Periodontal Health of Adult Obese Individuals In Khartoum, Sudan.

By:

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BDS (Punjab University, Lahore, Pakistan) 1994

A thesis Submitted in Partial Fulfillment for the requirements of the master Degree of Dentistry in Periodontology

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October 2008
This work is dedicated to my parents and to my wife and sons

To all those who love me, to all those who know me true

To the heaven that stands above me and awaits my spirit too

To the good that needs assistance, to the wrong that needs resistance,

To the future in the distance, and to the good that I will do.

Hisham
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Acknowledgements

I would like to express my sincere thanks and gratitude to my supervisor Professor Ibrahim Ghandour for his valuable advices, directions and support during the study, and for his great influence and insight throughout my work.

I would also like to express my great thanks to my teachers and colleagues in the department of periodontology, University of Khartoum, especially Dr Bakri Gobara and Dr Hanadi Elrayah for helping me bringing this work to light.

Lastly, but in no sense the least, I would like to thank Dr Ammar Khamis (Biostatistician) for his great help and kindness in making this work done.

Hisham
Abstract:

Oral health status of obese individuals is an important subject which was investigated by a number of researchers in different countries. Since this problem was not investigated in Sudan the present study was designed to investigate the periodontal health status of some obese subjects in Khartoum State.

A group of 206 obese male and female subjects were randomly selected from two fitness centers in Khartoum State (Police Club Fitness Center) and (Army Club Fitness Center) according to the following criteria:

1. Age range from 18-35 years.
2. Obese subjects without any other systemic conditions.

3. All Subjects were examined using the Body Mass Index, Plaque Index, Gingival Index, and Recession, frequency of tooth brushing, Probable Pocket Depth of ≥ 4 mm, and Clinical Attachment Loss.

The results revealed that gingival inflammation with a mean and standard deviation of (1.1 ± 0.5), and the prevalence of gingivitis among them was 96%.

However a correlation was done between gingival index and the body mass index there was a significant association (P= 0.029).

Another correlation was done between the level of Clinical Attachment Loss and the Body Mass Index also showed a significant association (P=0.000).

Taking into consideration the results of this study it is recommended that health providers must increase the awareness of obese individuals about their oral health status to improve their oral hygiene and should be advised to more professional and periodontal treatment.
المستخلص

صحة الفم لدى الأشخاص المصابين بالسمنة من الموضوعات الهامة التي تعرض لها العديد من الباحثين في مختلف البلدان.

وبما أن هذا النوع من الدراسة لم يتم إجراها في السودان تم إجراء هذا البحث لدراسة صحة الفم والأسنان لدى الأشخاص المصابين بالسمنة.

تم الكشف في هذه الدراسة على 206 شACL مصAB بالسمنة من بين الذين يترددون على مراكز التخسيس.

في مركزي نادي الشرطة الرياضي العالمي وفرع الرياضة العسكري بالخرطوم تم اختيار الأشخاص من الجنسين حسب الفئة العمرية من 18 - 35 سنة أصحاب لا يعانون من أي أعراض فيما عدا السمنة.

تم الكشف باستخدام مقياس كتلة الجسم ومقياس تحديد درجة Body Mass Index ومقاس Plaque Index ومقاس Gingival Index ومقاس Recession ومقاس Probable Pocket Depth تراكم اللامة الجرثومية ودرجة التهاب اللثة والجروح اللثوية في الأنسجة اللثوية ومقاس كتلة الجسم وكان هناك ارتباط ثفيق حيث أن الدالة الإحصائية لثوية شديدة حيث كان المتوسط والانحراف المعياري (0.5 ± 1.1…

تم إجراء مقارنة ملابسية في درجة التهاب اللثة ومقياس كتلة الجسم ودالة الإحصائية التالية ( P=0.029 ) ( P=0.000 ) من هذه نتائج بأن الأشخاص المصابين بالسمنة لديهم القدرة للإصابة بأمراض اللثة والتهاباتها وما يتبع ذلك من مضاعفات على مجمل صحة الفم والأسنان لذلك يجب زيادة الوعي لدى الأشخاص المصابين بزيادة في الوزن أو السمنة بأهمية الاهتمام بصحة الفم ونظافة الأسنان الدوري.

كما نوصي بتوفير الرعاية السنية المتخصصة لهذه الأشخاص مع علاج الأنسجة حول السنوية لمنع حدوث مضاعفات مستقبلية.
CHAPTER ONE

Introduction and Literature Review

1.1 Introduction:

Periodontitis is inflammation of the periodontium that extends beyond the gingiva and may lead to destruction of the connective tissue attachment of the teeth. It is no longer considered as a single disease. Periodontitis is now considered to exist in three primary forms; Chronic Periodontitis, Aggressive Periodontitis, and as a manifestation of systemic diseases and conditions. Among these conditions, chronic periodontitis is considered as the commonest form. (1)

Obesity is a major public health concern in both developed and developing countries. It has been defined by the WHO as “accumulation of excess body fat, to an extent that may impair health” (2). Obesity is considered a major risk factor for several conditions including diabetes mellitus, cardiovascular diseases including, hypertension, stroke, and osteoarthritis. (2)

Periodontal disease is one of commonest chronic diseases in the population across the world. Several systemic factors are associated with increased risk of periodontal diseases including smoking, diabetes, osteoporosis, stress, and age. (3)

It has been found that there is a significant association between obesity and increased risk of periodontal diseases. (3, 4)

Although the problem of periodontitis and other associated factors had been investigated in different studies in Sudan, no study has been conducted to investigate the relationship between periodontal diseases and obesity, for that reason this study was designed.
Justification

Periodontal diseases prevalence has always been considered to be low among Sudanese. In view of changing life style of the Sudanese population particularly dietary habits, different systemic diseases including, cardiovascular diseases and diabetes mellitus are on the increase, however obesity is also increasing. The association between cardiovascular diseases and diabetes mellitus on one hand and periodontal health on the other hand has been highlighted before; however no study has ever investigated obesity and periodontal health in Sudan. Hence such a study is highly needed to provide base line data.

Objectives of the study:-

General objective

– To study the periodontal health of a group of obese subjects in Khartoum State.

Specific objectives

1. To find any association between obesity and periodontal diseases.

2. To study the association between age, gender and frequency of tooth brushing and periodontal health status of obese subjects.

3. To find the association between measures of periodontal diseases and the Body Mass Index as a measure of obesity.
1.2 Literature Review

1.2.1 Obesity:-

Obesity is a condition in which the natural energy reserve stored in the fatty tissues of humans is increased to the point where it’s associated with certain health conditions or increased mortality, so it’s becoming of great public health concern.

Obesity and overweight are defined as an accumulation of excess body fat, to an extent that may impair health. Although obesity is an individual clinical condition, it is increasingly viewed as a serious and growing public health problem, since excessive body weight has been shown to predispose to various diseases, particularly cardiovascular diseases, diabetes mellitus type II, sleep apnea and osteoarthritis.\(^{(5)}\)

1.2.1.1 Measuring obesity:

In any particular setting, obesity is typically evaluated by measuring BMI (Body Mass Index) but also in terms of its distribution through waist circumferences (WC), or waist-hip circumferences ratio (WHCR).

1.2.1.2 What is the BMI:-

Body mass index is a number calculated from a person’s height and weight. BMI is a reliable indicator of body fatness for people, so calculating BMI is one of the best methods for population assessment of overweight and obesity.

Because calculations require just weight and height it is inexpensive and easy to use for clinical as well as for the general public.
The formula for calculating the BMI is as follows:

\[
\text{Weight (kg)} / [\text{height (m)}]^2 = \text{BMI}
\]

with the metric system the formula for BMI is weight in kilograms is divided by height in meters squared. Since height is commonly measured in centimeters. Divide height in centimeters by 100 to obtain height in meters.

US/Customary

\[
\text{BMI} = \frac{\text{lb - weight}}{\text{(inch)} \times \text{height}^2 \times 103}
\]

The standard weight status categories associated with BMI ranges for adults are shown in the following table: (6)

<table>
<thead>
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<th>BMI</th>
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<td>Below 18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5 → 24.9</td>
<td>Normal</td>
</tr>
<tr>
<td>25 → 29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>30 → 39.9</td>
<td>Obese</td>
</tr>
<tr>
<td>40 and above</td>
<td>Severely Or Morbidly Obese</td>
</tr>
</tbody>
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In clinical settings, one has to take into account race, ethnicity, lean mass muscularity, age and sex. BMI, overestimates body fat in persons who are very muscular (highly trained athletes may have a high BMI because of increased muscularity's rather than increase body fatness, BMI alone can not be used as a sole clinical and epidemiological predictor of obesity.
- At the same BMI women tend to have more body fat than men.
- At the same BMI older people on average tend to have more body fat than younger adults.

Body fat Measurement:

Another way to determine obesity is to assess percent body fat. Doctors and scientists generally agree that men with more than 25% body fat and women with more than 30% body fat are obese (7).

Waist Circumferences:

The absolute waist Circumferences is $\geq 102$ cm in men.

The absolute waist Circumferences is $\geq 88$ cm in women.

Waist hip Relation:

Waist hip ratio is $\geq 0.6$ for men and $\geq 0.85$ for women are also used as measures for central obesity. (7)

**1.2.1.3 Health Consequences of Obesity:**

A large number of medical conditions have been associated with obesity. They are categorized as being the result of either increased fat mass: - (osteoarthritis – obstructive sleep apnea – social stigma) or increased number of fat cells (diabetes, cancer, cardiovascular diseases, and non alcoholic fatty liver diseases) (8).

Mortality is increased with obesity. It has been considered that with a BMI of over 32 there is alteration in body response to insulin (insulin resistance), and increased tendency to thrombosis (9)
Central obesity which is characterized by high waist-hip ratio is an important risk factor for metabolic syndromes which may predispose to cardiovascular diseases, diabetes mellitus type II, high blood pressure, high blood cholesterol and triglyceride level combined hyperlipidemia). (10)

1.2.1.4 Causes of Obesity:

Most researchers have concluded that the combination of an excessive nutrient intake and sedentary lifestyle are the main causes of obesity in western societies in the last quarter of the 20th century. (11) Genetics may also play a role in the etiology of obesity but in combination to environmental factors. Various genetic conditions that feature obesity have been identified such as Prader-Willi syndrome. A 2007 study identified that FTO gene; heterozygote had 30% increased risk of obesity, while homozygote has 70% increased risk. (12) Medical illness such as physical and mental illness may predispose to obesity, hypothyroidism, Cushing's syndrome, growth hormone deficiency etc. (13) Smoking cessation is a known cause for weight gain, as nicotine suppresses appetite. Certain medications e.g. Steroids, atypical antipsychotic may also cause weight gain.

The role of bacteria colonizing the digestive tract in the development of obesity has recently become a subject of investigation. As bacteria participate in digestion of fatty acids and polysaccharides so alteration in the proportion of certain strains of bacteria may explain why certain people are more prone to weight gain than others. (14)
1.2.1.5 Therapy

The most recommended treatment for obesity is an energy limited diet and increased exercise. In studies, diet and exercise programs have consistently produced an average weight loss of approximately 8% of total body mass (excluding study drop-outs). While not all dieters will be satisfied with this outcome, studies have shown that a loss of as little as 5% of body mass can create large health benefits. A more intractable therapeutic problem appears to be weight loss maintenance. Of dieters who manage to lose 10% or more of their body mass in studies, 80-95% will regain that weight within two to five years, supporting the finding that the body has various mechanisms that maintain weight at a certain set point.

In a clinical practice guideline by the American College of Physicians, the following five recommendations were made (15):

1- People with a BMI of over 30 should be counseled on diet, exercise and other relevant behavioral interventions, and set a realistic goal for weight loss.

2- If these goals were not achieved, pharmacotherapy can be offered. The patient needs to be informed of the possibility of side effects and the unavailability of long term safety and efficacy data.

3- Drug therapy may consist of sibutramine, orlistat, phentermine, diethylpropion, fluoxetine, and bupropion. For more severe cases of obesity, stronger drugs such as amphetamine and methamphetamine may be used on a selective basis. Evidence is not sufficient to recommend sertraline, topiramate, or zonisamide.

4- In patients with BMI ≥ 40 who fail to achieve their weight loss goals (with or without medication) and who develop obesity-related
complications, referral for bariatric surgery may be indicated. The patient needs to be made aware of the potential complications.

5- Those requiring bariatric surgery should be referred to high-volume referral centers, as the evidence suggests that surgeons who frequently perform these procedures have fewer complications.

A clinical practice guideline by the US Preventive Services Task Force (USPSTF) concluded that evidence is insufficient to recommend for or against routine behavioral counseling to promote a healthy diet in unselected patients in primary care settings, but that intensive behavioral dietary counseling is recommended in those with hyperlipidemia and other known risk factors for cardiovascular and diet related chronic diseases. Intensive counseling can be delivered by primary care clinicians or by referral to other specialists, such as nutritionists or dietitians. (16), (17)

Counseling:

A meta-analysis of randomized controlled trials concluded that "compared with usual care, dietary counseling interventions produce modest weight losses that diminish over time." (18)

Diets:

Various dietary approaches have been proposed, some of which have been compared by randomized controlled trials: A comparison of Dr. Atkins', Slim-Fast's, Weight Watchers', and Rosemary Conley's diets found no significant differences. (19)

A comparison of Atkins diet, Zone diet, Weight Watchers, and Ornish diet noted (20) "All four diets tested resulted in modest statistically significant weight loss at one year, with no statistically significant differences between different diet regimens"
"The higher discontinuation rates for the Atkins and Ornish diet groups suggest that many individuals found these diets to be too extreme"

Low carbohydrate versus low fat:

Many studies have focused on diets that reduce calories via a low-carbohydrate diet (Atkins diet, Zone diet) versus a low fat diet. The Nurses' Health Study, an observational cohort study, found that low carbohydrate diets based on vegetable sources of fat and protein are associated with less coronary heart diseases.\(^{(21)}\)

A meta-analysis of randomized controlled trials by the international Cochrane Collaboration in 2002, concluded that fat-restricted diets are no better than calorie restricted diets in achieving long term weight loss in overweight or obese people.

The Women's Health Initiative Randomized Controlled Dietary Modification Trial,\(^{(22)}\) found that a diet of total fat to 20% of energy and increasing consumption of vegetables and fruit to at least five servings daily and grains to at least six servings daily resulted in:

- No reduction in cardiovascular diseases\(^{(23)}\)

- An insignificant reduction in invasive breast cancer.\(^{(24)}\)

- No reductions in colorectal cancer. \(^{(25)}\)

However additional recent randomized controlled trials have found that in young adults "Reducing glycemic [carbohydrate] load may be especially important to achieve weight loss among individuals with high insulin secretion."\(^{(26)}\) This is consistent with prior studies of diabetic patients in which low carbohydrate diets were more beneficial.\(^{(27)}\)
Glycemic index:

"The glycaemic index factor is a ranking of foods based on their overall effect on blood sugar levels. Low glycaemic index foods, such as lentils, provide a slower more consistent source of glucose to the bloodstream, thereby stimulating less insulin release than high glycaemic index foods, such as white bread." (28)

1.2.2 The Normal Periodontium:

The normal periodontium consists of the investing and supporting tissues of the tooth (gingiva, periodontal ligament, cementum, and alveolar bone). It has been divided into two parts: the gingiva, whose main function is protection of the underlying tissues, and the attachment apparatus, composed of the periodontal ligament, cementum, and alveolar bone. (29,30). Cementum is considered part of the periodontium because, with bone, it serves as the support for the fibers of the periodontal ligament.

The oral mucosa consists of three zones: the gingiva and the covering of the hard palate, termed the masticatory mucosa; the dorsum of the tongue, covered by the specialized mucosa; and the oral mucous membrane lining the remainder of the oral cavity. The gingiva is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth.

The marginal, or unattached, gingiva is the terminal edge or border of the gingiva surrounding the teeth in collar like fashion. In about 50% of cases, it is demarcated from the adjacent, attached gingiva by a shallow linear depression, the free gingival groove (31). (Usually about 1 mm wide, it forms the soft tissue wall of the gingival sulcus. It may be separated form the tooth surface with a periodontal probe.)
The gingival sulcus is the shallow crevice or space around the tooth bounded by the surface of the tooth on one side and the epithelium lining the free margin of the gingiva on the other. It is V shaped and barely permits the entrance of a periodontal probe. The clinical determination of the depth of the gingival sulcus is an important diagnostic parameter. Under absolutely normal or ideal conditions, the depth of the gingival sulcus is or is about 0. These strict conditions can be produced experimentally only in germfree animals or after intense, prolonged plaque control. (32, 33)

The attached gingival is continuous with the marginal gingiva. It is firm, resilient, and tightly bound to the underlying periosteum of alveolar bone. The facial aspect of the attached gingiva extends to the relatively loose and movable alveolar mucosa, from which it is demarcated by the mucogingival junction.

The interdental gingiva occupies the gingival embrasure, which is the interproximal space beneath the area of tooth contact. The interdental gingiva can be pyramidal or have a "col" shape. In the former, the tip of one papilla is located immediately beneath the contact point; while the latter presents a valley like depression that connects facial and lingual papillae and conforms to the shape of the interproximal contact. (34)

The attachment apparatus of the tooth includes the periodontal ligament, cementum, and alveolar bone.

The periodontal ligament is the connective tissue that surrounds the root and connects it to the bone. It is continuous with the connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone. (35, 36)

Cementum is the calcified avascular mesenchymal tissue that forms the outer covering of the anatomic root. The two main types of
cementum are Acellular (primary) and Cellular (secondary) cementum (37). Both consist of a calcified interfibrillar matrix and collagen fibrils. The two sources of collagen fibers in cementum are Sharpey's (extrinsic) fibers, which are the embedded portion of the principal fibers of the periodontal ligament (38), and are formed by the fibroblasts, and fibers that belong to the cementum matrix per se (intrinsic) and are produced by the cementoblasts. (39) Cementoblasts also form the noncollagenous components of the interfibrillar ground substance, such as proteoglycans, glycoproteins, and phosphoproteins.

Acellular cementum is the first to be formed and covers approximately the cervical third or half of the root; it does not contain cells.

Cellular cementum, formed after the tooth reaches the occlusal plane, is more irregular and contains cells (cementocytes) in individual spaces (lacunae) that communicate with each other through a system of anastomosing canaliculi. Cellular cementum is less calcified than the acellular type (40).

The alveolar process is the portion of the maxilla and mandible that forms and supports the tooth sockets (alveoli). It forms when the tooth erupts to provide the osseous attachment to the forming periodontal ligament; it disappears gradually after the tooth is lost.

The alveolar process consists of the following:

1. An external plate of cortical bone formed by Haversian bone and compacted bone lamellae.

2. The inner socket wall of thin, compact bone called alveolar bone proper, which is seen as the lamina dura in radiographs. Histologically, it contains a series of openings (cribriform plate)
through which neurovascular bundles link the periodontal ligament with the central component of the alveolar bone, the cancellous bone.

3. Cancellous trabeculae, between these two compact layers, which act as supporting alveolar bone. The interdental septum consists of cancellous. Supporting bone enclosed within a compact border.

In addition, the jaw bones consist of the basal bone which is that portion of the jaw located apically but unrelated to the teeth.

1.2.2.1 Diseases of the Periodontium:

Diseases affecting the periodontium are collectively called periodontal diseases. Periodontal diseases are one of the most prevalent chronic diseases affecting humans. It has been estimated that at least 35% of the United States adults have periodontal diseases (41).

Periodontitis is inflammation of the periodontium and produces destruction of the connective tissue attachments of teeth, of the three forms of periodontitis (chronic periodontitis, aggressive periodontitis, and periodontitis as a manifestation of systemic diseases), chronic periodontitis is the most common form.

Periodontal diseases are a group of infectious diseases caused by predominantly gram negative bacteria. (42), periodontal disease refers to both gingivitis and periodontitis. Periodontitis involves the destruction of the supporting structures of the teeth including the periodontal ligaments, bone and soft tissue and is the most significant of these because it may cause tooth loss. (43)
The presence of bacteria in the oral cavity has been known since the time of Anton von Leeuwenhoek, as reported by Tatakis and Kumar (44) (2005), who described the presence of "animalcules" in dental plaque. The bacterial etiology of periodontal diseases has been explored for over 100 years. However, the identification of specific causative species has been hampered by some of the unique features of periodontal diseases. The foremost of these features is that disease occurs in a site already colonized by a bacterial population. Thus, disease might be caused by overgrowth of one or more species in the resident population or by colonization by exogenous pathogens. (44)

The plaque biofilm consists largely of microbes and host proteins that adhere to the teeth within minutes of a dental prophylaxis (4,5). No one knows how many bacterial species coexist in the dental plaque, but the number is very large. (46). Some of the most common organisms associated with periodontal diseases are Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus, Campylobacter rectus, Actinobacillus actinomycetemcomitans and Treponema denticola. (41,43). Microbial biofilm is the etiological factor of periodontal diseases. Supragingival plaque contains many bioactive end products, such as fermented organic acid, sulfur component, tissue digesting enzymes, peptidoglycans and lipopolysaccharides. These components are diffused from supra-gingival plaque to the surface of gingival epithelium and increase the flow of crevice fluid and inflammatory fluid from periodontal tissues. This new nutritional supply, which is delivered from serum, changes the ecosystem of the plaque adjacent to the inflamed gingiva. In this new environment, proteolytic bacteria in the plaque expand their ecologic niche and produce proteases, which accelerate tissue damage. (47) The junctional epithelium acts as a gate keeper,
selectively allowing the passage of antigens and cells as well as producing a range of defensive molecules. Once the epithelial barrier with its antimicrobial peptides is breached, adaptive immune response comes into play. Cytokines are central to this response, such that production of "appropriate" cytokines results in development of protective immunity and production of "inappropriate" cytokines leads to tissue destruction. Exactly how the immune system chooses these cytokines is unclear, although the genetic factors are most likely involved.\(^{(48)}\)

In a review, Loesche and Grossman\(^{(46)}\) (2001), concluded that most, if not all, forms of periodontal diseases are specific, albeit chronic infections. Regardless of whether the host is genetically predisposed to periodontal disease, or if the host is compromised by leukocyte defects, or diabetes, or is a smoker, or has poor oral hygiene, the clinical symptoms are almost always associated with the overgrowth of a finite number of anaerobic species.

### 1.2.2.2 Chronic Periodontitis: -

Gingival inflammation takes place as a result of bacterial challenge from dental plaque. (Being the primary etiological factor for gingivitis and periodontitis)

Chronic periodontitis is a slowly progressing disease, however in the presence of systemic or environmental factors that may modify the host response to dental plaque such as diabetes, smoking and stress the disease progression may become aggressive.
Chronic periodontitis has recently been defined as "an infectious disease resulting in inflammation within the supporting structure of teeth, progressive attachment loss, and bone loss" (49).

This definition outlines the major clinical and etiological features of the disease. Periodontal pocket formation is a common sequel of the disease which is pathological deepening of the gingival sulcus. The most characteristic clinical findings in patients with chronic periodontitis include supragingival and subgingival plaque accumulation, gingival inflammation, pocket formation and loss of periodontal attachment and bone loss.

Chronic periodontitis is clinically diagnosed by the chronic inflammatory changes in the marginal gingiva and radiographically by evidence of bone loss based on age of the patient, rate of disease progression over time, family history and presence of local factors.

Chronic periodontitis can be considered as localized when < 30% of sites assessed in the moth demonstrate attachment loss and bone loss, and it can be considered generalized periodontitis when >30% of the assessed sites represent attachment loss and bone loss.

The severity of chronic periodontitis can be described as being slight (mild), moderate, or severe according to the following criteria:

- **Slight (Mild)** periodontal destruction is when 1-2 mm of clinical attachment loss has occurred.
- **Moderate** when 3-4 mm of clinical attachment loss has taken place.
- **Severe** periodontitis when 5 mm or more of clinical attachment loss has taken place.
Risk factors for chronic periodontitis:

Local Factors:

Plaque accumulation on teeth and gingival surfaces is considered the primary initiating factor in the etiology of chronic periodontitis.

Systemic Factors:

The rate of progression of plaque-induced chronic periodontitis is considered to be slow; however, when it occurs in patients who suffer from systemic diseases that modify host response. The rate of periodontal destruction is significantly increased. For example, diabetes mellitus type II. Non Insulin dependent Diabetes mellitus (NIDDM) is the most prevalent form of diabetes and account for 90% of diabetic patients. An increase in type II diabetes mellitus in teenagers and young adult has been observed and may be associated with an increase in juvenile obesity and periodontal diseases.

Environmental factors:

Smoking has been shown to increase the severity of periodontal diseases. Emotional stress also may influence the extent and severity of chronic periodontitis through its effect on immune function.

Genetic Factors:

Genetic predisposition through family members has been found, suggesting the possibility of some genetic basis for periodontal diseases.

1.2.2.3 Aggressive periodontitis:

Aggressive periodontitis is characterized by a young age of disease onset and severe periodontal tissue destruction. Meng et al. have reviewed the determinants of host susceptibility in aggressive
periodontitis, focusing on family aggregation, single nucleotide polymorphisms, polymorph nuclear leukocytes, antibodies, and smoking, stress and root morphology abnormalities. They suggest that increased host susceptibility in patients with aggressive periodontitis may be caused by the combined effect of multiple genes interacting with environmental factors.

1.2.2.4 PERIODONTAL DISEASES AND SYSTEMIC DISEASES:

The concept that oral infections, such as chronic inflammatory periodontitis, may contribute to various systemic diseases is not new. Indeed, the possibility that a localized or focal infection could have systemic effects was a popular idea at the turn of the last century. A theory of focal infection developed had proposed that local "foci" of infection were responsible for the initiation and progression of various inflammatory conditions, such as arthritis, appendicitis and peptic ulcers. A focal infection is a chronic, localized infection that can disseminate microorganisms or toxic microbial products to contiguous or distant tissue and can adversely affect the distant target organs.

A symposium at North Carolina Chapel Hill entitled 'Periodontal diseases and human health: New edition in periodontal medicine' advanced the notion that oral infections may have powerful and multiple influences on the occurrence and severity of systemic conditions and diseases. Since then numerous publications in dental and medical journals have focused on the relationship between periodontal disease and systemic conditions, especially cardiovascular diseases, diabetes mellitus and respiratory pathosis. Progress in this field within the last few years has been tremendous. High serum antibody levels to Porphyromonas
gingivalis predict myocardial infarction, antibodies to periodontal pathogens and stroke risk. (56) 'Early carotid atherosclerosis in subjects with periodontal diseases'. (57) 'Gender differences in the relationship between periodontal diseases tooth loss and atherosclerosis'. (58) Periodontal diseases and biomarkers related to cardiovascular diseases. (59) 'Periodontal disease and mortality in type II diabetes' (60) and 'oral bacteria in the occluded arteries of patients with Buerger disease' (61) are titles of published articles in medical journals, which have identified a positive correlation between periodontal disease and systemic diseases. Stamm (54) mentioned that practitioners of obstetrics, cardiovascular disease and periodontics are collaborating in scientific progress in ways not previously imagined.

Recently, it has been suggested that herpes viruses comprise an important source for triggering periodontal tissue destruction. Herpes virus infections may initiate or accelerate periodontal breakdown via their ability to stimulate cytokine release from host cells, or they might impair host defense mechanisms, resulting in heightened virulence of resident periodontopathic bacteria. (62, 63)

Microbial antigens and virulence factors elicit an immediate inflammatory and immune response from the host. The host reacts to microbial insults by producing cytokines, kinins, complement activation products and matrix metalloproteinase. Some of these inflammatory mediators participate in periodontal ligament and bone destruction. In turn, the inflammatory response is regulated by genetic and environmental modifiers. As reported by Page et al, (64) bacteria are essential in, but not sufficient to cause, periodontitis, and host factors, such as heredity, and environmental factors, such as smoking, are important determinants of periodontal disease occurrence and severity.
Bacteria may cause periodontal tissue destruction indirectly by activating various components of the host defense system. When activated, these host systems may provide protection at the cost of some level of periodontal destruction. Periodontal disease researchers have extensively studied this double-edged sword phenomenon, and the concepts thus derived consider the involvement of intrinsic and induced host factors. In addition, cell specific receptors and their pathways have been identified.

In order to investigate the pathogenesis of periodontitis, it is essential to understand how the immune system reacts towards microbial infections. To familiarize readers with this topic, Mahanonda and Pichyangkul, \(^{(65)}\) published an article on the fundamental mechanisms of host responses to bacterial and viral infections \(^{(65)}\). The authors concentrated on two topics: the cross-talk between innate and adaptive immunity mediated by dendritic cells via Toll-like receptors, and the antigen specific immunoregulation by particular subsets of dendritic cells and T cells. T cells play an important immunoregulatory role in the pathogenesis of periodontal diseases. In a separate article dealing with T cells in periodontal diseases, Gemmell et al, \(^{(66)}\) suggest that T cells have a homeostatic role and that autoimmunity is an important component of chronic periodontal inflammation. They discuss various mechanisms by which T cells are involved in the destruction and repair processes of chronic periodontitis. In their article, the authors had advanced the concept that T cells have a homeostatic role, rather than defensive or destructive role in periodontal diseases \(^{(66)}\).

Periodontitis is characterized by destruction of the periodontal ligament and bone. Alveolar bone resorption occurs as a result of uncoupling of the normally balanced processes of bone resorption and bone formation. Cytokines, chemokines and prostaglandins have been
identified as regulators of the immune inflammatory process in periodontitis. Activated lymphocytes, macrophages and neutrophils infiltrate inflamed gingival tissue and secrete inflammatory mediators including interleukin-1 and prostaglandin E2. In addition, T helper 1 and T helper 2 lymphocytes, which are present in periodontal lesions, up-regulate the production of the pro-inflammatory interleukin-1 and tumor necrosis factor-α. These cytokines can induce bone resorption indirectly by promoting the differentiation of osteoclast precursors and by activating osteoclasts. Udagawa et al. (67) conducted a study on signal transduction of lipopolysaccharide induced osteoclast differentiation. Lipopolysaccharide, being the major constituent of gram negative bacteria, is proposed to be a potent stimulator of bone resorption in inflammatory periodontal disease (67). Prostaglandin E2 stimulates pro-inflammatory responses, including osteoclastogenesis, by enhancing the expression of receptor activator of nuclear factors. However, prostaglandin E2 also has anti-inflammatory effects, including inhibiting the production of pro-inflammatory cytokines, such as tumor necrosis factor, and can elicit anabolic actions on bone. An article by Noguchi and Ishikawa (68) discussing the periodontal roles of prostaglandin E2 and cyclo-oxygenase-2, has been included. The authors suggest that prostaglandin E2 has the ability to down regulate hyper-inflammation occurring in periodontal lesions, and to stimulate wound healing.

Similarly to environmental factors, host factors, such as heredity, are a major determinant of infectious disease occurrence and severity. With recent advances in the science of genetics, genes responsible for various diseases have been identified and mapped. Genetic traits that may be associated with an increased risk for periodontitis include abnormal phagocyte function, tumor necrosis factor-α polymorphism, interleukin-1 α polymorphism and prostaglandin. (64) Yoshie et al, (69) have reviewed
the possible role of genetic polymorphisms in periodontitis. In addition to listing the evidence linking genetics and aggressive and chronic forms of periodontitis, the authors have discussed the polymorphisms identified in the cytokine family and receptor genes, and addressed the issues and concerns about the candidate gene approach of study in periodontitis. Oxidative stress has been implicated in periodontitis, obesity, type II diabetes mellitus, cardiovascular diseases and the events that lead to adverse pregnancy outcome. In recent years, reactive oxygen species have also been implicated in the pathogenesis of periodontitis. The overproduction of reactive oxygen species may be a key component of hyper-inflammation associated with periodontitis. Chapple and Matthews (70) explored the role of reactive oxygen and antioxidant species in periodontal tissue destruction.

Among the environmental factors known to affect the incidence and severity of periodontitis, smoking stands out as one of the most significant and pre-ventable risk factors. Tobacco smoking increases the risk of periodontal destruction by stimulating inflammatory responses and impairing protective and reparative host responses. In an elaborate review, Ryder (71) deals with the influence of smoking on the immune response in periodontal disease. He suggests that host modulating agents, such as tetracycline, may partly alleviate the destructive effects of tobacco products on host responses. Research in the field of tobacco smoking and its effects on the host immune system may also help to identify host modulating agents that may be beneficial in the treatment of periodontal disease in both smokers and non smokers. Diabetes is an extensively researched risk factor for periodontitis. Diabetic subjects are known to respond to microbial challenges in an exaggerated manner compared with non diabetic subjects. Exaggerated inflammatory responses in diabetic subjects appear to increase the risk for vascular
disorders. Nassar et al.\(^{(72)}\) have proposed the interesting hypothesis that long term hyperglycemia supports anaerobic infection in periodontal sites owing to an environment of exacerbated innate immunity. Persistent hyperglycemia may lead to chronically activated innate immunity and chronic inflammation in the periodontium by either blocking or suppressing pathways of resolution. Nassar et al.\(^{(72)}\) examined the role of innate immunity, inflammation and resolution of inflammation as essential components in the development of diabetic complications, including periodontal disease. They also proposed several therapeutic approaches, including host modulation therapy. In a separate study, Nishimura et al.\(^{(73)}\) summarized the current knowledge on the bidirectional relationship between diabetes mellitus and periodontal diseases, and discuss future strategies for the prevention and treatment of periodontal diseases in diabetic subjects. Periodontal diseases have been considered to be the sixth complication of diabetes mellitus.

There is substantial evidence that the relationship between periodontal diseases and systemic diseases may be bidirectional. That is, not only the systemic conditions have oral manifestations, but periodontal diseases can also affect certain systemic conditions. It has been found that people with periodontal diseases have higher levels of circulating bacterial pro-inflammatory components, compared to people with healthy gums\(^{(52,53,74)}\). The local inflammatory response to these bacterial products is the infiltration of periodontal tissues by the inflammatory cells including polymorphonuclear neutrophils, lymphocytes, macrophages and plasma cells. Activated macrophages release cytokines which can result in local and systemic elevation of pro-inflammatory mediators such as IL 1, IL6, PGE2 and TNF-\(\alpha\).\(^{(75,76,77,78)}\)
Elevated levels of these cytokines can also initiate a systemic acute phase response (APR). The acute phase response (APR) represents an early and highly complex reaction of an organism to a variety of injuries such as bacterial, viral or parasitic infection; mechanical or thermal trauma, ischemic necrosis or malignant growth. The acute phase refers to physiological and metabolic alterations that ensue immediately after the onset of infection or tissue injury the hepatic synthesis and intravascular secretion of many plasma proteins with a wide range of actions is also increased. The components of the acute phase response are largely non-specific as compared to cellular or humoral immunity and purpose of these responses is to restore homeostasis and to remove the cause of disturbance. Characteristic features of systemic acute phases response include fever, neutrophilia, altered lipid metabolism, increased gluconeogenesis, increased (muscle) protein catabolism and transfer of amino acids from muscles to liver activation of complement and coagulation pathways and induction of acute phase proteins.

The major acute phase proteins include C-reactive proteins (CRP), serum amyloid A, fibrinogen and hepatoglobin, whose concentrations increases with inflammation, and albumin and transferrin whose concentrations decreases with inflammation. The serum C-reactive protein concentrations closely follow the course of the acute phase response to inflammation; therefore its measurement can provide a valuable and timely barometer for many disease processes.

Proinflammatory cytokines and mediators are significantly elevated with gingival inflammation during destructive periodontitis. Cytokines appears to play a major role in the clinical symptoms and tissue destruction with progressive periodontitis. There is also strong
evidence for cytokines eliciting the systemic acute phase response in various inflammatory conditions.\(^{(79)}\)

From many studies on the systemic effects of serum pro-inflammatory cytokine levels potentially elevated by periodontitis, researchers have hypothesized that periodontitis induced elevation of IL-1 and TNF-\(\alpha\) may play a major role in the development of a variety of systemic conditions and diseases. In fact some studies have shown that in advance periodontitis, the levels of IL-1 and TNF-\(\alpha\) are sufficiently elevated in gingival crevicular fluid to be detectable systemically by biological serum assay. Thus patients with advanced periodontitis could be considered systemically compromised even in the absence of overt clinical symptoms of disease.\(^{(74, 79, 82)}\)

**Periodontal diseases and cardiovascular diseases**:

Coronary heart disease is the leading cause of adult mortality and morbidity throughout the world.\(^{(83)}\) The major contributing factor in the majority of the cases of cardiovascular disease and cerebrovascular disease (stroke) is atherosclerosis.\(^{(84)}\) Hypercholesterolemia, dyslipidemia, obesity, hypertension, diabetes mellitus and smoking are well established risk factors for atherosclerosis and its complications.\(^{(89)}\). One of the outcomes of atherosclerotic process is the narrowing of the arteries resulting from subendothelial deposition of cholesterol, cholesterol esters and calcium within the vessel wall.\(^{(84)}\) The cause of acute coronary syndrome is the destabilization of pre-existing atherosclerotic plaque resulting in clot formation on the surface of plaque. Inflammation within the atheromatous plaque is a factor which may lead to its destabilization\(^{(85)}\).
Chronic infections, including dental infections have been linked to increased risk for cardiovascular diseases. (86) The research documenting the association between periodontal diseases and cardiovascular diseases is relatively recent, with the first study published in 1989. (87) Several mechanisms have been proposed to explain such an association. One of them is based on the potential for the inflammatory phenomenon of periodontitis to have effects by the systemic dissemination of locally produced mediators such as C-reactive protein (CRP), interleukin 1beta(IL-1β) and IL-6 and tumor necrosis factor alpha (TNF-α).

Elevated levels of CRP have recently gained special attention as a risk factor for cardiac and cerebrovascular events. Elevated levels of IL-6 have also been associated with unstable angina. (65).

Majority of the studies published have suggested that periodontitis may be associated with cardiovascular events. (88) In a recent case control study, Geerts et al (89) (2004), concluded that periodontitis is a significant risk factor for cardiovascular disease after adjusting for other confounding factors. Similarly, Czerniue et al (85) (2004), found that the periodontal status of patients admitted to the coronary care unit due to acute coronary syndrome was unacceptable.

Beck and Offenbacher, (87) in their review article published in 2001, suggested that at that time there is no enough evidence to state that periodontal infection is a cause of cardiovascular disease. On the other hand, in a recent systematic review, Scannapieco et al (84) (2003), concluded that periodontal disease appears to be moderately associated with atherosclerosis induced diseases such as coronary artery disease, stroke and peripheral vascular disease. Although the extent to which the initiation and/or progression of atherosclerosis is influenced by periodontal infection is presently unknown.
Periodontal diseases and diabetes mellitus:

Diabetes mellitus is a metabolic derangement characterized by impairment in glucose use. Diabetes mellitus occurs in two major forms; type 1 diabetes mellitus which is the result of a reduction in or the elimination of insulin production by beta cells in the pancreas. While type II diabetes mellitus results from defects in the insulin molecules or from altered insulin cell receptors and represent the impaired insulin function.

Periodontal diseases are a complication of diabetes mellitus. The presence of periodontal disease in a diabetic patient is a serious health hazard, which will lead to more severe diabetes related complications. The association between diabetes mellitus and periodontitis has long been discussed. Most of the early studies tended to consider the relationship as unidirectional, noting a higher incidence of periodontitis in diabetic patients. More recent work has reported the converse relationship. Recent evidence suggests that chronic subclinical inflammation play an intermediary role in the pathogenesis of type II diabetes. Elevated levels of the inflammatory markers, such as C reactive protein (CRP), interleukin-6 (IL-6) and tumor Necrosis factor-α (TNF-α) are reported to be significant risk indicators of type II diabetes.

Several recent studies have suggested that oral infections, particularly those associated with destructive periodontal diseases may induce elevated serum levels of C-reactive proteins and other pro-inflammatory mediators. Engebretson et al (2004), reported that poor glycemic control is associated with elevated levels of IL-1 β in gingival crevicular fluid.
Many studies have been conducted to see the effect of periodontal therapy on glycemic control in type II diabetes mellitus patients. In a recent study, Kiran et al (94) (2005), concluded that non surgical periodontal therapy is associated with improved glycemic control in type II diabetic patients. Similarly Rodrigues et al (90) (2003), showed in their study that effective periodontal treatment resulted in lower glycemic levels. Lwamoto et al (92) (2001), evaluated the effect of antimicrobial periodontal treatment on circulating TNF-α and glycated hemoglobin levels in patients with type II diabetes and found that anti infectious treatment is effective in improving the metabolic control in diabetics possibly through reduced serum TNF-α and improved insulin resistance.

Periodontal diseases and adverse pregnancy outcomes:

Preterm delivery and low birth weight is one of the major social and economic public health problem not fully explained by the currently established risk factors (95). Preterm delivery is the most significant cause of neonatal mortality and long term health problems including respiratory distress syndrome, cerebral palsy, pathologic heart conditions, epilepsy and severe learning problems. (95, 96) Despite the significant improvement in medical care and vigorous attempts to reduce preterm birth by wide spread use of drugs and public health interventions, the preterm birth rate have not declined but instead have increased over the last two decades. (95, 97)

Offenbacher et al 98, were the first to report that periodontitis was a possible risk factor for preterm low birth weight. After adjusting for other known risk factors they suggested that pregnant women with periodontitis were at a higher risk of delivering a preterm low birth weight infant as compared to women without periodontitis.
Medianos et al (99) (2002), hypothesized that translocation of periodontal pathogens to foetal-placental unit induce a maternal and/or foetal response that results in preterm birth. The bacteria involved in chronic periodontal inflammation are similar to those found in women with bacterial vaginosis. (99)

The potential of *C. rectus* and *P. gingivalis* in mediating the adverse pregnancy outcomes was recently studied in a mouse model. In a most recent study by Yeo et al (100), (101) (2005), on pregnant mice, the authors concluded that remote subcutaneous maternal infections with *C. rectus* increase foetal resorption and foetal growth restrictions.

Maternal serum levels of some cytokines have been reported to be associated with preterm low birth weight. Such cytokines include interleukin 6 (IL-6) and tumor necrosis factors, showed poorer periodontal condition and elevated serum levels of interleukin-8 as compared to controls. Lopez et al (102), in one of their study conducted in (2002), suggested that inflamed periodontal tissues produce significant amounts of pro-inflammatory cytokines including interleukin 1 beta, interleukin-6 (IL-6), prostaglandin E2 (PGE2) and tumor necrosis factor alpha (TNF-α). In the most recent study by Dortbudak et al (103) (2005), concluded that pregnant women with elevated levels of PGE2, IL-6, and IL-8 in their amniotic fluid in the 15-20 weeks of pregnancy and with periodontitis are at a higher risk for preterm birth.

Most of the recent studies on the possible relationship between periodontal diseases and risk of preterm low birth weight infants suggest periodontal disease as a risk factor for preterm low birth weight deliveries. (98, 99, 103, 104) On the other hand Noack et al (96) (2005), concluded that periodontitis was not a detectable risk factor for preterm
low birth weight in pregnant women in the population they included in their study.

In conclusion, it can be summarized that although a number of studies suggest that periodontal infections are associated with systemic diseases but the exact mechanism by which periodontal diseases have systemic effects still remains unclear and speculative. Therefore, such periodontal systemic associations must be investigated through large, prospective randomized clinical studies as well as interventional studies.

1.2.2.5 Obesity and Periodontal diseases:

Researchers in a recent study on apparently healthy Japanese adults found a significant association between obesity and increased risk of periodontitis, the authors carried out a study in the United State population to examine whether or not there is an association between obesity and periodontal diseases using the Body Mass Index and waist circumferences. The authors found a significant association between the measures of body fat and periodontal diseases among younger adults. They also concluded that obesity is a major contributor to the development of type II diabetes mellitus, and in the United Stats it is increasing at an alarming rate. The number of diagnosed cases of diabetic patients has increased 30% in less than 10 years.

Another study was performed among 241 healthy Japanese subjects 20-59 years of age who attended a health promotion program at Fukuoka Health Promotion Center in Japan from May 1995 to April 1997. The authors examined the periodontal health status by using the Community Periodontal Index for Treatment Needs (CPITN) and the
Body Mass Index (BMI) to measure obesity. The authors of the study concluded that obesity and periodontitis are strongly related and that the later may be exacerbated by some conditions associated with obesity e.g. metabolic syndrome and insulin resistance diabetes.\(^{(105)}\)

Another study was done among 706 subjects aged 30-65 years from south Brazil. The subjects were examined clinically for obesity and periodontal diseases. The authors found that obesity was significantly associated with periodontitis. They also reported that in adults, smoking may attenuate the association of periodontitis to obesity.\(^{(106)}\)

Recent studies suggest that obesity is associated with periodontal disease. Obesity increases the risk of various systemic diseases and of type II diabetes mellitus, and raises patient mortality. Conversely, periodontal disease has been reported to influence the serum glucose level, not only in diabetic patients but also in non diabetic subjects. Taking account of all these issues, the relationship among obesity, diabetes and periodontal diseases is complicated and needs to be further evaluated. Saito and Shimazaki,\(^{(105)}\) have reviewed the relationship between obesity and periodontal diseases. The influence of periodontal diseases on obesity related metabolic disorders, such as glucose and lipid metabolism, and associated glucose intolerance and dyslipidemia were highlighted.\(^{(106)}\)

Al-Zahrani et al. (2003), conducted a study among a USA general population, in the Third National Health and Nutrition Examination Survey (NHANES 111). The subjects were both males and females and the age range was 18-34 years. The authors used the Body Mass Index as a measure for obesity, and the Probable Pocket Depth and Clinical Attachment Loss as measures for periodontitis. The authors concluded that the relationship between parameters of obesity and insulin resistance
with the parameters of periodontal diseases were significantly associated. (107)

Wood et al. (2003), conducted a cross sectional study among participants in the Third National Health and Nutrition Examination Survey (NHANES 111), in which they compared the Body Mass Index with parameters of periodontal diseases using nutritional techniques. A significant association was found between the variables of the two conditions. (108)
CHAPTER TWO

MATERIAL AND METHODS

2.1 Study Population:

All obese subjects aged between 18-35 years available at the study areas during the examination period, and who fulfilled the inclusion criteria were included in the study.

2.2 Study Area:

The subjects were selected from two fitness centers. The two centers are:

1. The Police Club Fitness Center.

II. The Army Club Fitness Center.

2.3 Study Design:

This is a cross sectional study.

2.3.1 Sample Size:

The sample size was determined by a statistician using the following equation:-

\[ N = \frac{Z \times Z \times (P \times (1-P))}{D \times D} \]

Where:

- **Z**: The value of reference normal distribution for the desired confidence interval (Z= 1.96 for the 95% CI).
- **P**: The expected prevalence.
- **D**: The highest acceptable error in the estimate (half width of CI measurement of precision).
In this study the expected prevalence was obtained from a study conducted in a similar country which was 43%. Accordingly N was calculated to be 188 subjects.

Adding the 10% compensation N will be 188+18= 206.

N= 206 subjects.

2.4 **Inclusion Criteria:**

1- Obese subjects who were apparently healthy without any systemic conditions.
2- Both genders were selected.
3- Age range between 18- 35 years.

2.5 **Exclusion Criteria:**

1- Non obese subjects.
2- Subjects with systemic conditions.
3- Subjects beyond the age range.

2.6 **Study Time Plan:**

The examination was done during the period June- September 2008.

2.7 **Ethical Approval:**

I- Ethical committee: The study was approved by the Faculty of Dentistry, University of Khartoum Postgraduate Research Board.

II- Informed consent was obtained from all the participants before the commencement of any clinical examination.

2.8 **Examination:** All subjects were examined under artificial light, on an ordinary chair using a William’s graduated periodontal probe and a plain
mouth mirror. The examination was carried out by one examiner (Hisham). Full mouth examination was done. Kappa analysis was performed on a group of subjects to check acceptable level of consistency to control the intraexaminer variability. Pre examination training and calibration with a trained Periodontist was done before the start of the study.

2.9 Questionnaire and Parameters Used:

For all subjects included in this study a precoded pretested structured questionnaire was done for collection of qualitative data consisting of questions on demographic data and lifestyle characteristics. In addition to the quantitative data collection, clinical examination was done using the following parameters:-

1- PLAQUE INDEX (PI) (Silness and Løe 1964):

According to the following criteria:

0 = Absence of plaque.

1 = Plaque seen after probing the gingival margin.

2 = Visible plaque.

3 = Abundance of plaque.

2- GINGIVAL INDEX (GI) (Løe and Silness 1963):

0 = Absence of signs of inflammation.

1 = Slight change in color and texture.

2 = Visual inflammation and bleeding on probing.

3 = Overt inflammation and spontaneous bleeding.
3-PROBABLE POCKET DEPTH (PPD) (IN mm)

Probable Pocket Depth was measured in mm as the distance from the gingival margin to the base of the periodontal pocket.

4-GINGIVAL RECESSION (IN mm)

Gingival Recession was measured in mm as the distance from the gingival margin to the cementoenamel junction.

5- CLINICAL ATTACHMENT LOSS (IN mm)

Clinical Attachment Loss was measured in mm as the distance from the cemento-enamel junction to the base of the sulcus.

2.10 Statistical analysis:-

The data was analyzed with the help of a biostatistician using the SPSS analysis, with the confidence interval (CI) of 95% and significant level of 5%. P value equal or less than 0.05 was considered as statistically significant.
CHAPTER THREE

3. Results

General Descriptive Data:

Two hundred and six obese subjects, 140 females and 66 males age range was 18 – 35 years attending the two fitness centers (Police, and Army Club Fitness Centers) were included in the study.

The examination was done during the period July 1st to September 6th 2008. All participants were obese with the BMI ≥30 and otherwise healthy. Pregnant ladies where excluded to avoid the effects of pregnancy on the periodontal health. Examination was done using a William’s graduated periodontal probe, mirror and artificial light. The examination was done by one examiner (Hisham).

The Prevalence of Gingivitis among the study group:-

The prevalence of gingivitis in this study was 96%. A total number of 198 subjects where shown to have various grades of the gingival index.

The prevalence of Periodontitis among the study group:-

The prevalence of periodontitis in this study was 62%. A total number of 129 subjects were shown to have either Probable Pocket Depth of ≥4 mm or Clinical Attachment Loss.
Distribution of the study group according to age:-

The mean and standard deviation of age in all obese subjects examined was 26.2±5.4 years, (Table 1).

Distribution of the study group according to the degree of obesity (The Body Mass Index) (BMI):-

According to the degree of obesity measured by the BMI the mean and standard deviation for participants was 34.4±3.6, (Table 2).

The mean Plaque Index among the study group:-

The mean and standard deviation of Plaque Index among all participants was 1.1±0.5  (Table 2).

The mean Gingival Index among the study group:-

The mean and standard deviation of Gingival Index among all participants was 1.4±0.5 (Table 2).

The mean Probable Pocket Depth among the study group:-

The mean and standard deviation of Probable Pocket Depth among all participants was 6.5±1.2 mm and the number of subjects having Probable Pocket Depth of ≥4 mm was 95 subjects (Table 2).
The mean Clinical Attachment Loss among the study group:-

The total number of subjects with Clinical Attachment Loss was 97 subjects. The mean and standard deviation of Clinical Attachment Loss was 8.7±1.3 mm (Table 2).

Correlation between Plaque Index and the Body Mass Index:-

A correlation was done between Plaque Index and the Body Mass Index (BMI). A significant association was found P =0.002 (Table 3).

Correlation between Gingival Index and the Body Mass Index:-

A correlation was done between Gingival Index and the Body Mass Index (BMI). A statistically significant association was found P=0.029 (Table 4).

Correlation between Clinical Attachment Loss and the Body Mass Index:

A correlation was done between Clinical Attachment Loss and the Body Mass Index. A statistically significant association was found P=0.000 (Table 5).
Correlation between Probable Pocket Depth and the Body Mass Index:

A correlation was done between Probable Pocket Depth and the Body Mass Index. A statistically significant association was found $P = 0.000$ (Table 6).

Correlation between Clinical Attachment Loss and different age groups:

The participants were divided into three groups according to age range:

Group 1  18 – 24 years  
Group 2  25 – 29 years  
Group 3  30 – 35 years  

The mean and standard deviation for Clinical Attachment Loss of each age group are seen in (Table 7).

To find the association of Clinical Attachment Loss and different age groups ANOVA test was done which showed a statistically significant association between Clinical Attachment Loss and Group 3 (30-35 years) $P = 0.000$

No association was found between Clinical Attachment Loss and Group 1 (18-24 years) and Group 2 (25-30 years). $P = 0.253$ (Table 7).
Correlation between Probable Pocket Depths and different age groups:

A correlation between Probable pocket depth and different age groups was done. It showed no significant association in Group 1 and 2 respectively \( P = 0.567 \).

A statistically significant association was found between Probable pocket depth and group 3 (30-35 years) \( P = 0.002 \) as seen in (Table 8).

Correlation between frequency of tooth brushing, Clinical Attachment Loss, and Probable Pocket Depth:-

The participants were divided into two groups:

Group 1 = Those who brush once a day.

Group 2 = Those who brush more than once.

A statistically significant association was found between frequency of brushing and Clinical Attachment Loss \( P = 0.003 \).

A statistically significant association was found between frequency of tooth brushing and Probable Pocket Depth \( P = 0.002 \) (Table 9).
Obesity and periodontal health in Khartoum State

Table (1)

Distribution of the study group according to age:-

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Magnitude (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean±SD</td>
<td>26.17±5.37</td>
</tr>
<tr>
<td>Median</td>
<td>27.00</td>
</tr>
<tr>
<td>Range</td>
<td>18-35</td>
</tr>
</tbody>
</table>

N=206 Subjects
Table (2)

Mean and standard deviation of PI (Plaque Index), GI (Gingival Index), PPD (Probable Pocket Depth), CAL (Clinical Attachment Loss), and the BMI (Body Mass Index) among the study sample.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>PI</td>
<td>1.1 ± 0.5</td>
</tr>
<tr>
<td>GI</td>
<td>1.4 ± 0.5</td>
</tr>
<tr>
<td>PPD</td>
<td>6.5 ± 1.2</td>
</tr>
<tr>
<td>CAL</td>
<td>8.7 ± 1.3</td>
</tr>
<tr>
<td>BMI</td>
<td>34.4 ± 3.6</td>
</tr>
</tbody>
</table>
Table (3)

Correlation between the Plague Index (PI) and the Body Mass Index (BMI):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No. (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>206 subjects 100(%)</td>
<td>0.002 (Statistically Significant)</td>
</tr>
<tr>
<td>PI</td>
<td>189 subjects 91.7(%)</td>
<td></td>
</tr>
</tbody>
</table>
Table (4)

Correlation between the Gingival Index (GI) and the Body Mass Index (BMI):-

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>206 subjects</td>
<td>0.029 (Statistically Significant)</td>
</tr>
<tr>
<td></td>
<td>100 (%)</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>198 subjects</td>
<td>96(%)</td>
</tr>
</tbody>
</table>
Table (5)

Correlation between Clinical Attachment Loss (CAL) and the Body Mass Index (BMI):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>206 subjects</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>100(%)</td>
<td>(Statistically Significant)</td>
</tr>
<tr>
<td>CAL</td>
<td>97 subjects</td>
<td>47(%)</td>
</tr>
</tbody>
</table>
Table (6)

Correlation between Probable Pocket Depth (PPD) and the Body Mass Index (BMI):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>206 subjects 100 (%)</td>
<td>0.000 (Statistically Significant)</td>
</tr>
<tr>
<td>PPD</td>
<td>95 subjects 46 (%)</td>
<td></td>
</tr>
</tbody>
</table>
Table (7)

Correlation between Clinical Attachment Loss (CAL) and different age groups:-

<table>
<thead>
<tr>
<th>Age group (Years)</th>
<th>No (%)</th>
<th>Mean± SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-24</td>
<td>35 subjects</td>
<td>16.9 (%)</td>
<td>2.7±1.5 mm</td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25-29</td>
<td>37 subjects</td>
<td>17.9 (%)</td>
<td>6.9±1.5 mm</td>
</tr>
<tr>
<td>Group 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-35</td>
<td>25 subjects</td>
<td>12.1 (%)</td>
<td>7.6±1.3 mm</td>
</tr>
</tbody>
</table>
Table (8)

Correlation between Probable Pocket Depths (PPD) and different age groups:

<table>
<thead>
<tr>
<th>Age group (Years)</th>
<th>No (%)</th>
<th>Mean ± SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>33 subjects</td>
<td>4.1±1.3</td>
<td>0.567</td>
</tr>
<tr>
<td>18-24</td>
<td>16.02 (%)</td>
<td>mm</td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>38 subjects</td>
<td>7.7±1.3</td>
<td>0.567</td>
</tr>
<tr>
<td>25-29</td>
<td>18.4 (%)</td>
<td>mm</td>
<td></td>
</tr>
<tr>
<td>Group 3</td>
<td>24 subjects</td>
<td>7.9±1.7</td>
<td>0.002</td>
</tr>
<tr>
<td>30-35</td>
<td>11.6 (%)</td>
<td>mm</td>
<td></td>
</tr>
</tbody>
</table>
Table (9)

Correlation between frequency of tooth brushing, Clinical Attachment Loss (CAL), and Probable Pocket Depth (PPD):

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency of tooth Brushing</th>
<th>Mean ± SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAL</td>
<td>Once</td>
<td>5.4±1.3 mm</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>&gt; once</td>
<td>7.3±1.4 mm</td>
<td></td>
</tr>
<tr>
<td>PPD</td>
<td>Once</td>
<td>6.00±1.3 mm</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>&gt; once</td>
<td>4.00±1.2 mm</td>
<td></td>
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</tbody>
</table>
CHAPTER FOUR  
Discussion, Conclusions and Recommendations

4.1 Discussion

A sample of 206 obese subjects who fulfilled the inclusion criteria and consented to examination, were examined for their periodontal health status.

All confounding factors that may affect the periodontal health were excluded from the study.

Obese subjects at two fitness centers [Police Club Fitness Center and Army Club Fitness Center] were included because of their records that give essential information about their general health condition (medical history) and personal data and their Body Mass Index scores. The clinical examination included the Plaque Index, the Gingival Index, Probable Pocket Depth, and Clinical Attachment Loss. The results were analyzed and correlations were done for each periodontal parameter with the Body Mass Index of the study group.

Plaque Index:-

Plaque biofilm consists largely of microbes and host proteins that adhere to teeth surfaces, dental plaque contain many bioactive end products e.g.; fermented organic acids, sulphur compounds and tissue digesting enzymes as well as peptidoglycans and lippolysaccharides these compounds diffuse or perpetrate the gingival epithelium and cause inflammation of the gingival tissues (gingivitis) \(^{45,46,47}\) which is one of the primary stages in the development of periodontal diseases.
However researchers concluded that excessive nutrient intake and sophisticated life style are the two main causes for obesity. *Al – Zahrani, et al,* (107) mentioned that obesity in young adults might be a greater source of chronic stress. Stress and how a person copes with might be a source of poor oral hygiene as a young adult might become negligent to oral hygiene measures. (107)

In the present study the mean Plaque Index was (1.1±0.5) and the mean Body Mass Index was (34.4±3.6).

A correlation was done between the Plaque Index and the Body Mass Index of the study group the result was statistically significant (P=0.002). This agrees with the findings of Saito et al (1998) (105), who concluded that obesity may negatively affect oral hygiene measures.

**Gingival Index:**

In the present study the mean gingival index was (1.4±0.5). A correlation was done between the Gingival Index and the Body Mass Index as a measure of obesity of the participants. A statistically significant association was found (P =0.029).

A suggestion can be made accordingly that there is a link between obesity and periodontal disease, gingival disease being one of them. *Saito, et al* (2001) (105) concluded the same in a study conducted among 241 healthy Japanese subjects. However from the results of the Plaque Index of this group of obese subjects, it is expected that a negative effect on gingival health will occur.
**Probable Pocket Depth:**

The mean pocket depth in the present study was (6.5±1.2 mm). The number of subjects presented with Probable Pocket Depth of ≥4 mm were accounting for 95 with 46% of the entire study group.

The number of subjects presented with Probable Pocket Depth and Clinical Attachment Loss were 129 with a percentage of 62% among all subjects in the study which suggest that the prevalence of periodontitis among this study group was relatively high. Probable Pocket Depth being a diagnostic parameter for periodontitis is a result of pathological deepening of the gingival sulcus by the action of firstly plaque biofilm and the microbial community within it, secondly by the action of host response towards that microbial challenge.

The host secrets, both anti-inflammatory and proinflammatory mediators, but in obese subject the amount of proinflammatory cytokines are high because adipose tissues secrets certain adipokines (cytokines and hormones) which are involved in the overall inflammatory process including the periodontitis. \(^{(107)}\)

In the present study a correlation was done between Probable Pocket Depth and the Body Mass Index which showed a statistically significant association (P = 0.000). This result agrees with the results of Saito, et al (2001) \(^{(105)}\) in their cross- sectional study which suggested that there is a strong relation linking obesity to chronic inflammatory periodontal diseases. However these results are also higher than the prevalence of chronic inflammatory periodontitis found in the general Sudanese population. \(^{(109)}\)
Clinical Attachment Loss:

In the present study the mean Clinical Attachment Loss was (8.7±1.3 mm), and the total number of subjects with Clinical Attachment Loss was 97 subjects (47% of the whole study group).

A correlation was done between Clinical Attachment Loss and the Body Mass Index a statistically significant association was found (P =0 .000). This agrees with the findings of Al – Zahrani, et al (2003) \(^{(107)}\)

The explanation for this may be that adipose tissue cell like adipocytes, preadiocytes and macrophages secretes more than 50 bioactive molecules known as a adipokines like: leptin, tumor Necrosis factor alpha (TNF\(\alpha\)), Interlukin – 6 ( IL – 6 ) and Prostaglandin E 2 (PGE2). These proinflammatory cytokines lead to insulin resistance and poor health outcomes and general systemic inflammation including the chronic periodontitis. \(^{(107)}\)

From the results of the present investigation it was clear that obese, relatively young subjects in Khartoum State who are attending Fitness Centers were found to have an alarming prevalence of inflammatory periodontal diseases. This was clear in spite of the fact that, this group of subjects is aware of the health problems resulting from obesity since they are attending a Fitness Center. However what they should be aware of is the importance of oral hygiene care.
4.2 Conclusions and Recommendations

4.2.1 Conclusions

From the results of the present study we may conclude the followings:-

1- The prevalence of gingivitis among obese subjects in this study was found to be 96%, and the prevalence of chronic inflammatory periodontitis was 62%.

2- A significant association was found between both Probable Pocket Depth and Clinical Attachment Loss and the Body Mass Index, as well as with the Plaque Index, frequency of tooth brushing and the Gingival Index.

3- It was clear from this study that the obese subjects examined had more periodontal diseases compared to the general Sudanese of the same age.

4.2.2 Recommendations:-

From the results of the present study we may recommend the following:-

1- Improvement of public health concern to obesity by establishment of well equipped fitness centers paying great attention to medical and dental monitoring for participants is important.

2- Dietary counseling and physical activities are essential for overall health and may help improve gingival and periodontal health.

3- Dental health education to raise the awareness of obese subjects is needed. However it is also important to increase the dentist’s awareness towards possible oral health problems that may affect obese individuals.
References


93) Engebretson SP, Hey-Hadavi J, Ehrhardt FJ, Hsu O, Celenti RS, Grbic JT & Lamster lB.. Gingival crevicular fluid levels of interleukin-1β and glycemic control in patients with chronic


Examination Sheet

1- Date: - ……………….        Serial No: ……………………

2- Name: - ……………………………………………………………………….

3- Age:-……………………

4- Gender: - (1) Male   □    (2) Female    □

5- Address……………………………………………………………………...

6- Oral Hygiene Habit:-

<table>
<thead>
<tr>
<th>Tool</th>
<th>Frequency</th>
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<tbody>
<tr>
<td>0</td>
<td>None.</td>
</tr>
<tr>
<td>1</td>
<td>Tooth Brush</td>
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<tr>
<td>2</td>
<td>Muswak.</td>
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<tr>
<td>3</td>
<td>Both.</td>
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PLAQUE INDEX (PI) (Silness and Löe 1964)

0 = Absence of plaque.

1 = Plaque seen after probing the gingival margin.

2 = Visible plaque.

3 = Abundance of plaque.

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38
**GINGIVAL INDEX (GI) (Löe and Silness 1963):**

0 = Absence of signs of inflammation.

1 = Slight change in color and texture.

2 = Visual inflammation and bleeding on probing.

3 = Overt inflammation and spontaneous bleeding.

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**PROBABLE POCKET DEPTH (PPD) (IN mm)**

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**GINGIVAL RECESSION (IN mm)**

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**ATTACHMENT LOSS (IN mm)**

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</tbody>
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Appendix

بحث استمرار

- : لفظ كلمتين

Obesity and Periodontal Health in Khartoum state

بعض الله الرحمن الرحيم

Appendix

بحث استمرار

- : لفظ كلمتين

Obesity and Periodontal Health in Khartoum state