University of Khartoum
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Periodontal Health of Diabetic Patients in Khartoum

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Supervisor:
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Dedication

To my Husband, Children ...
To my Father and Mother ...
To all who can benefit from this study ...

Mona
January 2003
Acknowledgement

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Abstract:

Diabetes has been reported repeatedly in association with periodontal disease specially in younger people, but no one was able to prove that diabetes is a specific cause of severe periodontal disease. As a matter of fact many people with diabetes has normal periodontal tissues. Although the relationship between diabetes and periodontal disease appears to be strong, the corresponding variables inherent in studying two chronic diseases may result in less clear association in a selected population.

The present investigation is carried out in Khartoum State during the year 2002, one hundred dentate patients (50 for test and 50 for control) with an age range 30 - 55 years. 50 of them were diabetic patients randomly selected from patients routinely attending Jabir Abu Elizz Diabetic Center. The control group (non-diabetic) was selected randomly from patients attending the Dental Clinic of the Faculty of Dentistry and Khartoum Teaching Hospital. The study group was assessed, examined and investigated for diabetic status. The periodontal health of the test and control groups was examined using a graduated periodontal probe and a mouth mirror. For all subjects the plaque index, gingival index, calculus, probable pocket depth and attachment loss were recorded. Higher mean gingival scores and higher mean levels of attachment loss were recorded among diabetic patients, which is not related to the amount of plaque or calculus.

The results of this study agree with studies elsewhere in the world that diabetic patients are at a higher risk for the development of periodontitis, hence the control of periodontitis is beneficial to diabetics and vice versa. It can be recommended that periodontists, dental hygienists should be partners in diabetes
control teams, and dental clinics should be part of every diabetic center, as well as the need for continuous oral health education and motivation programmes for diabetics.
البحث الخلاصة

الأمراض

السكري

ذكر ورد وتكراراً من الضامة والأنسجة.

ونجد ذلك عليه ورغم أنه لا أعطي بالسكري أنه على قاطع الأساسي،

الأنسجة للأمراض السنية.

مصابل كثيرون أشخاص هنا كثيرة، والسكري نسيجة سليمة سنية ضامة.

و عليه الرغم بين العلاقة أنه من المرض الأنسجة والأمراض كيرة تبدو الضامة كأن تكون النتائج أنه نجد الأحياء كثير في غير

الدراسة هذه في البحث أجريت السكر مريض، ماما اختياراً الشخصية

المرضية مرن وكسارية مريض علاج.

يعاني الآخرون وخمسن مريضة من السكر وشوى أخذ في السكر وفحص المريضة من المجموعة فحم تمت المريضة الكلية في الصحة.

للحال السكري مقرر الفحص الأخصائي ومقابلة المريض تاريخ بأخذ عندهم السكر والفحص المسؤول.

وغير المريض من الجمعية في الفحص تم المريضة الثلة الأمراض للحال بسكي.

الجرثومية الويتوبة استعمال تم) سيلن، ولو و1964 (اللوتي والدبليو)

واللوكس، وسو 1963 (الجرية المواد وترسيب الطالقية الأتصال فقداً أن الأسنان، جيوب وعمق،

تم أسنان ومرأة رذران باتخدام الفحص.

كالأتي الدراسة النتائج كأن -

: أوعى السكر الرغبة

: أوعى السكر الرغبة

: أوعى السكر الرغبة
تأثير السكر على الأمراض السائبة.

قد يؤثر السكر على الأنسجة الأمراض والبائية.

لقد أ настоящее الأنسجة، الأمراض السائبة تطور على تأثير السكر وقد يصبح المشوير.

توصيحة:

1. ينبغي أن يكون الأنسجة، الأمراض السائحة في أشراف الأسنان، والكود، الأنسجة، الأنسجة.

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Introduction and Literature Review

1-1 Introduction

The relationship between periodontal disease and diabetes has been described in many clinical studies. Most investigators reported a higher incidence and severity of periodontal disease among diabetics. Several other investigators found no association between this metabolic disorder and the degree of periodontal disease. An interesting link joins diabetes and periodontal disease, that their management depend on patient education and motivation and clinicians do nothing without patients taking their part in prevention and elimination of the etiologic factors. It is suggested that diabetes long term duration, long term poor control and organ complications may be associated with the severity of periodontal disease.

But as there is no consistent results on the effects of diabetes on periodontal disease in Sudan, new data collected from Sudanese population is needed. Therefore the present study has been designed to collect such data.

The objective of the study:

1. To evaluate periodontal health of diabetic patients.
2. To compare periodontal health status of diabetics and non-diabetic patients.
3. To compare periodontal health of controlled and uncontrolled diabetic patients.
1-2 Literature Review

1.2.1. Periodontal disease:

Periodontal disease has been defined as "any pathological process affecting the periodontal tissue" (1).

The term periodontal disease has been given different meanings and its is used in a general sense to encompass all diseases of the periodontium. Traditionally periodontal disease have been divided into two major categories gingival diseases and periodontal diseases (2).

The prevalence of destructive disease follow a linear progression from a adolescence to old age. The strong correlation with age probably reflects the cumulative effect of the disease rather than diminished resistance (3).

The primary cause of periodontal disease is bacterial plaque irritation. However small amounts of plaque are compatible with gingival and periodontal health, and some patients may resist larger amounts of plaque for long periods without developing destructive periodontitis (4).

A number of other factors both local and systemic predispose towards plaque accumulation or alter the tissue response to plaque. They may be regarded as secondary aetiological factors (5).

Systemic factors are those factors which act on the body as a whole and they do not cause periodontal disease but alter tissue response to plaque irritation (6).

Periodontal disease is a direct consequence of colonization of the gingival sulcus by the organisms of dental plaque (1). However this is an over simplification and it is more complex than this statement as it may involve:

- Host defense mechanisms.
- Oral environment
- Pathogencity of organisms
1.2.2 Diagnosis of Periodontal Disease:
In spite of the increased understanding of the etiology and pathogenesis of periodontal infection, diagnosis is still based almost entirely on traditional clinical assessments. (7)

1.2.3 Treatment of periodontal diseases:
Dental plaque causes periodontal disease, so control of plaque accumulation is the means by which preventive programmes can be made effective (8). Meticulous removal of plaque will usually result in resolution of the chronic gingivitis lesion without residual tissue destruction (9). Therapeutic approaches for periodontitis fall into two major categories: anti-infective treatment, which is designed to halt the progression of periodontal attachment loss by removing etiologic factors (10). And regenerative therapy which is intended to restore structures destroyed by disease.

Host responses may also have an effect on treatment outcome and patients with systemic conditions may not respond well to therapy that is directed solely at controlling local factors. In such patients it is important that an attempt be made to control the contributing systemic factors (11). Maintenance procedures should be part of the long term treatment plan (12).

1.2.4 Diabetes mellitus:
Diabetes mellitus is a disease syndrome due to deficiency of insulin available to the tissues. It is thus a fundamental disorder of carbohydrate metabolism with
consequential changes of widespread nature affecting every tissue and organ in the body (13).

The increased glucose contents of gingival fluid and blood of diabetics could change the environment of the micro-flora inducing qualitative changes in bacteria that could affect periodontal changes.

Diabetes Mellitus occur in two principal types primary (type 1 & 2), and secondary. Type 1, sometimes called juvenile diabetes, begins most commonly in childhood or early adolescence. In this form of diabetes the body produces little or no insulin. It is characterized by sudden onset (14).

The more common form of diabetes is type 2, sometimes called adult onset diabetes. This form occurs more common in people who are overweight and who do not exercise (14).

Secondary diabetes can also develop as a result of pancreatic disease, alcoholism, malnutrition or other severe illnesses that may stress the body (15). Sometimes another form of diabetes is called gestational diabetes which may develop during pregnancy and resolve after the baby is delivered (15).

Traditional symptoms of diabetes mellitus are polyphagia, polydipsia, polyuria, predisposition to infection and anorexia (16).

Many findings are described among uncontrolled diabetics in the oral mucosa including, cheilosis, drying and cracking, burning sensation, xerostomia, alteration in the flora of oral cavity with predominance of candida albicans, hemolytic strepto cocci and staphylo cocci (17).

And the most striking changes are the reduction in defense mechanisms and increased susceptibility to infection leading to destructive periodontal disease (17).

On the other hand among controlled diabetics none of the above changes is found and there is normal tissue response and normal defense against infections.
However the possibility that the control may be inadequate makes it advisable to exercise special care in the periodontal treatment of individuals with controlled diabetes (17).

A variety of periodontal changes have been described among diabetic patients such as tendency towards abscess formation (Fig. 5), diabetic periodontoclasia, enlarged gingiva (Fig. 4), sessile or pedunculated gingival polyps, polypoid gingival proliferation and loosened teeth (17).

Diagnosis of suspected individuals is based on symptoms, urine and blood tests can be used to confirm the diagnosis of diabetes (15).

1.2.5 Treatment of DM:

There is currently no cure for eradication of diabetes; the condition however can be managed so that patients can live a relatively normal live (15).

Treatment focuses on two goals, keeping blood glucose level within a normal range and preventing the development of complications (15).

Careful monitoring of diet, together with exercise are as important as the use of insulin or oral medication in preventing the complications of diabetes (15).

1.2.6 Complications of diabetes:

The complications of diabetes are related to long term elevation of blood glucose concentration (hyperglycaemia) (18).

Long term complications may occur in both type 1 and type 2 diabetes. Hyperglycemia is now known to lead to chronic microvascular and macrovascular complications. Macrovascular complications include coronary artery disease, cerebrovascular disease and peripheral vascular diseases (18).

Microvascular complications include retinopathy, nephropathy and neuropathy. Retinopathy may lead to blindness, whereas progressive renal disease can lead
to kidney failure. Peripheral neuropathy may lead to gangrenous infections leading to amputations of limbs and dyesthesias. In terms of oral manifestations, patients may experience delayed wound healing and xerostomia as well as periodontal destruction (18).

1.2.7 Dental Therapy and Effects on Diabetes:
The initial dental therapy for patients with diabetes must be directed towards control of acute oral infections. At the same time communication may be established with the patients physician so that a treatment plan can be developed to obtain control of blood glucose level (19).
It is important to advise the physician on the periodontal status, since the presence of infections including periodontal disease may increase insulin resistance and contribute to worsening of the diabetic state (19).

1.2.8 Timing of Dental Treatment for Diabetic Patients:
Patients with well controlled diabetes can be treated similarly to non-diabetic patients for most routine dental needs. Procedures should be short, atraumatic and as stress free as possible (19).
Patients should be instructed to take their medications as prescribed and to continue on diet control and self monitoring of glucose levels during the course of dental treatment. Early morning appointments are often preferred (19).

1.2.9 Diabetes and periodontal disease:
Microbial dental plaque is the initiator of periodontal disease, but whether it affects a particular subject, what form the disease takes, and how it progress are all dependent on host defenses to this challenge (20).
The periodontium is an interesting model for the manifestation of hormone mediated action on connective tissue and bone, owing to its communication with the oral cavity and vulnerability to inflammatory changes (21). Systemic factors may modify all forms of periodontitis principally through their effect on the normal immune and inflammatory defenses. Some good example of this effect is when there is reduction in number or function of polymorphonuclear leukocytes (PMNL), that may result in increased rate and severity of periodontal destruction. Many other systemic factors are much less clear cut and are difficult to causally link to periodontitis. In many cases the literature is insufficient to make a definite statement on links between systemic factors and periodontitis (20).

The possible role of systemic disease and systemic exposures (such as smoking) initiating or modifying the progress of periodontal disease is clearly a complex issue (20).

It is however generally agreed that several conditions may give rise to an increased prevalence, incidence or severity of gingivitis and periodontitis (20). Diabetes in general has a significant impact on tissues throughout the body, including the oral cavity. Research indicates that diabetes specially when poorly controlled increases the risk for periodontitis (16).

In fact it must be recognized that in uncontrolled diabetes many metabolic processes are affected including those which make up resistance to infection or trauma. For example the uncontrolled diabetic patients may suffer from persistent chronic ulcers of skin of legs, presumably because resistance is lowered and any minor irritation such as trauma or bacterial infection will result in injury greater than in a normal person. Also the effectiveness of healing process is decreased probably as a result of disturbance in cellular carbohydrate metabolism (6).
Considering that the periodontium is located in the oral cavity with its many factors predisposing to disease including bacteria, calculus and trauma, it is not surprising that this structure appear to breakdown more readily in patients with uncontrolled diabetes than in normal persons.(21).

However, diabetes does not cause gingivitis or periodontitis but it may:

- Alter tissue response to local irritation.
- Hasten bone loss
- Retard post surgical healing of periodontal tissues.

And in the presence of plaque associated inflammatory changes, diabetes mellitus and many other systemic diseases influence the presentation and progression of periodontal disease (6).

A majority of studies showed higher prevalence and increased severity of periodontal disease in diabetics than non-diabetics with similar local irritation (17).

The interrelationship between diabetes mellitus and periodontal disease provide an example of systemic disease predisposing to oral infection. Once the oral infection is established, it exacerbates the systemic disease. To understand this we must identify common physiological changes associated with diabetes and periodontal disease which produce synergy when the two conditions coexist (22).

In many cases the literature is insufficient to make definite statement on links between systemic factors and periodontitis (20).

Periodontal disease has been called the sixth complication of diabetes. Studies conducted throughout the world suggest that some diabetics are at increased risk for periodontitis (20).

Also it has been suggested that severe periodontitis may be an important risk factor in the progression of diabetes and that physicians should consider the
periodontal status of their diabetic patients having difficulty with glyceamic control (12). Despite the fact that there is little scientific evidence to support the concept, it has been generally accepted that treatment of periodontal disease in a diabetic patient may reduce insulin requirement and improve the metabolic balance (23). The prevalence of diabetes and its probable influence on periodontal disease suggests that, diabetics will very likely become an increasing part of the patients population seen by both general dentists and periodontists (16).

1.2.10 Factors Potentially Contributing to the Development of Periodontal Disease in Diabetics:

1.2.10.1 PMN leukocyte function:
PMN leukocytes are very important for the maintenance of periodontal health because they are the principal protective cells of the normal periodontium through out life. The function of these cells especially in relation to pathogenesis of periodontal disease have been comprehensively reviewed in adults by Genco and Slots (24), and in children by Kalkwarf and Mcley (25). There appears to be little doubt that quantitative or qualitative defects of PMNL in an individual, predispose to rapid and severe periodontal destruction (26). The depression of PMNL functions particularly chemotaxis but also phagocytosis, are held to be responsible for the severe periodontal disease entities such as those seen in diabetes mellitus (Golub et al 1983) (27). Reduced PMNL function has been found in patients with diabetes. This impairment of function was noted in assays of PMNL chemotaxis, adherence and phagocytosis. This PMNL defect suggests that this dysfunction could lead to impaired host resistance to infections (19).
The severity of periodontitis has been correlated with defective chemotaxis. Further decrease in PMNL chemotaxis has been reported in a family with a history of diabetes and severe periodontitis suggesting that the PMNL defect is of genetic origin (19).

A local effect has been suggested since PMNL phagocyte activity of gingival crevicular fluid (GCF) was less than that of peripheral blood, irrespective of the diabetic state. The functional activity of PMNL collected from diseased sites was less than that of healthy sites (19).

PMNL defect has been studied in rats chemically treated to induce diabetes. The reduction in peak chemotactic response of crevicular PMNL was 45%, 66%, 71% at days 4, 14, and 20 respectively. Uncontrolled diabetes of 20 days duration reduce the peak neutrophil response by 83%. Importantly, diabetic rats receiving insulin showed a reduction of only 34% in the chemotactic response of crevicular PMNL. (19).

1.2.10.2 Collagen Metabolism and AGE:

Collagen synthesis, maturation and homeostasis appear to be affected by glucose level. Skin fibroblast studies has shown that hyperglycaemic conditions reduce fibroblast cell proliferation and growth and reduce synthesis of both collagen and glycosaminoglycans. In addition gingival fibroblasts from diabetic patients synthesize less collagen compared to non-diabetic subjects (19).

Rats with experimentally induced diabetes have impaired production of bone matrix components by osteoblasts and decreased collagen production in association with diabetes (19).

Investigators have also found increased collagenase activity in gingival tissue. Crevicular fluid collagenolytic activity has increased in diabetic patients. This activity appear to be primarily of neutrophil origin. These results indicate that
the increase of collagenolytic activity was indigenously derived, independent of bacterial factors (19).

In the hyperglycaemic environment numerous proteins including collagen undergo non-enzymatic glycosylation process to form Advanced Glycation End product (AGE). The formation of AGE play a central role in diabetic complications. AGE accumulate with chronic hyperglyceamia (19).

AGE formation alter the function of numerous extra cellular matrix components modifying matrix – matrix and cell – matrix interaction. This alteration have an adverse effect on target tissues especially collagen stability and vascular integrity (19).

AGE formation on collagen result in increased cross linking of collagen molecule which contribute to reduced solubility and decreased collagen turn over rates (19).

Consistent with these results, the gingival collagen fibers of diabetics shows decreased solubility properties (19).

1.2.10.3 Monocytes, Macrophages, and Endothelial Cells:

These cells have high affinity receptors for AGE. AGE binding to monocytes and macrophages receptors may induce hyper responsive cellular state resulting in an increased secretion of IL-1, insulin like growth factors and TNF-α. While endothelial cells binding result in pro-coagulatory changes leading to focal thrombosis and vasoconstriction (19).

Monocytes from patients with diabetes produce significantly greater amounts of TNF- α, IL-1β, PGE2 in vitro than do non-diabetic controls (19).

Clinically, diabetic patients with periodontitis have significantly higher GCF of both IL-1 β and PGE2 compared to non-diabetic controls (19).
AGE mediated events are of primary importance in the pathogenesis of diabetic complications such as retinopathy, nephropathy, neuropathy and atherosclerosis. They may also be involved in tissue changes within the periodontium rendering the diabetic patient with poor glycaemic control and elevated AGE susceptible to increased tissue destruction (19).

AGE formation result in production of reactive oxygen intermediate. AGE in gingival tissue have been shown to increase oxidant stress in these tissues when compared to non-diabetic individuals. This enhanced oxidant stress may be responsible for the vascular injury common to diabetic complications (19).

1.2.10.4 Infection and diabetes:

Diabetes mellitus may predispose to many bacterial, viral and fungal infections, including those affecting the urinary and respiratory tract, oral cavity, periodontium, soft tissue and bone (13).

In particular infected foot ulcers and osteomyelitis, oro-pharyngeal and oesophageal candidosis. Rare infections such as necrotizing fasciitis, malignant otitis externa, mucor mycosis and fungal meningitis may occur. However no strong association between diabetes and increased susceptibility to infection has yet been demonstrated, although there is strong association between poorly controlled diabetes and various infections (18).

This increased susceptibility to infections, is probably multi factorial and include:

- the effect of hyperglycemia on inflammatory and humoral immune response with impaired chemotaxis, phagocytosis and intra cellular killing of microorganisms
- changes in the salivary flow and composition in diabetes may predispose patients to oral infections.
Some of these changes may improve as diabetes control improves. The incidence of loss of periodontal attachment in patients with diabetes is no greater than in the average population if metabolic control and oral hygiene were satisfactory (18).

It is generally accepted that infections in diabetic patients are more severe than the same infections in non-diabetic individuals. However conclusive studies supporting these clinical impressions do not currently exist.

Insulin resistance is the condition that exist during acute infections, this is independent of the diabetic state. Hyperglycemia and hyperinsulinemia after oral glucose administration are hallmark findings of insulin resistance. Significantly insulin resistance has been found to exist 1-3 weeks in the non-diabetic state after resolution of infection.

Vascular changes are common in patients with diabetes, basement membrane protein become glycosylated in a hyperglycemic environment, thickening and changes in physical properties (19).

Gingival capillaries of diabetic subjects show thickened basement membrane as well as disruption of collagen fibres within basement membranes, with swelling of the endothelium. These changes can be hypothesized to impede oxygen diffusion, metabolic waste elimination, PMNL migration and diffusion of serum factors including antibodies (19).

Other studies have failed to show any difference in thickness of basement membranes of gingival vascular tissue in diabetic patients.

Collectively defects of PMNL, induction of insulin resistance and vascular changes can all contribute to increased susceptibility to infections.

Importantly control of serum glucose level appear to partly reverse these factors and should therefore be closely monitored with infections (19).
1.2.10.5 Wound Healing:
The mechanisms leading to compromised wound healing in individuals with diabetes are unknown. It is probable that the cumulative effects of altered cellular activities which play part in susceptibility to infections also affect wound healing (19).
In addition decreased collagen synthesis by fibroblasts, increased collagenase production found in diabetics may play a role in wound healing. Glycosylation of existing collagen at the wound margins may result in reduced solubility and delayed remodeling of the wound site. Increased collagenase can readily degrade the newly synthesized less completely cross linked collagen, this contribute to further defective wound healing.
The late inflammatory response to wound healing has been found to be altered in diabetics (19).
Wound champers in diabetics, showed marked difference in both cellular infiltration of PMNL and cytokine levels of TNFα and IL-6.
The skin wounds in diabetics are analyzed bio-chemically for strength, toughness and elasticity and it was found that wounds in diabetics are the weakest and had the lowest elasticity.
The mitogenic activity of platelets from patients with diabetes had been found to be decreased. Platelets induce significantly less proliferation of fibroblasts than did platelets from non-diabetics (19).

1.2.11 Periodontitis and Diabetes Inter-relationship, Role of Inflammation:
Diabetes mellitus is a systemic disease with several major complications affecting both the quality and duration of life, periodontal disease is one of these complications. Recent data indicate that periodontitis may cause changes in systemic physiology, and the inter relationship between periodontitis and
diabetes provide an example of systemic disease predisposing to oral infections, and once that infection is established, the oral infection exacerbate the systemic disease. It may also be possible that oral infection predispose to systemic disease (22).

Many cellular/molecular mechanisms are responsible for such cyclical association. There are common physiological changes associated with diabetes and periodontitis that produce synergy when the conditions co-exist (22).

A potential mechanistic link involve the broad axis of inflammation especially immune cell phenotype, serum lipid levels and tissue homeostasis (22).

Diabetes induce changes in immune cell function produced up regulation of pro-inflammatory cytokines from monocytes/PMNL, and down regulation of growth factors from macrophages. This predispose to chronic inflammation, progressive tissue breakdown and diminished tissue repair capacity (22).

Periodontal tissue frequently manifest these changes, because they are constantly wounded by substances emanating from bacterial bio-film (22).

Diabetic patients are prone to elevated low density lipoproteins and triglyceride (LDL/TRG) even when blood glucose levels are well controlled. It is recently demonstrated that hyperlipidemia may be one of the factors associated with diabetes induced immune cell alteration (16).

Recently some evidence suggested that periodontitis itself may lead to elevated LDR/TRG (22).

Periodontitis induced bacteremia/endotoxemia has been shown to cause elevation of serum pro-inflammatory cytokines such as IL-1β and TNF-α which have been demonstrated to produce alteration in lipid metabolism leading to hyperlipidemia (22).

Within this, periodontitis may contribute to elevated pro-inflammatory cytokines/serum lipids and potentially to systemic disease arising from hyper
lipidemia and/or increased inflammatory mediators. This cytokines can produce an insulin resistance syndrome similar to that observed in diabetes and initiate destruction of pancreatic β cells leading to development of diabetes. Thus there is potential for periodontitis to exacerbate diabetes induced hyperlipidemia, immune cell alteration and diminished tissue repair capacity. It may also be possible for chronic periodontitis to induce diabetes (22).

Diabetes is a risk factor for severe periodontal disease, whereby, severe periodontal disease increases the severity and complicate metabolic control. It is proposed that it is a two-way relationship between periodontal disease and diabetes (28).

This effect is via two ways:

- Infection mediated up regulation of cytokine synthesis and secretion by chronic stimuli, which amplify and add to.

This model helps to explain the increase in tissue destruction seen in what may be called diabetic periodontitis and how periodontal disease may complicate the severity of diabetes and the degree of metabolic control resulting in a two-way relationship (28).

This proposed dual pathway of tissue destruction, suggests that the control of periodontal infection is essential for achieving long term control of diabetes mellitus (Fig. 1) (28).
Proposed Model for 2-Way Relationship Between Periodontal Disease and Diabetes Mellitus

PERIODONTAL DISEASE

- Periodontal pathogen
- Endotoxin, toxins, cell membrane products
- Proinflammatory cascades
- Secretion TNF-α + IL-1β
- Connective tissue destruction, Bone resorption

DIABETES MELLITUS

- AGE – protein
- Macrophage AGE - receptor
- Synthesis + Secretion TNF-α + IL-1β
- Degradative cascade
  - Hydrolase, MMP, collagenase secretion
  - Connective tissue degradation

Insulin resistance
hyperglycemia
Diabetes has been reported repeatedly in association with periodontal disease specially in younger people (18).

One can not say that diabetes is a specific cause of severe periodontal disease and as a matter of fact many people with diabetes has normal periodontal tissues (6).

The overall impression of clinicians point to the fact that, periodontal disease in diabetics follow an inconsistent pattern. These inconsistent results may be due to patient sample, periodontal indices used, and their management and diabetic status of the patient (17).

A cross sectional study of 1420 subjects reported that diabetes mellitus was the only systemic disease positively associated with attachment loss. As there are numerous conflicting studies which do not support this association between periodontal disease and diabetes, the relationship appear to be very strong within special population such as Pima Indians.

A study involving 75 diabetic patients evaluated the association between long term control of diabetes (as evaluated from multiple glycosylated hemoglobin measurement) and periodontitis. In that study the prevalence, severity and extent of periodontitis increased with poor control of diabetes (20).

The established correlation between diabetes and periodontal disease and the increasing prevalence of type 2 diabetes in the general population indicates that dental practitioners will probably treat an increasing number of diabetic patients (23).

It has been previously shown in both human and animal models that some of the host defects routinely observed in patients with diabetes are risk factors for periodontal disease.
The presence of diabetes increases the risk of developing periodontitis three folds. Although the relationship appear to be strong, the corresponding variables inherent in studying two chronic diseases may result in a less clear association in selected populations (28).

A substantial body of literature supports the conclusion that patients with diabetes mellitus have an increased susceptibility to infection. It is well documented that acute infections and inflammatory conditions increase glucose and insulin utilization and therefore complicate the metabolic control of diabetes. This factor is of great importance, since it has been shown that diabetic patients who are poorly controlled may have an increased risk for diabetic complications such as ocular and vascular lesions (12).

Although it has been generally thought that management of periodontal disease is beneficial to diabetic control and may reduce the insulin requirement, there is minimal evidence to support this conclusion or to suggest what magnitude of an effect might be expected from periodontal therapy (12).

William and Mahan in (1976) (29) found that patients require less insulin following stabilization of their diabetic condition and periodontal treatment (12).

However the evidence for existence of high risk groups to periodontal disease, was reviewed by Johnson et al in (1988). Some individuals are susceptible to one or other type of periodontal disease (26).

1.2.12 The Relationship Between Reduction of Periodontal Inflammation and Diabetes Control:

Lawrence et al (1992) studied the relationship between the reduction of periodontal inflammation and diabetes control. Clinical evaluation of the patients included, assessment of radiographic findings and the recording of
clinical indices. Blood glucose levels were evaluated by means of glycated hemoglobin. Two blood samples were drawn six to fourteen weeks prior to any oral hygiene instructions or periodontal treatment. These two samples were used to determine the expected changes in glucose metabolism without any periodontal intervention. Two other samples were taken post treatment at four and eight weeks (30).

The results of the study showed that, some patients with consistent improvement in bleeding showed a decrease in glycated hemoglobin levels post treatment (pretreatment 8.7%, post treatment 7.8%). Patients with no change or improvement in bleeding showed no change or an increase in glycated hemoglobin values following therapy (30).

The impact of diabetes on periodontal disease is evident during therapy, but conventional periodontal therapy that is directed to reduce the bacterial challenge is less predictable in poorly controlled diabetics. And the optimal clinical outcome in diabetic patients with periodontitis must include control of both the diabetes and the bacterial challenge (30).

1.2.13 The Relation Between Control of Diabetes and Gingival Bleeding:
Tellevero Ervasti et al in 1985 studied the relation between control of diabetes and gingival bleeding. In that study the periodontal health status of fifty adult diabetic patients and fifty three healthy control subjects was examined. The diabetic group was further divided into three subgroups according to the degree of control of diabetes. Comparisons between the entire diabetic group and control group did not reveal any significant differences in either the amount of etiologic factors or in the degree of gingival changes. On the other hand when the subgroups of diabetic patients were examined, those with poorly controlled diabetes suffered significantly more gingival bleeding than those with good or
moderate control. The difference in the amount of gingival bleeding between diabetics with poor control and the control group was also statistically significant. The amount of etiologic factors was similar in all studied diabetic subgroups. No correlation was found that could be attributed to duration, complication and medication of diabetes and gingival bleeding (31).

The reason for increased bleeding in poorly-controlled diabetics could be either inflammation or vascular changes in the gingiva.

The results show that there were no statistically significant differences in the periodontal status of diabetics and the control group. There was a significant positive correlation between bleeding and plaque as well as calculus in both groups (31).

Metabolic imbalance in the tissues may lower the resistance of diabetics to infection, and thus influence initiation, development and progression of periodontal disease (31).

1.2.14 Cell Mediated and Humoral Immune Responses in Diabetic Patients with Periodontitis:

In a study of cell mediated and humoral immune responses in diabetic patients with periodontitis carried by Anil et al. in 1990, fifty type 2 diabetic patients and fifty non-diabetic patients with adult periodontitis were examined. All subjects were screened clinically, bio-chemically and biophysically to exclude subjects with other infections. Periodontal status of patients and control subjects was assessed using the periodontal disease index (PDI). Venous blood was collected from each subject for immunological examination (32).

The results indicated that, cell mediated immunity did not show much variation from that of control subjects. Serum immuno globulin levels (except Ig D) showed significant alteration from those of control subjects. Serum Ig D values
of the patients were within the normal range. Serum IgA values of non-diabetic subjects were significantly higher than those of diabetic patients. There was no much variation in any of the other immunoglobulin contents between diabetic and non-diabetic groups. The slightly elevated value obtained for Ig G in the diabetic patients was not significant when compared to that of non-diabetics (32).

The study revealed that periodontitis is associated with alteration in immune responses in both diabetic and non-diabetic subjects. There is marked alteration in humoral immune responses whereas the alteration in cell mediated immunity is marginal. The changes in immune responses may be the cause of all the effects of periodontal disease. The increased incidence of periodontitis in diabetic patients suggests that the alteration in immune responses may be one of the factors in the pathogenesis of periodontitis (32).

1.2.15 The Effect of Improved Metabolic Control on Periodontal Condition and The Subgingival Microflora:

Sastrowijoto et al (1990) studied the effect of improved metabolic control on the clinical periodontal condition and the subgingival microflora of diseased and healthy periodontal pockets in six IDDM patients. During the study patients maintained personal oral hygiene measures as they did before the study without supplementary dental prophylaxis. Long term metabolic control (Hb A1c improved significantly with intensive insulin treatment) (33).

Gingival redness decreased significantly whereas gingival swelling showed no trend to decrease (33).

It is suggested that microvascular changes associated with improved metabolic control in diabetes mellitus may mediate the observed change in gingival
redness. No effect could be demonstrated for probing pocket depth, attachment level, bleeding on probing and plaque index (33).
Analysis of the effect of improved metabolic control on subgingival microflora revealed that only the percentage of streptococci increased significantly in diseased periodontal pockets (33).
The results of that study indicated that improved metabolic control in IDDM may have no potential impetus for improved clinical periodontal condition, nor on the subgingival bacterial flora (33).
The authors concluded that the periodontal condition in IDDM patients may ameliorate when local oral hygiene measures are applied (33).

1.2.16 Relationship Between Diabetes Mellitus and Oral Health Status:
Lawrence et al (1991) studied the relationship between diabetes mellitus and oral health status in Pima Indians from Gila River. This tribe has the world highest incidence and prevalence of NIDDM (this tribe has been under continuous investigation since 1995 by the National Institute of Diabetes). The prevalence and severity of destructive periodontal disease was determined by measuring probing attachment loss and inter proximal crestal alveolar bone loss (23).
The diabetic status of the subjects was determined by the results of modified two hours glucose tolerance tests. The objective of this study was to investigate the relationship between the prevalence and severity of periodontal disease and diabetes (23).
The results of the study indicated that there was an increasing prevalence of destructive disease seen with increasing age up to 45 - 54 years. There was a higher prevalence of periodontal disease in the diabetic subjects among all age groups. The risk indicators for periodontitis that were highly statistically
correlated using both measures of periodontal tissue destruction are identified. Diabetes, age and calculus were associated with an increased risk to periodontitis. The diabetic subjects had a risk of periodontitis 2.81-3.43 times higher than non-diabetics (23).

Age was analyzed by decades and it was found that for each decade of life, the risk for developing periodontitis was 3.1 and 4.28 using attachment loss and bone loss scores respectively as indicators of periodontitis.

The generalization of the finding of more severe periodontal disease in diabetic Pima Indians compared to other racial and ethnic groups will await further confirmation. However there is evidence in several Caucasian groups for greater severity of periodontal disease among diabetic subjects (23).

Further more it is reasonable to assume that findings of the Pima Indians periodontal status are generalizable. Since most other complications of diabetes in this group are comparable to those in other racial and ethnic groups. The comparability of periodontal disease in diabetics and non-diabetics was supported by other findings that the common periodontal pathogens *P. gingivalis*, *P. intermedius* are predominant members of the subgingival flora in Pima Indians suffering from periodontitis and there is specific serum antibody response to these organisms as is also seen in other populations (23).

Diabetes is consistently and strongly related to destructive periodontal disease among young and middle age adults. It is reasonable to suggest that diabetes is a predictor of periodontal disease and further more periodontal disease can be considered a complication of diabetes mellitus (23).
1.2.17 Relationship Between the Severity of Periodontal Disease and Organ Complications:

Organ complications in diabetes mellitus may be associated with severity of periodontal disease (retinopathy, nephropathy, incidence of higher blood pressure). There would appear to be a fixed chronology of complications beginning with retinopathy, culminating in many subjects having a number of such complications concomitantly (34).

Organ complications are associated with long term duration of diabetes and poor long term metabolic balance. However even long duration of diabetes and/or poor metabolic balance do not always result in complications (34).

Some diabetics with good metabolic control experience complications after a few years duration of their diabetes. It is not yet known whether the individual variations are genetically determined or not. Genetics may be a reason for variation in individual susceptibility to alteration in basement membranes, collagen metabolism and glycosylation of tissue proteins. These are behind the development of complications. Such alterations could also affect resistance to local etiologic factors increasing risks of periodontal disease. Diabetic patient motivation and attitude towards diabetes treatment are critical, and at least partly determined whether optimal glucose balance is achieved and complication prevented. Periodontal health as well, is largely dependent on dental health behavior (34).

Kaisa et al. (1994) studied the relationship between the severity of periodontal disease and organ complications in long term type 1 diabetic patients (34).

The population studied consists of a group of type 1 diabetics 26-34 years old who had diabetes for at least 10 years. As not all diabetic subjects can be regarded as at increased risk for periodontal disease than non-diabetic individuals, therefore many investigators have concentrated on diabetics
comparing severity of periodontal disease with diabetes related factors such as systemic complications, duration of diabetes and metabolic balance (34). The results of the study demonstrated the importance of taking account of interdependence between complications, duration of diabetes and level of metabolic control in studying association between periodontal disease and diabetes. Diabetic patients with severe periodontal disease were found to be mostly those who have diabetic organ complications. Complications seem to relate with periodontal disease better than glucose balance alone, possibly because the existence of complications take account of the subject individual susceptibility (34).

High levels of plaque and subgingival calculus in poorly controlled diabetic patients and in patients with complications were seen in this study. They may have been related to poor dental behavior of those patients (34). Tervonen and Oliver (1994) have reported more subgingival calculus in poorly controlled diabetic patients than in well controlled patients. The extent to which the periodontal disease observed in advanced complication patients may be the result of lack of dental care (34).

The severity of periodontal disease based on metabolic control only is less obvious than those based on the presence of advanced complications. The reason was that many patients with organ complications were in the well or moderately controlled group. This supports the assumption that comparisons based on metabolic balance alone may be inappropriate. Although the level of metabolic balance induce the development of complications but this vary from individual to the other (34).
1.2.18 Microbial Incidence in Poorly Controlled Diabetics:

Ropert et al (1992) studied the total microbial level, microbial incidence and percent levels of selected periodontal microorganisms in a group of poorly controlled IDDM patients (35).

The influence of diabetes on the risk of developing periodontal disease has been the subject of much discussion. According to a majority of authors, individuals suffering from IDDM are at increased risk for the development of periodontal disease (35).

Fifteen Caucasian patients with a mean age of 34.6 years were selected for the study. The duration of IDDM was 20.6 years. Medical histories indicated that the mean level of glycosylated hemoglobin was 10.4%. Subgingival plaque samples were taken from the base of the periodontal pocket with a sterile scaler inserted into the depth of the pocket, transferred and processed by certain methods to determine the highest recoverable bacterial counts. To study the clinical characteristics of the sample sites, two sites per patient were sampled for the panel of periodontal pathogens. The sites were selected so that both diseased and healthy sites for each patient would be examined (35).

Increased levels of periodontal pathogens, *P. intermedius*, *P. melaninogenica* spp., *bacteriod Gracilis*, *Eiknella corrodens*, *fuso bacterium nucleatum* and *campylobacter rectus* were found at the periodontal diseased sites. Increased prevalence of the organism *P. intermedius*, *P. melaninogenica* and *campylobacter rectus* were found at diseased sites. A significantly higher percentage of *P. intermedius* was found at the site exhibiting deep pockets and greater attachment losses (35).

In another study done by Mashimo et al (1983) (36) 14 IDDM patients aged 13-27 years were examined. The samples consisted of 9 periodontitis, 3 gingivitis and 2 periodontally healthy patients. The diseased patients whose condition
resembled juvenile periodontitis were found to harbor flora with high levels of *Actinobacillus actinomycetomacitans* (á á), *Capnocytophaga* spp. and anaerobic vibrios (35).

Sastrowijoto *et al* (1989) examined diabetic adults grouped by virtue of their glycosylated hemoglobin values into normal and poorly controlled patients. They reported that the organisms *Actinobacillus actinomycetomacitans, P. gingivalis, P. intermedius* were recovered at proportionally higher levels from the diseased versus control sites (35).

These results from IDDM patients with periodontal disease indicate that organisms implicated in other forms of periodontal disease are also found within the IDDM population. The work done by Mashimo and that done by Robert have a common finding indicating increased levels of black pigmented species and anaerobic vibrios. The difference from Mashimo study lies in that Mashimo found *Actinobacillus actinomycetomacitans* important while Robert study found *Actinobacillus actinomycetomacitans* at levels usually seen in the general population. The difference may be due to age, race of the population examined. A second difference from Mashimo is in the level of *Capnocytophaga* spp. which in Robert study does not differ from healthy and diseased sites (35).

Sastrowijotto *et al.* (1989) study also failed to confirm the association of *Capnocytophaga* spp. The author also found only the *P. intermedius* was significantly elevated in diseased versus healthy pockets in the poorly controlled IDDM group (35).

Finally it is possible that poor control make these patients susceptible to periodontal pathogens associated with their disease, perhaps as a result of neutrophil suppression (35).
Tervonen et al (1994) studied the prevalence of five periodontal pathogens in individuals with diabetes mellitus (37).

The study was part of a comprehensive investigation of periodontal disease in diabetic patients. The rationale for evaluating the relationship between the metabolic control of diabetes and prevalence of periodontal bacterial pathogens, was that in several earlier studies an increase has been found in the prevalence and extent of periodontitis with poor metabolic control (Tervonen and Knuuttila, 1986 (38), Miller et al, 1992 (39).

It has been speculated that the increase in Gingival Crevicular Fluid (GCF) during hyperglycemia may favour growth of some bacterial species (Zambon et al, 1988) (40).

Subgingival plaque samples was taken from each subject from single sites exhibiting the greatest inflammation based on the assumption that diabetic control at the time of sampling is reflected at such a site. The evaluation of bacterial pathogens was based on an immunoassay utilizing bacterial specific antibodies for five periodontal pathogens (Actinobacillus actinomycetemcomitans, F. nucleatum, E. Corrodens, P. gingivalis, P. intermedius) (38).

The results showed that the most frequent periodontal pathogens in this diabetic population was P. gingivalis 34.6% of the sites, 28% harbored F. nucleatum and 20.6% E. corrodens, Actinobacillus actinomycetemcomitans and P. intermedius were both found in less than 10% of the subjects and approximately half of the sites had ≥ 1 of the 5 bacterial pathogens (38).

The prevalence of most of the selected pathogens increased with decreasing metabolic control of diabetes. No significant difference in the prevalence of these pathogens was found between type 1 and type 2 diabetes nor with respect to duration of the disease (38).
The presence of bacterial pathogens at sites based on stratification of probing depth is that bacterial pathogens were more frequently seen at sites with probing depth $\geq 4\text{mm}$ than sites with shallow probing depth i.e. $\leq 3\text{mm}$. However the difference is significant for only $P. \text{gingivalis}$. Also the likelihood of harboring $E. \text{corrodens}$ was significantly associated with the presence of probing depth $\geq 6\text{mm}$ (38).

Non of the diabetic factors including type, duration and metabolic control of the disease entered the final multi-variate model at the significant variable (38).

The primary conclusion of the study was that in diabetic populations with mostly early periodontal disease, no relationship existed between metabolic control or other diabetic variables and the prevalence of sub-gingival periodontal pathogens. This finding together with earlier studies (Zambon et al, 1988 (40), Sastrowijoto et al, 1989, Mandel et al, 1992) (41) indicate that the same periodontal pathogens that are found in adult periodontitis are also implicated in periodontal disease in both type 1 and 2 diabetes. And the observation of increased periodontitis in poorly controlled diabetics is due to factors other than increased prevalence of pathogens. Like diabetic micro-angioapthies in the gingival area and defects in function of PMN making these patients more susceptible to periodontal disease progression as a result of non-specific as well as specific bacterial colonization (38).

1.2.19 Humoral Immune Response and Selected Subgingival Plaque Microorganisms in Diabetics:

Takanobu Morinushi et al (1986) studied the humoral immune response to selected subgingival plaque microorganisms in IDDM children (42).
The study was performed to determine if bacterial antibody titres to selected periodontal disease associated microorganisms might be helpful in revealing changes in plaque flora at the onset and conclusion of puberty.

Sera are selected from 35 subjects from a population of IDDM patients. Antibody titres to A. naeslundii (A. N.), P. intermedius, P. gingivalis, F. nucleatum (FM), Actinobacillus actinomycetomacomitans, C. ochracea (CO) and T. denticola (TD).

Stratification of antibody titres by age group (≤ 12 years, 12-15, >15 years) revealed that titres to A. N. increased significantly and progressively with increasing age. In contrast titres to FN were maximal less than 12 year age group and decrease with age. There were no significant variation in titres observed for other microorganisms (42).

Populations of IDDM are selected for this study because study of periodontal disease in normal circum puberty population is difficult due to low frequency of gingivitis at this age group. Depending on the fact that previous reports had indicated that IDDM have increased level of gingivitis when compared to non-diabetic population (42).

1.2.20 HLA & T lymphocyte Reactivity to Specific Periodontal Pathogens in Diabetics:

As considerable evidence has become apparent in recent years documenting the existence of abnormalities in the immune system associated with type 1 diabetes including reduced T lymphocyte proliferation and IL-2 production, similar immune defects may predispose diabetic patients to periodontitis. So it is therefore possible that the etiologic factors in type 1 diabetics, periodontitis involve a combination of certain bacterial infections (Capnocytophaga) and
altered host immune response and selective susceptibility which may suggest genetic control (43).

An association between diseased and a known genetic marker imply means to identify individuals at increased risk of developing the condition. Human leukocyte antigen (HLA) have been investigated for their role in type 1 diabetes in conjunction with the development of various types of periodontal disease. It appears that different HLA specificities may be associated with type 1 diabetes and periodontitis susceptibility, while others promote resistance (43).

HLA – D may play a role in periodontitis susceptibility through their ability to bind and present bacterial antigens. The results of histological studies indicate that stable periodontal pockets or healthy sites display primarily T lymphocytes while progressive lesions have significantly decreased T/B cell ratio. Thus the immuno regulatory effect of T lymphocytes particularly those acting locally may be important in maintaining equilibrium at the periodontal site (43).

Catherine Segren et al (1993) studied the HLA and T lymphocyte reactivity to specific periodontal pathogens in type 1 diabetic periodontitis. In this study sixty subjects who met the diabetic periodontal and medical criteria were selected. They were then divided into four groups, diabetic type 1 with periodontitis (DP), type 1 with no periodontitis (Dn P), non-diabetic periodontitis (nDP), non-diabetic with no periodontitis (nDnP). Periodontal evaluation of all subgroups consisted of bacterial plaque, bleeding on probing, probing depth and clinical attachment loss (44).

HLA – DR/DQ were performed using lymphotoxicity and fluorescent protocols. The purpose of this study was to investigate the association among periodontitis and type 1, HLA – DR/DQ phenotype and T lymphocyte immune response to specific periodontal pathogens (P. gingivalis and C. sputigena). The results of clinical periodontal parameters indicated that plaque levels and bleeding on
probing was higher in periodontitis patients. Probing depth and clinical periodontal attachment loss was also higher and similar in both periodontitis groups (44).

HLA – DR/DQ types varied among subjects, HLA – DR3 occur more frequently in diabetics (DP = 53.3%, DnP = 43.7% while nDP=26.7% and nDnP=33.3%) but the differences were not significant. HLA – D4 frequencies were significantly increased in diabetics (DP 73.3%, DnP=46.7% and nDP= 66.7% over the general population and nDnP = 6.7% (44).

HLA – DR 53 follow identical distribution among groups. HLA – DQ3 was also significantly related to periodontitis.

Poor plaque control alone does not account for severe periodontal destruction and not all individual were at equal risk (43).

A depressed spontaneous proliferation of peripheral blood lymphocytes has been found in patients with advanced periodontitis. No significant differences were found among groups in spontaneous T lymphocyte proliferation. T lymphocytes in diabetic patients remained reactive against antigens of both P. gingevalis and C. sputigena (43).

A number of previous studies indicated that type 1 diabetes occur at significantly higher frequencies in HLA – DR3 and or DR4 (and associated HLA – DQ) genes (43).

Katz et al (1987) (45) demonstrated that 80% of patients with rapidly progressive periodontitis possessed HLA – DR4. this confirm the potential of HLA – DR4 as a marker of periodontal susceptibility in young adults. Results also imply that HLA – D phenotype has stronger influence on periodontal susceptibility than on diabetic status (43).
1.2.21 Microbiological and Immunological Status of Periodontitis in Diabetics:

Zambon et al (1988), investigated the microbiological and immunological status of adult periodontitis in patients with NIDDM. 55 adults ranging in age from 17-64 years were selected for the study. Each subject demonstrated clinical signs of moderate to severe periodontitis (43).

The predominant cultivable micro-flora in periodontitis patients with NIDDM revealed large proportions of black pigmented *Bacteroid* species. The predominant isolates were *P. intermedius* 16% in a single site, *Wollinella recta* and *P. gingivalis* accounted for 13% of the total isolates. The most prevalent microorganism was *Streptococcus sanguis* which was present in 75% of sites and accounted for 7.8% of the total isolates. This study indicated that sub-gingival microflora in severe periodontitis patients with NIDDM is similar to that found in non-diabetic adults with severe periodontitis. But the study also indicated (as previous studies did) that *Bacteroids* species may be an important microorganism in the etiology of extraoral infections in adult diabetics. This species was reported to cause a variety of lesions in diabetics including endopelvic fascia and gangrene. There is also evidence that anaerobes in general and *Bacteriod fragilis* in particular may have increased pathogenicity and may persist for longer periods of time in diabetic as compared to non-diabetic host (40).

Crevicular space in patients with IDDM may comprise a unique environment favoring anaerobic microbial ecology. The capillaries and small vessels are thicker in subjects with diabetes compared to non-diabetics, inhibiting the diffusion of oxygen from the circulation into the gingiva (40).

Serum IgG antibody level to selected oral microorganisms supports the results of the microbial studies (40).
Both *P. gingivalis* and *P. intermedius* serum antibody levels were higher in patients with periodontal disease than in periodontally normal individuals regardless of the subject diabetic status. Further more serum IgG were elevated in both antigenic types of *P. gingivalis*, but were elevated to a greater extend to specific antigenic types. Further more measurements of antibody levels to the other microorganisms were found to be associated with periodontal disease in the Pima Indians such as *W. recta*, *Capnocytophaga*, *Fusobacterium* species, *Propionibacterium* species and *Eubacterium* species (40).

1.2.22 Glycaemic Control and Alveolar Bone Loss in diabetics:

Gorge *et al* (1998) studied the glycaemic control and alveolar bone loss progression in type 2 diabetes. In the study the authors tested the hypothesis that the risk for alveolar bone loss is greater, and bone loss progression is more severe for subjects with poorly controlled type 2 diabetes compared to those without type 2 or better controlled diabetes. Subjects for the study were 359 individuals, age range 15-57 years. 338 subjects were non-diabetics, the other 21 had type 2 diabetes (14 with well controlled diabetes and 7 with poorly controlled diabetes). Radiographic bone loss at base line was <25%. The median time of follow up was 2.3 years. Additional dental, behavioral, medical and demographic variables were evaluated at their base line values for confounding and effect modification. Age, calculus, plaque index, gingival index and time, alcohol consumption, smoking, obesity, coronary heart disease and gender were also evaluated (45).

The results indicated that there was a tendency for the subjects with poor control to have higher proportion with more severe bone loss. The pattern of increasing severity of bone loss increased with age. These results provide strong evidence to support the hypothesis. The final conclusion of this study was that
the risk for poor control > risk for better control > risk for those without type 2 diabetes (45).

Papapananou and Wennstrom (1990) also found an inverse relationship between base line radiographic bone level and the amount of bone loss at follow up examinations (46). However, Albandar (1990) reported a direct relationship between degree of bone loss at base line and at follow up examinations (47).

**1.2.23 Effect of Antimicrobial Periodontal Therapy on Metabolic Control of Diabetes:**

Yoshihiro et al (2001) studied the effect of antimicrobial periodontal therapy on serum TNF – α concentration and subsequent metabolic control of diabetes. They examined the periodontal and diabetic status on 13 type 2 diabetic patients. These patients were treated with local minocycline administration in every periodontal pocket around all existing teeth once a week for a month. Before and after the treatment the number of total bacteria in the periodontal pockets and circulating TNF – α concentration were measured and HbA1c value was assessed (48).

The results concluded that antimicrobial therapy significantly reduces the number of microorganisms in periodontal pockets. After treatment the circulating TNF – α was significantly reduced, HbA1c was also reduced significantly and in addition to that patients not receiving insulin therapy demonstrated decreased fasting glucose level. The average reduction in circulating TNF – α were 0.49 Pg/ml, and the average reduction for HbA1c was 0.8% (48).
1.2.24 Periodontal Treatment and Effect on Metabolic Control of Diabetes:
Concerning the question of whether periodontal treatment may have an effect on metabolic control of diabetes, a recent review of available literature concluded that periodontal therapy may not be associated with glycemic control in well controlled diabetes, but may result in improved metabolic control in poorly controlled diabetes (49). Re-examination of studies that address the effect of periodontal treatment on metabolic control of diabetes mellitus, six of the studies included type 2 patients, diabetes duration ranged from one year to a mean of 18 years, degree of control ranged from poor to well controlled, periodontal status from gingivitis to severe periodontitis. Periodontal treatments included mechanical depridement with or without administration of systemic antibiotic (49).
Seppälä et al (1994), studied the mechanical debridement only as a treatment of periodontal disease and the results indicated that no effect on blood glucose or HbA1c (49).
Aldridge et al (1995) conducted two studies. In study one, the results showed that no significant change in probing depth, but significant reduction in bleeding on probing. In study two the results indicated that there was a significant reduction in periodontal parameters but not in HbA1c. So there was no significant association between periodontal treatment and metabolic parameters in either group (50).
Smith et al (1996) found that there was significant improvement in mean periodontal disease, reduction and gain in attachment level, but no effect on HbA1c or other systemic parameters (51).
Christgau et al (1998) found that there was a reduction in median percent of pockets > 4mm and in bleeding on probing, but no effect on HbA1c (52).
Gross et al (1997) in his study including mechanical treatment only as the above studies concluded that there was a reduction in periodontal disease, gain in clinical attachment loss (CAL), but no effect on HbA1c (53).

In contrast to the above, all three studies incorporating systemic antibiotic with mechanical periodontal therapy reported an improvement in diabetic metabolic control (53).

Grossi et al (1997) in a randomized clinical trial in Pima Indians with type 2 diabetes and severe periodontal disease demonstrated that periodontal treatment incorporating topical antimicrobials with systemic doxycycline (100 mg/day for 14 days) resulted in elimination of P. gingivalis infection, significant gain in attachment level and a reduction of almost 1% in level of HbA1c at three months after treatment. This reduction constituted approximately 10% of the initial HbA1c concentration and was not dependent on the traditional methods of control in type 2 diabetes (53).

Williams et al (1960) (29) and Miller et al (1992) (39) in their studies collectively concluded that, the effect of periodontal therapy on diabetes metabolic control is dependent on the mode of therapy, mechanical versus a combination of mechanical and systemic antibiotics (39).

So successful elimination of periodontal infection with systemic antibiotics may significantly reduce the systemic bacterial challenge with reduction in secretion of inflammatory mediators affecting the catabolic cascade. Therefore one may propose that control of periodontal infection should be part of the standard care of diabetic patients (39).

The biologic basis for the systemic effect of periodontal treatment of diabetes, are likely the results of antimicrobial level of these drugs (39).

Doxycycline is a broad spectrum antibiotic, its concentration is 7-10 folds in the gingival fluid over serum levels thus providing an important adjunct. It has
also been demonstrated that tetracycline and their derivatives have a modulatory
effect on the host response, independent of their antimicrobial effect, by
suppressing the collagenolytic process and increasing protein synthesis and
secretion. Recently it has been reported that tetracycline prevents the release of
TNF-α by membrane retention. It has also been reported that it blocks protein
kinase C activity, an important step in the secretion of IL-1β and TNF-α. It also
inhibits collagen degradation, decrease levels of glycated hemoglobin by
inhibiting the glycation of proteins. A potential therapeutic role for tetracycline
in the treatment of periodontitis is by inhibiting tissue destructive enzymes (39).
In conclusion diabetic patients with periodontitis benefit from systemic
administration of tetracycline derivatives in many ways including:

- eliminating most periodontal pathogens
- a potent host response modulator
- possible inhibition of non-enzymatic glycation

Sarra (2001) in study on the treatment of periodontal disease and control of
diabetes, reached an evidence which points to an increase in the cytokines
response in type 2 diabetes, especially the pro-inflammatory cytokines
interleukin (IL-1β, IL-6 and TNFα). Genetics, age and nutrition are important
signals for this increased response also infections and inflammations. Persistent
elevation of IL-1β, IL-6, TNF-α in the diabetic state have an effect on liver,
stimulating the release of acute phase proteins, produce the characteristic dys-
regulation of lipid metabolism associated with type 2 diabetes and have an
effect on pancreatic beta cells as well (54).
In addition TNF-α a potent inhibitor of the tyrosine kinase activity of the insulin
receptor, has been implicated as an etiologic factor of insulin resistance.
Collectively the evidence supports a role for cytokine elevation in the
pathophysiology and metabolic abnormalities associated with diabetes (54).
Periodontitis is an infection that is twice as prevalent in diabetic individuals compared to non-diabetics. *P. gingivalis* one of the microorganisms responsible for this infection, is able to invade endothelial cells and is a potent signal for monocyte and macrophages activation. Thus once established in the diabetic host this chronic infection complicates diabetes control and increase the occurrence and severity of microvascular and macrovascular problems sometimes associated with diabetes (54).

Unlike treatment of acute infections, modalities of treatment for chronic infections are a matter of debate (54).

Mechanical removal of subgingival infection does not result in complete elimination of periodontal infection and consequently there is no effect on diabetes control (54).

Studies incorporating systemic antibiotics as an adjunct to mechanical debridement result in reduction of *P. gingivalis* to non-detectable levels and concomitant reduction in glycated hemoglobin, independent of the hypoglycemic effect of diabetes drug or insulin (54).

This evidence supports the notion that treatment of periodontal infection is essential in the diabetic patients. So assessment of infection status in diabetic patients is fundamental for appropriate treatment decision (54).

### 1.2.25 Bidirectional Inter-relationship Between Diabetes and Periodontal Disease:

Gorge (2001) in study on bidirectional inter-relationship between diabetes and periodontal disease, and epidemiologic perspective, conducted a comprehensive midline search to identify primary research reports of relationship between diabetes and periodontal disease. Reports included the adverse effects of
diabetes on periodontal health (DM $\rightarrow$ PD) were restricted to comparisons of periodontal health in subjects with and without diabetes (55). Observational studies reporting (DM $\rightarrow$ PD) provided consistent evidence of greater prevalence, severity, extent or progression of at least one manifestation of periodontal disease in a large majority of reports 44/48, 37/41 cross sectional and 7/17 Cohort (33). On the other hand treatment studies provided a direct evidence to support the idea that periodontal infection is having an adverse modifiable effect of glycaemic control after periodontal treatment (55).

The evidence reviewed support viewing the relationship between diabetes and periodontal disease as bidirectional. Further rigorous, systematic studies are warranted to establish that treating periodontal infections can be influential in contributing to glycaemic control management and possible to reduction of the burden of complication of diabetes mellitus (55).

### 1.2.26 Periodontal Treatment Need in Diabetic Patients:

Miljenko *et al* (1988) Investigated the periodontal treatment need in diabetic patients using CPITN. The study aimed also to shed additional lights on the possible effects of duration and control of diabetes of periodontal status of these patients (56).

A comparison was made between 222 diabetic patients and 189 control subjects. The results indicated that diabetic patients demonstrated significantly more missing teeth and higher missing sextants compared to non-diabetics i.e. the mean number of extracted teeth was found to be higher in diabetics than in the control group subjects. This finding is difficult to compare with other authors because corresponding data are rare and the differences in the age of the subjects studied (56).
Glavind, Lund and Løe (1968) found that an average of 9.5 and 7.5 teeth to be missing in a group of diabetics and controls groups respectively (57).

The other finding is that pathologic pockets of 6mm or more were found in 1.3 and 0.3 sextants in the diabetic and control groups respectively in subjects over 34 years of age (56).

Concerning the type of diabetes, no difference related to CPITN was observed between diabetes type 1 and type 2. Neither were any differences found in the periodontal condition that could be related to duration and control of diabetes. Whereas diabetics with advanced retinopathy demonstrated more sextants with deep pockets (56).

In this study CPITN recordings for control subjects were comparable to both CPITN data of 40 countries, in the findings obtained by Ainamo, *et al* (1986) (56).

The study confirmed that the prevalence and severity of periodontal disease increases with age in both healthy and diabetic subjects. The data also indicated that periodontal disease occur earlier and assume more severe forms in diabetic patients and in non-diabetics if left untreated (56).

There was no relationship between the control of diabetes and periodontal status. This result was explained on the fact that destruction of teeth supporting structures is a long term process. It is difficult to make a definite conclusion that periodontal status is related to diabetes control on the basis of one daily blood glucose value. This findings was also supported by Nickols *et al* (1978) (58). However these results contradict with the results from other studies (56).

Monitoring of the control of diabetes using HbA1c determination is more accurate to study the association between HbA1c and the state of the periodontium. Gislen *et al* (1980), (59) and Ervasti *et al* (1985) found strong
correlation between the two, whereas Gusberti et al (1983), (60) and Barnett et al (1984) (61) did not confirm these findings (56). So diabetes control may reflect a short term improvement and therefore may be of limited value when assessing the degree of control over many years for purposes of epidemiologic studies of chronic periodontitis by CPITN in adults (56).

1.2.27 Manifestation of IDDM in the Periodontium:
Belem et al (1991) in study on the manifestations of IDDM in the periodontium of young Barazilian patients examined, thirty diabetic children aged 5-18 years compared with thirty non-diabetic subjects, and correlated the results with gender and age (62). The gingival condition was carefully assessed, plaque index gingival index probing depth, and alveolar bone level were measured (62). The results showed that the mean plaque index in diabetics was 1.23 while in the control group it was 0.81. the difference was statistically significant whereas no significant difference was found between values for control males and control females. There were no statistically significant differences in the near pocket depths between the groups and the bone loss was different only for the upper and lower anterior region in diabetic patients compared with control subjects. No differences were detected in terms of age (62).

1.2.28 Health Behaviours and Their Relationship to Metabolic Control:
Although it has been reported that diabetic control is beneficial in managing periodontal disease, there are few studies in the literature where the relationship between singular behavioral factors and periodontal status has been examined (63).
Motokoto et al (2001) studied the health behaviours and their relationship to metabolic control and periodontal status in type 2 diabetic patients. Their results indicated a significant co-relation between oral behaviour and calculus accumulation. The authors concluded that oral health behaviour affected periodontal status throughout its effects on behaviours forwards oral health and diet (63). This conclusion is supported by the fact that diet behaviour affects both plaque accumulation, metabolic control and periodontal status (63).

1.2.29 Overview:

Epidemiological studies have shown that, the risk for developing periodontal disease is higher in patients with diabetes mellitus than in non-diabetic individuals (Cianciola et al 1982 (64), Hugoson et al 1989 (65), Emrich et al 1991) (23). Diabetic patients with poor metabolic control are reported to be at highest risk (Ervasi et al 1985 (66), Tervonen and Knuuttila 1986 (38), Miller et al, 1992 (39), Safkan – Seppala and Ainamo, 1994 (67) and Tervonen and Oliver, 1994) (37).

Grossi et al (1994) in a large cross sectional study showed that diabetic patients were twice as likely as non-diabetic subjects to have attachment loss (53).

Firatli (1997) followed type 1 diabetic patients and healthy controls for five years. The group with diabetes has significantly more clinical attachment loss than controls (68).

Bridges et al (1996) found that diabetes affects negatively all periodontal parameters (69).

In fact one study of type 1 diabetes and oral health assessment of tooth loss and edentulism, by Moore et al (1998) has shown that diabetic patients are five times more likely to be partially edentulous than non-diabetic subjects (70).
There are many other factors involved in the high prevalence of periodontal disease usually associated with diabetes. The relationship appears to be strong within certain populations indicating genetic components. Smoking was found to increase the risk of periodontal disease by nearly 10 times (as the study done by Moore et al, 1999). According to these results the management of diabetic patients should include strong recommendation to quit smoking (70).

Although it has been recognized for decades that adult diabetics are more likely to develop periodontal disease, only recently this observation has been scientifically established. Papapanou (1996) in meta analysis of data from four studies including 3524 adults > 18 year demonstrated significant relation between diabetes and periodontal disease (71).

Cross sectional studies by Knowler (1978) (72) and Shlossman (1990) (73). And longitudinal studies by Nelson et al (1990) (74) and Emirch et al (1991) (23) utilizing regression analysis controlling from a number of confounders or covariates have concluded that diabetes increased the risk for developing periodontitis in a manner that cannot be explained on the basis of age, gender or dental plaque levels (54).

Löe (1993) in his study of periodontal disease, the sixth complication of diabetes, helped to evidence periodontal disease as a complication of diabetes mellitus. In addition for older diabetics >40 years of age the evidence demonstrated that the severity of periodontal disease increases with longer duration of diabetes. This parallels findings from other complications of diabetes, that the longer the duration of diabetes the greater the prevalence and severity of the complication (75).

The relationship between infectious periodontal disease and diabetes has been described in many clinical studies. Several investigators reported a higher incidence and severity of periodontal disease in type 1 diabetic adults (Belting

Several other investigators found no association between this metabolic disorder and the degree of periodontal disease as assessed by clinical methods Benveniste et al 1967 (82), Hove and Stallard, 1970 (83), Barnett et al, 1984 (61), Goteiner et al, 1986) (84).

The suggestion that the impaired metabolic control of diabetes influences the severity of periodontal disease is widely accepted in dentistry.

Finestone and Boorujy, (1967) (85) reported a positive association between variation in blood sugar levels and the degree of periodontal disease in type 1 and type 2 diabetes mellitus (33).

Ervasti et al, (1985) reported more gingival bleeding on probing in poorly controlled diabetic adults than in moderately, and well controlled groups (66).


Cohen et al (1970) in their longitudinal two years observation of diabetes and periodontal disease noted that gingival index of diabetic patients was significantly higher than for control subjects. This non-positive correlation between gingival and soft deposit indices lead the authors to conclude that the higher gingival indices of diabetics may reflect a reduced resistance of diabetic patients (77).

Shlossman et al (1985) compared the prevalence and severity of periodontal disease in Pima Indians between the age of 15-90 years. They found that destructive periodontitis was more prevalent, and had an earlier age of onset in subjects with IDDM (86).
Cianciola *et al* (1982) studied a population of subjects with IDDM and found that periodontitis was found in 16% of subjects between 13-14 years of age, and 26% of subjects 17-18 years of age, while only 2% of 125 control subjects demonstrated periodontitis (64).

Sandler and Stahl (1960) studied the prevalence of periodontal disease in a hospitalized population. They suggested that patients with controlled diabetes may not have more periodontal disease when compared with normal subjects (87).

Gusberti *et al* (1983) reported a bacterial shift in the microbial composition at the onset of puberty in type 1 diabetic children, resulting in a higher percentage of *Capnocytophaga* species and *Actinomyces naeslundii* (60).

However, Sandholm *et al* (1986) reported no raised serum antibody titres to *Capnocytophaga* in 133 type 1 diabetic patients. By contrast diabetic patients progressive periodontal disease in adult non-diabetic patients is frequently associated with a high proportion of cultivable *A.A.*, *P. intermedius* and *P. gingivalis* Slot *et al* 1986 (89), Slot and Listgarten 1988 (90), Van winkelhoff *et al* 1988) (91).

Kjellman *et al* (1970) came to the conclusion that diabetics with poor co-operation in the care of the disease (uncontrolled) had more gingivitis than those with good control (controlled) (92).

Sheriden (1959) showed that pocket formation and tooth loss occurred with greater frequency in patients with poor controlled diabetes (93).

Firatli (1997) followed type 1 and healthy control for 5 years, came to a conclusion that people with diabetes has significantly more attachment loss than controls (68).
Cianciola (1982) and Tervonen and Karjalainen (1997) (94) found no significant difference in the periodontal condition of diabetics and non-diabetics including attachment loss (94).
Material and Methods

One hundred dentate patients as a sample size (50 for test and 50 for control) with an age range 30 to 55 years were included in this study. Following an introduction letter ensure cooperation (Annex i).

50 diabetic patients were randomly selected from patients routinely attending Jabir Abu Elizz Diabetic Center in Khartoum (Fig. 2), during the period May to September 2002, excluding those who are admitted, in acute complications and those who came for surgical dressing as well as patients with edentulous sextants.

The control group was selected randomly from non-diabetic patients attending the Periodontal Clinic of the Faculty of Dentistry University of Khartoum and Khartoum Dental Teaching Hospital, during the same period.

The study group was assessed, examined and investigated for diabetic status including:-

- control of diabetes and mode of therapy
- presence of complications

The information was collected through direct patient interviewing, study of patients' hospital records and discussion with the patient's physician.

Laboratory investigation of random blood sugar which is done routinely in the diabetic centre for all diabetic patients before they meet their physician was recorded.

All participants were examined for periodontal status dividing each jaw into three sextants, and the highest score was recorded for each sextant.

All sextants were examined and the following parameters were recorded:

1- Plaque index according to the criteria of Silness and Loe, (1964):

0- absence of plaque deposits

1- plaque seen after probing gingival margin
2- visible plaque
3- abundant plaque

2- Gingival index according to the criteria of Loe and Silness, (1963):
   0- normal gingiva
   1- mild inflammation, mild change in color & texture.
   2- Moderate inflammation, redness oedema & bleeding on probing.
   3- Severe inflammation ulceration tendency towards spontaneous bleeding

3- Calculus according to the following criteria:
   0- no calculus
   1- supra gingival calculus
   2- sub-gingival calculus

4- Probable Pocket Depth:
   Measured in mm, as the distance from the gingival margin to the base of the pocket (Fig. 3).

5- Attachment loss:
   Attachment loss was measured with recommended probing pressure of 25 gr. approximately and recorded as the distance from the cemento-enamel junction to the base of the gingival sulcus / periodontal pocket.
   All parameters were recorded using a Williams Graduated Periodontal Probe. All readings were done by one examiner (Mona) with the help of an assistant for recording.
   All data was recorded on special chart (Annex ii). Data was analyzed by statistician using SPSS. Comparisons between test and control groups were done using the \( t \) test and the significance level was pre-decided at the 5% level and Confidence Intervals at the 95% level.
Fig. (2) Jabir Abu El Izz Diabetic Center
Fig. (3) Periodontal Probing
(source: Clinical Periodontology, Carranza, 8th Ed.)
Fig. (4) Red enlarged gingiva of adult diabetic patient
(source: Clinical Periodontology, Carranza, 8th Ed.)

Fig. (5) Suppurating abscess of adult diabetic patient
(source: Clinical Periodontology, Carranza, 8th Ed.)
Results

Table 1 shows the distribution of sample according to age and gender. Males were 32% of the study and 54% of the control groups, and male total sample was 43%. Where as females were 68% of the study group, 46% of the control group and 57% of the total sample. The age range of participants was 30-55 years and the mean age was 45 years and 39 years for the study and control groups respectively.

Table 2 indicates the frequency and percent of systemic complications of diabetes mellitus (retinopathy and neuropathy) among the study group showing that 22% of the diabetic patients had complications while 78% are free from complications.

The frequency and percent of tooth brushing among participants is shown in Table 3. 90% of the participants in the study group brush their teeth once to twice daily, while only 10% brush more than twice. 88% of the control group brush their teeth once to twice daily and only 12% brush more than twice.

The previous treatment received by patients or participants is shown in Table 4. The participants in the study group show that 10% received scaling, 60% extractions and 30% fillings. The control group showed 26% for scaling, 28% for extractions, 18% for fillings and 28% for other treatments.

The frequency and percent of previous periodontal problems among study and control groups is shown in Table 5. Among the study group 40% experienced bleeding, 34% loosened teeth, 14% abscess formation and 12% other periodontal problems. In the control group 38% had bleeding, 42% loosened teeth, 8% abscess formation and 12% other periodontal problems.

The number and percent sextants with different scores of plaque index is shown in Table 6. The study group showed 24% of the sextants with plaque score 0,
53% plaque score 1, 17% plaque score 2, 6% with plaque score 3. The control group showed 29% with plaque score 0, 40% plaque score 1, 20% plaque score 2, and 11% plaque score 3.

Table 7 shows the mean plaque index for participants. There was no statistically significant difference between the study and control groups (p=0.3).

Table 8 indicates the number and percent sextants with different scores of gingival index. The study group showed 12% gingival score 0, 28% gingival score 1, 46% gingival score 2, and 14% gingival score 3. The control group showed 25% gingival score 0, 34% gingival score 1, 33% gingival score 2, and 8% for gingival score 3.

Table 9 shows the mean gingival index for participants. There was statistically significant difference between the study and control groups (p=0.009).

The number and percent sextants with different scores of calculus is shown in Table 10. The study group showed 60% with no calculus, 21% showed supra gingival calculus, and 19% showed supra and subgingival calculus. The control group showed that 61% have no calculus, 14% showed supra gingival calculus only, and 25% showed supra and subgingival calculus.

Table 11 indicates the mean calculus for all participants. There was no statistically significant difference between the study and control groups (p=0.6).

Table 12 shows the mean probable pocket depth for participants according to group and gender. The study group showed a mean and standard deviation for males 2.8±2.4 mm while for females was 2.1±2.0 mm. The control group showed a mean and standard deviation for male participants of 2.7±2.3 mm while for females it was 2.0±1.9 mm.

Table 13 is showing the mean probable pocket depth for all participants. There was no statistically significant difference between the study and control groups (p=0.6)
Table 14 indicates the mean attachment loss of participants according to group and gender. The study group showed a mean and standard deviation for male participants of 6.2±3.7 mm, while for females it was 4.5±2.9 mm. The control group showed a mean and standard deviation for male participants of 3.9±3.5 mm while for females it was 3.0±2.6 mm.

Table 15 is showing the mean attachment loss for all participants. There was statistically significant difference between the study and control groups (p=0.002).

Tables 16, is showing the mean plaque index, gingival index, probable pocket depth, attachment loss and calculus for controlled and uncontrolled diabetics: The mean plaque index for the controlled diabetics was 1.1±0.5, while for the uncontrolled diabetics it was 1.0±0.5. The mean gingival index for the controlled diabetics was 1.5±0.7, while for the uncontrolled diabetics it was 1.7±0.4 mm. The mean probable pocket depth for the controlled diabetics was 2.3±1.8, while for the uncontrolled diabetics it was 2.1±1.3mm. The mean attachment loss for the controlled diabetics was 5.1±2.6 mm, while for the uncontrolled diabetics it was 5.0±2.3mm. The mean calculus for the controlled diabetics was 0.6±0.5, while for the uncontrolled diabetics it was 0.6±0.4. There was no statistically significant difference between controlled and uncontrolled diabetics in all periodontal parameters investigated.
Table 1: The distribution of sample according to age and gender

<table>
<thead>
<tr>
<th>Group</th>
<th>Gender</th>
<th>Age (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male No. (%)</td>
<td>Female No. (%)</td>
</tr>
<tr>
<td>Study</td>
<td>16 (32)</td>
<td>34 (68)</td>
</tr>
<tr>
<td>Control</td>
<td>27 (54)</td>
<td>23 (46)</td>
</tr>
<tr>
<td>Total</td>
<td>43 (43)</td>
<td>57 (57)</td>
</tr>
</tbody>
</table>

Table 2: The frequency and percent of systemic complications (retinopathy and neuropathy) of diabetes mellitus among study group

<table>
<thead>
<tr>
<th>Complications</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>11 (22)</td>
</tr>
<tr>
<td>Absent</td>
<td>39 (78)</td>
</tr>
<tr>
<td>Total</td>
<td>50 (100)</td>
</tr>
</tbody>
</table>

Table 3: The number and percent of frequency of tooth brushing among study and control groups

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Once to twice</td>
<td>45 (90)</td>
<td>44 (88)</td>
</tr>
<tr>
<td>More than twice</td>
<td>5 (10)</td>
<td>6 (12)</td>
</tr>
</tbody>
</table>
Table 4: The types of different previous treatments done for study and control groups

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scaling</td>
<td>5 (10)</td>
<td>13 (26)</td>
</tr>
<tr>
<td>Extractions</td>
<td>30 (60)</td>
<td>14 (28)</td>
</tr>
<tr>
<td>Fillings</td>
<td>15 (30)</td>
<td>9 (18)</td>
</tr>
<tr>
<td>Other Treatments</td>
<td>- (0)</td>
<td>14 (28)</td>
</tr>
</tbody>
</table>

Table 5: The frequency and percent of previous periodontal problems among study and control groups

<table>
<thead>
<tr>
<th></th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding</td>
<td>20 (40)</td>
<td>19 (38)</td>
</tr>
<tr>
<td>Loosened teeth</td>
<td>17 (34)</td>
<td>21 (42)</td>
</tr>
<tr>
<td>Abscess Formation</td>
<td>7 (14)</td>
<td>4 (8)</td>
</tr>
<tr>
<td>Others</td>
<td>6 (12)</td>
<td>6 (12)</td>
</tr>
</tbody>
</table>
Table 6: Number and percent sextants with different scores of plaque index

<table>
<thead>
<tr>
<th>Plaque score</th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>74 (24)</td>
<td>88 (29)</td>
</tr>
<tr>
<td>1</td>
<td>159 (53)</td>
<td>121 (40)</td>
</tr>
<tr>
<td>2</td>
<td>50 (17)</td>
<td>61 (20)</td>
</tr>
<tr>
<td>3</td>
<td>17 (6)</td>
<td>30 (11)</td>
</tr>
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</table>

Table 7: The mean plaque index for study and control groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Plaque Index $\bar{X}$+SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>1.0+0.8</td>
</tr>
<tr>
<td>Control</td>
<td>1.2+1.3</td>
</tr>
</tbody>
</table>

$t = -1.018$

$df = 49$

$p = 0.3$
Table 8: The number and percent sextants with different scores of gingival index

<table>
<thead>
<tr>
<th>Gingival score</th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>36 (12)</td>
<td>75 (25)</td>
</tr>
<tr>
<td>1</td>
<td>83 (28)</td>
<td>102 (34)</td>
</tr>
<tr>
<td>2</td>
<td>140 (46)</td>
<td>98 (33)</td>
</tr>
<tr>
<td>3</td>
<td>41 (14)</td>
<td>25 (8)</td>
</tr>
</tbody>
</table>

Table 9: The mean gingival index for study and control groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Gingival Index $\bar{x}$ ±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>1.6 ± 0.9</td>
</tr>
<tr>
<td>Control</td>
<td>1.2 ± 0.9</td>
</tr>
</tbody>
</table>

$t = 2.702$
$df = 49$
$p = 0.009$
Table 10: The number and percent sextants with different scores of calculus

<table>
<thead>
<tr>
<th>Calculus</th>
<th>Study No. (%)</th>
<th>Control No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>181 (60)</td>
<td>182 (61)</td>
</tr>
<tr>
<td>1</td>
<td>61 (21)</td>
<td>42 (14)</td>
</tr>
<tr>
<td>2</td>
<td>58 (19)</td>
<td>76 (25)</td>
</tr>
</tbody>
</table>

Table 11: The mean calculus scores recorded for study and control groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Calculus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\bar{x} + SD$</td>
</tr>
<tr>
<td>Study</td>
<td>0.6+0.4</td>
</tr>
<tr>
<td>Control</td>
<td>0.7+0.6</td>
</tr>
</tbody>
</table>

$t = -0.558$
$df = 49$
$p = 0.6$
Table 12: The mean probable pocket depth for study and control groups according to gender

<table>
<thead>
<tr>
<th>Group</th>
<th>Male $\bar{X}$+SD</th>
<th>Female $\bar{X}$+SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>2.8+2.4</td>
<td>2.0+2.1</td>
</tr>
<tr>
<td>Control</td>
<td>2.7+2.3</td>
<td>2.0+1.9</td>
</tr>
</tbody>
</table>

Table 13: The mean probable pocket depth and range for study and control groups

<table>
<thead>
<tr>
<th>Group</th>
<th>P.P.D range (mm)</th>
<th>P.P.D $\bar{X}$+SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>0-12</td>
<td>2.2+2.2</td>
</tr>
<tr>
<td>Control</td>
<td>0-12</td>
<td>2.4+2.1</td>
</tr>
</tbody>
</table>

$t = -0.471$
$df = 49$
$p = 0.6$
Table 14: The mean attachment loss for study and control groups according to gender

<table>
<thead>
<tr>
<th>Group</th>
<th>Male $\bar{X}$±SD</th>
<th>Female $\bar{X}$±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>6.2±3.7</td>
<td>4.5±2.9</td>
</tr>
<tr>
<td>control</td>
<td>3.9±3.5</td>
<td>3.0±2.6</td>
</tr>
</tbody>
</table>

Table 15: The Mean Attachment Loss for study and control groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Attachment Loss $\bar{X}$±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>5.0±2.3</td>
</tr>
<tr>
<td>Control</td>
<td>3.5±2.3</td>
</tr>
</tbody>
</table>

$$t = 3.315$$  
$$df = 49$$  
$$p = 0.002$$
Table 16: The mean plaque index, gingival index, probable pocket depth, attachment loss and calculus for controlled and uncontrolled diabetics

<table>
<thead>
<tr>
<th>Group</th>
<th>Plaque Index</th>
<th>Gingival Index</th>
<th>P.P.D</th>
<th>Attachment Loss</th>
<th>Calculus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\bar{x}$+SD</td>
<td>$\bar{x}$+SD</td>
<td>$\bar{x}$+SD</td>
<td>$\bar{x}$+SD</td>
<td>$\bar{x}$+SD</td>
</tr>
<tr>
<td>Controlled</td>
<td>1.1+0.5</td>
<td>1.5+0.7</td>
<td>2.3+1.8</td>
<td>5.1+2.6</td>
<td>0.6+0.5</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>1.0+0.5</td>
<td>1.7+0.4</td>
<td>2.1+1.3</td>
<td>5.0+2.3</td>
<td>0.6+0.4</td>
</tr>
</tbody>
</table>

$P=0.6$ $P=0.2$ $P=0.6$ $P=0.9$ $P=0.9$
Discussion

4.1 Introduction:
The relationship between diabetes mellitus and periodontal disease appears to be strong, but a survey of the literature discussing the possible relationship between the two showed wide variation in opinion.

Periodontitis is a complex multi-factorial disease, similarly diabetes mellitus is a complex metabolic syndrome. It may be the complexities of both of these disease processes which may contribute to the controversy found in the literature about their relationship.

In this study, the periodontal health of diabetics was studied versus a control of patients attending a specialized periodontal department.

Taking into consideration that Sudanese patients are not motivated towards dental and periodontal health, the control group represents non-routine attenders of dental clinics. They attend only for the purpose of pain relief, which is a late symptom in periodontal disease if ever present.

However, a positive association in the results between the test and control groups in this study may support that, diabetic patients are at higher risk for periodontitis. This is because diabetic patients were not compared with healthy controls, but with patients attending for periodontal treatment. This fact indicates that, the control patients suffered a periodontal problem which reached a stage enforcing them to present themselves to a clinician for treatment.

In this sample diabetics were more females compared to males, this however does not entail gender predilection of the condition. In fact it may be because the examination was carried out during the day where most males are at work.

Of the diabetic group 22% showed complications, and while all participants brush their teeth, 90% of them brush 1-2 times a day. In fact all participants had been to a dentist before, but diabetics had more visits for extraction of teeth than
other treatments including prophylaxis, inspite of the fact that at least more than 40% of them suffered a periodontal problem before (Tables 4 and 5). However comparing periodontal parameters between test and control groups revealed the following:

4.2 Plaque index:
The mean plaque index for diabetics is $1.0\pm0.8$ while the mean plaque index for non-diabetics is $1.2\pm1.3$, statistical testing concluded that there is no statistically significant difference between the two groups ($p=0.3$).
This results is consistent with those of Benveniste (1967) who found no significant difference between the diabetic and non-diabetic patients in the amount of bacterial plaque (82). The results are also in agreement with Bernick et al (1975) who found no significant difference between diabetic and non-diabetic groups in plaque deposits (80).
However, these results are contradictory to Arthur (1991) who concluded that the mean plaque indices were significantly higher in the diabetic group of patients when compared with non-diabetics (62). It seems that while the diabetic condition may play a role in the amount of plaque deposition, oral hygiene practices may lead to the differences usually noticed between the different groups investigated.

4.3 Gingival index:
The results of this study indicated that the mean gingival index of diabetic patients is higher than the mean gingival index of the control group $(1.6\pm0.9$ and $1.2\pm0.9$ respectively).
This higher mean for diabetics can not be justified by the differences in the amount of plaque alone as this difference in plaque index for the two groups is of no significance.
So this gingival alteration in diabetics could be the result of diabetes vascular changes which alter the tissue response to bacterial insults. The difference between the two groups was statistically significant (p=0.009). These results are consistent with the results of Cohen et al (1970) and Bernik et al (1975) who found that diabetics had an increased degree of inflammatory changes compared with healthy control (77).

Cohen et al (1970) noted that gingival index of the diabetic patients was significantly higher when compared with the control, but the authors obtained no positive correlation between gingival index and soft deposits index and explained this only in the suggestion of reduced resistance of diabetics to infection, which may also be consistent with the results of the present study (77).

As for the uncontrolled diabetics the results disagree with the results of Kjellman et al (1970) who came to the conclusion that diabetes with poor cooperation in the care of the disease (uncontrolled) had more gingivitis than those with good control (controlled) (92). However the results also agree with those of Arther et al (1991) who concluded that the mean gingival index among diabetics was significantly higher when compared with non-diabetic controls (62). The same is true with the results of Hove and Stallard (1970) who discovered more vascular gingival changes in diabetics than in non-diabetics (83).

Many cross sectional studies Knowler (1978) (72) and Shlossman (1990) (73) and longitudinal studies Nelson et al (1990) (74) have concluded that diabetes increases the risk for developing periodontitis in a manner that can’t be explained on the basis of the dental plaque or age.

These conclusions support the results of the present study. However the results are inconsistent with other investigations including those of Bay et al (1974)
who found no statistically significant difference between diabetic patients and non-diabetic subjects in terms of gingival index.

4.4 Probable Pocket Depth:
Concerning the parameter of probable pocket depth (PPD) the mean PPD for diabetics was 2.2±2.2 mm and for non-diabetics was 2.4±2.1 mm. There was no statistically significant difference in the PPD between diabetics and non-diabetics (p=0.6). This result can be explained by the fact that periodontal disease and diabetes are two chronic diseases with too many variables inherent in studying these two disease which may result in less clear association in selected populations.

These results are consistent with the results of Benveniste et al (1967) who found that formation of pockets in diabetics and non-diabetics show no statistically significant differences (82). The same is true when the results are compared with those of Rylandar et al (1980) who found no statistically significant difference in pocket depth between diabetics and non-diabetic groups (79).

Sheriden (1959) showed that pocket formation and tooth loss occurred with greater frequency in patients with poor controlled diabetes (93). However in the results of the mean PPD according to gender, it was found that the:

mean PPD for diabetic males was 2.8±2.4 mm, while the mean for diabetic females was 2.1±2.0 mm. The results for non-diabetic males was 2.7±2.3mm, while for non-diabetic females it was 2.0±1.9mm.

This difference can only be explained by the fact that females are more aware about their dental health, and show better oral hygiene practices.

4.5 Attachment Loss:
In this study it was found that the mean attachment loss among diabetics was greater than non-diabetics the mean attachment loss for the two groups was 5.0±2.3 mm and 3.5±2.3 mm respectively. The results show that recession is a problem among both groups but there are high differences within the groups as indicated by the high standard deviation. The results also showed statistically significant difference between the study and control groups (p=0.002).

This result can only be explained by the suggestion that the healing process is decreased as a result of disturbance in cellular carbohydrate metabolism. So the periodontium inflames more, this in addition to faulty brushing that may lead to increased gingival recession.

These results are consistent with those of Grossi et al (1997), as they showed that diabetic patients were twice as likely as non-diabetics subjects to have attachment loss (53). The results are also in agreement with those of Firatli (1997) who followed type 1 and healthy controls for 5 years and came to a conclusion that people with diabetes has significantly more clinical attachment loss than controls (68).

However, the results also support the findings of Bridges et al (1996) who concluded that diabetes affects all periodontal parameters including bleeding, probable pocket depth, loss of attachment and missing teeth (69).

On the other hand the results disagree with other investigator who found no significant difference in the periodontal condition of diabetics and non-diabetics including attachment loss, including those of Ciacciola (1982) (64), Ervasti (1985) (66) and Tervonen and Karjalainen (1997) (94).

4.6 Calculus:

Concerning calculus the mean for diabetics was 0.6±0.4 while for non-diabetic it was 0.7±0.6. This difference was statistically insignificant.
The results agree with those noted by Cohen et al. (1970) who found no relation between gingival index and hard and soft deposits indices, among diabetics (77). Tervonen and Oliver (1994) have reported more subgingival calculus in poorly controlled diabetic patients than in well controlled patients (37). However, few studies examined calculus deposition among diabetics and most investigators concentrated on soft deposits because of their importance in the initiation and development of periodontal disease.

4.7 Controlled versus Uncontrolled diabetes:
The parameters for controlled and uncontrolled diabetics is shown in table 16. For all parameters there was no statistically significant difference between the two groups.

It is the long term poor control which affect periodontal parameters. The test used in this study is the random blood sugar which is an instantaneous estimation of the blood sugar, so it is unsuitable for evaluation of long term poor control. This is in addition to that 17% of the controlled diabetic patients in this study has complications which is another risk factor for severe periodontal destruction. It may be the complexity of diabetes with many variables resulted in this insignificant difference between controlled and uncontrolled diabetic patients. This support the assumption that comparisons based on metabolic control alone may be inappropriate.

However these results disagree with those of Ervasti et al. (1985) who studied the relationship between diabetic control and the reduction in gingival bleeding (66). In their study they found a significant reduction in gingival bleeding among controlled compared to uncontrolled diabetics. However the same authors indicated no significant increase in gingival swelling, an important sign of gingivitis.
The same results were also obtained by Sastrowijoto et al (1990) who studied the effects of improved metabolic control on the clinical periodontal condition and subgingival microflora among diabetics (33). The authors concluded that gingival redness decreased significantly whereas gingival swelling showed no trend to decrease. However while these results seems contradicting to the results of the present investigation, careful reading of the results may show some agreements. The fact that in this study the gingival index was used (Loe and Silness, 1963) rather than a bleeding index may explain the non-significant difference in the periodontal parameters specially the gingival index between controlled and uncontrolled diabetic patients.

While the results of the present investigation indicated that diabetics have more gingivitis and attachment loss compared to non-diabetics.

The results still requires further in depth analysis. However the results of this investigation can only be applied for the group under investigation and for populations with similar conditions and the results should not be generalized.
Conclusions and Recommendations:

4.8 Conclusions:
In the studied population the results show that:

1- Diabetes mellitus does not significantly affects bacterial plaque deposition and pocket formation, but it affects negatively the gingival condition leading to gingivitis and periodontal condition leading to increased attachment loss presenting as gingival recession.

2- Both study and control groups showed more recession compared to pocket depth indicating that other factors may have lead to this picture of periodontal disease example oral hygiene methods.

3- Uncontrolled diabetics showed more periodontal disease parameters compared to controlled diabetics, but the results are statistically not significant.

4.9 Recommendations:

1- Diabetics should be instructed in meticulous oral hygiene measures and be informed on the importance of routine dental checkups.

2- A dental unit and a dentist and dental hygienist must be part of any setup caring for diabetic patients whether in general hospitals or specialized centers.

3- The problem of increased gingival recession among diabetics and non-diabetics should find more attention and should be investigated, perhaps in large scale samples.
References

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84. Goteinner D, Vogel R, Deasy M, Goteiner C. Periodontal and caries
30: 298.

Annex i

جمهورية السودان
كلية طب الأسنان
جامعة الخرطوم

الпїЅلسأرلإم

Republic of Sudan
Faculty of Dentistry
University of Khartoum

تذكر - محدد مرض جورج أبو العفر لمرضى المستشفى

"سلام عليكم ورحمة الله تعالى وبركاته"

الموضوع:

بخصوص الموضوع أعلاه أفيد سيدتكم بأن الدكتورة منى عوض كمال تحتاج لعمل

5 (خمسون) ىن بمرضى السكري فوق الثلاثين عاماً من المرضى الدائمين للمركز.

نرجو تسهيل مهامها ونسأل الله أن يوفق الجميع لما فيه الخير للجميع.

ولكم الشكر والتقدير

[ลาย]

P.O Box: 102 Khartoum
Telephone Fax : 780088 Email : Kh_dent@hotmail.com

78 - 198-888
Annex ii
Questionnaire

NAME:                                         Age:

Residence and address:

Gender:         Male     Female

Systemic complication of D.M       Present     Absent

Random blood sugar

Frequency
of cleaning the mouth
None    Once    twice    more

Previous visits to dentist for:
Scaling     extraction     filling

Other treatment

Previous periodontal problem
Bleeding    Loosened teeth
Abscess formation    other

Plaque index  Gingival index

P.P.D  Attachment loss

Calculus