

Comprehensive Evaluation of Correlation Between Clinical Parameters of Peri-Implantitis and Periodontitis: An Original Study

Gurinder Bir Singh Thind^{1*} Amita Kashyap² Ashish Loomba³ Ankita Jaiswal⁴ Sharib Abdus Salam⁵ Rajalakshmi⁶

¹Private Consultant, S. Sewa Singh Memorial Dental Care Clinic, Kharar, Punjab, India.

²Private Consultant, S. Sewa Singh Memorial Dental Care Clinic, Kharar, Punjab, India.

³Private Consultant, Dr. Loomba's Family Dental Clinic, Mohali, Punjab, India.

⁴Senior Lecturer, Department of Orthodontics, Vyas Dental College, Jodhpur, India.

⁵Lecturer, Department of Periodontics, Patna Dental College & Hospital, Patna, Bihar, India.

⁶PG Student, Department of Oral Pathology, Consultant Oral Pathologist and Dental Surgeon, India.

ABSTRACT

Background: As we all know that the microbial flora in the natural dentition sulcus/pocket and the implant crevice/pocket is very similar in both health and disease. In health, coccal forms predominate, and in disease, large numbers of Gram-negative pathogens are associated with both tooth and implant. This study was aimed to evaluate any correlation between clinical parameters of peri-implantitis and periodontitis.

Materials & Methods: This study was exclusively based on a cross sectional ideology. A total of 60 patients those attending department of prosthodontics were approached in their post treatment phases. We used post operative radiographs to compare the amount of bone loss before and after implant placement. Clinical symptomatic correlations were also attempted. Response was recorded and data was processed statistically to evaluate intended parameters.

Results: Statistical analysis was done using statistical software Statistical Package for the Social Sciences (SPSS). The recorded data was subjected to suitable statistical tests to obtain p values, mean, standard deviation, standard error and 95% CI. $P \leq 0.05$ was considered as statistically significant. Total 8 subjects were belonging to age group >75 years. Only 20 subjects were belonging to the age range of 41-50 years therefore we can presume that most of the subjects were belonging to older age groups. P value was significant in group III of age range 61-70 years.

Conclusion: Within the limitations of the study authors concluded that while periimplantitis and periodontitis share clinical characteristics, they represent distinct entities. They further concluded that approaches aiming to simplify the molecular mechanisms of peri-implantitis pathophysiology can only partially be addressed in periodontitis setups.

Keywords: Periodontitis, Peri-implantitis, Implant.

INTRODUCTION

Presently, implants are categorized as endosteal (implants within the bone), subperiosteal (framework placed on bone), or transosteal

(implants placed through the bone from the superior to the inferior aspect). The endosteal is the most commonly used at this time and can be either

Received: Aug. 5, 2019; Accepted: Sept. 2, 2019

*Correspondence Dr. Gurinder Bir Singh Thind.

S. Sewa Singh Memorial Dental Care Clinic, Kharar, Punjab, India.

Email: Not Disclosed

root-, blade-, or plate-form. Periodontitis can be considered the consequence of broken balances in bacterial components of the plaque. Its prevalence drives to its consideration as the most prevalent infectious disease in the community, with 75% of adults affected as reported in published studies. Several studies have identified similarities in the pathogenesis of late periodontitis and peri-implantitis, showing intra-oral translocation of periodontal pathogens from teeth showing chronic periodontitis to the peri-implant niche, producing at last the lost of affected teeth or implants.¹⁻² Peri-implantitis has been defined as an inflammematory condition involving dental implants, surrounding mucosa and bone, which lose supporting bone. Although high success rates for endosseous implants have been reported, failures occur, and some implants are lost or removed. At least 10% of the failures have been suggested to be the result of peri-implantitis. One of the major causes of the peri-implantitis is the bacterial colonization of implant surfaces but additional risk factors such as periodontitis, poor oral hygiene, tobacco consumption, prepost operative therapies and genetic susceptibility should be considered. However, the long term implant's stability depends on the integration between fixtures and the surrounding bone. Microbia presented in the oral cavity has a substantial impact on biofilm formation on newly placed implants. Since periodontally compromised patients have a higher risk of peri-implantitis than unaffected patients, a transmission of periodontal pathogens from periodontal sites to implants is possible. Some authors studied the possible association between a previous history of periodontitis and peri-implantitis, and indicated that subjects with a history of periodontitis might be at greater risk for peri-implant infections.³ Other authors evaluated the prevalence of peri-implant diseases around implants and the possible relationship with periodontal bone loss, systemic condition, and demographic profile. Presence of peri-implant diseases may be associated with generalized periodontal bone loss and with poor oral hygiene.⁴⁻⁵ This study was aimed to evaluate any correlation between clinical parameters of peri-implantitis and periodontitis.

MATERIALS & METHODS

The present study was conducted to evaluate any correlation between clinical parameters of peri-

implantitis and periodontitis. This study was exclusively based on a cross sectional ideology. A total of 60 patients those attending department of prosthodontics were approached in their post treatment phases. The contact details of the subjects were obtained from the registry of department digital record database. We used post operative radiographs to compare the amount of bone loss before and after implant placement. Clinical symptomatic correlations were also attempted. Response was recorded and data was processed statistically to evaluate intended parameters. We have decided to conduct this study since they are exceptionally useful to obtain detailed information about personal and group perceptions and opinions. They are also competent of saving time and money while analyzing the subjects at individual levels. In addition, they also give a broader range of clinical data with better clarification and understanding. The privacy policy and other rights of the study participants were absolutely ensured. Informed consent was obtained from the subjects those were voluntarily ready for participation. The importance of this study was explained in detail to all general dental practitioners. Results thus obtained was tabulated and subjected to basic statistical analysis. P value less than 0.05 was considered significant ($p < 0.05$).

STATISTICAL ANALYSIS AND RESULTS

Responses those obtained from questionnaire exercise were sent for statistical analysis using statistical software Statistical Package for the Social Sciences version 21 (IBM Inc., Armonk, New York, USA). The resulting data was subjected to suitable statistical tests to obtain p values, mean, standard deviation, chi- square test, standard error and 95% CI. Table 1 and Graph 1 showed that out of 60 subjects, males were 45 and females were 15. Total 8 subjects were belonging to age group >75 years. Only 20 subjects were belonging to the age range of 41-50 years therefore we can presume that most of the subjects were belonging to older age groups. P value was significant in group III of age range 61-70 years. Total 8 subjects were found in the age of >75 (Table 1, Graph 1). Analysis of clinical parameters in around preserved implant has been shown in Table 2. Bone losses of different grades around implant were also noted (Figure 1).

Table 1: Age & gender wise distribution of subjects.

Age Group (Yrs)	Male	Female	Total	P value
41-50	13	7	20	0.09
51-60	5	0	5	0.50
61-70	13	1	4	0.01*
70-75	10	3	13	0.90
>75	4	4	8	0.08
Total	45	15	60	*Significant

* $p < 0.05$ significant

Graph 1: Age & gender wise distribution of practitioners.

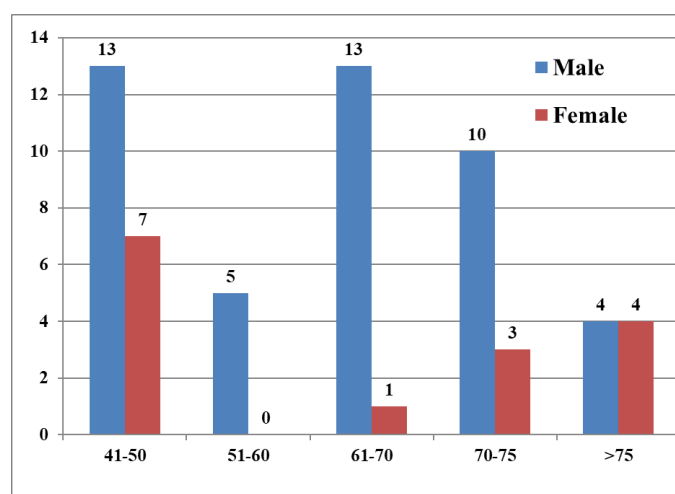


Table 2: Analysis of clinical parameters in around preserved implant.

Amount of Bone Loss					p value
Dental plaque (%)					
No plaque	16	12	18	14	0.20
Present on probing	18	10	18	14	0.01*
Visible plaque	16	18	19	7	0.00*
Pocket probing depth (mm)	Less than 1 mm (n= 15)	1-2 m (n= 15)	2-3 mm (n= 15)	More than 3 mm (n= 15)	-
Mesio-vestibular	1.0	1.90	2.80	3.50	0.70
Vestibular	0.80	1.80	2.60	3.30	0.10
Distovestibular	0.20	1.80	2.10	3.80	0.01*

Mesio-lingual	0.90	1.70	2.80	3.10	0.00*
Lingual	0.80	1.30	2.70	3.90	0.80
Disto-lingual	0.60	1.20	2.80	3.60	0.70
Implant mobility (%)	0.90	1.5	0.9	1.8	0.02*
Bleeding on probing (%)	24	26	23	27	0.01*
Pain (%)	0.0	0.2	0.8	0.3	0.09
Suppuration (%)	1.7	2.8	3.6	2.8	0.00*

* $p < 0.05$ significant

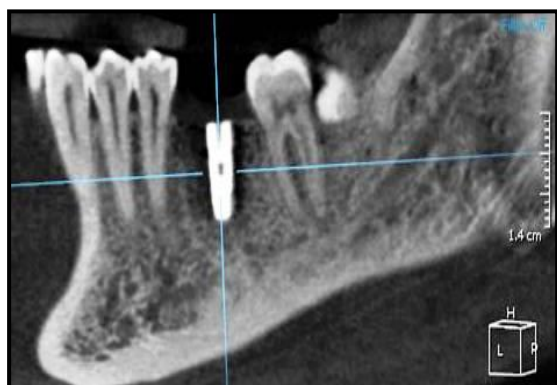


Fig 1: Bone loss around implant as illustrated on radiograph.

DISCUSSION

Periodontitis and peri-implantitis are two major oral diseases that we have to deal with in periodontal practice. They are polymicrobial inflammatory diseases that lead to the destruction of the tissue supporting the tooth/implant. Without treatment, they result in tooth/implant loss. Despite many discoveries over the past 20 years regarding the etio-pathogenesis of periodontal and peri-implant diseases, as well as significant advances in our understanding of microbial biofilms, the incidence of these pathologies still continues to rise. Since bacteria are the main cause of periodontitis and peri-implantitis, some methods have been used for microbiological testing in periodontitis.⁶ However, many techniques have not been fully accepted due to low sensitivity or specificity, moreover sometimes they are slow, expensive and

laborious. Polymerase chain reaction (PCR) test is rapid and sensitive especially if few but high sensitive bacteria are investigated such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*. Both *P. gingivalis* and *T. denticola* occur concomitantly with the clinical signs of periodontal destruction.⁷⁻⁹ They are considered the first pathogens involved in the clinical destruction of periodontal tissues. Moreover both them and *T. forsythia*, show a higher prevalence in disease than in health suggesting that these bacterial are associated with the local development of periodontitis and peri-implantitis. Peri-implantitis (PI) is a bacterial-induced inflammatory condition around dental implants, which leads to progressive crestal bone loss beyond the initial bone remodeling phase and can eventually lead to implant loss. Peri-implant health is characterized by the absence of clinical signs of inflammation, such as swelling, redness, and BoP.¹⁰⁻¹² It is not possible, however, to define a range of probing depths that are compatible with health. In addition, peri-implant health can exist around implants with reduced bone support. There are different scenarios in which peri-implant health may coincide with reduced bone support, as peri-implant health can be achieved at sites successfully treated for peri-implantitis. In addition, healing following implant placement in sites with ridge deficiencies may result in a bone level located apical of the implant margin and with parts of the peri-implant mucosa facing the intraosseous portion of the implant. Peri-implantitis is a plaque-associated pathological

condition that occurs in tissues around dental implants. It is characterised by inflammation in the peri-implant mucosa and loss of supporting bone.¹³⁻¹⁵ Peri-implantitis sites exhibit clinical signs of inflammation including bleeding on probing and/or suppuration, increased probing depths and/or recession of the mucosal margin, and radiographic bone loss compared to previous examinations. Peri-implantitis lesions extend apical of the junctional/pocket epithelium and are larger than those at peri-implant mucositis and periodontitis sites.¹⁶

CONCLUSION

Periodontitis and peri-implantitis are two major biofilm-induced inflammatory diseases that lead to the loss of teeth and oral implants if left untreated. These diseases are also associated with systemic conditions such as diabetes or cardiovascular diseases. Within the limitations of the study authors concluded that while periimplantitis and periodontitis share clinical characteristics, they represent distinct entities. They further concluded that approaches aiming to simplify the molecular mechanisms of peri-implantitis pathophysiology can only partially be addressed in periodontitis setups. Our study results must be treated as suggestive for predicting clinical outcomes for such critical situations. However, we expect some other large scale studies to be conducted that could further set certain standard norms.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

REFERENCES

1. Lang NP, Berglundh T. Periimplant diseases: where are we now? – Consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol* 2011; 38(Suppl 11):178–181.
2. Heitz-Mayfield LJ, Lang NP. Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontol* 2000 2010; 53:167–181.
3. Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. *Clin Oral Implants Res* 1992; 3:1–8.
4. Quirynen M, Vogels R, Peeters W, van Steenberghe D, Naert I, Haffajee A. Dynamics of initial subgingival colonization of “pristine” peri-implant pockets. *Clin Oral Implants Res* 2006; 17:25–37.
5. Heydenrijk K, Meijer HJ, van der Reijden WA, Raghoobar GM, Vissink A, Stegenga B. Microbiota around root-form endosseous implants: a review of the literature. *Int J Oral Maxillofac Implants* 2002; 17:829–838.
6. Kocar M, Seme K, Hren NI. Characterization of the normal bacterial flora in peri-implant sulci of partially and completely edentulous patients. *Int J Oral Maxillofac Implants* 2010; 25:690–698.
7. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. *Eur J Oral Sci* 1998; 106:527–551.
8. Kotsovilis S, Karoussis IK, Fourmoussis I. A comprehensive and critical review of dental implant placement in diabetic animals and patients. *Clin Oral Implants Res* 2006; 17:587–599.
9. van Winkelhoff AJ. [Consensus on peri-implant infections]. *Ned Tijdschr Tandheelkd* 2010; 117:519–523.
10. Carcuac O, Jansson L. Peri-implantitis in a specialist clinic of periodontology. Clinical features and risk indicators. *Swed Dent J* 2010; 34:53–61.
11. Furst MM, Salvi GE, Lang NP, Persson GR. Bacterial colonization immediately after installation on oral titanium implants. *Clin Oral Implants Res* 2007; 18:501–508.
12. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. *J Clin Periodontol* 1998; 25:134–144.
13. Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. *Clin Oral Implants Res* 1994; 5:254–259.
14. Demmer RT, Behle JH, Wolf DL, et al. Transcriptomes in healthy and diseased gingival tissues. *J Periodontol* 2008; 79:2112–2124.
15. Jonsson D, Ramberg P, Demmer RT, Kerschull M, Dahlen G, Papapanou PN. Gingival tissue

- transcriptomes in experimental gingivitis. J Clin Periodontol 2011; 38:599–611.
16. Sung HY, Guan H, Czibula A, et al. Human tribbles-1 controls proliferation and chemotaxis of smooth muscle cells via MAPK signaling pathways. J Biol Chem 2007; 282:18379–18387.