PATTERN OF PERIODONTAL DISEASES IN PERSONS LIVING WITH HIV INFECTION/AIDS AT THE KENYATTA NATIONAL HOSPITAL OUTPATIENT CENTER- NAIROBI, KENYA

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DECLARATION

I declare that this thesis is my original work and has not been presented for the award of a degree in any other university.

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DEDICATION

This work is dedicated to my wife Bhoke Chacha and my daughter Taraji Ghati .

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I thank the Lord God, for the love, good health and strength that He has given me in all aspects of life through-out the course of this programme. I also, sincerely express my utmost gratitude to my dear wife, Bhoke Chacha, for always being there for me. I am especially grateful to my supervisors, Prof. Evelyn Wagaiyu, Dr. Regina Mutave for their constant guidance, wisdom, patience, time, support and encouragement. My mentors and teachers, who molded me and established a foundation through which all of this has been possible.

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DEFINITION OF TERMS

CD4 cell counts: The number of CD4 positive T- lymphocytes per unit volume of blood. Determination requires the use of fluorescence activated cytometer.

Comprehensive Care Centre (CCC): Non-profit making, out-patient medical facility dedicated to providing health care to HIV infected patients by providing free HAART, psycho-social and nutritional counselling and palliative care.

Conventional periodontitis: Refers to forms of periodontal diseases that present with undistinguishable clinical findings from those that occur in non-HIV-infected populations.

Cross-sectional studies: Studies which the presence or absence of disease or other health related variables are determined in each member of the study population or in a representative sample at one particular time. This contrasts with longitudinal studies, which are followed over a period of time.

Highly active antiretroviral therapy (HAART): Drug regimens for patients with HIV infection, which aggressively suppress HIV replication. The regimen usually involves administration of three or more different drugs.

HIV seropositive: The development of neutralizing antibodies in individuals who have been exposed to the Human Immunodeficiency Virus (HIV).

Pattern of periodontal diseases: Refers to prevalence and severity of various forms of periodontal diseases as measured by various clinical parameters in HIV/ AIDS patients.

Pre-HAART era: Is the period before the implementation of highly active antiretroviral therapy (HAART) in HIV management; 1996.

Periodontal diseases: Pathological processes involving the periodontium including the gingiva, alveolar bone, cementum and periodontal ligament.

LIST OF ACRONYMS

AAP	American Academy of Periodontology
AIDS	Acquired Immune Deficiency Syndrome
ANUG	Acute Necrotizing Ulcerative Gingivitis
CCC	Comprehensive Care Centre
CD4	Cluster of Differentiation 4
CAL	Clinical Attachment Loss
CDC	Centers for Disease Control
CEJ	Cemento-Enamel Junction
CP	Chronic Periodontitis
DNA	Deoxyribonucleic Acid
EBV	Epstein-Barr virus
EC	European Commission
FI	Fusion Inhibitor
GP	Glyco-Protein
HAART	Highly Active Anti-Retroviral Therapy
HHV	Human Herpes Virus
HIV	Human Immunodeficiency Virus
LDL	Low Density Lipoprotein
LGE	Linear Gingival Erythema
MMPs	Matrix Metallo-Proteinases
NNRTs	Non-Nucleoside Reverse Transcriptase Inhibitors
NRTIs	Nucleoside Reverse Transcriptase Inhibitors
NUG	Necrotizing Ulcerative Gingivitis
NUP	Necrotizing Ulcerative Periodontitis

NUS	Necrotizing Ulcerative Stomatitis
PIs	Protease Inhibitors
PGE_2	Prostaglandin E_2
PLWHA	People Living With HIV infection/Aids
PMNs	Polymorphonuclear leukocytes
PD	Periodontal Diseases
PPD	Periodontal Probing Depth
RNA	Ribonucleic Acid
SPSS	Statistical Packages for Social Sciences
VCT	Voluntary Counselling and Testing
WHO	World Health Organization

ABSTRACT

Background: The Human Immunodeficiency Virus (HIV) infection is considered a modifier of periodontal diseases with numerous studies reporting increased prevalence of various forms of periodontal diseases in Persons Living with HIV infection/AIDS (PLWHA). Other studies have reported exacerbation of the pre-existing periodontal diseases with HIV induced immunosuppression. However, the pattern of periodontal diseases in PLWHA is not well documented especially in the Highly Active Antiretroviral Therapy (HAART) era.

Objective: To describe the pattern of periodontal diseases among PLWHA.

Study design: A hospital based descriptive cross-sectional study.

Study area: The Comprehensive Care Centre (CCC) at Kenyatta National Hospital (KNH) – Nairobi, Kenya.

Study population: Adult PLWHA whose status had been confirmed through serology test.

Patients and methods: 284 participants whose HIV infection status had been confirmed through serological tests were screened with 186 meeting the inclusion criteria. Data on socio-demographic variables, past medical and dental histories were collected from the patients through a structured interview. The CD4 cell counts and HAART treatment profile were obtained from the patients' register. Oral hygiene status and periodontal status were recorded in a modified World Health Organization (WHO) clinical examination form 1997 (Appendix II). The modified Quigley and Hein index (Turesky et al. 1970) and the gingival index (Loe and Silness 1963) were used to assess the oral

hygiene status and gingival inflammation respectively. Periodontitis was assessed using periodontal probing depth (PPD), recession and clinical attachment loss (CAL).

Results: 186 participants were recruited into the study among whom 73 (39.2%) were males and 113 (60.8%) were females. The age ranged between 20 to 65 years with a mean age of 40.25 ± 9.73 years. The prevalence of periodontal diseases associated with HIV infection was 6.5% with 3.2% having Linear Gingival Erythema (LGE), while 1.1 % and 2.2% having Necrotizing Ulcerative Gingivitis (NUG) and Necrotizing Ulcerative Periodontitis (NUP) respectively. All the participants with NUG and NUP had <200 cells/mm^3 CD4 cell counts while LGE was reported in participants with either <200cells/mm^3 or <500 cells/mm^3 CD4 counts. The prevalence of conventional periodontitis in the current study was 62.9% with 4.3% having severe periodontitis, 25.3% with moderate periodontitis and 33.3% with mild periodontitis. The presence of periodontitis was significantly associated with low CD4 cell counts, however, the severity of periodontitis showed no significant association with CD4 cell counts.

The CD4 cell counts of the study population ranged between 10 - 1309 cells/mm^3 with a mean of 435$\pm$ 250.99 cells/ mm^3. Thirty four (18.3%) had CD4 cell counts of <200 cells/mm^3 while the rest had >200 cells/mm^3. Only thirty two (17.2%) participants were not on HAART. The mean plaque score was 1.78$\pm$0.49 while the mean gingival inflammation score was 1.34$\pm$0.38. An association between CD4 cell counts and the mean gingival inflammation score was observed, with participants who had $\geq$ 200- 499 cells/mm^3 CD4 cell counts recording a higher mean gingival inflammation score than the rest.

Conclusion: The prevalence of periodontal diseases was high among PLWHA with conventional periodontitis being the most prevalent. The presence of periodontal diseases was associated with low CD4 cell counts (<200 cells/mm^3), however, the severity of periodontitis showed no association with CD4 cell counts.

Recommendation: Periodontal care should be incorporated as a component of comprehensive care for PLWHA.

CHAPTER ONE

INTRODUCTION AND LITERATURE REVIEW

1.1 Introduction

Periodontal diseases are chronic infectious disorders characterised by inflammation and destruction of the attachment apparatus of the teeth. The attachment apparatus include the gingiva, the periodontal ligament, the root cementum and the alveolar bone[1]. Periodontal diseases can be broadly grouped into gingivitis and periodontitis. Gingivitis is the inflammation of the gingiva in which the junctional epithelium remains attached to the tooth root at its normal anatomical level. Periodontitis on the other hand, occurs when there is destruction of the periodontal ligament and apical migration of the junctional epithelium[2]. Periodontitis is always preceded by gingivitis, however, gingivitis does not always progress to periodontitis[1].

Periodontal diseases affect human populations worldwide at high prevalence rates, second to dental caries[3]. Gingivitis is the most prevalent form of periodontal diseases and affects 50% to 90% of the adult population worldwide[4, 5]. On the other hand, studies have reported varied prevalence rates of periodontitis with as low as 5% and as high as 90% prevalence rates being reported[4, 6, 7]. This variation has been attributed to lack of standardised case definition and use of partial or full mouth examination[8].

The primary etiological factor of periodontal diseases is microbial plaque[9]. However, the development and progression of periodontal diseases is determined by an interplay of host immune defence mechanisms against the microbial plaque governed by genetics, environmental and behavioural factors[9, 10, 11, 12]. The Human Immunodeficiency Virus (HIV) induces immunological changes characterised by the

depletion of CD4 cell counts in the human body[13]. The CD4+ T-helper cells main function in the human body is to regulate and amplify the host immune response activity[13, 14]. Therefore, it is hypothesized that, CD4 cell depletion would lead to compromised host defence in the dento-gingival region, thus increasing the susceptibility to periodontal diseases [15, 16, 17].

Studies have described various forms of periodontal diseases in persons living with HIV infection/AIDS (PLWHA)[16, 18, 20, 21, 22]. Various studies done in the pre- Highly Active Anti-Retroviral Therapy (HAART) era reported higher prevalence of linear gingival erythema (LGE), necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP) among PLWHA[16, 18, 19]. However, recent studies have consistently reported lower prevalence rates of these diseases among the PLWHA than what was reported previously[21, 22, 23]. A study done by Kroidl et al. (2005), reported a prevalence of 9% for LGE and 3.6% for NUP and NUG[22]. The decreased prevalence of these lesions has been suggested to be due to the increasing use of HAART[16]. HIV infection has been reported to modify the course of the pre-existing periodontitis[24]. However, studies have reported contrasting findings on the prevalence and severity of conventional periodontitis[15, 25, 26, 27, 28]. Furthermore, it is still controversial as to whether the stage of the HIV disease, as expressed by the level of CD4 cell counts affects the severity of conventional periodontal diseases[25, 26, 27, 28, 29]. Several studies have reported an association between CD4 cell counts and the prevalence of periodontal diseases [25, 26] while others have reported no association[27, 28, 29].

With the overall number of PLWHA increasing due to HAART use and new infections occurring each year [30], the change in the pattern of periodontal diseases with higher prevalence and severe forms is likely to be witnessed. This is likely to increase the suffering of PLWHA because of real or perceived stigma, confidentiality concerns or economic and social factors that will limit their utilization of dental facilities. Therefore, this study was set to determine the pattern of periodontal diseases and the association between these diseases and CD4 cell counts in PLWHA in the era of HAART in Kenya. The data will hopefully aid in shaping policy regarding oral health care provision for PLWHA.

1.2 Literature Review

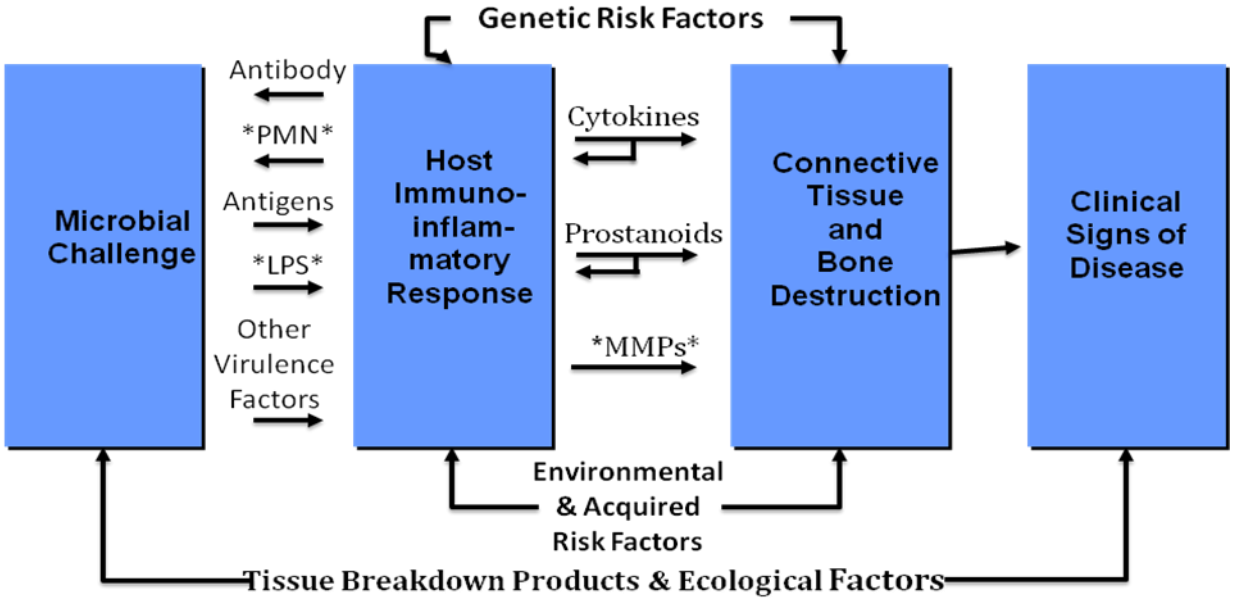
1.2.1 Overview of Periodontal Diseases

Periodontal diseases are chronic inflammatory conditions that are characterised by immuno-inflammatory host response against microorganisms of microbial plaque and their products[9]. This group of diseases is one of the two major dental diseases that affect human populations worldwide at high prevalence rates, second to dental caries[9]. Gingivitis, a form of periodontal disease affects 50% to 90% of the adult population worldwide[4, 5, 8]. On the other hand, the results on the prevalence of periodontal disease have been varied. This has been attributed to the use of different methodologies to measure the level of periodontal diseases, lack of standardised case definition and use of partial or full mouth examination[8]. Studies that defined periodontitis as the identification of at least one site with clinical attachment loss (CAL) of ≥2 mm, reported about 80% prevalence rate among the adults with around 90% of those aged 55 to 64 years having been affected[9]. However, the prevalence of periodontitis in those aged between 55 to 64 drops to around 50% when it is defined by the presence of at least one site with CAL of ≥4mm[9].

1.2.2 Pathogenesis of Periodontal Diseases

The current concept of the pathogenesis of periodontitis recognizes microbial plaque as the primary and direct factor that leads to the development of periodontitis[9, 10, 31]. Bacterial plaque causes periodontal tissue destruction by activating various components of the host defence systems[9, 10, 31]. This concept suggests that the disease is more likely to occur if specific aspects of the host defence mechanisms within the local tissue are altered [31]. This happens when the host factors become hyper-

responsive due to genomic variations and when induced by factors such as smoking, increased bacterial challenge within the tissues as a result of compromised host defence mechanisms at the gingival sulcus[31].



**PMNs*- polymorphonuclears, *LPS*-lipopolysaccharide, *MMPs*- Matrix metalloproteinases*

Fig 1: The pathogenesis of human periodontitis: (Adapted from Page R.C et al. 1997) 31

The development and progression of periodontal diseases, therefore, depends on the interaction between the resident oral microbiota found in the dento-gingival plaque and the host response. These interactions between the bacteria and the host result in a sequence of host immune mechanisms that may be activated even at the expense of damaging the periodontal tissues[3, 9, 10, 24, 31].

1.2.3 An overview of HIV infection and periodontal diseases

The Acquired Immune Deficiency Syndrome (AIDS) is a disease caused by the human immunodeficiency virus (HIV) which is classified as a retrovirus[13]. Primarily, HIV infects CD4+ T lymphocytes, which function as regulators and amplifiers of the immune response[13]. The overall result is a decrease in CD4+ T lymphocytes that eventually leads to a weakened immune system[13]. A weakened immune system results in the emergence of opportunistic infections and development of malignancies[13, 17, 32]. Oral diseases including periodontal diseases are among the numerous opportunistic diseases that have been reported to occur with continued deterioration of the immune system[33, 34].

1.2.3.1 HIV- Host interaction in the periodontal environment

Several studies have reported significant clinical, radiographic and biochemical constituents changes in the dento-gingival environment of HIV seropositive cases when compared to the HIV seronegative ones[17, 25]. On the contrary, other studies have reported no significant changes of these parameters between HIV seropositive and HIV seronegative groups[27, 28]. In general, it is poorly understood how HIV infection affects the periodontium, influences the innate and acquired immunity and how altered local or systemic immune response of these patients contributes to the pathogenesis of periodontal disease[15, 16, 35]. Microbiological analysis studies have shown the presence of high proportions of periodontal pathogens such as *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*[17]. Other studies have reported the presence of certain rarely found microbes in periodontal pockets such as the *Candida species, clostridium difficile, Bacteroides fragilis* and

Pseudomonas aeruginosa in PLWHA patients [36]. The changes in microbial profile in the dento-gingival environment together with the reported changes in inflammatory mediators such as prostaglandin E_2 (PGE_2) and matrix metalloproteinase (MMPs 1, 2, 3, 8, 9) are some of the factors considered to be responsible for higher levels of periodontal diseases in PLWHA as compared to HIV seronegative patients[16, 17, 25, 36, 37]. However, other studies have presented the exact opposite picture, that is, putative pathogens are less prevalent in PLWHA[36].

1.2.4 Periodontal changes associated with HIV infection

HIV infection is linked to various types of periodontal changes that manifest as periodontal diseases. These periodontal lesions include specific forms of gingivitis, necrotizing periodontal diseases as well as possible exacerbation of pre-existing periodontal disease[16, 17, 34, 38]. Some of these periodontal diseases have been identified as conditions strongly associated with HIV infection and include LGE, NUG and NUP. These diseases are among the seven cardinal oral lesions strongly associated with HIV infection[16, 34]. LGE, NUG and NUP are considered as the earliest clinical features of HIV infection and could possibly predict the progression of the HIV disease to AIDS[16, 34, 38] . The presence of these periodontal diseases in patients on HAART may suggest possible treatment failure[16]. LGE, NUG and NUP have characteristic clinical appearance and have been reported to occur at varied prevalence in HIV infected adults [21, 22, 23, 34].

1.2.4.1 Linear Gingival Erythema (LGE)

LGE is a form of gingivitis that is characterised by a fiery red (erythmatous) band of about 2-3 mm along the gingival tissue margin[16, 34]. The degree of erythema is inconsistent with the amount of plaque present[34]. Usually it presents in the anterior segment but may extend to involve the posterior teeth. In some cases it may be accompanied by bleeding and discomfort[39] but there is no evidence of pocketing or attachment loss[34]. The candida species and immune deterioration have been implicated in the etiopathology of LGE in HIV seropositive patients[16, 39, 40]. Studies have reported significant correlation between LGE and CD4 cells counts of below 200cell/mm^3, suggesting that LGE may be a marker of severe immune deterioration[20]. Varied prevalence for LGE both in the pre- HAART era and post- HAART era have been reported in various studies. The pre- HAART era studies report a prevalence range of 9- 50% of LGE among the PLWHA[40]. However, with the advent of HAART, the prevalence of LGE has decreased tremendously with as low as 1% to 17% being reported[22, 23, 41]. The presence of LGE in the era of HAART has been reported to indicate the loss of effectiveness of the therapy[16].

1.2.4.2 Necrotizing Ulcerative Gingivitis (NUG)

NUG is defined as the destruction of one or more interdental papillae that is limited to the gingival tissue with no loss of periodontal attachment[34, 42]. In the acute stage NUG presents with pain, ulceration, necrosis and sloughing of the interdental papillae. Areas of haemorrhage and a characteristic fetor oris are other clinical features[34, 42, 43]. Just like LGE, NUG has been associated with low CD4 cells counts in HIV seropositive patients[16, 44, 45, 46]. On the contrary, NUG has also been associated with

stress, poor diet, poor oral hygiene and tobacco smoking in HIV seronegative cases suggesting that these factors may potentiate its occurrence in HIV seropositive patients[46]. Several studies have reported NUG to have been more prevalent among HIV infected persons than in the general population with the prevalence ranging between 5%- 11%[42, 43]. However, low prevalence has been reported especially in the era of HAART, with one study reporting a prevalence of 3.6%[22].

1.2.4.3 Necrotizing Ulcerative Periodontitis (NUP)

NUP is characterized by periodontal attachment loss as a result of ulceration or necrosis[34, 43]. NUP presents with gingival bleeding, sharp pain, ulcerated gingival papillae, extensive soft tissue necrosis and advanced loss of periodontal attachment[16, 34]. Clinical studies have shown that NUP is more prevalent in HIV seropositive patients with less than 200 cells/mm^3 of CD4 cell counts as compared to patients with higher levels of CD4 cell counts[16, 20, 34, 44, 47]. However, not all HIV infected patients with less than 200cells/mm^3 suffer from NUP, suggesting that other factors in addition to immune-suppression are involved[20]. There have been inconsistent reports on the prevalence of NUP among the HIV infected patients with several studies reporting a prevalence range of between 1% and 18%[16, 21, 22, 24, 42]. The inconsistencies have been, partly due to the grouping of NUG and NUP together, differences in immune competence among the sampled population and mode of sample selection[21, 22].

1.2.4.4 Necrotizing Ulcerative Stomatitis (NUS)

NUS is a lesion that is less commonly associated with HIV infection in the adult population [34]. It is a painful, ulceronecrotic lesion of the oral mucosa that is associated with extensive destruction of the peri-oral tissue[48]. NUS develops from progression of NUP [46]. NUS is predominantly seen in children aged 1–4 years, although late stages can occur in adolescents and adults[49].

1.2.4.5 Conventional Periodontitis (CP)

Conventional periodontal diseases in HIV seropositive patients refer to forms of periodontal diseases that present with undistinguishable clinical findings from those that occur in HIV seronegative populations i.e. chronic periodontitis[50]. Unlike LGE, NUG and NUP, it is considerably uncertain as to whether or not conventional destructive periodontitis is exacerbated in HIV seropositive patients[15, 22, 26, 29]. Recent studies have reported an increase in the prevalence of conventional periodontitis among those who are HIV seropositive[25, 26, 51]. This increased prevalence has been attributed to the immune deterioration among PLWHA[22, 25, 26]. On the contrary, several other studies have found no association between conventional periodontitis and immune deterioration as indicated by the level of CD4 cell counts[15, 27, 28]. The findings on the prevalence of conventional periodontitis among HIV seropositive patients show considerable variations[22, 25, 26, 52,] with some studies reporting high[22, 25, 26] and others low prevalence[15]. These inconsistencies have been attributed to different definitions used to characterize periodontal diseases[25, 52] and methodologies used to collect clinical periodontal measurements (e.g. partial mouth versus full-mouth probing and the number of sites probed per tooth)[17, 25]. Furthermore, Patients'

characteristics, including the immune status (the level of CD4 cell counts), the oral hygiene status and utilization of dental care services are routinely not included [25].

1.2.5 Highly Active Antiretroviral Therapy (HAART) and Periodontal Diseases

The justification of HAART usage in the treatment of HIV infection is to induce reduction in the viral load and hence increases the CD4 cell level[53]. HAART is administered as a combination of at least three different antiretroviral drugs from at least two different classes; nucleotide reverse transcriptase inhibitor (NRTI), protease inhibitors (PI), non-nucleotide reverse transcriptase inhibitors (NNRTI) and entry or fusion inhibitors (FI)[16, 53]. These drugs act at various stages in the lifecycle of the virus thereby reducing the viral load to undetectable levels to allow immune restoration[53]. Because of the immune restoration that is associated with HAART use, a significant reduction in the oral lesions (including periodontal diseases) associated with HIV infections has been reported in several sudies[19, 28]. However, studies have reported higher prevalence of conventional periodontal diseases among PLWHA in the era of HAART[16, 22, 26, 54]. Usage of HAART is associated with an increase of metabolic problems such as increase in serum LDL, cholesterol and insulin resistance[25, 55, 56]. This in turn has been associated with the development and progression of periodontal diseases[25].

CHAPTER TWO

STATEMENT OF THE RESEARCH PROBLEM AND JUSTIFICATION

2.1 Statement of the Research Problem

The HIV infection prevalence in Kenya is estimated to be 7.1% among adults aged 15-64 with an estimated 1.4 million adults living with HIV infection/AIDS[30]. With the increasing use of HAART which modifies the course of HIV infection into a manageable chronic disease and new infections occurring each year, the number of PLWHA is likely to rise[16]. HIV infection has been reported to increase the prevalence and exacerbate the pre-existing periodontal diseases among PLWHA[22, 24, 25, 26]. Therefore, because of the increased numbers of PLWHA, the prevalence and severity of periodontal diseases among these patients is likely to be high. Furthermore, HAART which is increasingly becoming available in Kenya has been shown to promote adverse oral effects some of which enhance the development and progression of periodontal diseases in PLWHA[30, 57].

The importance of dental care for PLWHA must, therefore, be prioritized since periodontal diseases may compromise the general health of the dentition that will eventually have negative effects in the overall general health. Thus the effective management of these patients requires a multidisciplinary approach.

2.2 Study Justification

Most previous studies have reported varied prevalence of periodontal diseases among PLWHA[22, 23, 25, 26, 29]. This variation has been attributed to the inconsistencies in case definition, methodologies applied in data collection of clinical periodontal parameters (full mouth versus partial mouth) and variation in immune competence among the PLWHA. The differences reported on prevalence and the methodologies applied, highlight some of the complexities of comparing periodontal diseases in PLWHA. It is, therefore, difficult to make a meaningful comparison between studies.

Studies have reported increased prevalence and severity of periodontal diseases with the decrease of CD4 cell counts among PLWHA[22, 25, 26]. However, other studies have reported no association of increased prevalence and severity of periodontal diseases with a decrease of CD4 cell counts[27, 28, 29]. Therefore, the question of whether the stage of HIV diseases, as expressed by the CD4 cell counts affects the prevalence and severity of periodontal diseases still remains unresolved.

In view of this, the study is set to provide baseline data on the pattern of periodontal diseases in PLWHA in Kenya using the CDC/AAP consensus case definition[8] and to investigate the association between periodontal diseases and the CD4 cell counts. The data will thus be useful for better understanding of the problem and help formulate prevention and management protocols.

2.3 Objectives

2.3.1 Broad Objective

To determine the pattern of periodontal diseases in PLWHA with various levels of CD4 cell counts.

2.3.2 Specific Objectives

1. To determine the prevalence of periodontal diseases in PLWHA.
2. To determine the level of CD4 cell counts in PLWHA
3. To determine the relationship between CD4 cell counts and periodontal diseases in PLWHA
4. To determine the usage of HAART among PLWHA
5. To determine the relationship between periodontal diseases and HAART use in PLWHA.
6. To determine the oral hygiene status of PLWHA

2.4 Hypothesis

2.4.1 Null Hypothesis.

There is no difference in the pattern of periodontal diseases among PLWHA with various levels of CD4 cell counts.

2.4.2 Alternative Hypothesis.

There is a difference in the pattern of periodontal diseases among PLWHA with various levels of CD4 cell counts.

Table 1: Study Variables

Variable	Measurement
Socio-demographic Variables	
• Age	Number of years.
• Gender	Whether Female or Male.
• Residence	Where they live.
• Occupation	Type of work done.
• Education	Highest level of education.
Independent Variables:	
• CD4+ cell Count	Level of CD4 cells /mm^3.
Dependent variables:	
Periodontal diseases	
• Gingival inflammatory status	Presence and severity of gingival inflammation.
• Clinical attachment loss	Presence or absence of attachment loss from CEJ to the base of the pocket in mm.
• Periodontal probing depth	Pocket depth in mm.
Confounders:	
• Oral hygiene status	Presence and amount of plaque present.
• HAART	ART regimen and duration of use.
• Tobacco smoking	Whether they smoke tobacco or not.

CHAPTER THREE

MATERIAL AND METHODS

3.1 Study Area

The study was carried out at the Comprehensive Care Centre (CCC) at the Kenyatta National Hospital (KNH). KNH is the country's largest referral hospital located in the capital city, Nairobi. Though situated in a metropolitan city, it serves both urban and rural populations from most of the surrounding districts and provinces in Kenya. It also serves a large spectre of the socio- economic profile of patients.

The CCC is a specialized unit within the hospital that was established to provide comprehensive health care to HIV infected patients by providing free HAART, psycho-social and nutritional counselling and palliative care. The KNH- CCC deals with PLWHA drawn from KNH wards, Voluntary Counselling and Testing centres (VCT) and referrals from other facilities throughout the country. PLWHA are given regular appointments for organised follow-up and management. An Average of 50 patients (excluding patients attending prevention of mother to child transmission) are attended to daily.

3.2 Study Design

This was a hospital-based descriptive cross-sectional study.

3.3 Study Population

Adult HIV infected persons whose status was confirmed through serology.

3.4 Sample Design and Procedure

3.4.1 Sample Size Determination

The desired sample size was calculated from the prevalence of periodontal diseases among PLWHA as reported by Kroidl et al 2005, of 76% [22].

Using the Fishers formula for prevalence when the study population is 10000 or above, the desired sample size was thus determined as follows:

$$n = \frac{Z^2 (p)(q)}{\alpha^2}$$

Where;

n = sample size

p = estimated prevalence of periodontal diseases in people living with HIV/ AIDS (0.76)

Z = confidence level at 95% (corresponding to a standard Z value of 1.96)

q = 1- p

α = level of significance (standard value of 0.05)

The sample size was thus calculated as:

$$n = \frac{1.96^2 x 0.76(1-0.76)}{0.05^2}$$

= **280**

However, since the total numbers of adult participants who were registered and had appointment for the eleven days that the data was collected were 550, the sample size used for this study was calculated with the formula used when total study population is less than 10000:

$$nf = \frac{n}{(1+\frac{n}{N})}$$

Where:

nf = the desired sample size when population is less than 10,000

n = the desired sample size when population is more than 10000.

N = the estimate of population size.

$$nf = \frac{280}{(1+\frac{280}{550})}$$

=186

3.4.2 Sample Selection

Systematic random sampling method was used to select the participants because of its simplicity and good spread across the population. Usually, patients are seen on appointment at the KNH CCC, except for emergencies and new cases. On arrival, patients are given numbers which provide the order in which they are attended to. Each day when data were collected, the registered adult patients booked for that day was established and there after an interval was set using the following formula;

$$k = \frac{N}{n}$$

Where

k is the sampling interval (sometimes known as the *skip)*

n is sample.

N is the population size (booked patients for each particular day when data were being collected).

At KNH- CCC, about 50 adult patients (excluding patients for prevention of mother to child transmission) are seen daily. For the eleven days that data were collected, a population of 550 was expected; therefore, the interval for the current study was 550/186. Thus every 3rd patient was included in the study.

3.4.3 Inclusion Criteria

- HIV infected persons confirmed through serology (ELISA test or western blot).
- Persons with CD4 cell counts results which were 3 months old or less.
- Persons who consented to the study.
- Persons who were 18 years of age and above.

3.4.4 Exclusion Criteria

- Persons whose serology (ELISA test) results were unknown.
- Persons with no CD4 cell counts results which were more than 3 months old.
- Persons who did not consent to the study.
- Persons who were below the age of 18 years.
- Persons who were too ill to have periodontal examination performed on them.
- Persons who had a history of cardiovascular diseases, type I or II diabetes mellitus and any uncontrolled systemic illnesses.
- Persons who had a diagnosis or treatment of cancer in the past 5 years.
- Persons with less than 20 teeth.
- Persons who were pregnant
- Persons who had undergone periodontal surgery/treatment (full mouth scaling, root planing and polishing) within the last six months.

3.4.5 Participant Recruitment

Out of the 284 persons living with HIV/ AIDS screened at the KNH CCC, 98 were excluded while 186 qualified for inclusion according to the criteria set. Fig.2 below shows different reasons for exclusion of the participants who fell within the sample.

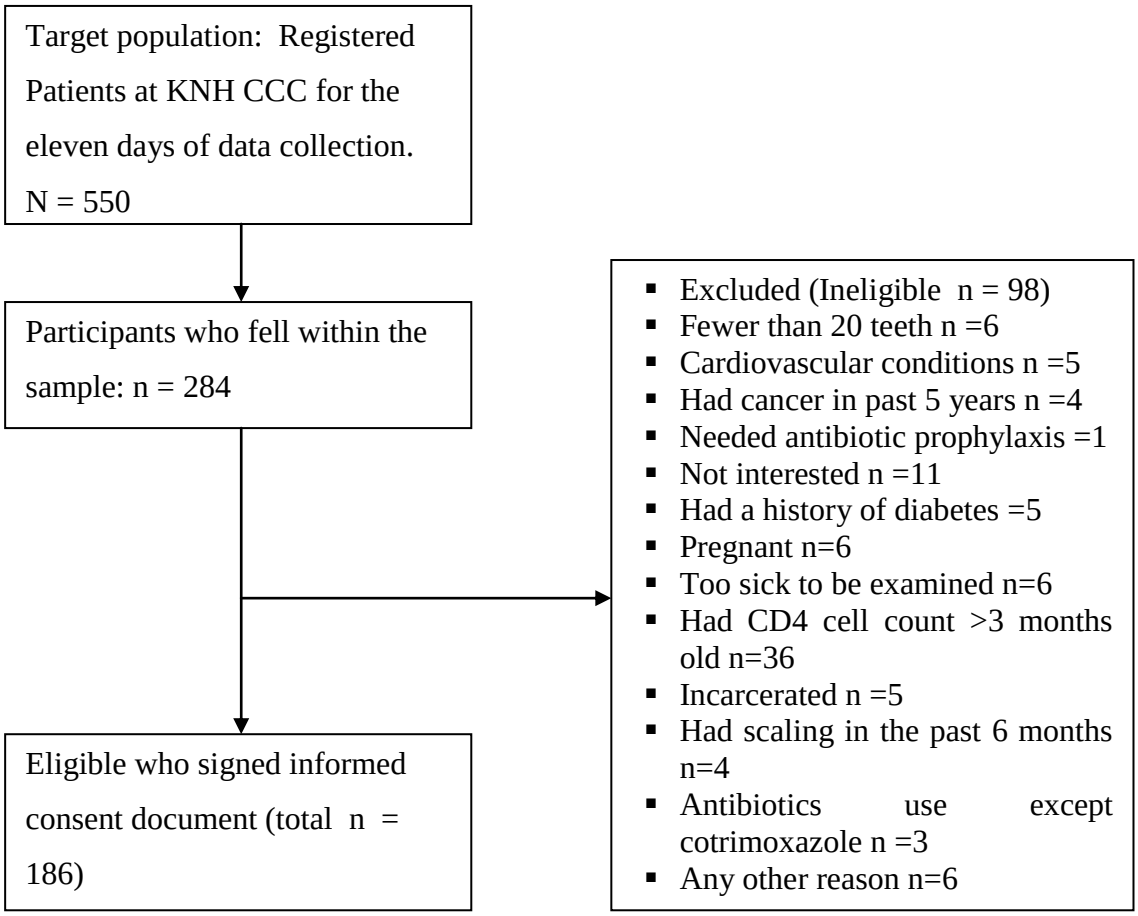


Fig 2: Participants recruitment flow diagram.

3.5 Data Collection Instruments and Techniques

Data was collected using various tools and techniques described as follows.

3.5.1 Data Collection Tools

A clinical examination form (modified World Oral Health assessment form 1997- Appendix II) was used to record data on various oral hygiene and periodontal status of

the participants. The following indices were used to assess the oral hygiene and periodontal status;

Table 2: Indices used for Various Clinical Parameters during Data Collection

Index	Variables
Quigley Hein Index - (Modified by Turesky et al, 1970) index	Oral hygiene- plaque score.
Loe and Silness gingival index- 1963	Gingival inflammation
CDC and AAP definition (2007)	Clinical attachment loss
CDC and AAP definition (2007)	Periodontal pockets

3.5.2 Preliminary Phase

Preliminary visit was made to the selected study site, which is KNH-CCC clinic in order to work out logistics and geographical mapping.

3.5.3 Calibration

Data collection was done by the Principal investigator who was calibrated by one of the supervisors (a Periodontist) before data collection. Clinical examination was done to determine the inter-examiner reproducibility. Kappa values were calculated for plaque score, gingival inflammation, periodontal probing depth (PPD) and clinical attachment loss (CAL). An almost perfect agreement was obtained with Kappa scores range of between 0.8-0.93 (0.9 for plaque score, 0.93 for gingival inflammation, 0.80 for PPD and 0.82 for CAL). For intra-examiner variability, repeated examinations of every fifteenth patient to adjust for intra - examiner errors was done (kappa score 0.82). This showed an almost perfect agreement.

3.5.4 Actual Data Collection Phase

Socio-demographic variables such as age, gender, residence, level of education and form of employment were collected from the participants through an interview by a

trained research assistant. The information on smoking status, past dental treatment and medical history were also obtained from the participants and recorded in the modified WHO- clinical examination form 1997.

Clinical Examination

Examination of the recruited participants was carried out at the CCC clinic with the participants seated upright on a chair and head bent backwards. The examination was done under natural light. Sterile Hu- Friedy (Marquis) periodontal probes and mouth mirrors were used in the examination. Clinical findings including plaque, gingivitis, periodontal probing depth and recession were obtained and recorded in the modified WHO- clinical examination form 1997 (Appendix II).

Measurement of Gingivitis

Examination for the presence or absence of LGE, NUG and NUP was done according to the EC-Clearinghouse (1993) criteria. Thereafter, gingival inflammation was assessed using the gingival index by Loe and Silness (1963) (appendix II). This assessment was done by sweeping the Hu-Friedy (Marquis) periodontal probe under light finger pressure at the gingival sulcus of the designated 'Loe and Silness' index teeth and recorded after 15 seconds in the modified WHO- clinical examination form 1997.

Plaque measurement

Plaque levels were assessed using the Turesky modification of plaque index by Quigley and Hein (1970) (appendix II). Disclosing tablets (Produits dentaires Vevey, Switzerland) was used to assess the plaque levels and to increase the sensitivity of detection and visual quantification of plaque. The plaque levels on the buccal and lingual surfaces of 'Ramfjord' index teeth, that is, 16, 21, 24, 36, 41 and 44 (FDI

nomenclature) was assessed and recorded in the modified WHO- clinical examination form 1997.

Periodontal Disease Measurements

Periodontal Probing Depth (PPD) - the height of the free gingival margin to the most apical location of the periodontal pocket was determined at six sites per tooth (mesio-facial, mid-facial, disto-facial, disto-lingual, mid lingual, and mesio-lingual) using a Hu-Friedy (Marquis) periodontal probe. The measurements obtained were recorded on the modified WHO- clinical examination form 1997 by a trained assistant.

Recession- the distance between the heights of the free gingival margin to the CEJ was also measured at six sites per tooth (mesio-facial, mid-facial, disto-facial, disto-lingual, mid lingual, and mesio-lingual). Positive values were given when the gingival margin was located apical to CEJ.

Clinical Attachment Loss (CAL) - PPD plus recession yielded the clinical attachment loss (CAL). PPD and REC values were rounded up to the next whole millimeter value. CAL in this study was only determined for teeth with recession.

These periodontal parameters were only measured for teeth with at least one-half of a remaining clinical crown (i.e. at least three contiguous sites in which PPD and REC were measurable).

CD4 Cell Counts and Treatment Profile

CD4 cell counts and HAART use information among the participants was obtained from their treatment files and registers after periodontal examination. Participants

who had CD4 cell count that were more than three months old were requested to have the CD4 cell counts done at the time of examination. Participants who did not oblige or the management declined to grant the request, had their data disregarded during analysis.

3.5.5 Definitions of Periodontal Disease

The definition used in this study was proposed by a working group with representatives from both the Center for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) [8]. However, since the CDC/AAP case definition (2007)[8] classify patients with mild periodontitis and no periodontitis together a definition for mild and no diseases was set. Mild periodontitis was defined as ≥2 sites with either CAL of ≥2 mm or PPD ≥4mm not on the same tooth (third molars excluded) while no periodontitis was defined as having not more than one sites with either CAL ≥2mm or PPD ≥4mm.

Table 3: Periodontal Diseases Case Definition (Modification of CDC/AAP Case Definition (2007)[8].

Disease category	***CAL***	***PPD***
Severe periodontitis	≥2 interproximal sites with CAL of ≥6mm (not on the same tooth)	**AND** ≥1 interproximal site with PPD of ≥5mm
Moderate Periodontitis	≥2 interproximal sites with CAL of ≥4mm (not on the same tooth)	**OR** ≥2 interproximal sites with PPD of ≥5mm (not on the same tooth)
Mild periodontitis	≥2 sites with CAL of ≥2 mm (not on the same tooth)	**OR** ≥2 sites with PPD ≥4mm (not on the same tooth)

**Third molars excluded*. *PPD* periodontal probing depth, *CAL*,clinical attachment loss;*

Infection control

Disposable face masks, cups and gloves were used. A set of autoclaved instruments (a periodontal probe and a dental mirror) was used for each patient.

3.6 Minimizing Errors and Biases

Systematic random sampling method was used to select the sample. This ensured that every person living with HIV/AIDS had an equal chance of being included in the study. Only individuals who met the inclusion criteria were enrolled. The Investigator was calibrated by a Periodontist.

3.7 Data Analysis and Presentation

The data collected were entered into a computer (statistical package for social sciences computer package) SPSS version 17.0 (SPSS Inc, Chicago, Illinois, USA). Data cleaning was done by checking frequencies and re- entering missing data. Data were analysed using the same SPSS version 17.0. Descriptive and inferential statistics were used. Descriptive statistics were measures of central tendencies and dispersions for continous variables (age, plaque scores, gingivitis, PPD, CAL, CD4 cell count and duration on HAART). Chi-square test was used to determine the association between key variables. Significance levels were accepted at $\alpha=0.05$. The data were presented in the form of tables and figures.

3.8 Main Outcome Measures

1. The prevalence of periodontal diseases among PLWHA.
2. Oral hygiene status (measured by mean plaque score and mean gingival score) in PLWHA
3. The levels of CD4 cell counts.
4. The HAART treatment among the PLWHA.

3.9 Ethical Considerations

Ethical approval was obtained from Kenyatta National hospital and University of Nairobi Ethics and Research committee (Appendix III). Permission for data collection was granted by the director of KNH CCC. The purpose of the study and expected benefits were clearly explained to the participants. Only participants who gave an informed written consent were recruited. Voluntary participation, confidentiality and withdrawal privilege were observed at all times during the study. Only the participants' file numbers were recorded in the data forms to ensure confidentiality. The entire examination was carried out maintaining universal standard precautions, and those requiring dental treatment were referred accordingly.

Study benefits

The patients received free dental check-ups and were informed of their dental health status and advised accordingly. Referral to Kenyatta National Hospital (KNH) dental department or the University of Nairobi (UON) School of Dental Sciences for treatment was done appropriately. Prescriptions of antibiotics, analgesics and antiseptics were given where indicated. Results on the pattern of periodontal diseases among PLWHA in Kenya will be published, with the aim of creating awareness and improving knowledge among both professionals and patients. The results will also form a basis for the development of viable management protocols to help prevent or minimise periodontal disease in PLWHA. This study as a partial fulfillment for the award of the Master of Dental Surgery degree in Periodontology has personal benefit to me in my efforts to enhance my career development.

Disclosure

The cost of the study was met by the principal investigator for academic purposes. Assistance was also obtained from UON School of Dental Sciences.

CHAPTER FOUR

RESULTS

4.1 Socio-Demographic Characteristics of the Participants

Out of the 186 PLWHA examined, there were 73 (39.2%) males and 113 (60.8%) females giving a sex ratio of 1:1.55. The male participants were significantly older than the females (t=3.78, P=0.00) with the male participants having a mean age of 40.25±9.73 years as compared to the females who had a mean age of 38.16±9.29 years. Table 4 shows the socio-demographic characteristics of the participants enrolled in the study.

Table 4: Socio-demographic characteristics of the participants enrolled in the study.

Variables		**Proportions of Participants n (%)**
Sex:	Male	73
	Female	113
Age Groups (Years):	20 - 29	21 (11.3)
	30 - 39	72 (38.7)
	40 - 49	62 (33.3)
	50 -59	22 (12)
	> 60	9 (4.7)
Residence	Lived outside Nairobi	54 (29.0)
	Lived in Nairobi	132 (71.0)
Employment	Formal	39 (21)
	Informal	147 (79)
Marital status	Single	33 (17.7)
	Married	115 (61.8)
	Widowed	27 (14.5)
	Divorced	7 (3.8)
	Separated	4 (2.2)
Education	None	8 (4.3)
	Primary	51 (27.4)
	Secondary	80 (43)
	College/University	47 (25.3)

Seventeen (9%) of the participants reported to have been smoking cigarettes at the time of the examination while 17 (9%) were former smokers and 152 (82%) had never smoked.

4.2 CD4 Cell Counts

The CD4 cell count for the participants enrolled in this study ranged between 10-1309 cells/mm^3 with a mean of 435.10$\pm$250.99 SD cells/mm^3. The CD4 cell counts were grouped in to three categories using the referent cell coding as CD4 cells <200, 200–499 and >500 cells mm^3. Table 5 shows the distribution of participants according to the CD4 cell counts in each of the three categories.

Table 5: Distribution of participants according to the CD4 cell counts.

CD4 cell counts/mm^3	Proportions of participants n (%)
<200	34 (18.3)
$\geq$200-499	90 (48.4)
$\geq$500	62 (33.3)

Table 6. Shows the distribution of the CD4 cell counts among the participants with regards to the socio- demographic characteristics. There was a statistically significant difference between the CD4 cell counts and gender with the male participants having lower CD4 cell counts as compared to the females (X^2=7.37, P=0.03).

Table 6: Distribution of CD4 cell count by socio-demographic variables of the participants

Variable		CD4+ cell counts/mm^3			X^2	P-Value
		<200 n (%)	≥ 200-499 n (%)	≥500 n (%)		
Gender	Male	17 (23.3)	40 (54.8)	16 (21.9)	7.37	0.03
	Female	17 (15.1)	50 (44.2)	46 (40.7)		
Age Groups (Years)	20-29	4 (19)	4 (19)	13 (62)	17.19	0.03
	30-39	15 (20.8)	36 (50)	21 (29.2)		
	40-49	10 (16.1)	37 (59.7)	15 (24.2)		
	50-59	4 (18.2)	7 (31.8)	11 (50.1)		
	> 60	1 (11.1)	6 (66.7)	2 (22.2)		
Employment	Formal	9 (23)	15 (38.5)	15 (38.5)	2.02	0.37
	Informal	25 (17)	75 (51)	47 (32)		
Marital status	Single	6 (18.2)	15 (45.5)	12 (36.4)	13.23	0.10
	Married	16 (13.9)	60 (52.2)	39 (33.9)		
	Widowed	7 (25.9)	11 (40.7)	9 (33.3)		
	Divorced	2 (28.6)	4 (57.1)	1 (14.3)		
	Separated	3 (75)	0	1 (25)		
Education	None	0	3 (37.5)	5 (62.5)	9.375	0.1
	Primary	13 (25.5)	20 (39.2)	18 (35.3)		
	Secondary	13 (16.3)	46 (57.5)	21 (26.3)		
	College/ university	8 (17)	21 (44.7)	18 (38.3)		

4.3 Highly Active Anti-Retroviral Therapy usage

Of the 186 participants enrolled in the study, 32 (17.2%) of the participants were not on HAART as at the time of examination while 154 (82.8%) were on HAART. With regards to the duration of HAART usage among the participants, the median duration of HAART usage was 41 months, mean 42.90$\pm$32.03 months while the range was between 1-132 months. Thirty four (22.1%) of the participants had used HAART for twelve or less months while 120 (77.9%) had used the drugs for more than twelve months. A combination of two NRTIs and one NNRTIs was the most common regimen with 136 (88.3%) of the participants using it while the rest were on two NRTIs and PIs.

With regards to the CD4 cell counts, there was no statistically significant difference observed between the CD4 cell counts and the therapy they were on (not on HAART, 2NRTIs+NNRTIs and 2NRTIs+PI) (X^2=3.78, P=0.44). However, a statistically significant difference was observed between the duration use of HAART and CD4 cell counts, with those who had used HAART for more than one year having higher CD4 cell count.

Table 7. Shows the CD4 cell counts and HAART regimen distribution among the participants in the study.

Table 7: CD4 cell counts and HAART Regimen Distribution among the Participants

HAART REGIMEN	CD4+ cell counts/mm^3			X^2	P-Value
	<200 n (%)	≥ 200-499 n (%)	≥500 n (%)		
2NRTIs+NNRTIs	25 (73.5)	70 (77.8)	41 (66.1)	3.78	0.44
2NRTIs+PI	3 (8.8)	9 (10)	6 (9.7)		
Not on HAART	6 (17.6)	11 (12.2)	15 (24.2)		
Duration on HAART					
0-12 months on HAART	15 (44.1)	15 (44.1)	4 (11.8)	21.61	0.00
>12 months on HAART	13 (10.8)	64 (53.3)	43 (35.8)		

4.4 Oral Hygiene status (Plaque Score)

All the participants had plaque with the overall mean plaque score being 1.78±0.49. There was a statistically significant difference between the mean plaque scores and gender with the male participants recording higher mean plaque scores than the female participants (X^2=10.52, P=0.01). A statistically significant difference was also observed between the mean plaque score and age groups (X^2=14.92, P=0.00) and smoking status (X^2=22.92, P=0.00). The use of HAART among the participants had no statistical influence on the mean plaque scores (X^2=0.09, P=0.83). Table 8 shows the distribution of the mean plaque scores among the participants.

Table 8: The Distribution of the Mean Plaque Scores among the Participants

Variable		Mean plaque score n (%)		X^2	P-Value
		<2	≥2		
Gender	Male	42 (57.5)	31 (42.5)	10.52	0.01
	Female	90 (79.6)	23 (20.4)		
Age groups (years)	20-29	18 (85.7)	3 (14.3)	14.92	0.00*
	30-39	55 (76.4)	17 (23.6)		
	40-49	45 (73.8)	16 (26.2)		
	50-59	11 (50.0)	11 (50.0)		
	>60	3 (30.0)	7 (70.0)		
Smoking Status	Current smokers	5 (28.4)	12 (70.6)	22.92	0.00
	Former smokers	8 (47.1)	9 (52.9)		
	Never smokers	119 (78.3)	33 (21.7)		
CD4 Cell Count	<200	20 (58.8)	14 (41.2)	3.69	0.16
	200-499	64 (71.1)	26 (28.9)		
	≥500	48 (77.4)	14 (22.6)		
On HAART		110 (71.4)	44 (28.6)	0.09	0.83
Not on HAART		22 (68.8)	10 (31.2)		

* Fishers exact test*.

4.4.1 Gingival Inflammation.

All the participants showed signs of inflammation in at least one site examined. The mean gingival inflammation score was 1.34±0.38. Participants with CD4 cell counts of ≥200<500 cells/mm^3 had statistically higher mean gingival inflammation score than the rest (X^2=17.52, P=0.00). Table 9 shows the distribution of the mean gingival inflammation score among the participants.

Table 9: The Distribution of Mean Gingival Inflammation Score Among the Participants

Variable		Mean Gingival Score			X^2	P-Value
		0-0.99	1-1.99	≥2		
		n (%)	**n (%)**	**n (%)**		
Gender	Male	12 (16.4)	58 (79.5)	3 (4.1)	1.74	0.49
	Female	26 (23.0)	80 (70.8)	7 (6.2)		
Age Group	20 - 29	7 (33.3)	13 (61.9)	1 (4.8)		
(years)	30 - 39	16 (22.2)	55 (76.4)	1 (1.4)		
	40 - 49	10 (16.1)	49 (79)	3 (4.8)	13.63	0.06*
	50 - 59	5 (22.7)	14 (63.6)	3 (13.6)		
	> 60	0	7 (77.8)	2 (22.2)		
Smoking	Current Smokers	4 (20.4)	13 (76.5)	0		
Status	Former smokers	3 (17.6)	13 (73.7)	1 (5.9)	0.84	0.97*
	Non- Smokers	31 (20.4)	112 (73.7)	9 (5.9)		
Mean	<2	34 (25.8)	96 (72.7)	2 (1.5)	18.05	0.00
Plaque score	≥2	4 (7.4)	42 (77.8)	8 (14.8)		
CD4 cell	<200	13 (38.2)	21 (61.8)	0		
count/mm^3	≥200 <500	8 (8.9)	75 (83.3)	7 (7.8)	17.47	0.00*
	≥500	17 (27.4)	42 (67.7)	3 (4.8)		
	On HAART	28 (18.2)	116 (75.3)	10 (6.5)	4.45	0.11
	Not on HAART	10 (31.3)	22 (68.8)	0		

Fishers exact test

4.5 Periodontal Diseases Associated With HIV Infection

Periodontal diseases that are associated with HIV infection (LGE, NUP, NUG) were significantly found in 12 (6.5%) of the participants enrolled in the study. When these diseases were categorized as separate entities, LGE was observed in 6 (3.2%) of the participants while NUG and NUP were observed in 2 (1.1%) and 4 (2.2%) of the participants respectively.

4.5.1 Distribution of Periodontal Diseases Associated with HIV Infection among the Participants

Periodontal diseases associated with HIV infection were more common among the male participants, those with a history of tobacco smoking, those with higher mean plaque score and participants with CD4 cell counts <200 cells/mm^3. Necrotizing periodontal diseases (NUG and NUP) were more prevalent in participants who were males, had a history of tobacco smoking, had higher mean plaque score and had CD4 cell counts <200 cells/mm^3. On the other hand, LGE was reported in participants who had no history of tobacco smoking and in participants with either <200 or >200-<500 cells/mm^3 CD4 cell counts categories. Table 10 shows the distribution of periodontal diseases associated with HIV infection among the participants.

Table 10: The Distribution of Periodontal Diseases Associated with HIV Infection among the Participants

Variables		LGE n (%)	NUG n (%)	NUP n (%)
Gender	Male	2 (25)	2 (25)	4 (50)
	Female	4 (100)	0	0
Smoking Status	Current Smokers	0	2 (50)	2 (50)
	Former Smokers	0	0	2 (100)
	Non- Smokers	6 (100)	0	0
Mean Plaque Score	<2	4 (100)	0	0
	≥2	2 (25)	2 (25)	4 (50)
CD4 cell counts/mm^3	>200	4 (40)	2 (20)	4 (40)
	200-499	2 (100)	0	0
	≥500	0	0	0
On HAART		6 (60)	2 (20)	2 (20)
Not on HAART		0	0	2 (100)

4.6 Conventional Periodontitis

The mean periodontal probing depth (PPD) was 2.2±0.8mm while the mean clinical attachment loss (CAL) among the participants was 3.5±1.2mm.

4.6.1 Prevalence of Conventional Periodontitis in PLWHA

A total of 117 (62.9%) of the participants had conventional periodontitis (≥2 sites with CAL of ≥2 mm **OR** ≥2 sites with PPD ≥4mm (not on the same tooth) while 69 (37.1%) had no conventional periodontitis. When the two clinical parameters were considered separately, 84 (45.2%) of the participants had ≥2 sites with PPD ≥4mm while 97 (52.2%) had ≥2 sites with CAL of ≥2 mm. Table 11 shows the prevalence of conventional periodontitis among the participants.

Table 11: The Prevalence of Conventional Periodontitis among the Participants

Periodontal measures (mm)	n (%) of participants
PPD	84 (45.2)
CAL	97 (52.2)
Either CAL ≥2 or PPD ≥4mm in ≥2 sites	

**PPD*- periodontal probing depth; *CAL*, clinical attachment loss;*

4.6.2 Distribution of conventional periodontitis among the participants.

The presence of conventional periodontitis showed statistical significant difference between gender, with the disease being more prevalent among the male participants (X^2=11.86, P=0.001). Conventional periodontitis was statistically higher among participants of older age groups (X^2=38.26, P=0.001), participants with higher mean plaque score (X^2=15.45, P=0.001), those with a history of tobacco smoking (X^2=9.01, P=0.01) and low CD4 cell counts (X^2=10.50, P=0.001). The HAART usage among the participants had no statistically significant influence on the presence of conventional periodontitis in the study. Table 12 shows the distribution of conventional periodontitis among the participants.

Table 12: The Distribution of Conventional Periodontitis among the Participants

Variable		No Periodontitis n (%)	Periodontitis n (%)	X^2	P Value
Gender	Male	16 (21.9)	57 (78.1)	11.86	0.001
	Female	53 (46.9	60 (53.1)		
Age (years)	20-29	17 (81)	4 (19)	38.26	0.001
	30-39	34 (47.2)	38 (52.8)		
	40-49	17 (27.4)	45 (72.6)		
	50-59	1 (4.5)	21 (95.5)		
	≥60	0	9 (100)		
Mean plaque score	<2	58 (43.9)	74 (56.1)	15.45	0.001
	≥2	11 (20.4)	43 (79.6)		
Smoking status	Never Smokers	64 (42.1)	88 (57.9)	9.01	0.01
	Current Smokers	2 (11.8)	15 (88.2)		
	Former Smokers	3 (17.6)	14 (82.4)		
CD4 Cell Count	<200	9 (26.5)	25 (73.5)	10.50	0.001
	200-499	27 (30)	63 (70)		
	≥500	33 (53.2)	29 (46.8)		
	On HAART	57 (37)	97 (63)	0.00	0.96
	Not on HAART	12 (37.5)	20 (62.5)		

4.6.3 Severity of Conventional Periodontitis among the Participants

Only 8 (4.3%) of the participants in this study had severe periodontitis, 47 (25.3%) had moderate periodontitis while 113 (70.4%) had mild/ no periodontitis (AAP/CDC case definition) [8].

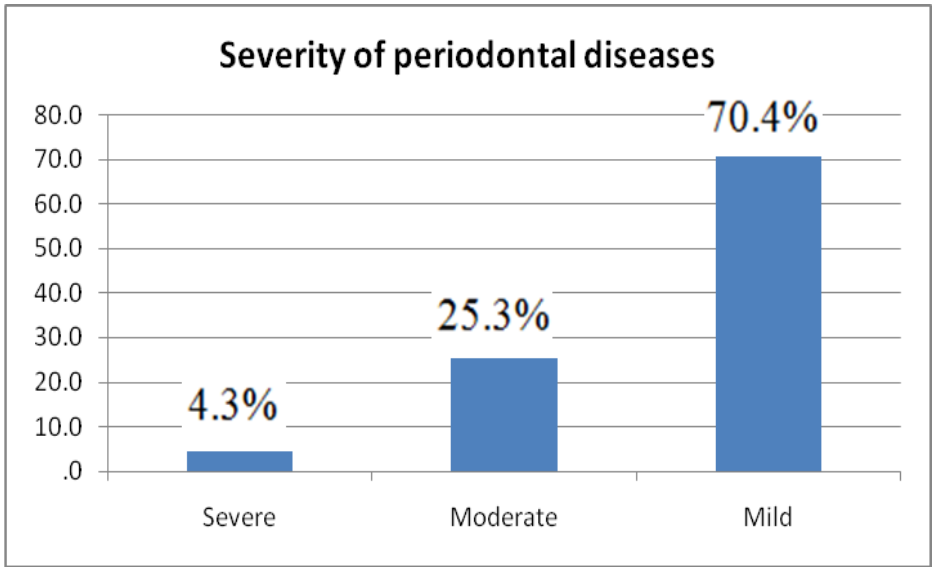


Fig 3: Distribution of Conventional Periodontitis by Severity (CDC/ AAP Case definition)

4.6.4 The Distribution of Conventional Periodontitis by Severity Among the participants (CDC/AAP Case Definition)

Regarding the CDC/AAP consensus case definition[8], a statistically significant difference between the severity of periodontitis and gender was observed with more males having severe periodontitis as compared to the females (X^2=14.99, P=0.001). A statistically significant difference was also observed between the mean plaque scores and the severity of periodontitis with participants having higher mean plaque scores recording severe periodontitis (X^2=26.77, P=0.001). However, there was no statistically significant difference between the severity of conventional periodontitis and CD4 cell counts (X^2=0.83, P=0.94) and HAART use among the participants (X2=0.42, P=0.81). Table 13 shows the distribution of conventional periodontitis according to severity among the participants (CDC/AAP consensus definition[8]).

Table 13: The Distribution of Conventional Periodontitis According to Severity among the Participants (CDC/AAP Consensus Definition)

Variable		The CDC/AAP Consensus Definition			X^2	P Value
		Severe PD	Moderate PD	Mild/no PD		
		n (%)	n (%)	n (%)		
Gender	Male	6 (8.2)	27 (37)	40 (54.8)	14.99	0.001
	Female	2 (1.8)	20 (17.7)	91 (80.5)		
Age groups (years)	20-29	1 (4.8)	2 (9.5)	18 (85.7)	48.11	0.001
	30-39	2 (2.8)	9 (12.5)	61 (84.7)		
	40-49	0	17 (27.9)	44 (72.1)		
	50-59	3 (13.6)	12 (54.5)	7 (31.8)		
	≥60	2 (20)	7 (70)	1 (10)		
Mean plaque score	<2	1 (0.8)	25 (18.9)	106 (80.3)	26.77	0.001
	≥2	7 (13.0)	22 (40.7)	25 (46.3)		
Smoking status	Never smokers	3 (2)	31 (20.4)	118 (77.6)		
	Current smokers	3 (17.6)	9 (52.9)	8 (29.4)	25.94	0.001
	Former smokers	2 (11.8)	7 (41.2)	8 (47.7)		
CD4 Cell Count	<200	2 (5.9)	9 (26.5)	23 (67.6)		
	200-499	4 (4.4)	24 (26.7)	62 (68.9)	0.83	0.94
	≥500	2 (3.2)	14 (22.6)	46 (74.2)		
	On HAART	2 (4.5)	40 (26)	107 (69.5)	0.42	0.81
	Not on HAART	1 (3.1)	7 (21.9)	24 (75)		
On HAART	≤12 months	3 (8.8)	11 (32.4)	20 (58.8)	3.15	0.21
	>12 months	4 (3.3)	29 (24.2)	87 (72.5)		

CHAPTER FIVE

DISCUSSION

Significantly more females (60.8) than males (39.2) participated in the study. This unequal sex ratio (Female to male ratio 1.55:1) could be a reflection of the HIV infection prevalence rate which has been reported to be higher in females than males in a ratio of 1.5:1 in Kenya[30]. Most of the participants in this study were in the 30-39 year age-group with the majority of the female participants having been in this category while the male participants were equally distributed between age-groups 30-39 and 40-49 years. This is in agreement with the current HIV infection epidemiology in Kenya where the peak prevalence of the disease is 30-34 years for females and 40-44 for males[30].

Most of the participants enrolled in this study (61.8) were married. Marriage has been identified as a risk factor in HIV transmission with extra-marital incidences being implicated as the main reason[58]. When the level of education was considered, the majority of the participants were educated with about 68.3 having had at least secondary education and above. This may have been partly due to the urban setting of the study. However, this implies that the awareness campaign against the spread of HIV infection might have not been adequately assimilated and, therefore, ineffective in reducing the rate of infection in the educated class[59]. Majority of the participants (79) were drawn from the informal sector of employment while 21 were in formal employment. Regarding the place of residence, 71 of the participants lived in Nairobi while the rest lived outside Nairobi.

Periodontal Diseases

Gingival Inflammation

The overall mean for gingival score in the current study was 1.34±0.38. This was slightly lower than the mean obtained in a study done by Anand Induchoodan (2008),[56] but higher than the mean gingival score obtained in a study done by Doshi et al. (2008) and Ali et al. (2009) which found a mean of 0.97 and 1.1 respectively[19, 60].

The mean gingival score among the participants showed a significant relationship with the CD4 cell counts. Participants with CD4 cell counts between ≥200- 499 cells/mm^3 had a higher mean gingival score than those with either <200 or ≥500 cells/mm^3. This finding is in agreement with the findings of Vastardis et al. (2003)[29] and Anand Induchoodan (2008) [56] who reported a significant relationship between lower bleeding index score and CD4 cell count of <200 cells/mm^3. However, this contradicts the findings of the study done by Doshi et al. (2008) which reported an increase in mean gingival score with a decrease in the CD4 cell count[60]. The plausible explanation for the decreased mean gingival scores in participants with <200cells/mm^3 as compared to those with >200cell/mm^3 may be that, gingival inflammation requires some level of operating host defense which in severe CD4 cell depletion (<200 cells/mm^3) may not be operative[61].

Although participants who were on HAART had a higher mean gingival inflammation score (81.8 of them with a mean ≥1) compared to participants who had never used HAART (68.8 of them with a mean of >1), the difference was not statistically

significant. This contradicts the findings of the study by Ulrich et al. (2012)[54] which showed a significantly higher level of gingival inflammation among the participants who were not on HAART as compared to those who were on HAART. The difference observed in the current study may be due to other factors such as mean plaque scores and the variation of immune competence of the participants.

Periodontal Diseases Associated with HIV Infection

The prevalence of periodontal diseases associated with HIV infection was 6.5 with 3.3 for LGE, 2.2 for NUP and 1.1 for NUG. The findings in the current study were lower than the prevalence reported in the pre-HAART studies which reported a prevalence range of between 9 and 50 for LGE, 11 and 25 for NUG and 1 and 18 for NUP[16]. In the HAART era, studies have reported a decrease in prevalence. Therefore, the prevalence of NUP and NUG in this study was in agreement with the findings from Kroidl et al. (2005) study, although the finding on the prevalence of LGE in their study was relatively higher than this study. Several other studies, both local and international have reported slightly higher prevalence of between 5 and 9.9 for NUG and NUP[21, 23, 61]. The variations observed in different studies can be attributed to differences in immune competence among the sampled population, mode of sample selection, problems of diagnosing periodontal changes associated with HIV infection and studies being carried either out or in a non-dental setting[21, 42, 65].

Necrotizing periodontal diseases were observed in the male participants while LGE was observed in both genders. This observation may be partly due to the high level of risk factors such as higher mean plaque score and smoking status that were more evident among the male participants as compared to the female participants. These two factors in addition to immunosuppression are considered as the risk to the

development of these types of periodontal diseases[20]. Based on the CD4 cell count, participants with low CD4 cell counts; <200 cell/mm^3, were at risk of developing necrotizing periodontal diseases (NUG and NUP) and LGE. This finding is in agreement with several previous studies[22, 44, 45, 47] that have reported a significant correlation between necrotizing periodontal diseases and CD4 cell counts of <200 cells/mm^3 [45, 47]. Some of the participants with LGE had CD4 cell counts of ≥200<500 cells/mm^3. This suggests that LGE may occur in HIV seropositive patients with either severe or moderate immunosuppression. A similar finding on the occurrence of LGE was reported in the study done by Kroidl et al. (2005) which reported an occurrence of these lesions in participants with even higher CD4 cell counts >500cells/mm^3 [22].

In the current study there was an association of periodontal diseases associated with HIV infection with higher mean plaque scores and tobacco smoking. These findings are in agreement with several previous studies which have reported the two factors as strong risk factors for the development of NUG and NUP[16, 46, 21, 22, 46, 65]. Therefore, there is a special need for advice on smoking cessation and better oral hygiene when managing HIV seropositive patients. An exploration of the prevalence of periodontal diseases associated with HIV infection, in relation to age, HAART use and duration on this treatment did not uncover any significant association.

Conventional Periodontitis

The prevalence of conventional periodontitis was 62.9 in the current study. This is slightly higher than the prevalence reported by Robinson et al. (1996) of 59.6 [61] but lower than the reported prevalence by Scheutz et al. (1997)[15] and Ali et al. (2009)[23] of 82 and 90.8 respectively. This variation may be attributed to the different case

definitions used in these studies, methodologies applied in data collection (partial mouth versus full mouth) and the divergent characteristics of the examined cohorts.

The mean PPD in this study was 2.2±0.8 while the mean CAL was 3.5±1.2. The means of these two clinical parameters in the current study were lower than the means obtained in the study done by Anand Induchoodan in 2008[56]. However, the study in question had a sample size of 60 participants making it almost impossible to compare the two.

Based on the consensus definition (CDC/AAP) [8], 4.3 of the participants had severe periodontitis, 25.3 had moderate periodontitis while the rest had mild or no periodontitis. This contradicts the findings by Lance Vernon et al. (2010), who reported over 90 prevalence of moderate to severe periodontitis when this consensus definition was applied in their study[25]. The low prevalence of moderate to severe periodontitis in this study may be due to the systematic random sampling method that was applied in participants recruitment as compared to self-referral that was applied by Lance Vernon et al. 2010[25]. Self-referral applied in their study may have resulted in participants who had poorer periodontal health and/or those interested in obtaining dental care being selected. Therefore, it is difficult to make a meaningful comparison between the findings of this study and several other studies that examined the severity of periodontitis. The difficulty may be due to the inconsistencies in case definitions and methodologies used to collect the clinical periodontal measurements (e.g. partial mouth versus full-mouth probing).

In the current study, the severity of periodontitis as measured by PPD and CAL showed significant relationship with gender, age, tobacco smoking and high mean plaque score. The male participants had more severe periodontitis as compared to

their female counterparts. Similar findings have been reported in other studies and have been explained by the better oral hygiene practices[63, 64, 65, 66] and/or more utilization of oral health care services among females. Age was significantly associated with the severity of periodontitis, suggesting the potential role of age as a risk factor of periodontitis in PLWHA. These findings of higher prevalence and severity of periodontitis with increasing age has been reported by previous investigators[16, 35]. However, age may just indicate the cumulative effect of prolonged exposure to true risk factors rather than being a risk factor[63].

Tobacco smoking and poor oral hygiene had a significant association with conventional periodontitis. This is in agreement with the findings of previous studies[16, 18, 35, 37].

In the current study, low CD4 cell counts (<200 cells/mm^3) showed a significant relationship with the presence of periodontitis but not the severity. The finding concerning the impact of CD4 cell count on the presence of periodontitis is in agreement with several previous studies[22, 26, 66]. With regards to the severity of periodontitis, the current study finding is in agreement with several studies[23, 28, 29] but contradicts other studies [25, 26, 62] which have reported that decreased CD4 cell count predicted both the presence and severity of periodontitis. In the study by Lance Vernon et al. (2010) an association between the severity of periodontitis and CD4 cell counts was reported. However, in their study they dichotomized the CD4 cell counts into <200 and $\geq$200 cells/mm^3 and used three outcome measures (PPD, Recession and CAL) as continuous variable to characterize periodontitis. These differences highlight

some of the complexities of comparing periodontal diseases in PLWHA and making the importance of thorough reporting of the severity of periodontitis difficult.

Despite the beneficial role of HAART reported in previous studies of reducing the prevalence of HIV-related oral manifestations[12], this study found no significant difference between periodontal diseases in people on HAART and those not on HAART. However, the initiation of HAART could be beneficial since it aids in immune restoration thus playing an indirect role in preventing the occurrences of some of the periodontal diseases.

CD4 cell counts and HAART profile

In the current study, participants were categorized into three groups according to CDC classification system for HIV Infection, (1993)[67] based on the level of their CD4 cell counts; <200, ≥200-499 and ≥500 cells/mm^3. About 18.3 of the participants had CD4 cell counts <200 cells/mm^3 while 48.4 and 33.3 had a count of ≥200<500 and >500 cells/mm^3 respectively. A similar proportion of the participants with CD4 cell counts of <200 was also reported in a study done by Lance Vernon et al. (2009)[25], who had 17 of their participants with <200 cells/mm^3. However, they categorized their participants into two categories, that is <200 and ≥200 cells/mm^3. A study by Kroidl et al. (2005) grouped the CD4 cell counts into three categories but had varied proportions of participants in each category [22]. The low proportion (18.3) of the participants with <200 cell/mm^3 in this study, may be due to the recruitment of the participants from a center that offers comprehensive care including HAART.

HAART is the gold standard mode of management for HIV infection[52]. In this study, 82.8 of the participants were on HAART with the median duration of HAART use having been 41 months. This was closely in agreement with the findings in studies done elsewhere that reported over 90 of their participants having been on HAART[22, 25]. However, this contradicts the findings from Kenyan studies done in the same center[23, 68], that reported 13.8 and 57 of the participants being on HAART respectively. This difference may be due to the fact that the current study recruited participants who were already registered and undergoing therapy.

Oral Hygiene Status

The overall mean plaque score in the current study was 1.78±0.49. This was slightly higher than the mean plaque index obtained in a study done by Anand Induchoodan in 2008, which reported a range of between 1.34 ±0.22-1.59±0.39 among the three categories of participants in that study[56]. The difference may be due to the use of Silness and Loe index (1964) in that study as compared to the Turesky modification of Quigley and Hein index (1970) applied in the current study. More males in the present study had higher mean plaque score as compared to the females. This difference was also observed by Doshi et al. (2008)[60]. This was thought to be a reflection of better oral hygiene practices[64, 65] or more utilization of oral health care services among females than males[62].

The use of HAART among the participants had no significant relationship with the mean plaque score. This contradicts the finding that attributes the use of HAART to oral side effects like dry mouth which tend to promote plaque accumulation [57].

Conclusion

The prevalence of periodontal diseases is high among PLWHA with conventional periodontitis being the most prevalent. The presence of periodontal diseases is associated with decreased CD4 cell counts, however, the severity of periodontal diseases is not associated with CD4 cell counts.

Limitation

- The prevalence and severity of periodontal diseases may have been slightly overestimated or underestimated by rounding up to the next whole millimeter in the periodontal probing depth and clinical attachment loss measurements. The use of a Hu-Friedy (Marquis) periodontal probe which is graduated at intervals of 3 mm may also have been a source of limitation.

Recommendations

- Periodontal care is an important component that should be incorporated in the comprehensive care of PLWHA. A greater emphasis on treatment and prevention of periodontal disease is indicated for persons with low CD4 cell count.

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APPENDICES

APPENDIX I

INFORMED CONSENT FORM

This is to certify that, ----------, hereby agree to participate in this educational and research study of Dr. Riro, Masters student, University of Nairobi P.O. BOX 19676 Nairobi.

I understand this study will involve a mouth examination using a dental mirror and a periodontal probe where all teeth excluding the third molars will be examined for plaque, gum bleeding, periodontal pocket depth and recession. Examination will also include recording of any periodontal diseases present.

I understand that my current oral health status will be evaluated and oral hygiene instructions given accordingly. I will be referred to KNH or UON dental school for treatment if need be.

Risks

I understand there is a chance of slight discomfort and bleeding in my gums during the mouth examination.

Benefits:

I understand that I will be made aware of any abnormal findings in my mouth so that I may voluntarily seek the care I need.

I understand that the results obtained from the study will provide baseline information for development of a protocol to help prevent or minimise periodontal diseases in PLWHA.

Cost and Payments:

I understand that this study is voluntary and no monetary compensation will be given.

Confidentiality:

I understand that all personal information obtained about me during this research will be kept strictly confidential.

Withdrawal Previledge

I understand that I may refuse to participate in or withdraw from the project at any time without penalty or prejudice. If I do this, I will continue to receive health care at KNH CCC as I would normally receive.

Voluntary Consent:

I certify that I have read all this consent form or it has been read to me and that I understand it. Any questions pertaining to the research have been answered to my satisfaction. My signature means I freely agree to participate in this study.

---------- ----------

Signature of the participant Date

Investigator Statement:

I certify that I have explained to the above individual the nature and purpose of this study, potential benefits and possible risks associated with participation in this study. I have answered any questions raised and explained the above in the best language understood by the participant on the date on this consent form.

_____ _____

Investigator Date

APPENDIX II

CLINICAL EXAMINATION FORM

CLINICAL EXAMINATION FORM

Socio-demographic variables

Identification number_____Sex_____Residence _____

Age in years as at last birthday _____Level of Education_____

Marital status _____Occupation _____

Past medical History (yes/ no)

Cardiovascular disease_____

Diabetic_____

Cancer_____

Cancer chemotherapy in the past 5 years_____

Use of any antibiotics except cotrimoxazole (septrin) _____

Joints prosthesis_____

Pregnant_____

Past history of periodontal therapy

Last date of periodontal therapy_____

Quigley Hein Index - (Modified by Turesky et al, 1970). The Plaque Index System

Scores	**Criteria**
0	No plaque
1	Separate flecks of plaque at the cervical margin of the tooth.
2	A thin continuos band of plaque (up to 1 mm) at the cervical margin of the tooth.
3	A band of plaque wider than 1 mm but covering less than one-third of the crown.
4	Plaque covering at least one-third but less than two-thirds of the crown.
5	Plaque covering two-thirds or more of the crown of the tooth

Tooth	16		21		24		36		41		44	
Surface	F	P	F	P	F	P	F	L	F	L	F	L
Score												

Loe and Silness, 1963 Gingival index

Scores	**Criteria**
0	Absence of inflammation
1	Mild inflammation; slight change in colour
2	Moderate inflammation; bleeding on probing.
3	Severe inflammation; tendency towards spontaneous bleeding

Tooth	16		12		24		36		32		44	
Surface	F	P	F	P	F	P	F	L	F	L	F	L
Score												

Periodontal probing depth, Recession and clinical attachment loss

Probing depths gives an indication of the extent of loss of attachment. However, this measurement is unreliable when there is gingival recession that is when CEJ is visible. Therefore, in cases where CEJ is visible Clinical attachment loss is recorded from CEJ to the base of the gingival sulcus.

Periodontal diseases case definition (Modification of the clinical case definitions proposed by the CDC working group for use in population-based surveillance of periodontitis-2007) [8]**.**

Disease category	**CAL**	**PD**
Severe periodontitis	More than 2 interproximal sites with CAL of more or equal to 6mm (not on the same tooth)	**AND** More than 1 interproximal site with PD of more or equal to 5mm
Moderate Periodontitis	More than 2 interproximal sites with CAL of more or equal to 4mm (not on the same tooth)	**OR** 2 or more interproximal sites with PD of more or equal to 5mm (not on the same tooth)
Mild periodontitis	$\geq$2 sites with CAL of $\geq$2 mm (not on the same tooth)	**OR** $\geq$2 sites with PPD $\geq$4mm (not on the same tooth)

Third molars excluded

Maxillary Arch

Periodontal Probing Depth (PPD), Recession, Clinical Attachment Loss (CAL)

Tooth	17			16			15			14			13			12			11			21			22			23			24			25			26			27		
Palatal																																									
PPD																																									
Recession																																									
CAL																																									
Facial																																									
PPD																																									
Recession																																									

Mandibular Arch

Periodontal probing depth (PPD), Recession and clinical attachment loss (CAL)

Tooth	47			46			45			44			43			42			41			31			33			33			34			35			36			37		
Lingual																																									
PPD																																									
Recession																																									
CAL																																									
Facial																																									
PPD																																									
Recession																																									

Patient's status and treatment profile

CD 4 cells count_____

HAART (yes/no) _____

If yes, type of HAART _____

Duration on HAART_____

Any other relevant medical History_____

APPENDIX III

ETHICAL APPROVAL

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.
Tel: 726300-9
Fax: 725272
Telegrams: MEDSUP", Nairobi.
Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/ A/30

24th February 2011

Dr. Riro Sibuti
Dept. of Periodontology, Community and Preventive Dentistry
School of Dental Sciences
University of Nairobi

Dear Dr.Sibuti

RESEARCH PROPOSAL: "PATTERN OF PERIODONTAL DISEASES IN PERSONS LIVING WITH HIV INFECTION/AIDS IN NAIROBI" **(P352/10/2010)**

This is to inform you that the KNH/UON-Ethics & Research Committee has reviewed and **approved** your above revised research proposal for the period 24th February 2011 – 23rd February 2012.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimens must also be obtained from KNH/UON-Ethics & Research Committee for each batch.

On behalf of the Committee, I wish you a fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of the data base that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF A N GUANTAI
SECRETARY, KNH/UON-ERC

c.c. The Deputy Director CS, KNH
The HOD, Records, KNH
The Dean, School of Dental Sciences, UON
The Chairman, Dept.of Periodontology, Community and Preventive Dentistry, UON
Supervisors: Dr. Evelyn Wagaiyu, Dept.of Period/Community and Preventive Dentistry, UON
Dr. Mutave R. J. Dept.of Period/Community and Preventive Dentistry, UON