University of Khartoum
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Periodontal Health Status of Children with Type 1 Diabetes mellitus in Khartoum

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Dedication

Dedicated to every one who tries to do some thing for people despite his disabilities.

Shadia
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Last but not least thanks to every one who have assisted in this work.
Abstract

This study was intended to fulfil the following main objectives:-

1- To study the periodontal health status of type 1 diabetic children
2- To compare the periodontal health status of diabetic children with non-diabetic children of comparable age.
3- To investigate the severity of periodontal disease in relation to the duration of diabetes, metabolic control and presence of systemic complications.
4- The study sample was 100 subjects, 40 diabetic type 1 children and 60 healthy non-diabetic children in Khartoum State. They were aged 7-15 years with a mean age of 13.5 years.
5- The study group had been further subdivided into two subgroups according to metabolic control (30% controlled diabetic and 70% non-controlled).
6- Periodontal parameters were investigated including Plaque Index (Silness and Løe 1964). Gingival index (Løe and Silness 1963). Periodontal pocket depth using Michigan 0 type probe and calculus on the bases of present or absent.
7- Indicators of oral health behavior were detected (frequency of cleaning the mouth / day and visits to the dentist).
8- Information about the systemic disease including duration, systemic complications and laboratory investigation of the level of metabolic control, were also obtained.

The results of this study indicated that:-

1- Higher plaque index scores, gingival index scores and calculus in diabetic test group. They were statistically highly significant.
2- Both groups (test and control) presented mostly with sulcus depth between 0 to 3, but the test group presented higher sextants percentage with pocket depth of more than 3 mm. The difference was statistically highly significant.

3- The controlled diabetic subgroups showed higher plaque index scores and gingival index scores but the pocket depth greater than 3 mm was higher in the non controlled diabetic subgroup.

4- While the control of diabetes has its effect on the periodontium there is no correlation with the duration or systemic complications of diabetes mellitus.

From the results of this study it was concluded that diabetes mellitus affects the periodontal health of affected children in the presence of local etiological factors (plaque and calculus).
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The main information is as follows:

1. Findings:
   
   a. Children in the upper age group were found to have high levels of glucose and the presence of second-degree fever, pus, and white blood cell counts.

2. Depth:
   
   a. Between the sections, the number of children reached 3.0.
   b. When the height exceeded the upper age limit, the glucose level in the afflicted group was significantly high.

3. Mean:
   
   a. The mean of the affected group showed significant findings, indicating a high level of glucose, pus, and white blood cell counts.
   b. The mean of the affected group showed significant findings, indicating a high level of glucose, pus, and white blood cell counts.

4. System:
   
   a. The system had an impact, while the treatment had little impact on the glucose level of the affected cases.
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1.1 Introduction: -

Periodontal disease is one of the most widespread diseases affecting mankind. It is evident in all countries at various degrees of prevalence. The main cause of the periodontal diseases is known to be bacterial plaque. The systemic disorders either pathological or physiological conditions usually does not initiate periodontal disease, but it may modify the host response towards bacterial plaque, affecting the severity and course of the disease.

Diabetes mellitus is a complicated chronic metabolic disorder. It is a major worldwide health problem. As a systemic problem it has its effect on the immunity and the vascular system. It might affect the host response towards bacterial plaque, leading to exaggeration of the inflammatory response.

In spite of the low prevalence of periodontal diseases among children, the effects of periodontal disease observed in adults have their inception earlier in life. Gingival disease in childhood may progress to Jeopardize the periodontium of the individual later in adult life.

Unfortunately diabetes mellitus affects children. However, the plaque biofilm is usually present and gingivitis is common in children, so, the periodontal health of the child may be affected widely and complicated by diabetes. It is important to know the periodontal health status in diabetic children to evaluate the magnitude of the problem and thus manage it at the appropriate time in order to prevent undesired complications including sever periodontal destruction and loss of teeth in the future.

Diabetes mellitus was investigated extensively in Sudan. However, little was done concerning the study of the periodontal health of diabetic children. Hence the present investigation was planed to:-

1) Study the periodontal health of type 1 diabetic children in Khartoum.
2) Compare the periodontal health of diabetic with non-diabetic children.

3) Investigate the severity of periodontal disease in relation to the effect of the metabolic control and duration of the diabetic condition.
Literature Review
1.2 Periodontal Disease: -

1.2.1 Epidemiology: -

Periodontal disease comprises a variety of conditions affecting the health of the periodontium (which consists of the gingiva, periodontal ligament, alveolar bone and cementum). Gingivitis affects over 80% of young children and almost the entire adult population has experienced gingivitis, periodontitis or both. Research and clinical evidence indicates that the damage caused to the supporting structures of the teeth by periodontal disease in early adult life is irreparable. While in middle age it destroys a large part of the natural dentition and deprives many people of all their teeth long before old age\(^1\).

The total effect of the periodontal disease on the general health of the population cannot yet be assessed. The prevalence and severity of periodontal diseases vary according to geographical, social, local and systemic factors and oral habits. Early evidence of periodontitis is frequently seen by the second decade of life. The advanced destruction is commonly observed after the age of 40 years. The prevalence of destructive diseases follows a linear progression from adolescence to old age. The strong correlation with age probably reflects the cumulative effects of the disease rather than the diminishing resistance of older people. In most surveys females have been found to have lower degrees of severity of the disease than males. This can only be explained by better oral hygiene among females. However, in some less developed countries periodontal conditions are worse in females than in males and it has been suggested that this is due to frequent child birth and poor nutrition.
Racial differences in both severity and prevalence of periodontal disease have often been observed. The disease is more prevalent in Asia and Africa than in Europe, Australia and the U.S.A. Other factors such as differences in oral hygiene practices, education, economic level, and environment factors must also be considered. However, variations have also been noted between urban and rural groups (1).

1.2.2 Periodontal Disease in Sudan: -

Since 1966-2001 only two studies had been performed investigating periodontal health in Sudan. The first study was done by Emslie (1966) who found that chronic periodontal disease was prevalent and closely correlated with poor oral hygiene (2). Ghandour et al (2001) investigated 5405 subjects who were examined for periodontal disease in seven groups of states across the country. The subjects were of the age groups (12-15, 16-34, 35-64 and >65 years). Females showed better periodontal health compared to males. The subjects in the age group 12-15 years from Southern States showed better periodontal health, the same was true for Khartoum and Central States. Drafour states showed the worst periodontal health followed by the Eastern states. For 16-34 years Central States showed the best periodontal health, among the 34-64 years of age groups Central States showed less periodontal disease compared to other states. However Khartoum, Northern and Eastern states showed more periodontal disease compared to other states. In general periodontal disease in Sudan is still low compared to other countries of the region (3).

1.2.3 Etiology of Periodontal Disease: -

Microbial dental plaque is the initiator of periodontal diseases, but whether the disease affects a particular subject, what form of the disease
takes and how it progresses, are all dependent on the host defenses to this
date challenge\(^4\).

Dental plaque can be defined as the soft deposits that form a biofilm
adhering to the tooth surface and other hard surfaces in the oral cavity.
Plaque is different from other deposits that may be found on the tooth
surface such as materia alba and calculus. Materia alba is a soft
accumulation of bacteria and tissue cells that lack the organized structure of
dental plaque and can easily be displaced with a water spray. Calculus is a
hard deposit that forms by mineralization of dental plaque and is generally
covered by a layer of unmineralized plaque. Dental plaque is composed
primarily of microorganisms. It has been estimated that more than 325
different bacterial species may be found in plaque. Non-bacterial
microorganisms that are found in dental plaque include *mycoplasma species*,
*yeast*, *protozoa*, and *viruses*. The microorganisms exits within an
intercellular matrix that also contains a few host cells, such as epithelial
cells, macrophages and leukocytes. The intercellular matrix accounts for
20\%-30\% of the plaque mass. It consists of organic and inorganic materials
derived from saliva, gingival sulcular fluid and bacterial products.
Glycoproteins from saliva are important components of the pellicle, that
initially coats clean tooth surfaces, but they also become incorporated into
the developing plaque biofilm\(^5,6\).

In the mid 1900 periodontal disease was believed to result from an
accumulation of plaque over time in conjunction with diminished host
response and increased host susceptibility with age. This thinking was
supported by epidemiological studies that correlated both age and amount of
plaque with evidence of periodontitis. It was thought that all plaque was
alike and equally capable of causing disease. However, later several
observations contradicted this conclusion. Some individuals with considerable amounts of plaque and calculus as well as gingivitis never developed destructive periodontitis. Furthermore, individuals who did present with periodontitis demonstrated considerable site specificity in the pattern of disease. Some sites were unaffected while advanced disease was found in adjacent sites, in the presence of a uniform host response, these findings were inconsistent with the concept that all plaque was equally pathogenic. This finding led to renewed search for specific pathogens in periodontal diseases and a conceptual transition from the non-specific plaque hypothesis to the specific plaque hypothesis(7-9).

Walter Löesche (1976) delineated the specific and non-specific plaque hypothesis. The non-specific theory manifested that disease results from the elaboration of noxious products by the entire plaque flora. Accordingly when only small amounts of plaque are present, the host neutralizes the noxious products, a large amount of plaque would present large amounts of noxious products, which would essentially overwhelm the host defenses. On the other hand the specific plaque hypothesis states that (only certain plaque is pathogenic, and it’s pathogenicity depends on the presence of, or increase in specific micro-organisms). This concept predicts that plaque harboring specific bacterial pathogens results in specific types of disease, because these organisms produce substances that mediate the destruction of host tissues(10).

Several local factors previously considered to be of direct etiologic significance in periodontal disease are now known to act only by favoring plaque accumulation. These include calculus, faulty restorations, partial removable prostheses, food impactions, malocclusion and others.
Some systemic factors may modify all forms of periodontitis, but in many cases, the literature is insufficient to make definite statement on links between systemic factors and periodontitis\(^4\). Systemic diseases do not initiate chronic destructive periodontitis but may accelerate its progression and increase tissue destruction\(^11\).

Genetics in The Etiology of Periodontal Diseases: - There is no evidence to date linking early onset periodontitis in diverse populations to any gene or marker. There is also no evidence for major gene effects on adults onset periodontitis.

There are significant racial differences in both the prevalence of early onset periodontitis and associated host factors. It is currently unclear whether these differences are due to genetic or environmental factors\(^12\).

Smoking in the Etiology of Periodontal Diseases: - Data is strong indicating that smoking is an important risk factor for both adult onset and early onset periodontitis. Smoking has been shown to decrease serum IgG and alter PMNs function.

The general biomedical literature indicates that smoking has an effect on the vasculature, connective tissue and immune cells. These effects influence wound healing, immune response and inflammatory response.

Smoking may modulate the sub-gingival micro-biota and increases the prevalence of certain pathogens. Products of cigarette smoke are present in the sulcular fluid, and the clinical expression of the disease is altered in smokers, including increased calculus formation, recession of the palatal gingiva and decreased gingival inflammation. Smoking increases the risk of periodontal disease by nearly 10% in diabetic patients\(^13\text{-}16\).

Periodontal Pathogens: - In light of the advances made since the 1989 World Workshop on Clinical Periodontics, it has been determined that there are sufficient data
to consider the following three microorganisms as etiological agents in periodontal diseases:- 

*Actinobacillus actinomycetemcomitans; Porphyromonas gingivalis;* and *B. Phorsythus.*

There is also moderate evidence for the role of the other bacteria these are *Campylobacter rectus, Eubacterium nodatum, Fusobacterium nucleatum, prevotella intermedia/nigresence.*

Some evidence is available for the role of *Eikenella corroden*, *enteric rods, Pseudomons, Selemonans, Staphylococcus,* and *yeasts* associated with HIV periodonitis and peri-implantitis\(^{(17)}\).

**1.2.4 Classification of Periodontal Diseases:**

In order to scientifically study the etiology, pathogenesis and treatment of periodontal disease, a classification system must be agreed upon. A classification was adopted in 1989 by the World Workshop on Clinical Periodonitics. Subsequently a similar classification was agreed upon in the European Workshop of Periodontology (1993). These classifications have many shortcomings so the need for a revised classification system for periodontal diseases was emphasized during the 1996 World Workshop on Periodontics. In 1997 the American Academy of Periodontology formed a committee to plan and organize an International Workshop to revise the classification system for periodontal diseases in October 30-November 2\(^{nd}\) (1999), the International Workshop for Classification of Periodontal Diseases and Conditions was held and a new classification was agreed upon which differs from the classification system developed in the 1989 Workshop in Clinical Periodontics, 1989 classification was as follow\(^{(18)}\):-

- Adult periodontitis
- Early onset periodontitis
- Prepubertal Periodontitis Generalized and localized
- Juvenile periodontitis Generalized and localized
- Rapidly progressive periodontitis
- Periodontitis associated with systemic diseases
- Down’s syndrome
- Diabetes mellitus type 1
- Papillon Lefevre syndrome

The changes in 1989 classification system included the addition of a section on gingival diseases and lesions that are either dental plaque induced or not related to plaque.

The addition of a category on periodontal abscesses because, in the view of the workshop participants, periodontal abscesses present special diagnostic and treatment challenge.

A category on periodontal–endodontic lesions and a category on developmental or acquired deformities and conditions were also added. The changes also included replacement of the term (adult periodontitis) with chronic periodontitis, clearly the old term designation created problems e.g. in adolescent with this type of periodontitis.

Also replacement of early onset periodontitis with aggressive periodontitis. Patients with the category prepubertal periodontitis, under the new classification system would be placed under the heading of periodontitis as a manifestation of systemic diseases.

Necrotizing ulcerative periodontitis was replaced with necrotizing periodontal diseases. The new classification eliminated a separate disease category for refractory periodontitis.

The designation periodontitis as a manifestation of systemic disease has been clarified, the consensus report for this portion of the workshop
contained a list of systemic diseases in which periodontitis is a frequent manifestation. It should be noted that diabetes mellitus is not on this list. However, In the collective view of the workshop participants, diabetes can be a significant modifier of all forms of periodontitis but there is insufficient data to conclude that.

Similar to 1989 classification the new one does not contain a separate disease category for the effect of cigarette smoking\(^{(19)}\).

**1.2.5 Pathogenesis of Periodontal Diseases: -**

The development of periodontal diseases depends on the interaction between the resident microbiota and the host response. In certain types of periodontal diseases such as desquamative gingivitis, the lesion may frequently result from the host response\(^{(20)}\).

The histopathology of chronic gingivitis has been described chronologically by Page and schröeder\((1976)\) in a number of stages, the initial lesion 2-4 days followed by an early gingivitis and then the established lesion followed by the advanced lesion. These changes were described after examining biopsies of experimental gingivitis lesion at different time interval\(^{(21)}\). They were described as follows: -

*I- Initial-lesion:* -Represents an acute inflammatory response with characteristic infiltration of neutrophils, vascular changes and collagen changes (prevascular collagen degradation). These changes are likely due to chemotactic attraction of nutrophils by bacteria and host system constituents. No clinical signs and symptoms\(^{(22)}\).

*ii- Early lesion:* -Characterized by 75%T lymphocytes, few plasma cells and macrophages infiltration.
Changes occur both in junctional and crevicular epithelium where there are signs of cell separation and dento-gingival fiber groups breakdown, so that the seal of the marginal cuff of the gingiva is weaken.

Clinically the gingiva appears healthy because the lesion, which becomes more chronic in nature occupies a very small area of the gingiva, also the sign of acute inflammation is reduce as the lesion become chronic.

**iii- Established gingivitis:** -If satisfactory oral hygiene is not reestablished, clinically obvious gingivitis become established within 7-14 days(the papilla become swollen and bleed on probing).

*Histologically:* The number of lymphocytes increases and B- lymphocytes become more dominant, many of these are mature cells and produce specific antibodies mainly IgG, but there is secondary stimulation of acute inflammation produced by complement activation. resulting in emigration of PMNs through the junctional epithelium into the gingival sulcus and the flow of GCF increases. There is now degradation of the cells of the junctional epithelium and some proliferation of its basal layers into the underlying connective tissues, but at this stage there is no significant migration of epithelial cells onto the root surface. As the inflammation spreads there may be some resorption of the alveolar crest which is reversible on resolution of the inflammation. At more distant sites there is proliferation of fibrous tissues and blood vessels characteristic of chronic lesions.\(^{(23)}\)

**iv- Advanced lesion:**- extension of the lesion into the alveolar bone characterizes the forth stage that has been termed the advanced lesion or phase of periodontal breakdown.

Pathological changes in the gingival tissues consistent with clinically chronic or acute gingivitis have been noted in a number of systemic
conditions some of these may mimic the vascular alterations seen in plaque induced gingivitis or result in cellular infiltration by aberrant leukocytes or other vascular elements. These include acute leukemia, hemophilia, Sterge Weber syndrome, Wegener’s granulomatosis. In some other conditions defective host responses to bacterial infection may be manifested as an over expression of gingival inflammation or caused by an alteration in the usual bacterial microflora. Such conditions include Addison’s disease, diabetes mellitus, thrombocytopenia, HIV and combined immune deficiency diseases. A third group of conditions is related to hormonal changes manifested as exaggerated inflammatory response to plaque as well as an alteration in the subgingival microflora, these include cases related to pregnancy, puberty, steroid therapy and the use of hormonal contraceptives. Finally a large number of drugs many of which are associated with therapy for seizure disorders, hypertension or transplant rejection cause gingival enlargement in the presence of bacterial plaque (24).

Periodontitis is clinically differentiated from gingivitis by the loss of connective tissue attachment of the teeth in the presence of concurrent gingival inflammation, loss of the periodontal ligament and disruption of its attachment to the cementum, as well as resorption of the alveolar bone, with migration of the epithelial attachment along the root surface. The histopathology of the periodontitis lesion is in many ways similar to that of the established lesion of gingivitis, with predominance of plasma cells, loss of soft connective tissue elements and in addition bone resorption.

Despite the histopathologic similarities between gingivitis and periodontitis evidence indicating that periodontitis is an inevitable consequence of gingivitis is lacking. Further more, the pathogenic mechanisms explaining the progression of gingivitis lesions to periodontitis
lesions are not clear, and the factors that lead to initiation of periodontal lesions are still not well understood. Clinical models of disease activity in periodontitis, range from a continuous progression of disease during which loss of attachment occurs at a slow rate over long periods of time to an episodic burst model in which loss of attachment occurs relatively rapidly during short periods of disease activity.

Although two pathogenic bacteria have been shown to invade the superficial layers of the periodontal tissues, it is readily apparent from pathological observations that pathologic effects on connective tissues and alveolar bone occur at sites deep to the sub-gingival plaque and invading micro-organisms. For this reason in addition to the direct pathologic effects of bacteria on periodontal tissues, it is clear that the damage to the periodontium must also occur by indirect means, bacterial products must gain access to the cellular constituents of the gingival tissues and activate cellular processes that are destructive to collagenous connective tissue and bone. It is likely that direct pathological effects of bacteria and their products on the periodontium are significant during the early stages of the disease. It is likely that bacteria can contribute to the pathogenesis of periodontal diseases. Once the major protective elements in the periodontium have been overwhelmed by bacterial virulence mechanisms, a number of host mediated destructive processes are initiated. PMNS which normally provide protection, can themselves contribute to tissue pathology. During the process of phagocytosis, these cells typically (spill) some of their enzyme contents extra cellularly during a process known as degranulation. Some of the enzymes are capable of degrading surrounding host tissues, namely collagen and basement membrane constituents, contributing to tissue damage.
There is increasing evidence that the bulk of tissue destruction in established periodontitis lesions is a result of the mobilization of the host tissue via activation of monocytes, lymphocytes, fibroblasts and other host cells. Engagement of these cellular elements by bacterial factors in particular bacterial lipopolysaccharides (LPs), is thought to stimulate production of both catabolic cytokines and inflammatory mediators including acid metabolites, cytokines and inflammatory mediators in turn promote the release of tissue derived enzymes. The matrix metalloproteinases are a family of zinc and calcium dependent endopeptidases secreted or released by a variety of infiltrating cells (i.e. PMNS and macrophages) and resident cells (i.e. fibroblasts, epithelial cells, osteoblast and osteoclasts) formed in the periodontium. A number of physiologic processes (embryonic development and tissue remodeling) and pathologic conditions are characterized by matrix metalloproteinasis (MMP) activity. It was recognized that endogenous MMPs are primarily responsible for tissue destruction and not bacterial proteins \(^{22,25}\)

1.3 Periodontal Health and Disease in Childhood: -

i- Periodontal Health in Childhood

The gingiva of the deciduous dentition is pale pink, firm and may be either smooth or stippled. Stippling is found in 35% of children between 5 and 13 years of age \(^{26}\). The interdental gingiva is broad faciolingually and tend to be relatively narrow mesiodistally, in conformity with the contour of the approximal tooth surface. It’s structure is comparable to that of the gingiva. It consists of a facial papilla with intervening depression or col. The mean gingival sulcus depth for the primary dentition is 2.1mm ± 0.2mm. The width of the attached gingiva is greater in the incisor area than in the
area of the primary molars. The attached gingiva increases in width with age. (27,28)

Microscopically the stratified squamous epithelium of the gingiva presents well differentiated rete pegs with a parakeratinized surface which is correlated with the stippling. The connective tissue is predominantly fibrillar, but the well-differentiated collagen bundles in the adults are not present in childhood. The periodontal ligament of the deciduous teeth is wider than that of the permanent teeth. During eruption the principal fibers are parallel to the long axis of the tooth, the pundles arrangement occur when teeth encounter their functional antagonists.

Radio-graphically the alveolar bone in relation to the deciduous dentition shows prominent lamina dura both in crypt stage and during eruption. The trabeculae of the alveolar bone are fewer but thicker than in adults and the marrow spaces tends to be larger and the alveolar crests are flat (29).

ii- Periodontal Diseases in Childhood: -

Etiology of periodontal diseases in children is as in adults i.e. bacterial plaque. It appears to form more rapidly in children aged 8-12 years than in adults. The calculus is uncommon in infants and it occurs in approximately 9% of children 4-6 years in 18% of 7-9 year olds and in 33% to 43% among those 10-15 years old (30).

Chronic marginal gingivitis is the most prevalent type of gingival changes in childhood. Gingival color changes and swelling appear to be a more common expression of gingivitis in children than are bleeding and increased pocket depth (31).

Gingivitis associated with tooth eruption is frequent and has given rise to the term eruption gingivitis. However, tooth eruption per se does not
cause gingivitis, but the inflammation results from plaque accumulation around erupting teeth.

Partially exfoliated loose deciduous teeth frequently cause gingivitis. The eroded margin of partially restored teeth favor plaque accumulation, which cause gingival changes varying from slight discoloration and edema to abscess formation with suppuration. Other factors that favor plaque accumulation include carious cavities. Gingivitis increases in children with excessive over-bite and over-jet, nasal obstruction and mouth breathing habits.

According to Maynard and Wilson (1980), mucogingival problems in the primary dentition may occur as a consequence of developmental aberrations in eruption and deficiencies in the thickness of the periodontium if there is inadequate plaque control or excessive tooth brushing trauma it will lead to mucogingival problems. However, when the width of attached gingiva increases with age, such problems may resolve (32).

Early Onset Periodontitis: -

(i) Perpubertal periodontitis: Severe gingivitis and destructive periodontitis in the primary dentition.
(ii) Juvenile periodontitis: Sever localized attachment loss in permanent first molars and incisors, which is known as localized juvenile periodontitis (LJP). Sometimes generalized involvement of these teeth and few or many other teeth may occur which is known as generalized juvenile periodontitis.
(iii) Rapidly progressive periodontitis: -with generalized rapid attachment loss in the permanent dentition usually occur in young adults.
Perpuertal Periodontitis: -
This is a rare form of periodontal disease. It has its onset before the age of four years, and is characterized by rapid destruction of the periodontium of the primary dentition. The gingiva is grossly inflamed, and the patient commonly has bacterial infections. In some cases the condition affects the permanent dentition. In many instances there is a familial pattern of the disease, and most if not all cases are probably genetically mediated. Pre pubertal periodontitis is usually associated with Papillon-Lefevre syndrome, Down’s syndrome, Hypophosphatasia, acute and sub acute leukemias and leukocyte adhesion deficiency (LAD), and may be other syndromes (33).

Juvenile Periodontitis: -
Chaput introduced the term juvenile periodontitis in 1967 and Butler in 1969. Baer 1971 described juvenile periodontitis as a well defined clinical entity different from adult periodontitis in that it appears to start around the age of puberty. It seems more common in girls, appears to occur in families, and is rapidly progressive. The amount of destruction manifested is not commensurate with the amount of local irritants. Two forms of the diseases were described, localized and generalized. In the localized form the tissue destruction is restricted to the first molars and incisors, and is characterized by a symmetrical distribution. Gradients between these two extremes are often seen. The variability of these forms was recently demonstrated by Yosof (1990) (34) in a study of 47 Malaysian children (22 boys and 25 girls) affected with the condition, he divided the children into four groups according to the distribution of the bone loss.

1) Type one:- Bone destruction limited to first molars and incisors (14.9%).

2) Type two:- Bone destruction involved first molars, incisors and some other teeth (25.9%).

3) Type three:- Generalized destruction but worse around the first molar and incisors (14.9%).

4) Type four:- Generalized involvement of more than 14 teeth (44.7%).

Prevalence:-
Juvenile periodontitis occurs in approximately one in thousand adolescents and seem to have racial predisposition, occurring more frequently in people of West African origin. Recent studies resulted in a prevalence of up to 0.2%. It has been stated that JP is found most commonly in people of low socioeconomic classes.

Age:

The patient is usually an adolescent at the time of diagnosis, but may be much younger. (The onset may be several years before the time of examination).

Sex ratios:

Many of the earlier studies reported that the condition appeared more commonly in females than males, at a ratio of about 3:1 (Bear, 1971, Mason and Lehn 1974). But Sax (1987) and Melvin et al (1991) found almost equal sex prevalence (1.1:1) and this is twice female to male ratios respectively.

There is a familial tendency in this disease. Bone destruction classically is advanced, localized to the incisors and molars, showing symmetrical or mirror image distribution, with deep angular or crescent bone defects.

The sub-gingival microflora in JP is scanty compared with adult periodontitis. It is dominated by coccoid and straight non-motile rods. The dominant cultivable micro flora consist of gram negative capnophilic and facultative rods. The principle bacteria is *actinobacillus – actinomycetemcomitans*, *cabnocytophaga-species* and *Eikenella-corrodens*.(34-37.)

Regarding periodontal health status in children a number of studies have been performed. In one study by Blankenstien (1978), 1731 English and Danish school children 13-15 years of age were examined to assess the
prevalence of chronic periodontitis. The results indicated that only one child was affected i.e. 0.06% of the number studied\(^{(38)}\).

Krumova et al (1994) assessed the oral hygiene and periodontal health in 1596 school children (777 boys and 819 girls), aged 7-14 years. The oral hygiene status was assessed using the plaque index and was found to be rather poor. The relative share of children with plaque index of 0.00 to 1.00 was found to increase with age for both sexes. A correlation between oral hygiene and chronic gingival inflammation in 14 year’s old school children was found\(^{(39)}\).

Bhowate et al (1994), assessed the caries experience and periodontal health in 11-15 years old rural school children in Sevagram, 59.35% of the 802 children examined had gingivitis\(^{(40)}\).

Bjarnason et al (1995), assessed caries, oral hygiene and periodontal health in a representative sample in 15 years old Lativain school children. Visible dental plaque was found in 98.4% of the children. In the majority (88%) abundant plaque deposits were recorded. CPITN was assessed by standard WHO methods, deviation from periodontal health was observed in 90.7% of the children. Calculus was recorded in 26.1% gingival pockets in 25.9% of the sample and 38.7% of the children had gingival bleeding. The mean number of sextants with healthy periodontium was 2.5%. Only calculus and pocketing averaged 0.6 and 0.4 sextants respectively. Attachment loss was recorded in 11.7% of the subjects, but didn’t exceed 3mm\(^{(41)}\).

According to WHO, in any particular country 5 yearly epidemiological surveys should be carried out to monitor changes in oral health. International base line examination was carried in 1985 in Hungary, followed by another one in 1991. In 1998 using the pathfinder methodology,
1800 children aged 5 - 6 and 12 years were examined. More than two thirds (72%) of the children examined had gingivitis and need oral hygiene instruction, one third of them (32.3%) had dental calculus (42).

1.4 Diabetes Mellitus: -

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia due to defective secretion or activity of insulin. Diabetes mellitus can be classified into four broad categories according to signs and symptoms. Currently, the old term insulin dependant and non-insulin dependant diabetes mellitus (IDDM) and (non-IDDM) are not in use, because they are related to treatment rather than to diagnosis. Type I diabetes mellitus encompasses diagnosis of the disease resulting primarily from destruction of the beta cells in the Islet of Langerhans of the pancreas, leading to absolute insulin deficiency. The precise etiology of this destruction is unclear, but it appears to involve genetic factors related to HLA (MHC) genetic zone in chromosome 6, and has some features of autoimmune diseases. This may be explained by the large number of type I DM patients who have anti-bodies against coxackie 6B virus. It has been suggested that peptide sequences from viral proteins mimic those in certain protein antigens in the islet cells. T-cell immune responses directed against these antigens would kill or damage the islet cells. This would probably occur in a group of susceptible individuals with the appropriate HLA antigens, which would present the appropriate peptide sequence from the virus (43). Type 1 DM onset, is often abrupt, patients with this disease are more prone to ketoacidosis and wide fluctuation in plasma glucose levels. If untreated the patient is more likely to manifest the classic signs and symptoms of diabetes, which are ploy-urea, ploy – dipsia, ploy-phagia and loss of weight (44). Type one diabetic patients are more likely to suffer severe
systemic complications as a result of the disease. Brittle diabetes is a subgroup of type one where the patient have frequent and rapid swings of blood sugar levels between hyperglycemia and hypoglycemia. Such patients may require several injections of different types of insulin during the day to keep the blood sugar level within a fairly normal range. Patients are almost exclusively, young women. The high prevalence of psychosocial difficulties and demonstrable manipulation of therapy suggests that the primary cause of the syndrome may be psychosocial. Little is known about the natural course of brittle diabetes\(^{45,46}\). The more common form of diabetes, is type II diabetes, and is sometimes known as age-onset or adult-onset diabetes and is associated with obesity and lack of physical activity. It is considered a milder form of diabetes because of its slow onset and easy control with diet, oral medication and sometimes insulin injections. However, if uncontrolled it is just as serious as type I. The cause of type II ranges from insulin resistance with relative insulin deficiency to a predominantly secretory defect accompanied by insulin resistance. Another form of diabetes is the gestational diabetes, which can develop during the second or third trimester of pregnancy in about 2% of pregnant ladies. Usually these ladies are at a higher risk of developing type II diabetes within 5-10 years. A wide variety of relatively uncommon conditions fall into the categories of other specific types. These includes genetically defined forms of diabetes and diabetes associated with other diseases or drug use and pancreatic disease as well as alcoholism, malnutrition or other sever illnesses that affects debilitate individuals.

Diagnosis of Diabetes:

The diagnosis of diabetes is based on symptoms. Urine and blood tests can be used to confirm the real definitive diagnosis. These tests are also used
to monitor the disease. Paper strips or dipsticks that change color when
dipped in urine are also used. The test strip is compared to a chart, which
shows the amount of glucose in the urine based on the changes in color.
Blood tests are used as fasting glucose test after a period of at least eight
hours when the patient is fasting. Plasma levels of 7-8 mmol/L (200 mg/L)
or greater indicate diabetes. Usually tests are repeated to confirm the result.
Other tests are postprandial glucose test, where the blood is taken after the
patient has eaten a meal. In The glucose tolerance test, a blood sample is
taken following ingestion of a concentrated syrup of glucose or other sugars.
In diabetics plasma glucose level of 11.1 mmol/L or higher two hours after
the syrup and at one another point during the two – hours test period
confirms the diagnosis of diabetes\textsuperscript{(47)}.

The glycosylated hemoglobin test gives an idea about the diabetes
control during the last three months.

Complications of Diabetes: -

Complications of the diabetes are related to long elevation of blood
glucose concentration (hyper-glycemia). This results is formation of
advanced glycation end – products (AGEs). These (AGEs) act to “prime”
endothelial cells and monocytes, making them more susceptible to stimuli
that induce the cells to produce inflammatory mediators. Long term
complications may occur in both type I and type II diabetes macrovascular
complications including, coronary artery diseases, cerebrovascular and
peripheral vascular disease.

Microvascular complications include retinopathy, which may lead to
blindness, nephropathy which may lead to renal failure and neuropathy
which may lead to loss of limbs and dyesthesias (burning sensations). In
terms of oral manifestations the patient may experience delayed wound
healing and xerostomia, as well as increased susceptibility to periodontal disease and more opportunistic infections (e.g. candidiasis). Oral pareshesia including burning mouth tongue and altered taste sensation may also occur. (44)

**Diabetes in its Psychosocial Context:**

Patients with diabetes make up the most diverse group imaginable. This diversity along with the fact that diabetes can occur at any time within the life span of a person and does not go away, accounting for the wide range of associated bio-psycho-social issues. Multiple stresses, which range from insulin reactions to permanent physical complications, occur in three phases of health and function. The first is the first year after the onset of diabetes within emotional upheaval attendant on diagnosis. The mid phase of relatively well being and full function. The third phase begins when the person needs to make allowances for one or more permanent physical complication (48).

**Treatment of Diabetes Mellitus:**

There is currently no definitive cure for diabetes. The condition however can be managed so that patients can live a relatively normal life i.e. treatment focuses on two goals, keeping blood glucose within normal range and preventing the development of permanent long term complications. Careful monitoring of diet, blood glucose levels, and exercise are as important as the use of insulin or oral medication in preventing the complications of diabetes (44).

**1.5 The Relationship Between Periodontal Diseases and Diabetes Mellitus:**

Epidemiological studies were used to explain the relationship between periodontal diseases and the severity of diabetes, occurrence of
complications, mode of therapy, duration of diabetes and age of the patient.

Glavind et al (1968), studied the periodontal status of diabetic males aged 20-40 years. The authors found that the character of periodontal diseases among diabetics and non-diabetics was the same. The rate of periodontitis was the same up to 30 years of age but among the 30-40 years age group, diabetics showed an increased amount of periodontal breakdown compared to non-diabetics. Patients with a history of diabetes of longer than 10 years showed greater loss of periodontal structures than those with shorter duration. Insulin dose did not appear to be related to the degree of periodontal breakdown. Greater loss of attachment was detected among patients with retinal changes resulting from diabetes.

Tellervo et al (1985), compared the oral health of diabetic and non-diabetic patients and investigated the effects of diabetes on gingival health. They examined the periodontal health status of 50 adult diabetic patients and 53 healthy controls. The diabetic group was further divided into three subgroups according to the control of diabetes. Comparison between the entire diabetic group and the control group did not reveal any difference in either the amount of etiologic factors or in the degree of gingival changes. When the subgroup of diabetic patients was examined, poorly controlled diabetics showed significantly more gingival bleeding. The difference in the amount of gingival bleeding between poorly controlled diabetics and healthy controls was also statistically significant. No correlation was found between duration, complication and medication of diabetes and gingival bleeding.

Wilton (1988), reviewed the evidence for systemic predisposition to periodontal diseases in relation to cellular and humeral immunity, drug therapy, diet and nutrition. He concluded
that apart from defects of polymorphonuclear neutrophils and Ehlers–Dandlos Syndrome, little firm evidence exists for other diseases. Though insulin dependant diabetes and AIDS may accelerate and/or potentiate the damage of existing periodontal disease. He also concluded that whilst diabetes probably predispose to periodontal diseases which may be more severe than among-diabetics. However, the mechanism underlying this is still unclear. The author further concluded that it is increasingly believed that increased predisposition of diabetics to infections of all kinds may result from the synergism of immunological defects (51).

Milijenko etal (1988), investigated the periodontal treatment needs of diabetic patients and the possible effects of the duration and control of diabetes on the periodontal status of these patients using the CPITN. A comparison was made between 222 diabetic patients (mean age 46.9 years) and 189 control subjects (mean age 43.9 years). The result showed that on average 1.3 sextants in 50.9% of the diabetic patients and 0.3 sextants in 17.9% of the control subjects required complex periodontal treatment. Up to the age of 34 years no differences were observed between the diabetic and controls regarding pockets of 6mm or more. Above this age diabetics demonstrated significantly more sextants with deep pockets, and no differences were found in the periodontal condition related to the duration and control of diabetes. The results of this study indicated that oral hygiene instructions and scaling were required for all patients of both groups (52).
Satrowijoto et al (1989), studied the relationship between the degree of diabetes control and the clinical periodontal condition of the patients. The presence of possible pathogenic microorganisms in both diseased and healthy periodontal pockets and the influence of metabolic control on the subgingival periodontal microflora was also investigated. Twenty-two type 1 diabetic adults were grouped into patients with accepted control and poor control on the basis of glycosylated hemoglobin (HbA.C) values. Forty-four subgingival sites were examined for the bacteria. *Actinobacillus actinomycetemcomitans, black pigmented bacteroides species and capnocytophaga species*. No significant difference could be demonstrated between patients in the two test groups with regard to their periodontal condition. Neither age of the diabetic patients nor duration of diabetes influenced the periodontal parameters in both test groups. Pocket depth of 4mm or more was found to be significantly associated with increased swelling, bleeding on probing and amount of marginal plaque. It was concluded from this study that diabetes control seemed to have no direct effect on the periodontium. *Actinobacillus actinomycetemcomitans and black pigmented bacteroides species* may be important pathogens in periodontal disease in Type 1 diabetics as they are known to be in non-diabetic periodontal patients(53).
Sastrowijoto et al. (1990), in a prospective study, examined the effects of metabolic control on the clinical periodontal condition and the subgingival microflora of diseased and healthy periodontal pockets in 6 ambulatory type 1 diabetic patients. The authors concluded that periodontal condition in type 1 diabetics might only ameliorate when local oral hygiene measures were applied (54).

Robert et al. (1992), examined a group of poorly controlled insulin dependent diabetics in a cross-sectional study for total microbial levels, microbial incidence, and the percent levels of selected periodontal microorganisms on the basis of prior reports that associated them with either periodontal disease or health. One periodontal healthy and one periodontal diseased sites were examined in each of the type 1 diabetic patients. The purpose of that investigation was to compare a selected group of periodontopathogenic organisms. Significantly detected total count levels of *P. intermedia*, *P. melaninogenica* species, *B. gracilis*, *F. corrodens*, *F. nucleatum* and *C. rectus* were seen in the disease pockets as compared to the healthy control pockets. A significantly elevated level of the organism *P. intermedia* was seen in the diseased versus healthy periodontal pockets. The significantly elevated levels of these organisms may be due to an increased susceptibility in the poorly controlled type 1 diabetics.
This increased susceptibility to infection could be associated with depressed neutrophil function including chemo taxis, phagocytosis and killing reported in the diabetic patients and may contribute to the increased severity of periodontal disease reported in type 1 diabetic patients\(^{(55)}\).

Wilson and Reeves (1986), found that in vitro neutrophil phagocytosis and killing of *Candida albicans* was inhibited by increased concentration of glucose and beta-hydroxybutyrate thus this killing of candida by the diabetic neutrophil is impaired under conditions of hyperglycemia and ketosis. Diabetics also displayed an angiopathy affecting various blood vessels. Changes in the periodontal vasculature have been documented. Histological studies of oral diabetic patients have reported splitting, thickening and deposition of polysaccharide substances in the basement membrane of the small gingival blood vessels. This finding may suggest that local blood supply to the gingiva is diminished among diabetics resulting in a reduced repair ability\(^{(56)}\).

Tervonen et al (1994), studied the prevalence of periodontal pathogens with varying metabolic control of diabetes mellitus. One hundred and seven individuals with diabetes mellitus 20-70 years of age were studied for the occurrence of periodontal pathogens including *Actinobacillus actinomycetemcomitans, F. nucleatum, E. corrodens, P. gingivitis, P. intermedia*. The result
revealed that most frequently present periodontal pathogen in this diabetic population was *p. gingivalis* (34.6%). However, 28% harbored *F. nucleatum*. 20.6% *F. corrodens*, *Actinobacillus actinomycetemcomitans* and *p. intermedia* were both found in less than 10% . In diabetic patients with mostly early periodontitis, no significant relationship seems to exist between the prevalence of periodontal pathogens and diabetic factors such as metabolic type and duration of the disease. Thus, the increased prevalence and extent of periodontitis, observed in poorly controlled diabetes is due to factors other than increased pathogenicity of the sub gingival microbial flora\(^{(57)}\).

In a study conducted to investigate the relationship between periodontal disease and organ complications in Type 1 diabetic patients, Kaisa et al (1994) examined twenty six type 1 diabetics, 26-34 years of age who had diabetes for at least 10 years. The results revealed that patients with advanced complications had significantly more bleeding on probing, more pockets > 4mm deep and more attachment loss than patients with incipient or no complications. Indicators of dental health behavior including amount of plaque, subgingival calculus, oral hygiene practices and regularity of visits to the dentist were worse among subjects with poor diabetes control and complications\(^{(58)}\).
Seppala et al (1997), examined cellular and vascular changes in gingival connective tissue samples by serologic point – counting procedure and interactive digital analyzing systems in long term type 1 diabetic patients. Gingival connective tissue capillaries representing clinically healthy sulci with no evidence of periodontal disease at the site of biopsy, were studied in 29 patients with diabetes. 19 were identified as poorly controlled and 10 as controlled insulin dependant diabetics. Ten non-diabetic, age and gender matched individuals served as controls. For each individual site specific recordings were made for the plaque index, bleeding index, probing depth, attachment loss and radiographic interproximal alveolar bone loss were recorded. The results showed that swollen and proliferated endothelial cells were frequently found in poorly controlled diabetic patients and the mean distance from the lumen to the outer border of basement membrane was greater among poorly controlled patients than among the controlled. The findings of this Study indicated that, cellular, vascular and connective tissue changes indicative of increased catabolism rather than anabolism detected in the gingiva are especially associated with poorly controlled long term insulin – dependant diabetes (59).

Ludovico etal (1998), performed a 3-year longitudinal study assessing the periodontal status and sub gingival micro biota of
type 1 diabetics. A group of 16 patients were compared with their 16 healthy cohabiting siblings. Patients were monitored every three months for levels of glycosylated hemoglobin. Clinical and microbiological parameters were measured six weeks before drawing blood to determine levels of HbA1c. Periodontal parameters including probing depth, attachment level, sulcus bleeding index were measured at base line (To), year 2(T2) and year 3 (T3). Two sites in each patient were selected for microbial sampling. The result revealed no significant difference in clinical parameters between diabetics and healthy siblings at any examination. However, there was a significant increase in *P. intermedia* at T3 as compared with base line results for deepest sites in the diabetic group. The data suggested no significant differences in clinical parameters between the diabetic and non-diabetic siblings throughout this 3-year longitudinal study (60).

Paul et al (1999), evaluated the periodontal disease status of 320 dentate adults with a mean age of 23.7 years who were previously diagnosed as Type 1 diabetics. These patients had been monitored at a 2-year interval during one of their regularly scheduled medical examinations. They received a periodontal examination as part of a comprehensive oral health assessment program. For the periodontal assessments three facial sites (mesial, mid cervical and distal) of the teeth in the right maxillary and left
mandibular sites or vice versa were evaluated for calculus, bleeding on probing and loss of attachment. The results indicated that attachment loss was significantly greater for older patients, whereas, bleeding on probing and calculus levels were constant for other age groups. Univariate analysis of factors possibly related to extensive periodontal disease indicated an association with older age, lower income, level of education, past and current cigarette smoking, infrequent visit to the dentist, tooth brushing less than once per day, age of onset, longer duration of diabetes and diabetes complication of neuropathy (61).

Because periodontal ligament cells (PDL) play a significant role in maintenance and regeneration of mineralized tissues, the success of procedures such as guided tissue regeneration was found to be directly related to the ability of these cells to augment mineralized tissues, Hana et al (1999), examined the ability of PDL cells from a longstanding type 1 diabetes to form mineralized tissue and to determine whether these cells would exhibit altered responses to exogenously added growth factors. PDL cells were isolated from 4 diabetic patients treated with insulin for at least 5 years and from other systemically healthy donors. The cells isolated were tested for their ability to form mineralized nodules in vitro and to express alteration in alkaline phosphate activity in response to exogenously added growth factors. All PDL cells
isolated formed mineralized nodules, but PDL cells from diabetics formed the nodules slowly than did the controls. Alkaline phosphate activity was significantly higher in non-diabetics compared to diabetics. The results suggested that population of PDL cells in insulin dependant diabetics may be altered in their ability to form mineralized tissues and to respond to growth factor functions affecting the maintenance and regeneration of the periodontium (62).

Soory (2000) in an article discussing the role of hormonal factors in periodontal diseases, referred to the factors in the development of periodontal diseases and poor healing among diabetic patients. He reported that reduced neutrophil function, proteolytic enzyme activity, accumulation of advanced glycation end products, vascular changes such as thickening of the basement membrane, metabolic waste elimination, neutrophil migration and diffusion of antibodies may impaire wound healing due to their effects on cellular functions (63).

It has been reported in a position paper on diabetes mellitus prepared by Brian (2001), for the Research, Science and Therapy Committee of the American Academy of Periodontology that among the first references on diabetes and periodontal diseases was an article describing the inflamed gingiva from patients with diabetes to have sessile or pedunculated proliferations or polyps. It
was suggested that this gingival change was of significance in diagnosing patients with diabetes. The author indicated that the synthesis, maturation and homeostasis of collagen appear to be affected by glucose levels. Gingival fibroblasts from diabetic patients synthesize less collagen compared to non-diabetic subjects. Rats with experimentally induced diabetes have impaired production of bone matrix components by osteoblasts and decreased collagen synthesis by gingival and periodontal ligament fibroblasts. The investigator also found increased collagenase activity in gingival tissue in animals. Crevicular fluid and collagenolytic activity was also increased among diabetic patients. Collectively these results may indicate that the increased collagenase was endogenously derived independently of bacterial factors. Interestingly, the increased crevicular fluid and collagenase levels found in patients with diabetes can be inhibited in vitro by tetracycline(64).

In a hyperglycemic environment, numerous proteins including collagen undergo a non-enzymatic glycosylation process to form (AGEs) which play a central role in diabetic complications. It alters the function of numerous extra cellular matrix components, modifying matrix–matrix and cell-matrix interactions. AGEs formation result in collagen increased cross-linking between collagen molecules. This results in significant
reduction in solubility and decreases the turnover rate of collagen. Monocytes, macrophages and endothelial cells possess high affinity receptors for AGEs which bind to macrophage and monocytes inducing a hyper-responsive cellular state resulting in increased secretion of IL-1, insulin like growth factor, and tumor necrosis factor TNF-α. While endothelial cell binding result in pro-coagulatory changes leading to focal thrombosis and vasoconstriction. Clinically, diabetic subjects with periodontal diseases have significantly higher gingival crevicular fluid levels of both IL-1 B and PGE2 compared to non diabetic controls matched for periodontal disease severity. AGEs formation may result in the production of reactive oxygen intermediate and increases oxidant stress in gingival tissues of diabetic patients. This enhanced oxidant stress may be responsible for the vascular injury more common with diabetic complications. It is generally accepted that patients with diabetes are more susceptible to the development of infections than those without diabetes. Infections among diabetics are more sever than among non diabetics(44).

Debora (2002), in a review article reported that periodontitis has been referred to as the sixth complication of diabetes. A number of studies found higher prevalence of periodontal diseases among diabetic patients than among healthy controls. In a large cross-sectional study Grossi and others showed that diabetic
patients have attachment loss twice as non-diabetic subjects. In another cross-sectional study, Bridges and others found that diabetes affects all periodontal parameters including bleeding scores, probing depths, loss of attachment and missing teeth. In fact one study has shown that diabetic patients were five times more likely to be partially edentulous than non-diabetic subjects. People with type 1 and 11 diabetes appear equally susceptible to periodontal diseases and tooth loss. Moore et al, reported that for both type 1 and 11 diabetes there doesn’t appear to be any correlation between the prevalence or the severity of periodontal diseases and the duration of diabetes.  

1.5.1 Other Aspects of the Relationship Between Periodontal Diseases and Diabetes Mellitus:-

Periodontitis is a complex multi–factorial disease, similarly is diabetes mellitus, It is the complexities of both disease processes, which may contribute to the controversy found in the literature about their relationship. Many investigators have studied oral manifestations of diabetic patients including Periodontal disease severity, immunological responses of diabetic patients and controlling the effects of diabetes on the periodotium. Diabetes has long been identified by periodontists as a complicating factor in periodontal therapy. Both epidemiological studies and case reports have shown diabetes to be a major risk factor for periodontitis .A
substantial amount of literature supports the conclusion that patients with diabetes mellitus have an increased susceptibility to infections. It is also well documented that acute infections and inflammatory conditions increase glucose and insulin utilization, and therefore complicate the metabolic control of diabetes. Despite the fact that there is little scientific evidence to support the concept, it has been generally accepted that treatment of periodontal diseases in diabetic patients may reduce insulin requirements and improve metabolic balance. Lawrence et al (1992) performed a pilot study to evaluate the effect of controlling gingival inflammation on blood glucose level as determined by glycosylation of hemoglobin and albumin. The authors found it reasonable to conclude from their pilot study that controlling periodontal inflammation can alter the metabolic control of diabetes mellitus $^{(65)}$. Three considerations appear to be important in the appropriate interpretation of such conclusions. Firstly, caution must be used in interpreting results from such a small number of patients (200 patients). Secondly, periodontal disease is only one factor, and potentially relatively minor factor in the management of the metabolic control of diabetics. The third consideration is that it may not be possible or practical to control periodontal diseases in all poorly-controlled diabetic patients, but still the conclusions should not be overlooked.
Frank (1998), investigated periodontal diseases as a potential risk factor for systemic diseases in a paper presented to the Research, Science and Therapy Committee of the American Academy of Periodontology. The author reported that despite the legacy of the theory of focal infection, there has been a renewed interest over the last several years in the relationships between systemic and oral health. This may be in part due to the notion that dental medicine must become more integrated with general medicine, and to accumulating evidence that oral diseases may have clinically significant effect on general health. It is also clear that a number of systemic diseases and conditions are risk factors for periodontal diseases including diabetes mellitus. The association between diabetes and periodontal diseases is well documented. However, The converse possibility that periodontal diseases either predispose to, or exacerbates the diabetic condition has received only little attention. One study has noted that 7 out of 9 diabetics treated for periodontitis subsequently required reduced needs for insulin. Another study showed a reduction in the need for insulin following periodontal treatment of 9 diabetic subjects. These studies though preliminary, still provide intriguing clues to suggest that periodontal diseases may influence the course of diabetes.
A recent study of Pima Indians investigated the effect of periodontal treatment on the course of diabetes. Subjects were randomly assigned to 1 of 4 groups. All subjects received subgingival additional treatments, including systemic doxycycline and sub-gingival irrigation with chlorhexidine, systemic doxycycline and sub-gingival irrigation with povidone iodine or sub gingival irrigation with water alone (placebo). Glycated hemoglobin concentration was monitored at baseline and throughout the study. The results indicated that while all subjects experienced a reduction in periodontal disease, the groups receiving systemic doxycycline and sub-gingival irrigation with the anti microbial agents were clearly more improved relative to the placebo treated groups. All subjects treated with doxycycline experienced reduction in glycated hemoglobin. These results suggest that periodontal anti microbial treatment has the potential to reduce the level of glycated hemoglobin in diabetic subjects \(^{(67)}\).

Yoshihiro et al (2001), hypothesized that the TNF-\(\alpha\) produced due to periodontal inflammation synergistically affects insulin resistance as well TNF-\(\alpha\) produced from adipose tissue in insulin resistance type 11 diabetes. Therefore, to understand the effects of antimicrobial periodontal therapy on serum TNF –\(\alpha\) concentration and subsequent metabolic control of diabetes. 13 type II diabetics were examined. The results indicated that
antimicrobial treatment was effective in improving metabolic control in diabetic patients, possibly by reducing serum TNF-α and improved insulin resistance(68).

Anthony (2001), in a comprehensive report concluded that periodontitis is much more than a localized oral infection. Recent data indicated that periodontosis may cause changes in the systemic physiology. The interrelation between periodontitis can provide an example of systemic disease predisposing to oral infection, and once that infection is established the oral infection exacerbates the systemic disease. Hyperlipidemia may be one of the factors associated with diabetes induced immune cell alterations. Recent human studies have established a relationship between high serum lipid levels and periodontitis. Some reasonable evidence now suggests that periodontitis may lead to elevated low density lipoprotein cholesterol and triglycerides. Periodontitis cause elevation of serum proinflammatory cytokines IL-IB, TNF- α which have been demonstrated to produce alterations in lipid metabolism leading to hyperlipidemia. within this context, periodontitis may contribute to elevated cytokines/ serum lipid and potentially to systemic disease. Periodontitis exacerbate diabetes induced hyperlipidimia, immune cell alteration, diminished tissue repair capacity and may also be possible for chronic periodontitis to induce diabetes(69).
Grossi (2001) investigated the persistent elevation of IL-IB, IL6, TNFα in diabetes mellitus. She found that this elevation have an effect on the liver to stimulate the release of acute phase proteins, and produce dys-regulation of lipid metabolism. Collectively the evidence supports a role of cytokines elevation in the pathophysiology of a metabolic abnormality associated with diabetes (70). Grossi also have suggested that effective control of periodontal infection in diabetics reduces the level of AGES in the serum (44).

Debora (2002), in a review article referred to recent investigations, which attempt to determine if the presence of periodontal disease influences the control of diabetes (44).

1.6 Diabetes Mellitus and Periodontal Diseases in Children:

Few studies examined the relationship between diabetes mellitus and periodontal diseases in children.

Takanobu etal (1989), performed a study to determine if antibacterial antibody titers for selected periodontal disease associated microorganisms might be helpful in revealing changes in plaque flora at the onset and conclusion of puberty on 35 subjects aged 7-18 years. They selected a population of insulin-dependant diabetics (IDD) based on previous reports the results indicated that IDD, have increased level of gingivitis when compared to non-diabetic populations. They concluded that the relationship between oral flora and diabetes is unclear. Clinical measurements revealed that the number of bleeding sites increased with age and sexual maturation of children (71).
Sandholm et al (1989), studied the type of sub-gingival microflora from eighty-five 12-18 years old Finnish adolescents who are insulin dependant diabetics. A comparison was made with subgingival plaque samples from paired age and sex matched healthy controls. In conclusion the proportions of cocci and total gram-positive bacteria in those patients were lower than in control subjects. The proportion of periodontaly more pathogenic forms (gram-negative rods and total gram-negative bacteria), were higher in the diabetic patients. No difference was found in the spirochetes and flagellated bacteria between the two groups (72).

The clinical periodontal status of the same subjects had been reported in a separate study. All subjects were assessed clinically and radiographically. Data was also collected on the systemic diseases including duration of diabetes, daily insulin dose/kg and hemoglobin AIc-peptide and diabetic complications. The periodontal examination of all subjects was performed using the plaque index (Sinless and Löe, 1964), gingival index (Löe and Sinless, 1963) and the retentive calculus index. The parameters were recorded on the index teeth of Ramfjord (1959) and the number of teeth with gingival recession on the labial sites in the upper and lower arches was recorded and so was the number of overhangs. Pocket depth was measured on 4 sites on each tooth to the nearest mm with a standard probe. The number of points with bleeding on probing was recorded. Radiographic results showed that sites with bone loss around first incisors were 13 in 8 patients, non-of the subjects had radio-graphical bone loss exceeding 1mm.

The main results of the clinical examination revealed that the study group had more gingivitis than the control. There was no signification difference in plaque index between diabetics and control. The patients had no calculus although the retentive calculus index was low in both groups.
Gingival recession in one or more teeth was found in 11 patients and 17 controls, and no periodontal pocket exceeding 6mm was found in the 170 subjects studied. The age of the control didn’t correlate with any clinical or radiographical factors. The only corresponding correlation in the patient group was the positive correlation in the age with CPITN scores. The duration of diabetes was found to correlate positively with radio-graphical bone loss on the distal aspects (73).

Novaes et al (1991), evaluated the periodontal condition of insulin dependent diabetic children. Thirty insulin dependent and 30 controls aged 5-18 years were included in the study. Data was tested for correlation with age and sex in each group. The examination included evaluation of the gingival index, plaque index, radiographical examination for bone level assessment, pocket depths measurement using Michigan 0 type probe from the free gingival margin and recorded at six locations on each tooth. The results indicted higher accumulation of plaque among diabetic females and older patients, and higher gingival index among diabetics than controls with no significant difference with respect to age and sex. Pocket depth didn’t differ significantly between diabetics and controls in relation to increasing age, but in relation to sex. Diabetic females showed greater pocket depth on the palatal region. Alveolar bone loss was significantly greater in diabetics than in controls only at the anterior upper and lower regions (74).

Dominguez (1993), studied periodontal disease in type I diabetics and non-diabetic children. The investigation was a cross–sectional study on 383 individuals, 11-18 years old. The results indicated that periodontal disease prevalence of 37.53% and an epidemiological and statistical association was found between insulin –dependent diabetes mellitus, low social class, poor dental hygiene and plaque index over 0.2. The author concluded that type I
diabetic patients must be considered a high-risk group for periodontal diseases\textsuperscript{(75)}.

In an investigation on serum fructosamin and gingival health in insulin dependent diabetic children (IDDM), Firatili et al (1994) compared fructosamin assay (which is used in diagnosing and monitoring diabetics) with the hemoglobin and plasma glucose assays in children and adolescent type I diabetics. The authors demonstrated that, gingival index scores correlated with fructosamin values in insulin-dependent diabetes mellitus patients but not in non diabetic controls. They also found that there was no correlation between gingival scores and fasting plasma glucose and HBA1c values. Periodontitis was found to be rare in diabetic children and adolescents\textsuperscript{(76)}.

Pinson etal (1995), studied periodontal diseases in type I diabetic children and adolescents. The study compared the periodontal status of a type I diabetic group consisting of 26 type I diabetic patients with an age range 13-42 years and 24 control subjects of similar age and sex. The study group consisted of 26 type I diabetic subjects who were evaluated with glycosylated hemoglobin (GHb) to obtain a measure of diabetes control. Clinical periodontal evaluation was performed for all teeth in each subject, and included plaque index, gingival fluid flow, gingival index, probing depths, clinical attachment levels, recession, and gingival bleeding. Analysis of the data demonstrated no statistically significant differences in the overall parameters for the two groups. However, the comparisons were based on site-specific measurements and showed that the gingival index was somewhat higher among the diabetics (p= 0.0002)\textsuperscript{(77)}.

Ludovico etal (1995), studied the periodontal health status and sub gingival microflora of type I diabetic children with a mean age of 11.3 years
and compared that with healthy siblings with a mean age of 13.2 years. Patients were monitored every three months for levels of glycosylated hemoglobin (HbA1c). Clinical and microbial parameters were measured six weeks before drawing blood. Clinical indices measurements for the entire permanent dentition included probing depth, attachment level, sulcus bleeding index and plaque index. Subgingival plaque samples were obtained at two sites from each subject. Significant differences were not detected in the clinical parameters and subgingival micro-flora between the population of type I diabetics and their non-diabetic siblings. However, the diabetic patients in this study were well controlled and under continuous medical supervision with quarterly analysis for levels of (HbA1c). In this regard there is strong evidence that the degree of glucose control reflected by HbA1c levels is associated with, if not causally related to specific complications of diabetes. The fact that the diabetics in this study were well controlled may have reduced the possibility of the development of theoretical diabetes complication of periodontitis(78).

Firatili (1997), reported his five years study on the clinical periodontal status of 44 type I diabetic children and adolescents and 20 healthy controls. Fasting blood glucose, fructosamine and glycosylated hemoglobin values were determined at base line, and 5 years later. The differences between clinical and laboratory parameters were compared during the study period. The differences between the two groups were also evaluated. The only statistically significant difference observed in the diabetic group was clinical attachment loss (CAL). Statistically significant positive correlation was observed between the duration of diabetes and CAL. The author concluded that diabetes mellitus modifies the clinical status of the periodontal tissues and increase clinical attachment loss(79).
Material and Methods:-

Following approval of the protocol for this study, search started for centers where diabetic patients could be found with appropriate medical records. Jabir Abu Elizz center for diabetic patients management was the most appropriate study area. A pilot search indicated that the flow of patient might not be enough for the study. It was then decided to include Khartoum Paediatric Hospital and Omdurman General Hospital. Letters were then send to the authorities in these centres explaining the purpose of the study and asking permission for undertaking the investigation as well as ensuring co-operation (Annex i). Following permission, the study started by selecting subjects for the investigation.

Subjects:-

The test group was 40 insulin dependent diabetic children (16 boys and 24 girls) with an age range of 7 – 15 years. The study area was Khartoum at Jabir Abu Elizz diabetic center, Khartoum Pediatric Hospital and Diabetes Mellitus Patients Society in Omdurman Hospital. The later patients have been examined in Wad Nobawi dental center, on a dental chair with day light for illumination. All other patients were examined at the diabetic clinic, using the same criteria and methods.

The control group was 60 healthy non-diabetic children of the same age range (7- 15 years) (30 boys and 30 girls) at a basic school at the same area of Omdurman.

For type I diabetic children information about the systemic condition concerning age of diabetes onset, presence or absence of systemic complications, laboratory investigations for the level of diabetes control had been obtained from the routine follow up charts on the day of examination. The test group (diabetic type 1 children) was then divided according to metabolic control (Random Blood Sugar 120mg /dl and below this level was
considered as controlled). 30% of the test group were designated as controlled and 70% as non-controlled.

100% of the diabetic patients did not present with systemic complications (renal, retinal and other systemic diabetes complications). For both groups information regarding age of the individual, oral hygiene habits and current periodontal treatment was obtained.

Clinical Examinations:-
Examinations for both the healthy controls and the diabetic patients were performed by a single examiner, (Shadia), using a dental mirror and a periodontal probe (Michigan 0 type). Interest was only to determine the periodontal status of both groups at the time of examination.

The oral cavity was divided into sextants. The highest score of each periodontal parameter was recorded for each sextant.

The examinations for each tooth started by dental plaque detection according to the method of Silness and Löe (1964) (80):-
0- Absence of plaque deposits.
1- Plaque seen after probing the gingival margin.
2- Visible plaque.
3- Abundance of plaque.

Followed by periodontal pocket depth measurement in mm using Michigan 0-type probe from the crest of the free gingival margin to the bottom of the gingival sulcus, for the buccal proximal area. During the pocket probing bleeding from the gingival sulcus was observed to determine the gingival index according to the method of Löe and Silness (1963) (81).
0- Normal healthy gingiva.
1- Mild inflammation, mild change in color.
2- Moderate inflammation, redness, edema and bleeding on probing.
3- Sever inflammation, edema, ulceration and tendency to spontaneous bleeding.

Calculus was recorded on the basis of present or absent in the oral cavity of each individual. All data was recorded on special form (Annex ii) with the help of a trained assistant.

Data was then analysed with the help of statistician. The level of significance was pre decided at the 5% level (P <0.05) and the Confidence Interval at the 95% level.
Table 1 shows the distribution of the study sample according to age and sex. The sample comprised 100 subjects of which 40 test (diabetic type 1 children) and 60 control groups (non-diabetic children). The sample was subdivided according to age into two subgroups 7-11 with a mean age of 9 years, and 12-15 years with a mean age of 13.5 years.

Table 2 shows the number and percentage of sextants with different scores of the plaque index. Percentage of sextants with score 0 and 2 were almost similar in both test and control groups. Score 1 was higher in the control group than in the test group (60% and 40% respectively). While score 3 was higher in the test group compared to the control group (21% and 3% respectively). The difference was statistically highly significant (P = 0.00).
Table (1) The Distribution of the Study Sample According to Age and Sex.

<table>
<thead>
<tr>
<th>Age group/year</th>
<th>Mean age/years</th>
<th>Male No (%)</th>
<th>Female No (%)</th>
<th>Male No (%)</th>
<th>Female No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 -11</td>
<td>9.00</td>
<td>6(15)</td>
<td>12(30)</td>
<td>16(27)</td>
<td>18(30)</td>
</tr>
<tr>
<td>12 –15</td>
<td>13.50</td>
<td>10(25)</td>
<td>12(30)</td>
<td>14(23)</td>
<td>12(20)</td>
</tr>
</tbody>
</table>

Table (2) The Number and Percentage Sextants with Different Plaque Index Scores. (Test / Control)

<table>
<thead>
<tr>
<th>Subject (No)</th>
<th>Sextants</th>
<th>Plaque Scores No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>Test (40)</td>
<td>240</td>
<td>17 (7) 107 (45) 65 (27) 51 (21)</td>
</tr>
<tr>
<td>Control (60)</td>
<td>360</td>
<td>29 (8) 219 (60) 100 (28) 12 (3)</td>
</tr>
</tbody>
</table>

P = 0.00
Table 3 shows the number and percentage sextants with different scores of the gingival index.

For the test group only 13% presented with healthy gingiva (GI score 0), while the healthy gingiva represents 32% of the sextants examined in the control group. The highest GI score was score 1 in both groups (44% in test group and 59% in control group). The percentage of score 3 in the test group was 13% while it was less than 1% in the control group the difference was statistically highly significant (P= 0.00).

Table 4 presents the periodontal pocket depths in mm number and percentage of different depths in different sextants. Both groups presented mostly with pocket depth 0 - 3 mm. Only 15% of the test group showed pocket depth more than 3 mm but among the control group only 3% showed pockets more than 3 mm. The difference was statistically highly significant (P =0.00).
Table (3) The Number and Percentage Sextants with Different Scores of the Gingival Index (Test /Control).

<table>
<thead>
<tr>
<th>Subject (No)</th>
<th>Sextants</th>
<th>Gingival Scores No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Test (40)</td>
<td>240</td>
<td>30(13)</td>
</tr>
<tr>
<td>Control (60)</td>
<td>360</td>
<td>117(32)</td>
</tr>
</tbody>
</table>

P= 0.000

Table (4) Periodontal Pocket Depth and Sextants with Different Depths in mm (Test / Control)

<table>
<thead>
<tr>
<th>Subjects (No)</th>
<th>sextants</th>
<th>Depth mm No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0mm</td>
<td>1mm</td>
</tr>
<tr>
<td>Test (40)</td>
<td>240</td>
<td>85(36)</td>
</tr>
<tr>
<td>Control (60)</td>
<td>360</td>
<td>22(6)</td>
</tr>
</tbody>
</table>

P = 0.00.
Table 5 Shows the presence of calculus in the test and control groups.
In 70% of the test group calculus was present while in the control group it was only 32%. The difference was statistically highly significant. (P=0.00)

Table 6 Shows the frequency of cleaning the mouth per day.
Most of the subject in both groups used to clean the mouth once per day (57% of the test and 76% of the control).
The difference between the two groups was statistically not significant.

Table 7 Shows previous or current periodontal treatment.
100% of the test group did not experience any periodontal treatment and so did 98% of the subjects in the control group.
Table (5) Presence of Calculus, No and Percentage.(Test / Control)

<table>
<thead>
<tr>
<th>Subjects (No)</th>
<th>Calculus No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>present</td>
</tr>
<tr>
<td>Test (40)</td>
<td>28(70)</td>
</tr>
<tr>
<td>Control(60)</td>
<td>19(32)</td>
</tr>
</tbody>
</table>

P=0.00

Table (6) Frequency of Cleaning the Mouth /day

<table>
<thead>
<tr>
<th>Subject (No)</th>
<th>Frequency of cleaning / day No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non</td>
</tr>
<tr>
<td>Test (40)</td>
<td>1(3%)</td>
</tr>
<tr>
<td>Control(60)</td>
<td>1(2%)</td>
</tr>
</tbody>
</table>

P=0.263

Table (7) The Number and percentage of Subjects who Experienced Periodontal Treatment

<table>
<thead>
<tr>
<th>Subject (No)</th>
<th>Periodontal Treatment No(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Test (40)</td>
<td>0.0 (0%)</td>
</tr>
<tr>
<td>Control (60)</td>
<td>1 (2 %)</td>
</tr>
</tbody>
</table>
Table 8 Shows the duration of diabetes mellitus for the study sample.

88% of the patients experienced the disease for less than 5 years. Only 12% experience it for more than 5 years, non-of the test group experienced the disease for 10 years or more.

Table 9 Shows the plaque index scores of different sextants for the controlled and non-controlled diabetic subgroups.

14% of the sextants in the non-controlled sub group were plaque free while only 1% of the controlled sub group present plaque.

The scores 1, 2 and 3 were high in the controlled sub group compared to the non-controlled. The difference was statistically significant (P= 0.026).
Table (8) The Duration of Diabetes

<table>
<thead>
<tr>
<th>Duration</th>
<th>subjects No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 5 years</td>
<td>35 (88)</td>
</tr>
<tr>
<td>≥ 5 years</td>
<td>5 (12)</td>
</tr>
<tr>
<td>Total</td>
<td>40 (100)</td>
</tr>
</tbody>
</table>

Table (9) The Number and Percentage Sextants with Different Plaque Index Scores for the Controlled and Non-controlled diabetic Subgroups.

<table>
<thead>
<tr>
<th>Diabetic Subgroup (No)</th>
<th>Sextants</th>
<th>Plaque score No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Controlled group (12)</td>
<td>72</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Non controlled (28)</td>
<td>168</td>
<td>23 (14)</td>
</tr>
</tbody>
</table>

P = 0.26

Table (10) Shows the gingival index scores of different sextants for the controlled and non-controlled diabetic subgroups.

15% of the sextants of the non controlled subgroups presented with score 0 gingival index while only 5% of the controlled diabetics presented with clinically healthy gingiva (score 0). Gingivitis was sever in the
controlled subgroup, score 3 was present in 21% of the sextants examined in this subgroup. Only 10% showed score 3 of the sextants examined in the non controlled subgroup. The results were statistically significant (P = 0.006).

Table (11) shows the periodontal pocket depth in mm for the controlled and non-controlled diabetic subgroups.

Twenty five percent of the sextants examined in the controlled subgroup and 43% of the sextants examined in the non controlled subgroup showed pockets of less than 3 mm, 8% of the sextants examined in the controlled sub group showed pocket depth greater than 3 mm, while 18% of the sextants examined in the non controlled sub group showed pocket depth greater than 3 mm. The difference was statistically highly significant (P = 0.00).
Table (10) Number and Percentage Sextants with Different Scores of the Gingival Index in the Controlled and Non controlled Diabetic Subgroups:

<table>
<thead>
<tr>
<th>Diabetic Subgroup (No)</th>
<th>Sextants</th>
<th>Score No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Controlled (12)</td>
<td>72</td>
<td>4 (5)</td>
</tr>
<tr>
<td>Non controlled (28)</td>
<td>168</td>
<td>25 (15)</td>
</tr>
</tbody>
</table>

P=0.006

Table (11) Periodontal Pocket Depth and sextants with different depth in mm for the Diabetic Subgroups

<table>
<thead>
<tr>
<th>Diabetic Subgroup (No)</th>
<th>Sextants</th>
<th>Depth in mm No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0mm</td>
</tr>
<tr>
<td>Controlled (12)</td>
<td>72</td>
<td>18 (25)</td>
</tr>
<tr>
<td>Non controlled (28)</td>
<td>168</td>
<td>73 (43)</td>
</tr>
</tbody>
</table>

P = 0.00
Table (12) shows the calculus presence in the controlled and non-controlled sub groups.

Seventy-five percent of the sextants examined in the controlled subgroup showed calculus, while 68% of the sextants examined in the non controlled subgroup showed calculus. The difference was statistically not significant (P = 0.651).

Table (12) The Presence of Calculus Number and percentage in the Diabetic Subgroups

<table>
<thead>
<tr>
<th>Diabetic subgroup (No)</th>
<th>Calculus No (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present</td>
</tr>
<tr>
<td>Controlled group (12)</td>
<td>9(75)</td>
</tr>
<tr>
<td>Non controlled (28)</td>
<td>19(68)</td>
</tr>
</tbody>
</table>

P = 0.651
4.0 Discussion

1- **Study sample:** The study sample was one hundred subjects, distributed according to age and sex as shown in table 2. The sample is comprised of 40 diabetic children and 60 non-diabetic controls. The number of diabetic children is reasonable because the whole number of diabetic patients (children and adults) registered in the diabetic centers visited was 300 subjects of different ages.

2- **Plaque Index:** In the present study the plaque index scores were higher among diabetics than among the control group. The difference was statistically significant as shown in table 2. (P= 0.00) This result may be due to the fact that, while parents of the children examined are oriented towards the systemic problem of their children they are unaware of the importance of oral cleanliness. The present result agrees with those obtained by Novaes et al (1991) and Dominguez et al (1995), and disagree with Tellervo et al (1985) who studied an adult population and also disagree with that of Sandholm et al (1989) who studied a socially and economically different population, as well as with that of Pinson et al (1995) and Ludovico et al (1995).

The results of the plaque index scores for the diabetic controlled and non-controlled subgroups is shown in table 9. While the plaque index was generally higher in the diabetic group, score 1,2 and 3 were higher in the controlled subgroups than among the non controlled, and the difference was statistically significant (P=.026). No reason could be speculated for this difference. However, the issue requires further detailed in depth investigation.
3- **Gingival Index:** The results of the gingival index showed that score 3 was higher in the test than in the control group (13% of the test and 1% of the control group). As shown in table (3), the difference was statistically highly significant (P= 0.00). This result is in agreement with that of Takanobu et al (1989) (71), Sandholm et al (1989) (72), Novaes et al (1991) (74), Firatili et al (1994) (76) and Pinson et al (1995) (77), but disagrees with the results of Tellervo et al (1985) (50), because they studied an older adult population and also disagree with Ludovico et al (1995) (78).

The gingival index of the diabetic subgroups showed that gingivitis was sever among the controlled group. Score 3 presented in 21% of the sextants examined while 10% obtained score 3 in the non-controlled group, and the difference was statistically significant (P=.006). As shown in table 10, the result disagree with those of Tellervo et al (1985) (50), and Seppala et al (1997) (59). This difference may be due to oral hygiene practices and available professional care. However, these results can not be explained and may require further investigations.

4- **Periodontal Pocket Depth:** Both test and control groups presented mostly with pocket depth between 0-3mm. However, 15% of the test group showed pocket depth greater than 3mm while only 3% of the control group showed pocket depth more than 3 mm. The difference was statistically significant as indicated in table (4). The results disagree with those of Milijenko et al (1988) (52) who studied an adult population of diabetics using CPITN. The results also disagree with those of Ludovico et al (1995) (78), (1998) (60), Novaes et al (1991) (74), Pinson et al (1995) (77), and Firatili et al (1997) (79).
Periodontal pocket depth for the diabetic controlled and non-controlled subgroups, showed that 18% of the sextants examined in the non controlled subgroup showed pocket depth greater than 3mm, while only 8% of the controlled sub group presented with pocket depth greater than 3mm. The difference was statistically highly significant (Table 11). This result indicates that the level of metabolic control affects the periodontal health in diabetic children, though the oral hygiene and gingival inflammation was better among the controlled subgroup. This result agrees with those of Seppala et al (1997) (59), and Ludovico et al (1995) (78), and disagree with the results of Sastrowijito two studies in (1989) (53) and (1990) (54).

5- Calculus: In 70% of the test group calculus was not present, while in only 32% of the control it was present. The difference was statistically highly significant (P= 0.00). The results disagree with those of Sandholm et al (1989) (73). The only explanation for these results may be the degree of oral hygiene neglect leading to increased amount of dental plaque.

As for the controlled and non-controlled diabetic subgroups there was a small difference in calculus between the two subgroups (75% and 68% for the controlled and non-controlled subgroups respectively). However, while no reasoning could be given for this results, the available literature is showing no studies examining calculus presence among diabetes in relation to disease control.

6- Indicators of Oral Health Behavior: The results of the frequency of cleaning the teeth showed that most of the subjects examined in both the test and control groups used to clean their teeth once per day and the difference between the two groups was statistically not significant. However, still other periodontal disease indicators show that rushing practiced was not efficient.

The entire test group and 98% of the controlled group didn’t experience periodontal treatment before. This may indicate lack of dental care
and/or lack of awareness of the importance of visits to the dentist. The results agree with those of Paul et al (1999)\(^{(61)}\) and disagree with those of Kaisa et al (1994)\(^{(58)}\).

7- **Systemic Complications and Duration of Diabetes**: All the diabetic patients didn’t present other systemic complications of diabetes. 88% of the patients experienced the disease for less than 5 years and only 12% of the patients for more than 5 years.

These findings indicate that the results of this study were not influenced by the duration and/or complications of diabetes mellitus. The present results are in agreement with that of Kaisa et al (1994)\(^{(58)}\), Ludovico et al (1995)\(^{(78)}\) and Firatili et al (1997)\(^{(79)}\). And disagree with the results of Tellervo et al (1985)\(^{(50)}\), Milijenko et al (1988)\(^{(52)}\) Sastrowijoto et al (1990)\(^{(54)}\), and Tervonen et al (1994)\(^{(57)}\).

In general the results of the present investigation show that periodontal health of diabetic children may be affected by the diabetic disease process and its control. So both families and the profession should give especial attention for those children in order to help them keep an intact dentition for life and help control the diabetic condition because both periodontal diseases and diabetes affect each other.
Conclusions:

From the results of this study we can conclude the following:

1. Plaque index scores are higher among diabetic children than among the healthy controls.

2. Gingivitis was more prevalent among diabetics compared to healthy controls.

3. Periodontal pocket depth was generally rare in both test and control groups, but it was significantly higher among the diabetic children.

4. The level of diabetes control may have an effect on the periodontal health.

Recommendations:

From the conclusions of this study we may recommend the following:

1. Special oral health care should be availed for diabetic children at both family and professional levels.

2. Dentists and physicians should be made aware of the periodontal problems facing diabetic patients especially children so as not to underestimate their role in motivating and encouraging them for better oral hygiene practices.

3. A longitudinal study should be carried out to further investigate the periodontal health in relation to diabetes control, duration of diabetes and oral hygiene measures among the diabetic children.
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