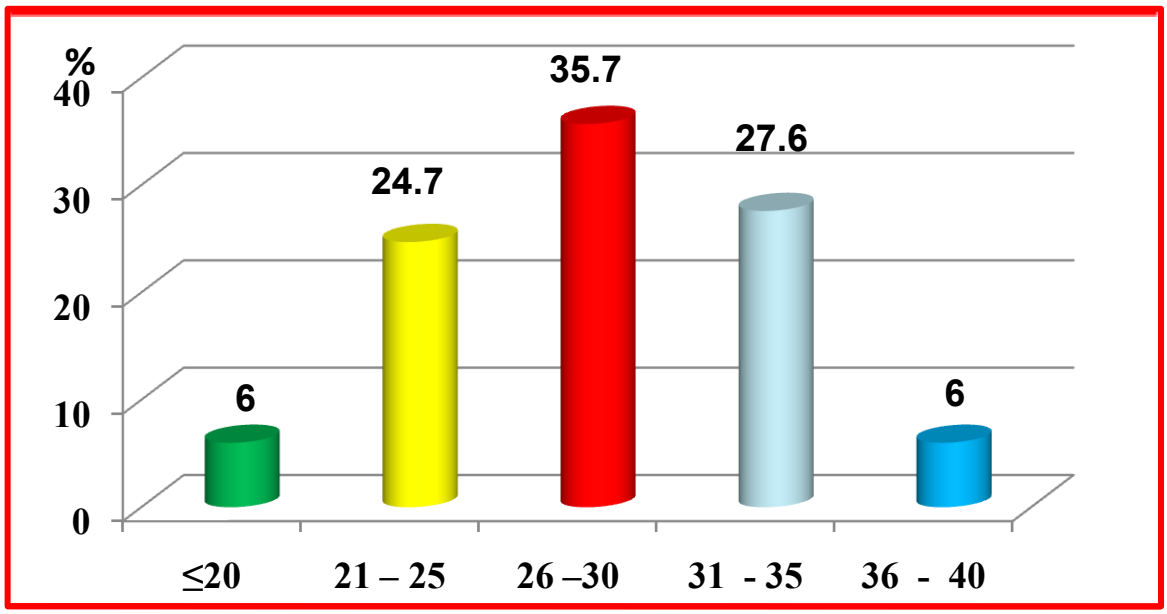

Figure 5.1: Categorization of subjects according to the age.

Table 5. 2: Categorization of subjects according to the residence.

Residence.	No.	%
Benghazi	215	71.7
Outside Benghazi	85	28.3
Total	300	100

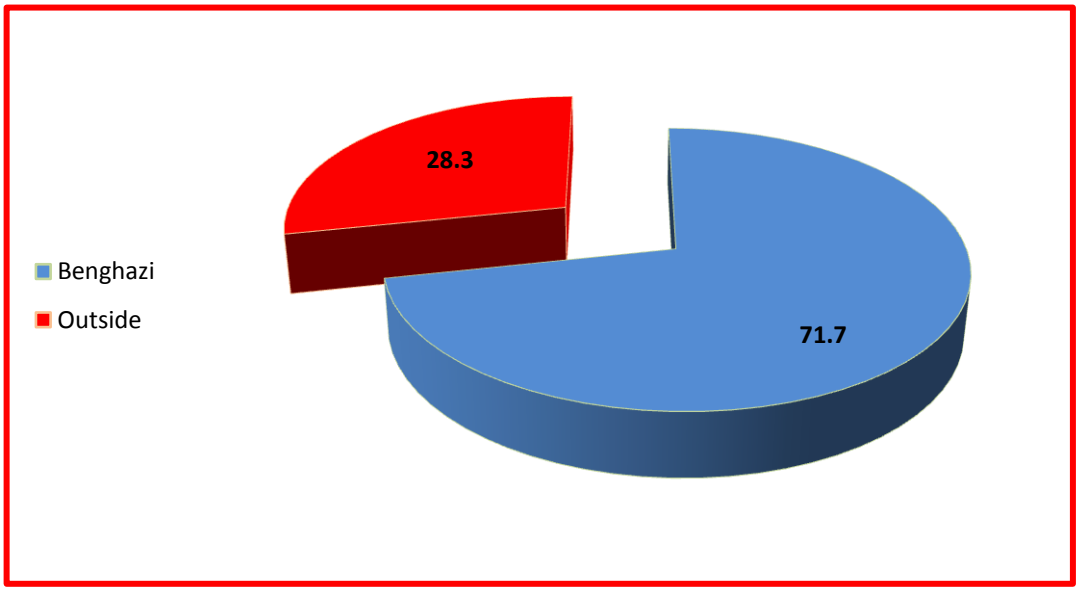

Figure 5.2: Categorization of subjects according to the residence.

Table 5. 3: Categorization of subjects according to the prenatal visit.

Booking	No.	%
Booked	297	99
Unbooked	3	1
Total	300	100

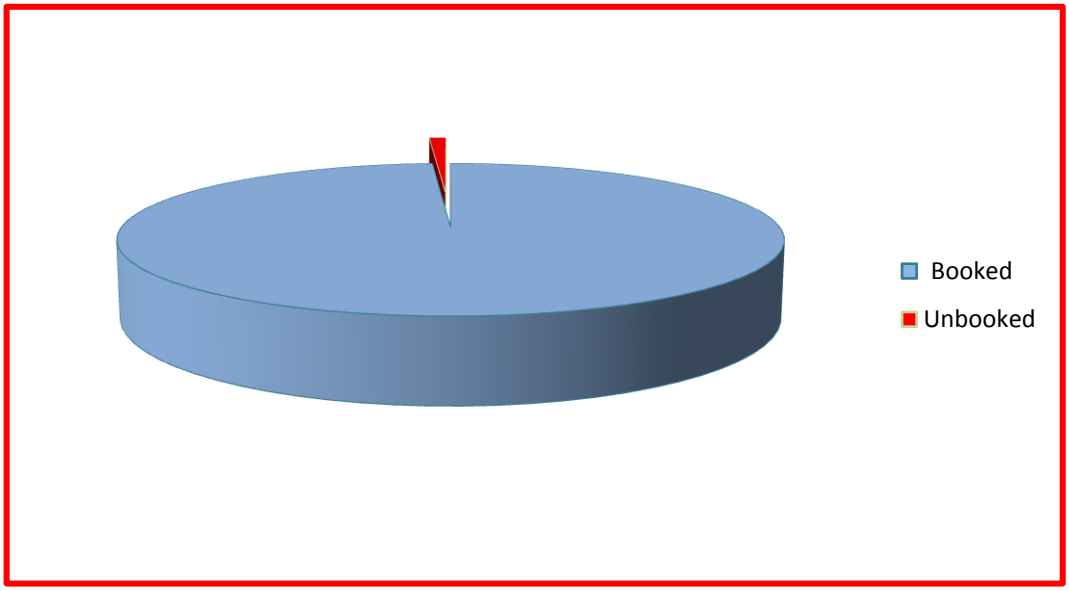

Figure 5.3: Categorization of subjects according to the prenatal visit.

4- High vaginal swap.

Ninety four percent of the sample had negative vaginal swap result. That means 281 cases was negative for local infection as shown in table (5.4) and figure (5.4).

5- Culture result.

Table (5.5) and figure (5.5) show that negative culture with percentage of 40, 53.7 percent with normal flora and 6.3 percents with pathological culture distributed as 3.7 percent staph aurous and 2.3 percent streptococcus while 0.3 percents was Candida.

6- Gravidity.

Eighty four point seven percents had 1-4 pregnancies and 15.3 percent had 5 or more pregnancies. As shown in table (5.6) and figure (5.6) mean=2.6, Std. Deviation = 1.7. Median=2. Mode = 1. Minimum= 1. Maximum = 9.

7- Parity.

Table (5.7) and figure (5.7) show that 44.3 percent of sample was nulipara and 54.75 percents had from 1 to 5 babies while 1 percent had more than 5 babies. Mean=1.2. Std. Deviation = 1.4 .Median=1. Mode= 0. Minimum= 0. Maximum = 7.

Table 5. 4: Categorization of subjects according to the high vaginal swap.

High vaginal swap result	No.	%
Positive	19	6
Negative	281	94
Total	300	100

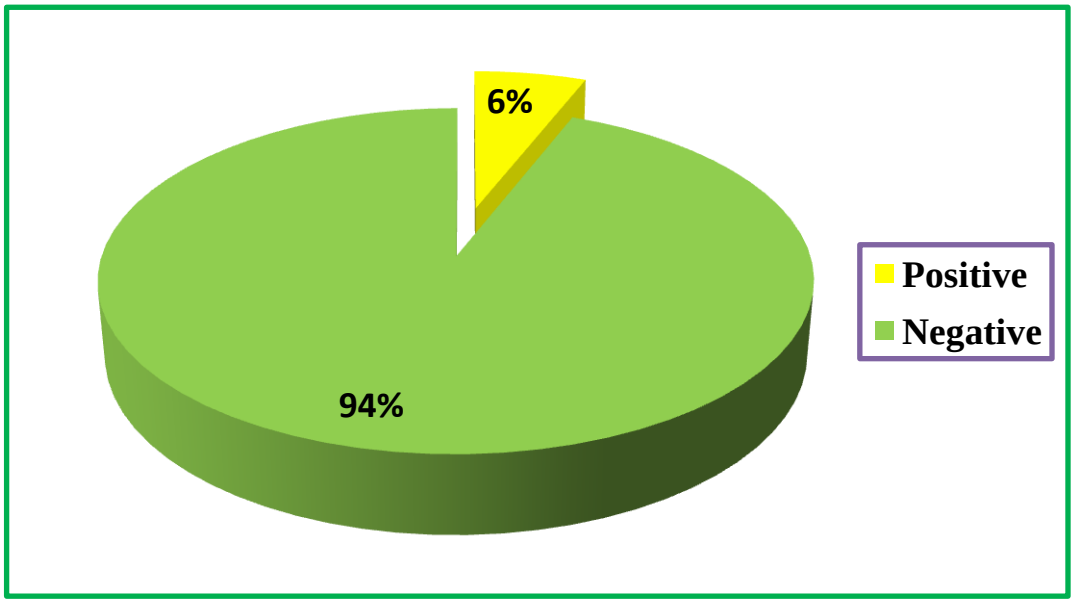

Figure 5.4: Categorization of subjects according to the high vaginal swap.

Table 5. 5: Categorization of subjects according to the culture result.

Culture result of high vaginal swap result	No.	%
Negative	120	40
Normal flora	161	53.7
Staph aurous	11	3.7
Streptococcus	7	2.3
Candida	1	0.3
Total	300	100

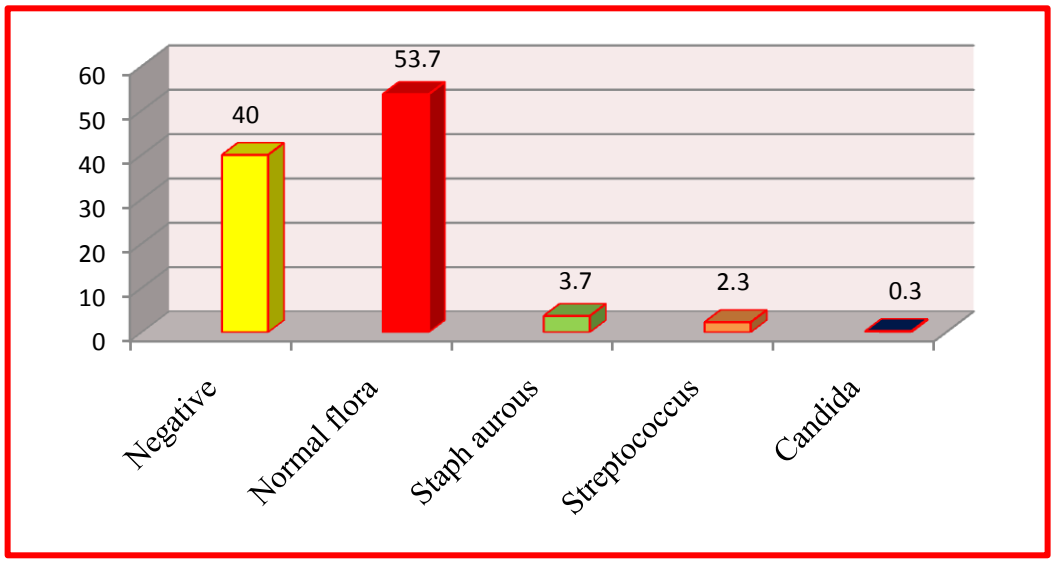

Figure 5.5: Categorization of subjects according to the culture result.

Table 5. 6: Categorization of subjects according to gravidity.

Number of pregnancy	No.	%
1 - 4	254	84.7
≥ 5	46	15.3
Total	300	100

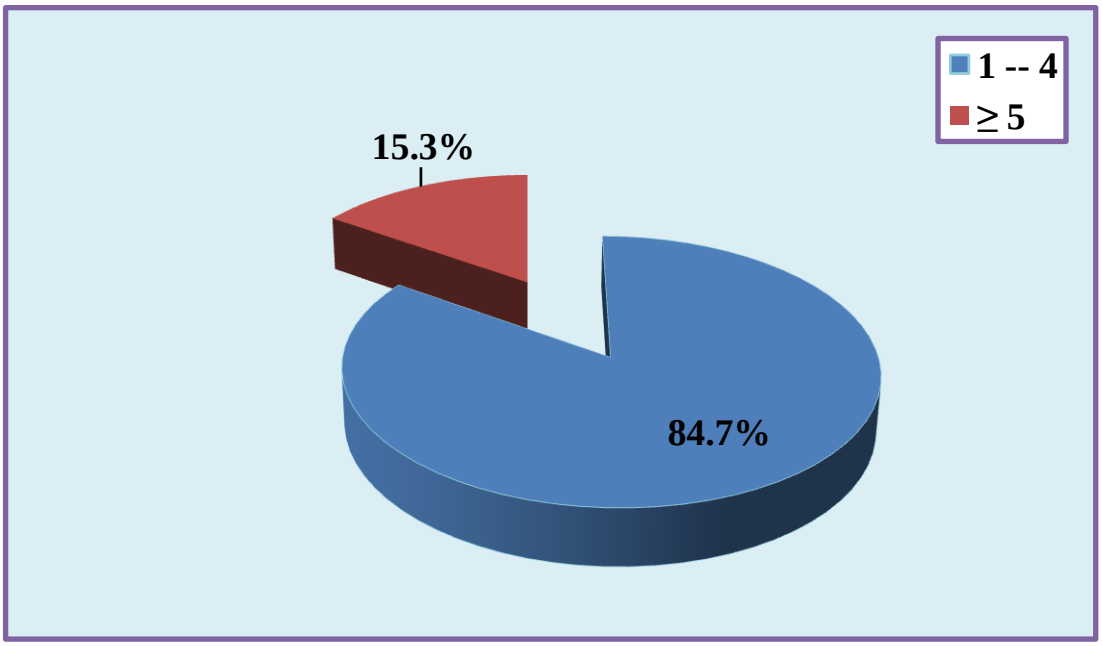

Figure 5.6: Categorization of subjects according to gravidity.

Table 5. 7: Categorization of subjects according to parity.

parity	No.	%
Primi	133	44.3
1-5	164	54.7
>5	3	1
Total	300	100

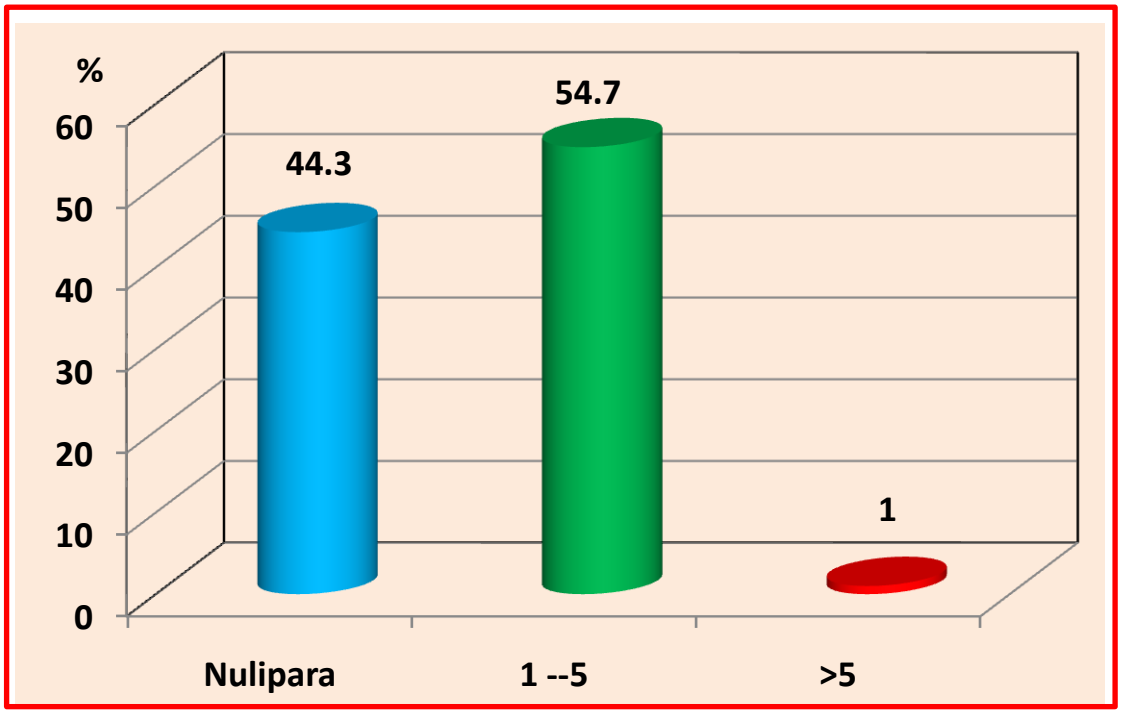

Figure 5.7: Categorization of subjects according to parity.

8- History of abortion.

A percentage of 74 of the sample had no history of abortion and 26 percents had history of abortion as show in table (5.8) and figure (5.8).

9- History of passive smoking.

Fifty one percent of the sample had history of passive smoking (second hand smoking) from their husbands. As shown in figure (5.9) and table (5.9).

10- History of LBW.

Table (5.10) and figure (5.10) show that 89 percent of the sample had no history of low birth weight (LBW). While 11 percent had history of LBW.

11- History of preterm.

Table (5.11) and figure (5.11) show that 93.7 percent of the sample had no history of pre-term delivery.

12- Medical condition.

Table (5.12) and figure (5.12) show that 88 percent of the sample had no medical finding, while the rest of the participant who had a positive medical history mostly for ″Anaemia, Asthma and UTI″ as shown in figure (5.13) table (5.13).

Table 5. 8: Categorization of subjects according to the history of abortion.

History of abortion	No.	%
No	222	74
Yes	78	26
Total	300	100

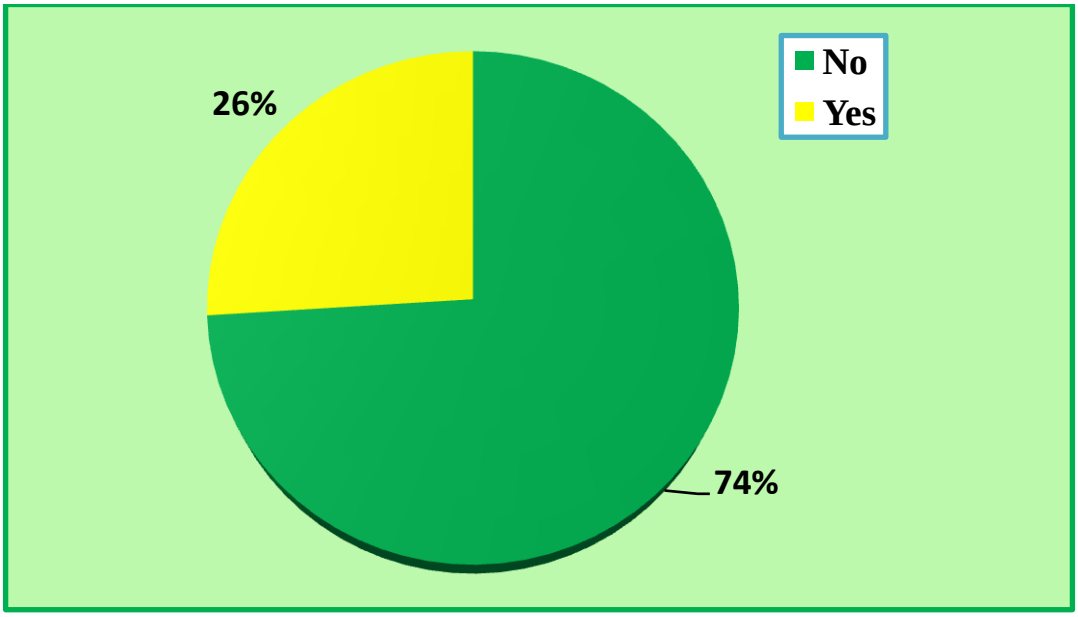

Figure 5.8 Categorization of subjects according to the history of abortion.

Table 5. 9: Categorization of subjects according to the history of passive smoking.

Passive smoking	No.	%
No	147	49
Yes	153	51
Total	300	100

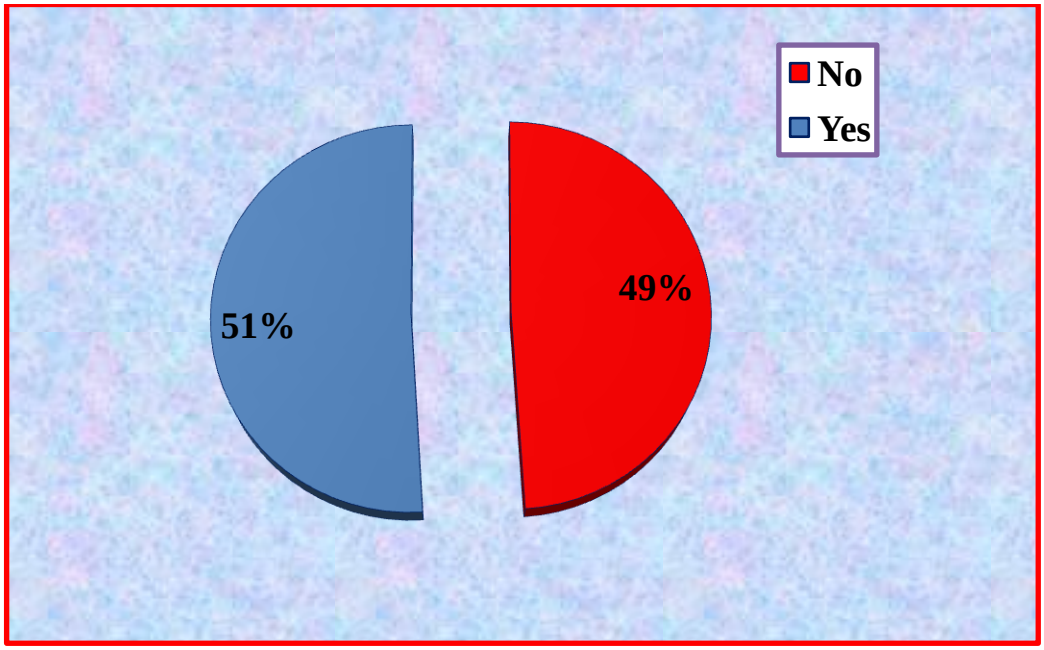

Figure 5.9: Categorization of subjects according to the history of passive smoking.

Table 5. 10: Categorization of subjects according to the history of LBW.

History of low birth weight	No.	%
Yes	33	11
No	267	89
Total	300	100

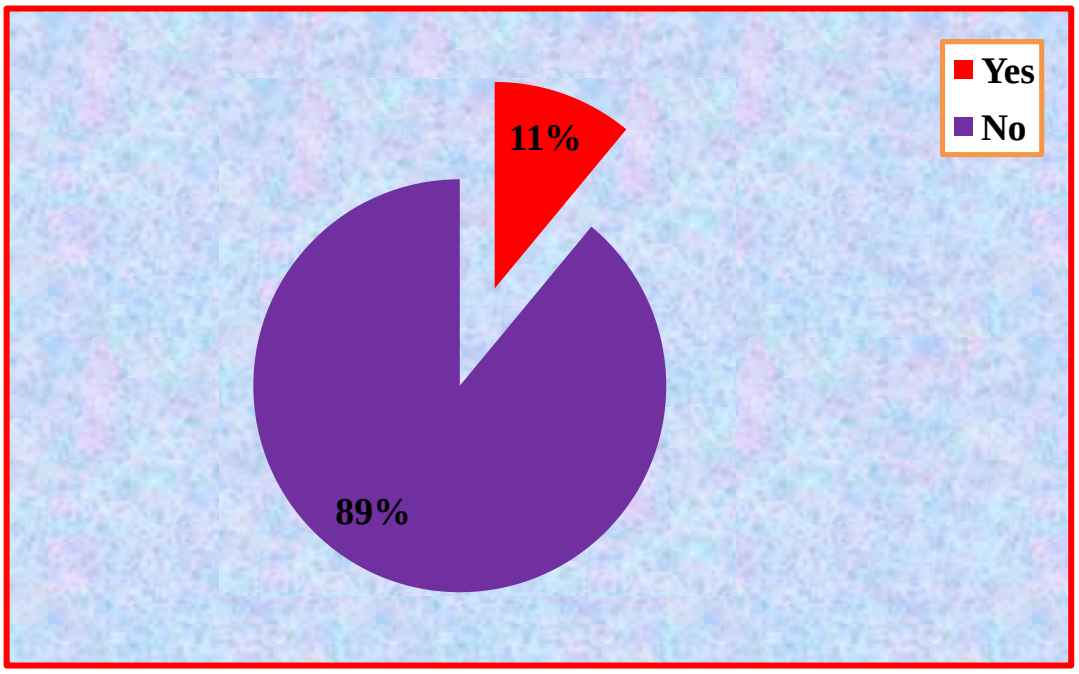

Figure 5.10: Categorization of subjects according to the history of LBW.

Table 5. 11: Categorization of subjects according to the history of preterm.

History of pre term delivery	No.	%
Yes	19	6.3
No	281	93.7
Total	300	100

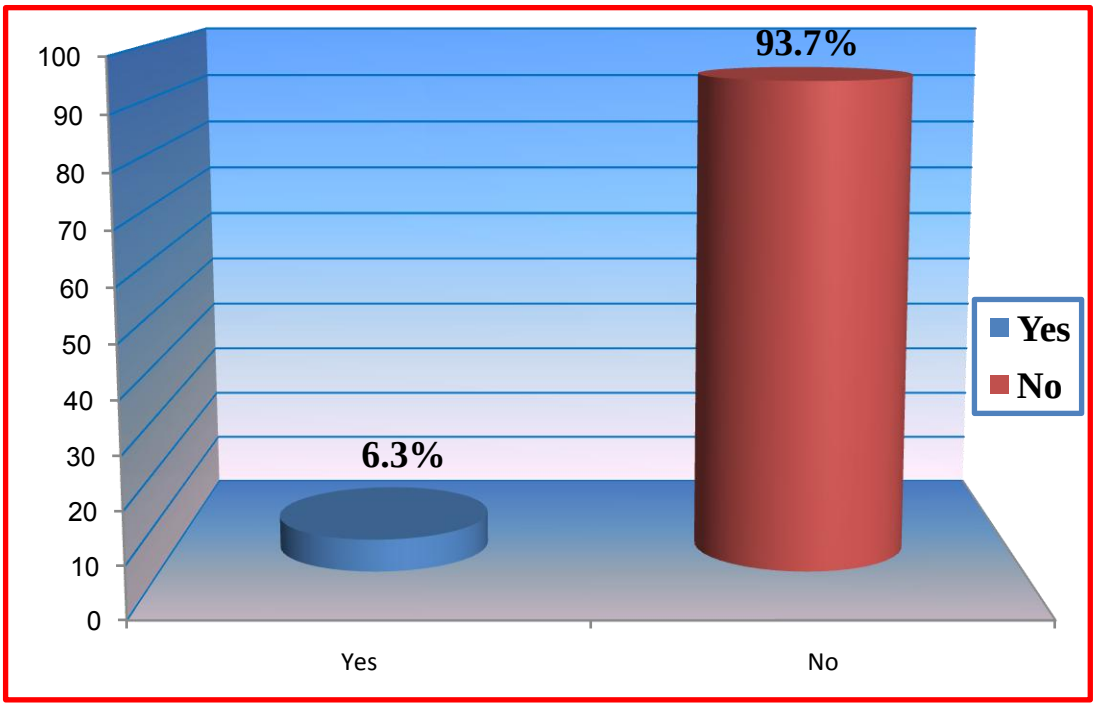

Figure 5.11: Categorization of subjects according to the history of preterm.

Table 5. 12: Classification of subjects according to the medical condition.

Medical history	No.	%
No	264	88
Yes	36	12
Total	300	100

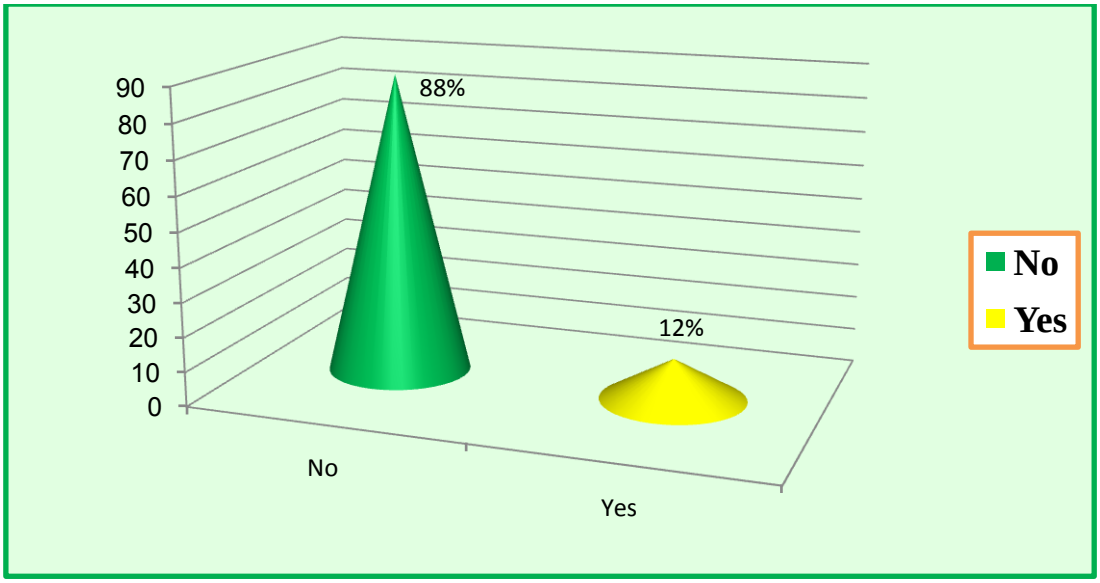

Figure 5.12: Classification of subjects according to the medical condition.

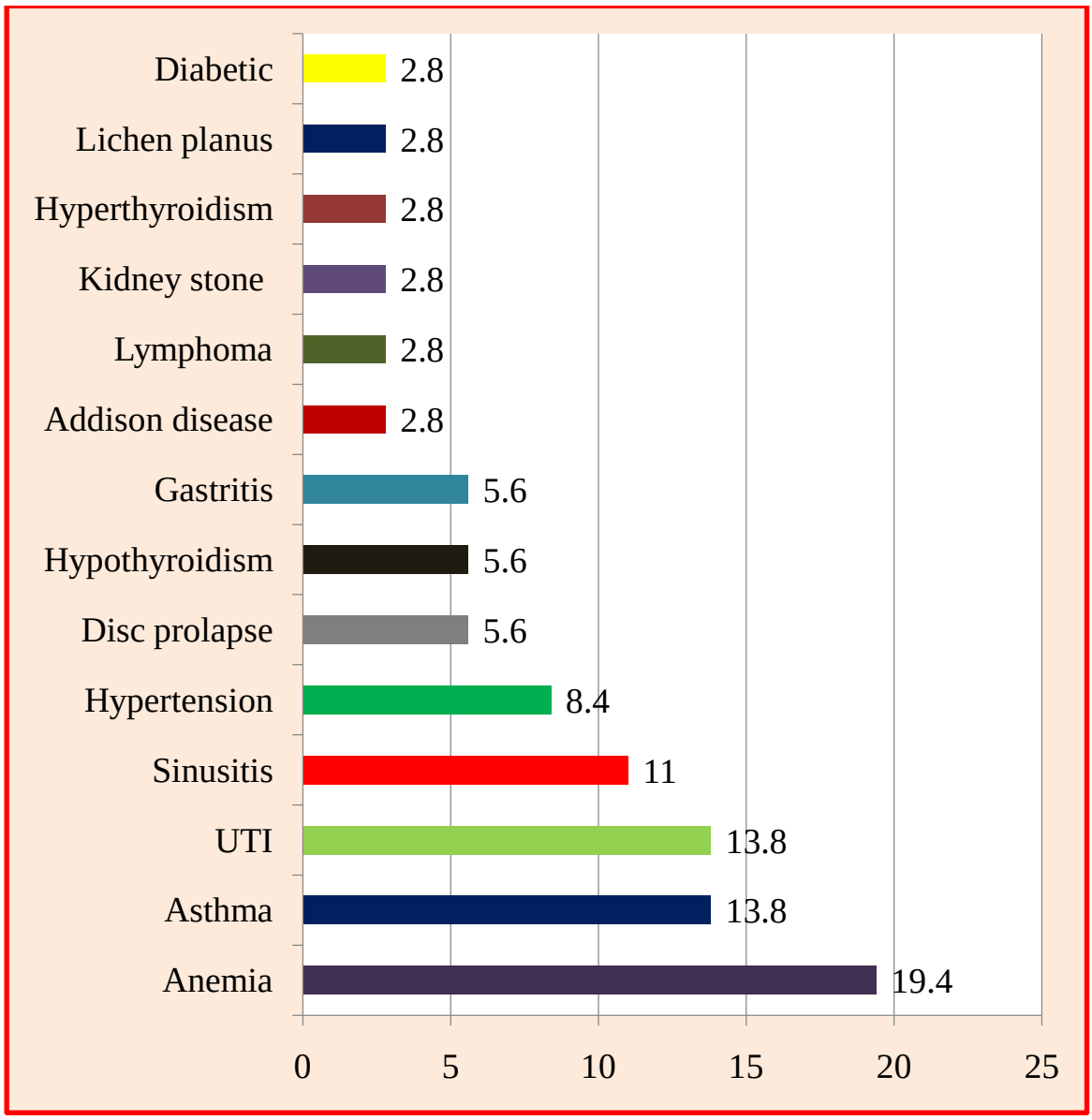

Figure 5.13: Classification of subjects according to the type of medical condition.

Table 5. 13: Classification of subjects according to the type of medical condition.

Type of medical history	No.	%
Anemia	7	19.4
Asthma	5	13.8
UTI	5	13.8
Sinusitis	4	11
Hypertension	3	8.4`
Disc prolapse	2	5.6
Hypothyroidism	2	5.6
Gastritis	2	5.6
Addison disease	1	2.8
Lymphoma	1	2.8
Kidney stone	1	2.8
Hyperthyroidism	1	2.8
Lichen planus	1	2.8
Diabetic mellitus	1	2.8
Total	36	100

14- Housing /accommodations

As illustrated in Table (5.14) and figure (5.14) 90 percent of the family had owned their house and only 9.3 percent are renting.

15- Type of family.

To assess socioeconomic status the participant was divided to:
Nuclear* It mean that the patients living alone separated from their parent.
Extended** those participant who lives with their parent at the same house. Table (5.15) and figure (5.15) showed 80.7 percent of the family were nuclear and 19.3 percent were extended.

16- Level of education.

The study sample presented 5.3 percent didn't receive education, 2 percent went to primary School, 22 percent to preparatory school and a percentage of 31 went to secondary school while 39 percent with higher education level as shown in table (5.16) and figure (5.16).

17- Type of subject occupation.

As shown in table (5.17) and figure (5.17) the sample showed different type of occupation; teacher 13.6 percent, civil servants 7.7 percent, nurse 1.7 percent and technician 0.7 percent, doctor 0.3 percent, 71 percent house wife and the rest a percentage of 5 are students.

Table 5. 14: Classification of subjects according to their housing /accommodations.

HOUSE OWNERSHIP	No.	%
Yes	272	90.7
No(renting)	28	9.3
Total	300	100

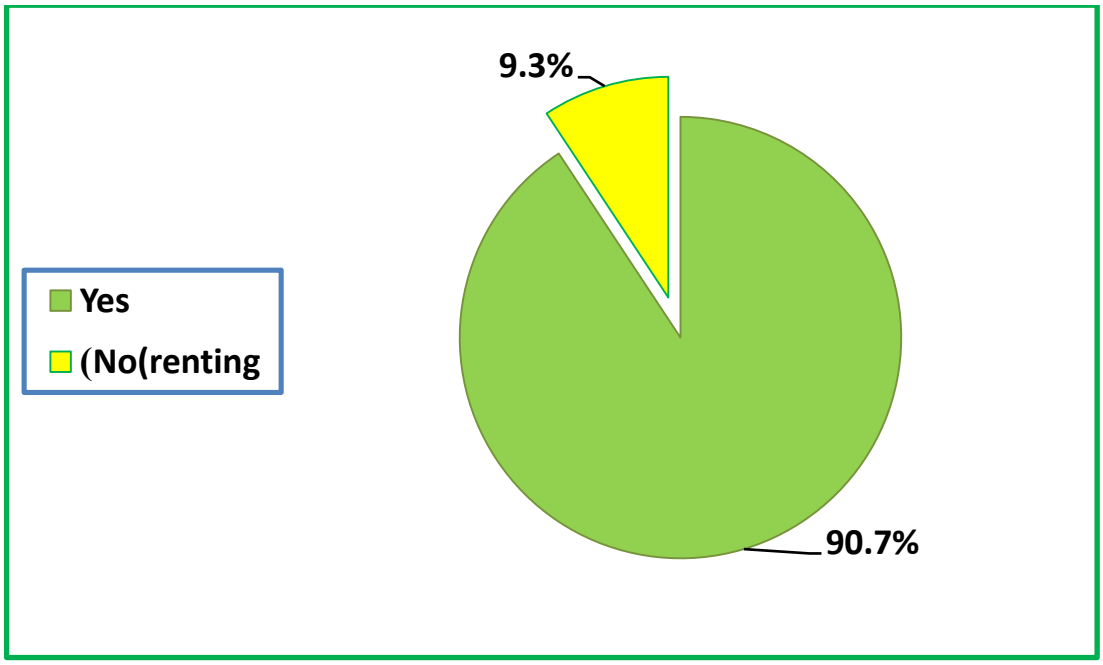

Figure 5.14: Classification of subjects according to their housing /accommodations

Table 5. 15: Classification of subjects according to the type of family.

Type of the family	No.	%
Nuclear*	242	80.7
Extended**	58	19.3
Total	300	100

Nuclear* patients live alone separated from their parent.

Extended** patients live with their parents at same house

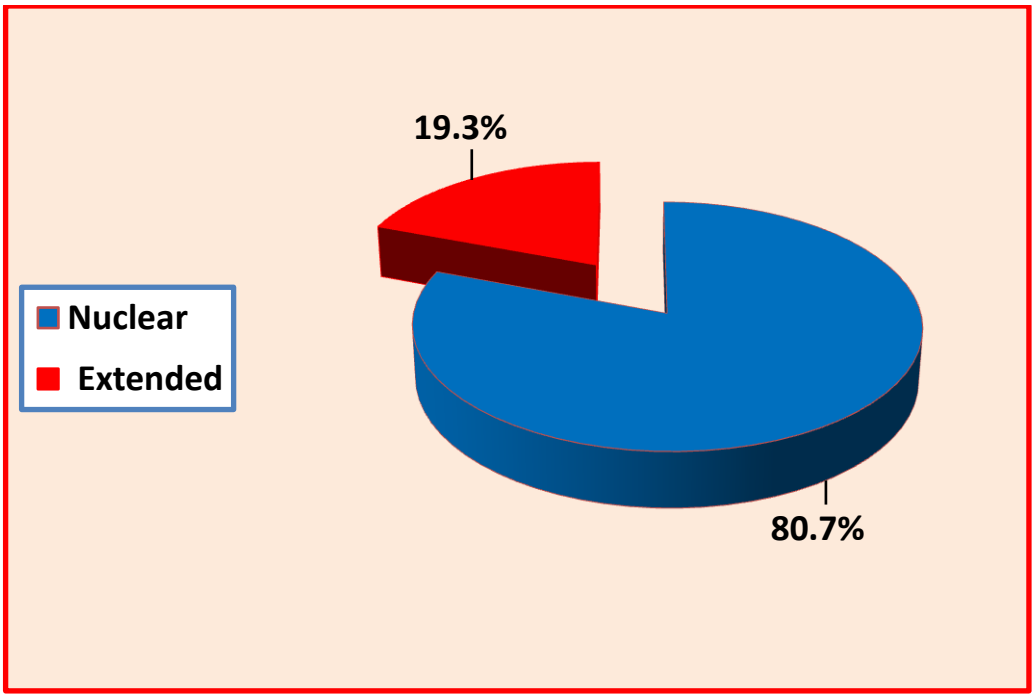

Figure 5.15: Classification of subjects according to the type of family.

Table 5. 16: Classification of subjects according to the level of their education.

Level of education of the patients	No.	%
Illiterate	16	5.3
Primary	6	2
Preparatory	68	22.7
Secondary	93	31
University	117	39
Total	300	100

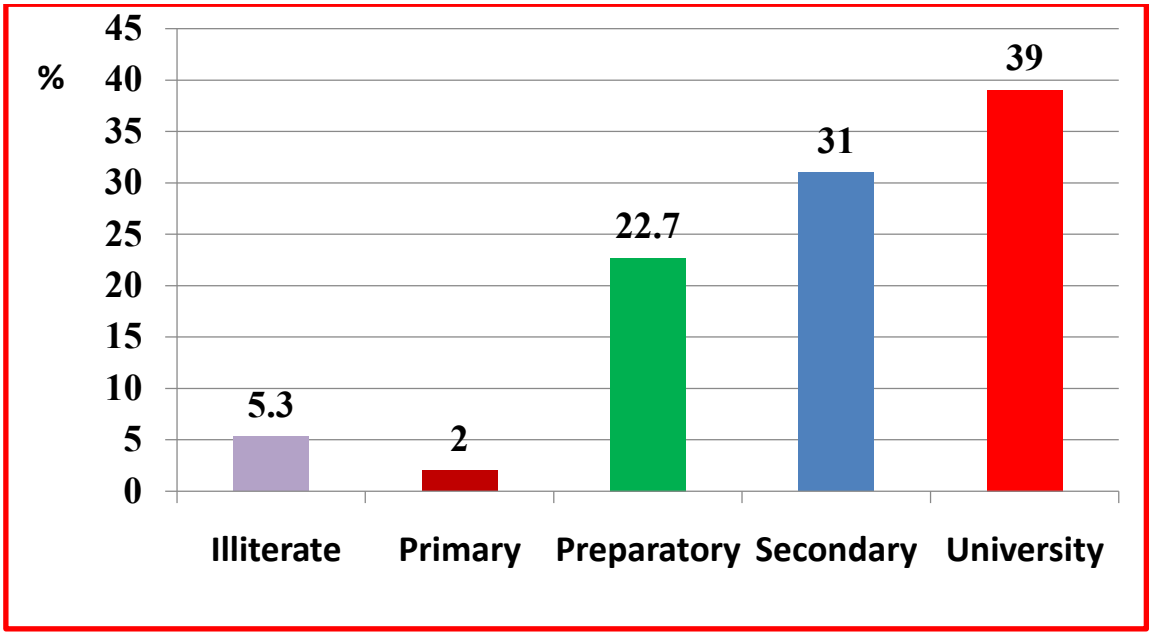

Figure 5.16: Classification of subjects according to the level of their education.

Table 5. 17: Classification of subjects according to the type of occupation.

Type of occupation	No.	%
House wife	213	71
Teacher	41	13.6
Civil servant	23	7.7
Student	15	5
Nurse	5	1.7
Technician	2	0.7
Doctor	1	0.3
Total	300	100

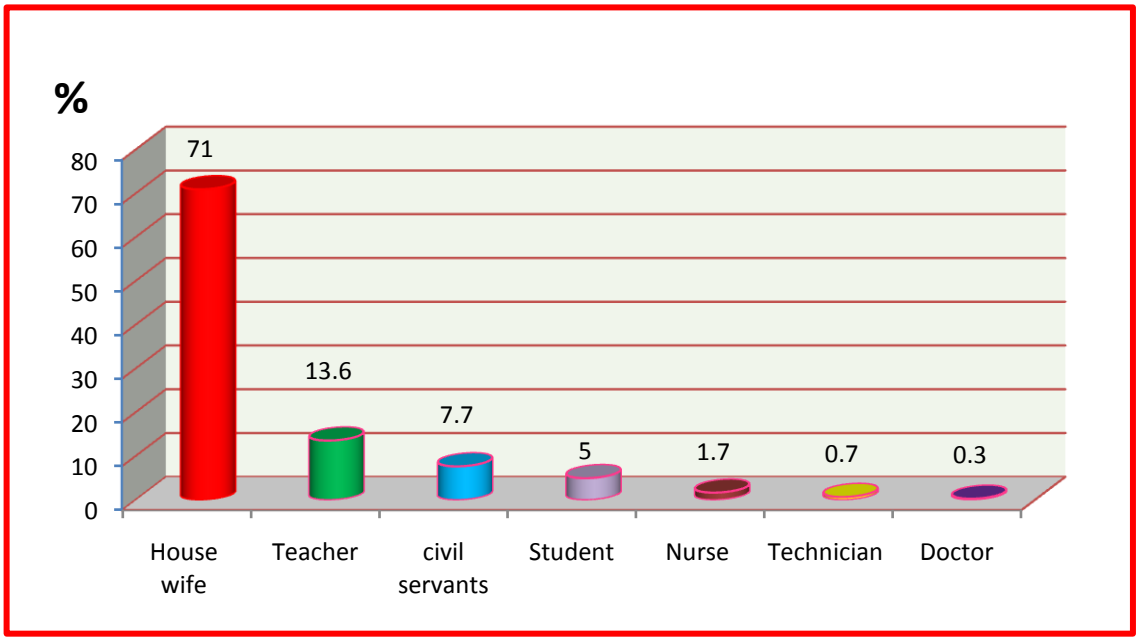

Figure 5.17: Classification of subjects according to the type of occupation.

18- Number of the family members.

The sample showed that 78.4 percent of the patients had 5 or less family member, 16.3 percent had from 6 to 10 members and 5.3 percent had more than 10 family member as shown in table (5.18) and figure (5.18). The mean was 4.313 member, Std. Deviation = 3.279 member. Median =3 member. Mode = 2 member. With minimum of 2 family member and maximum= 32 member.

19- Husband's occupation.

The study sample of husband's occupation showed a percentage of 42.7 self employed, 39.3 percent are civil servants, 16 percent are working at the army and 2 percent are technician. As shown in table (5.19) and figure (5.19).

20- Birth weight.

As shown in table (5.20) and figure (5.20), there were 12 subjects didn't deliver at AL Jomhuriya Hospital so these are missed from the sample. In addition to the missed subjects, there were 13 birth weights which are not sure about their birth weight due to technical problems (12+13) so 25 birth weight drops from the sample as showing in table (5.21) and figure (5.21). Mean ± SD (3.1± 0.66) kg. Median=3.2kg. Mode= 3kg, minimum= 0.700gm and maximum = 4.800kg

Table 5. 18: Classification of subjects according to the number of the family members.

Number of the family members	No.	%
≤5	235	78.4
6 - 10	49	16.3
>10	16	5.3
Total	300	100

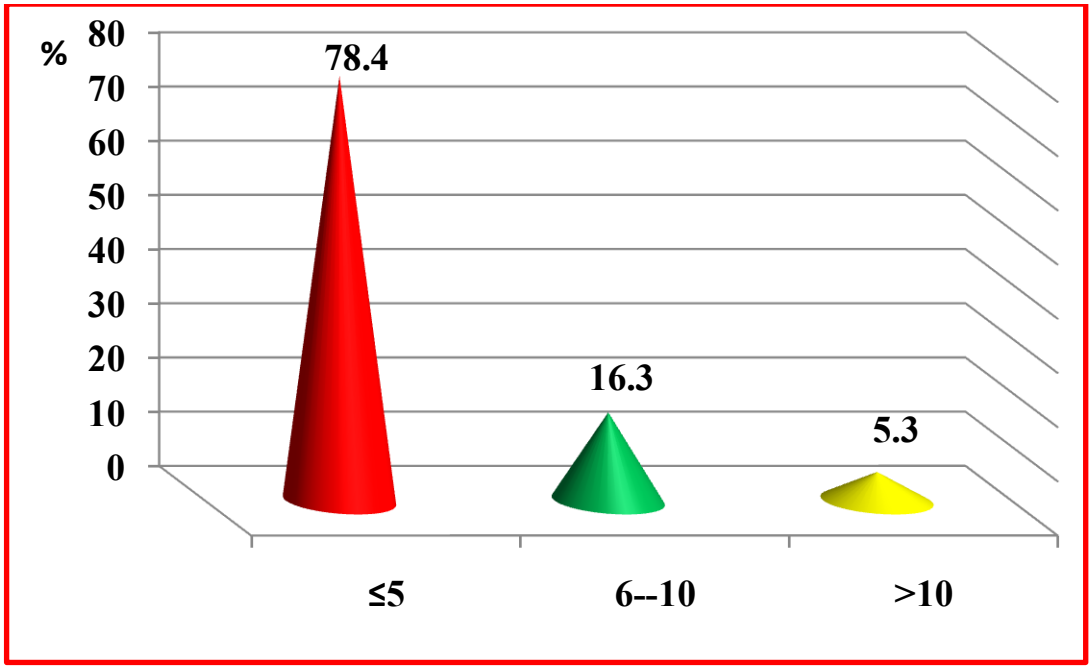

Figure 5.18: Classification of subjects according to the number of the family members.

Table 5. 19: Classification of subjects according to the husband's occupation.

Husbands occupation	No.	%
Self employed	128	42.7
Civil Servant	118	39.3
Army	48	16
Technician	6	2
Total	300	100

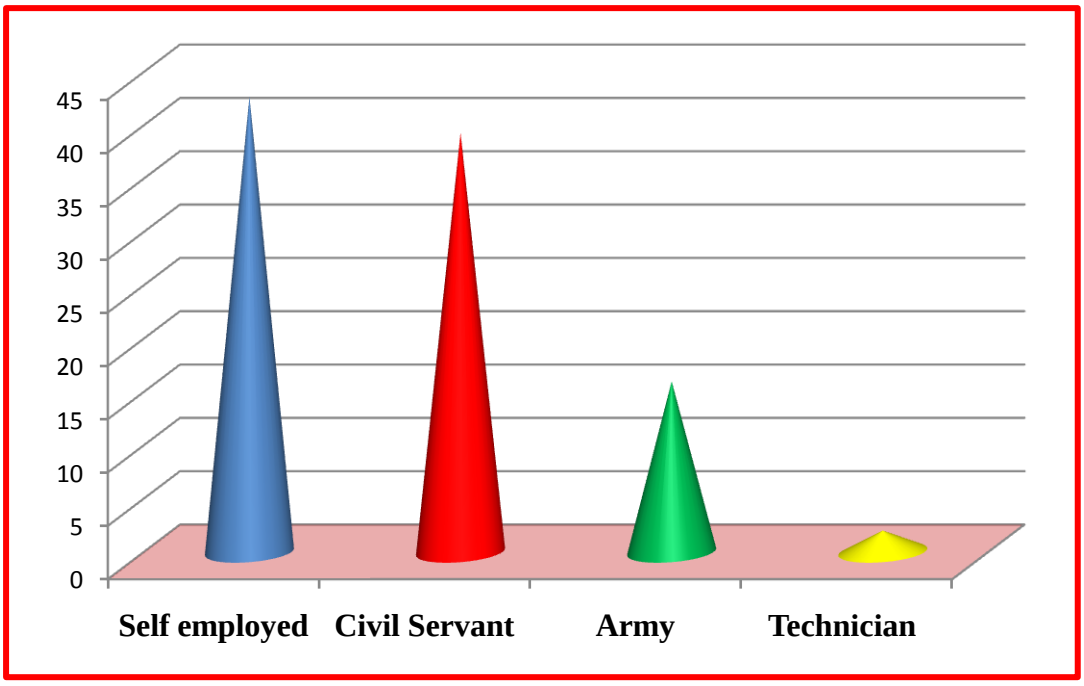

Figure 5.19: Classification of subjects according to the husband's occupation.

Table 5. 20: Classification of subjects according to the birth weight.

Birth weight of/ kg	No.	%
<2.500	32	10.7
≥ 2.500	243	81
Missing*	25	8.3
Total	300	100

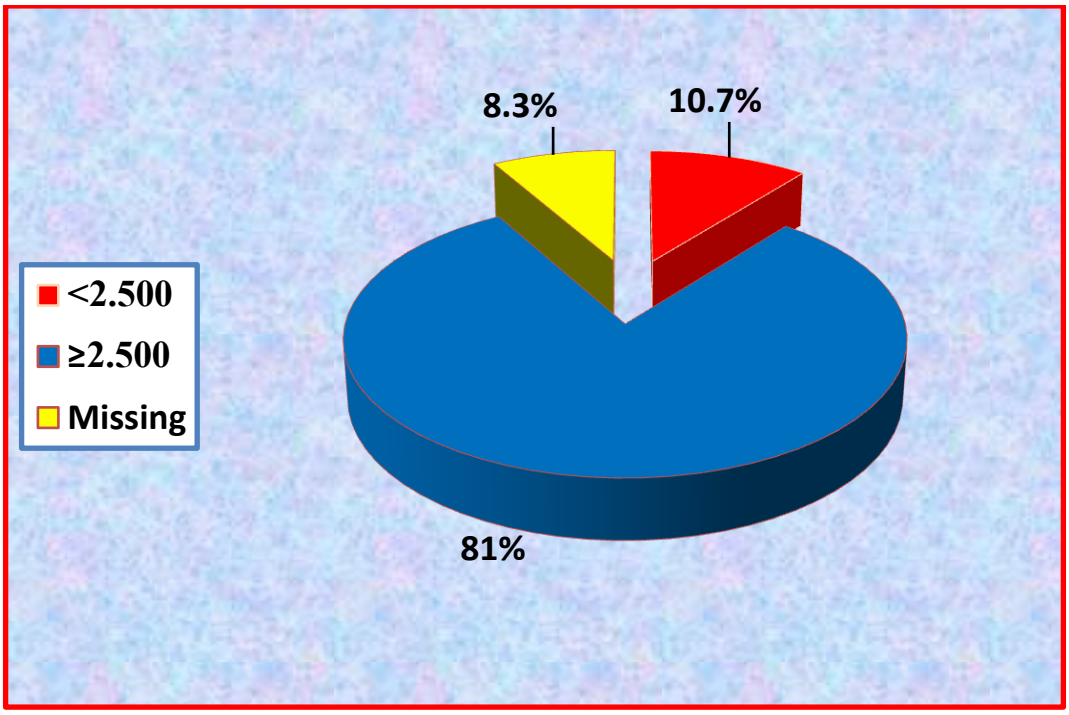

Figure 5.20: Classification of subjects according to the birth weight.

21- Gestational age

About 12 cases are considered missing as they didn't deliver at Al Jomhuriya Hospital (after answering the questionnaire, had a vaginal swap and went a periodontal examination). So the 288 participants showed; a percentage of 82 for full term pregnancy and 14 percent for preterm as shown in table (5.21) and figure (5.21). The mean = 39 .6 weeks. Std. deviation =1.6 weeks. Median =40 weeks. Mode =40 weeks. Minimum=32 weeks and the maximum= 43 weeks.

22-According to the birth weight and gestational age.

Table (5.22) and figure (5.22) show that, from a total 40 preterm cases; 27 participants had low birth weight (PLBW), while 60 participants from a total 235 full term cases had a low birth weight (FLBW), 13 cases dropout from the sample as shown in table (5.21) and figure (5.21); 2 cases from the preterm group and 11cases from the full term group.

Basic Characteristic Of The Study Population.

The basic characteristic of the study population like mother age and weight, parity, gravidity and birth weight had significant relationship with gestational age. Whereas other characteristics of the study population like; abortion, residence, prenatal visit, past low birth weight and past preterm delivery, passive smoking and socio economic status are not significant with gestational age, as show in table (5.23).

Table 5. 21: Classification of subjects according to the gestational age.

Gestational age	No.	%
Full term	246	82
Pre term	42	14
Missing	12	4
Total	300	100

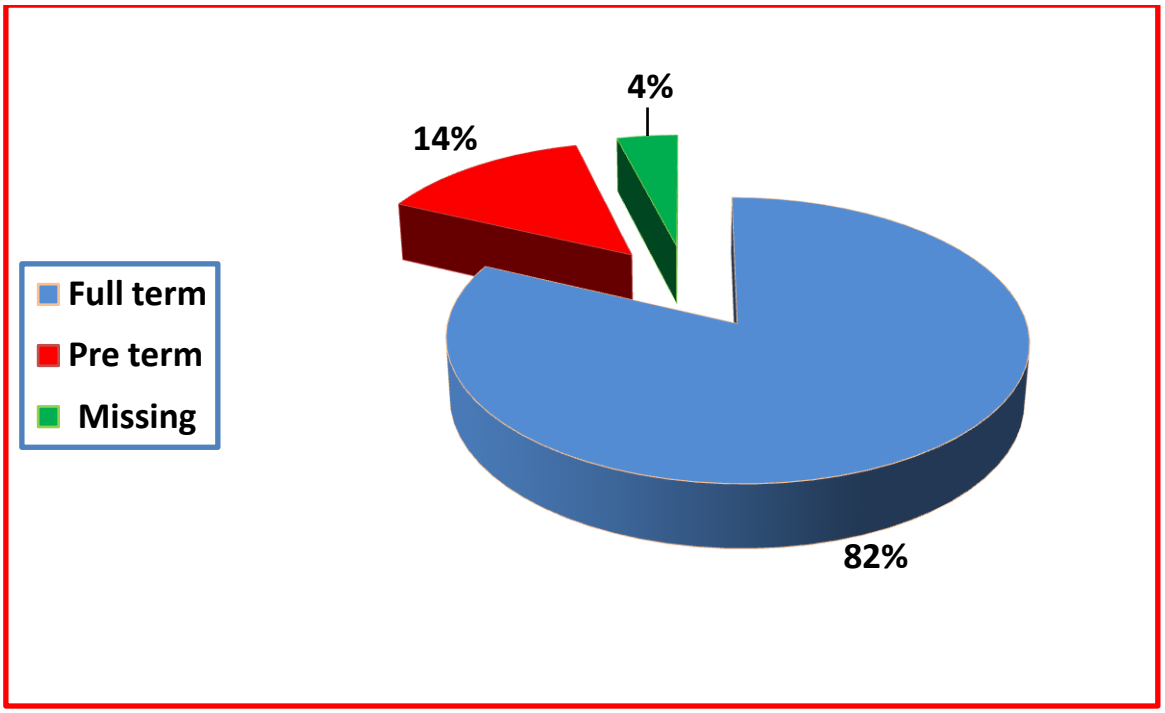

Figure 5.21: Classification of subjects according to the gestational age.

Table 5. 22: Classification of subjects according to birth weight and gestational age.

Gestational Age	<2.500	≥ 2.500	Total
preterm	27 (67.5%)	13 (32.5%)	40
Full term	60 (25.5%)	175 (74.5%)	235
Total	87 (31.6%)	188 (68.4%)	275

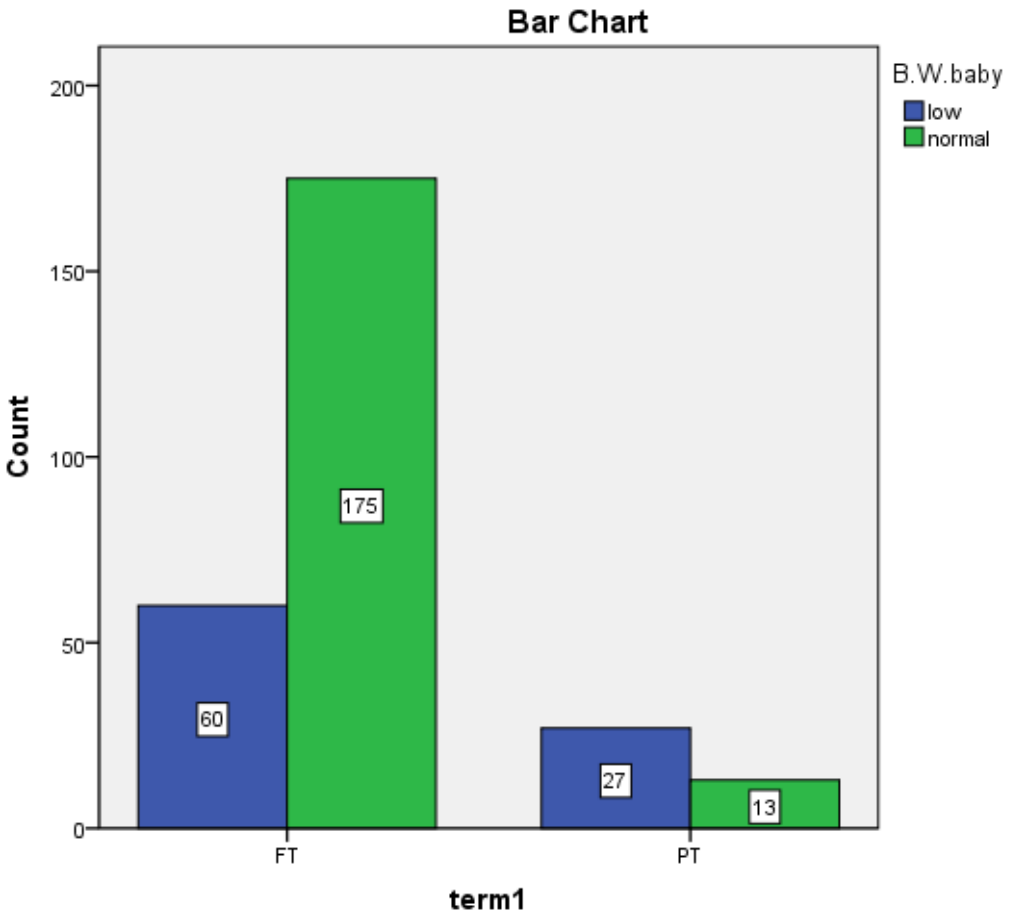

Figure 5.22: Classification of subjects according to the birth weight and gestational age.

Table 5. 23: Basic Characteristic's Of The Study Population.

Characteristics	Term delivery (n= 246) Mean ± Std.Deviation	Preterm delivery (n=42) Mean ± Std.Deviation	P Value from t- test
Age (Year)	27.8 ± 4.7	30.6 ± 4.7	0.0001*
Gestational age	39.6 ± 1.6	34 ± 4.6	0.0001*
Parity	1.15 ±1.3	3.2± 2.3	0.0001*
Birth weight	3.3 ± 0.55	2.2 ± 0.786	0.0001*
Gravidity	2.5 ± 1.6	2.9 ± 2.3	0. 001*
Mothers weight	78.7 ± 15.2	75.7 ± 14.1	0.010*

***Significant difference.**

5.2- Periodontal examination results

5.2.1- Plaque index results

As shown in table (5.24) figure (5.23), 153 subjects had moderate plaque deposition, and 78 subjects had mild plaque deposition and 21 subjects had poor plaque deposition. While 48 subjects normal according to Silness and Loe score criteria [(258)].

5.2.2- Gingival index results

As shown in table (5.25) and figure (5.24), 180 subjects had moderate gingivitis and 85 subjects had mild gingivitis and 24 subjects had sever gingivitis while 11 subjects had normal gingiva according to Loe and Silness criteria [(259)]

5.2.3- Periodontal disease index results

As shown in table (5.26) and figure (5.23), 141 subjects had moderate gingivitis, 87 subjects had mild gingivitis, 30 subjects had sever gingivitis and 33 subjects had mild periodontitis and no subject had moderate nor sever periodontitis while 9 subjects had normal PDI status according to periodontal disease index of Ramfjord [(260)].

5.2.4- Correlation between plaque & gingival index scores

The table (5.27) showed that 60 percent of subjects had moderate gingivitis which is related to a high percentage of plaque deposition which is (51%) as shown in figure (5.26).

Table 5. 24: Distribution of subjects according to the plaque index score.

Average plaque index score	No.	%
0 – 0.99(Normal)	48	16
1 – 1.99(mild)	78	26
2 – 2.99(moderate)	153	51
≥3 (poor)	21	7
Total	300	100

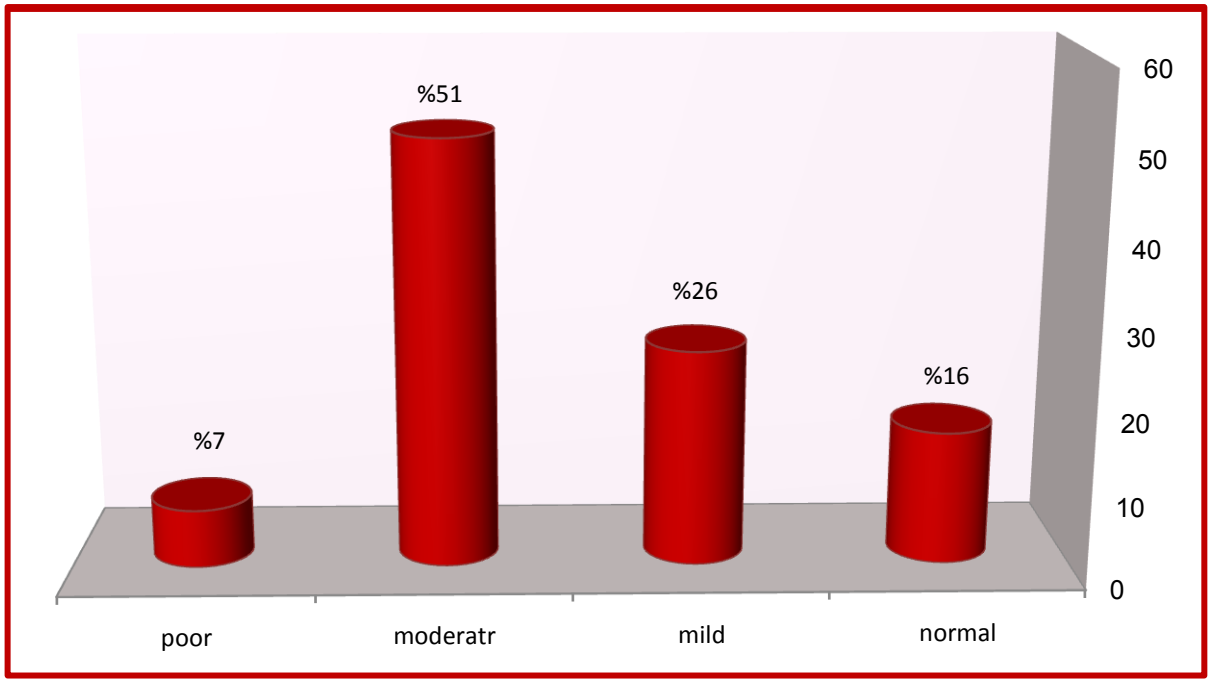

Figure 5.23: Distribution of subjects according to the plaque index score.

Table 5. 25: Distribution of subjects according to the gingival index score.

Average gingival index score	No.	%
0 – 0.99(Normal)	11	3.6
1 – 1.99(Mild gingivitis)	85	28.4
2 – 2.99(moderate gingivitis)	180	60
≥3 (Sever gingivitis)	24	8
Total	300	100

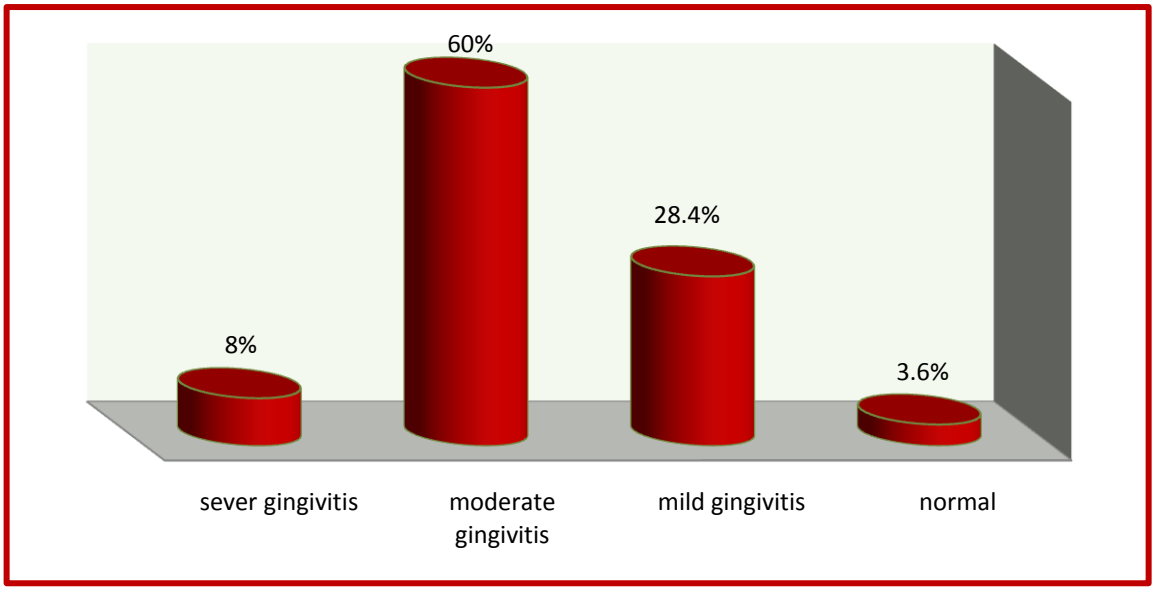

Figure 5.24: Distribution of subjects according to the gingival index score.

Table 5. 26: Distribution of subjects according to the periodontal disease index score.

Periodontal disease index score	No.	%
0 – 0.99(Normal)	9	3
1 – 1.99(Mild gingivitis	87	29
2 – 2.99(moderate gingivitis)	141	47
3 – 3.99(Sever gingivitis)	30	10
4 – 4.99(Mild periodontitis)	33	11
5 –5.99(Moderate periodontitis)	0	0
≥ 6 (Sever periodontitis)	0	0
Total	300	100

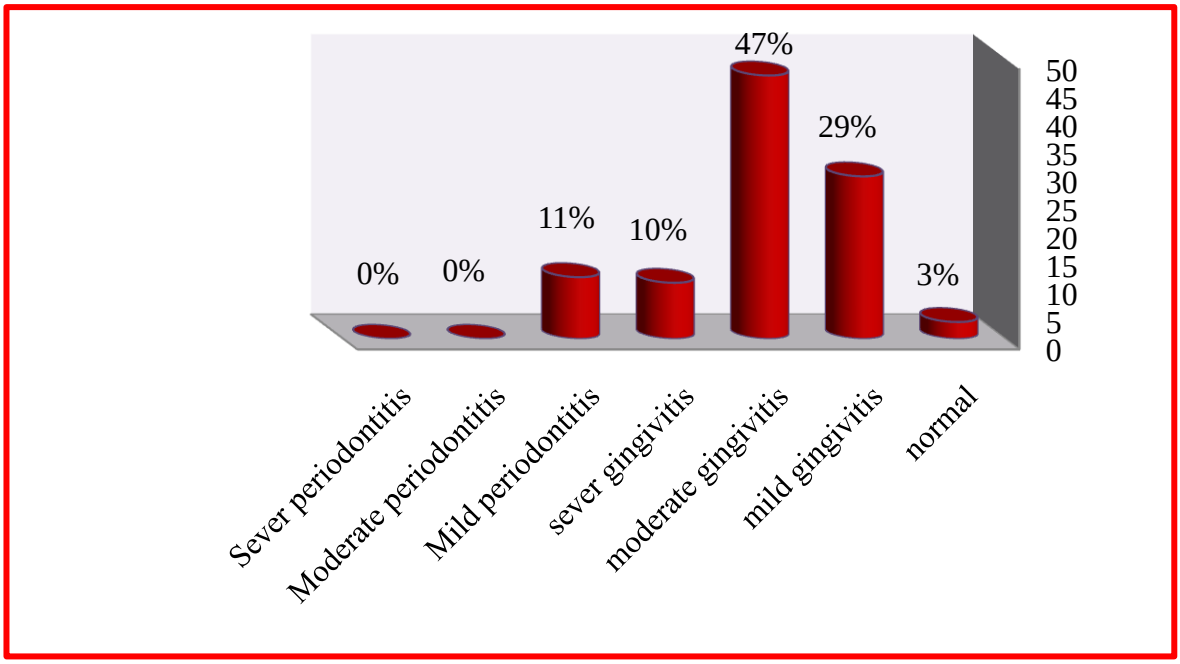

Figure 5.25: Distribution of subjects according to the periodontal disease index score.

Table 5.27: Correlation between the plaque index and the gingival index scores.

score	Plague index score	Gingival index score
0 – 0.99 (Normal)	16 %	3.6 %
1 – 1.99 (Mild)	26 %	28.4 %
2 – 2.99 (moderate)	51 %	60 %
3 – 3.99 (Sever)	7 %	8 %

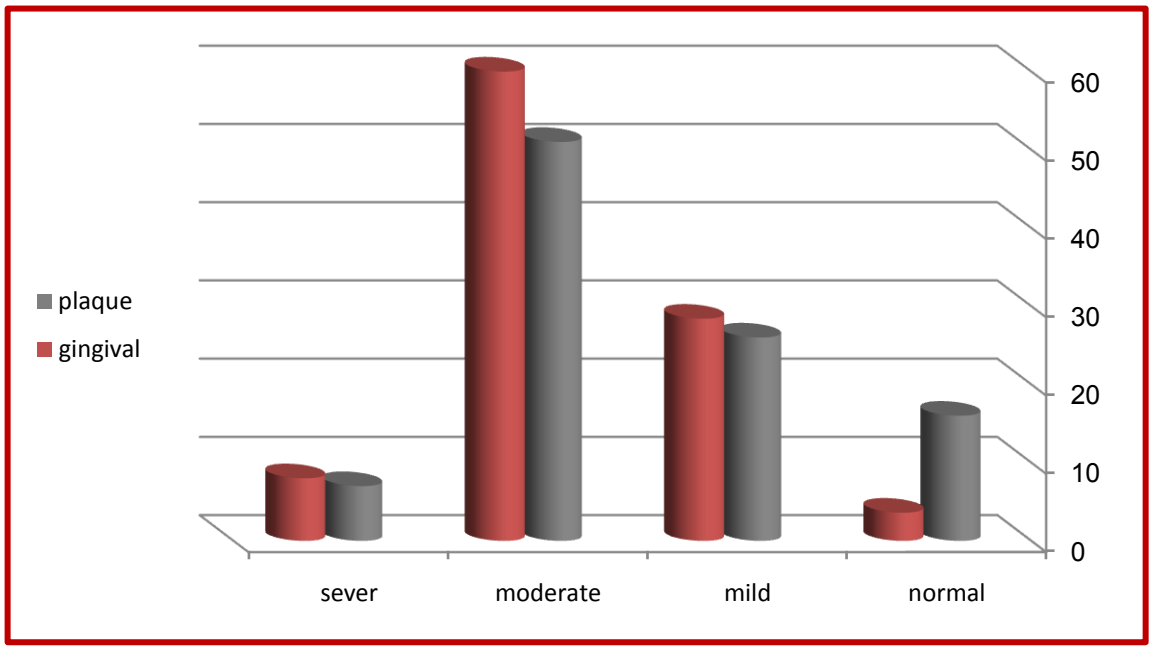

Figure 5.26: Correlation between plaque index and gingival index scores.

5.3- Socioeconomic status with birth weight and gestational age relationship results.

Chi-Square test (X^2) and p –value, used to determine the significant of the relationship between socioeconomic status of the subject with birth weight and gestational age.

Kind of subjects according to type of family showed no significant relationship with birth weight P-value= 0. 510 as seen in table (5.28). And also showed no significant relationship with gestational age P-value = 0. 521 as seen in table (5.29).

Kind of subjects according to level of their education showed no significant relationship with birth weight P-value = 0. 481 as seen in table (5.30). And also showed no significant relationship with gestational age P-value = 0.560 as seen in table (5.31).

Kind of subjects according to husband‘s occupation showed no significant relationship with birth weight P-value = 0.630 as seen in table (5.32). And also showed no significant relationship with gestational age P-value = 0.414 as seen in table (5.33).

Kind of subjects according to their occupation showed no significant relationship with birth weight P-value = 0.230 as seen in table (5.34). And also showed no significant relationship with gestational age P-value = 0.411 as seen in table (5.35).

Kind of subjects according to number of family member showed no significant relationship with birth weight P-value = 0. 169 as seen in table (5.36). And also showed no significant relationship with gestational age P-value = 0.129 as seen in table (5.37).

Kind of subjects according to their house ownership showed no significant relationship with birth weight P-value = 0. 475 as seen in table (5.38). And also showed no significant relationship with gestational age P-value = 0. 971 as seen in table (5.39).

Table 5.28: kind of subjects according to the type of family and birth weight.

Type of the family	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
	No.	%	No.	%		
Nuclear*	27	12.3	193	87.7	220	100
Extended**	5	9.1	50	90.9	55	100
Total	32	11.6	243	88.4	275	100

X^2 = 0.433 df= 1 P = 0. 510 (Not significant)

Nuclear* patients live alone separated from their parent.

Extended** patients live with their parents at same house.

Table 5.29: kind of subjects according to the type of family and gestational age.

Type of the family	Gestational age				Total	
	Full term		Pre term		No.	%
	No.	%	No.	%		
Nuclear	198	86.1	32	13.9	230	100
Extended	48	82.8	10	17.2	58	100
Total	246	85.4	42	14.6	288	100

X^2 = 0.412 df= 1 P = 0. 521(Not significant)

Table 5. 30: kind of subjects according to their level of education and birth weight.

Level of education of the patients	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
	No.	%	No.	%		
Illiterate	2	12.5	14	87.5	16	100
Primary	0	0	6	100	6	100
Preparatory	8	13.1	53	86.9	61	100
Secondary	6	7.2	77	92.8	83	100
University	16	14.7	93	85.3	109	100
Total	32	11.6	243	88.4	275	100

X^2 = 3.481 .df= 4 P= 0. 481 (Not significant)

Table 5. 31: kind of subjects according to their level of education of patients and gestational age.

Level of education of the patients	Gestational age				Total	
	Full term		Pre term		No.	%
	No.	%	No.	%		
Illiterate	15	93.8	1	6.3	16	100
Primary	5	83.3	1	16.7	6	100
Preparatory	53	80.3	13	19.7	66	100
Secondary	77	88.5	10	11.5	87	100
University	96	85	17	15	113	100
Total	246	85.4	42	14.6	288	100

X^2 = 2.984 .df= 4 P= 0.560 (Not significant).

Table 5.32: kind of subjects according to the husband's occupation and birth weight.

Husbands occupation	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
	No.	%	No.	%		
Free business	10	8.4	109	91.6	119	100
Civil Servants	19	18.1	86	81.9	105	100
Army	3	6.5	43	93.5	46	100
Technician	0	0	5	100	5	100
Total	32	11.6	243	88.4	275	100

$X^2 = 7.298$ .df=3 P = 0.630 (Not significant)

Table 5. 33: kind of subjects according to the husband's occupation and gestational age.

Husbands occupation	Gestational age				Total	
	Full term		Pre term		No.	%
	No.	%	No.	%		
Free business	109	81.3	14	11.4	123	100
Civil Servants	91	81.3	21	18.8	112	100
Army	42	87.5	6	12.5	48	100
Technician	4	80	1	20	5	100
Total	246	85.4	42	14.6	288	100

$X^2 = 2.858$. df=3 P = 0.414 (Not significant)

Table 5. 34: kind of subjects according to their occupation and B.W.

Type of occupation	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
House wife	17	8.8	177	91.2	194	100
Teacher	6	14.6	35	85.4	41	100
Employee	5	23.8	16	76.2	21	100
Student	3	25	9	75	12	100
Nurse	1	25	3	75	4	100
Technician	0	0	2	100	2	100
Doctor	0	0	1	100	1	100
Total	32	11.6	243	88.4	275	100

X^2 = 8.117. df= 6 P = 0.230 (Not significant)

Table 5. 35: kind of subjects according to their occupation and G.A.

Type of occupation	Gestational age				Total	
	Full term		Pre term		No.	%
House wife	181	87.9	25	12.1	206	100
Teacher	34	82.9	7	17.1	41	100
Employee	15	71.4	6	28.6	21	100
Student	10	76.9	3	23.1	13	100
Nurse	3	75	1	25	4	100
Technician	2	100	0	0	2	100
Doctor	1	100	0	0	1	100

X^2 = 6.107. df= 6 P = 0.411 (Not significant)

Table 5. 36: kind of subjects according to the number of family member and birth weight.

Number of the family members	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
	No.	%	No.	%		
≤5	24	11.1	192	88.9	216	100
6 - 10	8	17.8	37	82.2	45	100
>10	0	0	14	100	14	100
Total	32	11.6	243	88.4	275	100

$X^2 = 3.552$ df= 2 P = 0. 169 (Not significant)

Table 5. 37: kind of subjects according to the number of family member and gestational age.

Number of the family members	Gestational age				Total	
	Full term		Pre term		No.	%
	No.	%	No.	%		
≤5	197	87.6	28	12.4	225	100
6 - 10	38	79.2	10	20.8	48	100
>10	11	73.3	4	26.7	15	100
Total	246	85.4	42	14.6	288	100

$X^2 = 4.090$ df= 2 P = 0.129 (Not significant)

Table 5. 38: kind of subjects according to their housing /accommodation and birth weight.

Housing /accommodation	Birth weight				Total	
	<2.500		≥ 2.500		No.	%
	No.	%	No.	%		
Yes	28	11.2	222	88.8	250	100
No(renting)	4	16	21	84	25	100
Total	32	11.6	243	88.4	275	100

X^2 = 0.509 .df=1 P = 0. 475 (Not significant)

Table 5. 39: kind of subjects according to their housing /accommodation and gestational age.

Housing /accommodation	Gestational age				Total	
	Full term		Pre term		No.	%
	No.	%	No.	%		
Yes	223	85.4	38	14.6	261	100
No(renting)	23	85.2	4	14.8	27	100
Total	246	85.4	42	14.6	288	100

X^2 = 0.001.df=1 P = 0. 971(Not significant)

5.6- Correlation of preterm delivery with PDI, GI.

Correlation between preterm (PT) and periodontal disease as shown in table 5.40. There was an indirect relationship with GI and PDI. The P-value = 0.826 with periodontal disease index (PDI) which is not significant and P-value with gingival index (GI) = 0.936 which also not significant. Plaque index (PI) showed P-value = 0.030 which means that plaque index (PI) had statically significant relationship with preterm.

5.7- Correlation of birth weight with PDI, GI.

Table 5.41 showed that PDI had direct correlation with birth weight (B.W) but GI had indirect correlation with birth weight, P-value was (0.887, 0.525) with (PDI, GI,) respectively that is mean that PDI,GI were not significant with birth weight.

5.8- Correlation of premature low birth weight with PDI, GI.

Pearson correlation between premature low birth weight (PLBW) with PDI had direct correlation where as correlation of PLBW with GI was indirect correlation and P-value was (0.849, 0.302) for (PDI, GI) respectively that mean PLBW was not significant with PDI, GI as seen in table 5.42.

Table 5. 40: Correlation of preterm with PDI,GI.

Parameter	Pearson correlation	P- value
PDI	-0.013	0 .826 (Not Significant)
GI	-0.005	0.936 (Not Significant)
PI	-0.129	0.030 (significant)

Table 5. 41: Correlation of birth weight with PDI, GI.

Parameter	Pearson correlation	P-value
PDI	0.009	0.887(Not Significant)
GI	-0.038	0.525(Not Significant)

Table 5. 42: Correlation of premature low birth weight with PDI, GI

Parameter	Pearson correlation	P- value
PDI	0.048	0.849(Not Significant)
GI	- 0.258	0.302(Not Significant)

DISSCUSION

6- DISSCUSSION

In this study periodontitis had been examined in addition to different factors which may contribute to PLBW in a sample of 300 Libyan ladies gave their birth at obstetrics and gynaecology department at Al-Jomhuriya Hospital in Benghazi. In order to determine the relation between periodontitis and preterm low birth weight (PLBW) among Libyan pregnant ladies. After a granted permission for the study research to conducted at different department of Al-Jomhuriya Hospital obtained (Appendix I) and the permission agreement of the participant to participate in the study received (Appendix IЦ).

Three hundred Libyan ladies responded to many questions face to face in the questionnaire. Responded to the questions on; age, address, type of house, education level, family size, mother and father work also to smoking history, number of their pregnancy, number of their children, history of abortion in addition to past medical and obstetric history including previous PLBW and prenatal visit (Appendix IIII).

The entire participant's weight recorded in kilogram, as the weight is a risk factor of preterm delivery[256]. High Vaginal swaps collected using disposable speculum from each participant to excluded local infection after the interview and before the delivery to avoid contamination and all swaps sent to Microbiology department in Al-Jomhuriya Hospital laboratory.

After that, periodontal examination done before delivery using; Plaque index of Silness & Loe (PI) [258], Gingival index of Loe & Silness (GI) [259] and Periodontal disease index for Ramfjord (PDI) [260] to assess periodontal disease factor. Lastly, at the day of delivery the babies' weight was recorded from the hospital's record book in labour department.

Descriptive statistics were used to assess the characteristic of study obtained from data relating to; identification, socio-graphic data, obstetric history, gestational history, smoking and medical history obtained from questionnaires.

6.1 -Discussion of characteristic sample.

The preterm births data from "National, regional and worldwide estimates of preterm birth rates in the year 2010 for selected countries since 1990"; shows Preterm birth rate (2010) in Libya 8.3 percent[263]. While the preterm birth rate in the current study sample was found to be 14 percent. The prevalence of LBW as estimated by UNICEF/ WHO around the world in more developed country 7.0 percent, less developed 16.5 percent and least developed countries 18.6 percent[264].

In a study was done in neonatal department at Al –Jomhuriya- Hospital (2008) LBW was 9.55 percent[265], and in our study the LBW was 10.7 percent. Low incidence of LBW and preterm babies in our country may be due to that our Libyan mothers are non smoker, non alcoholic and most of time they are not practicing hard work[265,266].

To our knowledge this association was not explored among Libyan women (at least Benghazi). Women in Libya are of similar ethnic background, non-smokers, non alcohol drinkers, and has only one sexual partner all through their life. Such characteristics were found to be associated with PLBW. Additionally most of the confounding variables that well known as risk factors associated with PLBW were analyses, and controlled like; age, socioeconomic factors, previous preterm history, previous LBW history, vaginal infection, prenatal care and past medical and obstetric history. So the strength of our study lies in the fact that was adjusted for the most important confounders in addition the study was conducted among homogenous women.

In this study women with maternal age under 18 and over 40 years were excluded, since age outside this range is known as a risk factor for PLBW[213]. Women under 16 and those above 35 have a 2 to 4 percent higher rate of preterm birth[147]. To control the effect of maternal age as a risk factor for PLBW the highest percent of the sample is between 26-30 years old,27.6 percent of the sample is between 31-35 years old as well as 27.7 percent of the sample is between 21-25 years old. That means the highest percent of the sample is less than 30 years old. This pattern of age group was similar to the study conducted by Offenbatch et al.[206] and Zadeh-modarres et al.[267]. T-test show statically significant relationship between age and preterm (P-value=0.0001) in concordance with those reported in several other studies[268, 269, 270]. Even though some studies show insignificant relationship between age and PLBW[256,243].

Weight of Libyan ladies in this study showed significant relationship with PT (P-value=0.010) Similar results have been reported by Sekiya et al. 2007[271], Chan and Lao, 2009[272], Claude Bayingana, 2010[256]. This can be expected because the thinner the mother, the weaker she would be and thus less able to carry full term. It has been documented that women with a poor nutritional status are at greater risk for preterm birth[257].

Parity is an important risk factor for PLBW. High parity is likely to increase the risk of preterm delivery due to uterine changes such as myometrium stretching from previous pregnancies[273]. Parity in our study categorized into; nulipara, from 1-5, and more than 5. The Primi in our sample was 44.3 percent, from 1- 5 were 54.7 percent and more than 5 was 1 percent. Our study demonstrated that mothers with a parity >5 has significant association with preterm delivery (P-value=0.0001). This finding is similar to that of most studies which had shown that multiparaous women were more likely to deliver preterm[270, 274]. On contrary Davenport et al. found insignificant result[214].

Minimum gravidity (number of previous pregnancies) in our study was one, and Maximum gravidity was nine. About 84.7 percent of ladies had from

one to four gravida, and about 15.3 percent had ≥ 5. The present study showed association between the gravidity and preterm (P-value=0.001), which agree with Minkoff et al. (1984)[164] and in contrast to the study by D.Gandhimadhi and R. Mythili (2010)[275].

The sample showed significant association between B.W (birth weight) and PT (P-value=0.0001) and insignificant relationship between PDI with B.W (P-value= 0.119) this result in accordance of result conducted by Lunardelli and Peres[276] and in contrast with Jeffcott et al.[209].

In our sample about 74 percent had no history of abortion. Association between history of abortions and preterm low birth weight deliveries in our study were found insignificant. This is in accordance with Augeda et al.[277]. On the other hand, Marin et al.[278] Found significant results between number of abortions and preterm low birth weight. This is may be due to the presences of specific genetic and environmental factors influence the pregnant women to induce abortion[214].

An insignificant association (P-value> 0.05) was observed between history of previous preterm and preterm deliveries in our study, in accordance with Satheesh Mannem (2011)[279] and on contrary with Davenport et al.[214] who found significant result between previous history of preterm delivery and preterm. This could be because of our sample size of ladies had previous history of PT delivery were low about 6.3 percent (n=19).

To exclude local infection, vaginal swaps were collected from all participants before delivery. Our samples were; 281 cases show negative culture and only 19 cases show positive culture, distributed as Staph auras =11 cases, Streptococcus=7 cases and Candida=1 case. In particular, the causal relationship between BV (Bacterial Vaginosis) and PTB (preterm birth) among women from various ethnic groups has been consistently noted[280]. In our study there were no BV (Bacterial Vaginosis) reported, may be because BV was reported to be high among women from low socioeconomic strata, and those with low levels of

education, a history of smoking, and multiple sex partners[281,282], while our study population not fit this description. No association was observed between PTB and genital tract infection in our sample (P-value=0. 734). Similar negative associations had been reported[283] and some other studies have found a positive association[284]. Besides, distribution of patients according to culture result of high vaginal swap and birth weight showed insignificant relationship (P-value=0.675).

All Libyan ladies in the sample are non-smoker and distribution of patients according to history of passive smoking with birth weight and preterm are not significant (P-value=0.389, P-value=0.251) respectively. The findings of pooled studies conducted before mid-1995, were inconsistent, it showed that the risk of low birth weight was not increased in infants of SHS (second hand smoke) exposed women, but there was a somewhat increased risk for low birth weight at term[285]. Ward et al. Showed that SHS produced a smaller increase in prematurity, which was non-significant after adjustment for potential confounding[286].

Socio economic status of our studies determined by questions of; type of house, maternal education, maternal work, father work, other's family work and family size. Our result was; distribution of patients according to level of education of the patients and gestational age show P-value=0.560 (Not Significant), distribution of patients according to type of family and gestational age P-value=0.521(Not Significant), distribution of patients according to husbands occupation and gestational age P-value=0.414 (Not Significant), distribution of patient according to type of occupation and gestational age P-value=0.411 (Not Significant), distribution of patient according to number of the family members and gestational age P-value=0.129 (Not Significant). Also distribution of patient according to Owen their house and gestational age P-value=0.971 (Not Significant). These results show no significant relationship between socioeconomic status and preterm among Libyan ladies which in

accordance with most studies conducted in European countries and Canada[214,279].

A significant association between PLBW and socioeconomic status was observed in population groups with an incidence of preterm deliveries from economically disadvantaged families shown by Lopez et al.[55]. What's More in our study the distribution of patients according to birth weight with; type of family, house ownership, number of the family members, occupation of the patients, husbands occupation and level of education of the patients show (P-value=0. 510, P-value=0. 475, P-value=0. 169, P-value=0.230, P-value=0.630 and P-value=0. 481) respectively that's mean not only preterm birth but also birth weight showed insignificant relationship with socioeconomic status among Libyan ladies.

According to current knowledge, there are several risk factors for preterm low-birth weight (PLBW), e.g. maternal age of <18 years or >40 years, low socioeconomic status, alcohol/ drug abuse, smoking, multiple pregnancies or poor general health of the pregnant woman. High-risk gestation, hypertension, gestational diabetes and systemic disease, placenta previa and maternal thinness defined by a low body index are also considered as risk factors. Urinary tract infections, infections of the genital tract, such as (BV) and intra-uterine infections are believed to be etiologic for many PTBs. The other main risk factors for preterm labour (PTL) are previous PTL; pregnant women with a history of previous PTL presented a 15–80 percent risk of having another PTL in future pregnancies[287]. As well as Psychological stress, physical activity, gestational weight gain, violence, and social support are important risk factors for adverse pregnancy outcomes. However, a significant proportion of PT/LBW is of unknown aetiology[214].

All the studies testing the association between periodontal disease and adverse pregnancy outcomes were the inconsistency in controlling for confounders. It is possible that some residual confounding effects remain. This

is a major shortcoming and raises doubts as to the conclusions of all such studies. It is thus vital to control for as many confounding factors as possible by using different strategies like restriction, matching, in order to avoid spurious associations and all potential risk factors for PLBW should be included in the design of the study[240].

Moreover, both periodontitis and premature labour involve multi factor aetiology. It's probable that maternal periodontitis may interact synergically with other maternal risk factors to induce preterm births. Such as a short cervix is more closely associated with preterm births when the woman has also BV[288]. It is also to be remembering that, the risk factors of preterm birth appear to be similar to risk factors for periodontal diseases (tobacco, ethnicity, socioeconomic and educational levels) and may confound the association between periodontitis and preterm birth[50, 235, 289, 290]. Actually, smoking is recognized as one of the principal risk factors for both adverse pregnancy outcomes and periodontitis[134,291].

It is possible, also exists some unknown genetic or environment factors which locates the patient in the risk category of the periodontal disease and the premature labour at the same time[292]. Like gene-environment association between BV, PD, and PTB suggests that only pregnant women with BV and/or PD who have a genetic predisposition to mount a damaging inflammatory response to anaerobic oral or genitourinary flora will develop periodontitis, chorioamnionitis and PTB. Pregnant women who have BV and/or PD, but who do not have a predisposition to mount a damaging inflammatory response, are less likely to deliver preterm[293,294].

As a result, a woman with a genetic predisposition to mount a damaging inflammatory response to infection may have varying full term or preterm deliveries in their pregnancy history based upon their exposure to anaerobic bacteria during each individual pregnancy[295].

6.2-Discussion of Periodontal Status.

While the definitions of PB (preterm birth) and LBW (low birth weight) are well established. No consensus has yet been achieved on the definition of periodontitis in periodontal research, which is essential to optimize the interpretation, comparison and validation of clinical data [(238)]. In periodontitis most of the researchers used their own case definitions, mostly based on disease distribution within the study population[(50)]. Research on periodontitis has and is being plagued by the use of a variety of case definitions[(296)]. It seems, Selection of different diagnostic definitions and measurements of periodontal disease states will also lead to different outcomes [(234)].

Gomes-Filho et al. stated that the use of less strict definition leads to an insignificant association. When a strict definition of periodontitis is adopted, the number of cases is bound to become less for analysis [(297)]. Additionally, Dasanayake et al.[(211)], and Davenport et al.[(214)] used the CPITN and found that the relation between PLBW and periodontal disease was significant. But the use of CPITN doesn't seem appropriate as it is recommended for the estimation of treatment needs and can underestimate the prevalence of bleeding, periodontal pockets and loss of attachment[(296)].

Hence it is important to use a priori definition in accordance with the prevalence and severity of periodontitis in the studied population and/or to perform a sensitivity analysis in order to examine the effects of different definitions of periodontitis[(297)]. A relatively strict definition of periodontitis should be used in order to exclude false positives[(296)].

Furthermore, precise selection of exposure measurement will make the results consistent and confer greater safety in determining the association. In many studies[(206,162,214,298)], the periodontal examination of women was accomplished within a few days post-partum. The oral hygiene of the women

would be less than optimum and hence more gingival inflammation can be expected[296].

In this study; the periodontal examination performed when women came to the labour department to give their birth. We used three indices to assess periodontitis in pregnant Libyan ladies:-

1-Plaque index score of Silness & Loe (PI) to assess the oral hygiene of the participant.

3- Gingival index of Loe & Silness (GI) to assess gingival status and gingival inflammation.

4- Periodontal disease index for Ramfjord (PDI) to assess periodontal bone status.

The sample showed that 62 percent of pregnant Libyan ladies had gingivitis, this is in accordance with American Dental Association (ADA) "approximately 50-70 percent of pregnant women had gingivitis" [185]. In 2004 Salma Mahfoud found 56 percent of pregnant Libyan ladies had gingivitis in Benghazi[299].

6.2.1- Plaque index score of Silness & Loe (PI).

Plaque index (PI) used to assess oral hygiene of a patient by measuring plaque accumulation [300]. Our sample showed 51 percent had moderate plaque deposition, 7 percent had abundant plaque deposition and 26 percent had mild plaque accumulation, while 16 percent of our sample had scanty plaque accumulation. This explains the degree of gingival inflammation as sever with 8 percent and moderate with 60 percent and mild with 28.4 percent of the study sample size. During pregnancy the oral microflora uses the progesterone and estrogen hormones as a growth factors and they form plaque on the gingival margin and tooth surface[301].

In this study sample the statistic analysis showed significant relationship between PI and PT at P-value =0.030. Often plaque accumulates on teeth surface when stop daily oral hygiene such as brushing, ladies in the last trimester usually they are under increased anxiety and stress and may neglect their dental hygiene as brushing their teeth so the relation between PI and PT is expected. This result is in accordance with the studies conducted by Yalcin et al.[302] and Satheesh Mannem[279] In contrast to study conducted by Radnai et al. showed insignificant relationship between plaque and preterm birth[216].

6.2.2- Gingival index of Loe & Silness.

Gingival Index (GI) was developed to assess the severity and quality of gingival inflammation in individual or population [300]. Our sample showed 28.4 percent had mild gingivitis and only 8% had severed gingivitis while 60 percent had moderate gingivitis. Correlation between GI and PT by using logistic regression not significant (P-value=0.936). Also Pearson Correlation of GI with PLBW is in significant (P-value=0.302). In addition, correlation of GI with B.W (birth weight) also showed in significant relationship (P-value=0.525).

6.2.3- Periodontal disease index of Ramfjord.

Commonly accepted clinical measures of periodontal disease are clinical attachment level (CAL, the distance between the cemento-enamel junction and clinical pocket base) and probing depth (PD, the distance from the gingival margin to the apical part of the pocket)[50].

The periodontal pocket depth associated to gingival bleeding measurement could be considered as the best markers of periodontal disease activity or the inflammatory/infectious burden of periodontitis. The measurement of clinical attachment level (periodontal pocket depth plus gingival recession) and bone loss around teeth reflect more the history and the severity of periodontal disease[205].

The measurement of clinical attachment loss is an important and comparable periodontal parameter, as it allows estimating periodontal status regardless of inflammation. The pocket base and cementoenamel junction are explicit referential points, unlike the gingival margin, which depends on actual inflammation conditions. This is why the AAP assigned CAL as the basis of diagnosing periodontitis[303]. So case definition for periodontitis should include probing depth and clinical attachment level. Ramfjord's PDI is still considered as the "gold standard" method for determining the status of periodontium[300] which used in this study. It is used to measure incidence and prevalence of periodontal disease. It can be used in large survey because it is quick and easy.

The sample showed; about 11 percent of a sample had mild periodontitis, and no ladies had moderate or severe periodontitis. According to periodontal disease index 87 ladies had mild gingivitis mean about 29 percent of a sample, and 141 ladies had moderate gingivitis and 30 had sever gingivitis which are about 47 percent, 10 percent of the sample respectively.

According to Pearson correlation **(**P-value=0.826) which is in significant relationship between PDI with PT. Again Pearson correlation was used to test the relationship between PDI with B.W, It found no significant relationship between PDI and B.W (P-value=0.887). As well as the relationship between PDI with PLBW at (P-value=0.849) was not significant relationship.

The study's results conclude that PDI is not significant with PT/LBW or PLBW. Thus, these results do not support the hypothesis that periodontal disease is an independent risk factor of preterm delivery among Libyan women in Benghazi.

Generally speaking the link between periodontal health status of pregnant women and adverse pregnancy outcomes is still contentious as many studies found no association between periodontitis and pregnancy disorders[304]. Even there is no association between maternal periodontal disease and preterm delivery among Libyan ladies in Benghazi- Libya in this study. Based on our results, additional epidemiological studies are needed including a larger number and wider spectrum of participants from different hospitals in different areas in Libya. It is necessary to standardize the case definition and selection criteria, so that reasonable conclusions can be drawn when comparing various studies.

Future investigators in this area should also consider measuring markers of active periodontal disease and not rely solely on clinical examination. As periodontitis is episodic in nature, probing alone cannot determine whether the disease is active or quiescent and since periodontal disease is characterized by a relapsing / remitting pattern, identifying active disease may be an important factor in establishing associations with PT. Several tests had been developed to assess substances in the gingival crevicular fluid (GCF) which can be obtained from periodontal pockets. Like; neutrophil elastase, dipeptidylpeptidase and gingipain have proved to be good diagnostic enzymes[305].

Moreover chronic periodontitis is considered a site specific disease and the clinical signs are believed to be caused by direct site specific effects of subgingival plaque accumulation. As a result of this local effect, periodontal destruction may occur on one surface of the tooth while other surfaces maintain normal attachment levels[306]. That is why full chart of periodontal disease is ideal structure to diagnosis periodontitis as periodontitis is site specific disease. A potential link between periodontal infections and adverse pregnancy outcomes has been established based on 2 principles. First, periodontal bacteria can directly cause infections to the uteroplacenta and to the fetus; second, systemic inflammatory changes induced by periodontal diseases can activate responses at the maternal–fetal interface.

However, associative studies have produced different results in different population groups and no conclusive evidence has still been produced[307]. In 2007, Vergnes and Sixou published a systematic review where they concluded that PD may be an independent risk factor of PB or LBW/ PLBW, association does not imply causation, and it seems important to consider the possibility that there is some underlying mechanism causing both PD and APO[308].

CONCLUSIONS

7- CONCLUSIONS

Many risk factors are associated with premature low birth weight. This study was conducted to assess the relationship between the periodontal disease and premature low birth weight among Libyan population. The following has been concluded:-

1-The preterm rate in the pregnant Libyan sample was 14%. And the rate of low birth weight was 10.7%.

2-It showed that, maternal age, maternal weight, gestational age, birth weight, parity and gravidity had significant relationship with preterm delivery among the Libyan pregnant sample.

3- Abortion, previous preterm, previous low birth weight, local infection, prenatal visit and socioeconomic status had insignificant relationship with preterm delivery among the selected sample.

4- No significant relationship has been shown between periodontitis and preterm delivery, birth weight and preterm low birth weight among Libyan women in Benghazi.

5-The sample showed 62% of pregnant Libyan ladies had gingivitis.

RECOMMENDATIONS

8- RECOMMENDATIONS

1. It is still recommended that women who are pregnant or planning to become pregnant continue to maintain optimum periodontal health with professional cleaning and meticulous oral hygiene to prevent periodontal disease.

2. Based on the results of this study, future investigators in this area should consider full chart of periodontal disease and future studies that investigate specific characteristics of periodontal pathogens, as well as maternal immune and inflammatory responses, and used tests to identifying active disease, furthermore, future research to be focus on establishing why some women develop adverse pregnancy outcomes due to an oral inflammatory burden while others do not.

3. We need to conduct good-quality, multi-centre studies and intervention studies in different Libyan cities. Before this can assume to be a casual relationship among Libyan ladies.

4. Health professionals as part of their regular care should provide oral health care to pregnant women. At the same time, pregnant women, should have the knowledge of the obvious signs of oral disease.

5. A better communication between dentists and medical doctors is needed, and more responsibilities and effective team approaches in the clinical management of their shared patients for better oral health and general health.

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9- REFERENCES

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APPENDICES

10-APPENDICES

APPENDIX I

Form of a granted permission

الجماهيرية العربية الليبية الشعبية الاشتراكية العظمى
جامعة العرب الطبية
كلية طب وجراحة الفم والأسنان
بنغازي

التطور العلمي مكسب للإنسانية

الرقم الإشاري : ٧٨٦
التاريخ :
الموافق : ٢٠١٥/٥/١

الأخ / مدير مستشفى الجمهورية...

بعد التحية،،،

نود الإفادة بأن الأخت طبيبة الأسنان / نزهة عبدالحميد الدغيلي، هي إحدى طالبات الدراسات العليا بقسم (طب الفم) بالكلية.

لذا نأمل منكم تسهيل الإجراءات اللازمة بشأن فحص التهابات اللثة على السيدات الحوامل والولادات المبكرة ونقص حجم الجنين عند السيدات الليبيات.

شاكرين لكم حسن تعاونكم

والسلام عليكم ورحمة الله وبركاته

د. مفتاح علي أسعيد
أمين اللجنة الشعبية للكلية

لكل من :-
*الأخ منسق الدراسات العليا بالكلية
*الملف الصادر
* الملف المعني
م/ر/التوزيع

هاتف :45-9096046-9092239-9092219 - بريد مصور :00218619093771 - ص.ب :9504 - بريد عبد المنعم رياض

APPENDIX Ц

CONSENT PERMISSION FOR THE PARTICIPANTS

Iam: - Nuzha Eldegheli postgraduate dentist in oral medicine department. My study title is "the relationship between periodontitis and premature low birth weight among Libyan ladies"
Your participation in this study will help us to identify if the periodontitis is a factor associated with preterm delivery in this country. This will help in developing measures to prevent preterm low birth delivery so as to ensure as many babies as possible are born healthy at term.

I will give you information you need to help you decide whether to participate in the study or not. You may ask questions on the risks and benefits of the study on your baby and yourself.

((I will ask you questions related to your general health, obstetric and gynaecology history, your answer is yes or no response, weight you, and vaginal swap will be take to exclude local infection and oral examination will do here in reception labour bed to assess your oral hygiene. and instruction regarding oral hygiene will give and refer you to suitable dental department to begin treatment you need after delivery)).

All the information obtained will be held in strict confidentiality. And we will not publish or discuss in public that will identify you or your baby.

Appendix III

Structural questionnaire

Patient name: .....
Age .....
Nationality .....
Address .....
Past Medical History..... ....

Education Level :-

non.....Elementary.....Primary.....Secondary.....Higher

Type of house owner..... Rent.....

Maternal' work: - .....

Father's work: - .....

Other's in family work.....

Type of family Nuclure..... Extended.....

Family size:-.....

Body Weight:-.....kg

Gravid , Para ,Abortion G..... p..... A.....

Last menstral period (L.M. P) -.....expecting delivery day (E.D.D).....

Gestational age (US):- .....

Diabetic .....

Hypertension .....

Need AB prophylaxes before dental treatment.....

Smoking History .....

Passive smoking .....

Previous L.B.W. ....

Previous preterm. .....

Prenatal Visit .....

Past Obstetric History .....

Date

Appendix IV

Plaque index criteria

Score	Criteria
0	No plaque in gingival area.
1	Film of plaque adhering to free gingival margin and adjacent area of tooth, plaque may be noticed by running a probe across tooth surface.
2	Moderate accumulation of plaque on gingival margin, and or adjacent surface, which can be seen by naked eye.
3	Abundance of plaque along the gingival margin; interdental spaces filled with plaque

Appendix V

Gingival index criteria

score	Criteria
0	Normal gingival
1	Mild inflammation: Slight change in color, slight edema, no bleeding on probing.
2	Moderate inflammation: Erythema, edema, and glazing, bleeding on probing.
3	Severe inflammation: marked redness and edema, ulceration and tendency to spontaneous bleeding.

Appendix VI

Periodontal Disease Index Criteria

score	Criteria
0	Absence of inflammation.
1	Mild to moderate inflammatory gingival changes not extending all around the tooth.
2	Mild to moderately severe gingivitis extending all around the tooth
3	Severe gingivitis, characterized by marked redness, tendency to bleed, and ulceration.
4	Gingival crevice in any of the four measured areas (mesial, distal, buccal, lingual) extending apically to the cementoenamel junction but not more than 3 mm.
5	Gingival crevice in any of the four measured areas extending apically to the cementoenamel junction 3-6 mm.
6	Gingival crevice in any of the four measured areas extending apically more than 6 mm from cementoenamel junction.

الخلاصة باللغة العربيه

المقدمة: تم نشر العديد من الدراسات عن العلاقة بين أمراض اللثة والولاد ة المبكرة ونقص وزن الجنين. بعضها تشير إلى وجود علاقة إيجابية وبعضها الاخرى الى علاقة سلبية. اختلاف هذة النتائج يشير الى احتمال تاثر العلاقة بالخصائص السكانيه للمجتمعات.

الهدف: تقييم العلاقة بين أمراض اللثة و الولاد ات المبكرة ونقص وزن الجنين عند النساء الليبيات في مدينة بنغازي–ليبيا.

الطريقة: اختير عدد 300 من النساء الحوامل الليبيات بعد السيطرة على بعض العوامل المعروفه والمسببه للولادة المبكرة / ونقص وزن الجنين عند الولادة مثل : عمرالحامل ووزنها، وعامل التدخين، والرعاية السابقة للولادة، الحالة الصحية العامة للحامل، وحالة الجنين وتاريخ الولادة. تم فحص وتقييم حالة اللثة للسيدات الحوامل باستخدام مؤشر بلاك، ومؤشر التهاب اللثة ومؤشر أمراض اللثة. تم وزن الجنين و تحليل النتائج مع الوزن و يوم الولادة

النتائج: انتشار أمراض اللثة في السيدات الليبيات الحوامل يظهر ان 47 في المئة من التهاب اللثة معتدل، و ان 29 في المئة من العينة لديها التهاب لثة خفيف، و 10 في المئة التهاب لثة مفرط في حين أن 11 في المئة لديهم التهاب فى الجيوب اللثوية معتدل. فقط مؤشر بلاك له علاقة ايجابية مع الولادة المبكرة (معامل الاهمية = 0.030)

الخلاصة: تشير دراستنا إلى عدم وجود ارتباط بين التهاب اللثة و الولادات المبكرة ونقص وزن الجنين لدى السيدات الليبيات في بنغازي.

التوصيات: نتائج هذه الدراسة اولية ولكن هناك حاجة إلى مزيد من البحث فى مختلف المدن الليبية مع زيادة حجم العينة قبل أفتراض أن تكون هذه هى العلاقة عند السيدات الليبيات.

علاقة أمراض اللثة عند السيدات الحوامل بالولادات المبكرة ونقص حجم المواليد في عينة من النساء الليبيات

قدمت من قبل :

نزهه عبد االحميد الدغيلى

تحت إشراف :

د. سالمة محفوظ الورفلى

مساعدى المشرف :

د. موسى بوسريرة

د.محمد صالح حماد

قدمت هذه الرسالة استكمالا لمتطلبات الحصول على درجة الماجستير في طب الفم

جامعة بنغازي

كلية طب جراحة الفم والاسنان

2018