

Impact of Maintenance Therapy for the Prevention of Peri-implant Diseases: A Systematic Review and Meta-analysis

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Abstract

At the present time, peri-implantitis has become a global burden that occurs with a frequency from 1% to 47% at implant level. Therefore, we aimed herein at assessing the impact of peri-implant maintenance therapy (PIMT) on the prevention of peri-implant diseases. Electronic and manual literature searches were conducted by 3 independent reviewers using several databases, including MEDLINE, EMBASE, Cochrane Central Register of Controlled Trials, and Cochrane Oral Health Group Trials Register, for articles up to June 2015 without language restriction. Articles were included if they were clinical trials aimed at demonstrating the incidence of peri-implant diseases under a strict regime or not of PIMT. Implant survival and failure rate were studied as secondary outcomes. A meta-analysis was conducted to evaluate the influence of PIMT and other reported variables upon peri-implant diseases. Thirteen and 10 clinical trials were included in the qualitative and quantitative analysis, respectively. Mucositis was affected by history of periodontitis and mean PIMT at implant and patient levels, respectively. Similarly, significant effects of history of periodontal disease were obtained for peri-implantitis for both implant and patient levels. Furthermore, mean PIMT interval was demonstrated to influence the incidence of peri-implantitis at implant but not patient level. PIMT interval showed significance at both levels. For implant survival, implants under PIMT have 0.958 the incident event than those with no PIMT. Within the limitations of the present systematic review, it can be concluded that implant therapy must not be limited to the placement and restoration of dental implants but to the implementation of PIMT to potentially prevent biologic complications and hence to heighten the long-term success rate. Although it must be tailored to a patient's risk profiling, our findings suggest reason to claim a minimum recall PIMT interval of 5 to 6 mo. Additionally, it must be stressed that even in the establishment of PIMT, biologic complications might occur. Thus, patient-, clinical-, and implant-related factors must be thoroughly explored.

Keywords: peri-implantitis, periodontitis, mucositis, risk factors, dental implants, evidence-based dentistry

Introduction

Over the past decades, the utilization of dental implants for oral rehabilitation has been considered the standard treatment alternative in a broad variety of scenarios due to its apparent predictability (Jung et al. 2008). Nonetheless, the steady increases of biologic complications (i.e., mucositis and peri-implantitis) involving implants are triggering a shift in clinicians' decision making of saving questionable natural dentition (Rasperini et al. 2014). While mucositis is defined as the presence of reversible inflammatory soft tissue infiltrate, peri-implantitis involves the loss of bone beyond the physiologic crestal bone remodeling (Zitzmann and Berglundh 2008). Presently, peri-implantitis constitutes a global burden that occurs at a frequency from 1% to 47% at implant level (Zitzmann and Berglundh 2008; Atieh et al. 2013; Derks and Tomasi 2015; Jepsen et al. 2015). This fact may be due in part to the lack of consensus in terminology, etiology, and diagnostic criteria (Salvi and Lang 2004; Zitzmann and Berglundh 2008; Atieh et al. 2013). First, Mombelli et al. (1987) described it as an infectious disease that shares features with chronic periodontitis. Currently, although the hypothesis of bacterial infection due to plaque accumulation as

the etiologic factor is still accepted (Jepsen et al. 2015), it does not appear to be a unifactorial disease, where patient-, surgical-, and prosthetic-related indicators may contribute to its development

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and severity (Heitz-Mayfield 2008; Albrektsson et al. 2012; Konstantinidis et al. 2015).

In the arena of periodontology, supportive periodontal therapy has been demonstrated to be essential in preventing the incidence or recurrence of periodontal diseases (Lovdal et al. 1961; Rosling et al. 1976; Nyman et al. 1977; Ramfjord 1987; Axelsson et al. 2004). The protocol is tailored according to a patient's risk profiling (Tonetti et al. 2015). For instance, in the presence of history of periodontal therapy, subgingival microbiota containing large numbers of spirochetes and motile rods may recolonize the pockets 4 to 8 wk after scaling (Magnusson et al. 1984). Likewise, routine maintenance of dental implants has been recommended to prudently circumvent peri-implant inflammation (Wilson et al. 2014). Certainly, understanding the nature of peri-implant tissues (Berglundh et al. 1991; Moher et al. 2009) and their disease pattern (Albouy et al. 2008) would be important to consider, even surpassing importance. Hultin et al. (2007) conducted a systematic review to evaluate whether peri-implant maintenance therapy (PIMT) is effective in prevention of biologic complications. Although its possible role in peri-implant tissue stability was identified, they could not draw conclusive results due to the lack of evidence. Henceforth, because of the increasing research interest within this field and the paramount concern of peri-implant disease prevention as the utmost effective intervention to maintain tissue stability (Jepsen et al. 2015), the present systematic review aims at assessing the impact of maintenance therapy on the incidence of peri-implant diseases.

Material and Methods

This review was written and conducted according to the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-analyses; Moher et al. 2009; see Appendix Fig.).

Focused Question

What is the impact of PIMT upon the incidence of biologic complications (i.e., mucositis and peri-implantitis)?

PICO Question: Patient, Intervention, Comparative, Outcome (Stone 2002)

- P:** Mandibular and/or maxillary complete or partial edentulous healthy subjects in need of dental implants to restore oral function
- I:** Enrollment in regular recall interval for PIMT after implant placement/intervention for treatment of peri-implant disease
- C:**
 - C_1 : No regular interval for PIMT
 - C_2 : Longer interval for PIMT compared with the test group
- O:**
 - Primary outcome:* Incidence of biologic complications (i.e., peri-implant mucositis and peri-implantitis at implant and patient levels)

Secondary outcomes: Implant survival rate (ISR) and implant failure rate (IFR)

Information Sources and Data Extraction

Electronic and manual literature searches were conducted by 3 independent reviewers (A.M., L.A., K.T.D.) in several databases, including MEDLINE, EMBASE, Cochrane Central Register of Controlled Trials, and Cochrane Oral Health Group Trials Register, for articles up to June 2015 without language restriction. Three reviewers independently extracted the data from studies (L.A., K.T.D., A.M.). Publications that did not meet the inclusion criteria were excluded. In case of disagreement, consensus was reached by discussion with a fourth reviewer (M.A.A.)

Screening Process

For the PubMed library, combinations of controlled terms (MeSH and Emtree) and keywords were used whenever possible. In addition, other terms not indexed as MeSH and filters were applied. As such, the key terms used were as follows:

(((((dental implant [MeSH Terms]) OR implantation, endosseous [MeSH Terms]) AND maintenance [MeSH Terms]) OR prophylaxis, dental [MeSH Terms]) OR periodontal attachment loss [MeSH Terms])

This preliminary screening was limited to “humans” and “clinical trials.” A second, broader screening was conducted owing to the small number of articles found indexed with the preliminary screening strategy:

(((((dental implants [MeSH Terms]) OR endosseous dental implantation [MeSH Terms]) AND supportive periodontal therapy) OR maintenance) AND peri-implantitis).

Again, “humans” and “clinical trials” were applied as restricted studies. On the other side, for the EMBASE and Cochrane Libraries the key terms used were

(*Title, Abstract, Keywords*): dental implant AND supportive therapy OR maintenance AND peri-implantitis OR biologic.

The screening in such databases were limited to “clinical trials” AND “humans.” In addition, an electronic screening of the grey literature at the New York Academy of Medicine Grey Literature Report (<http://greylit.org>) was conducted as recommended by high standards for systematic reviews (i.e., Assessment of Multiple Systematic Reviews [AMSTAR] guidelines; Shea et al. 2009).

Additionally, a manual search of periodontics-related journals, including *Journal of Dental Research*, *Journal of Clinical Periodontology*, *Journal of Periodontology* and the *International Journal of Periodontics and Restorative Dentistry*,

from January 2014 up to February 2015, was performed to ensure a thorough screening process. References of included articles were also screened to check all available articles.

Eligibility Criteria

Articles were included in this systematic review if they met the following inclusion criteria:

- Prospective or retrospective, randomized or not, cohort or case series trials involving human subjects aimed at showing the incidence or recurrence of peri-implant diseases under a strict regime of PIMT or not
- Rough surface implant, with or without smooth surface collar
- Subjects, $N \geq 10$
- Clinical trials with >6-mo follow-up
- Articles where the frequency of PIMT could not be clearly extracted were included in the qualitative but not the quantitative analysis (meta-analysis)

Systematic reviews, animal trials, case reports, in vitro studies, and those studies that did not meet the inclusion criteria were excluded. Furthermore, for quantitative assessment, clear descriptions of PIMT intervals as well as incidence of biologic complications had to be reported. In case of unclear data, authors were contacted to provide the data.

Risk of Bias

Two reviewers (A.M. and M.A.A.) designed and assessed the proposal for the present project to make sure the PRISMA and AMSTAR guidelines were followed to avoid risk of bias and provide a high level of evidence. PRISMA consists of a 27-item checklist and a 4-phase flow diagram. Additionally, AMSTAR guidelines (Shea et al. 2009) were followed to ensure high quality regarding the methodology of this systematic review, with incidence of peri-implant diseases the primary outcome.

Qualitative Assessment

The Newcastle-Ottawa Scale (NOS) was used to evaluate the methodological quality of nonrandomized included studies. Two independent reviewers (M.A.A. and A.M.) evaluated all the included articles (Stang 2010). The topics evaluated were selection of study groups, comparability of patients, and outcome. Each included study received a maximum of 13 points for cohort studies and 10 points in case-control studies. The Cohen kappa coefficient was used to assess interrater agreement.

Statistic Analysis

A multivariate negative binomial regression was used to examine potential effects of PIMT; history of periodontitis and mean PIMT interval on incidence of mucositis (at patient and implant levels); and peri-implantitis (at patient and implant levels),

ISR, implant success rate, and IFR. The follow-up period was the exposure variable, and the number of participants (or implants, as appropriate) was used as weights. Robust standard errors were used. For the calculation of PIMT interval on incidence of biologic complications, PIMT range and mean PIMT were found to be highly correlated ($r = 0.80$); therefore, PIMT range was considered as a single independent factor. The range was computed as the difference among the 3 extremes of the PIMT treatment. A null range value was assigned to the control non-PIMT groups.

Results

Screening Process

A total of 1742 records were identified through the electronic search, 50 citations from the manual search, and 89 records from other sources (grey literature, references list, and unpublished articles). After exclusion of duplicates, there were 1877 records of potential interest to screen. Then, after excluding articles based on their titles and abstracts, 31 studies were left for full-text assessment. Finally, 18 studies were excluded for not meeting the strict inclusion criteria (Appendix Table 1), and 13 studies reporting the incidence or prevalence of biologic complications in patients under a clearly reported PIMT were considered for qualitative analysis (Fig. 1; Table).

Influence of PIMT upon primary outcomes (Figs. 2–5)

Implant level. For mucositis, a negative binomial model was fitted. History of periodontal disease showed negative effects ($z = -8.12$, $P < 0.001$; lower mucositis with larger number of patients with history of periodontitis). Moreover, PIMT interval was shown to significantly influence the incidence of mucositis at this level ($z = 8.64$, $P < 0.001$). Significant effects of treatment ($z = -19.04$, $P < 0.001$), history of periodontitis ($z = -14.64$, $P < 0.001$; increased peri-implantitis with larger number of patients with history of periodontitis), and mean PIMT ($z = -29.31$, $P < 0.001$) were obtained for peri-implantitis. Additionally, a significant effect was found for the intervals of PIMT on the incidence of peri-implantitis at this level ($z = -5.26$, $P < 0.001$).

Patient level. For mucositis, there were significant effects of treatment ($z = -14.36$, $P < 0.001$), history of periodontitis ($z = -5.83$, $P < 0.001$; lower mucositis with larger number of periodontal disease patients), and mean PIMT interval ($z = -21.07$, $P < 0.001$; lower peri-implantitis with larger interval). PIMT range did show an influence on mucositis at this level ($z = -3.07$, $P = 0.002$). For peri-implantitis, the same negative binomial model was applied. Significant effects of treatment ($z = -16.63$, $P < 0.001$), history of periodontal disease ($z = 3.79$, $P < 0.001$; increased peri-implantitis with larger number of patients with history of periodontal disease), and mean PIMT ($z = -3.94$, $P < 0.001$) were observed. In addition, PIMT interval demonstrated a significant effect on the incidence of peri-implantitis at this level ($z = -26.51$, $P < 0.001$).

Table. Descriptive Information of the Included Studies in the Qualitative Analysis.

Subjects, <i>n</i>	PIMT, Mean Interval, mo ^a	Implants, <i>n</i>	Author Year; Follow-up, mo							
			Mucositis Level, %		Peri-implantitis Level, %		MBL, mm	ISR, %	ISS, %	IFR, %
			Patient	Implant	Patient	Implant				
Aguirre-Zorzano et al. 2013; 12										
27	4	123	18.5	NR	3.7	NR	0.16 ± 0.15	100	NR	0
22	0	123	50	NR	22.7	NR	0.62 ± 0.94	99.18	NR	0.82
Costa et al. 2012; 60										
39	11	157	UC	UC	18	10.8	UC	99.37	NR	0.63
41	0	183	UC	UC	43.9	28.4	UC	98.4	NR	1.6
Frisch et al. 2013; 168										
22	7.5 (3 to 12)	89	36.4	21.3	9.1	8	1.8 ± 1.5	98.9	NR	1.1
Karoussis et al. 2004; 120										
89	4.5 (3 to 6)	179	NR	NR	NR	15.4	UC	92.4	69.8	7.6
Swierkot et al. 2012; 60 to 192										
35	3	22	74.2	56	42.8	26	NR	96	33	4
18	3	30	44.4	40	11.1	10	NR	100	50	0
Pjetursson et al. 2012; 95										
70	NR	165	NR	NR	38.6	22.2	UC	95.8	NR	4.2
Cho-Yan et al. 2012; 96										
30	NR	56	NR	NR	36.7	26.7	0.45 ± 0.94	98.4	NR	1.6
30	NR	61	NR	NR	16.7	13.1	0.26 ± 0.72		NR	
Degidi et al. 2012; 120										
59	6	210	NR	10.1	NR	8.2	1.93 ± 0.40	97.62	65.26	2.38
Aguirre-Zorzano et al. 2015; 63										
239	5 (4 to 6)	786	24.7	12.8	15.1	9.8	4.3 ± 1.9	NR	NR	NR
Ferreira et al. 2006; 6 to 60										
94	3.5 (1 to 6)	578	64.6	61.7	8.9	8.5	NR	NR	NR	NR
118	>6			66.95		9.3	NR			
Marrone et al. 2013; 102										
58	NR	266	31	38	34.5	23	3.8	NR	NR	NR
45	NR				40					
Mir-Mari et al. 2012; 76										
245	4.5 (3 to 6)	964	38.8	21.6	16.3	9.1	UC	NR	NR	NR
Rinke et al. 2011; 68										
58	4.5 (3 to 6)		43.1	NR	3.44	NR	UC	NR	NR	NR
31	5 (4 to 6)		48.3	NR	25.8	NR	UC			

IFR, implant failure rate; ISR, implant survival rate; ISS, implant success rate; MBL, marginal bone loss; NR, not reported; PIMT, peri-implant maintenance therapy; UC, unclear.

Outcomes: mucositis level, peri-implantitis level, marginal bone loss, implant survival rate, implant success rate, implant failure rate.

^aRange in parentheses.

Influence of PIMT upon secondary outcomes

Implant survival rate. Effects of mean PIMT ($z = -7.88$, $P < 0.001$) were obtained, as were marginal effects of history of periodontitis ($z = 1.91$, $P = 0.056$). Implants under PIMT have 0.958 the incident event than those with no PIMT.

Implant failure rate. Significant effects of history of periodontitis ($z = 38.03$, $P < 0.001$) and mean PIMT ($z = -30.59$, $P = 0.001$).

Quality Assessment

After the screening process, 13 studies included in the qualitative assessment were analyzed with NOS (see Appendix Table 2). A Cohen kappa interrater agreement rate of 0.89 was reached. After the disagreements were discussed between the examiners

(M.A.A. and A.M.), a mean NOS score of 5.30 ± 1.32 was obtained. Among the included observational studies, Cho-Yan Lee et al. (2012) obtained the highest quality, with 8 stars.

Discussion

Principal Findings

Periodontal supportive therapy adherence has shown to be of crucial importance for the long-term maintenance of natural dentition (Becker, Becker, et al. 1984; Becker, Berg, et al. 1984; Lindhe et al. 1984; Lindhe and Nyman 1984). Maintenance around dental implants should be considered of utmost importance due to the nature of peri-implant tissues (Tomasi et al. 2014); nonetheless, to date, there is no consensus on the ideal interval of PIMT for the adequate care of dental

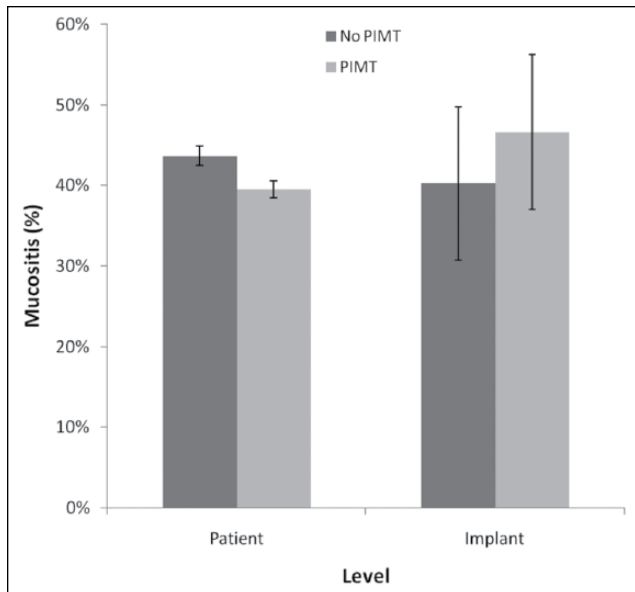


Figure 1. Mucositis at patient and implant levels as a function of treatment. PIMT, peri-implant maintenance therapy.

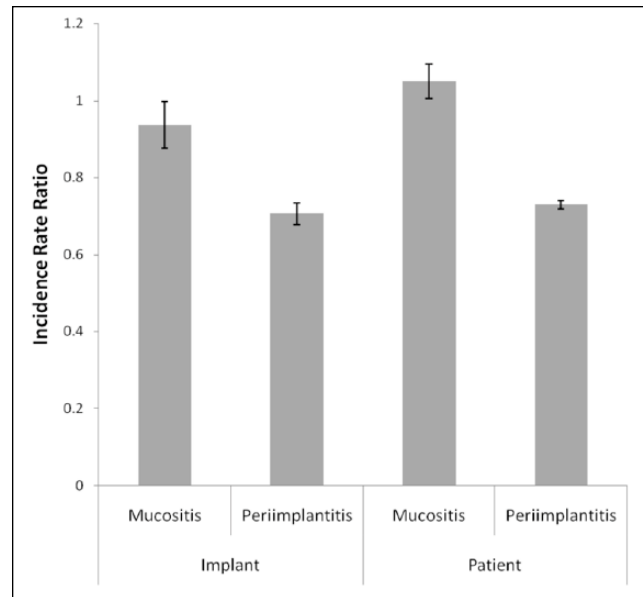


Figure 3. Incidence rate ratio of mucositis and peri-implantitis at patient and implant levels as a function of range (in months) of peri-implant maintenance therapy.

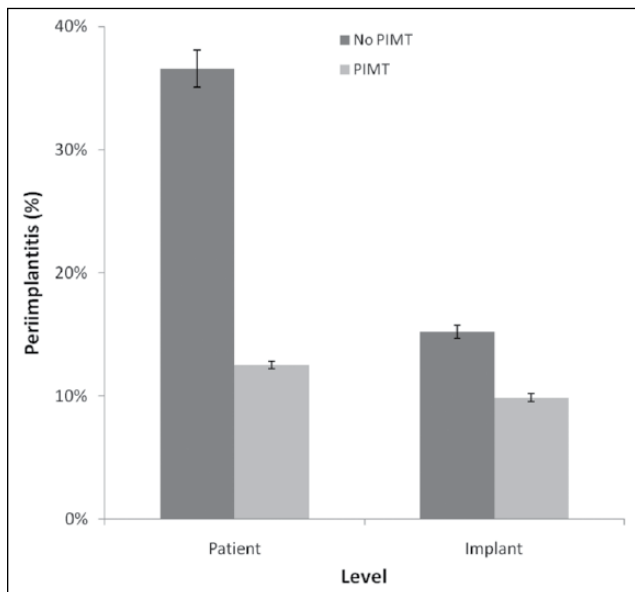


Figure 2. Peri-implantitis at patient and implant levels as a function of treatment. PIMT, peri-implant maintenance therapy.

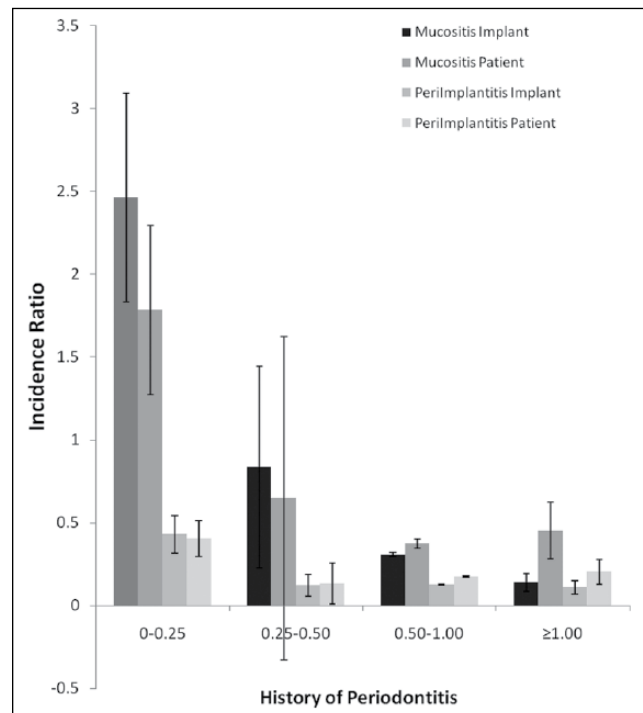


Figure 4. Incidence of mucositis and peri-implantitis at implant and patient levels according to history of periodontitis (years).

implants. The present systematic review showed the positive impact of PIMT on peri-implant tissue health as well as ISR. Moreover, these findings showed that the reasonable interval of PIMT might be 5 to 6 mo because of the positive significant impact on incidence of peri-implantitis. By any means, PIMT as supportive periodontal therapy must be customized according to patients' risk profiling. Furthermore, we have corroborated robust previous findings (Schou et al. 2006; Roos-Jansåker 2007; Pjetursson et al. 2012; Konstantinidis et al. 2015)

showing the critical role of periodontal disease history and that it contributes to the incidence of mucositis and peri-implantitis. Therefore, in some subjects, further implementation of PIMT could be suggested because of the higher risk of developing peri-implant diseases.

Agreements and Disagreements with Previous Studies

Owing to the nature of the matter investigated, the lack of bias-free studies (no randomized controlled trials due to ethical principles) allows us to compare our findings. A previous systematic review aimed at studying the influence of supportive peri-implant maintenance on long-term ISR could not draw clear conclusions because of the poor evidence available (Hultin et al. 2007). Atieh et al. (2013) more recently suggested the effect of supportive periodontal therapy upon the rate of occurrence of periodontal diseases. A current clinical trial suggested that the simple fact of enrolling subjects for preventive PIMT may reduce the risk of peri-implantitis from 43.9% to 18% at patient level (Costa et al. 2012). Likewise, Aguirre-Zorzano et al. (2013) noted that by the regular performance of PIMT with a mean recall of 4 mo, peri-implant diseases could be nearly prevented. Rinke et al. (2011) showed similar findings when comparing regular versus irregular PIMT recalls. Hence, in agreement with results from this systematic review, it demonstrated a relevant impact of PIMT and its frequency upon the prevention of peri-implant diseases.

Clinical Implications

Peri-implantitis and periodontal disease are entities that have been defined as infectious (Lang et al. 2011). Accordingly, tissue maintenance to prevent their occurrence is imperative. Long-term longitudinal studies in the area of periodontology showed the positive effect of maintenance therapy to minimize tooth loss (Hirschfeld and Wasserman 1978; Becker, Becker, et al. 1984; Becker, Berg, et al. 1984; Lindhe and Nyman 1984). In the arena of implantology on the contrary, there is still a lack of longitudinal studies to determine its actual significance. The cumulative interceptive supportive therapy was developed on the basis of certain clinical and radiographic parameters to monitor, detect, and arrest inflammation involving peri-implant tissues (Lang et al. 2000). However, to date, there is no evidence-based guideline/protocol for prevention of peri-implant diseases. Therefore, according to our results, their incidence can be minimized with routine control to identify any possible etiologic or contributing factor of peri-implantitis. Previously, several studies on experimental peri-implant mucositis in humans demonstrated the cause and effect between the accumulation of bacterial plaque and the development of mucositis and its reversibility once plaque control is reinstated (Pontoriero et al. 1994; Zitzmann et al. 2001; Salvi et al. 2012). In this sense, one other interesting fact that can be inferred from our results is that even in the strict enrollment of the PIMT, biologic complications may occur. As such, even now considering the plaque-dependent hypothesis as the primary etiologic factor (Jepsen et al. 2015), local contributing factors must be further explored. As a matter of fact, in the few longitudinal studies, an important aspect was pointed out: that implant-, clinician-, and patient-related factors might trigger a so-called foreign-body reaction jeopardizing peri-implant

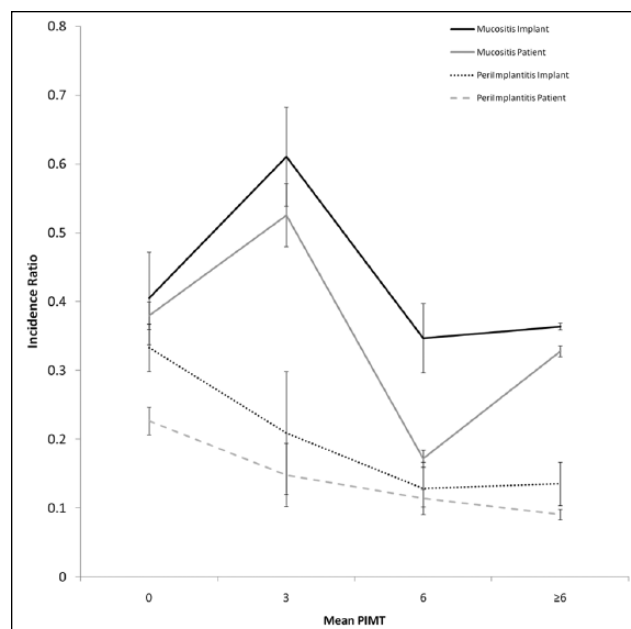


Figure 5. Incidence ratios for mucositis and peri-implantitis at implant and patient levels as a function of mean peri-implant maintenance therapy (PIMT).

bone-level stability and leading to a noninfective peri-implant lesion (Albrektsson et al. 2012).

Limitations and Recommendations for Future Research

It is important to highlight major limitations from the present work. First, because of ethical principles, peri-implantitis cannot be studied by restricting the patient from conducting oral hygiene to observe the appearance and development of the disease. Therefore, the questionable design of the studies within this matter may not reflect the state of the art on PIMT. Additionally, the large range of intervals evaluated may not accurately reveal its impact on the incidence of biologic complication. For instance, it does not seem logical that in a 2- to 3-mo PIMT program follow-ups, the incidence of peri-implantitis at patient and implant levels is higher as compared with that at 5 to 6 mo. Furthermore, recent evidence demonstrated that periodontitis and peri-implantitis lesions exhibit critical histopathologic differences that may help in the understanding in the onset and progression of peri-implantitis (Carcuac and Berglundh 2014). Also, it was shown that implant surface characteristics may influence spontaneous progression of peri-implantitis (Albouy et al. 2008). Therefore, because of our inclusion criteria to minimize risk of bias (i.e., rough surface implants with >6 mo of follow-up), caution must be exercised when interpreting and extrapolating our findings to the daily implant practice.

Thus, although of clinical relevance, findings from the present study must be cautiously interpreted. Since this review has demonstrated that PIMT is highly imperative, future longitudinal research should focus on the frequency of PIMT recall on

the incidence of peri-implant diseases and its burden on arresting mucositis. In addition, local as well as systemic contributing factors responsible for triggering an inflammatory process around dental implants in the lack of infection must be studied. Last but not the least, because of the lack of consistent consensus in the definition (Zitzmann and Berglundh 2008; Charalampakis et al. 2014), investigations must be conducted to determine to what extent bone loss around dental implants should be considered “peri-implantitis” as an entity and thus the need of nonsurgical or surgical intervention to arrest disease progression.

Conclusions

Within the limitations of the present systematic review, it can be concluded that implant therapy must not be limited to the placement and restoration of dental implants but to the implementation of PIMT to potentially prevent biologic complications and hence heighten the long-term success rate. Although it must be tailored to a patient’s risk profiling, our findings suggest reason to claim a minimum recall PIMT interval of 5 to 6 mo. Additionally, it must be stressed that even in the establishment of PIMT, biologic complications might occur. Hence, patient-, clinical-, and implant-related factors must be thoroughly explored.

Author Contributions

A. Monje, contributed to conception, design, and data interpretation, drafted and critically revised the manuscript; L. Aranda, contributed to data acquisition and interpretation, drafted and critically revised the manuscript; K.T. Diaz, M.A. Alarcón, R.A. Bagramian, contributed to data interpretation, drafted and critically revised the manuscript; H.L. Wang, contributed to conception, design, and data interpretation, critically revised the manuscript; A. Catena, contributed to conception, data acquisition, analysis and interpretation, critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

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References

Aguiar-Zorzano LA, Vallejo-Aisa FJ, Estefania-Fresco R. 2013. Supportive periodontal therapy and periodontal biotype as prognostic factors in implants placed in patients with a history of periodontitis. *Med Oral Patol Oral Cir Bucal*. 18(5):786–792.

Aguiar-Zorzano LA, Estefania-Fresco R, Telletxea O, Bravo M. 2015. Prevalence of peri-implant inflammatory disease in patients with a history

of periodontal disease who receives supportive periodontal therapy. *Clin Oral Implants Res*. 26(11):1338–1344.

Albouy JP, Abrahamsson I, Persson LG, Berglundh T. 2008. Spontaneous progression of peri-implantitis at different types of implants: an experimental study in dogs. I: clinical and radiographic observations. *Clin Oral Implants Res*. 19(10):997–1002.

Albrektsson T, Buser D, Chen ST, Cochran D, DeBruyn H, Jemt T, Koka S, Nevins M, Sennerby L, Simion M, et al. 2012. Statements from the Estepona Consensus Meeting on Peri-implantitis, February 2–4, 2012. *Clin Implant Dent Relat Res*. 14(6):781–782.

Atieh MA, Alsabeeha NH, Faggion CM Jr, Duncan WJ. 2013. The frequency of peri-implant diseases: a systematic review and meta-analysis. *J Periodontol*. 84(11):1586–1598.

Axelsson P, Nystrom B, Lindhe J. 2004. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults: results after 30 years of maintenance. *J Clin Periodontol*. 31(9):749–757.

Becker W, Becker BE, Berg LE. 1984. Periodontal treatment without maintenance: a retrospective study in 44 patients. *J Periodontol*. 55(9):505–509.

Becker W, Berg L, Becker BE. 1984. The long term evaluation of periodontal treatment and maintenance in 95 patients. *Int J Periodontics Restorative Dent*. 4(2):54–71.

Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. 1991. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res*. 2(2):81–90.

Carcuac O, Berglundh T. 2014. Composition of human peri-implantitis and periodontitis lesions. *J Dent Res*. 93(11):1083–1088.

Charalampakis G, Jansaker E, Roos-Jansaker AM. 2014. Definition and prevalence of peri-implantitis. *Curr Oral Health Rep*. 1(4):239–250.

Cho-Yan Lee J, Mattheos N, Nixon KC, Ivanovski S. 2012. Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis. *Clin Oral Implants Res*. 23(3):325–333.

Costa FO, Takenaka-Martinez S, Cota LO, Ferreira SD, Silva GL, Costa JE. 2012. Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *J Clin Periodontol*. 39(2):173–181.

Degidi M, Nardi D, Piattelli A. 2012. 10-year follow-up of immediately loaded implants with TiUnite porous anodized surface. *Clin Implant Dent Relat Res*. 14(6):828–838.

Derks J, Tomasi C. 2015. Peri-implant health and disease: a systematic review of current epidemiology. *J Clin Periodontol*. 42(Suppl 16):158–171.

Ferreira SD, Silva GL, Cortelli JR, Costa JE, Costa FO. 2006. Prevalence and risk variables for peri-implant disease in Brazilian subjects. *J Clin Periodontol*. 33(12):929–935.

Frisch E, Ziebolz D, Rinke S. 2013. Long-term results of implant-supported over-dentures retained by double crowns: a practice-based retrospective study after minimally 10 years follow-up. *Clin Oral Implants Res*. 24(12):1281–1287.

Heitz-Mayfield LJ. 2008. Peri-implant diseases: diagnosis and risk indicators. *J Clin Periodontol*. 35(Suppl 8):292–304.

Hirschfeld L, Wasserman B. 1978. A long-term survey of tooth loss in 600 treated periodontal patients. *J Periodontol*. 49(5):225–237.

Hultin M, Komiya A, Klinge B. 2007. Supportive therapy and the longevity of dental implants: a systematic review of the literature. *Clin Oral Implants Res*. 18(Suppl 3):50–62.

Jepsen S, Berglundh T, Genco R, Aass AM, Demirel K, Derks J, Figuero E, Giovannoli JL, Goldstein M, Lambert F, et al. 2015. Primary prevention of peri-implantitis: managing peri-implant mucositis. *J Clin Periodontol*. 42(Suppl 16):152–157.

Jung RE, Pjetursson BE, Glauser R, Zembic A, Zwahlen M, Lang NP. 2008. A systematic review of the 5-year survival and complication rates of implant-supported single crowns. *Clin Oral Implants Res*. 19(2):119–130.

Karoussis IK, Brägger U, Salvi GE, Bürgin W, Lang NP. 2004. Effect of implant design on survival and success rates of titanium oral implants: a 10-year prospective cohort study of the ITI Dental Implant System. *Clin Oral Implants Res*. 15(1):8–17.

Konstantinidis IK, Kotsakis GA, Gerdes S, Walter MH. 2015. Cross-sectional study on the prevalence and risk indicators of peri-implant diseases. *Eur J Oral Implantol*. 8(1):75–88.

Lang NP, Berglundh T; Working Group 4 of Seventh European Workshop on Periodontology. 2011. Periimplant diseases: where are we now? Consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol*. 38(Suppl 11):178–181.

Lang NP, Wilson TG, Corbet EF. 2000. Biological complications with dental implants: their prevention, diagnosis and treatment. *Clin Oral Implants Res*. 11(Suppl 1):146–155.

Lindhe J, Nyman S. 1984. Long-term maintenance of patients treated for advanced periodontal disease. *J Clin Periodontol*. 11(8):504–514.

- Lindhe J, Westfelt E, Nyman S, Socransky SS, Haffajee AD. 1984. Long-term effect of surgical/non-surgical treatment of periodontal disease. *J Clin Periodontol.* 11(7):448–458.
- Lovdal A, Arno A, Schei O, Waerhaug J. 1961. Combined effect of subgingival scaling and controlled oral hygiene on the incidence of gingivitis. *Acta Odontol Scand.* 19:537–555.
- Magnusson I, Lindhe J, Yoneyama T, Liljenberg B. 1984. Recolonization of a subgingival microbiota following scaling in deep pockets. *J Clin Periodontol.* 11(3):193–207.
- Marrone A, Lasserre J, Bercy P, Brex MC. 2013. Prevalence and risk factors for peri-implant disease in Belgian adults. *Clin Oral Implants Res.* 24(8):934–940.
- Mir-Mari J, Mir-Orfila P, Figueiredo R, Valmaseda-Castellón E, Gay-Escoda C. 2012. Prevalence of peri-implant diseases: a cross-sectional study based on a private practice environment. *J Clin Periodontol.* 39(5):490–494.
- Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. 2009. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *BMJ.* 339:b2535.
- Mombelli A, van Oosten MA, Schurch E Jr, Land NP. 1987. The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiol Immunol.* 2(4):145–151.
- Nyman S, Lindhe J, Rosling B. 1977. Periodontal surgery in plaque-infected dentitions. *J Clin Periodontol.* 4(4):240–249.
- Pjetursson BE, Helbling C, Weber HP, Matuliene G, Salvi GE, Brägger U, Schmidlin K, Zwahlen M, Lang NP. 2012. Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. *Clin Oral Implants Res.* 23(7):888–894. Published erratum in *Clin Oral Implants Res.* 2012;23(8):1004.
- Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. 1994. Experimentally induced peri-implant mucositis: a clinical study in humans. *Clin Oral Implants Res.* 5(4):254–259.
- Ramfjord SP. 1987. Maintenance care for treated periodontitis patients. *J Clin Periodontol.* 14(8):433–437.
- Rasperini G, Siciliano VI, Cafiero C, Salvi GE, Blasi A, Aglietta M. 2014. Crestal bone changes at teeth and implants in periodontally healthy and periodontally compromised patients: a 10-year comparative case-series study. *J Periodontol.* 85(6):152–159.
- Rinke S, Ohl S, Ziebolz D, Lange K, Eickholz P. 2011. Prevalence of peri-implant disease in partially edentulous patients: a practice-based cross-sectional study. *Clin Oral Implants Res.* 22(8):826–833.
- Roos-Jansåker AM. 2007. Long time follow up of implant therapy and treatment of peri-implantitis. *Swed Dent J Suppl.* 188:7–66.
- Rosling B, Nyman S, Lindhe J, Jern B. 1976. The healing potential of the periodontal tissues following different techniques of periodontal surgery in plaque-free dentitions: a 2-year clinical study. *J Clin Periodontol.* 3(4):233–250.
- Salvi GE, Aglietta M, Eick S, Sculean A, Lang NP, Ramseier CA. 2012. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clin Oral Implants Res.* 23(2):182–190.
- Salvi GE, Lang NP. 2004. Diagnostic parameters for monitoring peri-implant conditions. *Int J Oral Maxillofac Implants.* 19 Suppl:116–127.
- Schou S, Holmstrup P, Worthington HV, Esposito M. 2006. Outcome of implant therapy in patients with previous tooth loss due to periodontitis. *Clin Oral Implants Res.* 17(Suppl 2):104–123.
- Shea BJ, Hamel C, Wells GA, Bouter LM, Kristjansson E, Grimshaw J, Henry DA, Boers M. 2009. AMSTAR is a reliable and valid measurement tool to assess the methodological quality of systematic reviews. *J Clin Epidemiol.* 62(10):1013–1020.
- Stang A. 2010. Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. *Eur J Epidemiol.* 25(9):603–605.
- Stone PW. 2002. Popping the (PICO) question in research and evidence-based practice. *App Nurs Res.* 15(3):197–198.
- Swierkot K, Lottholz P, Flores-de-Jacoby L, Mengel R. 2012. Mucositis, peri-implantitis, implant success, and survival of implants in patients with treated generalized aggressive periodontitis: 3- to 16-year results of a prospective long-term cohort study. *J Periodontol.* 83(10):1213–1225.
- Tomasi C, Tassarolo F, Caola I, Wennstrom J, Nollo G, Berglundh T. 2014. Morphogenesis of peri-implant mucosa revisited: an experimental study in humans. *Clin Oral Implants Res.* 25(9):997–1003.
- Tonetti MS, Chapple IL, Jepsen S, Sanz M. 2015. Primary and secondary prevention of periodontal and peri-implant diseases: introduction to, and objectives of the 11th European Workshop on Periodontology consensus conference. *J Clin Periodontol.* 42(Suppl 16):S1–S4.
- Wilson TG Jr, Valderrama P, Rodrigues DB. 2014. The case for routine maintenance of dental implants. *J Periodontol.* 85(5):657–660.
- Zitzmann NU, Berglundh T. 2008. Definition and prevalence of peri-implant diseases. *J Clin Periodontol.* 35(8 Suppl):286–291.
- Zitzmann NU, Berglundh T, Marinello CP, Lindhe J. 2001. Experimental peri-implant mucositis in man. *J Clin Periodontol.* 28(6):517–523.