The Association of Periodontal Disease with Metabolic Control in Type 1 Diabetic Adolescents

UNIVERSITY *of the*
WESTERN CAPE

A mini-thesis submitted in partial fulfillment of the Requirement for Master's Degree in Periodontology, Department of Periodontology and Oral Medicine

Dr. Mohamed Abdelrahman

Student No.: 3820857 Supervisor:

Dr. Anthea Jeftha

Co-Supervisor: Dr. Marli Conradie-Smit

December 2020

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Dr. Mohamed Abdelrahman

KEYWORDS

Adolescents

HBA1C

Glycaemic control

Metabolic control

Periodontal disease

Periodontal status

Periodontitis Prevalence

Type 1 diabetes

ABSTRACT

Background: Chronic inflammation of the periodontium is known as periodontal disease. The inflammation can be contained only within gingival connective tissue or can progress, leading to the loss of gingival connective tissue and alveolar bone. Lately, periodontal disease is considered as a co-morbidity of diabetes mellitus (Polak, Sanui *et al.*, 2020). Though studies that have assessed the relationship of periodontal status with the glycemic control in type 1 diabetes mellitus adolescents, such studies have not been conducted in South Africa.

Aim: This study compared the periodontal status of adolescents with Type 1 diabetes mellitus (T1DM) with their diabetic metabolic control.

Method: This cross-sectional study was conducted in 58 adolescent patients diagnosed with T1DM. Disease of the periodontium was assessed using the American Academy of Periodontology as well as the European Federation of Periodontology 2017 classification. Periodontal parameters were assessed and the periodontal health of the diabetic adolescents was compared to their glycemic control.

Results: Out of the 58 diabetic adolescents, 44 (75.86%) had gingivitis, 6 (10.34%) had periodontitis and 8 (13.79%) adolescents showed a healthy periodontal status. Forty out of the 44 gingivitis patients had uncontrolled diabetes, 4 out of the 6 patients with periodontitis had uncontrolled diabetes, while 7 out of the 8 patients with healthy periodontal status, had uncontrolled diabetes. There was no association between periodontal diagnosis and metabolic control, p=0.231.

Conclusion: Though more adolescents in this sample had been diagnosed with periodontal disease, there was no association between periodontal diagnosis and metabolic control in T1DM adolescents.

DECLARATION

I, Mohamed Abdelrahman, hereby declare that the work contained in this dissertation titled; "The Association of Periodontal Disease with Metabolic Control in Type 1 Diabetic Adolescents*"* is my original work and has not been formerly in its totality or in any part submitted at any academic institution for degree or examination.

Dr. Mohamed Abdelrahman 14 December 2020

ACKNOWLEDGEMENT

First I would like to give thanks to Allah for affording me the opportunity, might and courage to complete this study.

Great thanks to my supervisor, Dr. Anthea Jeftha, for the kind supervision, help and encouragement in this thesis. It was an honor to work with her.

I want to acknowledge Dr. Conradie-Smit for her great advice, help and guidance in completing this research and to Dr. F. Kimmie-Dhansay for putting effort and assistance in my data analysis.

Lastly, I give special thanks to my beloved parents, fiancé and peers for their blessings and motivation, I am fortunate to have them as part of this journey.

ABBREVIATIONS

AAP	American Academy of Periodontology
AIDS	Acquired Immunodeficiency Syndrome
ANOVA	Analysis of variance
BMREC	Biomedical Research Ethics committee
CEJ	Cement-enamel-junction
Clinical AL	Clinical attachment level
DKA	Diabetes ketoacidosis
DM	Diabetes mellitus
EFP	European Federation of Periodontology
FBG	Fasting blood glucose
FBS	Fasting Blood Sugar
HbA1c	Glycated Haemoglobin
HIV	Human Immunodeficiency Virus
IDF	International Diabetes Federation
LOA	Loss of attachment
MM	Millimeters
N	Number
NA	Not applicable
P gingivalis	*Porphyromonas gingivalis*
PD	Periodontal disease
PPD	Probing pocket depth
RANKL	Receptor activator of nuclear factor kappa-B ligand
SD	Standard deviation

STATA	Statistical software
SU HREC	Stellenbosch University Health Research Ethics Committee
T1DM	Type 1 diabetes mellitus
T2DM	Type 2 diabetes mellitus
USA	United States of America

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CHAPTER ONE

Introduction

Chronic inflammatory disease of the attachment apparatus surrounding the teeth is known as periodontal disease. The inflammation can be contained only within gingival connective tissue or can progress causing loss of gingival connective tissue and alveolar bone (Socransky 1977, Kinane *et al.*, 2001, Roy *et al.*, 2004, Sjdin, *et al.*, 2012). It had been reported that 90% of the world's population is affected by gingivitis, where the chronic inflammatory disease process affects the gingival connective tissues only.

Periodontitis is an inflammatory breakdown of periodontal ligament, cementum, gingiva, and alveolar bone, distinguished by the loss of collagen and connective tissues in the gingiva followed by periodontal ligament and alveolar bone loss. With the progression of periodontitis, loss of tooth function, structure and stability occur. It may also trigger a generalized systemic inflammatory response and even sepsis, especially in patients with compromised immunity for example patients with diabetes mellitus (Loos, Van Dyke 2020).

In 2009, 8 % of patients that have been part of a British adult dental health survey accounted for having a pocket depth (a clinical parameter to diagnose periodontitis) of 6 millimeter (mm) or more (White et al., 2011). Other workers report that approximately 14% of the world's population suffers from periodontitis (Soskolne et al., 2001).

With reference to the 1999 American Academy of Periodontology (AAP) classification, chronic periodontitis is frequent in individuals diagnosed with (T1DM) and it can depreciate the glycemic control (American Academy of Periodontology 1999, Sakalauskiene et al., 2014, Borilova et al., 2016).

Diabetes mellitus (DM) is a metabolic disease, where there is increased glucose level in the blood) produced by deficiency in insulin secretion, insulin action or both (Novotna et al., 2015). It is generally categorized into four categories: type 1 diabetes mellitus (T1DM) (absolute insulin deficiency), type 2 diabetes mellitus (T2DM) (underlying insulin resistance, with concomitant loss of beta-cell function), gestational diabetes (detected in fourth to ninth

month of gestation and not evident before) and other sources have been linked to explicit types of diabetes (for example pancreatic destruction) (American Diabetes Association Standards of Care in Diabetes 2018).

T1DM is caused by an autoimmune destruction of pancreatic beta-cells and the consequent inability to produce insulin. T1DM is most prevalent in children. Around 3.8 % of the American population are affected by T1DM, which is around 5% to 10% of total cases of diabetes mellitus (Centers for Disease Control and Prevention 2011, Newman et al., 2011).

The lack of insulin, with resultant chronic hyperglycemia, may result in severe complications, both microvascular and macrovascular (Bluestone et al., 2010). Type 1 diabetic patients rely on the ingestion of exogenous insulin for regulating the disease and preventing both acute and chronic complications. Insulin treatment may however lead to hypoglycemia (Cryer 2008, Davis-Richardson et al., 2014).

Periodontal disease has recently been considered as a co-morbid condition to diabetes mellitus. The transfer from an interdependent microbiome to a microbial dysbacteriosis causes an inflammatory response in the periodontal membrane, cementum and alveolar bone. This inflammatory response is a common characteristic in both diabetes and periodontitis. Both respond to the dysbiotic microbiome by triggering an impaired inflammatory and immune response as well as impaired healing (Polak, et al., 2020).

Diabetes has a deleterious impact on developing periodontitis, as it was previously stated that diabetes influences the development of periodontal disease (Newman 2011, Takei et al., 2011).

Studies have demonstrated an association linking impaired glucose tolerance in individuals with diabetes mellitus and alternations in the microbiome of the periodontium. The majority of studies illustrated that individuals with inflammation of the periodontium and diabetes exhibit high levels of pro-inflammatory mediators in the blood, especially C-reactive protein, oxidative stress and tumor necrosis factor alpha, which may have an impact on controlling diabetes. A number of studies showed some evidence on improved diabetic control following periodontal therapy, as there is a decrease in the circulating pro-inflammatory mediators,

which also improve the lipid profiles (Sanz, et al., 2018).

In individuals with unsatisfactory control of their diabetes, chronic inflammation of the periodontium is three times as frequent as contempt sub-gingival biofilm (Sima et al., 2013). The national Health and Nutrition Examination Survey 2009-2010 reported that 12.5% that 12.5% of diabetic patients had inflammation of the periodontal tissues, whereas 6.3% had an intact periodontium. (The National Health and Nutrition Examination Survey 2009-2010, Arora et al., 2014).

The periodontal status of T1DM adolescents is basically under reported and, in South Africa, there is no such data available. Therefore, the purpose of this study is to focus on periodontitis combined with T1DM as well as to designate the status of the periodontium in this risk group.

CHAPTER TWO

Literature review

Periodontal disease

Periodontal disease (PD) comprises a spectrum of diseases that involve the tissues that constitute the attachment or support of teeth. These tissues include gingivae, periodontal ligament, cementum and alveolar bone. Microorganisms in the dental biofilm trigger the inflammatory lesions that ultimately lead to damage and destruction of the tooth supporting tissues (Denisse et al., 2017).

Periodontal health is described as the natural healthy state of the periodontium without any evidence of inflammation in the form of gingivitis or periodontitis. As part of homeostasis, the physiological immune markers are responsible for maintaining a healthy periodontium. Clinical gingival health is categorized on site level into clinical gingival health on a healthy attachment apparatus, and clinical gingival health on a reduced attachment apparatus (Chapple *et al.,* 2018).

Clinical gingival health on healthy attachment apparatus shows several clinical features such as no bleeding on probing, patient symptoms, edema, erythema and bone or clinical attachment level. Bone loss in a healthy individual varies from one to three millimeter (mm) apical to cement-enamel-junction (CEJ). Clinical gingival health on a reduced attachment apparatus may be found in individuals either with stable periodontitis or patients with gingival recession or crown lengthening surgery (non-periodontitis) (Chapple *et al.,* 2018).

It is distinguished by lack of erythema, edema, bleeding on probing and patient symptoms with the existence of attachment and bone loss. Individuals with stable periodontitis or those who have been fully treated are at high risk of recurrence. Therefore, patients with stable periodontitis on a reduced periodontium should be differentiated from those without periodontitis because of the increase in risk of progression of periodontitis (Chapple *et al.,* 2018).

Periodontal health on an intact periodontium, clinically shows no probing attachment loss and there is no alveolar bone loss, but has a probing pocket depth equal to or less than 3 mm, and bleeding on probing less than 10%. Periodontal health on a reduced periodontium in patients without inflammation of the attachment apparatus present loss attachment on probing with possible radiographic loss, probing pocket depth equal to or less than 3 mm and bleeding on probing less than 10%. Periodontal health on a reduced periodontium in individuals who have been fully managed or stable from periodontitis revealed radiographic bone loss and probing attachment loss, probing pocket depth less than or equal to 4mm and bleeding on probing less than 10%. (Chapple *et al.,* 2018).

There are three classes that can determine periodontal health Microbiological such as supra-gingival plaque composition and sub-gingival biofilm composition Environmental factors such as medication, stress, smoking and nutrition, and finally, host factors that is further categorized into focal predisposing factors such as periodontal pockets, restorations, root morphology tooth site and crowding, and systemic modifying factors such as genetics, general health and host immune function (Lang *et al.*, 2018).

Gingivitis is a category of periodontal disease, that is further classified into plaque-induced gingivitis and non-plaque induced gingivitis. Plaque-induced gingivitis is described as the reaction between the dental plaque and the human immune-inflammatory response that leads to an inflammatory lesion, limited to the gingiva and does not affect the muco-gingival junction and periodontal attachment such as periodontal ligament, alveolar bone or cementum. This can be treated by decreasing the amount of plaque on the tooth surface (Chapple *et al.*, 2018).

Furthermore, inflammation of the gingivae can also be categorized into gingivitis on an undamaged periodontium, gingivitis on weakened periodontium in treated periodontitis, and non-periodontitis individuals, according to either, the gingival inflammation caused by dental plaque on a healthy or on a weakened periodontium (Chapple *et al.*, 2018).Certain predisposing risk factor either (local) or modifying (systemic) risk factors are used to distinguish the intensity, extent and rate of development of gingivitis (Chapple *et al.,* 2018).

Local risk factors include mouth dryness that result in decreased teeth surface clean-up

which diminishes the removal of the dental plaque, thus encouraging inflammation of the gingiva. Another predisposing factor is the retention factors such as tooth anatomical factors that increases plaque deposition at the gingival margin and apically, allowing biofilm adhesion and growth making the removal of plaque more difficult. Systemic risk factors include smoking, metabolic disease and nutritional deficiencies. Smoking activates micro-vascular vasoconstriction and fibrosis that may conceal bleeding on probing clinically, despite the large number of inflammatory infiltrates. Hyperglycemia is a metabolic factor that passively activates mitochondrial stress leading to the induction of several pro-inflammatory mediator episodes. Nutritional factors such as lack of vitamin C which affects the synthesis of collagen leading to capillary wall frailty and a possible tendency of gingival bleeding (Chapple *et al.,* 2018).

Drugs may also function in several ways to increase the risk of gingivitis. Certain drugs may decrease the flow of saliva, leading to gingival hyperplasia, whereas other medications affect the level of hormonal secretion. During pregnancy the levels of estrogen, androgens and progestogens are highly elevated which produces abnormal gingival inflammation with a little amount of dental plaque, a similar condition is seen during puberty and in individuals taking oral contraceptives. Blood malignant and pre-malignant diseases such as leukemia and myelodysplasia respectively, both cause signs of pale purple swollen gingiva and small level of gingival bleeding (Chapple *et al.*, 2018).

The assessment of gingivitis can be made clinically by the presence of several inflammatory signs such as swelling, redness, lack of function, bleeding on probing and pain and the patients usually complain from several symptoms such as soreness, struggle on eating, bad breath and spontaneous bleeding, although gingivitis can be assessed clinically, it cannot be assessed radiographically (Chapple *et al.,* 2018).

Plaque-induced gingivitis on a healthy periodontium manifest clinically as bleeding on probing ≥10% on the total number of teeth and probing pocket depth ≤3 mm, does not reveal probing attachment loss nor alveolar bone loss. On a weakened attachment apparatus in individuals with stable periodontitis and those without inflammation of the periodontal tissues clinically there is loss of attachment on and bone loss can be detected

radiographically, it shows the same measurements for probing pocket depth and bleeding on probing as in gingivitis on an intact periodontium. Localized gingivitis represents bleeding sites of 10% to 30%, whereas generalized gingivitis represents bleeding sites greater than 30% (Chapple *et al.,* 2018).

Non-plaque induced gingival diseases are usually localized to the oral cavity but can also present as manifestations of systemic diseases. According to the lesion's etiology, it can be classified into specific infections (bacterial, viral and fungal), genetic disorders, reactive process, endocrine, nutritional and metabolic diseases, inflammatory and immune conditions and lesions, gingival pigmentation, traumatic lesions and neoplasms (Holmstrup et al., 2018).

Periodontitis is clinically diagnosed when the gingival tissues and alveolar bone are destroyed and the periodontal pockets are deep. In complex phases, it can trigger the teeth to be weak which eventually leads to its loss (Denisse *et al.,* 2017).

Based upon the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) 2017 classification, three divisions of periodontitis have been acknowledged this includes: necrotizing periodontal disease, periodontitis as a manifestation of systemic disease and periodontitis based on staging, grading, extent and distribution (Tonetti *et al.,* 2018).

The new 2017 classification updated the terminology of necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis to necrotizing gingivitis and necrotizing periodontitis, respectively (Herrera *et al*., 2018).

Necrotizing periodontal disease comprises a spectrum of inflammatory periodontal diseases including necrotizing gingivitis, necrotizing periodontitis and necrotizing stomatitis and noma. These inflammatory periodontal diseases are various phases of the same disease but share the same, etiology, clinical features and treatment (Herrera *et al*., 2018).

Necrotizing periodontal disease affects individuals with specific risk or predisposing factors such as improper oral hygiene and previously existing gingivitis or with history of necrotizing periodontal disease, stress, poor nutrition, immune-compromised conditions such as HIV and AIDS, tobacco smoking and alcohol intake, local factors, seasonal

variation and young age (Herrera *et al*., 2018).

Poor oral hygiene led to plaque deposition on the teeth that may result in necrotizing periodontal disease and can even progress because of the restricted brushing due to soreness. Necrotizing periodontal disease often takes place after having a history of periodontal disease (Herrera *et al*., 2018).

Stress changes the response of the immune system that may further result in the decrease of the flow of saliva and gingival blood flow, elevates the amount of *Prevotella intermedia* periodontal pathogen and quantity of 17-hydroxycorticosteroid in the urine and blood, and the role of specific white blood cells and polymorphonuclear leukocytes (Herrera *et al.*, 2018).

Malnutrition is common in poor countries that decreases the compounds that inhibit oxidation and changes the response towards infections, suppressor T-lymphocytes is inversely proportional to the helper, elevates free cortisol in saliva and serum and causes impaired intact mucosa (Herrera *et al*., 2018).

HIV patients are at high risk of disease worsening, are more susceptible to disease reoccurrence and have a weak response to treatment (Herrera *et al*., 2018).

Tobacco smokers are more likely to develop necrotizing periodontal disease while the intake of alcohol is related to psychological and physiological factors that may promote necrotizing periodontal disease (Herrera *et al*., 2018).

Orthodontic treatment and ornamental crowns are local factors that may result in rise of necrotizing gingivitis. Necrotizing periodontal disease is relatively high in the wet season in Africa but not in South Africa (Herrera *et al*., 2018)

Individual aged 15-34 with other predisposing factors and those less than 15 years old with other infections and poor nutrition in poor countries are highly susceptible to necrotizing periodontal disease (Herrera *et al*., 2018).

Necrotizing gingivitis is limited to the gingival tissue that represents several clinical features such as soreness, bleeding, bad breath, pseudo membrane development, erosion

and necrosis in the papillae and other extra oral features such as lymphadenopathy and pyrexia, and the loss of the attachment is not established, however children do not usually show features of pain or bad breath, but lymphadenopathy, pyrexia and hyper salivation is common (Herrera *et al*., 2018).

Necrotizing periodontitis is exemplified by loss of the periodontal attachment, focal mortification of the papillae that lead to the breakdown of the tissue generating a pit and also bone damage that may even progress to bone death in acute immune-compromised individuals. It may be due to several necrotizing gingivitis cascades or on a site with periodontitis history and been affected with necrotizing gingivitis (Herrera *et al*., 2018).

Necrotizing stomatitis is distinguished by an erosion greater than 1mm from the gingival margin and tissues following the muco-gingival junction leading to bone inflammation and death in immune-compromised patients and those with acute poor nutrition. It is an infectious disease caused from certain bacterial species such as *Selenomonas, Fusobacterium* and *Treponema* species (Herrera *et al*., 2018).

A variety of systemic diseases and conditions have a significant periodontitis manifestation such as systemic disorders having a great effect on the forfeiture of periodontal tissue by impacting the inflammatory periodontium, this in turn leads to severe early onset of periodontitis; disorders that impact periodontal disease pathogenesis thus increasing the prevalence and severity; and finally conditions that precede to loss of the periodontal tissue autonomous of plaque-induced periodontitis (Albandar, *et al*., 2018).

Periodontitis can be classified based on staging identified by the severity of periodontitis, complexity of management of the disease and extend and distribution as a describer for each stage. Grading periodontitis is used to estimate the risk of progression in periodontitis (Tonetti, *et al.,* 2018).

There are four different stages of periodontitis. Stage I periodontitis, known as initial periodontitis, that expresses the initial levels of attachment loss and stage 1 patients usually present with long-term inflammation of the gingiva and microbial imbalance in the oral biofilm. Clinically, stage 1 represents radiographic alveolar bone loss less than 15% in the

coronal part, interdental clinical attachment loss at site with significant loss of 1-2 mm to determine the severity and probing pocket depth of 4 mm of less with a horizontal bone loss (Tonetti, et al., 2018).

Moderate periodontitis or stage 2 has an uncomplicated therapy that requires the elimination of bacteria and observation to limit periodontal destruction, clinically shows attachment level of 3-4 mm, bone loss 15% - 33% in the coronal region and probing pocket depth less than or equal to 5 mm with a horizontal bone loss. Stage 1 and 2, both do not show any evidence of tooth loss caused by periodontitis (Tonetti, *et al.,* 2018).

Stage 3, known as severe periodontitis causes great defect in the tissues that assign the tooth to the alveolar process such as cementum, fibra periodontalis and alveolar bone, and lack of therapy may lead to tooth loss. It clinically represents a maximum loss of 4 teeth, bone loss that reaches mid-root, moderate ridge damage, class 2 or 3 furcation lesion and probing pocket depth of 6mm or more with a vertical bone loss more than or equal to 3 mm (Tonetti, *et al.,* 2018).

Stage 4, known as advanced periodontitis, result in greater defect in both attachment and bone, compared to stage 3 and with much greater tooth loss, it is clinically represented by loss of 5 or more teeth, bone loss that reaches mid- root and apical part, impairment in the function of the masticatory, alveolar ridge damage, loss of vertical dimension of occlusion, mesial shifting and excessive tooth mobility due occlusal trauma that requires several levels of oral therapy (Tonetti, *et al.,* 2018).Stage 3 and 4, both represent attachment loss more than or equal to 5 mm. Some local complexity factors for example furcation involvement, drifting and hypermobility may shift the initial stage to the other (Tonetti, *et al.,* 2018).

The extent and distribution describe each stage and is defined at each stage as molar-incisor pattern that was previously known in the older classification as localized juvenile periodontitis in children, localized form less than 30% of sites is involved , generalized form more than or equal to 30% of sites are involved (Tonetti, *et al.,* 2018).

Grading periodontitis enables the estimation of the rate of disease progression. The primary

criteria of periodontal grading depend on direct or indirect clues for disease progression; direct evidence depend on panel data such as clinical attachment loss and bone loss, while the indirect evidence depend on the percentage of alveolar bone loss as a unit of age and observational characteristics (Tonetti, *et al.,* 2018).

Risk factors that alter the grading of periodontitis are for example: glycemic control of diabetes mellitus and smoking may influence the rate of periodontitis worsening and elevate the transformation of one grade to the other (Tonetti, *et al.,* 2018).

There are three grades of disease progression; grade A, grade B and grade C that represent slow, moderate and rapid disease progression, respectively (Tonetti, *et al.,* 2018).

Patients with grade A, exhibit no alveolar bone loss or clinical attachment loss over five years but clinically represent a percentage of bone loss per unit of age of Less than 0.25 and great plaque accumulation with small amount of damage, theses patient are usually non-smokers and non-diabetic (Tonetti, *et al.,* 2018).

Grade B clinically, shows indication of alveolar bone loss or attachment loss less than 2 mm over five years, percentage of bone loss per unit of age of 0.25-1.0 and plaque accumulation is proportional to destruction. Patients with moderate disease progression usually smoke less than 10 cigarettes in one day and have well-controlled diabetes (Tonetti, *et al.,* 2018).

Rapid periodontitis progression or grade C patients exhibit alveolar bone loss or clinical attachment loss of 2 or more over 5 years, clinically represents a percentage of bone loss per unit of age greater than 1, plaque accumulation, heavy destruction and specific periodontitis pattern that led to poor response to the treatment of bacterial control. These patients usually smoke more than 10 cigarettes in one day and have uncontrolled diabetes as a risk factor (Tonetti, *et al.,* 2018).

To recognize the existence, sequence and severity of PD the following predisposing factors should be inspected namely, smoking, oral hygiene habits, medications, age and the existence of general diseases, such as diabetes (Garofalo 2008, Sanz-Snchez, *et al.,* 2008, Han *et al.,* 2012, Denisse *et al.,* 2017).

The oral microbial flora and the host response is susceptible to changes caused by smoking, smoking also elevates the number of periodontal microorganisms in the oral cavity (Nazir 2017). Porphromonas.gingivalis is found to interact with the nicotine found in cigarettes resulting in rise in the level of cytokine which leads periodontal tissue breakdown (Koshi, Rajesh *et al.*, 2012).

The development of periodontal disease is associated with certain factors such as poor oral hygiene habits, improper brushing and other hygiene measurements. These factors can stimulate the deposition of bacteria which leads to the development of dental plaque on the teeth surface and gums which changes the inflammatory level in the periodontal tissue. (Nazir 2017).

Certain medications are associated with the reduction of salivary flow in the oral cavity, these medications include Tricyclic antidepressants, atropine and beta blockers. Reduction in the salivary flow increases the patient's susceptibility to the development of periodontal disease and infections. Whereas other medications such cyclosporine, phenytoin and nifedipine are associated with gingival hyperplasia which renders the patient's ability to eliminate the dental plaque underneath the enlarged gingivae efficiently and therefore worsen the actual periodontal disease (Nazir 2017).

Studies have revealed that there is a direct correlation between age and the risk of development of periodontal disease. This may explain the increase in incidence of periodontal disease among those of 65 years and above (Nazir 2017).

There is a two-way correlation among diabetes and periodontal disease, both known as chronic complex diseases. Periodontal disease shows higher severity, prevalence and progression in diabetics compared to those non-diabetics, demonstrating diabetes as a significant risk factor for periodontitis. Conversely, periodontal disease has a momentous negative influence on the metabolic control of patients with diabetes mellitus (Lahariya, *et al.*, 2017).

An increased blood glucose level directly stimulates a pro-inflammatory host response in the periodontal tissues through an advanced glycation end product/receptor for advanced

glycation end product axis, which may assist in the function of fibroblast and leukocytes and change the ration of RANKL/osteoprotegrin. The correlation of advanced periodontal dysbacteriosis and increased blood glucose level may propose a connection, indirectly between the beginning and deterioration of periodontitis in uncontrolled diabetics. An increase in the toll-like receptors encourages a host response to dysbacteriosis which may elevate the inflammatory response (Polak, *et al.,* 2020).

Gingivitis is the predominant type of periodontal disease in adolescents. Furthermore, there is gingival proliferation as young adult turns to adult age (Duque, *et al.,* 2017). Focal irritants and infections change the inflammatory response of the mouth in individuals with systemic diseases, enhancing the development of PD and dental disease (Denisse *et al.*, 2017).

Diagnosis of periodontal disease

The AAP 1991 classification of periodontitis was as followed: chronic, aggressive (localized and generalized), necrotizing and as a manifestation of systemic disease, in 2017 it was reclassifies as followed necrotizing periodontitis, periodontitis as a manifestation of systemic disease the previously recognized forms "chronic" or "aggressive" have been changed now into a single category, known as "periodontitis". In 1991 the severity of the disease was classed into either mild, moderate or severe while in 2017 it was built on a staging system depending on the severity and complexity of management of disease onto (stage I to IV), and grading system (grade A, B and C). This new 2017 classification allows clinicians to treat individuals with periodontitis and to accomplish clinical results that were formerly not feasible.

It also carries direct documentation of individuals who are prone to necessitate greater effort to avoid or regulate their chronic disease long term. As 2017 classification was established on staging, Periodontitis staging is of benefit to clinicians regarding all appropriate components that aid to improve individual patient management and thus exemplifies a critical phase towards personalized care or precision medicine (Caton *et al.*, 2018).

Definition and classification of diabetes mellitus

Diabetes mellitus (DM) can be defined as a clinical syndrome with accompanying hyper-glycaemia due to a relative or absolute insulin deficiency. Four type of diabetes have be recognized namely type 1 diabetes mellitus also known as insulin dependent diabetes, type 2 diabetes mellitus also known insulin independent diabetes, gestational diabetes and other explicit genetic types (American diabetes association, 2018). The first and second type are the predominant forms, and they account for the majority of cases. With Type 2 DM being far more common (IDF data).

Epidemiology of diabetes mellitus

Diabetes mellitus (DM) is a global health emergency. More individuals are diagnosed annually with this disease resulting in lethal life-threatening difficulties (Diabetes UK 2015). In 2015, the incidence of diabetes was revealed to be 415 million worldwide aged 20-70years), by the year 2040 it is expected to rise to 642 million International Diabetes Federation 2015). In 2015 it was discovered that the occurrence of this disease to be higher than ten million among the following 7 countries, Brazil, China, India, Indonesia, Mexico, Russian Federation and the USA. Since 1996 the frequency of diabetics has expanded in Great Britain from 1.4 million to 3.5 million whereas 549,000 individuals were unaware of their diabetic status (Diabetes UK 2015).

The international diabetes federation reported in 2013 that, there are 2.5 million diabetics in South Africa and approximately 2.6 million have prompt indications of diabetes. And a death rate of 68,000 diabetic individuals has been reported (Federation 2013). Conferring to researches performed in 2013, diabetes appeared to be the fourth major source of death in KwaZulu-Natal (South Africa) the countries, second largest population (Africa 2014, Budlender *et al.,* 2011). In 2014 the incidence in Kwazulu-Natal was to be around 12.3% but in the past 3 years before 2014 it was less by 56% (Sahadew *et al.,* 2016). The burden of undiagnosed diabetes in Sub-Saharan Africa remains significant with estimates of up to 50%.

Type 1 diabetes mellitus (T1DM)

One of the most frequent metabolic syndromes in childhood is Type 1 diabetes mellitus,

occurring in 1 in 400-600 of children and adolescents (Centers for Disease Control and Silverstein *et al.*, 2005, Prevention 2011,). This precise type of diabetes arises due to the auto-immune interruption of the beta cells of the islet of Langerhans resulting in restriction of insulin secretion. This results in chronic high blood sugar. The management of the disease consist of the administration of exogenous insulin to prevent ketosis (Twetman *et al.,* 2005, Orbak, *et al.,* 2008, Malicka *et al.,* 2014). In addition, it causes interruption in the metabolism of carbohydrates, lipids and amino acids because of the reduction in insulin (Talat *et al.,* 2016).

Epidemiology of T1DM

In 2011, 1 million Americans have been diagnosed with diabetes (T1DM), frequently seen in adolescents even though it can also develop in adults (at times a slower onset) (Centers for Disease Control and Prevention 2011).

Worldwide, the prevalence of insulin dependent diabetes mellitus escalating significantly (Bialo *et al.,* 2015). Every year the incidence is growing by a rate of 3% worldwide (Skyler *et al.,* 2017). This disease is less frequently seen in Venezuela and China although it is rising every year in Finland (2.4%), Germany (2.6%), and Norway (3.3%) annually (Podar *et al.,* 2001, Thunander *et al.,* 2008, Patterson *et al.,* 2009, Ehehalt *et al.,* 2012).

A study considering young adults in in South Africa, Ethiopia and Tanzania showed that insulin dependent diabetes mellitus usually emerges later on in African children in comparison to European (Pinchevsky et al., 2015).

A study conducted in Soweto (South Africa) (2005) consisting of 88 T1DM of participants evaluating revealed that hypoglycemia, renal failure, retinopathy, infection and high blood pressure were the major cause of mortality in T1DM due to hypoglycemia (Gill et al., 2005).

Pathophysiology of T1DM

Metabolic instabilities related to insulin dependent diabetes mellitus that result as a consequence of auto immunological progression, the sequelae of this progression is destruction of beta-cell, with the interruption in the function of alpha cells that results in excretion of extreme quantities of glucagon that increases blood glucose. Insulin deficit leads to enhanced lipolysis and free fatty acids in the extracellular fluid. This influences

the development of ketone bodies to be used as a fuel source, resulting in diabetes ketoacidosis (DKA). Raju and Raju (2010) stated that diminished blood sugar, lipid and protein metabolism were the major metabolic instabilities in insulin dependent diabetic individuals (Omer 2016).

Complications of T1DM

Complications related to T1DM may be acute or chronic. Acute complications comprise of diabetic ketoacidosis (DKA) and hypoglycemia. DKA is primarily presented in insulin dependent diabetes mellitus although it happens as elimination or leaving out prescribed amount of their medication. DKA can be precipitated by stress such as surgery or infection. Hypoglycemia commonly occurs due to a difference between the insulin dose taken and the consumption of carbohydrates. Those with unregulated or insufficiently regulated diabetes for numerous years frequently develop chronic complications.

Retinopathy, which results in blindness, nephropathy, which can ultimately lead to lack of proper function of kidney and peripheral neuropathy with forfeiture of sensation in the limbs complicated at times by ulceration of lower limb are considered micro vascular complications. erebrovascular disease (stroke), acute coronary syndrome and peripheral vascular disease are considered macro vascular complications (American Diabetes Association 2003).

Patients diagnosed with diabetes have impaired immunity and are prone to numerous infections including periodontitis. Several skin lesions seems to be seen in young adults diagnosed with insulin dependent diabetes mellitus, those includes acquired ichthyosis, rubeosis faciei, diabetic hand, fungal infections and necrobiosis lipoidica (Pavlović, *et al.*, 2007). A study was performed to discover the association between periodontal health and metabolic control in individuals diagnosed with insulin dependent diabetes mellitus. The study consisted of 28 known diabetics and 20 healthy individuals, the results of which confirmed a positive correlation between periodontal disease and insulin dependent diabetes mellitus, and that periodontal inflammation was more frequent in those with a longer disease duration, inadequate glucose control and diabetic obstacles (Ajita *et al.*, 2013).

Diagnosis of Diabetes Mellitus

To confirm the diagnosis of DM one the following investigations should can be used Fasting plasma glucose, Random Plasma Glucose 2-h Postprandial Plasma Glucose test following a stand or glycated hemoglobin (HbA1c), with certain cut-off values for each test (American Diabetes Association 2018).

HbA1c (glycated hemoglobin) is a blood investigation that is utilized to detect the average control blood sugar over period of 90 days it is considered as an essential test in the recall visits of diabetic patients. An HbA1c value of 6.5% or above is considered as diagnostic of diabetes. HbA1c is also used in the follow-up of diabetic patients, with target ranges for individualized control in order to improve outcome (Denisse *et al.,* 2017). HbA1c will thus be utilized as an indicator of diabetic control within this study.

Epidemiological studies of periodontal disease in patients with diabetes mellitus

Periodontal disease is considered as the sixth frequent impediment of diabetes mellitus (DM) (insufficiently controlled) (Lahariya et al., 2017).

Studies have revealed that individuals diagnosed with DM are more suspected to periodontal diseases in comparison to non-diabetic individuals, engendering diabetes as a main etiological factor for periodontitis, in other words inadequately controlled diabetics have a higher chance to experience chronic periodontitis with time (Taylor et al., 1996 Mealey et al., 2006).

With regard to HbA1c that is confirmed that individuals with periodontitis have an escalated level in their HbA1c in comparison with individuals with an intact periodontium (Graziani *et al.,* 2018). The national health and nutrition examination survey (NHANES) (1970) in the USA found that the higher periodontal index increases the susceptibility of developing DM in the forthcoming years (Demmer *et al.,* 2008).

The interrelation between insulin dependent diabetes mellitus and periodontal disease added children and young adults to its awareness as insulin dependent diabetes mellitus does not result in inflammation of the attachment apparatus nevertheless it alters the reaction of the periodontal tissue to focal factors (Newman *et al.,* 2006). Larger quantities of plaque, gingival inflammation, bleeding sites, loss of attachments and higher level of destruction is

seen among both insulin dependent diabetes mellitus and non-insulin dependent diabetes mellitus patients in comparison to well controlled patients (Lalla *et al.,* 2006).

Studies conducted in periodontitis and diabetes

Individuals diagnosed with DM have lower tolerance to infections, with an increase in the quantities of salivary glucose and with developed capability in forming dental plaque (Mattson *et al.,* 2001, Zhou *et al.,* 2015).

Concurring to numerous studies, diabetic individuals with periodontal disease have impaired immune response related principally to T cells and have alterations in the function of polymorphonuclear cell such as granulocytes leading to infections in the mouth. Disproportionate tissue inflammation enhances the incidence n severity of PD in these individuals resultant in early damage of the periodontium. Studies revealed that, the management of the periodontal infection helps in diabetic regulation by reducing blood sugar level, high blood glucose and the essential of insulin (Denisse *et al.,* 2017).

Babu, *et al.,* (2018) studied 80 type 1 diabetic adolescents, and evaluated their gingival status and dental carries using world health organization criteria and gingival index as periodontal parameter and found that all 80 diabetics had a healthy gingival status. Periodontal parameters (sites with bleeding on probing, and plaque index, clinical attachment level and probing depth), were evaluated to assess the periodontal status of 168 adolescents with type 1 diabetes. Results showed that the prevalence of gingivitis and periodontitis were 20.8% and 5.9%, respectively and concluded that poor metabolic control had a great impact on the periodontal status (Xavier, *et al.,* 2009). In another study in Serbia, the periodontal health in young adults with insulin dependent type 1 diabetes mellitus was assessed. In 187 diabetics and 178 non-diabetics, periodontal disease was evaluated clinically. When comparing both groups, those with diabetes had more gingivitis than non-diabetic due to their glycemic control, severity and duration of disease (Dakovic 2008). Giuca, *et al.,* (2015) studied the impact of metabolic control on the periodontal status of adolescents with insulin dependent diabetes mellitus. 120 participants were grouped into 3 clusters in equal number according to their metabolic control into: poor controlled, well controlled and non-diabetics. Periodontal parameters such as, bleeding on

probing, clinical attachment level, gingival index, probing depth and plaque index, were assessed. Results showed that 27 (67.5%) out of the 40 poorly controlled patients had gingivitis, 1 (2.5%) had periodontitis and 12 (30%) had a healthy periodontal status. from the 40 well controlled adolescents 26 (65%) had gingivitis, 0 (0%) periodontitis and 14 (35%) healthy. 13 (32.5%) from the 40 non-diabetics had gingivitis, 0 (0%) periodontitis and 27 (67.5%) were healthy.

Jindal, *et al.,* (2015) studied 50 individuals with T1DM distributed into 3 clusters corresponding to glycemic control and the study revealed that those with good glycemic control had less indications of periodontal disease. Periodontal parameters (bleeding index, pocket depth, plaque index, and attachment level) were compared in equal numbers of diabetics and non-diabetics. The association between the advancement of complications, periodontal parameters, level of metabolic control and the period of the disease were assessed. The study revealed that T1DM individuals had higher periodontal disease burden, particularly individuals that have inadequate control and complications (Silvestre, *et al.,* 2009). In an additional study performed in Kuwait assessing the relationship between diabetics, salivary glucose and caries in young adults. Akpata, *et al.,* 2012 performed a study among DM patients (53) and non-diabetic controls (53), the results of which confirmed that caries experience was suggestively superior in children with insulin dependent diabetes mellitus than in non-diabetic controls. The periodontal status of 107 diabetic individuals and 40 controls was examined by Poplowska Kita 2014, in this study the Oral Hygiene Index (OHI), Community Periodontal Index (CPI) and tooth number were used. Those participants with a CPI values of 0–2 were classified as "non-periodontitis" and those with a CPI value of 3–4 were classified periodontitis, respectively.15% of medically healthy participants and 57.9% diabetics participants were found to have periodontitis, 40% of which had controlled diabetes and 59.5% had uncontrolled diabetes.

A study consisting of 95 diagnosed (T1DM) children, who were clinically examined for their periodontal health (Plaque index, gingival index, bleeding on probing and clinical ttachment loss) on the 6 Ramfjord index teeth in comparison to 61 healthy control subjects 4-14 years old) (Al-Khabbaz *et al.* 2013). The study of Al-Khabbaz *et al.* (2013) regarding children with healthy periodontal status diagnosed with insulin-dependent DM showed that 44% of

the 42 participants never went to the dentist before and 65% of the 62 participants had poor dental visits follow up.

In the diabetic cluster, periodontitis was substantially related to longer periods of diabetes. In another study in Iran, the relationship amongst dental and periodontal status and type 1 diabetes mellitus was assessed. They studied 50 adolescents with diabetes and compared them to 50 other healthy individuals and were grouped into 2 age groups. They compared

The periodontal and dental status of both groups, results showed an increase in the periodontal parameter (pocket depth) with age, with no considerable statistical variation between the 2 groups. They concluded that adolescents diagnosed with type 1 diabetes were more disposed to developmental of periodontal disease that became worse with aging (Sadeghi, *et al.*, 2017).

The studies reported in this mini-thesis literature review were based on a cross-sectional studies comparing the periodontal status of type1 diabetic adolescents with their diabetic control. Cross-sectional studies are convened to explore the connections between risk factors and the outcome of interest. It describes a population or a subgroup within a population with respect to an outcome and a set of risk factors.

Periodontal status has an impact on the metabolic control of diabetic patients. The treatment of periodontal disease is of benefits to diabetic patients because an effective treatment of periodontal disease improves blood glucose levels. This study will therefore compare the periodontal status of diabetic patients in a resource-limited setting to their glycemic status. In addition, we aim to demonstrate that glycemic status has an impact on periodontal status of patients diagnosed with Type 1 DM.

CHAPTER THREE

Aim and objectives

Research question:

Is there an association between metabolic control in type 1 adolescent diabetic patients and their periodontal status?

Null Hypothesis:

There is no relationship between the metabolic status and the presence of periodontal diseases in adolescent patients diagnosed with T1DM diabetes mellitus.

Aim (General objective):

To compare the periodontal status of Type 1 diabetic adolescents with their diabetic metabolic control.

Specific Objectives:

1. To determine the absence/presence of gingivitis.
2. To determine the absence/ presence of periodontitis.
3. To determine the metabolic (glucose) control by recording the HBA1c level.
4. To compare the metabolic control with the periodontal status of the patients.

CHAPTER FOUR

Methodology

Design of the study

This was a cross-sectional analytic study.

Rationale

The purpose of Cross-sectional studies is to find the prevalence of the outcome of interest, for the population or subgroups within the population at a given time point. It is relatively inexpensive and takes up little time to conduct and many outcomes and risk factors can be assessed. Therefore, it might be useful for public health planning, it might also give a better understanding of the disease etiology. The data obtained can be used for research purposes (Sadeghi *et al.,* 2017).

Sample size

58 participants were included in this study. This number of participants was selected obtain 90% power, which has an importance measure of 0.05 (Vidya *et al.,* 2018).

Convenient sampling that included 58 participants was conducted and all Type 1 DM adolescent patients from the Tygerberg Endocrinology clinic were examined and assessed at the Faculty of Dentistry (Tygerberg Oral Health Center).

Inclusion criteria

1. Type 1 Diabetic adolescents aged 12-18 years old diagnosed with diabetes mellitus for at least 12 months.

Exclusion criteria

1. Patients who do not match the determined age group.
2. Adolescent patients who have Type 2 Diabetes.
3. Patients on other drug therapy that affect the periodontium, (Phenytoin, Nifedipine and Cyclosporine).
4. Patients with other known chronic and or immunosuppressive systemic diseases or patients who have Human Immunodeficiency Virus (HIV)/Acquired Immunodeficiency Syndrome (AIDS) and also individuals with known

hematological deficiencies were also excluded.

Data collection

Diabetes-related parameters:

After completing a thorough medical history and consulting the physician for specific information, an evaluation of the diabetic control of the patients was done in accordance with the glycated hemoglobin value (HbA1c).

Oral examination:

A periodontal examination was carried out by a single examiner using Williams probe to determine the parameters. Clinical parameters were adjusted with an orthopantomogram to assess the alveolar bone levels (Kumar *et al.*, 2018).

Data analysis:

Adolescent diabetic patients were considered well controlled with an HbA1c less than 7% (120-150 mg/dL), and uncontrolled HbA1c greater than 7% (150-180 mg/dL) (Denisse *et al.*, 2017). The diabetic control was compared to the periodontal parameters measured and to the periodontal status of the participants.

The data was analyzed with STATA (Stata Corp. 2017. *Stata Statistical Software: Release 15*. College Station, TX: Stata Corp LLC). Descriptive statistics represented by histograms, bar charts, number (percentage), mean (standard deviation) or median depending on the distribution of data. For categorical data, a Chi square test was utilized. For continuous data, a t-test was utilized to demonstrate contrast between two groups else an ANOVA was utilized for group sizes greater than 2. In the event that the data does not fulfil the assumptions for the above stated tests, a non-parametric equivalent will be used. Level of significance was established at p-value of less than 0.05.

Ethical consideration

The study was revised and ratified by the Biomedical Research and Ethics Committee of the University of the Western Cape (BMREC) and Health Research ethic committee (HREC):BM18/9/14.Similarly, an ethical clearance was attained by the Stellenbosch University Health Research Ethics Committee (SU HREC) which recognized the UWC BMREC. Thereafter, permission was granted from the Dean of Dentistry, to access the patients.

The objectives and aspects of the study was made knowledgeable to all patients that joined the study. Informed written consent was given by each contributor (assent) along with a consent from the parent/s and or guardians of the participating adolescent in their first language as shown below in the appendix. Participation was voluntary and at any stage or time the patient was able to withdraw their participation without any prejudice.

Once the clinical examination was concluded, each patient was provided with the information of the clinical findings and referred to appropriate clinicians which was prearranged for both periodontal and other dental treatment needs.

Anonymity of data was ensured by giving each patient a code and only the researcher had access to the patient's identity which was captured on a separate sheet and stored on a computer with a secret code, only known to the researching clinician.

CHAPTER FIVE

Results

A complete number of 58 young adults diagnosed with type 1 DM were involved in the study. There were more females than males (37 versus 21) and their mean age was 15.4 years.

Figure 1 demonstrated that the majority of the sample studied had gingivitis 44 (75.86%), 6 (10.34%) had periodontitis and 8 (13.79%) had periodontal health. The majority of those with periodontitis (n=5) showed a localized distribution while only 1 patient was represented with generalized distribution. The majority of the patients that have been diagnosed by stage I periodontitis as previously mentioned 6 (10.34%) and due to their diabetic diagnosis, were categorized as either grade B (n=3, %) or C (n=3, %). (Figure 2) (Figure 3).

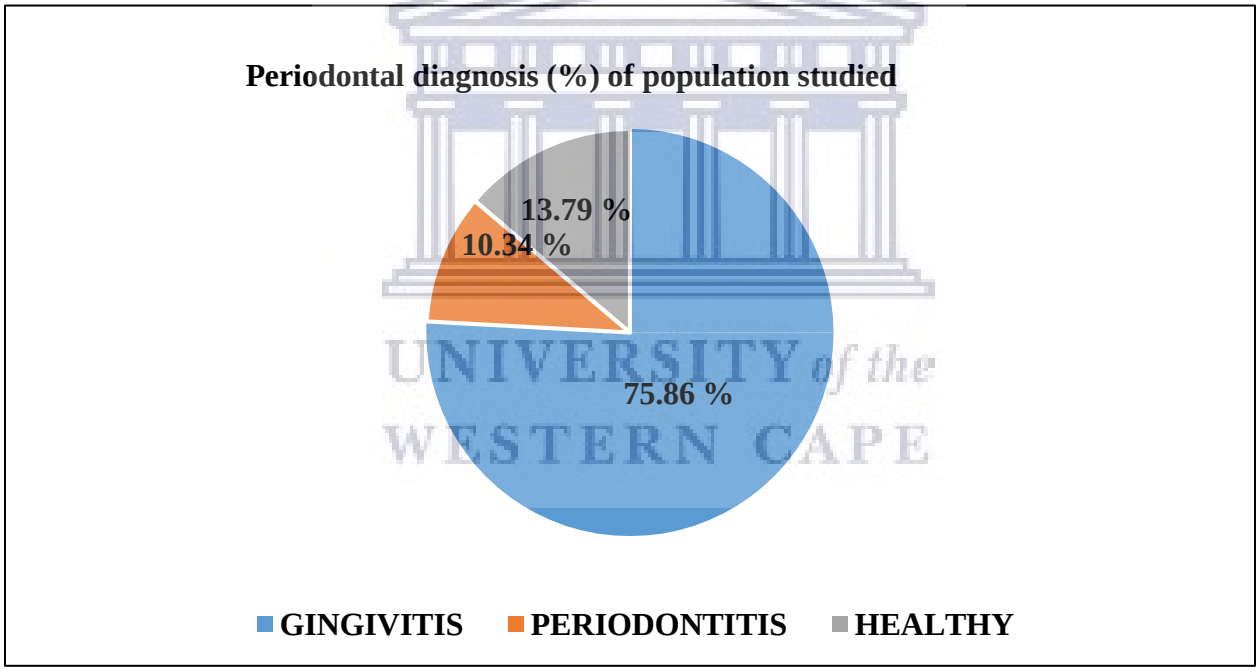

Figure 1. The percentage of periodontal diagnosis of the sample studied.

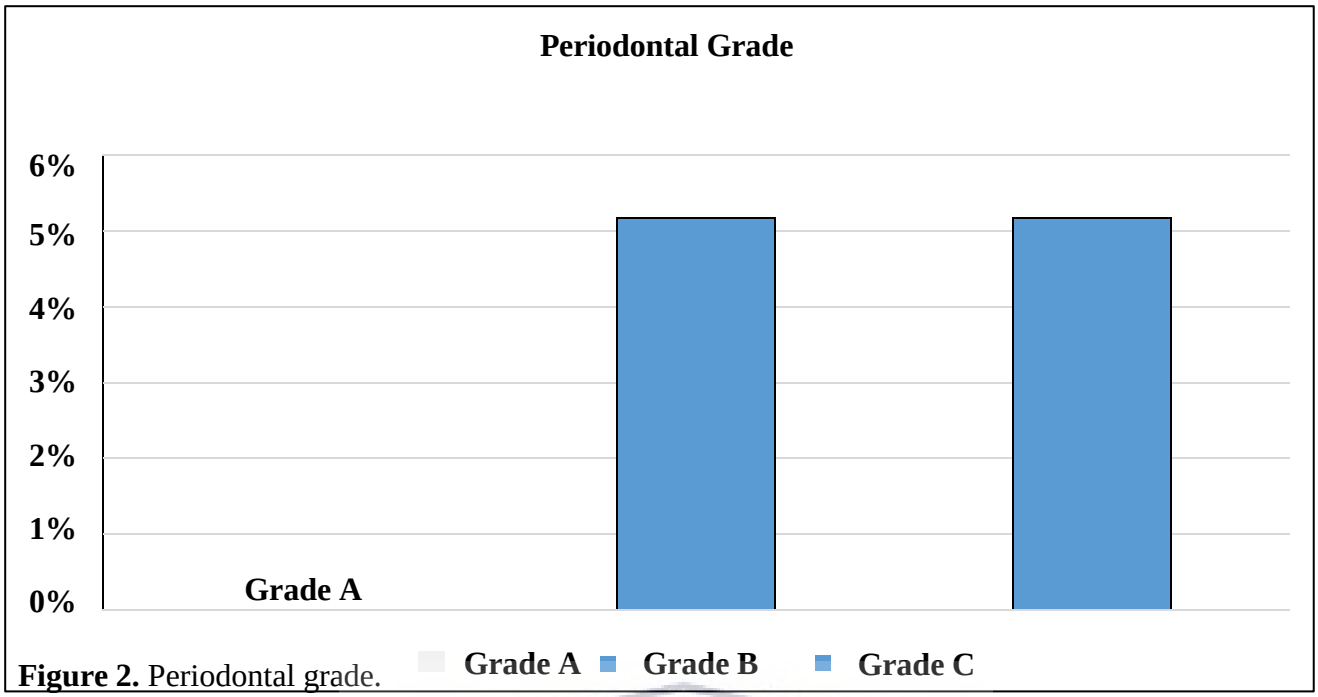

Figure 2. Periodontal grade.

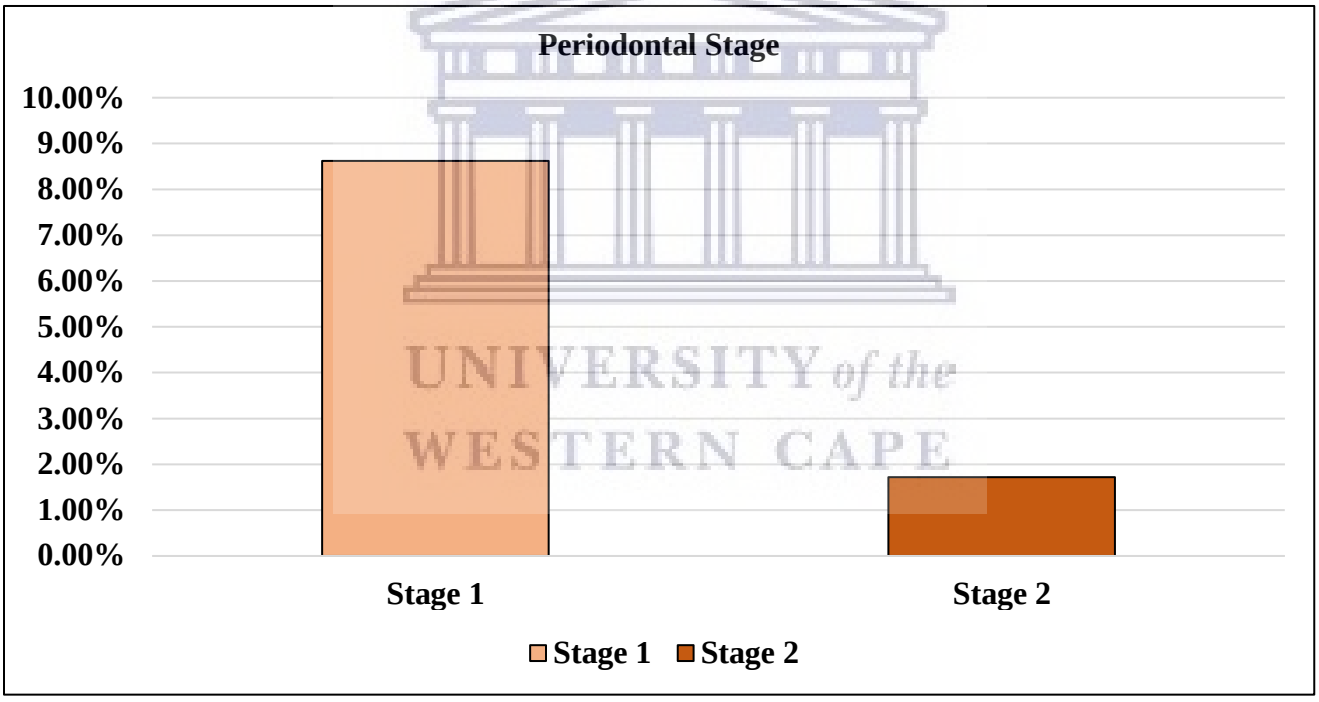

Figure 3. Periodontal stage.

The majority, 51 (87.92%) had uncontrolled diabetes with HbA1c ≥ 7% (Figure 4). The mean Hemoglobin of the population studied was found to be 9.24 (2.05) %. For controlled diabetics, the mean age was 14.85 (2.73) and uncontrolled diabetes 14.76 (2.00) years, and there was no remarkable differentiation in the mean age between among the groups, p =0.911.

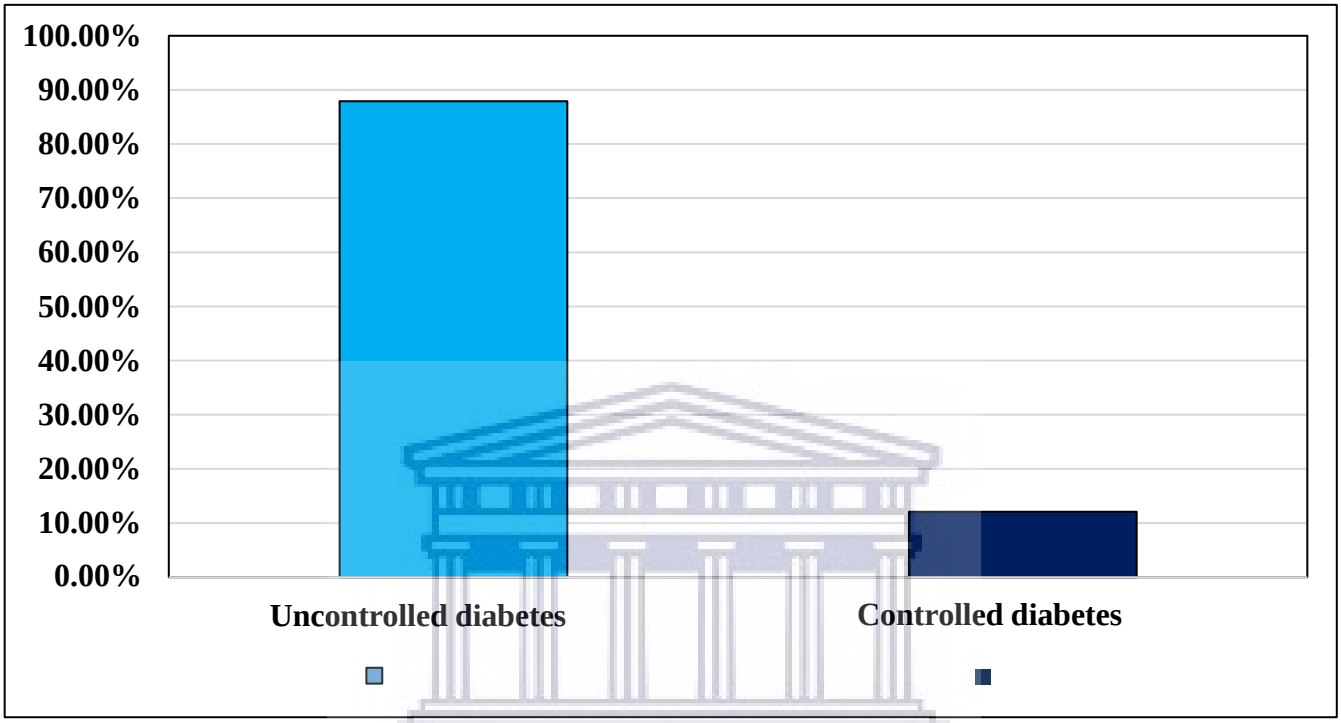

Figure 4. The metabolic control of the sample studied.

No statistically noteworthy association was detected between sex and diabetic control, p=0.231. Of the subjects, 17 males (33.33%) and 34 females (66.67%) had uncontrolled diabetes whereas 4 males (57.14%) and 3 females (42.86%) had controlled diabetes (Table 1).

Table 1. The sex distribution and overall diabetes control.

		N (%)	Prevalence (Uncontrolled Diabetes)	p-value
Gender	**Male**	21 (36.21)	17 (33.33)	0.231
	Female	37 (63.79)	34 (66.67)	

Table 2 showed the comparison of the metabolic control with periodontal status where, the majority of patients who had uncontrolled diabetes had gingivitis. 7 out of 8 patients with periodontal health had uncontrolled diabetes. 4 out of 6 patients with periodontitis had uncontrolled diabetes, but this was not statistically significant. The mean bleeding index was 46.37%.

Table 2. Comparison of the metabolic control with the periodontal status.

Statistical readings	**Periodontal diagnosis**		
	Health	Gingivitis	Periodontitis
Number of controlled diabetes (%)	8(13.79%)	44(75.86%)	6(10.34%)
Number of uncontrolled diabetes (%)	7 (87.5%)	7 (13.73%)	4 (7.84%)
P-value	0.232	0.232	0.232

CHAPTER SIX

Discussion

An association between periodontal disease and type 1 diabetes was not discovered in this sample. In this study, the majority of the sample was diagnosed with gingivitis (75.86%) while a few had periodontitis (10.34%). Periodontal disease in type1 diabetics have been studied by various authors and similar results were revealed Silvestre, et al., (2009), Al-Khabbaz et al., (2013), Poplowska-Kita 2014, Jindal, et al., (2015), and Sadeghi, et al., (2017). Similarly, Xavier, et al., (2009) assessed the periodontal status of 168 young adults T1DM in Brazil, and established that the frequency of gingivitis, periodontitis was 20.8% and 5.9%, respectively while in this study the gingivitis, periodontitis were 75.86% and 10.34%, respectively. Giuca, et al., (2015) studied 120 patients from Italy, and assessed their periodontal status and the majority of them had gingivitis. In contrast, Babu, et al., (2018) studied 80 type 1 diabetic adolescents from South India, and evaluated their gingival status, and found that all 80 diabetics had a healthy gingival status while in the present study only 13.79% of the sample had a healthy gingival status differed from the previously mentioned study. This is linked to the socioeconomic status of the population studied as the socioeconomic status influences the outcome of disease control. Medication, dental care and information on disease control are usually more readily available and easier to be accessed by population with high socioeconomic status.

The majority of the gingivitis patients in this study had an HbA1c more than 7% (uncontrolled). This can be explained by the fact that infection causes an increase in blood glucose and hence HbA1c. High blood glucose is a good media for flourishing infection. Adequate treatment of the infection and good blood glucose control will improve the HbA1c. Most of the patients had uncontrolled diabetes and this can be explained by the fact that the sample came from a tertiary center where only complicated cases are dealt with. Poplowska-Kita (2014) confirmed a connection between the number of areas affected by fasting blood glucose (FBG) and periodontitis. However, Denisse et al., (2017) explored the effect of treatment of periodontal infection on HbA1c and found good response, however in this study the effect of treatment on HbA1c was not studied.

Out of those with gingivitis and periodontitis in this sample, most of them had uncontrolled diabetes mellitus. Similarly, Jindal, et al. 2015 examined 50 participants with insulin dependent diabetes mellitus separated into 3 clusters depending on glycemic control, they found that those with good glycemic control had lower prevalence of periodontal disease. Silvestre, et al., 2009 found that type 1 diabetics were more prone to periodontal disease, especially diabetics that have inadequate control and complications that arise from this. (Dakovic 2008) evaluated the periodontal health in adolescents with type 1 diabetes and found that those with diabetes had increased gingivitis than non-diabetic as a consequence of their glycemic control, severity and duration of disease. Poplowska-Kita (2014) drew attention to the indicator that patients with insufficient metabolic control displayed significantly higher alveolar bone loss and loss of attachment.

CHAPTER SEVEN

Conclusion and recommendations

Conclusion

In the studied sample of Type 1 DM adolescents, gingivitis was most prevalent, followed by periodontal health and periodontitis was least prevalent. The majority of the sample had poorly controlled T1DM.

Limitation of this study

The restrictions includes the following; the study design (cross-sectional) meaning that the researcher was only able to seen and include participants that presented at that single point of time and thus he could have been missing patients that didn't present at that specific time, the second restriction in this study was that number of participants (small number).

The patients were sought from a tertiary hospital and hence the sample were mostly poorly controlled T1DM.

Recommendations

Ongoing efforts to improve the access to diabetes education initiatives which should include lifestyle recommendations, blood glucose self-monitoring and managing of insulin treatment. In addition, always consider periodontitis as an associated infection in patients with diabetes. Widespread patient education on dental hygiene as well regular visits to the dentist are suggested.

Further research is needed regarding the relation of periodontal disease with metabolic control in insulin dependent diabetes mellitus in adolescents, with larger sample size and include socioeconomic status with comparison of more periodontal parameters such as gingival index, plaque index, calculus index, tooth decay and amount of missing teeth.

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UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,
7505 South Africa

T: +27 21 9373184, F: +27 21 9312287
E: zsmith@uwc.ac.za
www.uwc.ac.za

Appendix 1 **Appendix**

Parental Informed Consent Document

PROJECT TITLE

The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

INTRODUCTION

Your child has been invited to join a research study to look at the relationship between the health of their gums and their diabetic control.

Please take whatever time you need to discuss the study with your family and friends, or anyone else you wish to. The decision to let you child join, or not to join, is up to you.

In this research study, we are evaluating if there is a relationship between gum health and control of diabetes mellitus. We know that the inflammation of gum disease can make it difficult to control the disease of diabetes. This information is not known for younger diabetic patients in South Africa.

WHAT IS INVOLVED IN THE STUDY?

Your child will be asked to have an examination of their teeth and gums. The dentist will look and take measurements around each tooth. If Radiographs are needed, they will also be taken. The findings will be recorded, and your child will be referred for any treatment needs diagnosed at this visit. The examination will take about 1 hour, and the treatment will differ according to the need of each child and will be conducted at a second and subsequent visit/s.

The investigators may stop the study or take your child out of the study at any time they judge it is in your child's best interest. They may also remove your child from the study for various other reasons. They can do this without your consent. Your child can stop participating at any time. If your child stops, he/she will not lose any benefits of access to dental treatment needs.

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RISKS

This study involves the following risks.

If your child already suffers from bleeding gums, the examination may cause minor bleeding at the margin of the gum line.

The examination may be sensitive in children who have severe forms of gum disease. There may also be other risks that we cannot predict.

BENEFITS TO TAKING PART IN THE STUDY?

It is reasonable to expect the following benefits from this research:

The diagnosis of unknown gum and other dental diseases and facilitated access to treatment is the greatest benefit of participating in this study. However, we can't guarantee that your child will personally experience benefits from participating in this study if they are uncooperative in the dental chair or do not keep set appointments.

Others may benefit in the future from the information we find in this study as we will discover the role that gum disease plays in the ability to affect diabetic control.

CONFIDENTIALITY

Your child's name will not be used when data from this study are published. Every effort will be made to keep clinical records, research records, and other personal information confidential.

We will take the following steps to keep information confidential, and to protect it from unauthorized disclosure, tampering, or damage: A code will be placed on the survey and other collected data and through the use of an identification key the researcher will be able to link your survey to your identity. All information collected will be locked in cabinets and password protected computer. No one has the right to access this information except the researcher. He is only access for the purpose of research.

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INCENTIVES

There are no incentives to participate in this study.

YOUR RIGHTS AS A RESEARCH PARTICIPANT?

Participation in this study is voluntary. Your child has the right not to participate at all or to leave the study at any time. Deciding not to participate or choosing to leave the study will not result in any penalty or loss of benefits to which your child is entitled, and it will not harm his/her relationship with the dentist or the diabetic clinic.

If your child decides to leave the study, the procedure is: to contact the researcher, whose name and number is at the bottom of this page.

CONTACTS FOR QUESTIONS OR PROBLEMS?

Call Dr. Abdelrahman at 0219373167/8/0827951014 or email 3820857@myuwc.ac.za if you have questions about the study, any problems, if your child experiences any unexpected physical or psychological discomforts, any injuries, or think that something unusual or unexpected is happening.

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email: nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21 9312287 E: zsmith@uwc.ac.za www.uwc.ac.za

Parental Consent Document

Title of Research Project:

The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

Permission for a Child to Participate in Research

As parent or legal guardian, I authorize _____

(Child's name) to become a participant in the research study described in the information form.

Child's Date of Birth _

Parent or Legal Guardian's Signature Date

Upon signing, the parent or legal guardian will receive a copy of this form, and the original will be held in the subject's research record.

BMREC	The Dean	The Head of Department
UWC	Prof. Neil Myburgh	Dr A Jeftha
Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	Faculty of Dentistry Tygerberg Campus 0219373001 Email: nmyburgh@uwc.ac.za	Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the
WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21 9312287

E: zsmith@uwc.ac.za
www.uwc.ac.za

PARTICIPANT INFORMATION LEAFLET AND ASSENT FORM

TITLE OF THE RESEARCH PROJECT:

The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

RESEARCHERS NAME (S): Dr Mohamed Abdelrahman

ADDRESS: Department of Oral Medicine and Periodontology, D Floor, UWC Faculty of Dentistry, Tygerberg Hospital. Francie Van Zyl Road. Cape Town.

CONTACT NUMBER: 0219373167/8

0827951014

What is RESEARCH?

Research is something we do to find new knowledge about the way things (and people) work. We use research projects or studies to help us find out more about disease or illness. Research also helps us to find better ways of helping or treating children who are sick.

What is this research project all about?

This project is about the relationship between gum disease and how your diabetes is controlled.

Why have I been invited to take part in this research project?

You have been invited because you have been diagnosed and treated for diabetes.

Who is doing the research?

I am Mohamed Abdelrahman, a student at the University of the Western Cape and I am studying further about how to diagnose and treat adults and children with gum diseases.

What will happen to me in this study?

I will look, feel and measure around your gums in your mouth. If I find any diseases, I will give you follow up appointments for treatment either in my clinic or other clinics within our dental hospital.

Can anything bad happen to me?

If you suffer from bleeding gums, your gums may bleed during the examination. You may have some pain if your gum disease is very bad. If you have any problems with what I do you are allowed to tell your parents and they can tell me if you experience any problems while I examine your mouth.

Can anything good happen to me?

Yes. You may have some diseases in your mouth that you are not aware of. We will then find them and send you for the proper treatment and also teach you how to prevent them from happening again.

Will anyone know I am in the study?

Only I and your parents will know that you are in the study. I will not put your name on any documents that I have to share with others, who help me to analyze what I find in your mouth. I will keep your name separate and save it on a computer that only I will have access to because it will require a password, that only I will know.

Who can I talk to about the study?

Me, Dr Abdelrahman, or My supervisor, Dr Jeftha 0219373158

Or BMREC UWC

Private Bag x17 Bellville

7535

Tel: + 27 21 959 4111

Email: research-ethics@uwc.ac.za

What if I do not want to do this?

You are free to refuse to be included in this study. You can stop your participation in this study at any time. Even if you choose to stop, we will still see that you get your dental treatment if you need it.

Do you understand this research study and are you willing to take part in it?

YES **NO**

Has the researcher answered all your questions?
YES **NO**
Do you understand that you can pull out of the study at any time?
YES **NO**

Signature of Child **Date**

BMREC UWC Private Bag x17	The Dean Prof. Neil Myburgh Faculty of Dentistry	The Head of Department Dr A Jeftha Faculty of Dentistry
Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

Note: parental informed consent form and participant consent form in English.

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21 9312287
E: zsmith@uwc.ac.za
www.uwc.ac.za

Appendix 2

Inligting en toestemmingsvorm vir Ouers

NAVORSINGS TITEL:

Die verhouding tussen periodontale siektes en die beheer van Tipe 1 Diabetes (Suikersiekte) in jong volwassenes.

INLEIDING:

Ons vra hiermee toestemming dat jou kind mag deelnaam aan n navorsingsprojek wat die verhouding van jou kind se vermoe om sy/haar Tipe 1 Diabetes to beheer te vergelyk met die gesondheid van jou kind se tandvleis.

Neem asseblief tyd Om hierdie inligling en jou besluit hieroor, met jou familie en of vriende te deel. Die besluit om jou kind by hierdie navorsing te betrek, of nie te betrek nie, is in jou beheer en jou keuse.

Vorige navorsing het betoon dat siektes van die tandvleis wel pasiente met Diabetes laat sukkel om hul diabetes te beheer. Hierdie navorsing is nog nie in jong volwassenes in Suid Afrika beskikbaar nie, en dus wil ons graag hierdie navorsing doen.

VERDERE INLIGTING OOR DIE NAVORSINGSPROJEK.

Ons gaan jou kind se tandvleis en tande ondersoek.

Die tandaarts gaan ook die spasie tussen die tand en die tandvleis meet, omdat dit die manier is waarop ons die gesondheid van die tandvleis kan bepaal.

As daar xstrale van die tande nodig is, sal ons dit ook laat doen. Alle informasie wat ons van jou kind se mond kry sal op dieselfde manier gedokumenteer word as waneer hy/sy vir n normale tandheelkundige ondersoek gaan.

Indien jou kind tandheelkundige behandeling nodig sou he, sal ons reelings tref om jou kind na n tandaarts

by ons Fakulteit te stuur vir hierdie behandeling.

Die ondersoek sal omtrent 1uur duur en enige behandeling sal op opvolg afsprake gedoen word. Die navorsers mag hierdie navorsing of jou kind se betrokkenheid by die projek stop as dit vir die beswil van jou kind is. Dit kan sonder jou bemagtiging gebeur.

Jou kind mag ook sy/haar betrokkenheid by hierdie projek enige tyd stop. Dit sal sy/haar verdere tandheelkundige behandeling nie negatief beinvloed nie.

RISIKOS

Hierdie navirsing behels die volgende risikos:

Jou kind se tandvleis mag gedurende die ondersoek bloei, dit sal definitief bloei as hy/sy al klaar by die Hui bloeding ervaar waneer hulle hul tande borsel. Hierdie bloeding sal nie jou kind of enige manier siek maak of benadeel nie.

Kinders met erge inflammatoriese tandvleissiektes mag miskien ongerief gedurende die ondersoek ondervind. Ander risikos kan op hierdie tydperk nie voorspel word nie.

VOORDELE VAN BETROKKENHEID BY HIERDIE PROJEK

Die volgende voordele kan verwag word:

Onbekende mond of tand siektes sal gediagnoseer en behandel word. Hierdie voordeel is alleenlik moontlik as jou kind wel samewerking tot behandeling sal gee en as hy of sy hul afprake nakom.

Ander jong volwassenes wat ook Tipe 1 diabetes het sal voordeel uit die resultate van hierdie navorsing he.

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VERTROULIKHEID

Alle besonderhede van jou kind, hul name en kliniese inligting sal vertroulik hanteer word. Jou kind se naam en identiteit sal nie saam met sy kliniese inligting bekend gemaak word waneer ons die uitslae van hierdie navorsing publiseer nie.

A kode sal aan die document van jou kind se kliniese inligting wees en nie hul name nie. Die navorser sal alleenlik die lys van name en kodes op sy persoonlike rekenaar bere. Dit sal deur mate van a wagwoord beskerm word.

AANSPORINGS

Daar is geen aansporings vir die deelname by hierdie navorsing nie.

DIE REGTE VAN DEELNAMERS

Betrokkenheid by hierdie navorsing is vrywillig. Jou kind het op enige tydperk die reg om te onttrek. Jou kind sal sy/haar toegang tot tandheelkundige en diabetes behandeling nie negatief afekteer nie, al besluit hul om te ontrek.

Indien jou kind besluit om te onttrek, moet u asseblief die navorser in kennis stel. Sy besonderhede is hieronder aangeheg.

KONTAKTE

Dr. Abdelrahman 0219373167/8/0827951014 or epos 3820857@myuwc.ac.za

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email: nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21 9312287 E: zsmith@uwc.ac.za

Inligting en toestemmingsvorm vir Ouers

NAVORSINGS TITEL:

Die verhouding tussen periodontale siektes en die beheer van Tipe 1 Diabetes (Suikersiekte) in jong volwassenes.

Toestemming vir my kind Om by die narvorsing deel te neem:

As ouer of wettige voog, gee ek hiermee toestemming dat

_____(volle naam van kind) by hierdie navorsingsprojek betrokke mag wees.

Geboortedatum van kind ____

Ouer of Wettige Voog se handtekening_____Datum _____

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg, 7505
South Africa

T: +27 21 9373184, F: +27 21
9312287 E:
zsmith@uwc.ac.za

DEELNAME INLIGTINGS EN INSTEMMINGSVORM

NAVORSINGS TITEL:

Die verhouding tussen periodontale siektes en die beheer van Tipe 1 Diabetes in jong volwassenes.

NAME VAN NAVORSER: Dr. Mohamed Abdelrahman

ADRES: Dept of Oral Medicine and Periodontology, D Floor, UWC Faculty of Dentistry, Tygerberg Hospital. Francie Van Zyl Road. Cape Town.

KONTAK NOMMERS: 0219373167/8

0827951014

WAT IS NAVORSING?

Navorsing is die manier waarop navorsers nuwe inligting bekom oor die manier hoe dinge en mense te werke gaan.

Navorsingsprojekte word gebruik Om ons te help om nuwe inligting oor siektes te bekom. Navorsing help ook om uit te vind wat die beset maniere van siekte behandeling in kinders en volwassesnes is.

INLIGTING OOR HIERDIE NAVORSINGSPROJEK?

Hierdie projek sal die verhouding tussen die beher van Tipe 1 Diabetes en tandvleis siektes ondersoek.

HOEKOM IS EK GEVRA OM HIERMEE BETROKKE TE RAAK?

Jy is betrokke omdat jy met Tipe 1 Diabetes gediagnoseer en behandel word.

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WIE DOEN HIERDIE NAVORSING?

My naam is Mohamed Abdelrahman, ek is n student by die Universiteit van Wes Kaapland en ek studier hoe Om volwassesnes en kinders met tandvleis siektes te diagnoseer en behandel.

WAT GAAN MET MY IN HIERDIE PROJEK GEBEUR?

Ek gaan in jou mond kyk, voel en by jou tandvleis meet. As ek wel mondsiektes diagnoseer, sale k jou vir verdure behandeling verwys.

KAN ENIG IETS SLEG MET MY GEBEUR?

As jou tandvleis bloei wanneer jy jou tande by die Hui borsel, mag di took bloei wanneer ek jou tandvleis meet. As jy tandvleis siektes wel het, mag die ondersoek n bietjie seer wees. Jy moet jou oers vertel as enigiets jou pla terwyl jy jou ondersoek ondergaan.

WAT SAL VIR MY HIERMEE GOED WEES?

Dit sal goed wees om uit te vind of jy enige siektes in jou mond het, en daarvoor behandeling te kan kry.

WIE SAL WEET DAT EK BY HIERDIE NAVORSING BETROKKE IS?

Jou ouers en die navorser sal allennlik van jou betrokkenheid bewus wees. Jou naam sal nie met ander gedeel word nie, behalwe as jy wel verdure behandel nodig het.

MET KAN EK HIERDIE NAVORSING BESPREEK?

Dr Abdelrahman, 0219373167/8 BMREC

UWC

Private Bag x17

Bellville

UNIVERSITY of the WESTERN CAPE 7535

Tel: + 27 21 959 4111

Email: research-ethics@uwc.ac.za

MAG EK NEE SE?

Jy mag beslis nie hiermee betrokke word as jy ni wil nie. Jy mag ook jou betrokkenheid op enige tyd onttrek. Jy sal nog steed's behandeling vir enige mondsiketes en jou diabetes ontvang.

Verstaan jy hierdie inligting, en is jy beried om deel te neem? Ja Nee

Het ons al jou vrae beantwoord? Ja Nee

Verstaan jy dat jy enige tyd mag onttrek?? Ja Nee

Handtekening van Kind Datum

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

Note: Parental informed consent form and participant consent form in Afrikaans.

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21
9312287 E:
zsmith@uwc.ac.za

Appendix 3

Iphepha-mvume lomzali

ISIHLOKO

The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

INTSHAYELELO

Umntwana wakho uyamenywa abeyinxalenye yophando, phado elo elijonga ubudlelwane kwimpilo yentsini kunye nendlela abalawula ngayo idiabethi.

Thatha ixesha elaneleyo ukuthetha nezihlobo, izizalwane malunga nesisifundo. Isigqibo sokuvumela umntwana wakho abeyinxalenye yesisifundo sixhomekeke kuwe.

Koluphando sijonga ubudlelwane kwimpilo yentsini kunye nendlela abalawula ngayo idiabethi, siyayazi ukuvutha kwesigulo sentsini kwenza kubenzima ukulawula idiabethi. Olu lwazi alaziwa kubantu abaselula eMzantsi Afrika.

YINTONI ECHAPHAZELEKAYO KWESISIFUNDO?

Umntwana wakho uzakucelwa ukuhlolwa intsini kunye namazinyo. Ugqirha wamazinyo uzakuhlola ,athathe umlinganiselo kwizinyo ngalinye. Ukubangaba imifanekiso iyadingeka nayo izakuthathwa. Iziphumo zizokushicilelwa umntwana wakho uzokusiwa okanye anikwe amachiza awadingayo koluphando. Olu hlolo lizokuthatha iyure enye kwaye amachiza azakohluka kumntwana ngamnye kuxhomekeke kwizidingo zomntwana ngamnye.

Umphandi angasimisa esisifundo okanye akhuphe umntwana wakho koluphando nangaliphi na ixesha ebona kufanelekile. Bangamkhupha umntwana koluphando nangezinye izizathu bangayenza lonto ngaphandle kwemvume yakho.

Umntwana wakho angarhoxa ukuthatha inxaxheba nangaliphi na ixesha. Nokuba umntwana

wakho utrhe wayeka ukuba kwesisifundo akuzukuphulukana nokufumana amachiza kugqirha wamazinyo.

MNGCIPHEKO

Oluphando luquka imingcipheko elandelayo:

Ukubangaba umntwana wakho uhlutshwa zintsini ezophayo, uhlolo lungenza kubekhona ukopha kwentsini. Uhlolo lungangabalungeli abantu abophelwa zintsini ngokumandla. Ingakhona neminye imingcipheko esingenoyibona okwangoku.

INZUZO YOKUBAYINXALENYE YESISIFUNDO

Ukuhlolwa kwentsini nezinye izifo ezinxulumana namazinyo, ukufumana amachiza zinzuzo azakuthi azifumane umntwana. kodwa asinokutsho ngokuphelelyo ukuba umntwana wakho akazukwazi ukusebenzisana nogqirha wamazinyo. Abanye bangafumana inzuzo ngolwazi oluzakufumaneka koluphando, lwazi olo luzakujonga indima edlalwa sisifo sentsini ekulawuleni idiabethi.

IMFIHLO

Igama lomntwana lizokuba yimfihlo, xakukhutshwa iziphumo zophando. Sizakuqinisekisa lonke ulwazi olushicilelweyo luyimfihlo. Sizakuthatha lamanyathelo alangelayo ukuqinisekisa uphando luyimfihlo nokulikhusela kumonakalo. Ikhowudi izakusetyenziswa njenngendlela umphandi azakuthi abonengayo inkcukhaca zomntwana ngamnye. Lonke uphando oluqokelelweyo lizakugcina kwikhabhathi kunye nekhompuyitha ezitixiweyo. Akukho bani onelungelo lokufumana olu lwazi ngaphandle komphandi.

INKUTHAZO

Akukho nkuthazo youkuyinxalenye yoluphando.

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ILUNGELO LAKHO NJENGOMNTU OTHATHA INXAXHEBA

Ukubayinxalenye yoluphando ayisosinyanzeliso. Umntwana wakho unelungelo lokurhoxa koluphando kwaye angayeka nangaliphi na ixesha. Ukuyeka kwakhe akuzukwenza umntwana aphulukane nenzuzo azifaneleyo, akuzubanafuthe libi kubudlelwane bakhe nogqirha wamazinyo.

Ukubangaba umntwana uthatha isigqibo sokurhoxa koluphando anagatsalela umxeba umphandi igama lakhe nenombolo yomxeba yakhe iyafumaneka ezantsi kweliphepha.

INOMBOLO MALUNGA NEMIBUZO OKANYE INGCACISO

Fowunela Dr.Abdelrahman kulenombolo 3820857@myuwc.ac.za. Ukubangaba unemibuzo ngoluphando, okanye ingxaki athi umntwana abenayo nezinye izinto ofuna ukuziqonda.

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21
9312287 E:
zsmith@uwc.ac.za

ISIHLOKO **Iphepha-mvume lomzali**

The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

Imvume yokuba umntwana abeyinxalenye yoluphando

Mna mzali, ndinikka imvume (igama lomntwana) athathe inxaxheba kwesisifundo sichaziweyo ngasentla.

Usuku lokuzalwa komntwana _____

Isistyikityo somzali _____ umhla _

Ngokusayina , umzali uzakufumana ikopi yeliphepha , eyokwenene ikopi igcinwe.

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

UNIVERSITY of the WESTERN CAPE

Private Bag X1, Tygerberg,

7505 South Africa

T: +27 21 9373184, F: +27 21 9312287 E: zsmith@uwc.ac.za

Ulwazi nesiqinisekiso sokubayinxalenye yoluphando

Isihloko sesifundo: The association of periodontal disease with metabolic control in Type 1 diabetic adolescents.

Amagama abaphandi: Dr Mohamed Abdelrahman

Idilesi: Dept of Oral Medicine and Periodontology, D Floor, UWC Faculty of Dentistry, Tygerberg Hospital. Francie Van Zyl Road. Cape Town.

Inombolo yomxeba: 0219373167/8

0827951014

Yintoni uphando?

Uphando yinto esiyenzayo ukufumana ulwazi olutsha ngendlela izinto ezisebenza ngayo. Sisebenzisa uphando ukufumana ulwazi oluphangaleleyo ngezifo okanye izigulo. Uphando lusinceda sifumane indlela ezingcono zokunceda nokunyanga abantwana abagulayo.

Lungantoni oluphando?

Koluphando sijonga ubudlelwane kwimpilo yentsini kunye nendlela abalawula ngayo idiabethi.

Kutheni ndiyinxalenye yoluphando?

Uyinxalenye yoluphando kuba ungumntu ophila nediabethi.

Ngubani owenza oluphando?

Ndingu Mohamed Abdelrahman, umfundi kwiDyunivesithi yseNtshona-koloni.Ndenza izifundo eziqwalasela indlela zokubona nokunceda abantu abadala kunye nabantwana abanesifo sentsini.

Kuzokwenzeka ntoni kum koluphando?

Ndizokujonga, ndive kwaye ndithathe umlinganiselo kwintsini ezisemlonyeni wakho. Ukubangaba ndifumana isigulo okanye isifo ndizakunika amachiza okanye ndikuthumele kugqirha wamazinyo.

Ingakhona into embi enokwenzeka kum?

Ukuba usokoliswa zintsini ezophayo, intsini zakho zingopha xa ndikuhlola. Ungava ubuhlungu ukuba isifo sakho sentsini sigqithisile. Nayiphi ingxaki engathi ivele ngexesha ndikuhlola, ungaxelela umzali wakho anditsalele umxeba.

Ingakhona into entle enokwenzeka?

Ewe. Kungakho isifo emlonyeni wakho ongasaziyo singasifumana sikuthumele kumachiza afanekileyo singaphindi sibekho kwakho.

Ukhona umntu ongayzi ndiyinxalenye yoluphando?

Mna kunye nabazali bakho kuphela. Andiyikulisebenzisa igama lakho kumaphepha endiwanika abantu endisebenza nabo. Ndizokuligcina liyimfihlo igama lakho.

Ndingathetha nabani ngoluphando? mna, Dr Abdelrahman,

okanye 0219373167

okanye BMREC UWC

UNIVERSITY of the WESTERN CAPE
Private Bag x17, Bellville 7535 Tel: + 27 21 959 4111

Email: research-ethics@uwc.ac.za

Ukubangaba andifuni kubayinxalenye yoluphando?

Unelungelo lokungathathi nxaxheba koluphando. Ungarhoxa nangaliphi ixesha ufuna. Nokuba uthe warhoxa sizokuqhubeka sikunika amachiza owadingayo.

Uyaluqonda oluphando, kwaye uyafuna ukubayinxalenye?

EWE HAYI

Ingaba umphandi uphendule yonke imibuzo onayo?

EWE HAYI

Uyayiqonda ungarhoxa koluphando nangaliphi ixesha?

EWE HAYI

Isityikityo somntwana Umhla

BMREC UWC Private Bag x17 Bellville 7535 Tel: + 27 21 959 4111 Email: research-ethics@uwc.ac.za	The Dean Prof. Neil Myburgh Faculty of Dentistry Tygerberg Campus 0219373001 Email:nmyburgh@uwc.ac.za	The Head of Department Dr A Jeftha Faculty of Dentistry Tygerberg Campus 021 9373158/ 3186 Email: ajeftha@uwc.ac.za

Note: Parental informed consent form and participant consent form in Xhosa.

Appendix 4

	A	B	C	D	E	F	G	H	I	J
1	Q_1	Q_2	Q_3	Q_4	Q_5	Q_6	Q_7	Q_8	Q_9	Q_10
2	PATIENT CODE	AGE	GENDER	HBA1C	TYPE OF DIABETES	ANTIBIOTIC	PHENYTOIN	OTHER	DISEASE	SMOKE
3	1									
4	2									
5	3									
6	4									
7	5									
8	6									
9	7									
10	8									
11	9									
12	10									
13	11									
14	12									
15	13									
16	14									
17	15									
18	16									
19	17									
20	18									
21	19									
22	20									
23	21									
24	22									
25	23									
	24									
	25									

	K	L	M	N	O	P	Q	R	S	T
1	Q_11	Q_12	Q_13	Q_14	Q_16	Q_17	Q_18	Q_19	Q_20	Q_21
2	No of cigare	BLEEDING	PROBING POCKET DEPTHS	RADIOGRAPHIC BONE LOS	GINGIVITIS	PERIODONTITIS	PERIODONTITIS STAGE	PERIODONTITIS GRADE	DISTRIBUTION	PERIODONTAL DIAGNOSIS

Sample of data collection sheet.

Note: The spread sheet was used during this study contains patient's information, periodontal parameters and diabetic parameters.