

The Crucial Role of Plaque Control in Peri-Implant Mucositis Initiation as Opposed to the Role of Systemic Health Condition: A Cross-Sectional Study

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Purpose: The aim of this study was to investigate the priority of periodontal plaque as a risk factor compared to other risk factors, namely hypertension and diabetes mellitus type II, regarding the initiation and severity of peri-implant mucositis, eventually reinforcing the importance of plaque control, periodic maintenance and supportive periodontic treatment after implant placement in order to prevent peri-implant diseases.

Patients and Methods: A total of 58 patients (84 implants) were enrolled; each individual implant was considered as a separate sample first, then sampling by patient was also applied, implants were divided into group A: systemically healthy patients and B: patients with hypertension and diabetes mellitus type II, the status of peri-implant tissue was followed after the healing abutment placement, with regard to implant mucosal index (IMI), probing pocket depth (PPD) and bleeding on probing (BOP); when sampling was done by patient, the mean of scores of all examined implants in each patient was taken to represent one sample.

Results: Group A implants showed higher mean scores of PPD (5.2 mm) than group B (4.2 mm) with significance ($P = 0.014$), and higher mean scores of BOP, group A = 0.71, group B = 0.45 with ($P = 0.015$); there was no statistical difference with regard to IMI, group A = 1.35, group B = 1.16 with ($P = 0.172$). Similar results were obtained when the sampling was calculated by patient; PPD: group A (5.31 mm), group B (4.75 mm) and $P = 0.008$, IMI: group A (1.34), group B (1.16) and $P = 0.131$, BOP: group A (0.75), group B (0.48) and $P = 0.03$.

Conclusion: In the absence of proper plaque control, systemic diseases showed no impact on the initiation and severity of peri-implant mucositis when compared to systemically healthy patients.

Keywords: mucositis, dental implant, dental plaque, risk factor

Introduction

Dental implants are globally well accepted as the best choice for replacement of missing teeth,¹ because they do not involve any other teeth preparation, have better psychological acceptance by patients,² better load distribution³ and a better preservation of periodontal tissue (alveolar bone and gingiva).⁴

The developments in the dental implantology field concerning a fixture's design, material composition, accessories, etc.,⁵⁻⁷ raised the challenge from functional survival of the implant to aesthetic survival of the implant, many dental implants can survive with reduced bone level functionally, but aesthetically that is not sufficient.⁸

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The aesthetic aspect of implants cannot be met without maintaining healthy soft tissue supported by a satisfactory height of alveolar bone level,⁹ preventing the exposure of unwanted titanium fixture or its metallic shadow in the cervical region especially in the anterior region.¹⁰

That being said, it does not underestimate the danger of serious deterioration and eventual loss of the implant and the surrounding bone due to true peri-implantitis, despite the advancement in implantology mentioned above, all these fearsome scenarios start with a reversible disease, namely the peri-implant mucositis,¹¹ that develops mainly due to lack of proper plaque control in addition to other systemic risk factors; mostly diabetes mellitus and behavioral risk factors such as smoking.^{12,13}

The mirroring similarity between periodontium and peri-implant tissue extends to the diseases too, the 2017 world workshop set some clear conditions for peri-implant mucositis by the presence of cardinal inflammatory signs like: bleeding on probing (BOP), increased probing level compared to previous readings (PPD) but with the absence of radiological bone loss (RBL),¹⁴ although in 55% of peri-implant diseased sites (*Staphylococcus spp.*, *enterics* and *Candida spp.*) were found exclusively and 19 different species, among which *Porphyromonas Gingivalis*, *Prevotella Intermedia* and *Tannerella forsythus* appear at higher rates, still the overall microbiological picture is highly similar.¹⁵ This disease affects nearly 50% of implants and if left untreated, develops into the more serious condition ie, peri-implantitis.¹⁶

It is beyond debate now that, with the exception of congenital and auto-immune problems, the plaque pathogens like *P. Gingivalis*, *Aggregatibacter Actinomycetemcomitans*, etc. are the key factor behind periodontal problems,^{17,18} this is true for peri-implant diseases too¹⁹ starting from simple plaque related gingivitis until more serious conditions such as loss of attachment related periodontitis in its most known form (previously named chronic and acute periodontitis) recently changed into only periodontitis.²⁰

It is believed too that systemic overall condition also plays a role in the prognosis of dental implant survival^{15,21} and that some dentists refuse to operate on a patient with uncontrolled diabetic patients (which is the right thing to do). The contraindication is related to delayed wound healing due to impairment of microvasculature and activation of inflammatory pathways that may affect a cell's apoptosis.^{22–24}

The American College of Cardiology and American Heart Association set new guidelines for hypertension in 2017, lowering the reading that is considered hypertensive to 130/80 mmHg less than the previous guideline (140/90 mmHg).²⁵

Singh et al found that 20.8% of 832 implants failed due to hypertension, several studies and meta-analysis showed a positive relationship between hypertension and periodontal problems.²⁶ Fernandes et al found *Prevotella intermedia*, a known periodontal pathogenic bacteria, in 92.8% and traces of other periodontal pathogens in atherosclerotic plaque samples taken from patients with vascular disease.²⁷

Despite the interlinking between several risk factors and behavior-related effects, plaque control measures are still the most decisive factor for periodontal health.^{28,29}

The condition in question (peri-implant mucositis) actually can be stopped and reversed back to normal if correct maintenance is performed, the removal of plaque will stop further deterioration, in three weeks, tissue can be restored to its normal condition, thus plaque control should be the main factor to focus on, as Salvi et al found.^{30,31} In order to do that, we need good compliance by patients; yet, several limitations arise, since patients cannot notice periodontal problems (as well as peri-implant problems) as it is mostly painless, other limitations might be dentist-related, as it is sometimes not easy to tell the difference between peri-implant mucositis and peri-implantitis on clinical bases.³² This can also be seen in the wide range of reported peri-implant mucositis prevalence in literature, ranging from 8–46% and peri-implantitis up to 25%.³³

Patients who are committed to regular supportive recall visits, professional and home plaque control measures lost (0.7) tooth per subject over 30 years, Matarese et al found.³³

This article investigates the role of plaque accumulation in peri-implant diseases initiation and progression as a risk factor as opposed to systemic diseases, aiming to raise the attention to the supportive periodontal therapy (SPT) that includes total examination of peri-implant tissue, plaque control and careful periodic observation following the prosthetic part placement.

The null hypothesis states that there is no difference between patients with systemic diseases and systemically healthy patients regarding the implant mucosal index, probing pocket depth, and bleeding on probing.

Patients and Methods

Principles of the Declaration of Helsinki were considered and met, all patients were informed about the idea of the research and signed a letter of consent, the research was also approved by the ethical committee of the College of Dentistry/University of URUK (no. 23 in 2020).

Study Design

This is a cross-sectional non-interventional study, the comparison took place between two groups.

Group A: patients that are systemically healthy.

Group B: patients suffering from hypertension and diabetes mellitus type II.

Both of them failed to follow proper plaque control measures, and had a mean plaque index ≥ 1 , they all received equal treatment protocols, oral hygiene instructions and motivation.

Patients' History Taking and Selection

Before final filtering, 139 patients (male and female) were interviewed aged between 18–50-years-old, before the start of the treatment, medical, dental and familial history was recorded in case sheets, then patients were followed through all stages of the implant procedure ranging from 3–8 months. Fractions of patients were excluded due to exclusion criteria at the interview (1), others dropped out due to certain surgical considerations (17), and some dropped out during the research for personal reasons (9).

Exclusion criteria were:

1. Pregnant women.
2. Mentally and psychologically challenged patients.
3. Women of post-menopausal period.
4. Cancer patients, patients undergoing chemo/radiotherapy.
5. Patients with synthetic bone/barrier implant procedure.
6. Smokers.

For this article, final filtration kept only fifty-eight patients (eighty-four implants) who failed to follow proper plaque control, and whose plaque index is more than 1 after three weeks of healing abutment placement, see [Figure 1](#).

Patient Systemic Diseases Diagnosis

All patients were sent to a physician to obtain a signed diagnosis paper with their blood pressure measurement, HbA1c percentage and fasting blood sugar level, once after the interview and again on the operation day.

Readings higher than 130/80 mmHg were considered hypertensive.²⁵

Readings higher than 6.5% HbA1c were considered diabetic.²²

Surgical Procedure

Samples included were subjected to conventional dental implantology protocol, Dentium implants, no sinus lift, no synthetic bone and/or membrane, Delayed exposure and delayed loading protocol, exposure was scheduled as:

1. Maxillary implants: 6–8 months.³⁴
2. Mandibular implants: 3–4 months.³⁴

Before exposure, osteointegration was examined using periapical x-ray, and that delayed the exposure by 1–2 months in some cases of mandibular arch.

Patients did not receive any antibiotic treatment during exposure procedure and healing abutment placement until the examination time.

Selection of Implants

When patients who failed to follow proper home plaque control measures, returned to complete the implant therapy (prosthetic part), three weeks after healing abutment insertion, which is enough to develop signs of peri-implant mucositis around the healing abutment,³⁰ the implants were carefully examined using peri-apical x-rays, OPG's and clinically with a periodontal probe (CPITN Probe) and mirror, the selected implants were fully integrated within the jaw bone, showing no signs of radiolucency, mobility, and the healing abutment was a mounted fit without any looseness ([Table 1](#)).

Samples Grouping

Samples were divided into two groups ([Table 1](#)) ([Figure 1](#)).

Group A: healthy patients.

Group B: patients with systemic diseases (hypertension and diabetes mellitus type II).

Sampling by Implant

Each individual implant was considered as an individual sample, divided between implants belonging to systemically healthy patients and implants belonging to patients with hypertension and diabetes mellitus Type II ([Tables 1](#) and [2](#)).

Sampling by Patient

The mean of scores of implants within the same patient was taken to represent one sample (as per patient), here each patient represented an individual sample ([Tables 1](#) and [3](#)).

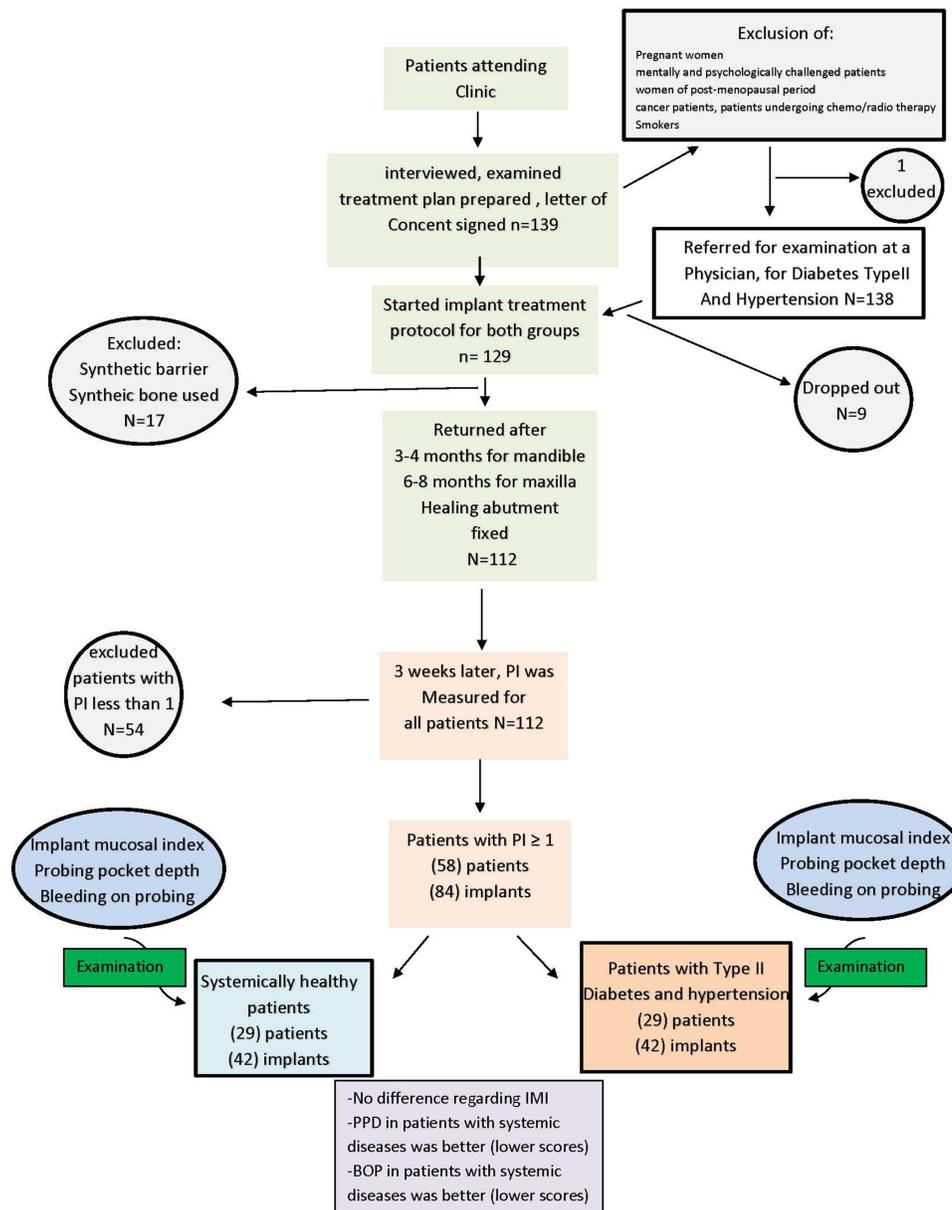


Figure 1 STORBE chart for patient flow and patient's selection.

- Measurement of plaque, implant mucosal index, probing pocket depth.

Plaque Index (PI)

After the healing abutment was attached to the implant, three weeks period of waiting elapsed before each peri-implant tissue was measured using a periodontal probe (Figure 2) PD-CPITN, model: PI-1304 New York, USA. Plaque index of Løe 1967 was used for evaluation of plaque accumulation and thus the overall oral hygiene status of the patient, its scores are as follows:

Score 0: absence of plaque by both vision and gentle probing.³⁵

Score 1: absence of plaque by vision but presence on probing.³⁵

Score 2: visible plaque accumulation.³⁵

Score 3: abundant amount of plaque exceeding the cervical third of the healing abutment.³⁵

Each one of the 4 surfaces (mesial, facial, lingua/palatal, distal) of healing abutment was measured by moving the probe gently on the surface in a sweeping motion, after dryness of the surrounding field, a mean of them was considered to represent the reading of the individual implant.

Table 1 Descriptive Numbers of Patients and Implants Characteristics That Participated in the Research

	Numbers			Percentages		
	Group A	Group B	Total	Group A	Group B	Total
Male	15	17	32	25.862%	29.310%	55.172%
Female	14	12	26	24.138%	20.690%	44.828%
Sum. of patients	29	29	58	50%	50%	100%
PD 3.6, L 10mm	7	4	11	8.333%	4.762%	13.095%
PD 3.6, L 12mm	5	5	10	5.952%	5.952%	11.905%
PD 3.6, L 14mm	3	3	6	3.571%	3.571%	7.143%
PD 4, L 12mm	6	10	16	7.143%	11.905%	19.048%
PD 4, L 10mm	8	11	19	9.524%	13.095%	22.619%
PD 4.5 10mm	6	5	11	7.143%	5.952%	13.095%
PD 4.5 12mm	2	1	3	2.381%	1.190%	3.571%
PD 5, L 8mm	5	3	8	5.952%	3.571%	9.524%
Sum of implants	42	42	84	50%	50%	100%

Notes: Group A: systemically healthy patients, group B: patients with hypertension and type II diabetes mellitus.

Abbreviations: PD, platform diameter size; L, length.

Table 2 Descriptive Statistics of Plaque Index, Implant Mucosal Index, Probing Pocket Depth and Bleeding on Probing for Two Groups When Each Implant Was Considered a Sample

	Plaque Index		Implant Mucosal Index		Probing Pocket Depth		Bleeding on Probing	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Number of samples	42	42	42	42	42	42	42	42
Mean	1.2619	1.2381	1.3571	1.1667	5.2381	4.8333	0.7143	0.4524
Mean rank	43.00	42.00	45.61	39.39	48.64	36.36	–	–
Sum of ranks	1806.00	1764.00	1915.50	1654.50	36.36	1527.00	–	–
Median	1.0000	1.0000	1.0000	1.0000	5.0000	5.0000	1.0000	0.0000
Variance	0.198	0.186	0.235	0.386	0.576	0.728	0.209	0.254
Std. Deviation	0.44500	0.43108	0.48497	0.62143	0.75900	0.85302	0.45723	0.50376
Minimum	1.00	1.00	1.00	0	4.00	4.00	0.00	0.00
Maximum	2.00	2.00	2.00	2.00	7.00	7.00	1.00	1.00
Range	1.00	1.00	1.00	2.00	3.00	3.00	1.00	1.00
Standard Error of Mean	0.06867	0.06652	0.07483	0.09589	0.11712	0.13162	0.07055	0.07773

Notes: Group A: systemically healthy patient, group B: patients with hypertension and type II diabetes mellitus.

Implant Mucosal Index (IMI)

After good dryness of field, the same type of periodontal probe was inserted gently in sulcus, no more than 1 mm, then moved in an encircling movement with a little pressure directed towards the soft tissue (Figure 2), after a few seconds, scores were observed as:

Score 0: no bleeding.³⁶

Score 1: single point bleeding.³⁶

Score 2: moderate multi points bleeding.³⁶

Score 3: profuse bleeding in multiple points on probing.³⁶

Score 4: suppuration.³⁶

All the four surfaces (lingual, distal, mesial and labial) of the healing abutment were measured and a mean of them was considered to represent the reading of the individual implant.

Bleeding on Probing (BOP)

With the periodontal probe, we encircle the sulcus around the implant gently with minimum pressure and then observe. Scoring the following:

Score 0: no bleeding.³⁷

Score 1: presence of bleeding in one or more surface(s).³⁷

Table 3 Descriptive Statistics of Plaque Index, Implant Mucosal Index, Probing Pocket Depth and Bleeding on Probing for the Two Groups When Patients are Considered as Samples

	Plaque Index		Implant Mucosal Index		Probing Pocket Depth		Bleeding on Probing	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Number of samples	29	29	29	29	29	29	29	29
Mean	1.2586	1.2184	1.3448	1.1667	5.3103	4.7529	0.7586	0.4828
Mean rank	29.86	29.14	32.34	26.66	35.17	23.83	–	–
Sum of ranks	866.00	845.00	938.00	773.00	1020.00	691.00	–	–
Median	1.0000	1.0000	1.0000	1.0000	5.0000	5.0000	1.0000	0.0000
Variance	0.172	0.133	0.216	0.250	0.561	0.614	0.190	0.259
Std. Deviation	0.41449	0.36489	0.46490	0.50000	0.74897	0.78379	0.43549	0.50855
Minimum	1.00	1.00	1.00	0.00	4.00	4.00	0.00	0.00
Maximum	2.00	2.00	2.00	2.00	7.00	6.50	1.00	1.00
Range	1.00	1.00	1.00	2.00	3.00	2.50	1.00	1.00
Standard Error of Mean	0.07697	0.06776	0.08633	0.09285	0.13908	0.14555	0.08087	0.09443

Notes: Group A: systemically healthy patients, group B: patients with hypertension and type II diabetes mellitus.

If a single surface showed bleeding, the sample was considered as (1) since it's a 1/0 measurement.

Probing Pocket Depth (PPD)

Using the same probe, insertion with minimal pressure into the interface between healing abutment and gingiva then the depth of pocket was recorded till the tissue stops the probe without further pressure, the deepest side was chosen to represent the implant³⁸ (Figure 3).

Calibration/Inter-Observer Reliability

Examination was run by the authors, one examiner was the lead, the other one was for intercalibration purpose, for plaque index the procedure was slightly complicated, since the plaque removed by the first examiner cannot be observed by the following examiner, thus we invited a third examiner (a periodontist) to observe the procedure while the lead examiner and co-examiner do the work.



Figure 2 Probing around the healing abutment to measure IMI and PI using a CPITN periodontal probe.



Figure 3 Measuring the pocket depth around healing abutment.

The scores of both examiners were tested using Cohen's Kappa test and the results were:

For all parameters (plaque index, implant mucosal index (IMI), bleeding on probing and probing pocket depth) the significance of Cohen's Kappa test was ($P = 0.000$).

PI kappa's value = 0.692 (good inter-rater reliability).

IMI Kappa's value = 0.929 (excellent inter-rater reliability).

BOP Kappa's value = 0.828 (excellent inter-rater reliability).

PPD Kappa's value = 0.715 (good inter-rater reliability).

Statistical Analysis

Statistical analysis was performed using SPSS[®] version 26 by IBM[®]. Null hypothesis proposed that there is no statistical difference between group A and group B with regard to implant mucosal index, probing pocket depth and bleeding on probing.

Cohen's Kappa test was used to test the inter-rater reliability between the scores of the two clinicians who did the measurements.

Normality test: Kolmogorov–Smirnov normality test's result was highly significant ($P = 0.00$) and data were considered as abnormally distributed.

Mann–Whitney test was used to test the null hypothesis during the comparison between IMI and PPD of group A and group B.

Chi-square test was used to test the null hypothesis during the comparison between BOP of group A and group B.

Results

When implants were considered as individual samples, the mean of PI of group A was = 1.26 (it was = 1.25 when patients were considered as individual samples) higher than mean of PI of group B = 1.23 (1.21 when patients were considered as individual samples) (Figure 4), when tested using the Mann–Whitney test, the result was non-significant $P = 0.802$ ($P = 0.84$ when patients were considered as individual samples) and thus the null hypothesis was accepted as there is no difference in the mean of PI between the two groups.

When comparing means of IMI of the 84 implants (58 patients) (Tables 2 and 3), group A = 1.35 (when patients were considered as individual samples group A = 1.34) and B = 1.16 (when patients were considered as individual samples group B = 1.16), although means of IMI in group A was higher than that of group B (Figure 5), non-significance test result was obtained as $P = 0.172$ ($P = 0.131$ when patients were considered as individual samples), accepting the null hypothesis that there is no statistical evidence of a difference between group A and group B (Tables 4 and 5).

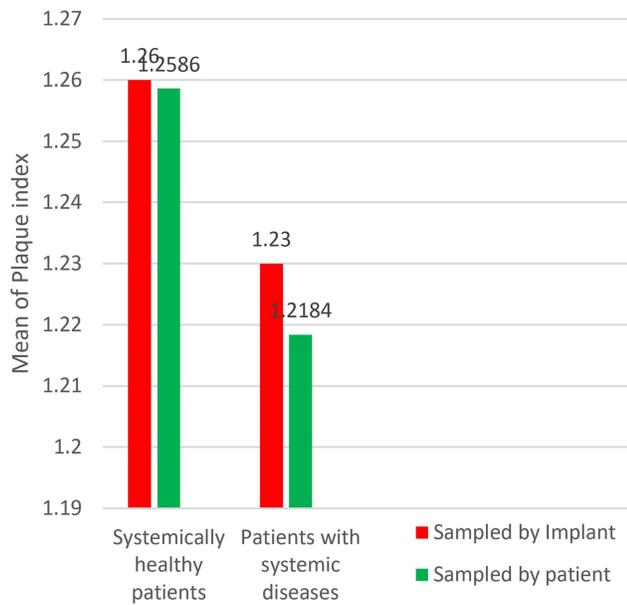


Figure 4 The PI of group A and group B.

When comparing the probing pocket depth (Tables 2 and 3), the results showed a higher mean of probing pocket depth in group A = 5.2 mm (5.31 mm when patients were considered as individual samples), as compared to group B = 4.5 mm (4.75 mm when patients were considered as individual samples) (Figure 6), upon testing the results using the Mann-Whitney test, we obtained a statistical significance $P = 0.014$ ($P = 0.008$ when patients were considered as individual samples) proving an existent difference between groups A and B (Tables 4 and 5).

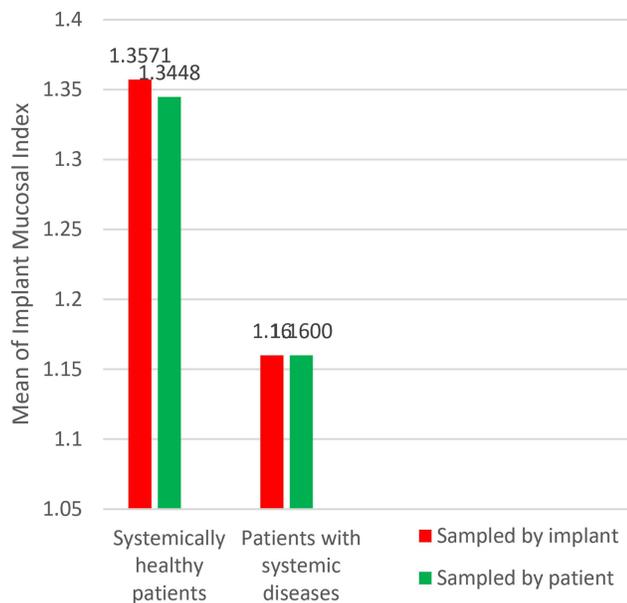


Figure 5 Means of IMI of group A and group B.

Table 4 Mann-Whitney Test for Means of Plaque Index (PI), Implant Mucosal Index (IMI) and Probing Pocket Depth (PPD) Comparison Between Group A and Group B (Each Sample Represents an Implant)

Statistical Test	PI	IMI	PPD
Mann-Whitney U	861.000	751.500	624.000
Wilcoxon W	1764.000	1654.500	1527.000
Z	-0.250	-1.367	-2.457
P value	0.802	0.172	0.014

Table 5 Mann-Whitney Test for Means of Plaque Index (PI), Implant Mucosal Index (IMI) and Probing Pocket Depth (PPD) Comparison Between Group A and Group B (Each Sample Represents a Patient)

Statistical Test	PI	IMI	PPD
Mann-Whitney U	410.000	338.000	256.000
Wilcoxon W	845.000	773.000	691.000
Z	-0.200	-1.509	-2.671
P value	0.841	0.131	0.008

Regarding the bleeding on probing (BOP) (Tables 2 and 3), descriptive statistics showed a higher tendency for bleeding on probing in healthy patients = 0.71 (0.75 when patients were considered as individual samples) than patients with hypertension and DM type II = 0.45 (0.48 when patients were considered as individual samples) (Figure 7); chi-square test showed a significant relationship of $P = 0.015$ ($P = 0.031$ when patients were considered as individual samples) (Tables 6 and 7).

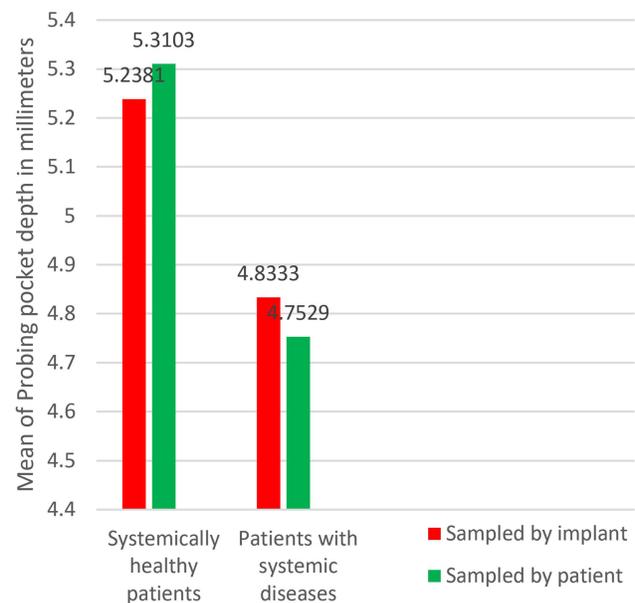


Figure 6 Means of probing pocket depth of group A and group B.

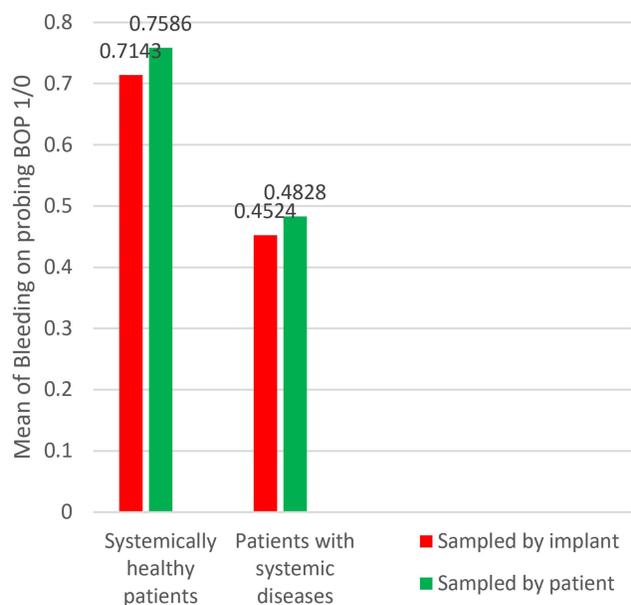


Figure 7 Means of BOP of groups A and B.

Discussion

Both groups A and B had a mean of PI more than 1 (Tables 2 and 3). Group A (systemically healthy patients) showed worse scores with regard to PPD and BOP when compared to group B (patients with systemic diseases) as seen in (Figures 6 and 7). While a difference between both groups' IMI scores could not be proven, when the implant was taken as an individual sample and when each separate patient was taken as an individual sample (Tables 4 and 5).

Most, if not all, studies agree on the fact that systemic diseases, especially diabetes mellitus and dental plaque, are causative and risk factors for the development of peri-implant diseases.^{11,27}

Results of this study partially agree with Schimmel et al who found no statistical effect of medical conditions on peri-implant tissue condition, instead he found that psychological factors played a more important role especially in geriatric patients.³⁹ In contrast, Singh et al found that 20.3% of implants in hypertensive patients failed (among 832 implants).²⁶

That can be answered by Seki et al who found a negative effect of anti-hypertensive drugs on dental implants and hypothesized that bone metabolism is affected and thus deeper pockets were observed and more bone resorption.⁴⁰ That contradicted other authors who believed that drugs used for systemic diseases namely the anti-coagulants (that could be taken by some hypertensive patients) might have some protective role against peri-implant diseases, due to their secondary anti-inflammatory effects, this is what Romandini et al believed,⁴¹ Nemati et al found out an anti-inflammatory effect for the anti-hypertensive drugs could also enhance the function of PMN's immune cells,⁴² this could contribute to a better understanding of the better scores of the patients with systemic diseases when compared to systemically healthy patients in this article.

The tendency of hypertensive patients to develop severe forms of gingivitis and even attachment loss was linked to the augmented immune response, this augmentation can be

Table 6 Chi-Square Test for Bleeding on Probing Means of the Two Groups A and B (Each Sample Represents an Implant)

	Value	df	P-value	Exact Sig. (2-Sided)	Exact Sig. (1-Sided)
Pearson Chi-Square	5.927	1	0.015	0.026	0.013
Continuity Correction	4.898	1	0.027		
Likelihood Ratio	6.007	1	0.014		
Fisher's Exact Test					
Linear-by-Linear Association	5.856	1	0.016		
N of Valid Cases	84				

Table 7 Chi-Square Test for Bleeding on Probing Means of the Two Groups A and B (Each Sample Represents a Patient)

	Value	df	P-value	Exact Sig. (2-Sided)	Exact Sig. (1-Sided)
Pearson Chi-Square	4.687	1	0.030	0.057	0.029
Continuity Correction	3.588	1	0.058		
Likelihood Ratio	4.770	1	0.029		
Fisher's Exact Test					
Linear-by-Linear Association	4.606	1	0.032		
N of Valid Cases	84				

traced back to the release of inflammatory mediators like interleukins, c-reactive proteins, tumor necrosis factor- α and metalloprotease in patients with hypertension and coronary heart disease.^{31,43} Isola et al observed an increase in the serum and salivary levels of Galectin-3 (which is an inflammatory mediator and a member of the beta-glycoside binding proteins expressed in fibroblasts) in patients with both periodontitis and coronary heart disease.⁴³

Uncontrolled diabetic patients, since the National Institutes of Health (NIH) conference in 1988, are prohibited from dental implantology, because of diabetes mellitus related microvasculature problems, as well as the delayed wound healing and liability to infection, Heber Arbildo et al found that well controlled diabetic patients have the same implant survival rate as non-diabetic patients.²⁴ This harmonizes with our findings that diabetic patients of poor oral hygiene did not show a worse IMI score. Even more; in terms of statistical significance, their PPD and BOP were less than systemically healthy patients as seen in Figures 6 and 7.

This research agrees with Jepsen et al in 2015 who stated that plaque accumulation led to the development of mucositis, and even more if left as it is without maintenance or re-enforcement on plaque control, 43.9% of cases were presented with peri-implantitis after five years.⁴⁴

One of the explanations of plaque-induced periodontal diseases is related to the isolation of several biomarkers from peri-implant mucositis sulcus; bio-markers such as: AdpB: which is a distinctive surface protein with broad-spectrum extracellular matrix binding abilities associated with *Prevotella* species and FadA: which is an adhesin involved in tissue invasion associated with *Fusobacterium* species.

Other Noxious/Proinflammatory Cytokines

Interleukin-1 β (IL-1 β), tumor necrosis factor- α (TNF- α), macrophage inflammatory protein (MIP-1 α), Interleukin-8 (IL-8), Interleukin-6 (IL-6), type I collagen degradation product (ICTP), matrix metalloproteinase (MMP-1) and cathepsins.

Proteins Associated with Connective Tissue Destruction

Keratins, titin, actin- and microtubule-associated proteins, I-plastin, histone (H4, H1.2) apolipoprotein and others.^{44,45}

These pro-inflammatory markers promote the inflammatory process in gingiva around the implant and peri-implant mucositis is initiated.

If plaque control is not followed properly, Pokrowiecki et al suggested more serious markers to be released, such as receptor activator of nuclear factor (NF)-kappa B ligand (RANKL) which is also known as the osteoclast differentiation factor that promotes bone resorption and the conversion from peri-implant mucositis to peri-implantitis,⁴⁵ the expression of RANKL in periodontal diseases was studied by Matarese et al who found out that RANKL and Osteoprotegerin (OPG) are linked to Transglutaminase-2 (TG2), an enzyme that is produced by periodontal cells in periodontal disease progression, OPG is produced by periodontal ligament fibroblasts to hinder/prevent bone resorption by inactivating pre-osteoclast differentiation, this enzyme (TG2) disturbs the RANKL/OPG ratio, favoring the RANKL expression, leading to serious tissue changes such as alveolar bone resorption.⁴⁶

Limitations

1. The use of the CPITN probe, a better result and also safer for the implants and implant/tissue interface is to use plastic and/or carbon probes designed for this purpose.
2. Cross-sectional design.
3. Limited external validity.

Conclusion

In absence of proper plaque control, systemic diseases showed no impact on the initiation and severity of peri-implant mucositis when compared to patients without systemic diseases, with regard to probing pocket depth and bleeding on probing. Thus, prioritizing plaque control and supportive periodontal/peri-implant therapy is recommended.

Disclosure

The authors declare that they have no conflict of interests related to this work.

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