Peri-implant health and disease: A Cross-sectional study

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Declaration

I, Sibongile Priscilla Mahlangu declare that this Research Report is my own, unaided work. It is being submitted for the Degree of Master of Dentistry (Oral Medicine and Periodontics) at the University of the Western Cape, Parow. It has not been submitted before for any degree or examination at any other University.

Signature of candidate

7th day of January 2024 in Parow, Cape Town.

Abstract

Introduction: Preventive strategies that address peri-implant diseases can only be developed appropriately when there is thorough knowledge of the prevalence of disease based on standardized assessment protocols and case definitions **Aims and objectives:** The aim of this cross-sectional study was to evaluate the prevalence of the peri-implant health and disease in patients who received implants during the period 2012-2018 in the Department Oral Medicine and Periodontics Clinic, University of the Western Cape. **Methods:** The information for the vectors of the Implant Disease Risk Assessment tool was obtained from the files of participants. A comprehensive periodontal examination was performed. **Results:** Peri-implant mucositis (PIM) was diagnosed in 19.0% (subject level) and 11.7% (implant level). Peri-implantitis (PI) was diagnosed in 14.3% (subject level) and 8.8% (implant level). Peri-implant health (PIH) was found to be 66.7% (subject level) and 79.6% (implant level). Subjects with the history of periodontitis in the PI group were 50% respectively. Only 2.3% subjects and 33.3% implants diagnosed with PI had a combination of periodontitis and diabetes mellitus. Subjects in the PI group who participated in supportive periodontal therapy (SPT) casually and those who were non-compliant were 83.3% and 16.6% respectively. Restorative margin to bone crest of <1.5mm observed in the PI group was 66.7%. **Conclusion:** The prevalence of health and disease was within the range of previous calculations from meta-analysis and systemic reviews. There was a positive correlation with function time, history of periodontitis, casual SPT visits, non-compliance to SPT, a distance of <1.5mm from restorative margin to bone crest in the PI group. No associations were found between peri-implantitis and smoking status, and diabetes mellitus. This is attributed to the low number of smokers and diabetic patients that participated in the study.

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Nomenclature

Aa: *Actinomyces actinomycetemcomitans*
AGEs: Advanced glycation end products
Av: *Actinomyces viscosus*

Ca: *Candida albicans*
CRP: C-reactive protein
CT: Connective tissue

EAO: European Association for Osseointegration

Fn: *Fusobacterium nucleatum*

HbA1C: Glycated hemoglobin

IDRA: Implant Disease Risk Assessment
IL-1α: Interleukin 1α
IL-1β: Interleukin 1β
Il-4: Interleukin 4
IL-6: Interleukin 6
IL-8: Interleukin 8

JE: Junctional epithelium

KMW: Keratinized mucosa width

MBL: Marginal bone loss
MMP: Matrix metalloproteinases

N: number
Ncm: Newton-centimeter

OE: oral epithelium

Pa: *Pseudomonas aeruginosa*
PD: Pocket depth
Pi: *Prevotela intermedia*
PI: Peri-implantitis
PIH: Peri-implant health
PIM: Peri-implant mucositis
Pg: *Porphyromonas gingivalis*
Pn: *Prevotella nigrescens*

RAGEs: Receptors for advanced glycation end products

Sa: *Staphylococcus aureus*
SPT: Supportive periodontal therapy

Tf: *Tannerella forsythia*
TNF*α*: Tumor necrosis factor-*α*
Td: *Treponema denticola*

Y: Years

CHAPTER 1

1. Introduction

The demand and the application of dental implants as a restorative procedure has increased over the years. Over 2 million implants are reported to be placed in the United States annually (Cosgarea et al., 2019). There is limited data reported in South Africa regarding the number of implants placed as well as the survival rates (i.e. the implants surviving or existing in the mouth). The estimated survival rate of implants that are in function for up to 20 years is reported to range from 90% - 95%. The cumulative survival rates of implants in systemically healthy individuals after 25 years were reported to be 83.8% and 96.1% after a period of 10 years. The cumulative survival rate in medically compromised patients after 20 years is 90.8% (Cosgarea et al., 2019). Despite these high long-term reported implant survival rates, there is an increase in patients exhibiting peri-implant diseases. The presence of peri-implant disease leads to various negative outcomes, including but not limited to discomfort, escalated expenses due to the need for supplementary treatment, and potential loss of the implant if untreated (Charalampakis et al., 2012; Daubert et al., 2015; Renvert & Quirynen, 2015; Cosgarea et al., 2019).

The increasing number of dental implants placed worldwide, can potentially contribute to an increase in patients presenting with technical, aesthetic and biological implant complications. These complications may contribute to failure to maintain bone-to-implant contact, which can ultimately lead to implant failure and loss. The most frequent implant complication is peri-implantitis (PI) (Charalampakis et al., 2012; Daubert et al., 2015; Renvert & Quirynen, 2015).

Peri-implant diseases demonstrate similar (not identical) characteristics to periodontal disease conditions affecting the gingiva, such as gingivitis and periodontitis. Peri-implant mucositis (PIM), if treated, is reversible, but can potentially progress to peri-implantitis if left

untreated. Peri-implantitis **progresses with more rapid destruction of bone and soft tissues, compared to periodontitis**. Therefore, early diagnosis and treatment should be the main objective to prevent implant complications and future costly interventions (Derks & Tomasi, 2015).

Various nonsurgical and surgical therapy protocols for peri-implant disease management have been suggested. Management of PI is challenging; remission is rarely the long-term clinical result; recurrences happen; and in certain instances, depending on the degree of bone loss, the implant may need to be removed. This demonstrates how crucial preventative measures and supportive therapies are for individuals receiving dental implants. The prevention strategy needs to be used in conjunction with approaches that deal with any modifiable risk factors (e.g., smoking, diabetes etc.) that patients may have (Romandini, Lima, et al., 2021).

The European Association for Osseointegration (EAO) Consensus Conference in 2012 reported estimated prevalence rates after at least 5 years of use at implant and subject for PIM level to be 30.7% and 63.4%, and for PI to be 9.6% and 18.8% respectively. The EAO also proposed that one in every five individuals would develop PIM within five years following of receiving an implant (Charalampakis et al., 2012; Daubert et al., 2015; Renvert & Quirynen, 2015; Klinge et al., 2012; Atieh et al., 2013).

Prior to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, 7 case definitions for peri-implant disease existed (Lee et al., 2017). Thus, studies reporting the frequency of peri implant health (PIH) and disease varied significantly as result of heterogeneity of study designs, inconsistency of case definitions utilized, different units for analysis, differences in function time of the implants and the absence of extent and severity of disease reporting. In these studies, no consistent threshold for alveolar bone loss and pocket depths were utilized. As reported in studies by Cosgarea,

Muñoz Giraldo and Renvert et al., the prevalence values of PI by implant and subject were reported to be 11% and 34% for pocket depths (PDs) greater than or equal to 4 mm; 10% and 12% for PDs greater than or equal to 5 mm; and 10% by implant and 18% by subject for PDs greater than or equal to 6 mm respectively (Cosgarea et al., 2019; Muñoz Giraldo, 2018; Renvert et al., 2018). Furthermore, other studies examined data using the implant as the only unit of analysis, leaving out the affected patients. Between 12% and 43% of implants were estimated to have peri-implantitis after ten years. Due to the differing case definitions from the various reporting groups, these estimates differed greatly. Future peri-implant health (PIH) and disease studies will become more consistent if the case definitions provided by the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions are utilized. Pooling data collected from studies applying standardized case definitions will provide a more precise estimation of the worldwide impact of PIH and disease (Cosgarea et al., 2019; Muñoz Giraldo, 2018; Renvert et al., 2018).

Many studies reported on limited convenience samples. In addition, data was analyzed only based on the implant as the only unit of analysis, not including the affected patients (Derks et al., 2016; Derks & Tomasi, 2015; Romandini, Berglundh, et al., 2021).

Convenience samples were employed in the majority of epidemiological studies reporting on the frequency and associated factors of PIM and PI. Thus, a skewed representation of risk factors may be reported in these studies, supporting the need for studies using representative samples from both private and university settings (Derks et al., 2016; Derks & Tomasi, 2015; Romandini, Berglundh, et al., 2021).

In summary, the recommendations for future epidemiological studies determining the occurrence of PIH and disease include, using the most currently prescribed case definitions, using representative samples rather than convenience samples, describing the prevalence

together with the severity and extent of diseases, and describing the function time of the implants (Derks et al., 2016; Romandini, Berglundh, et al., 2021).

Preventive strategies that address peri-implant diseases can only be developed appropriately when there is thorough knowledge of the prevalence of disease based on standardized assessment protocols and case definitions (Derks & Tomasi, 2015).

The purpose of this cross-sectional study is to investigate the prevalence of PIH and disease in patients who received dental implants during the period 2000-2019 in a university representative sample (Department Oral Medicine and Periodontics, Faculty of Dentistry, University of the Western Cape).

CHAPTER 2

2. Literature Review

The ability of clinicians to recognize peri-implant diseases is only achievable when equipped with the knowledge and comprehension of the clinical characteristics of the tissues around implants. During wound healing tissues develop around the osseointegrated implants. These tissues are composed of two compartments, namely, the soft tissue (i.e., mucosa) and hard tissue (i.e., bone). The basic function of the hard tissue (i.e., bone) is to establish a direct connection against the surface of the implant thus supporting and stabilizing the implant - by a process referred to as osseointegration. The primary role of the peri-implant soft tissue (mucosa) is to maintain the integrity of the bone around the dental implant by protecting it, and to form a soft tissue seal around the implant prosthesis (Araujo & Lindhe, 2018).

2.1 Characteristics of the Peri-implant Mucosa

Peri-implant mucosa has some common features with the mucosa around teeth. The reduced cellularity and vascularity in the peri-implant connective tissue makes the tissues to be more susceptible to disease compare to the mucosa around teeth. Adaptation of the epithelium and connective tissue around the implant during wound healing is influenced by many factors including the implant design features and surface characteristics (Ivanovski, 2018).

Several studies reveal structural and histological similarities between mucosa around teeth (gingiva) and mucosa around implants (peri-implant mucosa) (Schupbach & Glauser, 2007; Araujo & Lindhe, 2018; Ivanovski & Lee, 2018). The nature of the implant surface fixture and abutment connection differs between implant systems, which will impact the characteristics of the implant soft tissue (CT) abutting the implant and abutment structure. Histological features demonstrate **suboptimal characteristics** of mucosa around implants compared to that demonstrated around teeth (Guo et al., 2021).

Peri-implant mucosa is between 3mm and 4 mm in vertical dimension, and the length of the epithelium is 2 mm. Healed mucosa around implants have a CT which is 85% matrix

elements and collagen fibers, 3% fibroblasts, and 5% vascular elements. The oral epithelium is orthokeratinized, while the coronal epithelium facing the implant is thin. At the portion of the implant's coronal surface facing the CT, there is contact between the surface of the implant and CT (Araujo & Lindhe, 2018).

The epithelium in the peri-implant sulcus **bears resemblance to that around teeth**, with shedding occurring in the outermost cell layer allowing removal of attached bacteria together with degenerating cells. The epithelium in the sulcus has intercellular spaces that are sealed tightly and possess many desmosomes; these contribute to the poor permeability. The sulcular epithelial cells have numerous cytoplasmic extensions that allow it to stretch. Several hemidesmosomes are seen at the surface between these extensions. The desmosomal connections to the adjacent cell layer become less pronounced during desquamation of the superficial cells (Ivanovski & Lee, 2018).

The peri-implant junctional epithelium is made up of 2 strata, the basal layer interacting with the CT is composed of cuboidal cells, and a basal lamina possessing both fibrils and desmosomes is formed towards the CT. The junctional epithelium (JE) along the implant is composed of uniform, flat cells (Schupbach & Glauser, 2007).

A basal lamina with numerous hemidesmosomes is found at the surface of the deepest cell layer, which adheres to the implant directly. The coronal JE had expanded intercellular spaces, some of which are inhabited by neutrophilic granulocytes. Studies have demonstrated significantly weaker strength of the “internal basal lamina” and the hemi-desmosomes, the adhesive structures located the at transmucosal region around the implants compared to those around teeth (Guo et al., 2021).

The regenerating JE around implants develops from the oral mucosa epithelium, while the reduced enamel epithelium is responsible for the development of JE surrounding teeth. The structural properties of both these JE is the same. Thus, the junctional epithelium around

implants also serves to provide peripheral defense. The cellular turnover of peri-implant JE is twice that of the JE surrounding teeth. The apical sulcus epithelium has a high frequency of desquamation, reported to be fifty-fold greater compared to that of the oral epithelium, thus hindering bacteria from colonizing the sulcus. The JE is entirely recovered within 5 days following mechanical disruption (such as probing around implants) (Schupbach & Glauser, 2007).

Polymorphonuclear leukocytes migrate through the enlarged intercellular spaces of the peri-implant JE to establish a barrier at the sulcus base hindering pathogens from accessing the epithelium. Leukocytes that encounter bacteria are activated resulting in them releasing contents of their granules as well as engulfing the pathogens. The enzyme-rich lysosomes further offer defense by eradicating bacteria (Schupbach & Glauser, 2007).

The blood supply of the mucosa surrounding implants originate from the large supraperiosteal blood vessel on the alveolar ridge. This vessel gives branches to the supra-alveolar mucosa and forms capillaries beneath the oral epithelium as well as the vascular plexus lateral to the JE. This **plexus adjacent to the JE of mucosa** surrounding implants is sometimes referred to as crevicular plexus and has the same location and composition as that in gingiva around teeth (Lindhe et al., 2015).

There are two different vascular sources of the gingiva (around natural teeth), the first is the large supraperiosteal blood vessels and the second is the vascular plexus of the periodontal ligament. The supraperiosteal blood vessels has branches that form:

1. The capillaries of the connective tissue papillae. These are located under the oral epithelium
2. The vascular plexus. These are located lateral to the junctional epithelium. (Lindhe, 2015)

The transmucosal portion of an implant with a prosthesis resembles that of teeth, these include the components of the JE and CT. The key difference is the relationship between the surface of the implant and the CT. In teeth the fiber attachment directly inserts at right angles into the cementum, while in implants the collagen fibers adapt in an orientation that is parallel to the implant, with no true physiological attachment to the implant. There is also reduced vascularity and cellularity in the connective tissue of implants. These combined features compel the tissues to be highly susceptible to the onset and persistence of peri-implant disease, highlighting the need for regular implant monitoring and maintenance (Ivanovski & Lee, 2018).

2.2 Probing Periodontal and Peri-implant Mucosa

Research investigating the PD around teeth and implants in dogs using a force of zero point five Newtons, using the Electronic Periodontal Probe (model 200, Vine Valley Research, NY, USA) revealed a slight compression of gingival tissue while the peri-implant mucosa was not only compressed but also dislocated laterally. The tip of the probe, at sites with healthy gingiva (around teeth), stopped coronally to the apical cells of the JE. At implant tissues it entered the apical portion of JE that is dislocated laterally, ending adjacent to the alveolar bone. The average PD was deeper around implants than teeth. In this study, the deeper penetration around the implant sites was associated with and linked to the probing force that are high, which was suitable for soft tissue around teeth. The peri-implant soft tissue has less resistance compared to gingiva (around teeth) to probing forces. There are studies performed employing probing forces lesser than 0.5N, these demonstrated that the soft tissue composition and attachment attribute to deeper penetration (Ericsson, 1993; Ivanovski & Lee, 2018).

In another study a probing force of 0.2N was applied and it resulted in entry to identical depths in relation to bone crest at healthy tooth and implant. The PDs are affected by the

peri-implant CT density. The probe tip stops more apical in implants than teeth when there is inflammation (Ivanovski & Lee, 2018; Lang et al.,1994).

There was previously controversy around peri-implant probing, the concern was that it could compromise the soft tissue seal as well as the adhesion integrity at the implant-mucosa interface. It was established through studies that the regeneration of the soft tissue seal and adhesion reaches completion at 5 – 7 days after probing around implants (Araujo & Lindhe, 2018; Etter et al., 2002).

2.3 Keratinized mucosa around implants

Keratinized mucosa width (KMW) around implants refers to the apico-coronal dimension of keratinized mucosa, extending from the mucogingival junction to the margin of the mucosa around the implant. The presence of KMW aids in the maintenance of natural architecture and colour of peri-implant soft tissue, and it prevents mucosal recession and its progression. It also reduces the risk of inflammation of the peri-implant tissues (Wang et al., 2021).

Controversy exists with regards to the amount of keratinized mucosa required for the maintenance of peri-implant health. There are studies that demonstrated an association between plaque accumulation and mucosal inflammation to be most frequently observed around sites with keratinized mucosa less than 2mm. Other studies demonstrated no relationship between a lack of keratinized mucosa with inflammation of the peri-implant mucosa (Araujo & Lindhe, 2018; Boynueğri et al., 2013; Wennström et al., 2012).

2.4 Case Definitions

Case definitions with specific diagnostic criteria were described in the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. These definitions include diagnostic criteria in the absence of previous clinical and radiographic data. PIH is defined as a peri-implant tissue that is characterized by the absence of

inflammation, namely, swelling or edema, erythema, bleeding on probing, and suppuration. PIM is defined as an inflammatory lesion that is reversible, with the inflammation limited to the soft tissue surrounding the implant and does not extend or involve bone. This pathologic condition is biofilm associated. It has characteristics that are similar to those of gingivitis (Alani & Bishop, 2014; Berglundh et al., 2018; Caton et al., 2018)

PI is a plaque associated inflammatory process that involves the soft tissue and alveolar bone. It is similar (not identical) to periodontitis with regards to clinical presentation and pathogenesis. Soft tissue recession can occur accompanied by exposure of implant threads in peri-implantitis. Soft tissue enlargement, especially in non-keratinized mucosa, may develop. The case definition of PI in instances where the baseline data (clinical and radiographic) is not available has been described. In the absence of baseline data, PI is diagnosed when there is suppuration or bleeding on probing, presence of peri-implant pocket depths that are ≥6 mm, accompanied by bone loss presenting as a bone level that is ≥3 mm apical to the most coronal intra-osseous portion (Berglundh et al., 2018; Romandini, Berglundh, et al., 2021).

2.5 Clinical characteristics of peri-implant diseases

The clinical characteristics of inflammation are necessary for a diagnosis of peri-implant disease. These include edema, erythema, and bleeding on probing (BOP). The main clinical characteristic necessary for the diagnosis of PIM is BOP. Edema of the peri-implant soft tissue may not always be present. The case definition for PI is the presence of bleeding and/or suppuration on probing with a force of 0.25 Ncm, probing pocket depth that is increased accompanied by bone loss that is visible on a radiograph (Berglundh et al., 2018; Romandini, Berglundh, et al., 2021).

Soft tissue enlargement, especially in non-keratinized mucosa, may develop. Soft tissue recession can also occur accompanied by exposure of implant threads in PI (Alani & Bishop, 2014).

2.6 Radiographic features

The radiographic presentation of PIM varies. A funnel or saucer shaped bone loss pattern is demonstrated in cases where the bucco-lingual or bucco-palatal ridge is wide. In instances where the alveolar bone is thin the bone loss follows a horizontal pattern. A circumferential bone loss pattern is evident when these sites are explored surgically (Alani, Kelleher & Bishop, 2014; Schwarz et al., 2018)

2.7 Risk Factors

Multiple risk factors are attributed to the development and progression of peri-implant disease. These factors include the history of periodontal disease, smoking, presence of uncontrolled diabetes mellitus, the general health status of the patient, adequate oral hygiene measures and their motivation to attend regular plaque control and supportive therapy. The local risk factors include the implant surface, implant position (anterior/posterior), accessibility for adequate oral hygiene, excess cement, and deepening of the pockets. The association of these risk factors (e.g. implant surface, implant position, accessibility for adequate oral hygiene, excess cement, and deepening of the pockets) with PI have been described in several studies investigating the prevalence of peri-implantitis (Renvert & Quirynen, 2015; Schwarz et al., 2018; Ogata, 2020).

Biofilm accumulation results in inflammation around teeth and implants depending on the susceptibility of the host as well as the presence of various risk factors. The presence of risk factors substantially impacts the tissue stability around teeth and implants negatively, resulting in elevated susceptibility to inflammation in the presence of biofilm accumulation (Colombo & Tanner, 2019; Monje et al., 2017). History of periodontitis, smoking,

uncontrolled diabetes, and lack of supportive therapy have been implicated as risk factors affecting the long-term prognosis of implants (Karoussis et al., 2003). Other factors associated with peri-implant disease include pockets, and time of function (Apatzidou, 2022). An understanding of risk factors associated with peri-implant disease, equips practitioners with the ability to select patients for implant placement applying evidence-based practice, to focus on modifying the risk factors prior to implant placement, and make informed decisions that will promote long-term implant survival and success (Daubert et al., 2015).

2.8 Generalized Factors

2.8.1 Systemic Factors

Chronic conditions that affect the turnover of bone are considered as risk factors for PI. These would include osteoporosis and radiotherapy. Patients with poor control of blood glucose are also at a higher risk to develop PI (Alani, Kelleher & Bishop, 2014; Daubert et al., 2015)

2.8.2 History of Periodontitis

Periodontitis is a plaque-related disease. Periodontal pathogens can translocate from their niches, including the tongue, periodontal pockets, and saliva, to the peri-implant tissues. Individuals with a history of periodontitis are at a higher risk to marginal bone loss around implants, and implant failure (Renvert & Quirynen, 2015)

2.8.3 Smoking

Smoking impairs both the innate and adaptive host responses. Gingival blood flow and vascularity in patients that smoke is suppressed. Wound healing is also impaired in these individuals and inflammation is also masked. Signs of inflammation are thus masked; this is inclusive of BOP and erythematous mucosa. There is a controversy with regards to smoking being a risk factor for peri-implantitis. Some studies have reported no significant difference

in the incidence of peri-implantitis between non-smokers and smokers. (Costa et al., 2012; Marrone et al., 2013; Karroussis et al., 2003) Other studies have reported on a clear association between smoking and PI (Renvert & Quirynen, 2015; Hinode et al., 2006; Chen et al., 2013; Sgolastra et al., 2012)

2.8.4 Maintenance therapy

Supportive periodontal or maintenance therapy is performed in order to maintain tissue health. It should be tailor made for the patient considering the history of periodontitis, prosthetic design, the patient's ability to properly perform oral hygiene, and their systemic condition. Compliance with supportive therapy reduces the risk of biological complications, including peri-implant disease. Patients with a history of periodontitis complying with a maintenance program have demonstrated maintenance of PIH over a period of 10 years (Renvert & Quirynen, 2015).

2.9 Local Factors

2.9.1 Poor Plaque Control

Microorganisms are responsible for the initiation of the inflammatory response mediated by the host. Any factor that favors the accumulation of biofilm increases the risk of peri-implant disease development and progression. The prevalence of peri-implant disease in patients with a history of periodontitis was greater when the plaque score was >25% (Heitz-Mayfield et al., 2018; de Avila, van Oirschot & van den Beucken, 2020).

2.9.2 Excess Cement

Cement retained prostheses are used as an alternative to screw retained prostheses, and their use is frequently found to result in excess cement left in the peri-implant tissues even with careful clinical control. Studies have demonstrated that the deeper the position of the crown margin the greater the risk of undetected cement. Radiographic evaluation is not predictable in the detection of cement. Cement in the peri-implant tissues provokes an inflammatory

response as it acts a foreign body. The inflammatory response results in the development and progression of peri-implant disease. The excess cement also encourages microbial colonization resulting in peri-implant disease development (Daubert et al., 2015; Renvert & Quirynen, 2015).

2.9.3 Implant Surface

A rough, hydroxyapatite-coated surface in combination with lack or absence of keratinized gingiva is associated with a greater risk of PI (Hussain, Miloro & Cohen, 2021).

2.9.4 Implant Location

Implant diameter was suggested to be a risk factor for peri-implant disease. It is rather the position of the implant that should be considered. Posterior implants have been reported to have greater bone loss (Daubert et al., 2015; Hussain, Miloro & Cohen, 2021). Reasons for this were not expanded on, however that this may be related to accessibility constraints when the patients perform oral hygiene practice. This would in turn increase microbial colonization at the site and increase the risk of peri-implantitis.

2.10 Microbiology of peri-implant diseases

The main cause of peri-implant disease is the bacterial challenge in the peri-implant tissues. Peri-implantitis is caused by a mixture of anaerobes, and its composition is similar to that described in the previously classified chronic periodontitis cases. These microorganisms include *Tannerella forsythia (Tf), Porphyromonas gingivalis, Treponema denticola, Prevotella nigrescens (Pn), Prevotella intermedia (Pi),* and *Fusobacterium nucleatum.* Other microorganisms that are found in peri-implantitis are *Staphylococcus aureus (Sa), Enerobacteriaceae, Candida albicans (Ca),* and *Pseudomonas aeruginosa (Pa)* (Renvert & Quirynen, 2015). The microbial samples from peri-implant pockets > 5 mm (in peri-implantitis) demonstrate high levels of spirochetes, while in those from peri-implant pockets < 5 mm the coccoid microbiota were predominating. Microbial samples from failing

implants had an abundance of fusiform bacteria, spirochetes, and motile rods, while implants with peri-implant health demonstrated few rods and coccoid microbiota. The organisms that are at high levels are *Prevotela intermedia (Pi),* and *Fusobacterium* species. There are cases where *Actinomyces actinomycetemcomitans (Aa), Captnocytophaga* species, staphylococci and non-pigmented *Bacteroides* species were detected. *Actinomyces viscosus (Av) and Actinomyces actinomycetemcomitans (Aa)* were detected more in supragingival biofilm on teeth than implants (Mombelli & Décaillet, 2011).

2.11 Aetiopathogenesis of peri-implant diseases

The disease process in PIM and PI is similar but not identical to that of gingivitis and periodontitis, respectively. The most important etiologic agent identified in the initiation and progression of peri-implant disease is the bacterial biofilm (Mombelli, 2018; Schwarz et al., 2018; Heitz-Mayfield, Heitz & Lang, 2020).

The implant surface is coated by proteins from plasma and saliva immediately after placement. This protein adsorption forms a layer which becomes the main mediator of adhesion by bacteria on the surface. The initial colonizers are mainly the *Streptococcus* species. Co-aggregation of different species occurs and the interaction between the different species promotes accumulation of the biofilm. The biofilm formation and accumulation are not limited only to the implant surface, it extends and invades the peri-implant soft tissues. The action of microorganisms results in the release of enzymes, toxins, and lipopolysaccharides. The presence and accumulation of biofilm provokes an inflammatory response. The inflammatory response changes the environment resulting in the microbiological shift to anaerobic gram-negative and proteolytic bacteria (Souza et al., 2021) The severity of peri-implant disease depends on the host response to the microbial dysbiosis. Several factors were evaluated in order to ascertain their correlation to submucosal dysbiosis, these include marginal bone loss (MBL), peri-implant BOP and PD. The factor that was

proven to correlate with submucosal microbial dysbiosis was an increase in MBL (Shi et al., 2022).

The inflammatory response and bacterial activity results in the break of mucosal seal to the implant surface, and this promotes formation and further accumulation of biofilm. The inflammatory response is associated with signs of inflammation. It is assumed that PIM precedes PI (Lang et al., 2011; Schwarz et al., 2018).

There is a release of pro-inflammatory mediators (such as interleukin (IL) 1α, IL-1β, tumor necrosis factor α, IL-8 etc.) resulting in gene expression signaling, and mobilization of inflammatory cells. The mobilized inflammatory cells release enzymes such as collagenases, proteolytic enzymes such as metalloproteinases, which are responsible for degradation of the CT (Lang et al., 2011).

The triggered inflammatory response results in a lesion that is lateral to the barrier epithelium and a healthy CT zone separates the lesion from the crestal bone. There is marked proliferation of epithelium into the collagen-reduced inflammatory infiltrate and sparse inflammatory cells adjacent to the connective tissue that is collagen rich. The inflammatory cell infiltrate lateral to the barrier epithelium is a B and T cell-dominated infiltrate.

Histologic analysis of soft tissue around implants following 16 weeks of biofilm formation demonstrated a distance varying between 1.0 and 1.9 mm between the apical portion of the inflammatory cell infiltrate and the crestal bone. As the disease progresses more of the subepithelial CT becomes infiltrated resulting in the reduction of the healthy connective tissue zone. The apical extension of the inflammatory infiltrate is apical to the pocket epithelium in PI, and it is more pronounced in PI compared to periodontitis lesions. The exact mechanisms leading to the apical extension of the infiltrate and crestal bone loss is not yet determined (Berglundh et al., 2011; Schwarz et al., 2018).

The erythema of the peri-implant tissue arises because of the enlargement of blood vessels in the sub-epithelial CT. Loss of keratinization also contributes towards the erythematous appearance of the peri-implant tissue. The interfibrillar substance in the CT becomes semi liquid resulting in the swelling and reduced firmness of the peri-implant tissue. Another contributing factor is the loss of fibrous CT (Lang et al., 2011).

The PI lesion contains more neutrophils and a greater portion of B cells compared with peri-implant mucositis. The PI soft tissue lesions are predominated by lymphocytes and plasma cells and characterized by the large number of macrophages and leukocytes. The size of these soft tissue lesions under histology is two times greater than those seen at periodontitis lesions. Although the periodontitis and PI lesions are somewhat similar there is a characteristic greater number and densities of macrophages, neutrophils and plasma cells, increased vascular structure density lateral to and outside the cellular infiltrate in peri-implantitis lesions. The neutrophils are not only located in the pocket epithelium but are also in the central peri-vascular compartments. Large-sized bacteria are present in the apical portion of the inflammatory cell infiltrate that faces the pocket. The lesions also have a dominant IL-1α, which is an osteoclast activating cytokine. There is also a large number of IL-6, and a smaller number of TNF-α in PI lesions compared to periodontitis (Berglundh et al., 2011; Schwarz et al., 2018).

The inflammatory lesion results in destruction of tissue. Greater breakdown is seen at tissues around modified surface implants compared to implants with non-modified surfaces. Tissue destruction is partly due to the microorganism action and to a greater extent due to the host response (Lang et al., 2011; Schwarz et al., 2018).

2.12 Peri-implant disease and periodontal disease

PIM and PI share similarities with gingivitis and periodontitis respectively, in clinical features and etiology, and distinction in histopathology features. Peri-implant inflammation is regarded to be similar around teeth although there are some differences. Periodontitis is described as a periodontal destructive process that is plaque-initiated, mediated by the host, and influenced by factors that are modifiable and non-modifiable to result in matrix degradation and bone destruction. PI is similar. Destruction of tissue in PI is reported to be more severe and faster in contrast to periodontitis (Monje et al., 2017; Salvi et al., 2017). PI sites possess an inflammatory infiltrate that is larger than that in periodontitis sites. The infiltrate comprises greater plasma cells, neutrophils, and macrophages compared to periodontitis sites. The expression of tumor necrosis factor-α (TNF-α), interleukin 6 (IL-6), interleukin 8 (IL-8) is higher in peri-implantitis than in periodontitis. The prime immune response orchestrators that are strongly associated with peri-implant mucositis and gingivitis are interleukin 1α (IL-1α) and interleukin 1β (IL-1β). Lactoferrin is associated with gingivitis only, this is supported by the findings of elevated activation of neutrophils and the increase in cells that are elastase-positive in the junctional epithelium of gingivitis sites. There is a stronger induction of IL-1α, and IL-1β in the early stages of gingivitis compared to PIM. PIH sites possess elevated crevicular fluid cytokines compared to gingival health sites (Schincaglia et al., 2017).

2.13 Management of Peri-implant Disease

The first step in the management of peri-implant disease is evaluating the clinical and radiographic features to determine a diagnosis based on the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions (Berglundh et al., 2018). The interventions performed are dependent on the peri-implant disease diagnosis. The first phase of intervention starts with initial therapy (or nonsurgical therapy) for both peri-

implant mucositis and peri-implant mucositis, and this is followed by surgical therapy in the case of PI when indicated. During the initial or nonsurgical therapy, the clinician needs to identify, eliminate or modify the causative factors, reduce pathogenic micro-organisms and reduce infection. Non-surgical management includes prosthesis evaluation, removal and replacement of bulky prosthesis that are not accessible to patient oral hygiene practices. The treatment of PIM entails mechanical therapy with or without disinfection or use of an antiseptic. Nonsurgical therapy alone in the case of peri-implantitis has been reported to result in limited clinical improvement (Fu & Wang, 2015; Karlsson et al., 2019; Renvert & Polyzois, 2015). This appears to be caused by the restricted access to the implant surface, which makes it challenging to properly clean the contaminated implant surface. The implant surface unlike the mucosa is non-shedding thus requiring decontamination once contaminated (Schwarz et al., 2022). As a result, surgical interventions are frequently needed. The recommended management for PI is nonsurgical therapy in combination with surgical interventions. Surgical interventions are critical for accessibility and better assessment of the peri-implant defect, optimal debridement of the implant surface and the defect, and for promoting potential re-osseointegration as well as recreation of a peri-implant mucosal seal. The therapeutic options in surgical intervention include access flaps, resective techniques, regenerative techniques, and explantation (Fu & Wang, 2015; Karlsson et al., 2019; Renvert & Polyzois, 2015)

2.14 Supportive Implant Therapy

Regular supportive therapy is necessary for achieving a positive therapy outcome. The time intervals for supportive therapy varies from three monthly to six monthly. It is based on the risk assessment of the individual patient (Roccuzzo et al., 2018).

The regularity of supportive therapy relies on the risk factors associated with developing peri-implant disease as well as the degree and severity of periodontal disease if present (Smith et

al., 2017). There is a proposal to decrease the supportive intervals for individuals who have a prior history of periodontitis to less than 6 months, in contrast to those with a history of gingivitis. Research has indicated that different time frames have been utilized in various studies, such as an interval of 2 weeks, 2-3 monthly interval, a 3-4 monthly interval, a 3-6 monthly interval, a 4-6 monthly interval, and an interval of up to 18 months. Data provides evidence in favor of engaging in supportive therapy sessions on a minimum of four occasions annually (Renvert & Rutger Persson, 2004; Trombelli et al., 2020).

Tailored supportive therapy intervals can be determined for individual patients based on several factors, including the number of teeth or implants, patient cooperation, oral hygiene efficacy and compliance, systemic health, previous frequency of supportive therapy, instrumentation access, history of disease or complications, and distribution and depth of sulci (Position Paper Periodontal Maintenance, 2003; Trombelli et al., 2020). The periodontal risk assessment tool can be used to work out the intervals (Lang & Tonetti, 2003).

2.15 Risk assessment tool

Multiple factors are attributed to development and progression of peri-implant disease. Eight parameters have been incorporated in the Implant Disease Risk Assessment tool (IDRA)(see figure 1), namely:

1. History of periodontitis
2. Bleeding on probing percentage
3. Pocket depths ≥5 mm
4. Bone loss in relation to the age of the patient
5. Susceptibility to periodontitis as mentioned on the Classification of Periodontal and Peri-implant Disease 2017 World Workshop
6. Supportive periodontal therapy

7. Implant restorative depth
8. Factors related to the prosthesis

The IDRA assists with the comprehensive evaluation of the risk factors listed above, and it is used to identify the risk status of the patient and the factors that increase the risk of developing peri-implantitis. The clinician needs to bear in mind that control or modification of these factors can minimize the chances of developing disease (Heitz-Mayfield, Heitz & Lang, 2020).

2.15.1 History of periodontitis

Individuals with a history of periodontitis are at a higher risk of developing PI. It is important that the clinician determine the reason for previously extracted teeth. Individuals with a history of extractions due to periodontitis are at a high risk while those who had extractions because of trauma or caries are at low risk of PI (Heitz-Mayfield, Heitz & Lang, 2020).

2.15.2 Bleeding on probing percentage

The presence of bacterial biofilm is an important etiologic agent in the development of peri-implant disease. The response of the host to the biofilm results in inflammation, which is identified as redness, edema, suppuration and bleeding on probing (BOP). The BOP score is a reflection of active disease and is associated with progression of disease. A number of studies reported a higher risk for progression of disease in patients with the bleeding on probing score between 20% - 30%, thus it is used in the IDRA tool as a second risk indicator. Individuals with the score of >25% are considered to have a higher risk for peri-implant disease (Heitz-Mayfield et al., 2020; Karlsson et al., 2019).

2.15.3 Pocket depths ≥5 mm

Periodontal pockets (PD) that are ≥5 mm deep are associated with an increased risk of peri-implant disease. Periodontal pathogens from pockets around teeth can colonize the implant sites. Patients who present with one or two pocket depths of ≥5 mm are categorized as low risk. Individuals with pocket depth of ≥5 mm at more than six sites are categorized as high risk (Heitz-Mayfield et al., 2020).

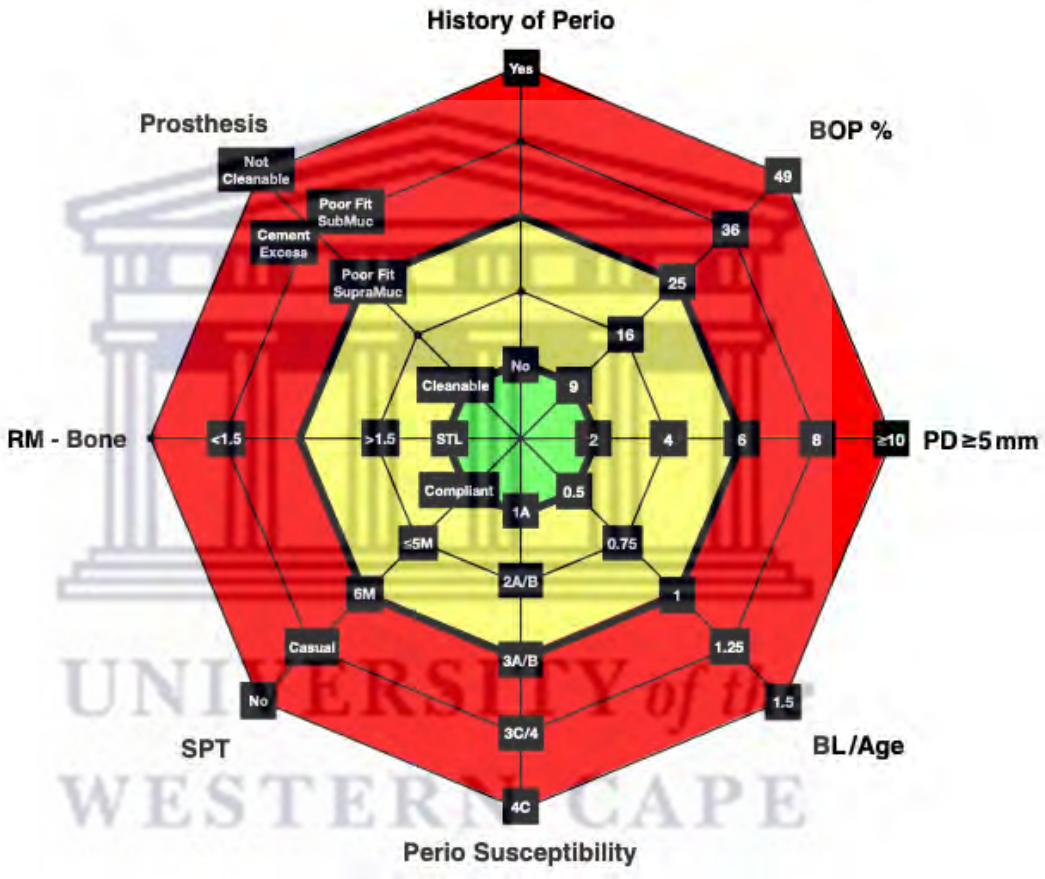

Figure 2.1 Implant Disease Risk Assessment (IDRA) functional diagram (Heitz-Mayfield et al., 2020)

2.15.4 Bone loss in relation to the age of the patient

Bone loss in relation to the patient's age indicates periodontal disease progression. Bone loss in relation to the patient's age is calculated by first measuring in millimetre (mm) the bone loss in the worst affected site using a periapical radiograph and calculated in percentage of

the length of the root. The percentage of bone loss is then divided by the patient's age (Heitz-Mayfield et al., 2020).

2.15.5 Susceptibility to periodontitis

The staging and grading for periodontal disease influences the development as well as the progression of peri-implant disease. Stage I Grade A is considered low risk. Stage II is moderate or high risk, and also stage III is considered moderate or high risk. Stage IV is high risk. Grade C is high risk, while Grade B is moderate or high risk. Diabetes and smoking are not placed as separate vectors in the IDRA tool. These factors are included when staging and grading the patient (Heitz-Mayfield et al., 2020).

2.15.6 Supportive periodontal therapy

Regular supportive periodontal therapy (SPT) is associated with peri-implant health. Patients who do not undergo SPT are at a high risk of developing peri-implant disease. Patients who comply with a regular SPT program are at a low risk of developing peri-implant disease (Heitz-Mayfield et al., 2020).

2.15.7 Distance from the restorative margin to the bone

Individuals with a "distance of ≤1.5 mm" from the "restorative margin to the crest of the bone" are at a higher risk of developing peri-implantitis. Individuals with a soft tissue level implant are at a low risk, while those with a distance of 1.5mm are at moderate risk of developing PI. A radiograph is used to determine this distance (Heitz-Mayfield et al., 2020).

2.15.8 Factors related to the prosthesis

The contour and design of the prosthesis can result in difficulty for the patient in conducting oral hygiene practices and then subsequently result in the accumulation of biofilms. A prosthesis should be easily accessible to cleansing for both the patient and clinician. A

prosthesis that fits well, is screw-retained or having no excess cement, and is cleansable is regarded as low risk for development of peri-implant disease. A prosthesis that is poorly fitting, inaccessible for cleaning, and has excess cement is regarded as high risk. A prosthesis that has margins that are supramucosal, thus accessible, but has a fit that is compromised is regarded as a moderate risk (Heitz-Mayfield et al., 2020).

CHAPTER 3

3. Methods and Materials

3.1 Aim of the study

The aim of this cross-sectional study was to evaluate, in a university-representative/setting sample, the prevalence of peri-implant health and disease in patients who received implants during the period 2000-2019 in the Department Oral Medicine and Periodontics Clinic, Faculty of Dentistry, University of the Western Cape.

3.2 Rationale for the study

The results of the study will guide clinicians to the risk of their patients developing peri-implant disease based on the risk or protective indicators.

3.3 Research objectives

- To report on the demographic characteristics (age, diagnosis, and gender), type and number of implants placed per individual.
- To quantify the prevalence of peri-implant health and disease in the university sample.
- To compare the diagnosis of peri-implant disease to the predicted disease occurrence and risk according to the Implant Disease Risk Assessment Tool (IDRA).
- To identify the associated patient and implant related factors in relation to the diagnosis of peri-implant disease.

- To contrast the prevalence of peri-implant disease with the compliance with a supportive therapy or implant maintenance programme.

3.4 Materials and methods

3.4.1 Study Design

This is a cross-sectional study.

3.4.2 Study population

The study population comprised of patients who received dental implants during the period of 2000-2019 in the Department Oral Medicine and Periodontics Clinic, Faculty of Dentistry, University of the Western Cape.

3.4.3 Sample Size

The sample size calculation was based on the null hypothesis that the prevalence of peri-implantitis will be the same as 45.0% in Derks el al., (2016) and Romandini et al., (2020). The sample size of 45% prevalence is 381 with a finite sample of 1000 implants placed between 2000 and 2019, with a finite sample of 200 participants between 2000 and 2019. The sample size was 90 implants. Therefore, the sample would result in 80% power to reject the null hypothesis for a 10% threshold in prevalence difference and with an alpha level set at 0.05(Derks et al., 2016). All the eligible patients who consent to be part of the study, who received implants during the period of January 2000 - December 2019 were included in the study.

3.5 Eligibility Criteria

3.5.1 Inclusion criteria

- Implant(s) placed during the period January 2000 - December 2019 in the Department Oral Medicine and Periodontics Clinic, Faculty of Dentistry, University of the Western Cape.
- Implant(s) with at least 1 year of loading with an implant prosthesis.
- All age groups.
- Radiographs taken with the placement visit of the final prosthesis.
- Patient records with available data on treatment modality performed.
- Patients who consent to be part of the study.

3.5.2 Exclusion criteria

- Missing data on relevant clinical characteristics e.g., medical history, baseline radiographs, implant hardware data and non-loaded implants.

3.6 Data collection

The study was based on records and data collected between 2000 and 2019. Patients that had implant(s) placed during this period were identified from the Department Oral Medicine and Periodontics Clinic database. The records were screened to identify and verify those patients that met the inclusion criteria. The eligible patients were contacted by phone using a phone script. Patients who gave consent to be part of the study were given an appointment for the clinical and radiographic examination.

Participants consenting to the study had a clinical and radiographic examination at the Department Oral Medicine and Periodontics Clinic, Faculty of Dentistry, University of the Western Cape.

3.6.1 Data extracted from files and implant database

The eligible patients' records were obtained, and the demographic characteristics, relevant medical history, clinical presentation, detailed radiographic findings, implant details, use of

antibiotics at placement, use of bone/soft tissue graft before or at implant placement, type of bone/soft tissue graft, smoking status at the time of implant placement and frequency of the supportive periodontal maintenance since the placement of the implant(s) were extracted from the patients' files as noted in the data collection sheet (Sheet 1). The extracted data was entered in a Microsoft Excel spreadsheet which was kept confidential. The extracted data was used to perform the assessment of risk factors associated with peri-implant disease and the Criteria for peri-implant disease (Heitz-Mayfield, Heitz & Lang, 2020). Each eligible patient was anonymized with an assigned study number.

The researcher (Dr Mahlangu) was calibrated by the primary investigator (Dr Suné Mulder van Staden) to ensure reproducibility of clinical examination. The researcher evaluated 10 patients first, assessing probing depths, bleeding on probing, soft tissue clinical description and plaque index around either the upper or lower first molars. After a period of 15 minutes the primary investigator (Dr Suné Mulder van Staden) evaluated the same patient to assess the degree of agreement. After a period of 30 minutes the same patient was evaluated for a second time by the researcher (Dr Mahlangu) to assess intra-examiner reproducibility (Stander et al., 2019).

3.6.2 Data extracted from clinical and radiological evaluation

Examinations were performed by a calibrated researcher (Dr Mahlangu) in the Department of Oral Medicine and Periodontics, University of the Western Cape. Photographs were taken and included intra-oral photographs in occlusion, occlusal views, and implant(s) site.

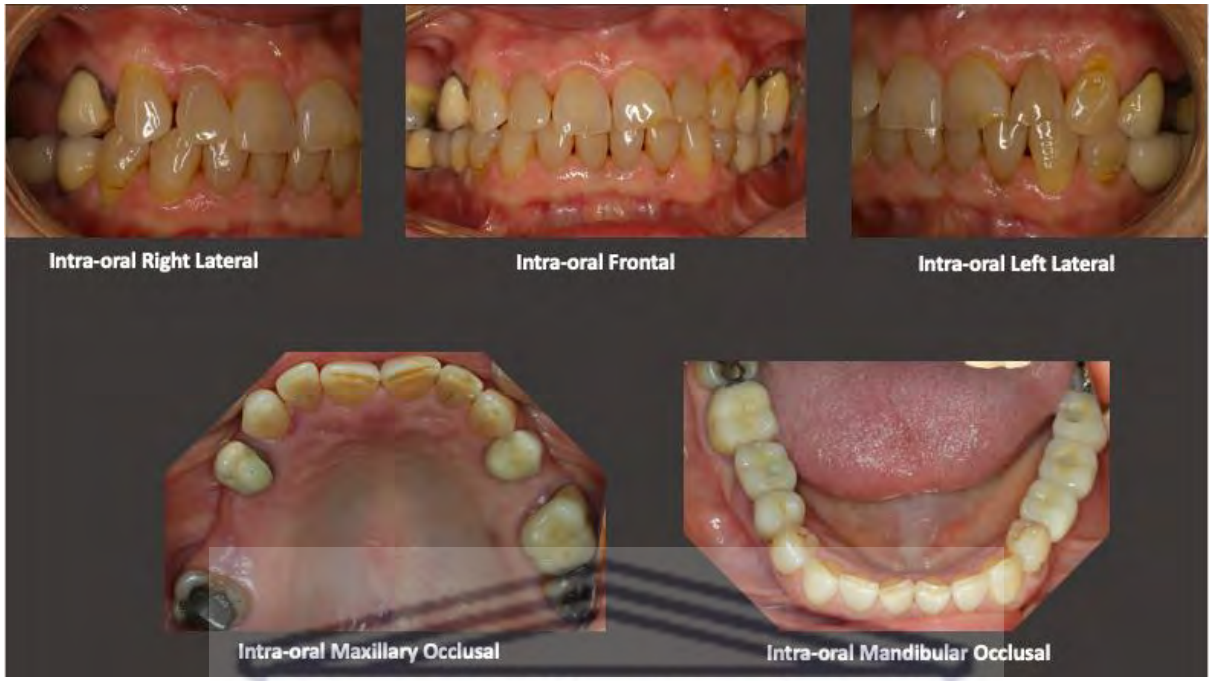

Figure 3. 1 Intra-oral pictures

A detailed medical history was obtained from each patient. The current status was verified by laboratory testing (HbA1C) with regards to all diabetic patients. The oral hygiene practice was obtained from each patient, and it included the brushing frequency, flossing frequency, type of toothbrush bristle used, type of floss used, type of mouthwash used and the frequency, use of other oral hygiene aids, and whether they had received oral hygiene instructions prior to implant therapy. The frequency of the supportive periodontal maintenance since the placement of the implant(s) was obtained from each patient and correlated with the patient records.

3.6.3 Clinical Parameters

A comprehensive periodontal examination was performed, and it included measuring the probing depths, gingival recession, mobility, bleeding on probing, plaque percentage, presence of suppuration, and excess cement around teeth and implants.

Edema was defined as abnormal swelling of the peri-implant tissues. Erythema was defined as redness of the mucous membranes. Tooth and implant mobility was defined as movement of either tooth or implant when a force was applied. Gingival recession was defined as marginal

soft tissue migrating apically to expose the cemento-enamel junction. All the above terminology was according to the Glossary of Periodontal Terms.

Probing depth around teeth

The periodontal pockets were assessed at six sites including the mesio-buccal, mid-buccal, disto-buccal, mesio-palatal/lingual, mid-palatal/lingual, and disto-palatal/lingual. The probing depth will be measure from the base of the pocket or sulcus to the gingival margin.

Probing around teeth

A graduated periodontal probe (UNC-15, Hu Friedy Group,Chicago, United States of America) with 1mm increments up to 15mm was used to measure probing depth at six sites per tooth.

Probing around implants

The probing was performed using a plastic probe (PerioWise Probe, Premier Dental Dental Products, Pennsylvania, United States of America) passed parallel to the implant or tooth long axis at 0.25 Ncm at six sites. Probing depth was measure at six sites per implant using a graduated plastic probe calibrated at 3mm, 6mm, 9mm and 12mm.

Assessment of soft tissue around implants

Description of the peri-implant tissues included clinical presentation (presence or absence of edema and erythema), presence or absence of keratinized mucosa, presence of frenal pull and amount of keratinized width. The amount of keratinized tissue around implants was measured using a periodontal probe (UNC-15, Hu Friedy Group,Chicago, United States of America) with 1mm increments up to 15mm.

Soft tissue dehiscence and Implant thread exposure

The presence of soft tissue dehiscence around implants was noted as either present or absent, and included the site (buccal or palatal/lingual). Implant thread exposure was noted as being present or absent.

Bleeding on probing

Bleeding on probing was recorded at six sites per tooth and implant. A formula was used to calculate the bleeding on probing percentage **(**O'Leary et al. 1972**).**

$$\text{BOP} = \frac{\text{Number of bleeding sites}}{\text{Number of sites evaluated}} \times 100$$

Plaque Percentage

A disclosing agent, 2-Tone® (Young Dental, Earth City, United States of America), was applied on teeth and implants. The old plaque stained blue, and it was recorded on six sites. The plaque percentage was worked out using a formula (Ainamo & Bay 1975**).**

$$\text{Plaque percentage} = \frac{\text{Number of sites with old plaque}}{\text{Number of sites evaluated}} \times 100$$

Implant Mobility

Implant mobility was assessed by using the end of two single-ended instruments (e.g., dental mirror and explorer probe) on the crown in a bucco-palatal/lingual direction. Distinction between true implant mobility and mobility of the supra structure was made. A rotational movement was employed to assess for mobility of crown or healing abutment, and a bucco-palatal direction for implants(Alani & Bishop, 2014).

3.6.4 Radiographic Assessment

Periapical radiographs of each implant were taken. Periapical radiographs allowed evaluation of the bone in the entire length of the implant, from most coronal past the most apical. Presence of excess cement was noted. Radiographic bone loss around teeth was recorded.

Periapical radiographs taken during implant placement, that were present and in good condition, were assessed and compared to current radiographs. Radiographic bone loss was defined as ≥3 mm apico-coronal or vertical increase in the distance between the supporting bone level and the shoulder of the implant. No radiographic bone loss was defined as <3 mm in the distance between the supporting bone level and the shoulder of the implant (Berglundh et al., 2018). Radiographic bone loss in relation to implant was recorded to include <25%, 25-50% and >50%. The presence of bone defects was recorded.

3.6.5 Implant Disease Risk Assessment

Some of the vectors from the risk assessment tool, Implant Disease Risk Assessment, (IDRA) which estimates the risk for a patient to develop peri-implantitis was utilized. The information for all the vectors of the assessment tool was obtained from the files. The eight vectors include the following (1) assessment of a history of periodontitis (2) percentage of sites with bleeding on probing (BOP) (3) number of teeth/implants with probing depths (PD) ≥5 mm (4) the ratio of periodontal bone loss (evaluated from a radiograph) divided by the patient's age (5) periodontitis susceptibility as described by the staging and grading categories from the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases (6) the frequency/compliance with supportive periodontal therapy (7) the distance in mm from the restorative margin of the implant-supported prosthesis to the marginal bone crest and (8) prosthesis-related factors including cleansibility and fit of the implant-supported prosthesis (Heitz-Mayfield, Heitz & Lang, 2020). Due to the records in the patient's files only vectors (1), (6), (7) and (8) were assessed.

3.6.6 Prophylaxis

The patients were given prophylaxis (scaling and polish) on the day of the examination, and a comprehensive treatment plan was drawn up to address and accommodate the needs of the

patient as per the findings of the clinical and radiographic examination. The patients with peri-implant mucositis or peri-implantitis were managed in the Oral Medicine and Periodontics Clinic. The patients presenting with other conditions were referred accordingly for management. The patients who were not on an implant maintenance program were given an appointment for implant maintenance.

3.7 Data Analysis

Data derived during this study was anonymized using the subject codes described in the data collection sheet. Subject data were entered in a Microsoft Excel spreadsheet. Descriptive statistics of the study participants were tabulated and presented using means and ranges for continuous values and percentages for discrete variables. Chi squared test of association was used to identify relationships between patient related factors and peri-implantitis, implant related factors and peri-implantitis, results of the IDRA tool and peri-implantitis. Chi squared test of association was used for between group comparisons (smoking, diabetes, history of periodontitis, history of peri-implantitis, poor oral hygiene, cement retained restorations, and implant surface roughness). All statistical analysis was performed with the assistance of a statistician.

3.8 Ethical Consideration and Data Management

3.8.1 Management of diseases or conditions

Each patient received prophylaxis after the assessment was completed. The patients with presence of disease were referred for an appointment accordingly.

3.8.2 Data management

Data was managed and stored as per University of the Western Cape Research Data Management Policy. Data in the written format was kept safe in the patients' hospital file. Data in the electronic format was kept safe in REDCap Project form. After completion of the

study the data was deposited in the Institutional Research Data Repository at graduation. Data will be appropriately preserved and remain accessible and usable for future research.

i) Ethical Compliance and approval

a. Permission to conduct the study

Ethics clearance was sought from the Biomedical Science Research and Ethics Committee of the University of the Western Cape (Certificate No. BM21/8/10). Permission was sought from the Manager of Dental services and the Head of Department of Oral Medicine and Periodontology.

b. Confidentiality Concerns

Each eligible patient was assigned a study number.

The data was assessed anonymously.

c. Obtaining Consent

Each patient was given a written consent form to read and complete in order to be part of the study.

d. Intellectual Property

The study was presented at the International Team for Implantology (ITI) Southern Africa 2022 conference and it will hopefully published in a journal article.

ii) Secure Storage and Backup

a. Raw and redefined data storage

Data in the written format will be kept safe in the patients' hospital file. The hospital files are stored in the Tygerberg Dental Hospital file room. Data in the electronic format will be kept safe in REDCap Project form. The main researcher's REDCap UWC account was utilised, and the supervisors were registered as account users.

Redefined data will be stored in the Institutional Research Data Repository.

b. Security and Backup

The electronic data will be safeguarded by passwords. The hospital files are accessible to health care professionals managing the patients.

iii) Data Sharing

Electronic data will be accessible via REDCap and will be safeguarded by passwords. The supervisors will be registered as users and will have access to the data.

Health care professionals managing the patients will have access to the data in the hospital files.

iv) Data Selection, Preservation and Retention

Data stored in the hospital files will stored as per Health Professional Council of South Africa's guidelines, and will be stored for a period of not less than six years. After completion of the study the data will be deposited in the Institutional Research Data Repository at graduation. Data will be appropriately preserved and remain accessible and usable for future research.

CHAPTER 4

4. Results

4.1 Patient Level Demographics

The patient level demographic data is presented in Figures 4.1, 4.2 and Table 4.1. The study population consisted of a total number of 42 patients, with the patient age ranging from 24.28 to 85.26 years and the gender distribution consisting of 61.9% females and 38.1% males.

Table 4. 1 Study population age distribution

Patient Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
42	24.28	85.26	62.82	63.98

n: number, y: years.

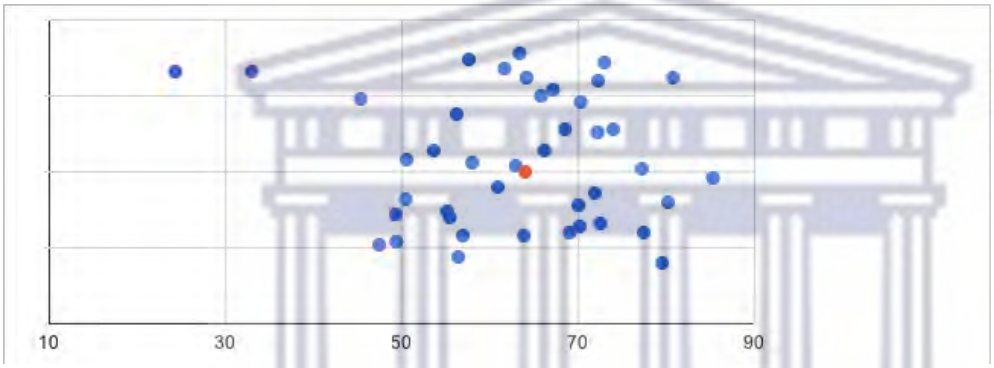

Figure 4. 1 Age distribution

Median in red

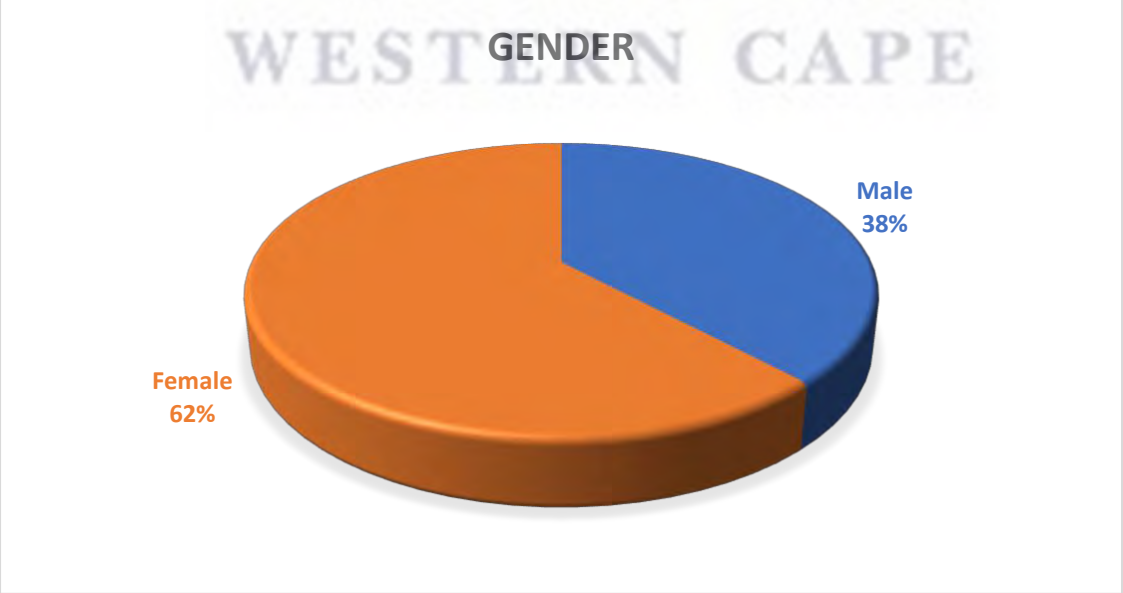

Figure 4. 2 Gender

Counts/frequency: Female (26, 61.9%), Male (16, 38.1%)

4.2 Factors associated with peri-implant health and disease

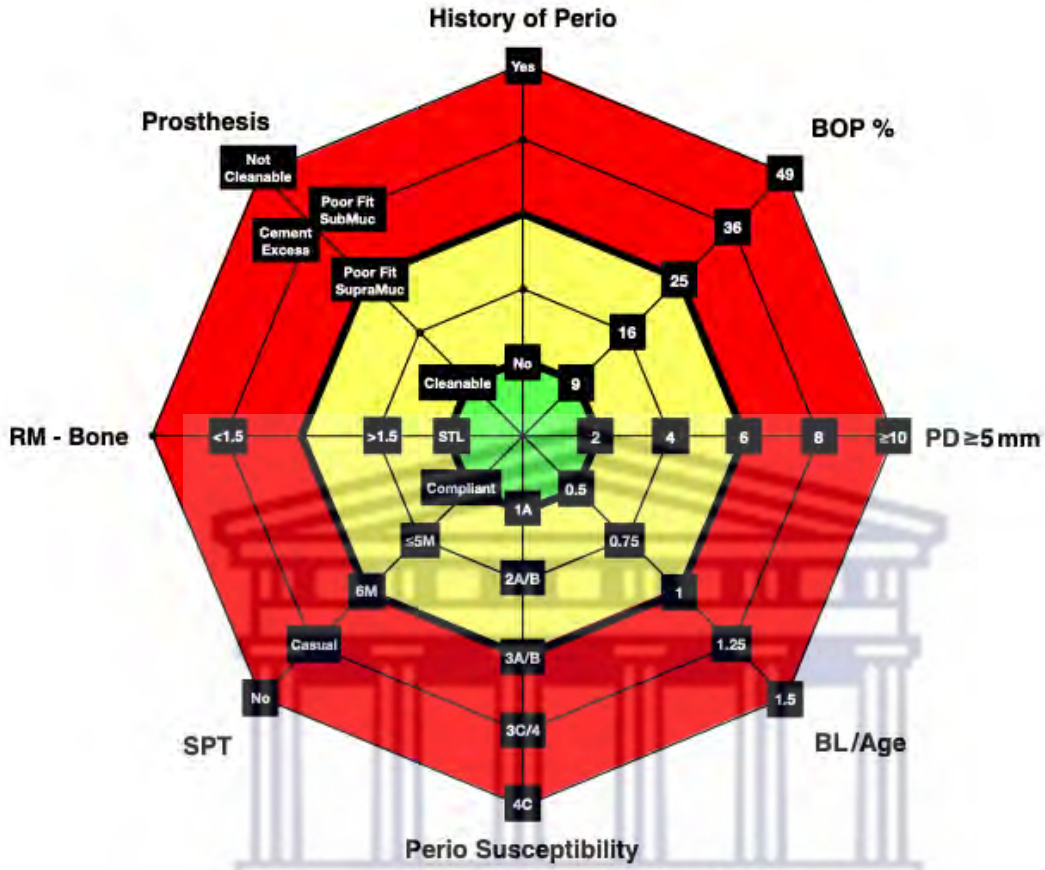

Figure 4. 3 Implant Disease Risk Assessment (IDRA) functional diagram (Heitz-Mayfield et al., 2020)

Data for some of the IDRA tool vectors was obtained from the files, these include history of periodontitis, RM to bone crest, prosthesis related factors, and SPT. These are depicted in the figures to follow.

The factors associated with peri-implant health and disease are summarized in Figures 4.3, 4.4 and 4.5. In the study population, 26.2% of the sample had a history of periodontitis. Diabetics comprised 4.8% of the study population. In the study population, non-smokers comprised 88.1% , current smokers 9.5% and past smokers 2.4% respectively.

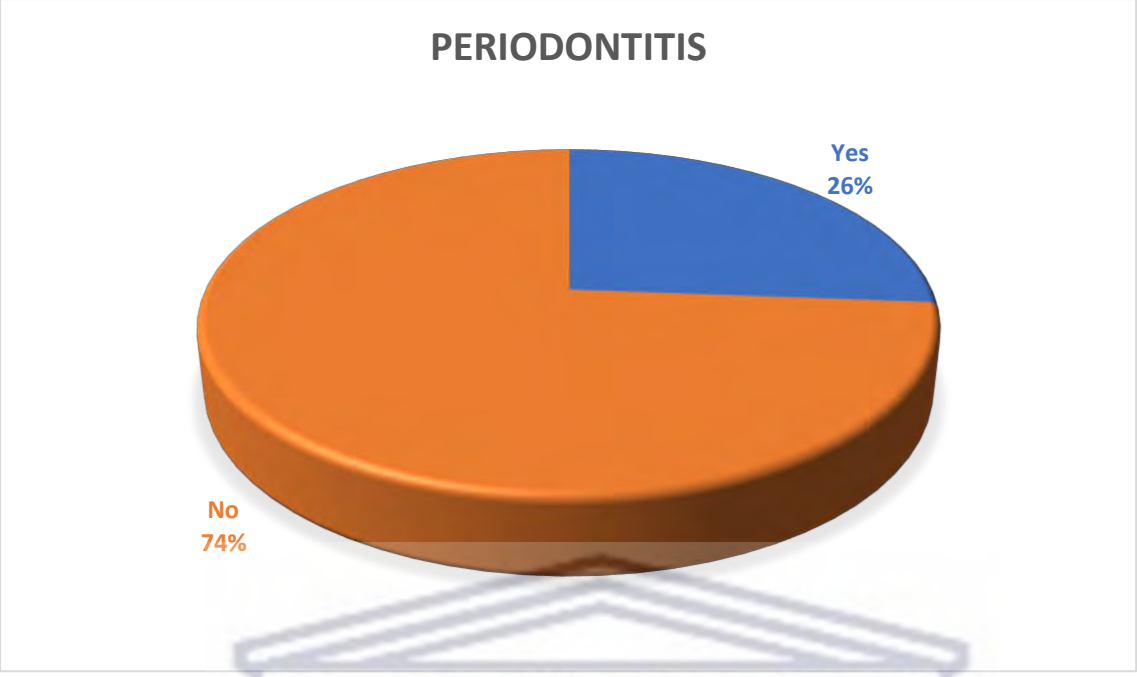

Figure 4. 4 History of periodontitis.

Counts/frequency: Yes (11, 26.2%), No (31, 73.8%)

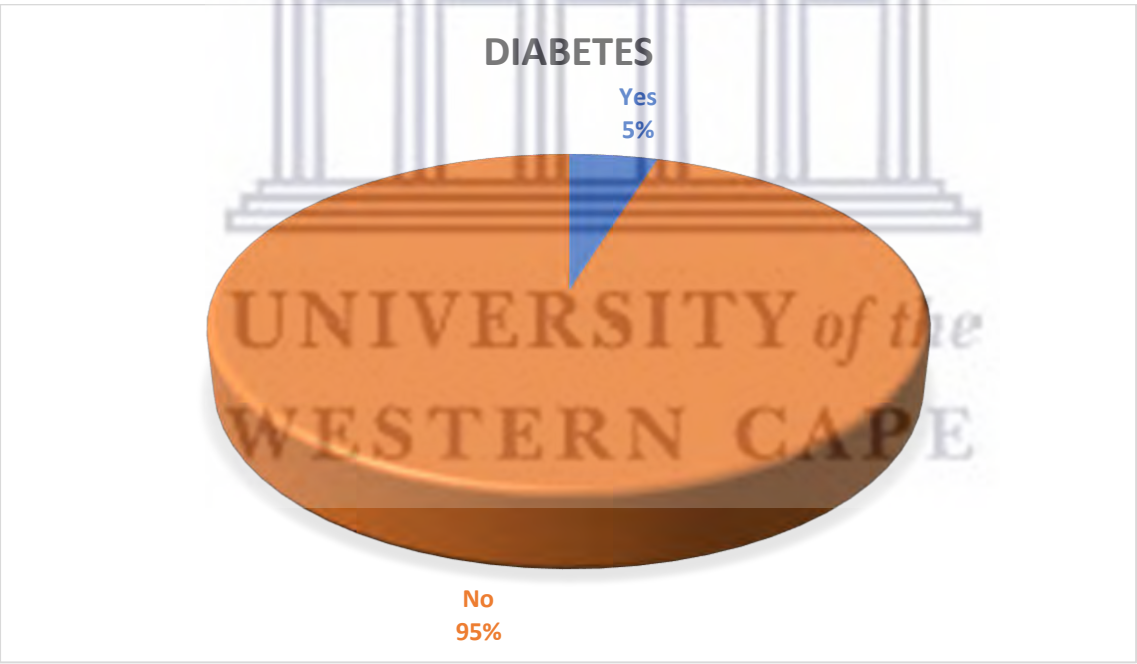

Figure 4. 5 History of diabetes mellitus.

Counts/frequency: Yes (2, 4.8%), No (40, 95.2%)

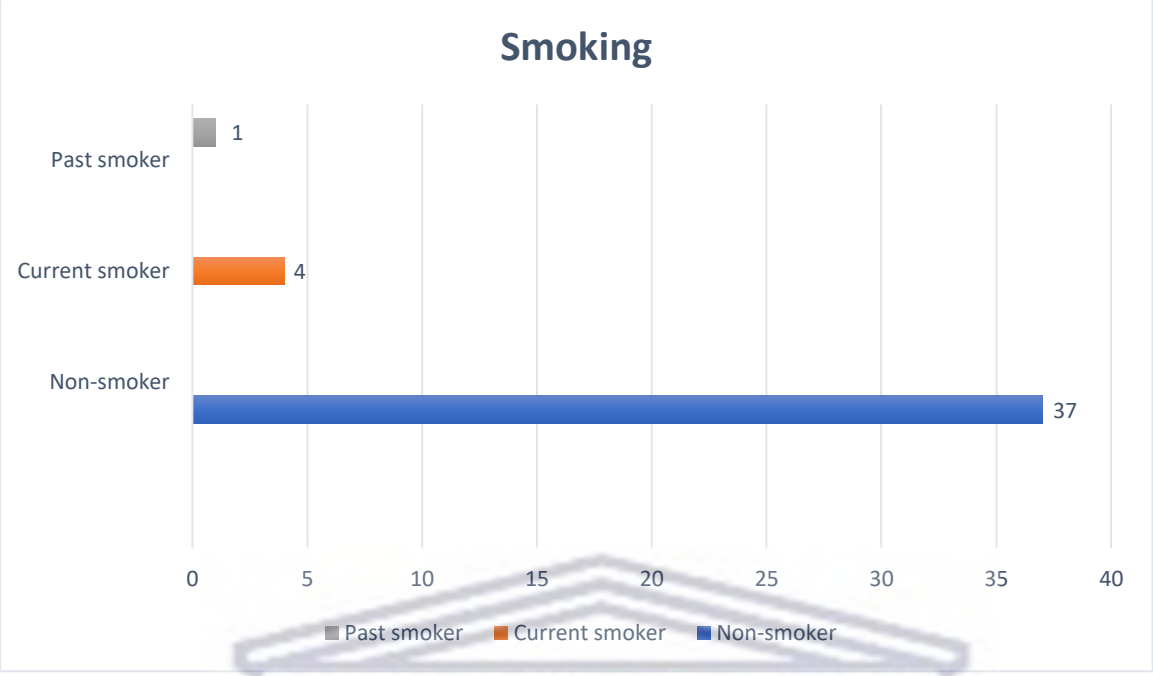

Figure 4. 6 History of smoking.

Counts/frequency: Non-smoker (37, 88.1%), Current smoker (4, 9.5%), Past smoker (1, 2.4%)

4.3 Prevalence of peri-implant health and disease

Peri-implant health was found in 66.7% of the study population at patient level, while peri-implant mucositis was diagnosed in 19.0% and peri-implantitis was diagnosed in 14.3% of the study population a respectively, see Figure 6.

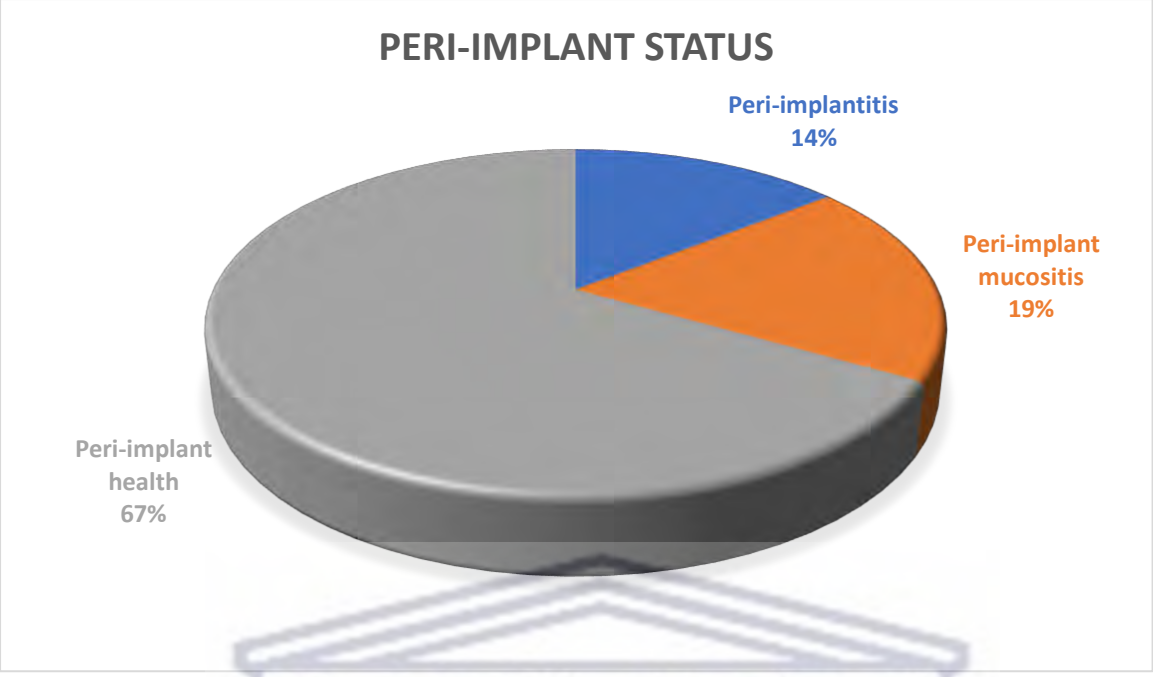

Figure 4. 7 Peri-implant status at patient level.

Counts/frequency: Peri-implant health (28, 66.7%), Peri-implant mucositis (8, 19.0%), Peri-implantitis (6, 14.3%).

4.4 Implant Level Demographics

The implant level demographic data are presented in Figures 4.7, 4.8 and Table 4.2. A total number of 137 implants made up the study population with the implant age ranging from 0.91 to 22.81 years.

Table 4. 2 Age of implant distribution at implant level

Implant Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
137	0.910	22.81	8.72	6.27

n: number, y: years.

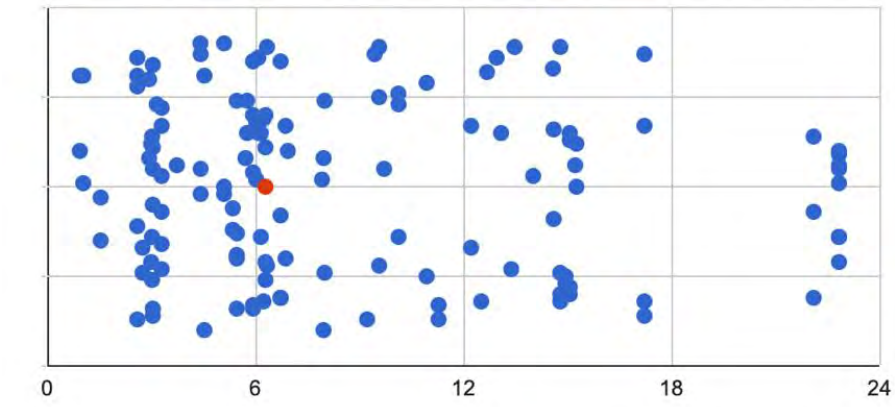

Figure 4. 8 Age of implant distribution. Median in red.

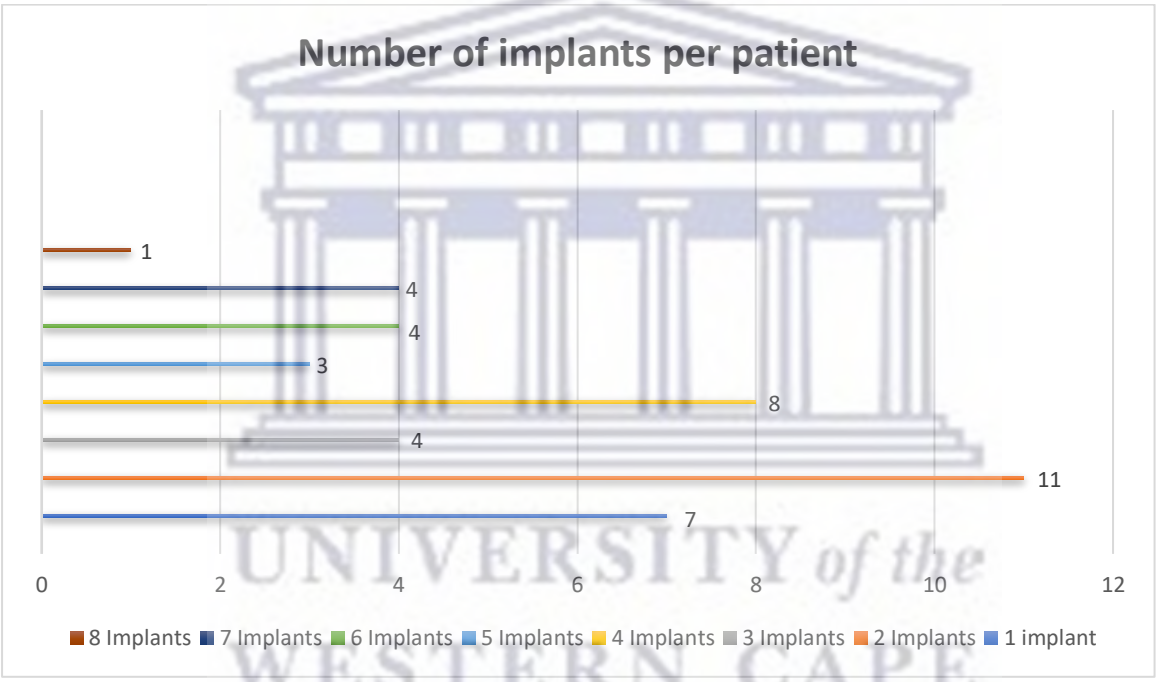

Figure 4. 9 Number of implants per patient.

Counts/frequency: 1 (7, 16.7%), 2 (11, 26.2%, 3 (4, 9.5%), 4 (8, 19.0%), 5 (3, 7.1%), 6 (4, 9.5%), 7(4, 9.5%), 8 (1, 2.4%)

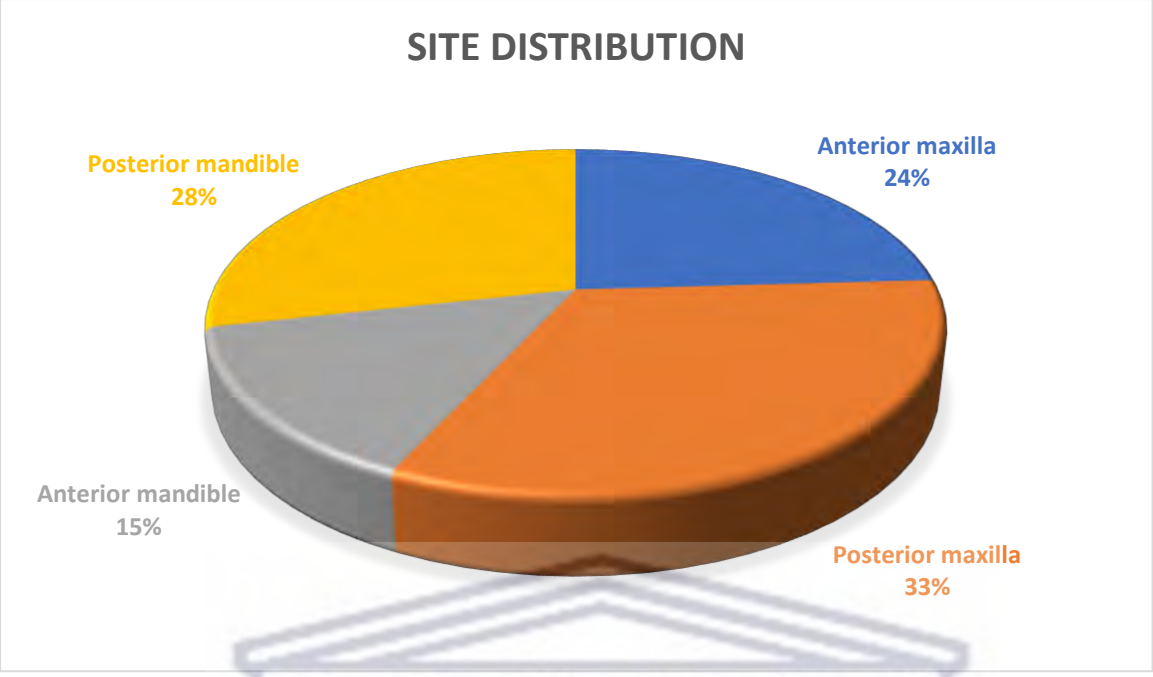

Figure 4. 10 Site distribution of implants.

Counts/frequency: Anterior maxilla (33, 24.1%), Posterior maxilla (45, 32.8%), Anterior mandible (20, 14.6%), Posterior mandible (39, 28.5%)

The number of implants per patient ranged from 1 to 8 implants per patient, see Figure 4.8. Approximately 32.8% of the implants were found in the posterior maxilla while only 14.6% were found in the anterior mandible, see Figure 4.9. The implant system distribution is depicted in Figure 4.10. The least represented implant systems were Bicon, Champions and Super Line with 0.9% individually.

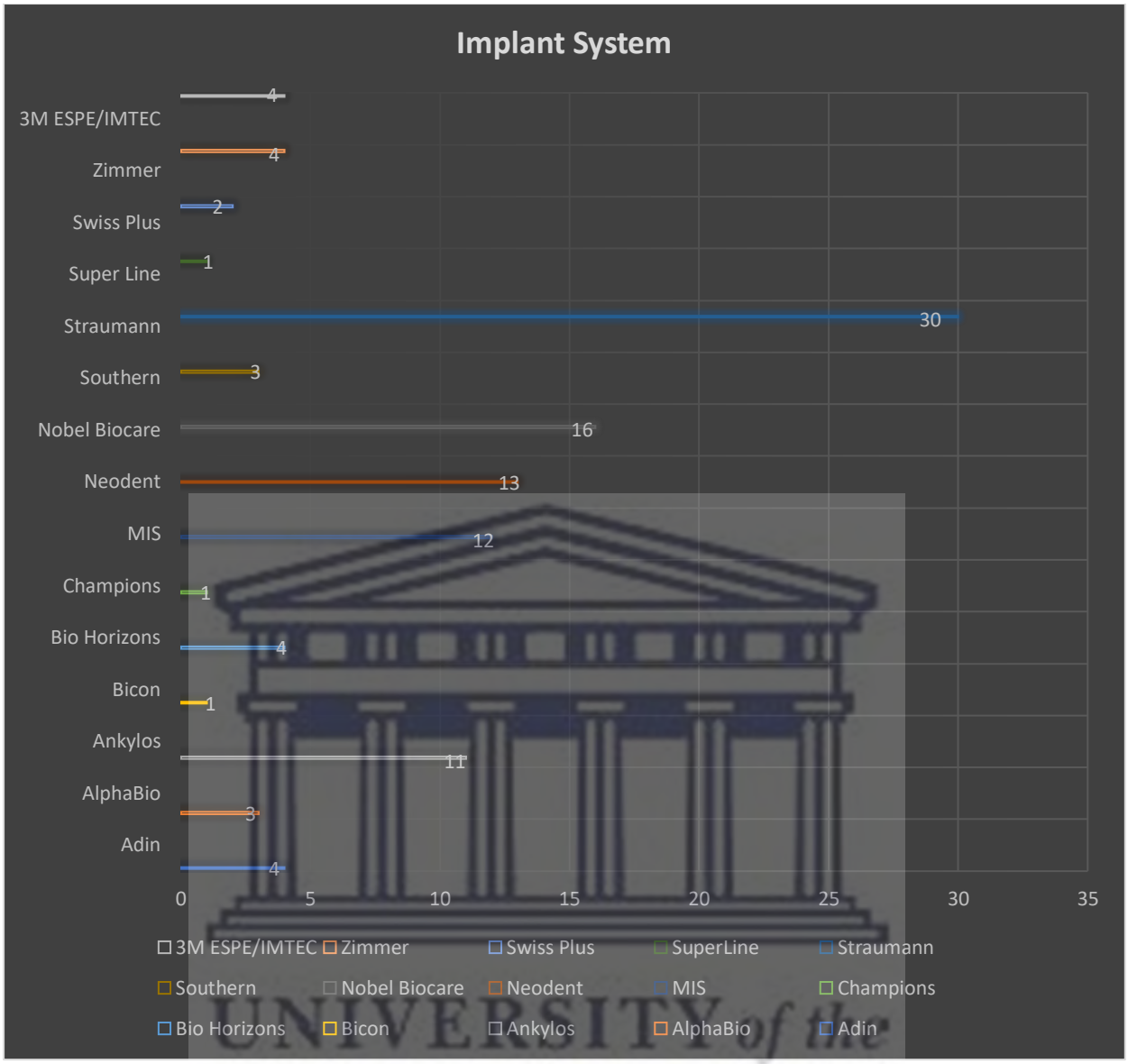

Figure 4. 11 Implant system distribution.

Counts/frequency: Adin (4, 3.7%), AlphaBio (3, 2.8%), Ankylos (11, 10.1%), Bicon (1, 0.9%), Bio Horizons (4, 3.7%), Champions (1, 0.9%), MIS (12, 11.0%), Neodent (13, 11.9%), Nobel Biocare (16, 14.7%), Southern (3, 2.8%), Straumann (30, 27.5%), SuperLine (1, 0.9%), Swiss Plus (2, 1.8%), Zimmer (4, 3.7%), 3M ESPE/IMTEC (4, 3.7%).

4.5 Peri-implant Status at Implant Level

Peri-implant status at implant level is depicted in Figure 4.11. PIH was diagnosed in 79.6%, while PIM in 11.7% and PI was diagnosed in found 8.8% of the study population respectively.

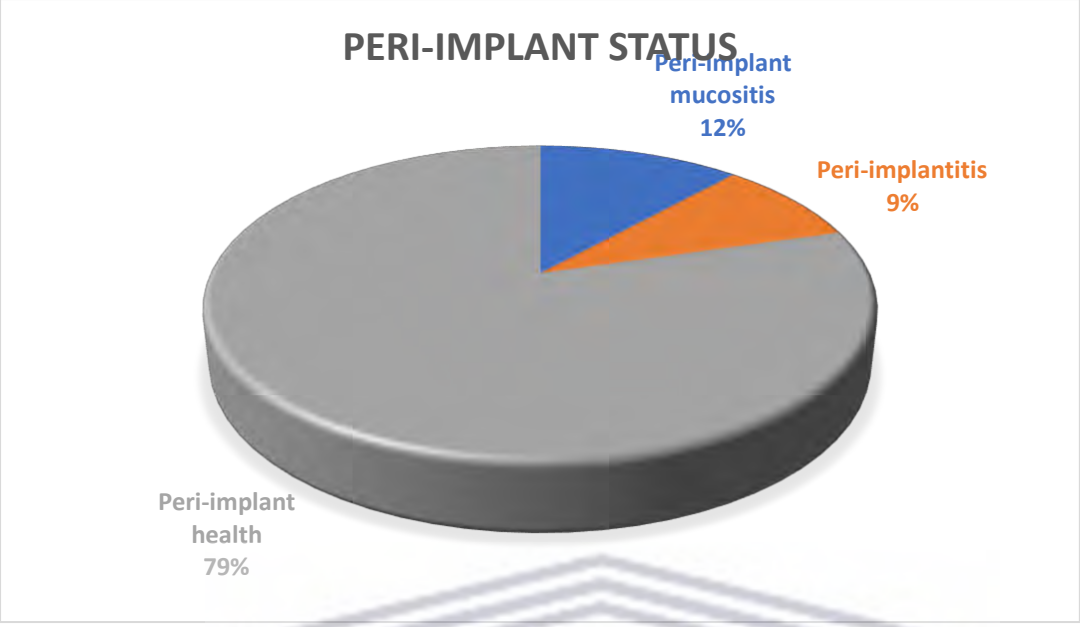

Figure 4. 12 Peri-implant status at implant level.

Counts/frequency: Peri-implant health (109, 79.6%), Peri-implant mucositis (16, 11.7%), Peri-implantitis (12, 8.8%)

4.6 Implant Level: Peri-implant health

4.6.1 Peri-implant Health Demographics

Patient and implant age distribution in the PIH group are depicted in Tables 4.3, 4.4, Figures 4.13, and 4.14. The patient age distribution ranged from 24.27 and 85.26 years, while the implant age ranged from 1.01 to 22.81 years. Approximately 60.6% of implants diagnosed with PIH were found in females, and 11.9% of the implants were in the anterior mandible and 34.9% were in the posterior maxilla, see Figure 4.15 and 4.16.

Table 4. 3 Patient age distribution in the peri-implant health group

Implant Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
109	24.27	85.26	64.49	66.15

n: number, y: years.

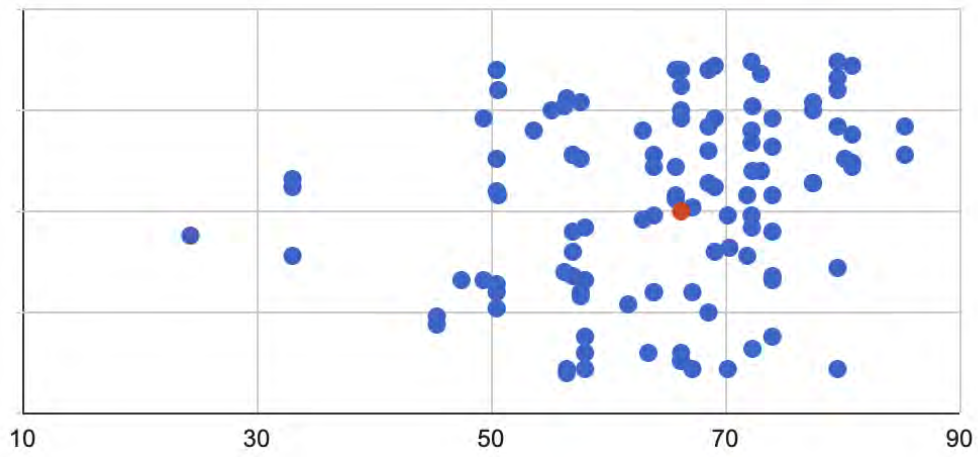

Figure 4. 13 Patient age distribution in the peri-implant health group.

Median in red.

Table 4. 4 Implant age in the peri-implant health group

Implant Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
109	1.01	22.81	8.62	6.36

n: number, y: years.

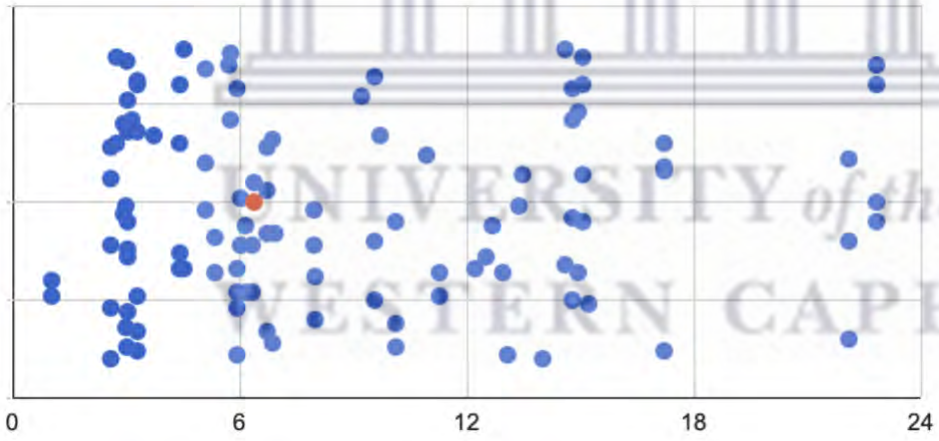

Figure 4. 14 Distribution of implant age in the peri-implant health group.

Median in red.

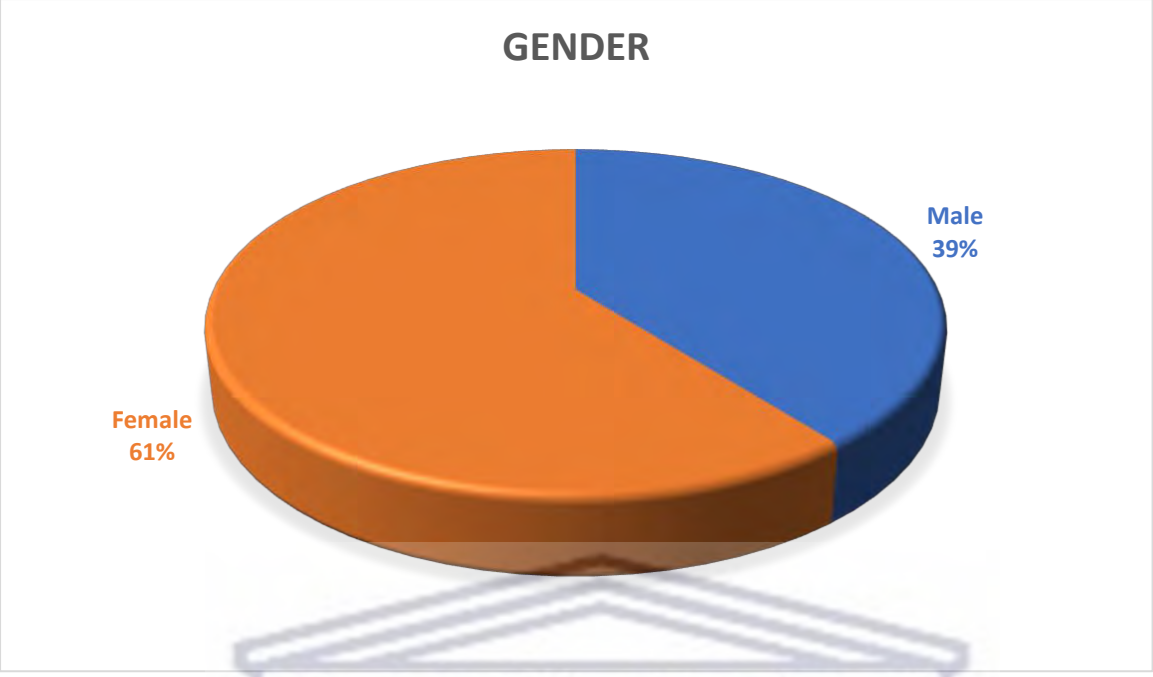

Figure 4. 15 Gender distribution in the peri-implant health group.

Counts/frequency: Female (66, 60.6%), Male (43, 39.4%).

4.6.2 Implant related factors

A total of 96 implants in the peri-implant health group were restored with fixed prostheses and 92 implants in this group were bone level implants. The implant systems that was represented the most in the peri-implant health group was Straumann at 27.5%, see Figure 4.17, 4.18 and 4.19.

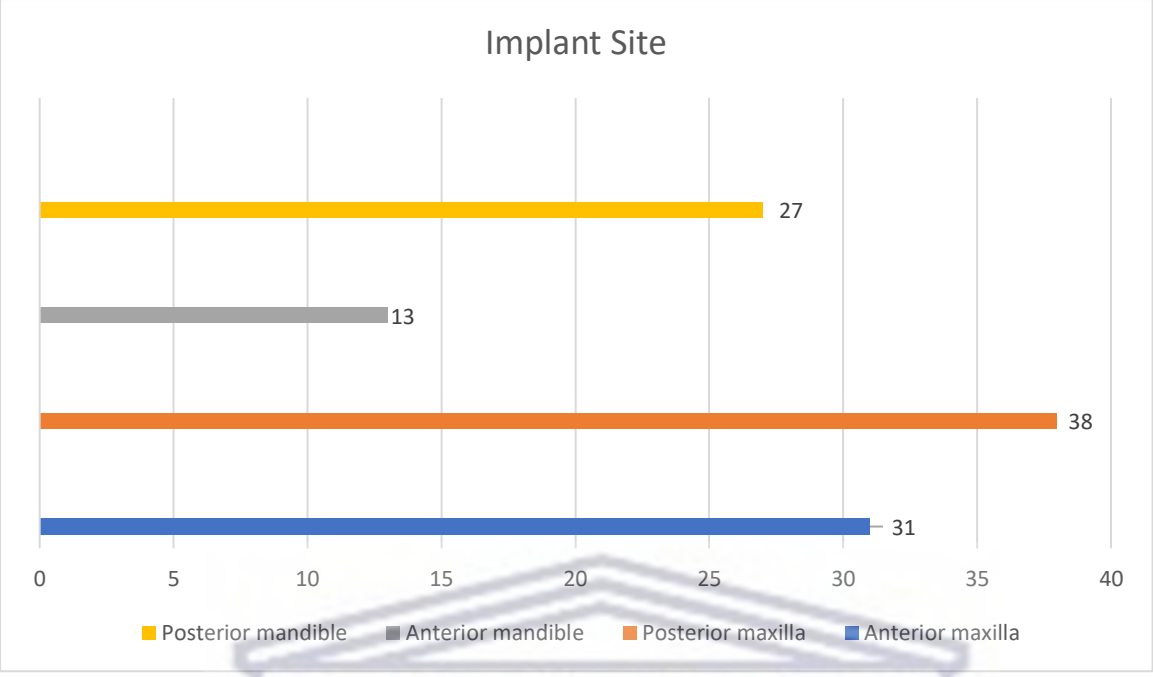

Figure 4. 16 Implant site distribution in the peri-implant health group.

Counts/frequency: Anterior maxilla (31, 28.24%), Posterior maxilla (38, 34.9%), Anterior mandible (13, 11.9%), Posterior mandible (27, 24.8%).

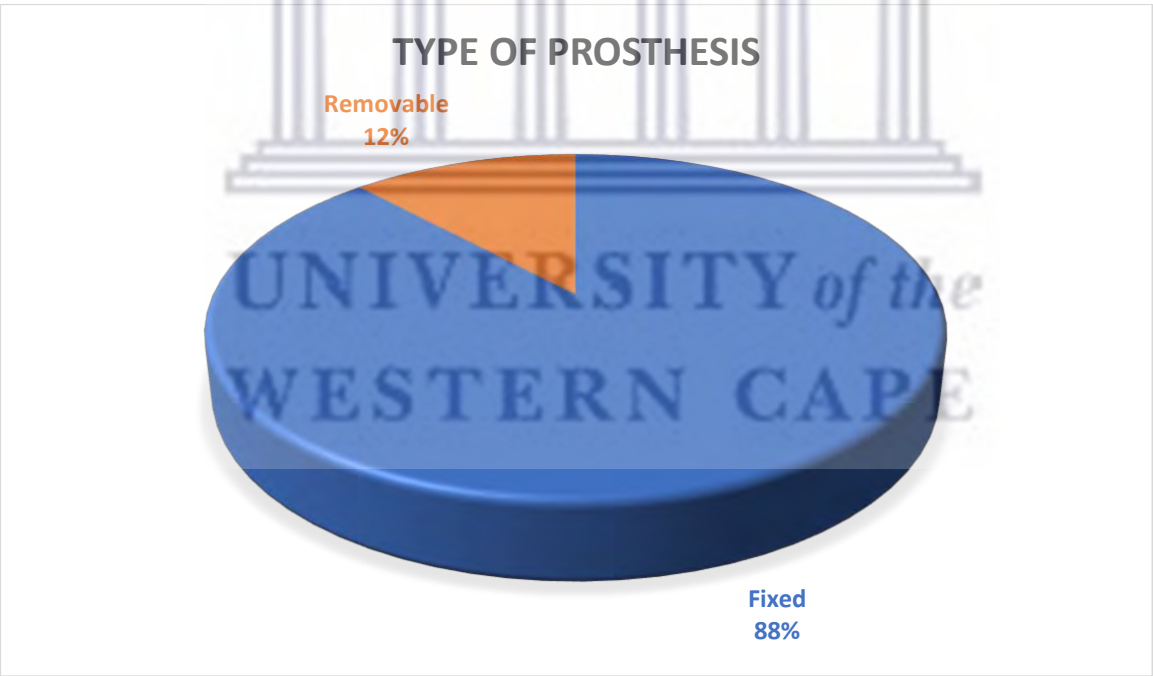

Figure 4. 17 Type of prosthesis in the peri-implant health group.

Counts/frequency: Fixed (96, 88.1%), Removable (13, 11.9%).

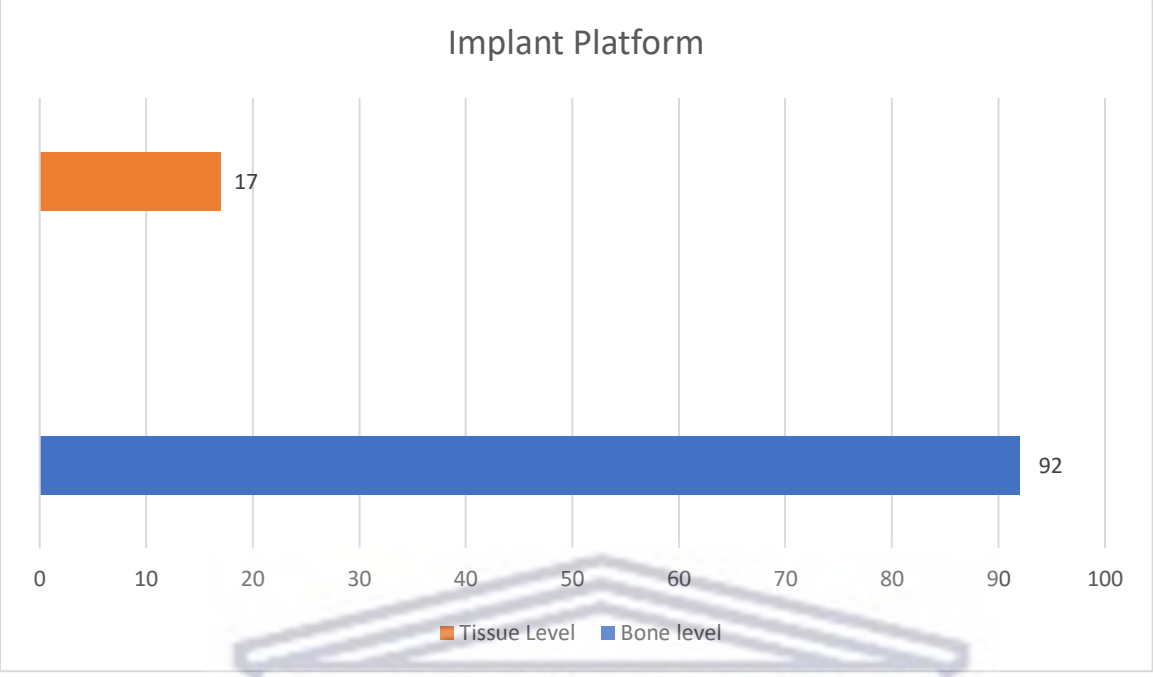

Figure 4. 18 Implant platform in the peri-implant health group.

Counts/frequency: Bone level (92, 84.4%), Tissue level (17, 15.6%).

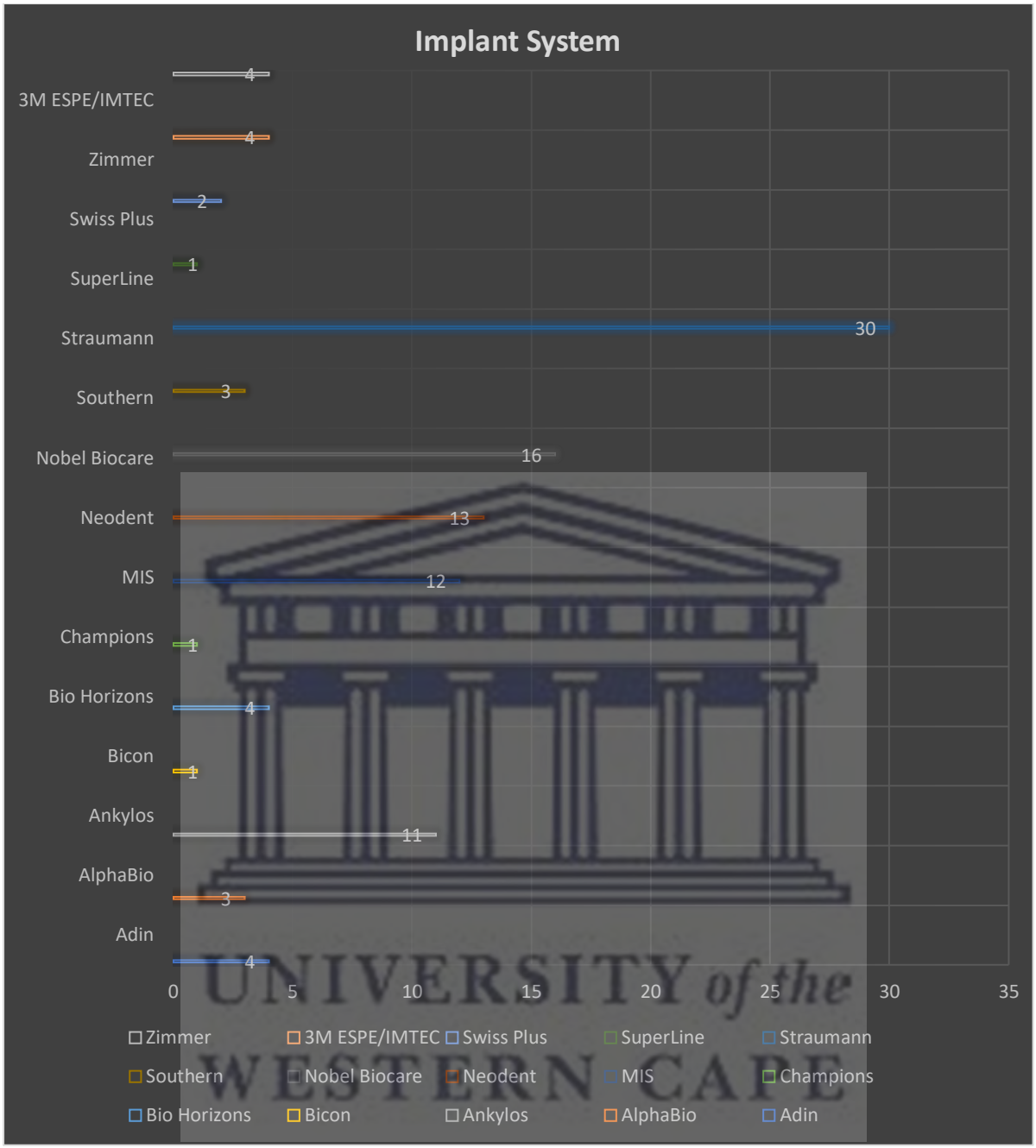

Figure 4. 19 Implant system distribution in the peri-implant health group.

Counts/frequency: Adin (4, 3.7%), AlphaBio (3, 2.8%), Ankylos (11, 10.1%), Bicon (1, 0.9%), Bio Horizons (4, 3.7%), Champions (1, 0.9%), MIS (12, 11.0%), Neodent (13, 11.9%), Nobel Biocare (16, 14.7%), Southern (3, 2.8%), Straumann (30, 27.5%), SuperLine (1, 0.9%), Swiss Plus (2, 1.8%), 3M ESPE/IMTEC (4, 3.7%), Zimmer (4, 3.7%).

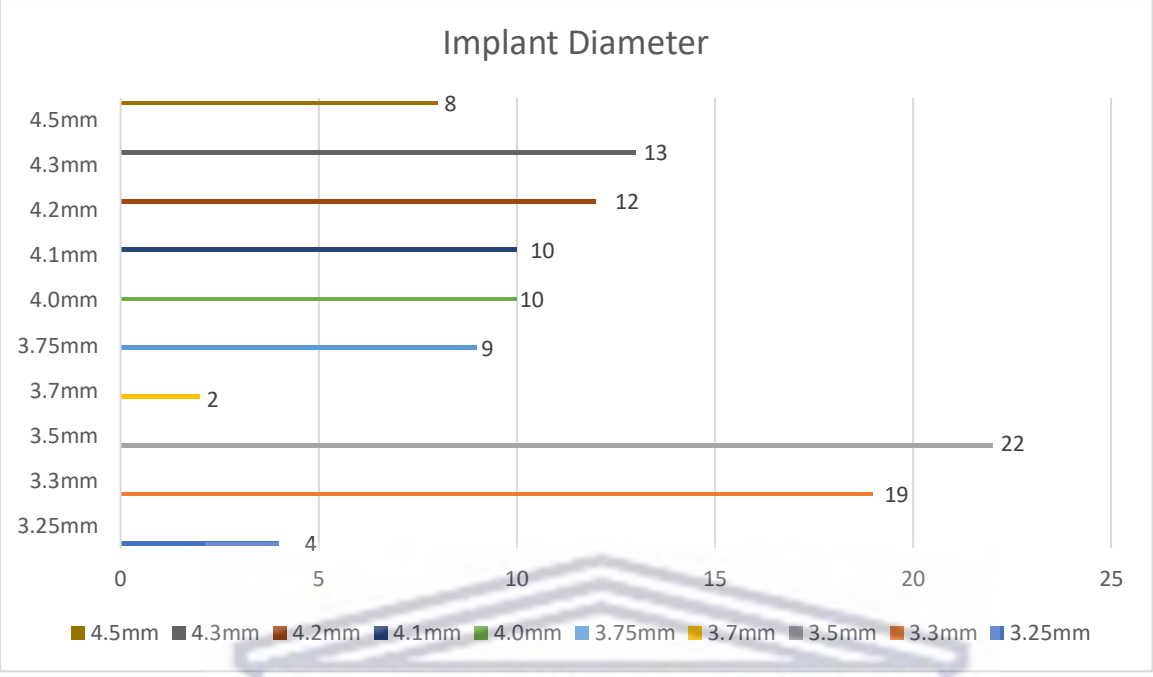

Figure 4. 20 Implant diameter in the peri-implant health group.

Counts/frequency: 3.25mm (4, 3.7%), 3.3mm (19, 17.4%), 3.5mm (22, 20.2%), 3.7mm (2, 1.8%), 3.75mm (9, 8.3%), 4.0mm (10, 9.2%), 4.1mm (10, 9.2%), 4.2mm (12, 11.0%), 4.3mm (13, 11.9%), 4.5mm (8, 7.3%).

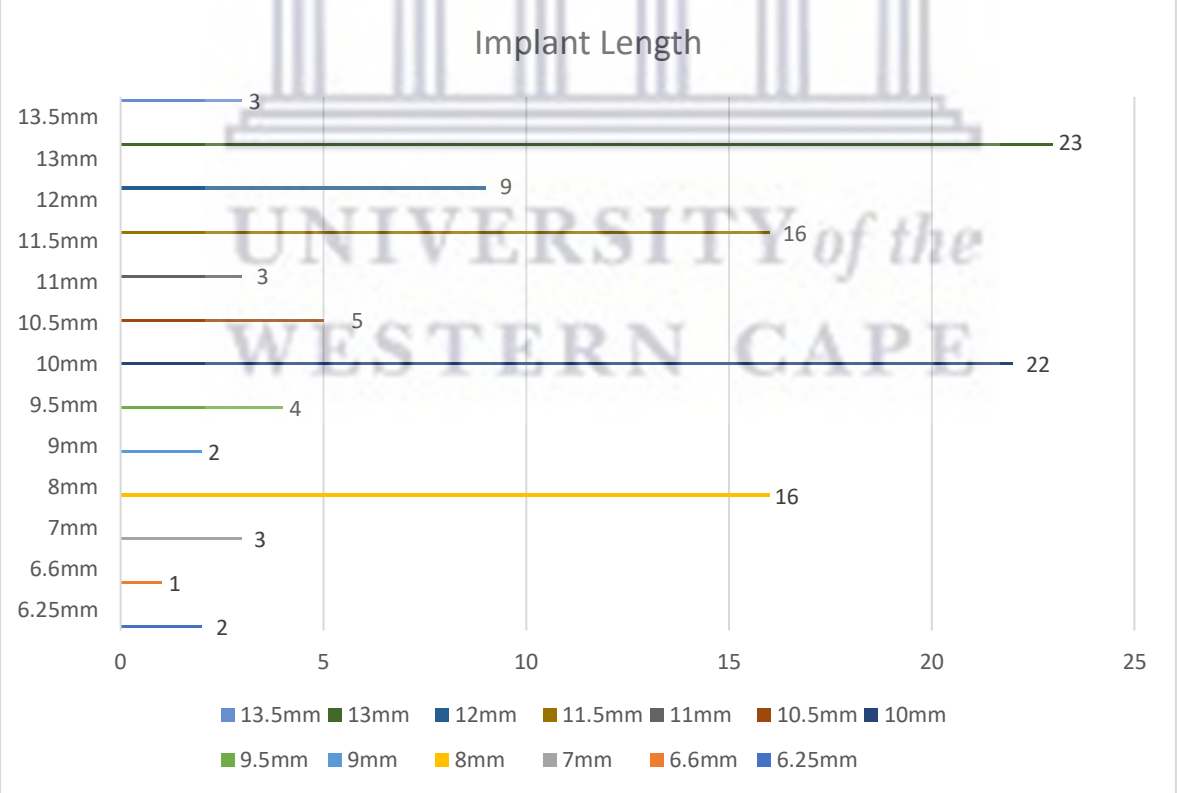

Figure 4. 21 Implant length in the peri-implant health group.

Counts/frequency: 6.25mm (2, 1.8%), 6.6mm (1, 0.9%), 7mm (3, 2.8%), 8mm (16, 14.7%), 9mm (2, 1.8%), 9.5mm (4, 3.7%), 10mm (22, 20.2%), 10.5mm (5, 4.6%), 11mm (3, 2.8%), 11.5mm (16, 14.7%), 12mm (9, 8.3%), 13mm (23, 21.1%), 13.5mm (3, 2.8%).

4.6.3 Systemic, Environment and Local Factors in the Peri-implant Health

The factors associated with peri-implant health are summarized in Figures 4.22, 4.23 and 4.24. In the study population 28.4% had a history of periodontitis, and 11.9% were smokers. The restorative margin to bone crest of <1.5mm was found in 37.6%, while that of >1.5mm was found in 50.5%, see Figure 4.25. Figure 4.26 outlines the compliance to supportive periodontal therapy distribution, 71.6% implants from the peri-implant health group were categorized in the non-compliant group.

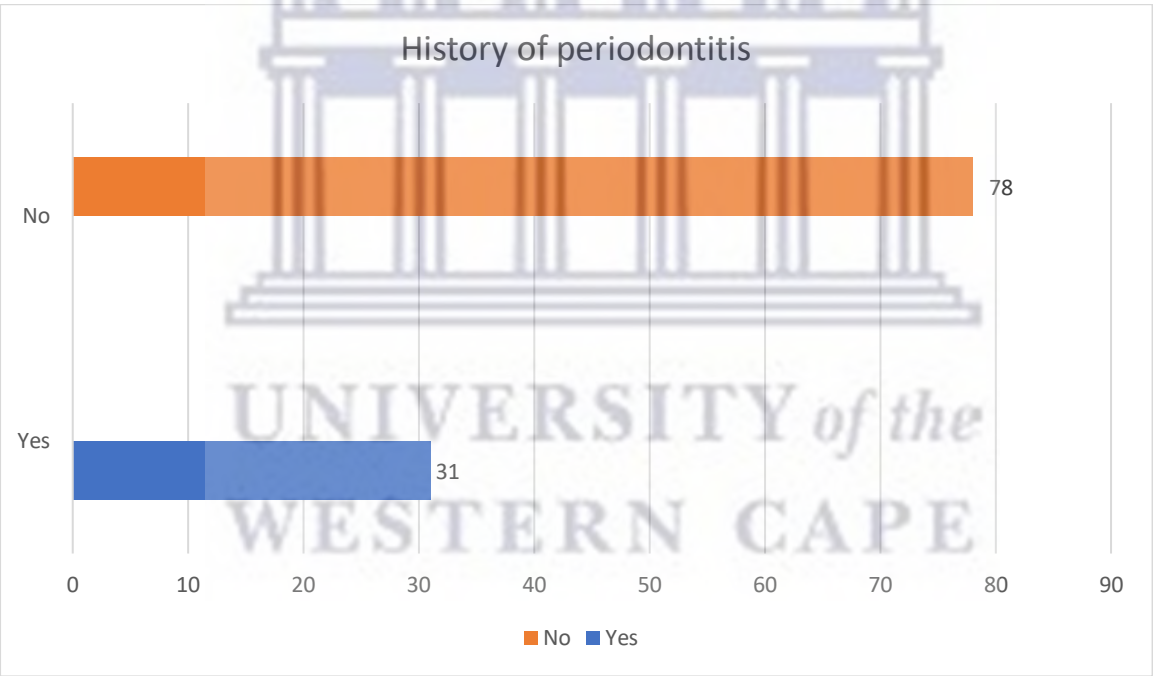

Figure 4. 22 History of periodontitis in the peri-implant health group.

Counts/frequency: Yes (31, 28.4%), No (78, 71.6%).

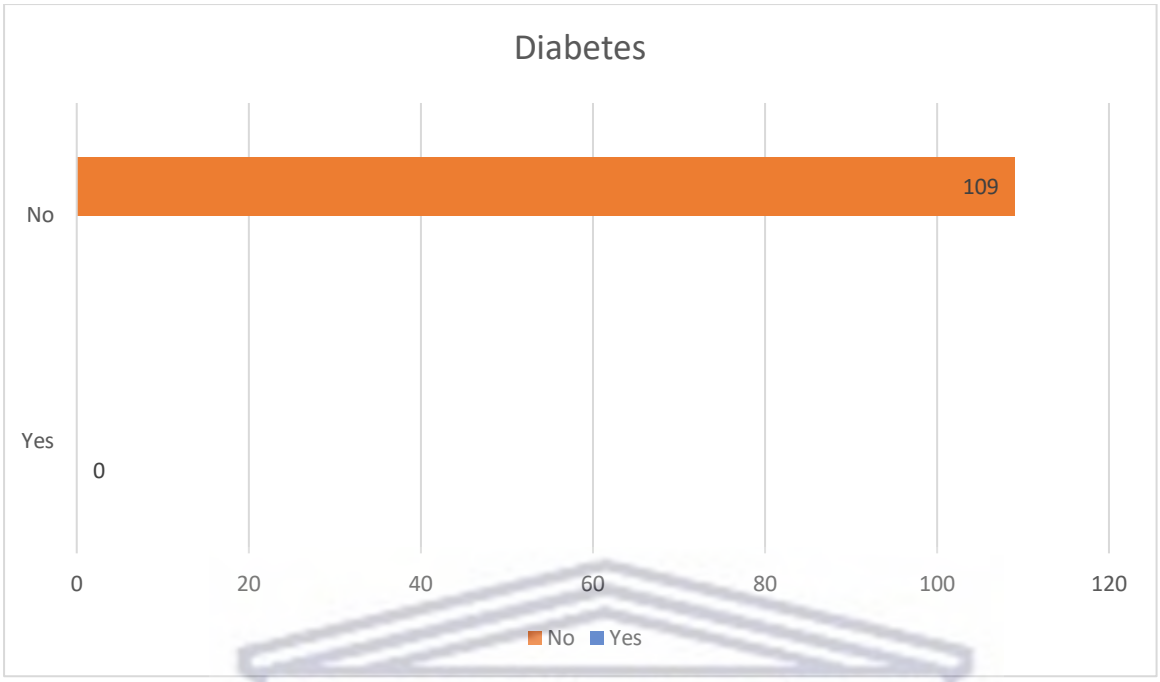

Figure 4. 23 Diabetes mellitus in the peri-implant health group.

Counts/frequency: Yes (0, 0.0%), No (109, 100.0%).

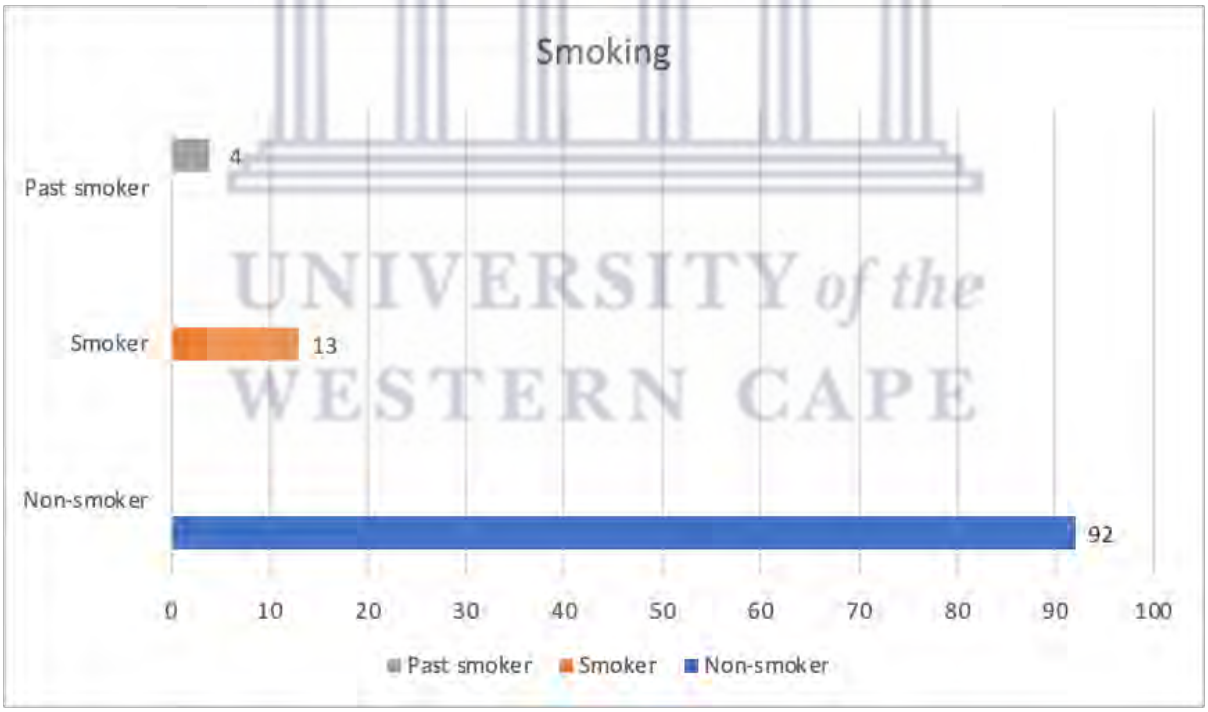

Figure 4. 24 Smoking in the peri-implant health group.

Non-smoker (92, 84.4%), Past smoker (4, 3.7%), Smoker (13, 11.9%)

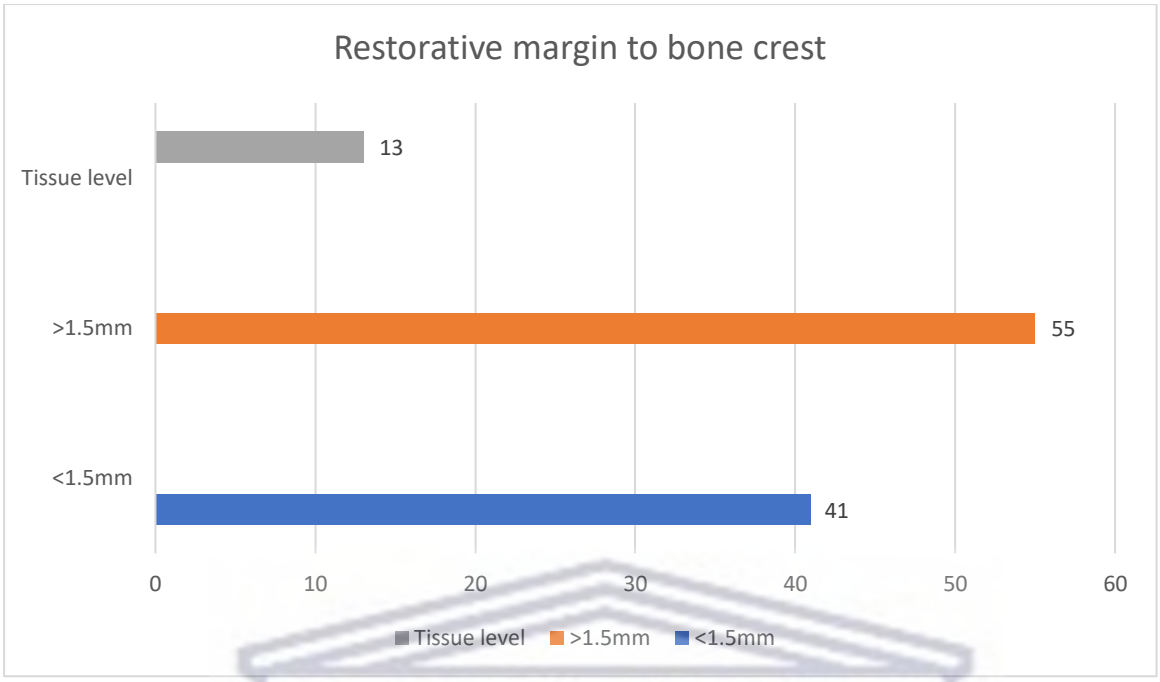

Figure 4. 25 Restorative margin to bone crest.

Counts/frequency: <1.5mm (41, 37.6%), >1.5mm (55, 50.5%), Tissue level (13, 11.9%).

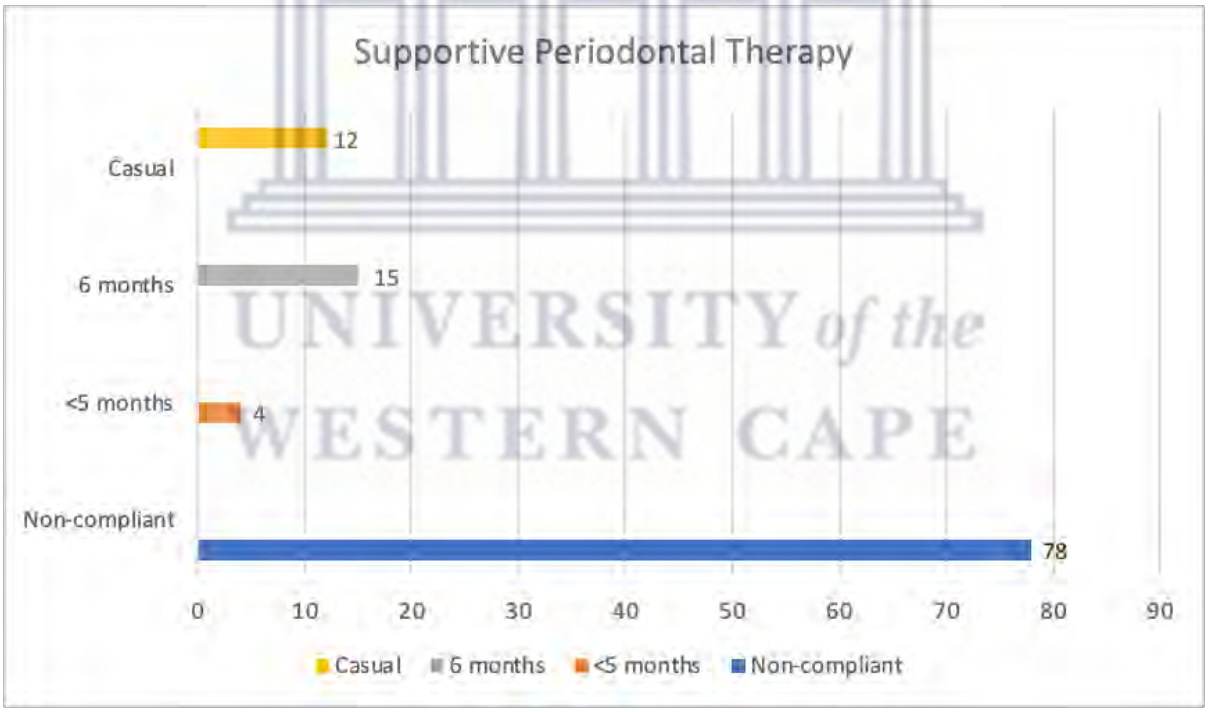

Figure 4. 26 Supportive periodontal therapy in the peri-implant health group.

Counts/frequency: Non-compliant (78, 71.6%), <5 months (4, 3.7%), 6 months (15, 13.8%), Casual (12, 11.0%).

4.7 Implant Level: Peri-implant Mucositis

4.7.1 Peri-implant Mucositis Demographics

Patient and implant age distribution in the PIM group are depicted in Table 4.4, 4.5, Figure 4.27, 4.28 and 4.29. The patient age distribution ranged from 49.20 and 80.10 years, while the implant age ranged from 0.9 to 15.20 years and there was no gender predilection in this group.

Table 4. 5 Patient age group in the peri-implant mucositis group

Total Implant Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
16	49.20	80.10	68.94	71.70

n: number, y: years.

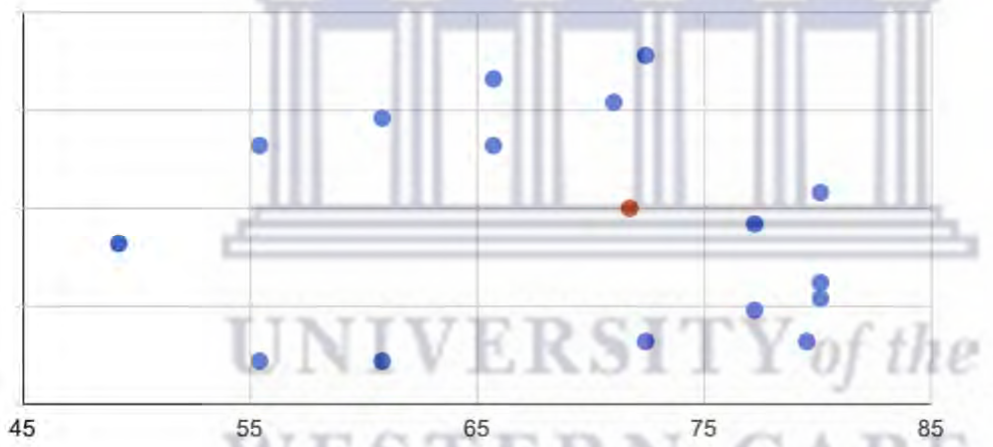

Figure 4. 27 Patient age group in the peri-implant mucositis group

Median in red.

Table 4. 6 Implant age distribution in the peri-implant mucositis group.

Total Implant Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
16	0.9	15.20	6.39	6.20

n: number, y: years.

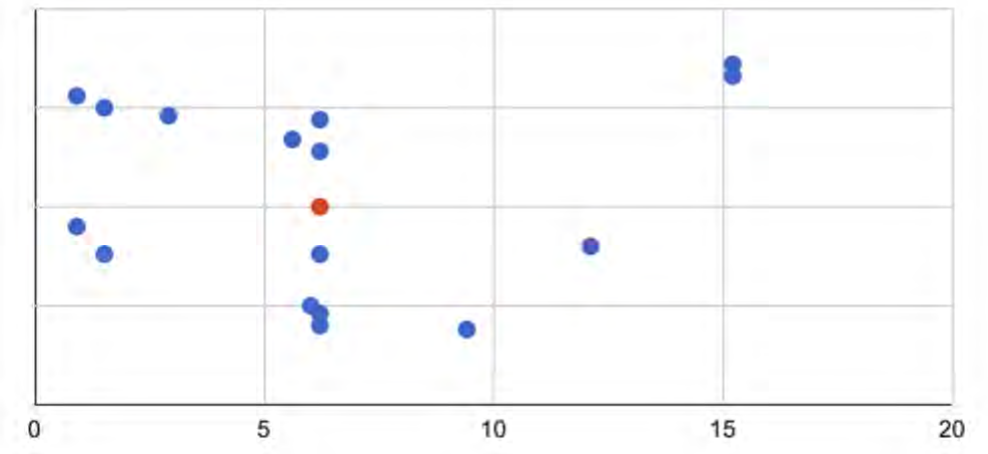

Figure 4. 28 Implant age distribution in the peri-implant mucositis group

Median in red.

Figure 4. 29 Gender distribution in the peri-implant mucositis group.

Counts/frequency: Female (8, 50.0%), Male (8, 50.0%).

4.7.2 Implant related factors

Bone level implants made up 81.3% while 43.8% of the total number of implants from this group were in the posterior maxilla, see Figure 4.30 and 4.31. A total of 11 implants in the PIM group were restored with fixed prostheses and the implant system that was represented the most in this group was Straumann at 31.3%, see Figure 4.32 and 4.33. Implant diameter

distribution ranged from 3.3mm and 5mm, and implant length distribution ranged from 8mm and 13mm, see Figure 4.34 and 4.35.

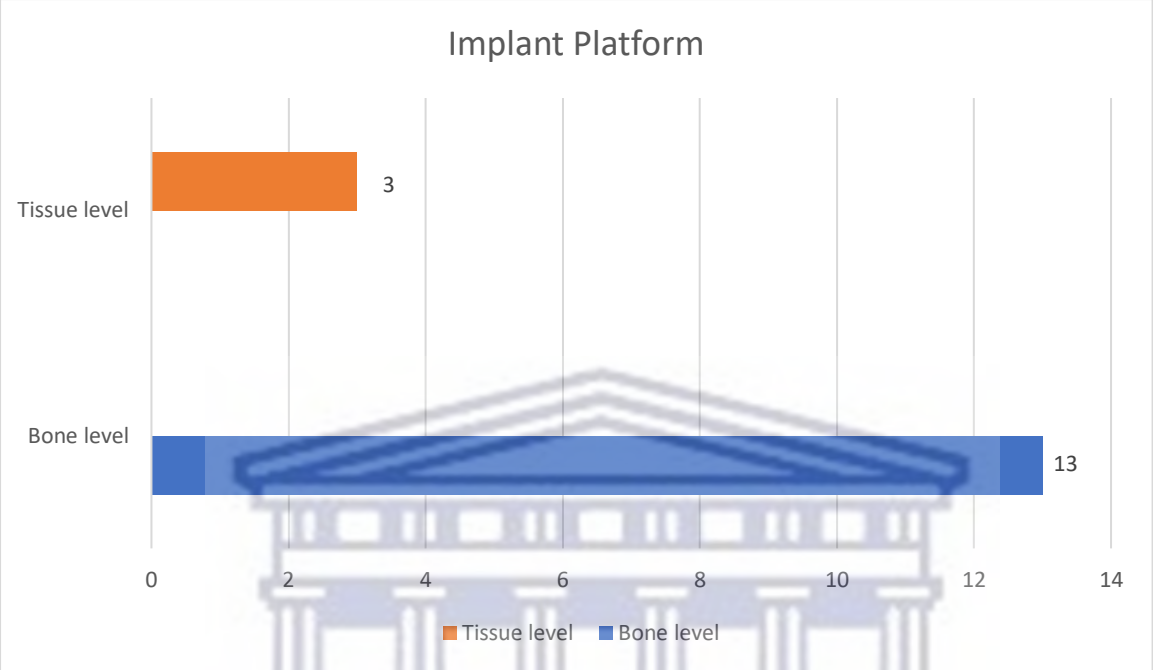

Figure 4. 30 Implant platform in the peri-implant mucositis group.

Counts/frequency: Bone level (13, 81.3%), Tissue level (3, 18.8%).

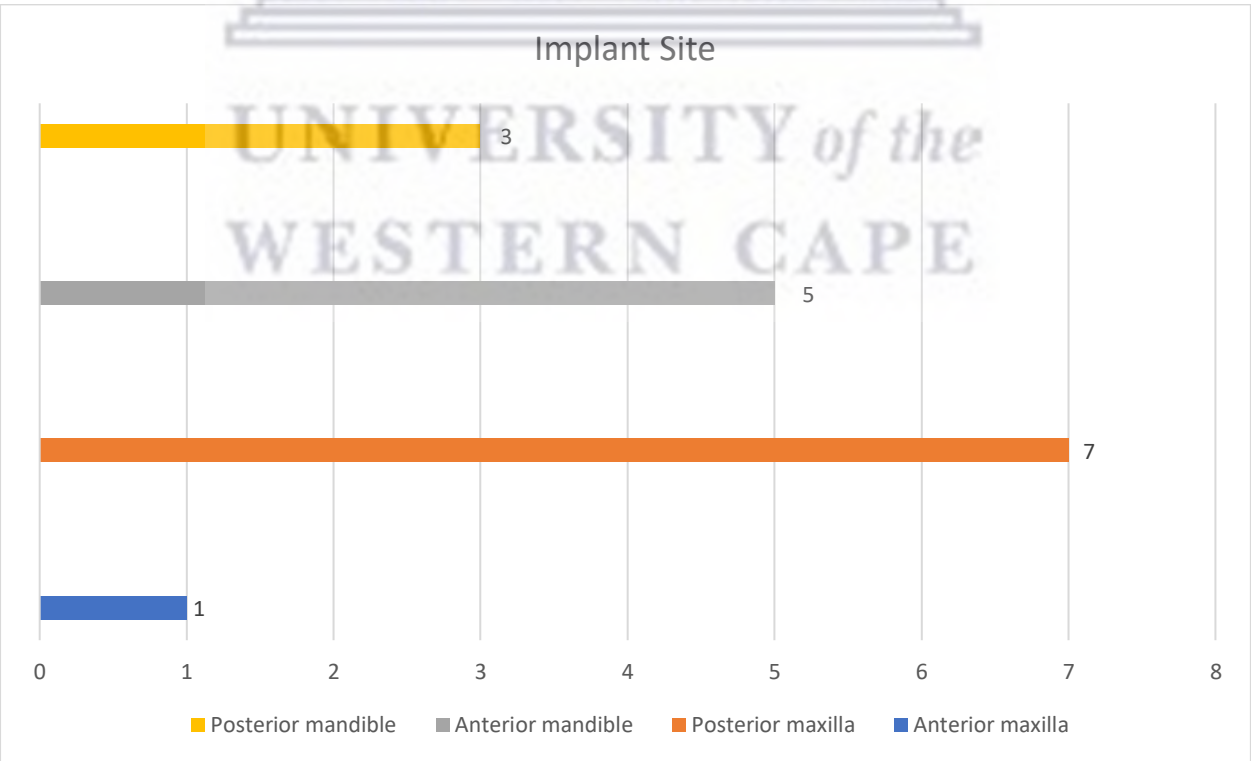

Figure 4. 31 Implant site distribution in the peri-implant mucositis group.

Counts/frequency: Anterior maxilla (1, 6.3%), Posterior maxilla (7, 43.8%), Anterior mandible (5, 31.3%), Posterior mandible (3, 18.8%).

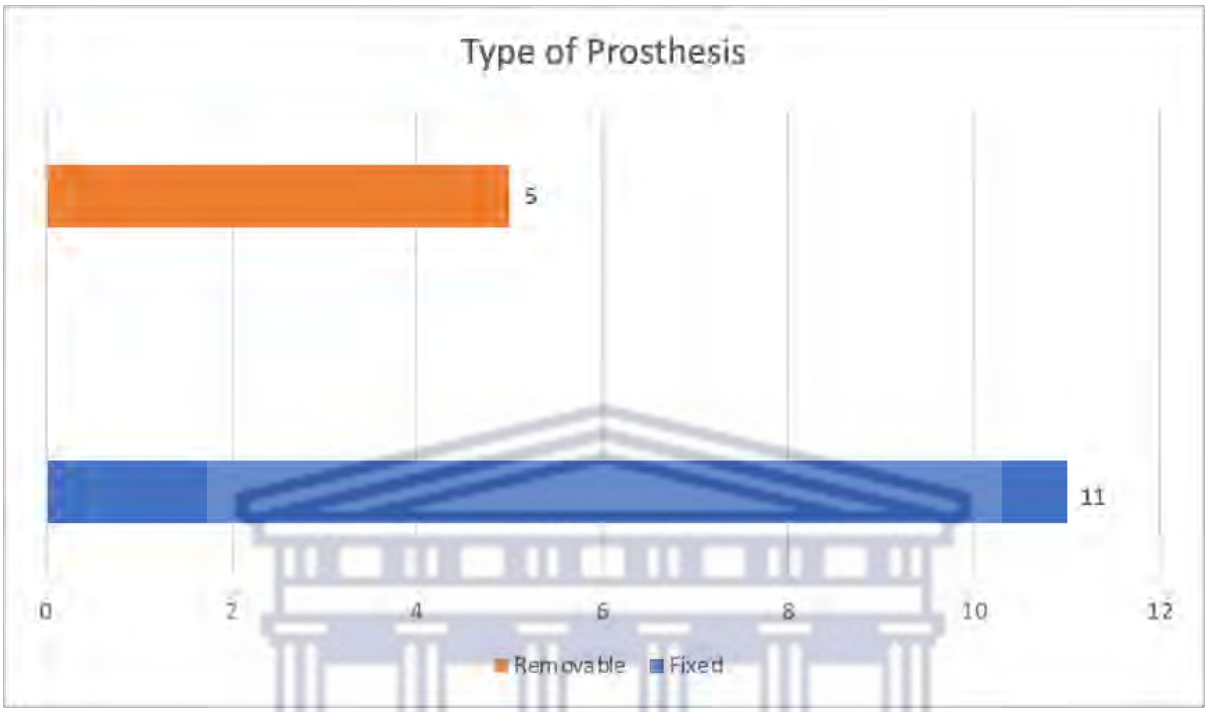

Figure 4. 32 Type of prosthesis in the peri-implant mucositis group.

Counts/frequency: Fixed (11, 68.8%), Removable (5, 31.3%).

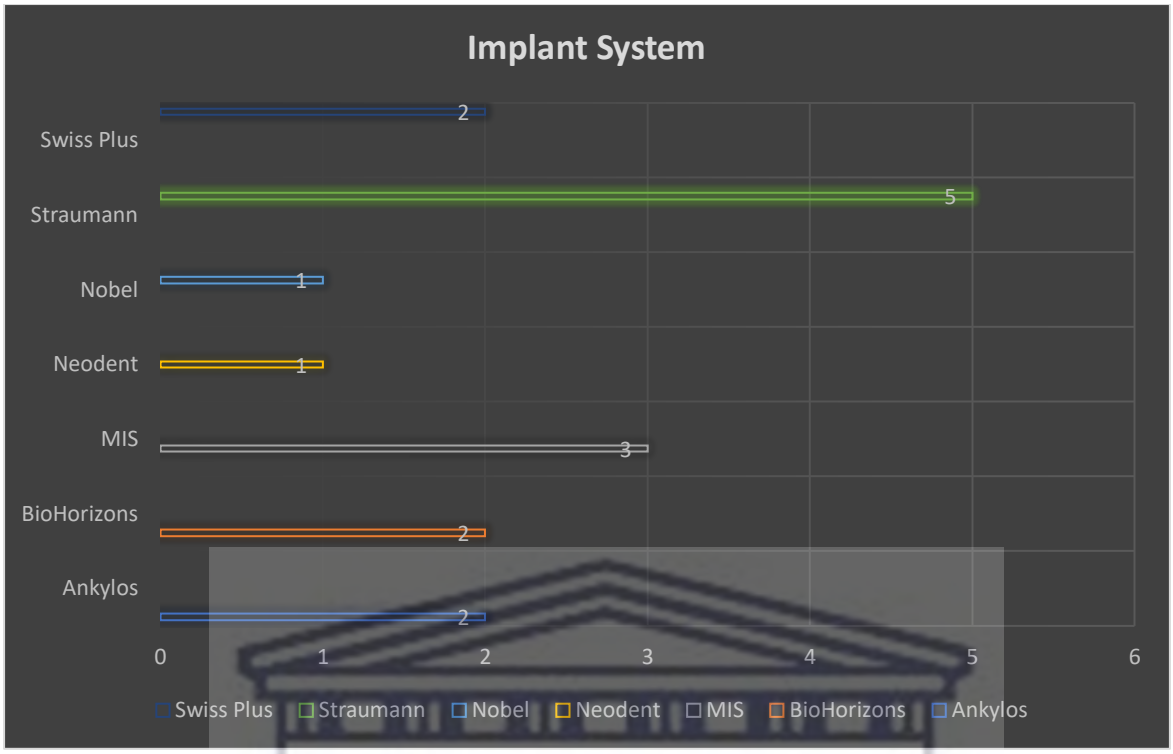

Figure 4. 33 mplant system distribution in the peri-implant mucositis.

Counts/frequency: Ankylos (2, 12.5%), BioHorizon (2, 12.5%), MIS (3, 18.8%), Neodent (1, 6.3%), Nobel (1, 6.3%), Straumann (5, 31.3%), Swiss Plus (2, 12.5%).

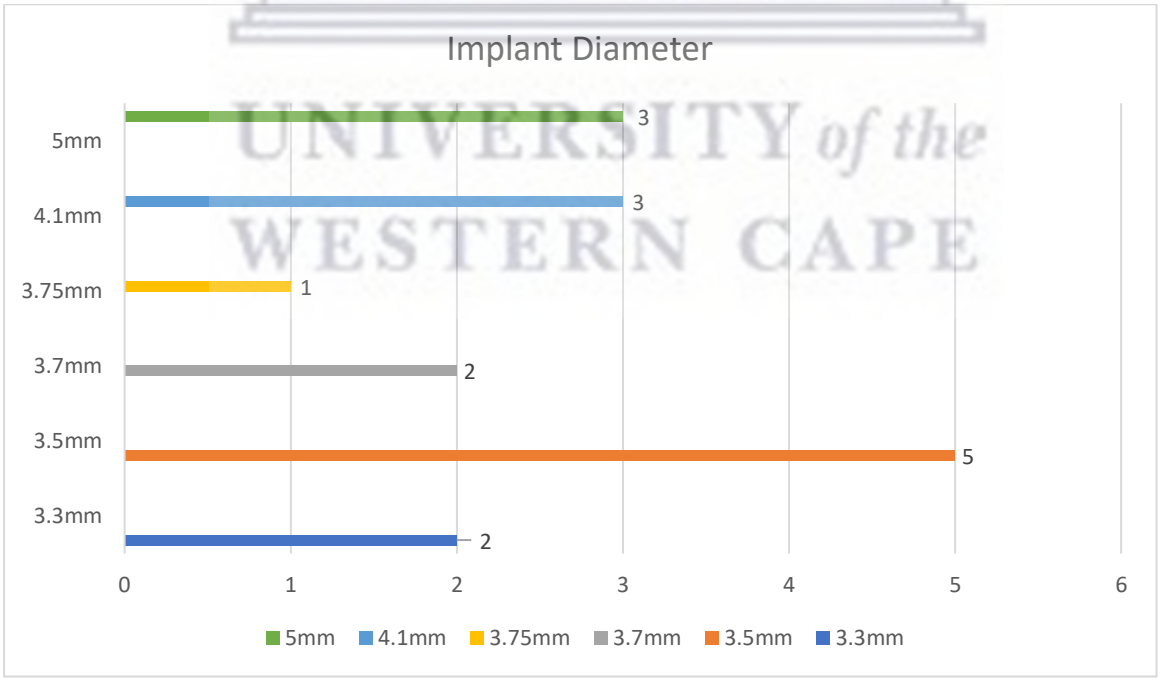

Figure 4. 34 Implant diameter distribution in the peri-implant mucositis group.

Counts/frequency: 3.3mm (4, 25.0%), 3.5mm (3, 18.8%), 3.7mm (2, 12.5%), 3.75mm (1, 6.3%), 4.1mm (3, 18.8%), 5mm (3, 18.8%).

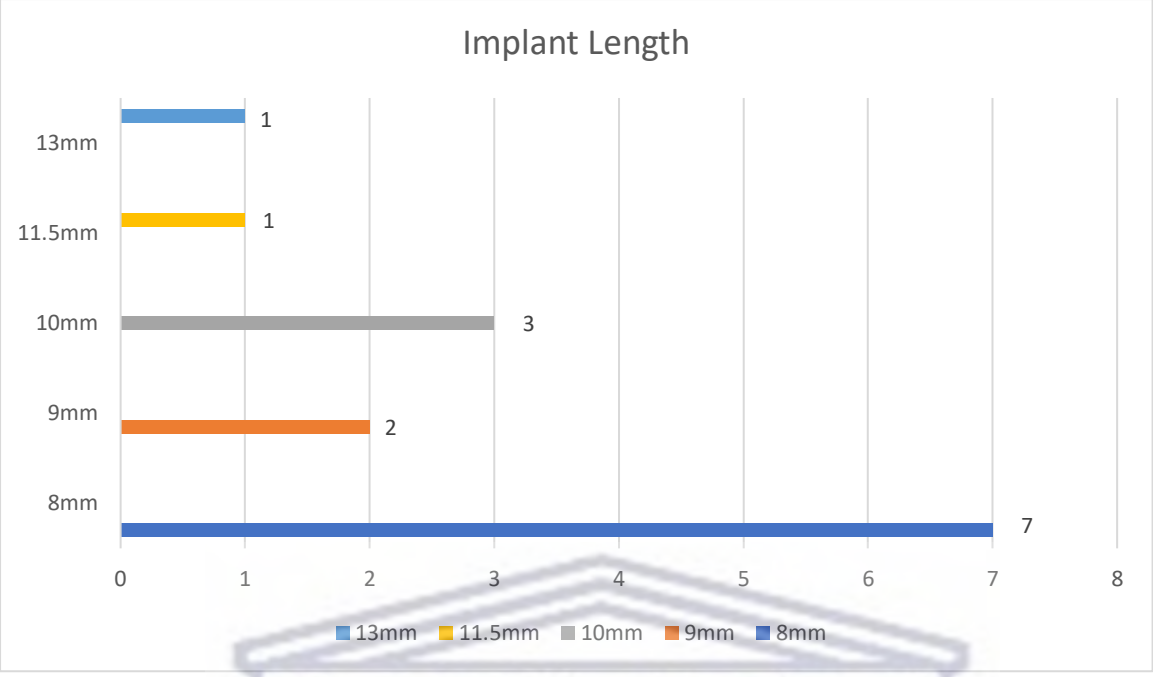

Figure 4. 35 Implant length distribution in the peri-implant mucositis group.

Counts/frequency: 8mm (7, 43.8%), 9mm (2, 12.5%), 10mm (3, 18.8%), 11.5mm (1, 6.3%), 13mm (1, 6.3%).

4.7.3 Systemic, Environmental and Local Factors in Peri-implant Mucositis

The factors associated with PIM are summarized in Figures 4.36, 4.37 and 4.38. A population of 6.3% had a history of periodontitis, and 12.5% were smokers. The restorative margin to bone crest of <1.5mm was found in 25%, while that of >1.5mm was present in 62.5%, see Figure 4.39. The compliance to supportive periodontal therapy is depicted in Figure 4.40, approximately 87.5% implants from the PIM were categorized as non-compliant.

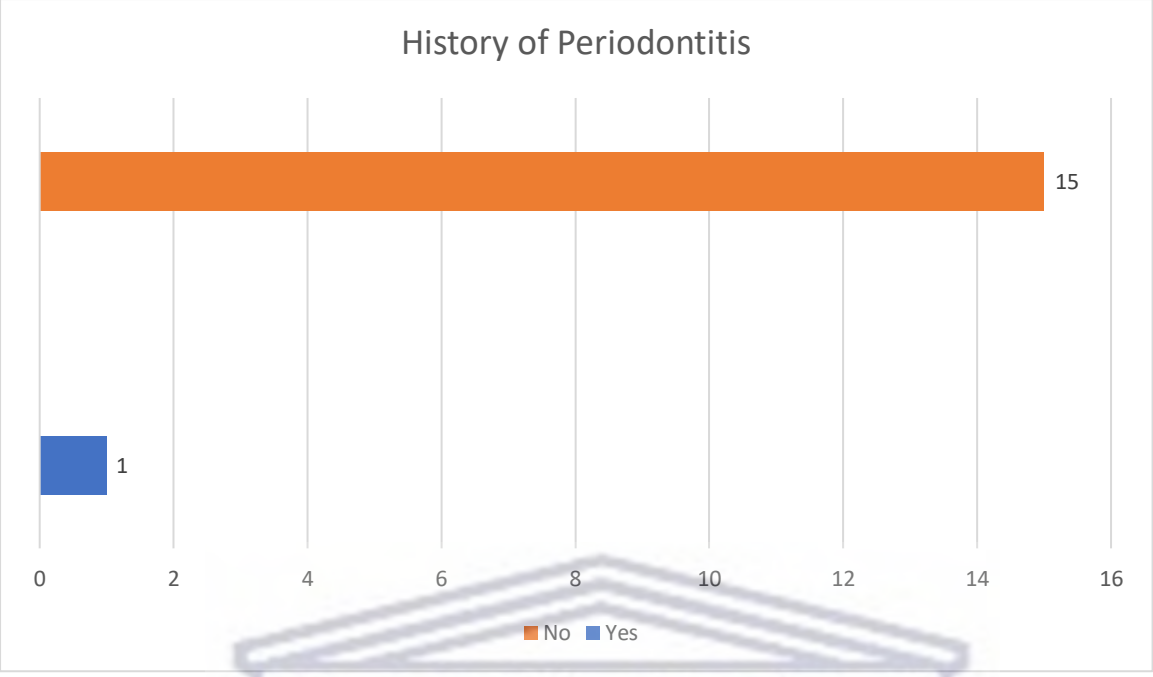

Figure 4. 36 History of periodontitis in the peri-implant mucositis group.

Counts/frequency: Yes (1, 6.3%), No (15, 93.8%).

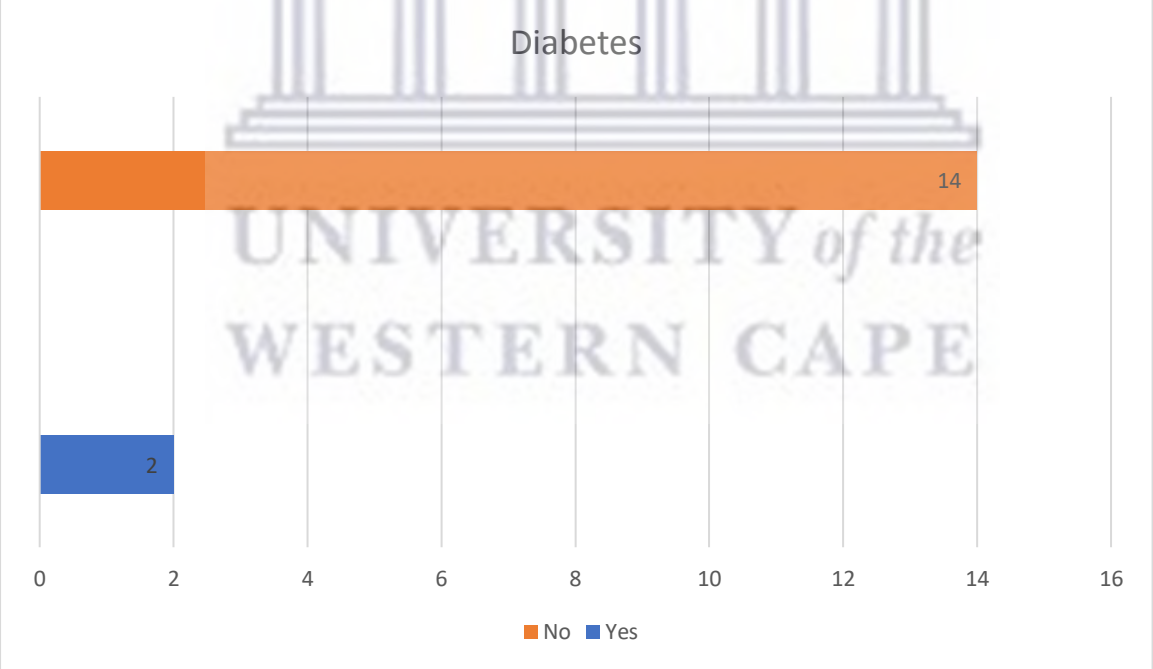

Figure 4. 37 Diabetes mellitus in the peri-implant mucositis group.

Counts/frequency: Yes (2, 12.5%), No (14, 87.5%).

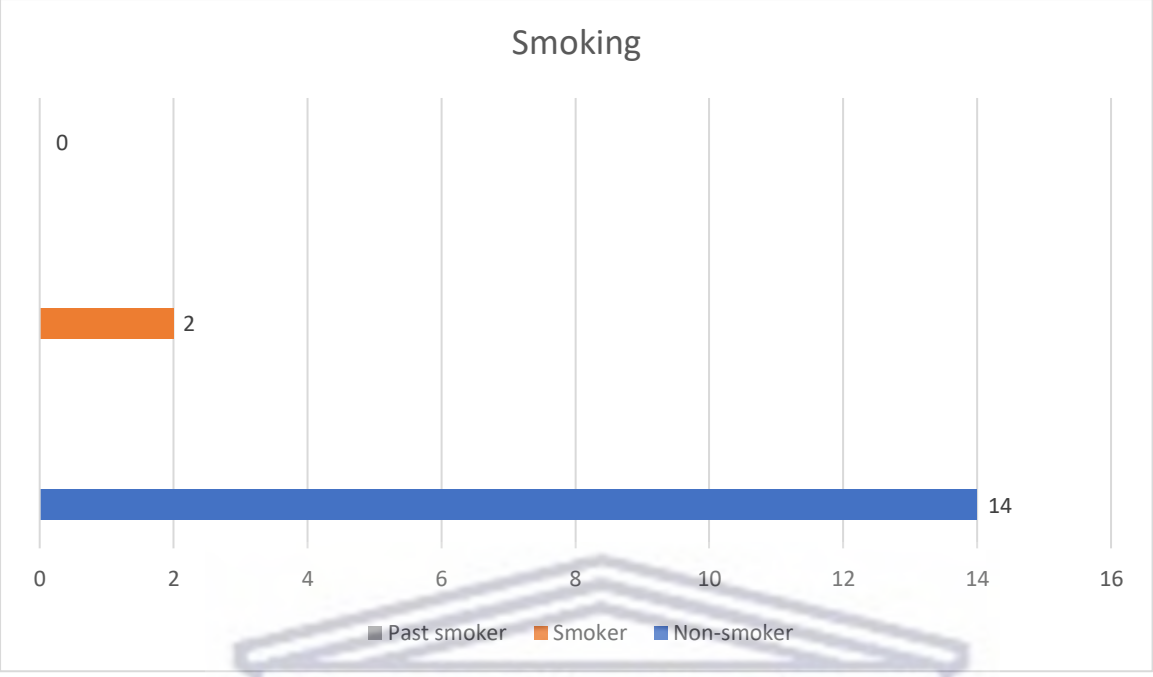

Figure 4. 38 Smoking in the peri-implant mucositis group.

Counts/frequency: Yes (2, 12.5%), No (14, 87.5%).

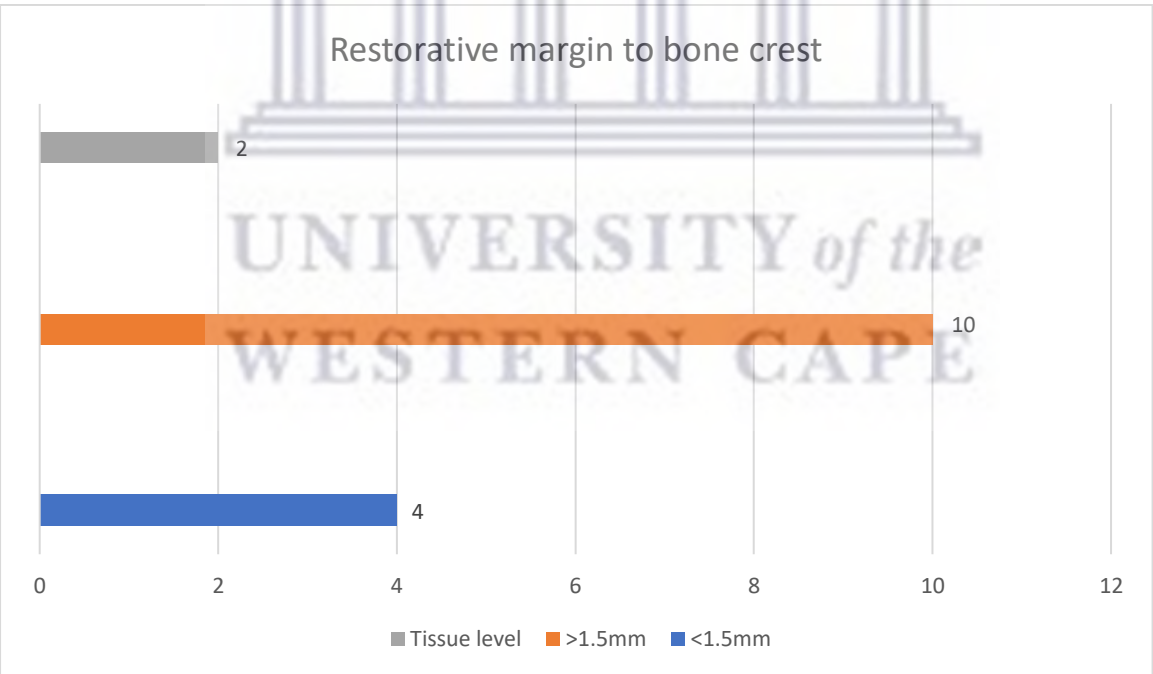

Figure 4. 39 Restorative margin to bone crest in the peri-implant mucositis group.

Counts/frequency: <1.5mm (4, 25.0%), >1.5mm (10, 62.5%), Tissue level (2, 12.5%).

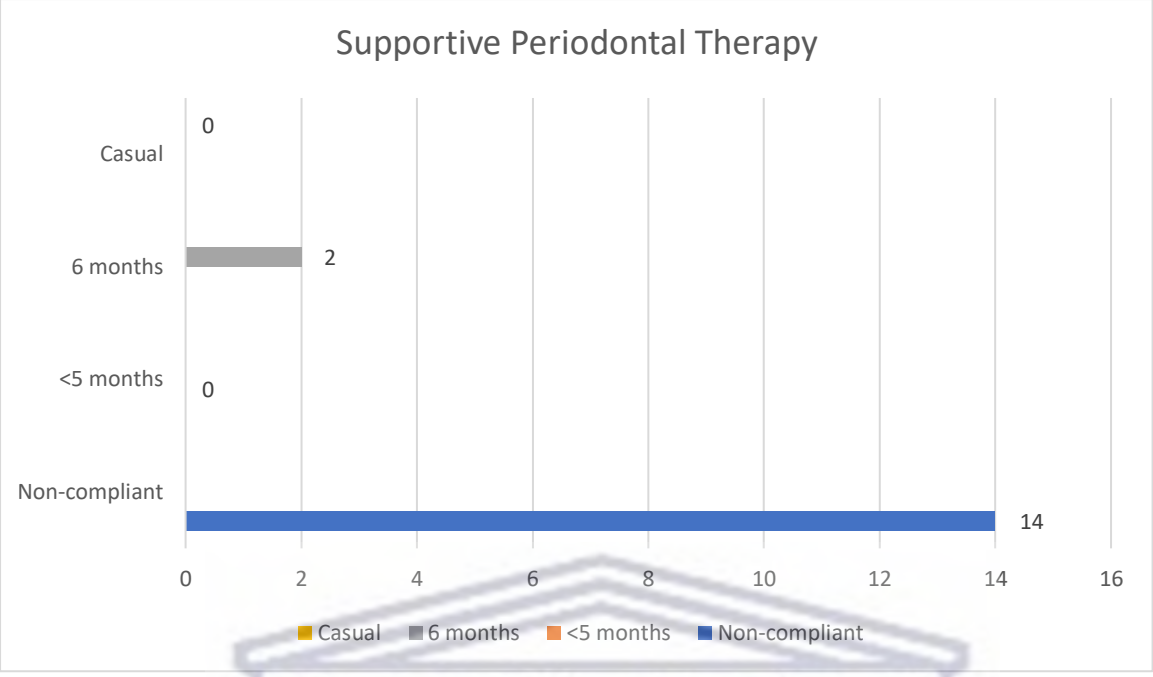

Figure 4. 40 Supportive periodontal therapy in the peri-implant mucositis group.

Counts/frequency: Non-compliant (14, 87.5%), <5 months (0, 0.0%), 6 months (2, 12.5%), Casual (0, 0.0%)

4.7.3 Clinical Finding in Peri-implant Mucositis

Edema and bleeding on probing was found in 68.8% implants while only 18.8% implants presented with erythema and BOP only, see Figure 4.41.

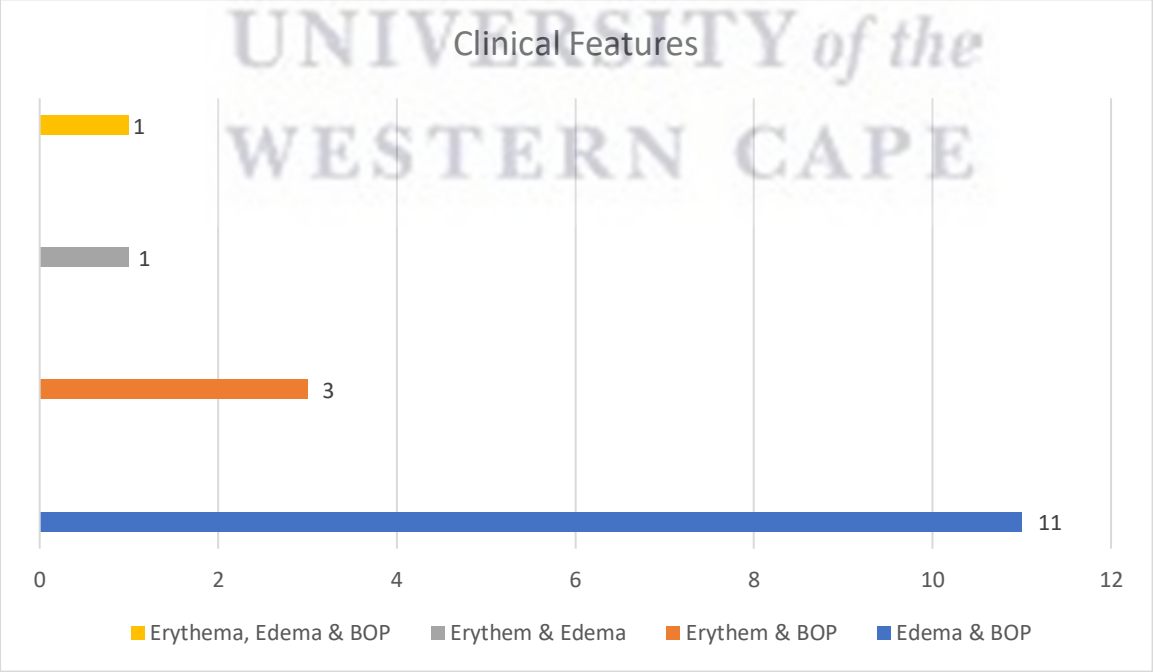

Figure 4. 41 Clinical features in the peri-implant mucositis group.

Counts/frequency: Edema and Bleeding on probing (11, 68.8%), Erythema and Bleeding on probing (3, 18.8%), Erythema and Edema (1, 6.3%), Erythema, Edema and Bleeding on probing (1, 6.3%).

4.8 Implant Level: Peri-implantitis Group

4.8.1 Peri-implantitis Demographics

Patient and implant age distribution in the PI group is depicted in Tables 4.6, 4.7, Figures 4.42, and 4.43. The patient age distribution ranged from 49.30 to 80.70 years, while the implant age ranged from 5.40 to 22.80 years. Females and bone level implants made up 100% individually in this group, and 66.7% of the bone level implants were in the posterior mandible, see Figure 4.44 and 4.45.

Table 4. 7 Patient age distribution in the Peri-implantitis group

Implant Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
12	49.30	80.70	69.64	68.55

n: number, y: years.

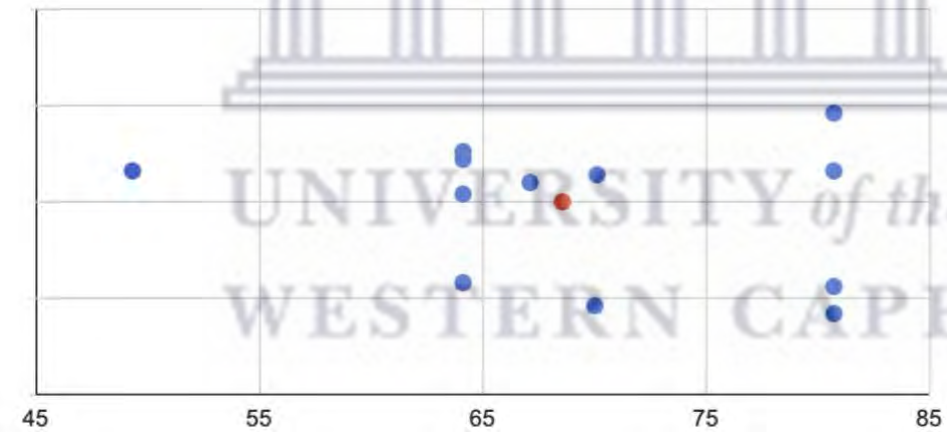

Figure 4. 42 Patient age distribution in the Peri-implantitis group

Median in red.

Table 4. 8 Implant age distribution in the Peri-implantitis group

Implant Total Count (n)	Minimum (y)	Maximum (y)	Mean (y)	Median (y)
12	5.40	22.80	12.62	9.35

n: number, y: years.

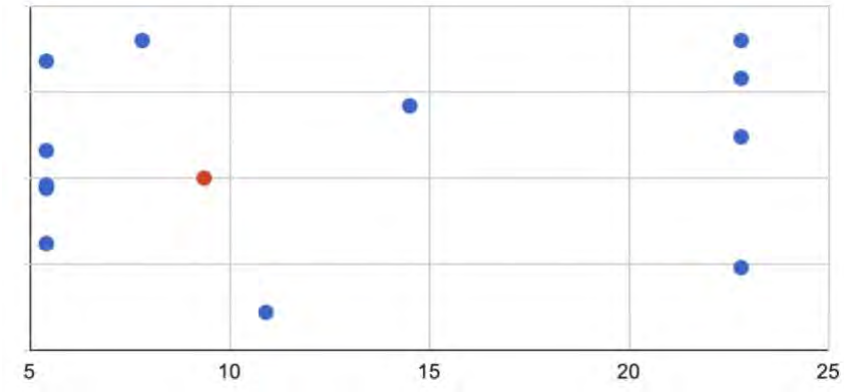

Figure 4. 43 Implant age distribution in the Peri-implantitis group

Median in red.

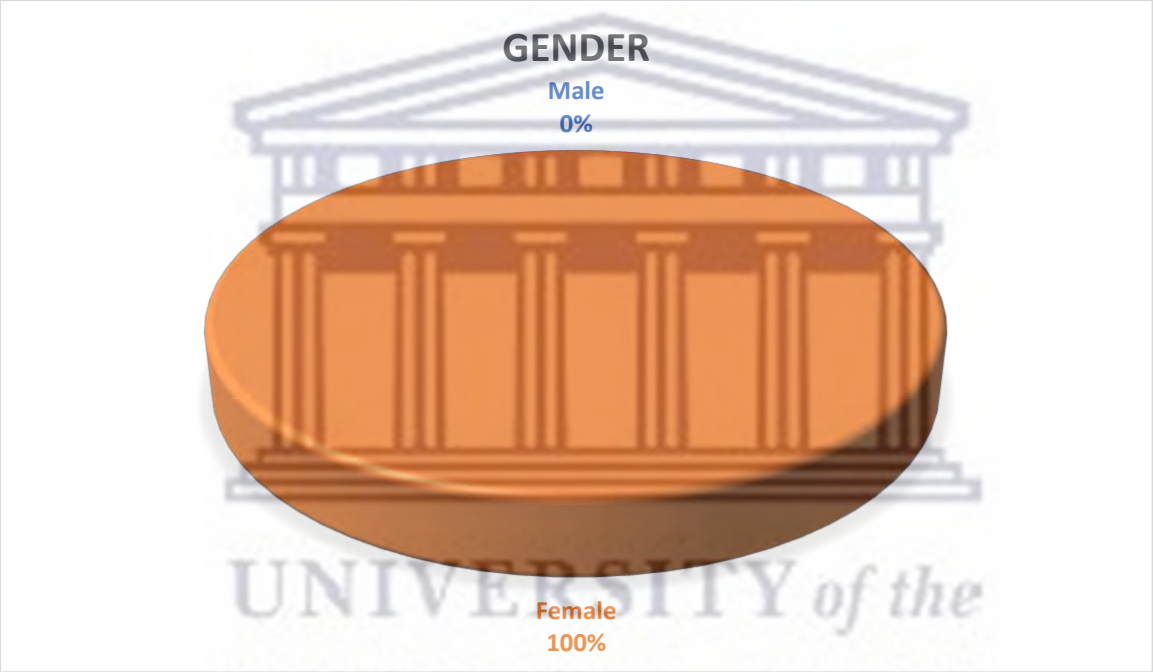

Figure 4. 44 Gender distribution in the Peri-implantitis group.

Counts/frequency: Female (12, 100%), Male (0, 0.0%).

4.8.2 Implant related factors

All the implants in the PI group were restored with fixed prostheses, and the implant system that was represented the most in this group was MIS and Zimmer at 31.3% individually, see Figure 4.46 and 4.47. Approximately 66.7% of the implants in this group were in the posterior mandible, see Figure 4.48. The implant diameter ranged between 3.25mm and 4.2mm, and the implant length ranged between 8mm and 13mm, see Figure 4.48 and 4.49.

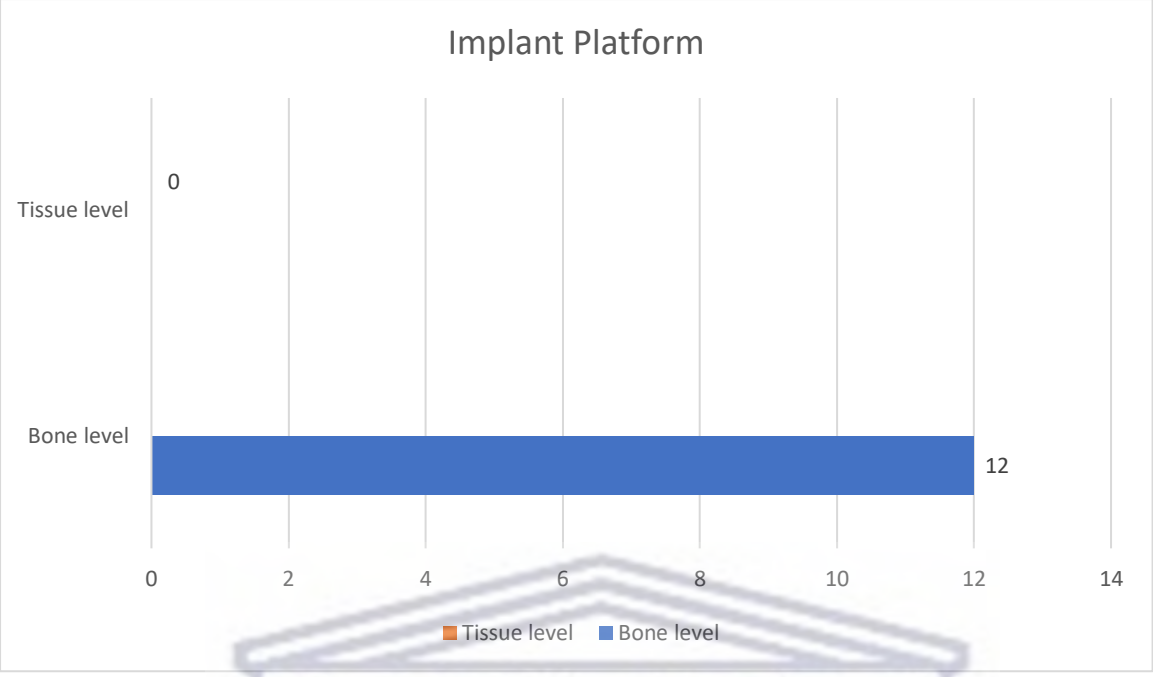

Figure 4. 45 Implant platform in the peri-implantitis group.

Counts/frequency: Bone level (12, 100%), Tissue level (0, 0.0%).

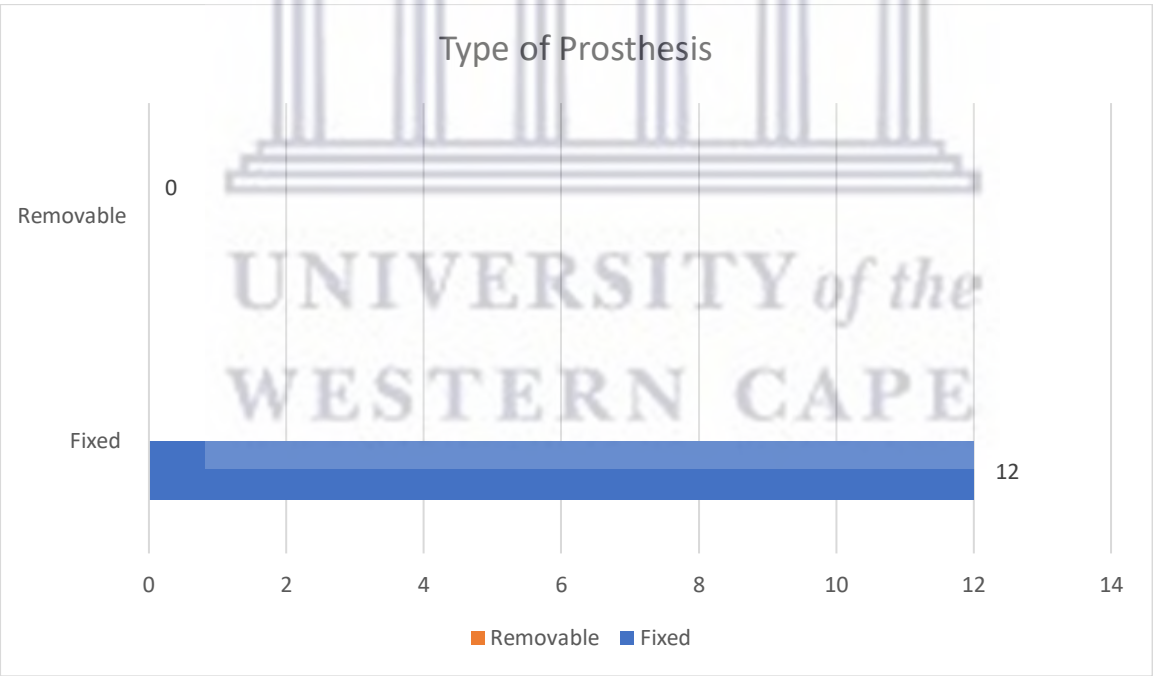

Figure 4. 46 Implant Prosthesis in the peri-implantitis group.

Counts/frequency: Fixed (12, 100.0%), Removable (0, 0.0%).

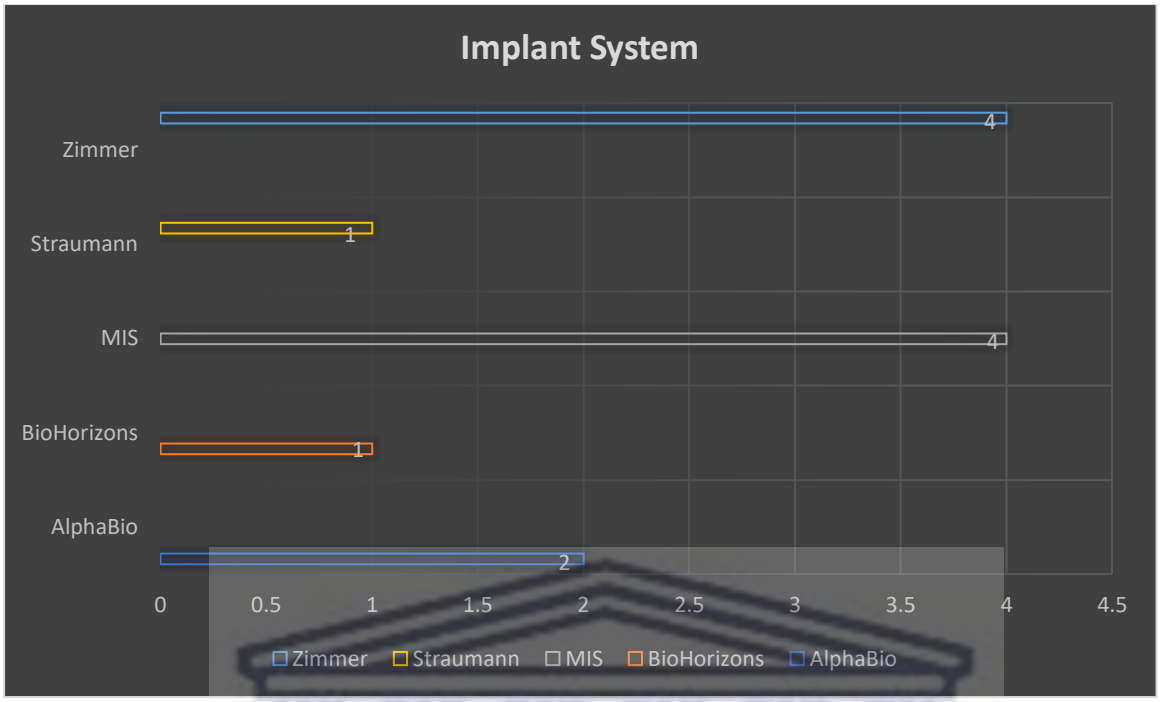

Figure 4. 47 Implant system distribution in the peri-implantitis group.

Counts/frequency: AlphaBio (2, 16.7%), BioHorizons (1, 8.3%), MIS (4, 33.3%), Straumann (1, 8.3%), Zimmer (4, 33.3%)

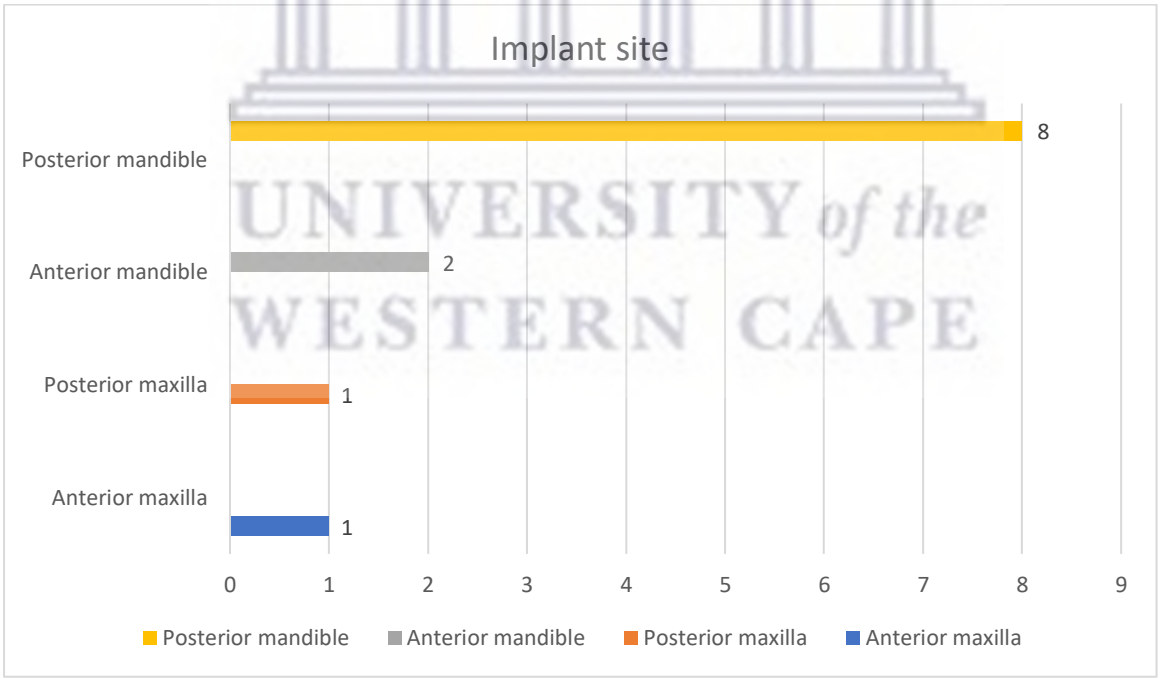

Figure 4. 48 Implant site in the peri-implantitis group.

Counts/frequency: Anterior maxilla (1, 8.3%), Posterior maxilla (1, 8.3%), Anterior mandible (2, 16.7%), Posterior mandible (8, 66.7%).

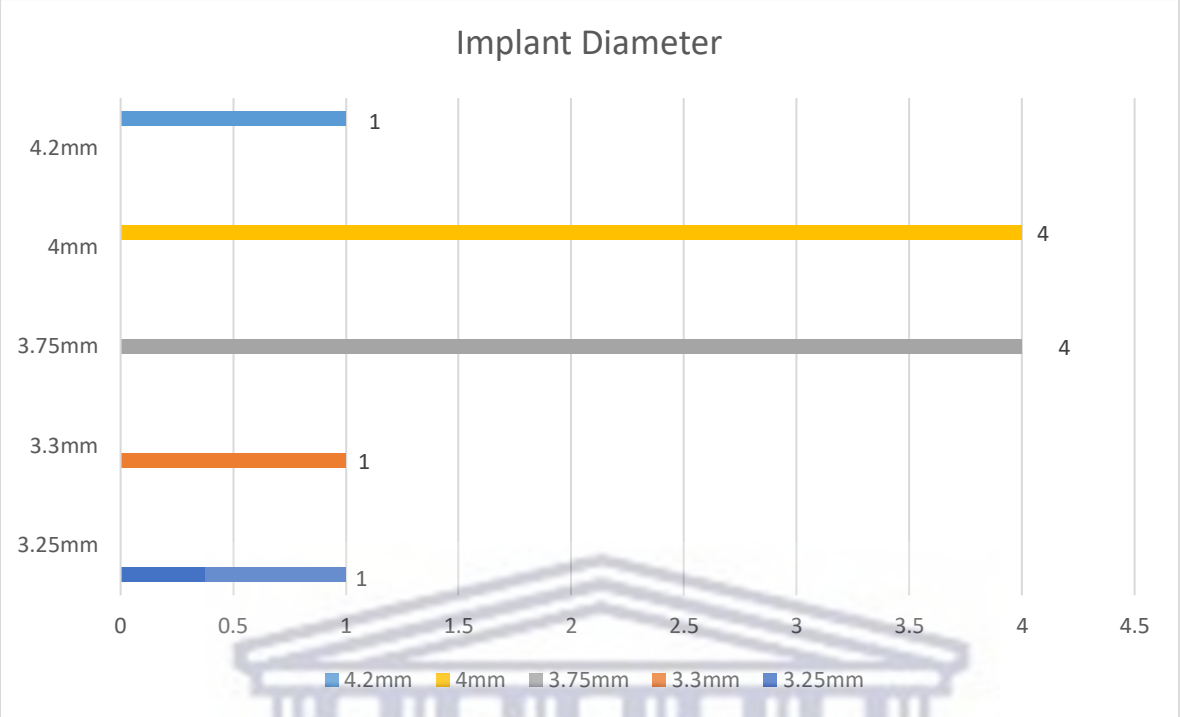

Figure 4. 49 Implant diameter distribution in the peri-implantitis group.

Counts/frequency: 3.25mm (1, 8.3%), 3.3mm (1, 8.3%), 3.75mm (4, 33.3%), 4.2mm (2, 16.7%).

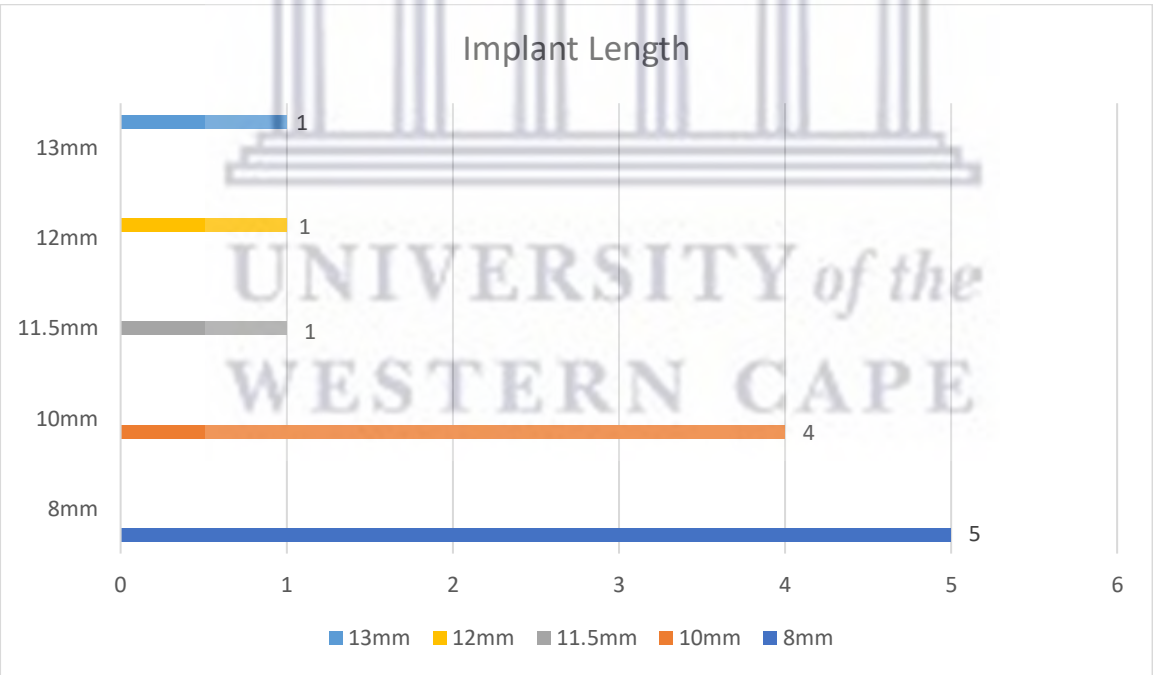

Figure 4.50: Implant length distribution in the peri-implantitis group.

Counts/frequency: 8mm (5, 41.7%), 10mm (4, 33.3%), 11.5mm (1, 8.3%), 12mm (1, 8.3%), 13mm (1, 8.3%).

4.8.3 Systemic, Environmental and Local Factors in the Peri-implantitis Group

The factors associated with PI are summarized in Figures 4.50, 4.51 and 4.52. A population of 50% had a history of periodontitis, 33.3% were diabetics with HbA1C >7% and 100% were smokers. The restorative margin to bone crest of <1.5mm was found in 66.7%, while that of >1.5mm was represented by 33.3%, see Figure 4.53. The compliance to supportive periodontal therapy is depicted in Figure 4.54 with 91.7% implants from the PI group were categorized as casual.

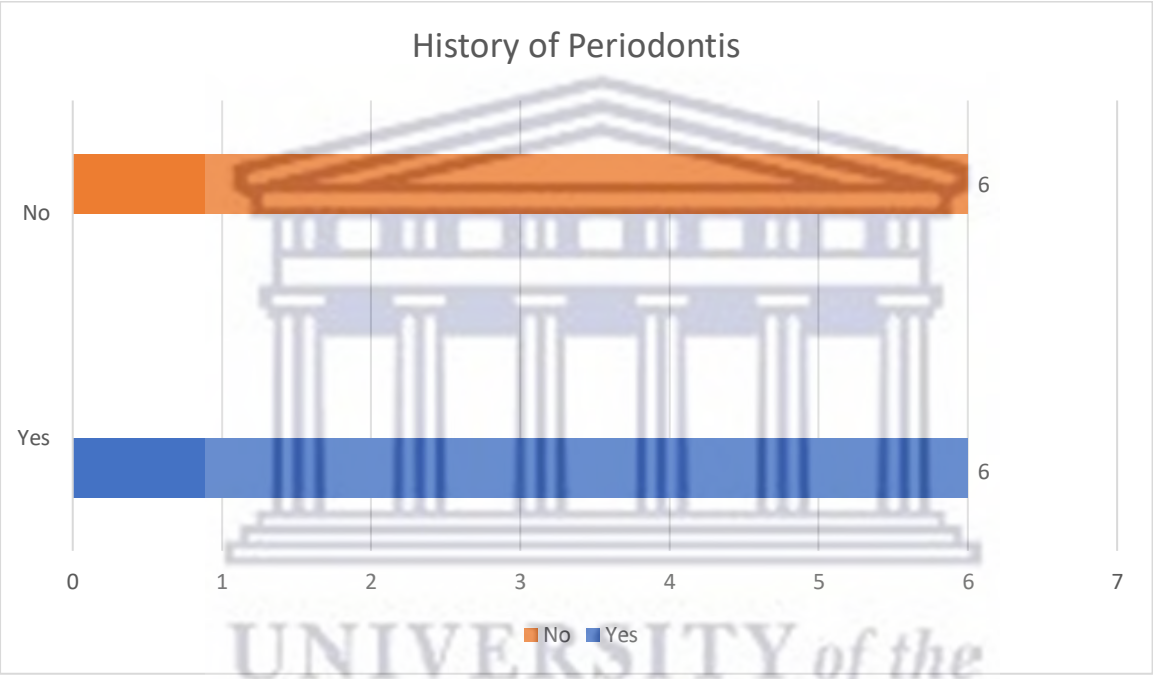

Figure 4. 51 History of periodontitis in the peri-implantitis group.

Counts/frequency: Yes (6, 50%), No (6, 50%).

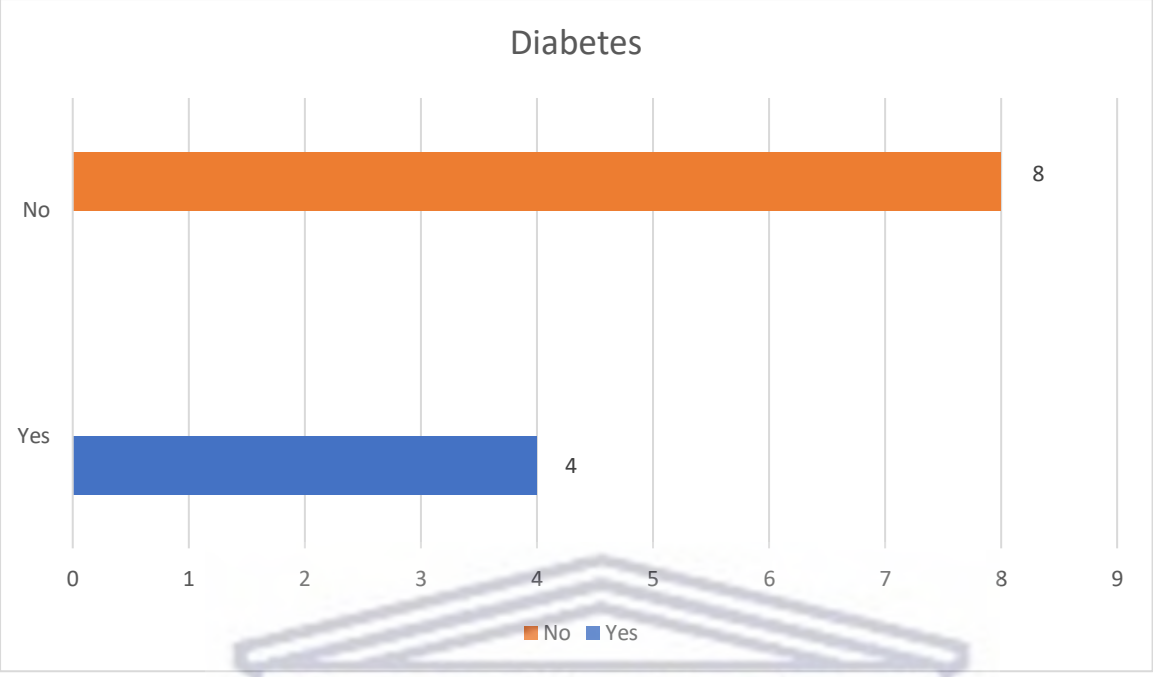

Figure 4. 52 Diabetes mellitus in the peri-implantitis group.

Counts/frequency: Yes (4, 33.3%), No (8, 66.7%).

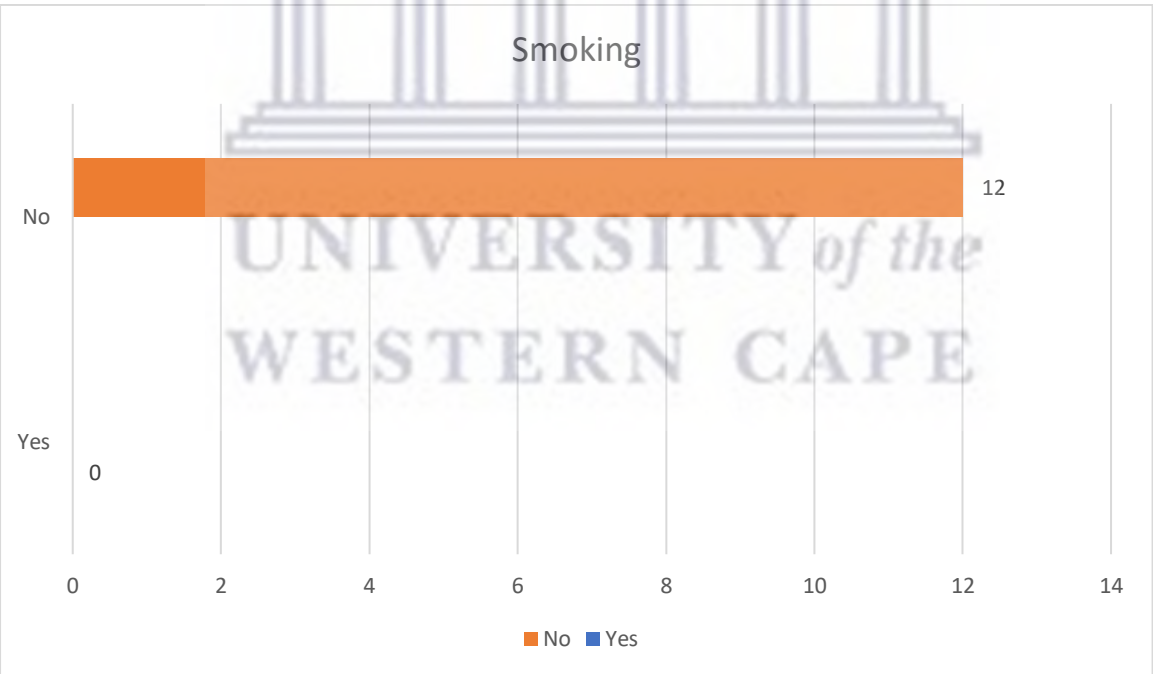

Figure 4. 53 Smoking in the peri-implantitis group.

Counts/frequency: Yes (0, 0.0%), No (12, 100%).

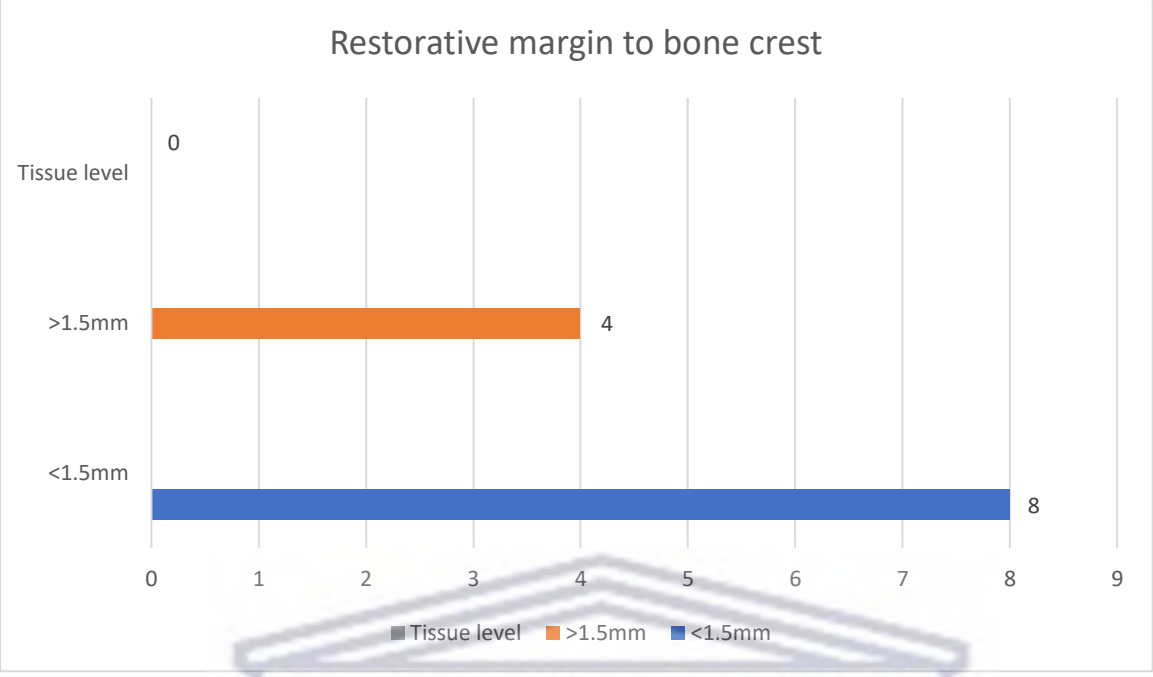

Figure 4. 54 Restorative margin to bone crest in the peri-implantitis group.

Counts/frequency: <1.5mm (8, 66.7%), >1.5mm (4, 33.3%), Tissue level (0, 0.0%).

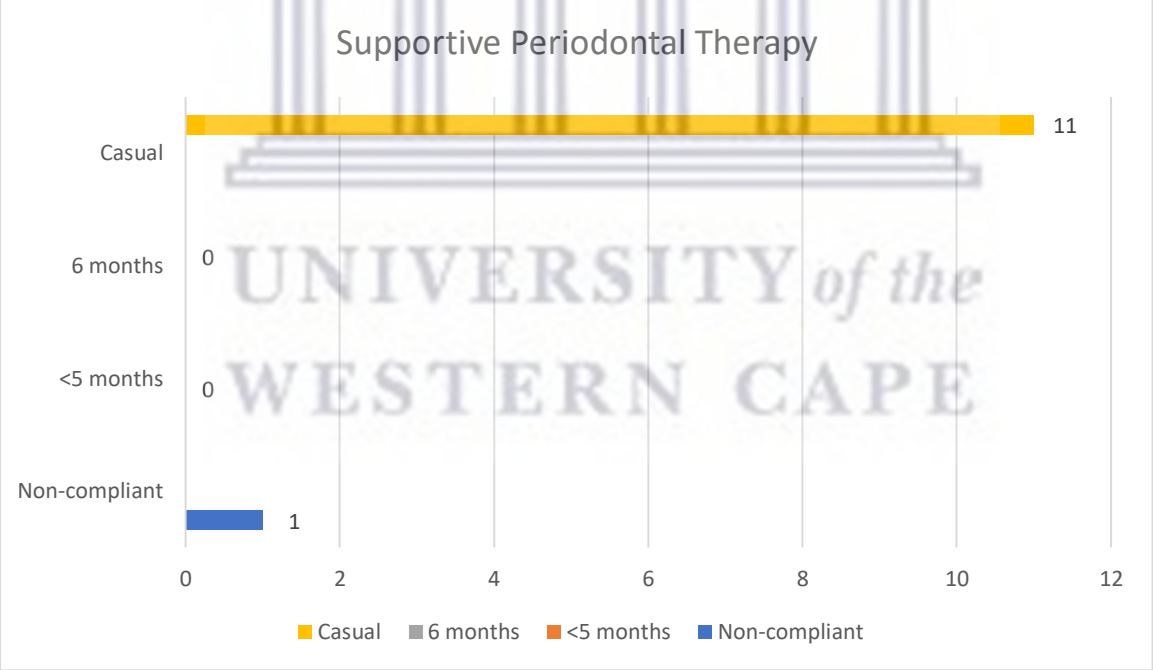

Figure 4. 55 Supportive periodontal therapy in the peri-implantitis group.

Counts/frequency: Casual (11, 91.7%), Non-compliant (1, 8.3%).

4.8.4 Clinical Findings in the Peri-implantitis Group

Clinical findings in 41.7% of the implants in this group were erythema, edema, bleeding on probing, suppuration and bone loss ≥ 3mm while 25.0% presented with only bone loss of ≥ 3mm, see Figure 4.56.

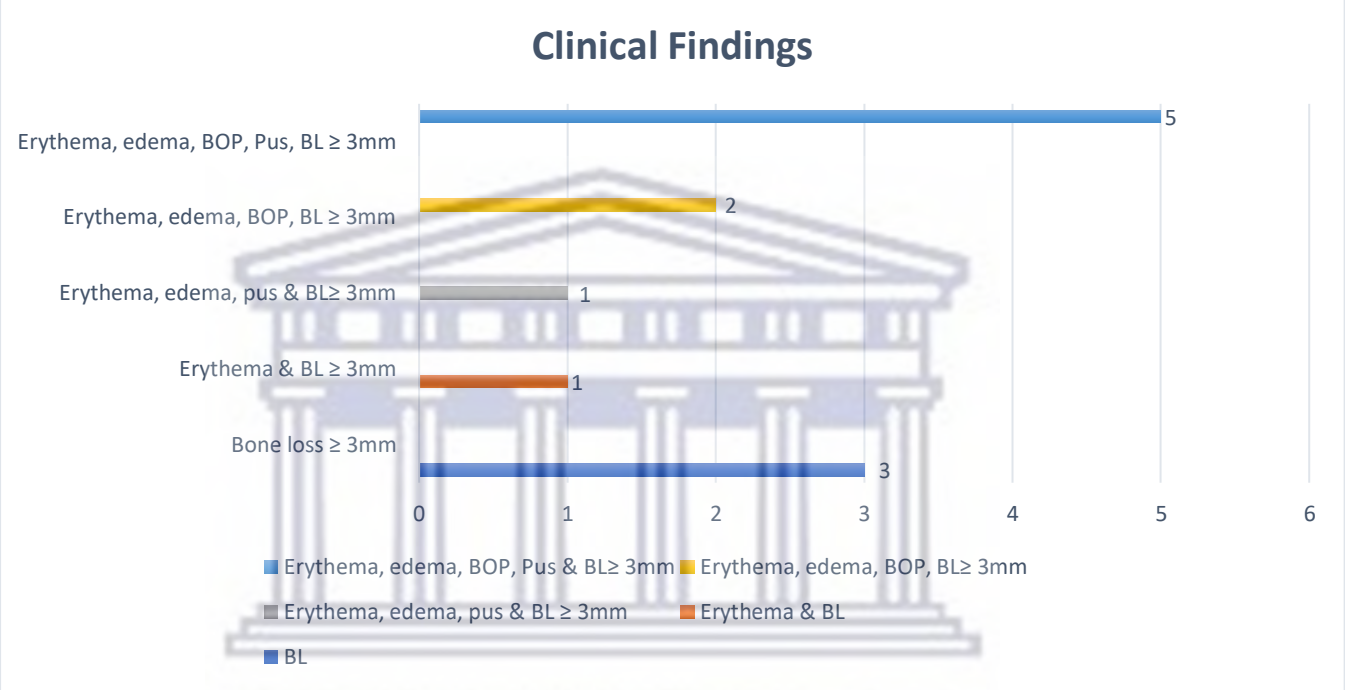

Figure 4. 56 Clinical findings.

Counts/frequency: Bone loss ≥ 3mm (3, 25.0%), Erythema and bone loss ≥ 3mm (1, 8.3%), Erythema, edema, suppuration, and bone loss (1, 8.3%), Erythema, edema, bleeding on probing and bone loss ≥ 3mm (2, 16.7%), Erythema, edema, bleeding on probing, suppuration, and bone loss ≥ 3mm (5, 41.7%). BOP: Bleeding on probing, BL: Bone loss.

4.9 Summary of the results

Table 4. 9 Cross-tabulation of peri-implant status and systemic risk factors at implant level

Total:	Peri-implant Health **(109)**	Peri-implant mucositis **(16)**	Peri-implantitis **(12)**
Gender			
Female	**66** (60.6%)	**8** (50.0%)	**12** (100%)
Male	**43** (39.4%)	**8** (50.0%)	**0**
Type of prosthesis			
Fixed prosthesis	**96** (88.1%)	**11** (68.8%)	**12**
Removable prosthesis	**13** (11.9%)	**5** (31.3%)	**0**
Site			
Anterior maxilla	**31** (28.24%)	**1** (6.3%)	**1** (8.3%)
Posterior maxilla	**38** (34.9%)	**7** (43.8%)	**1** (8.3%)
Anterior mandible	**13** (11.9%)	**5** (31.3%)	**2** (16.7%)
Posterior mandible	**27** (24.8%)	**3** 18.8%)	**8** (66.7%)
Risk Factors			

Smoker	**13** (11.9%)	**2** (12.5%)	**0** (0.0%)
Past smoker	**4** (3.7%)	**0** (0.0%)	
Non-smoker	**92** (84.4%)	**14** (87.5%)	**12** (100%)
Periodontitis	**31** (28.4%)	**1** (6,3%)	**6** (50.0%)
No Periodontitis	78 (71.6%)	**15** (93.8)	**6** (50.0%)
Diabetes mellitus	**0**	**2** (12.5%)	**4 (33.3%)** HbA1C >7%
Diabetes mellitus and Periodontitis	**0**	**0**	**4**
Smoking and Periodontitis	**0**	**0**	**0**
Smoking, Periodontitis & Diabetes mellitus	**0**	**0**	**0**
Bone crest to Resto margin <1.5mm	**41** (37.6%)	**4** (25.0%)	**8** (66.7%)
>1.5mm	**55** (50.5%)	**10** (62.5%)	**4** (33.3%)
Tissue level	**13** (11.9%)	**2** (12.5%)	**0**
Supportive periodontal therapy			
Non-compliant	**78** (71.6%)	**14** (87.5%)	**1** (8.3%)

<5 months	**4** (3.7%)		
6 monthly	**15** (13.8%)	**2** (12.5%)	**0**
Casual	**12** (11.0%)	**0**	**11** (91.7%)

CHAPTER 5

5. Discussion

The overall prevalence of PIH, PIM, and PI at a subject level in this university-based sample was 66.7%, 19.0%, and 14.3% respectively. The overall prevalence of PIH, PIM, and PI at the implant level was 79.6%, 11.7%, and 8.8% respectively. A positive correlation in the PI was demonstrated with the function time of the implants, history of periodontitis, casual SPT visits, non-compliance to SPT, and a distance of <1.5mm from restorative margin to bone crest. This study could not demonstrate an association in the PI group with smoking status, and diabetes. This was mainly due to the lack of a significant ratio of smokers and non-smokers, patients who are diabetic and those who are not diabetic.

5.1 Level of reporting

The prevalence of peri-implant health and disease was reported at both subject or patient level and implant level in this study. There has been discrepancies noted in studies that reported only at one level, i.e. either at implant or subject level, prevalence were higher in studies that reported only on patient level. Studies that reported only on implant level create a challenge in the estimation of the global impact of peri-implant disease. The results of this current study were derived from the case definitions that were published in 2018 (Derks et al., 2016; Romandini, Berglundh, et al., 2021; Sanz et al., 2012; Berglundh et al., 2018).

5.2 Prevalence

The prevalence of peri-implant disease is reported globally but we lack similar studies in South Africa. Only one study was performed in South Africa evaluating the prevalence of PIM as well as the associated risk factors. This cross-sectional cohort study had a total of 74 patients or subjects, and PIM was diagnosed in 52 subjects, and it was performed in the same setting as the current study i.e., Tygerberg Dental Hospital, Dental Faculty of the University of the Western Cape. The prevalence of PIM was 70.3% at a subject level. This study was performed prior to the recommendations of reporting at both subject and implant level (Stander et al., 2019).

In the current study PIM was diagnosed in 19.0% at subject level and 11.7% at implant level. PI was diagnosed in 14.3% at subject level and 8.8% at implant level. The current study also

reported on peri-implant health, it was found to be 66.7% at subject level and 79.6% at implant level. The prevalence of health and disease in this study was within the range of previous calculations from meta-analysis and systemic reviews. Worldwide PIM prevalence ranges from 19% to 65% at subject level while peri-implantitis ranges from 1 to 47%. Another meta-analysis and systemic review reported a range of 32% to 54% for PIM and a range of 14% to 30% for PI. The estimated mean prevalence reported for PIM and PI were 43% and 22% respectively (Derks & Tomasi, 2015). The estimated prevalence of PIM at subject level was 63.4% and at implant level it was 30.7%. The estimated prevalence of PI at subject and implant level was 18.8% and 9.6% respectively (Atieh et al., 2012).
A cross-sectional analysis study limited to two-piece implants with an internal connection reported peri-implant health in 44.5% subjects and 41.3% implants. PIM was found in 41.6% subjects and 41.3% implants, while PI was diagnosed in 13.9% subjects and 7.6% implants (Schwarz et al., 2018a). A study by Marrone reported PIM and PI at subject level to be 31% and 37%, respectively while at implant level PIM and PI were 38% and 23% respectively. PIH was reported to be 39% at implant level (Marrone et al., 2013). In the current study PIM was diagnosed in 19.0% at subject level and 11.7% at implant level. PI was diagnosed in 14.3% at subject level and 8.8% at implant level. The current study also reported on PIH, it was found to be 66.7% at subject level and 79.6% at implant level. The prevalence of health and disease in this study was within the range of previous calculations from meta-analysis and systemic reviews. Worldwide PIM prevalence ranges from 19% to 65% at subject level while PI ranges from 1 to 47%. Another meta-analysis and systemic review reported a range of 32% to 54% for PIM and a range of 14% to 30% for PI. The estimated mean prevalence reported for PIM and PI were 43% and 22% respectively (Derks & Tomasi, 2015). The estimated prevalence of PIM at subject level was 63.4% and at implant level it was 30.7%. The estimated prevalence of PI at subject and implant level was 18.8% and 9.6% respectively (Atieh et al., 2012)

5.3 Implant age range

The implant age range observed in this study was 0.9 to 15.2 years in peri-implant mucositis, and 5.4 to 22.8 years in PI. Studies have reported a positive correlation with function time in PI but no positive correlation in peri-implant mucositis. PI is reported to be less likely to be found in the initial 5 years of function. This observation was similar to the current study (Renvert & Polyzois, 2015; Ting et al., 2018). It was also noted that studies performed in

implants with a short function time may result in underestimation of the prevalence (Derks & Tomasi, 2015).

5.4 Implant system

The current study comprised of 137 implants from 15 different implant systems. There was no correlation between a specific implant system and peri-implant disease in the current study. Peri-implant disease in this study is associated with host susceptibility and risk factors, it is not system-related. There are studies that demonstrated a prevalence related to an implant system but with further assessment of the study it is more the characteristics of the implant surface that might favor retention of plaque than the actual implant system that influences disease (Ferreira et al., 2006).

5.5 Implant diameter and length

Implant diameter was not found to be associated with the development of peri-implant mucositis and peri-implantitis in this current study. Literature suggests that the implant site or location might be a possible risk factor rather than implant dimension (Daubert et al., 2015).

5.6 Implant site

The impact of the anatomical site or location of the implant on the risk of developing disease is unclear (Rodrigo et al., 2018). The current study comprised of a total of 137 implants, with 65 implants in the maxilla and 59 in the mandible, while the implants in the anterior maxilla and anterior mandible were 33 and 20 respectively. The current study demonstrated a higher prevalence of PIM in relation to the anatomical site or location to be in the posterior maxilla (43.8%).

The study also demonstrated a higher prevalence of peri-implantitis in relation to the anatomical site to be in the mandible than the maxilla, and the posterior mandible (66.7%) had a higher prevalence than the posterior maxilla (8.3%). Dalago et al reported a similar prevalence of PI, with a positive association between PI and the posterior mandible (Dalago et al., 2017). Rodrigo et al mentioned that the probability of developing PI in implants in the anterior mandible to be 4 to 5 times higher. Other studies that reported on PI in relation to the anatomical site of implants demonstrated a higher prevalence of PI in the maxilla than the mandible, 38.3% and 28.9% respectively. The prevalence between the anterior maxilla and

anterior mandible was rated at 21% (10.8% to 55.2% range) and 22.6% (4.54% to 90% range) respectively. The prevalence between the posterior maxilla and posterior mandible was rated at 18% (5.2% to 46.4% range) and 19% (7.2% to 48.6%) respectively. The data thus suggests that the anterior maxilla and mandible are the most prevalent regions for peri-implantitis. The posterior regions have advantages over the anterior regions. Bone density is higher in the anterior than in the posterior regions and a cortical bone that is thicker in the posterior regions than in the anterior regions. Occlusal forces are dissipated well with subsequent less stress around the implants with a thick cortical bone and less bone density. The masticatory loads in the posterior implants are in the vertical axis, while those in the anterior are in the horizontal direction. The latter masticatory loads received by an implant with a small diameter surrounded by a thin cortical bone results in compromised vascularity and marginal bone loss. Narrow diameter implants are used in the anterior regions as result of the anatomical characteristics and bone resorption pattern in the anterior regions. These are the regions that receive oblique forces dominantly (Moraschini et al., 2022; Rodrigo et al., 2018) .

5.9 Periodontitis

The patients with a history of periodontitis made up 26.2% of the study population, and 2.3% of the study population had a combination of periodontitis and diabetes. Subjects with the history of periodontitis in the PIM and PI group were 6.3% and 50% respectively in the current study. The subjects with the history of periodontitis in the PI group (50%) also presented with other risk factors (i.e., inadequate supportive therapy and uncontrolled diabetes or hyperglycemia) and none presented with only the history of periodontitis. There was a positive correlation between a history of periodontitis and PI in this study.
Literature has demonstrated that patients that are not periodontally stable have an increased risk of PI. There is higher success and survival rates in groups who do not have a history of periodontitis compared to groups with a history of periodontitis (Marrone et al., 2013). A study by Marrone demonstrated a higher prevalence of PI in subjects with a history of periodontitis compared to those with no history of periodontitis but there was no statistical significance of this finding (Marrone et al., 2013).
The reason for the aforementioned is the microbial plaque accumulation and the host response to the microbial challenge. It has been demonstrated that periodontal pathogens colonize the peri-implant sites approximately 3-6 months subsequent to placement of

implants and abutment connection. Thus, the microbial composition around teeth is reflected in the peri-implant sites, highlighting the significance of eliminating the etiological factor for peri-implant infection in these patients (Heitz-Mayfield et al., 2018; Karoussis et al., 2003). These factors outline the significance of consistent supportive therapy that is structured at correct time intervals to reduce the risk of recolonization of the peri-implant sites by the periodontal pathogens. Maintenance of health subsequent to effective periodontal therapy is important for the long-term survival and success of implants (Karoussis et al., 2003).

5.10 Diabetes Mellitus

The study performed by Stander (in the same institution as the current study) reported diabetes in 5% from the total study population, and diabetic patients in the PIM group were only 4%. The association of PIM with diabetes was found to be of no statistical significance in the study performed by Stander (Stander et al., 2019).

Diabetics made up 4.8% of the current study population. Subjects with diabetes in the PIM and PI group were 12.5% and 4% respectively in the current study. Only 2.3% of the subjects and 33.3% implants diagnosed with PI had a combination of periodontitis and diabetes, and the HbA1C was >7% and there was presence of active disease, i.e. PPD>4mm with BOP in this group. Furthermore, with regards to supportive therapy or maintenance they were categorized as casual. Thus, the subjects presented with hyperglycemia while periodontally unstable and lacking adequate supportive therapy.

Diabetes mellitus is associated with impaired function of macrophage and neutrophil, altered production of collagen, enhanced activity of collagenases and a heightened state of inflammation (Salvi et al., 2010). Diabetes mellitus is regarded as a risk factor for periodontal disease, namely gingivitis and periodontitis. It is not merely diabetes but hyperglycemia that has a significant role in the pathogenesis of these conditions (Monje et al., 2017).

Risk factors for both periodontitis and diabetes mellitus are largely identical, these include sedentary lifestyles, smoking, obesity, and genetic predisposition for impaired immune responses. Periodontitis and diabetes demonstrate enhanced or exaggerated inflammation and an immune response that is impaired. The consistent observation in periodontitis and type 2 diabetes includes elevated IL-6, IL-1β and C-reactive protein (CRP). Chronic hyperglycemia enhances the production of advanced glycation end-products and the expression of tissue-degrading enzymes in gingiva that is inflamed (Kocher et al., 2018). Hyperglycemia plays an

important role in the pathological process of periodontal disease and biologic complications of implants. Chronic complications of diabetes are as result of chronic hyperglycemia (Kocher et al., 2018; Monje et al., 2017).

The mechanism in periodontal and peri-implant disease includes the bacterial biofilm eliciting a continual chronic inflammatory host response, with the hyperglycemia and periodontitis affecting each other adversely and mutually. The inflammation is exaggerated and long-standing resulting in persistent resorption of bone, degradation and impaired repair or healing of periodontal tissue (Monje et al., 2017). Individuals with periodontitis and diabetes mellitus demonstrate decreased anti-inflammatory markers, adiponectin and IL-4 and increased CRP, a marker of acute inflammation. These circulating pro-inflammatory mediators play a role in both periodontitis and diabetes. The pro-inflammatory pathways activate oxidative stress and vice versa in both conditions (Hasturk & Kantarci, 2015). The receptors for advanced glycation end products (RAGEs) are activated by advanced glycation end products (AGEs) which is produced by elevated glucose levels in the bloodstream, this interaction contributes to impaired repair and wound healing. Other contributors to impaired wound healing in chronic hyperglycemia include impaired angiogenesis, enhanced expression of MMPs, impaired migration and proliferation of cells, oxidative stress that is elevated, chronic inflammation, and an innate immunity that is defective (Kocher et al., 2018).

A study by Daubert et al reported a significant association between peri-implantitis and diabetes mellitus, reporting a 95% prevalence in the study population with diabetes mellitus. An association between diabetes and PI was demonstrated (Daubert et al., 2015). A study by Ferreira demonstrated that poor glycemic control increased the risk of PIM and PI. The individuals with diabetes mellitus made 13.7% of the study population, individuals with diabetes in the PIM and PI were 58.62% and 24.13% respectively. The HbA1C was not indicated in both studies (Ferreira et al., 2006). A study performed by Renvert failed to demonstrate an association between diabetes mellitus and PIM and PI. Individuals with diabetes made up 4.07% of the study population, individuals with diabetes in the PIM and PI group were 3.7% and 0.37%. There is weak evidence to support the association of diabetes mellitus and peri-implant mucositis (Renvert & Polyzois, 2015; Renvert & Quirynen, 2015).

5.11 Smoking

In the current study, smokers at patient level in the PIM and PI group were 12.5% and 0% respectively. The smokers were smoking >10 cigarettes daily and were non-compliant to

SPT. The study did not find an association between smoking and PI, this can possibly be attributed to the proportion of smokers in the study population (9.5%) compared to non-smokers (88.1%) and past smokers (2.4%). Studies that demonstrate a positive correlation between smoking and PI or PIM have a more significant ratio of smokers and non-smokers. Smokers in the study performed in the same institution by Stander made up 22% of the study population. Smokers in the PIM group were 12%. The association between PIM and smoking was not statistically significant (Stander et al., 2019).

A study by Karbach et al had 28% smokers in the study population, smokers in the PIM group were 48%. This demonstrated a significant correlation between smoking and PIM (Karbach et al., 2009). Rinke et al reported a prevalence rate of 44.9% for PIM. Non-smokers in the peri-implant mucositis group made up 38.9% while the prevalence rate in smokers were 70.6%. The prevalence rate of smokers with a history of periodontal disease that presented with PIM was 80%, while smokers participating in SPT at irregular intervals that presented with PIM had a prevalence rate of 85.7%. It was determined that that smoking as an independent variable was a risk factor for PIM. It was concluded that smokers have a 3.7-fold higher probability to develop PIM compared to non-smokers. (Rinke et al., 2011) Compliance to SPT is considered to be an important risk factor (Rinke et al., 2011). Smoking is a risk indicator for PIM and PI (Heitz-Mayfield & Salvi, 2018; Karbach et al., 2009). The biological effects of smoking are dose-dependent and include vasoconstriction both in peripheral blood vessels as well as locally within the soft tissue, i.e. gingiva and peri-implant mucosa. The vascular dynamics as well as the cellular metabolism are altered by vasoactive components of smoking. Angiogenesis is also suppressed. Some of the aforementioned alterations can be see clinically by the reduction in gingival bleeding. Tissue homeostasis as well as the host defense and response are also affected by smoking, the latter being suppressed thus increasing the infection risk in smokers. Peripheral white blood cells are elevated and their function is altered, neutrophil migration and chemotaxis is impaired and the circulating immunoglobulins are decreased and the response of the antibodies to certain antigens is depressed (Apatzidou, 2022; Darby, 2022; Palmer et al., 2005).

5.12 Supportive periodontal therapy

Subjects that participated in SPT 6 monthly and <5 monthly intervals in the peri-implant health group were 28.5% and 3.57% respectively. Subjects from this group who participated casually in SPT were 7.14%, and those who were non-compliant were 60.7%. Subjects in the

PIM group who participated in SPT 6 monthly and those who were non-compliant were 12.5% and 87.5% respectively. Subjects in the PI group who participated in SPT casually and those who were non-compliant were 83.3% and 16.6% respectively. The findings in the PI and PIM group are in keeping with the recommendations for regular SPT with short intervals. Frisch et al reported rates of PI in subjects that was compliant to SPT and non-compliant group to be 4.08% and 17.25% respectively. PIM was observed in 67.77% of subjects in the non-compliant group and 30.61% in the compliant group. SPT in the aforementioned study was performed at least annual. The results confirmed that lack of SPT placed subjects at a higher risk of developing peri-implant disease (Frisch et al., 2020).

SPT refers to maintenance therapy of peri-implant tissues. Other terms used in literature include supportive therapy, supportive implant therapy, implant maintenance, cumulative interceptive supportive therapy, and supportive peri-implant therapy (De Ry et al., 2021; Frisch et al., 2020; Lang et al., 2000; Monje et al., 2016). The study used the definition of recall intervals as described by Heitz-Mayfield et al, patients who adhered to the 3 to 4 months recall interval were defined as compliant, those who adhered to a recall interval >6 months were defined as casual attenders, and those who did not attend supportive periodontal therapy were defined as non-compliant. The other intervals that is under the SPT vector in the IDRA tool are ≤ 5 months and 6 monthly recall intervals (Heitz-Mayfield et al., 2020). Recall intervals that are shorter have demonstrated reduction in plaque accumulation and bleeding on probing in dentate patients. Although an optimal interval will not be suitable for all cases, a minimal interval that is suggested is 5-6 months (Lin et al., 2019).

A systematic review by Atieh et al demonstrated the significance of supportive periodontal therapy in especially the periodontally compromised patients, a reduction in the frequency of peri-implantitis in these group of patients who adhered to regular supportive periodontal therapy. The risk of developing peri-implant disease increased by 11-fold for those who did not adhere to SPT (Atieh et al., 2012). The risk of developing peri-implantitis is reduced by regular SPT at patient level from 43.9% to 18%. Peri-implant disease can be prevented by following a recall of 4 monthly maintenance intervals (Monje et al., 2016).

SPT is performed to aid monitoring, detecting early, arresting peri-implant tissue inflammation, and maintaining peri-implant tissue health. It also aids identification of possible contributing factors and subsequently addressing those factors. A cause and effect have been demonstrated in literature between bacterial plaque and the development of peri-implant disease. SPT includes review of the medical history, clinical and radiographic

evaluation, reinforcing oral hygiene instructions, and removal of bacterial plaque making use of different devices (De Ry et al., 2021; Lin et al., 2019; Monje et al., 2016).

5.13 Distance from restorative margin of the implant-supported prosthesis to the bone

Restorative margin to bone crest of <1.5mm observed in the peri-implant mucositis and peri-implantitis group was 25.0% and 66.7% respectively. Derks et al observed a higher odds ratio (2.3) for distance of restorative margin to bone crest of ≤ 1.5mm in a peri-implantitis group. A distance of ≤1.5mm for restorative margin to the bone crest places a patient at high risk for development of peri-implantitis. Soft tissue level implants carry a low risk (Derks et al., 2016; Heitz-Mayfield et al., 2020). **The micro-gap at the implant-abutment interface is known to be colonized by bacteria and endotoxins. Shifting the micro-gap and inflammation away from the bone crest is recommended. The micro-gap in tissue level implants is moved coronal by more than 1.5mm to the bone crest** (Ganeles et al., 2021; Sasada & Cochran, 2017).

5.14 Limitations

The limitation of the current study includes the sample size, utilization of a larger sample size provides more strong conclusions as well as greater statistical power for analysis. The implants evaluated in this study were not placed in the same year therefor not all have been in function for more than 5 years. This study lacked a significant ratio of smokers and non-smokers, patients with a history of periodontitis and those with no history of periodontitis, and patients who are diabetic and those who are not diabetic. A correlation between the risk factors and peri-implant disease could not be drawn or deliberated upon.

5.15 Conclusion

The prevalence of peri-implant disease at subject level in this university-based population was 19.0% for PIM and 14.3% for PI, while at implant level it was 11.7% and 8.8% respectively. Only 2.3% of the subjects and 33.3% implants diagnosed with PI presented with hyperglycemia while periodontally unstable and lacking adequate SPT. There was a positive correlation with function time, history of periodontitis, casual SPT visits, non-compliance to SPT, a distance of <1.5mm from restorative margin to bone crest in the PI group in this study. No associations were found in this study between PI smoking status, and diabetes. This

is attributed to the low number of smokers and diabetic patients in the study. Prospective cohort studies are recommended to identify and demonstrate the risk factors and to prove the efficacy of prevention when these factors are modified (Romandini et al., 2020).

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Appendices

Appendix A: Ethics Clearance Certificate

UNIVERSITY of the WESTERN CAPE

60 YEARS of hope, action & knowledge

8 October 2021

Dr S Mahlangu
Oral Medicine and Periodontology
Faculty of Dentistry

Ethics Reference Number: BM21/8/10

Project Title: Peri-implant health and disease: Cross-sectional study

Approval Period: **7 October 2021 – 7 October 2024**

I hereby certify that the Biomedical Science Research Ethics Committee of the University of the Western Cape approved the scientific methodology and ethics of the above mentioned research project and the requested amendment to the project.

Any further amendments, extension or other modifications to the protocol must be submitted to the Ethics Committee for approval.

Please remember to submit a progress report annually by 30 November for the duration of the project.

For permission to conduct research using student and/or staff data or to distribute research surveys/questionnaires please apply via:
https://sites.google.com/uwc.ac.za/permissionresearch/home

The permission letter must then be submitted to BMREC for record keeping purposes.

The Committee must be informed of any serious adverse event and/or termination of the study.

Ms Patricia Josias
Research Ethics Committee Officer
University of the Western Cape

NHREC Registration Number: BMREC-130416-050

FROM HOPE TO ACTION THROUGH KNOWLEDGE.

Appendix B: Information Letter and Consent form

FACULTY OF DENTISTRY

Prevalence of Peri-implant disease and conditions

Project Registration Number: BM21/8/10

Patient Information Letter

Place patient's file sticker

I, Dr S. Mahlangu, plan a cross-sectional study to evaluate the implants that have been placed in your oral cavity. The evaluation of your implants will provide information regarding the prevalence of peri-implant disease and conditions of implants placed and restored at our clinic, Faculty of Dentistry, Department Oral Medicine and Periodontics. Participation in the study is on a voluntary basis and all information will be kept strictly confidential. The regular maintenance and monitoring of your implant restorations is extremely important to allow early detection and management of disease and the prevention of implant loss.Your participation will assist us in formulating a prognostic classification system for implant restored patients and to initiate an oral maintenance program for these patients.

You will be expected to present yourself for an evaluation appointment at the Department of Oral Medicine and Periodontics. There will be no cost for this evaluation visit (as per the

Manager Dental services: Dr E Prince). Patients requiring assistance with transport costs will be booked with the Healthnet System.

A full examination of the implant(s), soft tissue and bone around the implant(s) will be performed. A clinical and radiographic examination will be performed on your oral cavity at the evaluation visit.

The clinical examination includes the following:

- Measuring the depth between the implant and soft tissue (gum), and the depth between the tooth and soft tissue (gum) where applicable (called pocket depths).
- Measuring the amount of bleeding around teeth and implants.
- Measuring the amount of plaque present using a disclosing agent (wash off dye) that colours the old plaque differently to the new plaque.
- Measuring the soft tissue width above the implant restoration.
- Assessing if there is any pus draining around the implants.
- Assessing if there is any part of the implant that is visible clinically.
- Assessing if there is any mobility of the implant.
- A test will be performed (HbA1C test) to assess your glucose (blood sugar levels).

The radiographic examination will include the following:

- A radiograph (xray) of the implant(s) site that will assist in the assessment of bone loss around the implant.
- A radiograph (xray) of the tooth that has pocket depths above 3mm, which will assist in the assessment of bone loss around the tooth.

The examination will be followed by dental cleaning of your teeth and implants, and appropriate referral for further management.

In the scenario where peri-implant disease is detected at your implants - we will ensure adequate referral and management of any disease, depending on your clinical scenario. These additional visits will include costs that will be discussed with you.

Thank you.

Dr S. Mahlangu

Department of Oral Medicine and Periodontology

Faculty of Dentistry, University of the Western Cape

If you have any questions, please feel free to contact my supervisor, Dr S Mulder van Staden at smuldervanstaden@uwc.ac.za or Research Ethics Committee at BMREC, UWC, Private Bag X17, Bellville, 7535, Tel: +27 21 959 411, Email: researchethics@uwc.ac.za

UNIVERSITY *of the*
WESTERN CAPE

FACULTY OF DENTISTRY

Prevalence of Peri-implant disease and conditions

Project Registration Number: BM21/8/10

Patient Consent Form

Place patient's file sticker

I,.....(Name & Surname) am willing to participate in the above mentioned study and understand that the study is voluntary. This study is approved by the Ethical and Research Committee of the University of the Western Cape. Participation in this study is on voluntary basis. I have been adequately informed about the study. I also know that I have the right to withdraw from the study at any given stage which will not discriminate me in future treatments. My rights will be protected, and all my details will be kept confidential, and no details regarding my identity will be published.

Patient Name & Surname: ……
Signature: …… Date: …….

Witness Name & Surname:
……..

Signature: …… Date: …….

Researcher Name & Surname: Dr Sibongile Mahlangu

Signature: …… Date: …….

Department of Oral Medicine and Periodontology

Faculty of Dentistry, University of the Western Cape

If you have any questions, please feel free to contact my supervisor, Dr S Mulder van Staden at smuldervanstaden@uwc.ac.za or BMREC, UWC, Private Bag X17, Bellville, 7535, Tel: +27 21 959 4111, Email: research-ethics@uwc.ac.za

Appendix C: Consent to Clinical Photography

UNIVERSITY of the
WESTERN CAPE

FACULTY OF DENTISTRY

Prevalence of Peri-implant disease and conditions

Project Registration Number: BM21/8/10

Patient Consent to Clinical Photography

Place patient's sticker

I,.....(Name & Surname) consent to photographs being taken of me as requested. I understand that these photographs will treated with the utmost confidentiality, and they will be stored appropriately. I give permission for the images to be used for:

- Record purposes

The photographic images will form part of the information collected my care and treatment. This information will be handled in accordance with the HPCSA Guidelines on the keeping of patient records.

- Education and training purposes

The photographic images may be used for teaching purposes and viewed by health professionals outside of the UWC Faculty of Dentistry. They may be used in talks, conference presentations, posters or on the internet to help train other health professionals in the management of dental and oral diseases.

- Approved research purposes & publication

The photographic images may be used in medical or dental publications, journals, textbooks, conference material, e-publications and on the internet in professional journals and education. The images will not be used with identifying information such as name, however, full confidentiality is not guaranteed.

I understand that all efforts will be made to conceal my identity but that full confidentiality cannot be guaranteed.

I understand that my consent or refusal will in no way affect my dental care.

Patient Name & Surname: ……

Patient Signature: …….. Date: ……

Witness Name & Surname: ……..

Witness Signature: …… Date:……..

Requesting Clinician Name & Surname:

…… Date: …….

Department: ……. Phone: (021) 937 3167

Views required:

- Intra-oral frontal
- Intra-oral right lateral
- Intra-oral left lateral
- Intra-oral maxillary occlusal
- Intra-oral mandible occlusal

- Intra-oral implant site buccal and occlusal

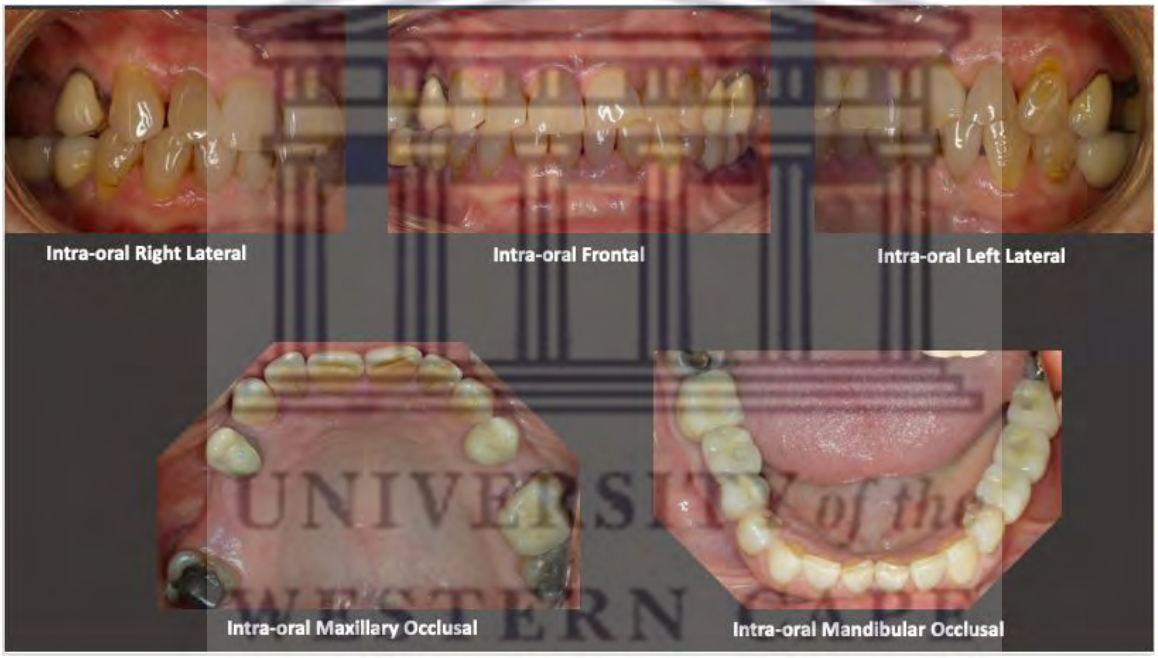
Required for:

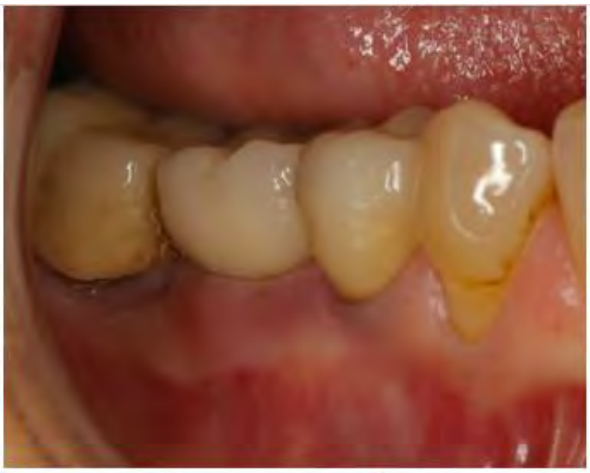
- Records
- Teaching and lectures
- Research
- Publication

Images taken by: …… Date: ……..

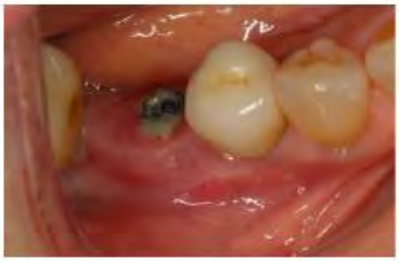
Location where copies will be stored: Department of Oral Medicine and Periodontology Google Drive account (accessible to the researcher, postgraduate students and consultants)

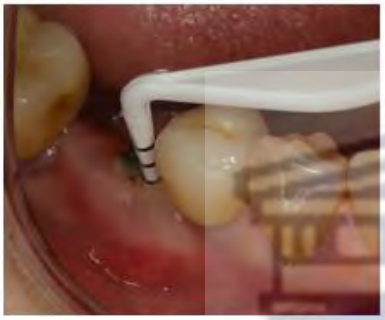
The example of the images to be taken:

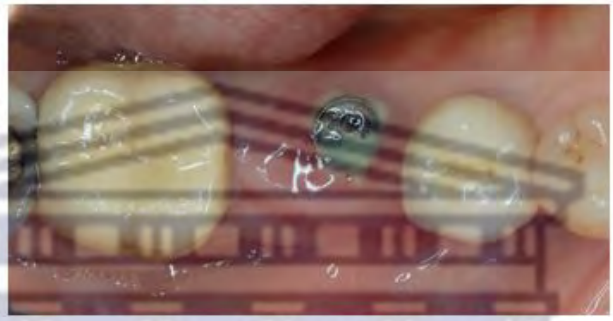




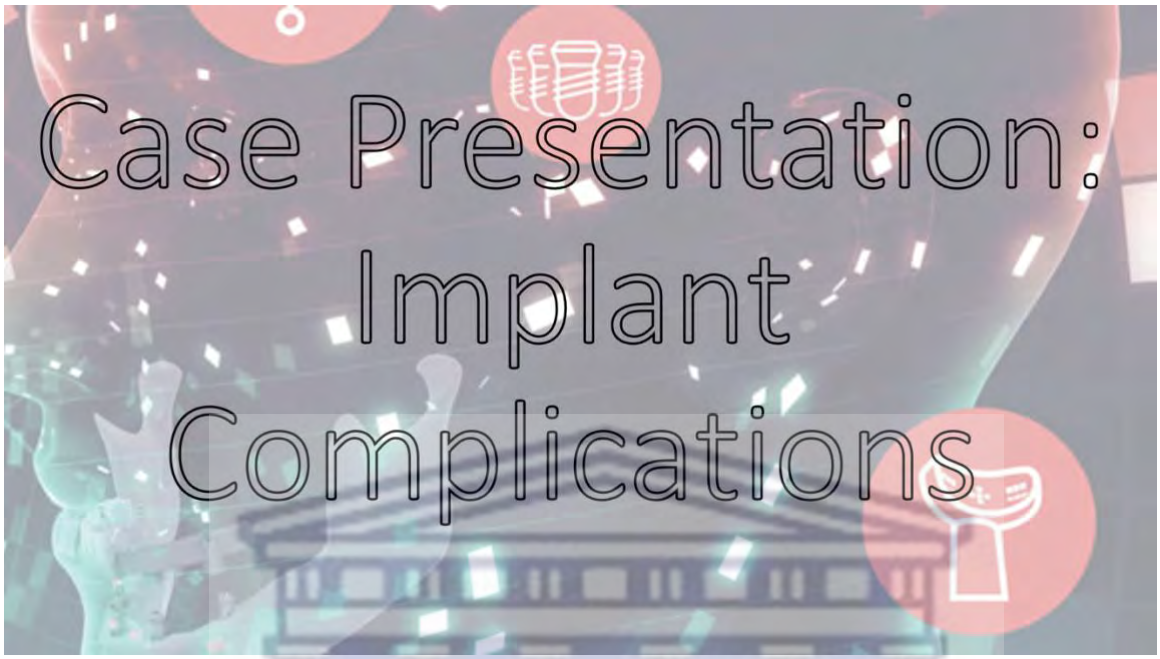
Example of Implant site buccal and occlusal view.



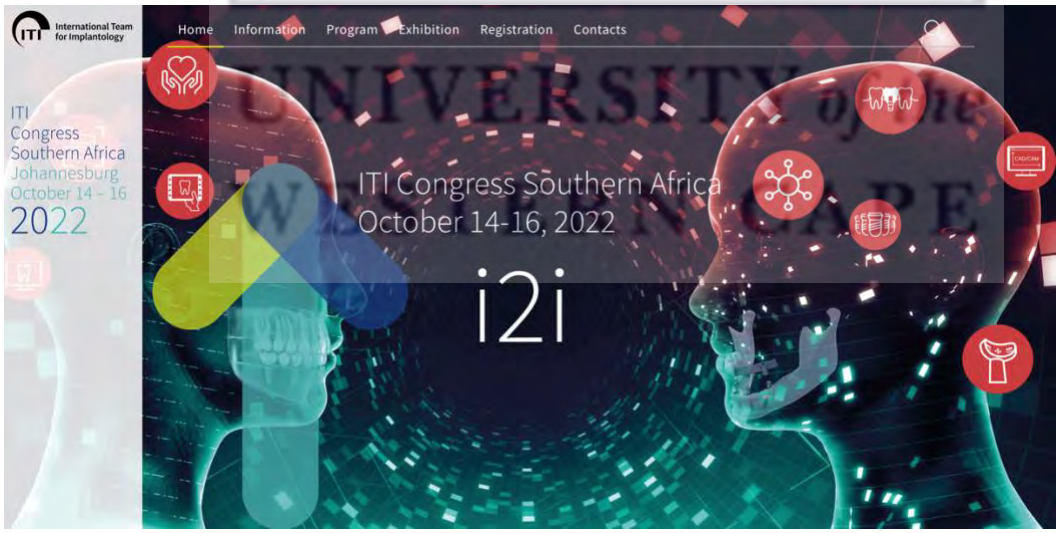




Appendix D: Presentation at the ITI Southern Africa Conference 2023



Two cases from the study population were presented at the ITI Southern Africa Conference 2023.



Appendix E: Data Collection Sheet

Sheet 1: Details from the Patient's Records:

Study code: patient	Study code per implant	Year of Placement	Patient Age at placement	Gender	Site	Use of antibiotics at placement

Cont.

Bone graft before implant placement	Bone graft at implant placement	Soft tissue graft before implant placement

Use	Type	Use	Type	Use	Type

Cont.

Soft tissue graft at implant placement		Soft tissue graft after implant placement		Implant system	Bone level vs Soft tissue level implant	Implant diameter	Implant length	Type of prosthesis
Use	Type	Use	Type					

Sheet 2: Clinical Examination

Current age	Site	Peri implant mucosa: Redness	Peri implant mucosa: Edema	Peri implant mucosa: Keratinized Tissue Width	Pocket depth: chart	Bleeding on probing %	Pus Present/Absent	Implant mobility Present/Absent

Implant thread exposure Present/Absent	Type of bone defect	Mobility of mucosal margin Present/Absent	Presence of frenal pull Present/Absent	Peri-implant mucosa phenotype	Radiographic bone loss in relation to implant:

					<25 % or 25-50% or >50%

Sheet 3: Patient Related Factors

Study code: patient	Study code: implant	History of periodontitis/peri-implantitis	Diabetes	Smoking	Poor oral hygiene: Plaque index	Supportive Therapy: Frequency

Sheet 4: Implant related factors

Study code: patient	Study code: implant	Implant surface roughness as per manufacturer	Crown: cemented	Crown: screw retained	Crown: difficult to access (home care or plaque control)	Distance from the restorative margin to the bone ≤ 1.5 mm

Oral hygiene practice & Frequency of Maintenance

Study code: patient	Study code: implant	Brushing frequency	Flossing		Mouthwash	Supportive Therapy/ Maintenance		Implant disease risk assessment	Oral hygiene instructions delivered after implant placement
			Type of floss			attendance	Planned Intervals		