



CORRELATION BETWEEN SLEEP AS ASSESSED USING PITTSBURG SLEEP QUALITY INDEX (PSQI) AND CHRONIC PERIODONTITIS - A CROSS – SECTIONAL ANALYTICAL STUDY

Periodontology

Eshani Yeragi* PG Student, YMT Dental College and Hospital, Navi Mumbai *Corresponding Author

Kavita Pol Nalawade Reader and Guide, YMT Dental College and Hospital, Navi Mumbai

Saurabh Gotmare PG Student, YMT Dental College and Hospital, Navi Mumbai

Swati Rathod PG Student, YMT Dental College and Hospital, Navi Mumbai

ABSTRACT

Sleep plays a crucial role along with the diet and exercise for overall wellbeing. According to FitBit Survey India ranks 2nd worldwide for being sleep deprived country. Sleep deprivation is known to adversely affect cognition and motor performance. This might impair an individual's capacity to perform adequate oral hygiene practices, thus increasing the risk of periodontal disease. **Aim and Objectives:** - To assess if there is an association of sleeping less than 7 hours a night and chronic periodontitis in individuals of age 25 years and above. **Methodology:** - 94 subjects were divided equally into 2 groups (n=47): Clinically healthy and periodontitis. Clinical parameters assessed were Gingival index, Plaque index and Pocket probing depth. All the study subjects were administered PSQI questionnaire for the assessment of sleep deprivation. Results: Present study revealed that mean PSQI was highest in periodontitis group as compared to healthy group and the difference among the two groups was statistically significant. **Conclusion:** The present study suggests strong association of sleep deprivation with severity of periodontal disease.

KEYWORDS

Sleep deprivation, Chronic periodontitis, Pittsburgh Sleep Quality Index.

1. Introduction

Sleep as defined by Cambridge University the resting state in which the body is not active and the mind is unconscious¹. A good night sleep is incredibly important for health and it's just as important as eating healthy and exercising. Unfortunately, the current environment and modern lifestyle is interfering with natural sleep patterns. People are now sleeping less than they did in the past, and sleep quality has decreased as well. Inadequate sleep has its effects on learning, memory processing, the repair of cell damage, brain development, neurobehavioral performance, hormonal regulation, risk of depression, increased cortisol, and ghrelin, impaired glucose metabolism, and increased inflammatory and proinflammatory markers among many other influences². It is also known that there has been a worldwide decrease in the average number of hours that people sleep since the mid-1970s. Currently, the average number of hours that a person sleeps is less than seven hours a night¹. As compared to earlier generation people are sleeping fewer hours.

Short sleep duration has been independently linked to several diseases, particularly to diabetes mellitus, metabolic syndrome, hypertension, stroke and coronary artery disease (Yaggi et al. 2006; Kim et al. 2015; Gottlieb et al. 2006; Qureshi et al. 1997; King et al. 2008; Kanagasabai & Ardern 2015)^{3,4,5,6,7,8}. Periodontitis is a ubiquitous chronic inflammatory disease affecting the supporting structures of the teeth and if not promptly diagnosed and correctly managed can ultimately lead to tooth loss⁹.

Due to its potential to influence systemic inflammation and oxidative stress, which are the two main pathogenetic mechanisms actually recognized in the periodontal damage, and to predispose to bacterial infections, extreme sleep durations could potentially be a risk factor for periodontitis (Kanagasabai & Ardern 2015; Patel et al. 2009; Patel et al. 2012)^{8,10,11}. Due to its modifiability, it would be particularly interesting to highlight such a link, if present.

Hence the aim of the study was to assess if there is an association of sleeping less than 7 hours a night and chronic periodontitis in individuals of age 25 years and above.

Null hypothesis - There is no association of sleeping less than 7 hours a night and chronic periodontitis in individuals of age 25 years and above.

2. Material and Methods

A total of 94 subjects were categorized in 2 groups (n = 47 each). The subjects were identified and selected from among the patients visiting the Department of Periodontology and Oral Implantology, Y. M. T.

Dental College and Hospital. All study subjects underwent detailed medical history and periodontal examination before enrolment.

Inclusion Criteria:-

1. Subjects within age group of 25 to 55 years.
2. Subjects with chronic periodontitis.
3. For Group A – Subjects with Gingival Index score ≤ 1 , PPD ≤ 3 .
4. For Group B – Subjects with Gingival Index score > 1 , PPD ≥ 4

Subjects were excluded if they were pregnant, edentulous, or lactating, smokers, suffering from known systemic diseases which could alter healing response of periodontium, who had received any periodontal therapy in 6 months before study or those who had history of medication in 3 months before study (Anti-inflammatory/Antibiotics). Subjects were examined by a single examiner for the assessment of gingival index (GI) and pocket probing depth (PPD). William's periodontal probe was used to measure the PPD from the gingival margin to the bottom of the periodontal sulcus.

[Sample size was determined using the correlation estimates from literature using the formula

$$\text{Total sample size} = N = [(Z_{\alpha} + Z_{\beta})/C]^2 + 3$$

where Z_{α} is the z variate of alpha error i.e. a constant with value 1.96,

Z_{β} is the z variate of beta error i.e. a constant with value 0.84

$$C = 0.5 * \ln[(1+r)/(1-r)]$$

Approximate estimates:

1. 80% power
2. Type I error to be 5%
3. Type II error to be 20%
4. Minimum correlation between the 2 techniques as 0.4

Approximately **47 samples per group** need to be taken in the present study.

i.e. 47 healthy

&

47 Subjects with chronic periodontitis]

Subject grouping:

Group A – Healthy patients : GI score: ≤ 1 , PPD ≤ 3 mm

Group B – Chronic Periodontitis patients: GI score > 1 , PPD ≥ 4 mm

Both the groups were given Pittsburgh sleep quality index questionnaire for sleep analysis.

The Pittsburgh Sleep Quality Index:-

The PSQI was developed in 1988, by Buysse and his colleagues, to create a standardized measure designed to gather consistent

information about the subjective nature of people's sleep habits and provide a clear index that both clinicians and patients can use^{12,13,14}. It gained popularity as a measure that could be used in research that looks at how sleep might be associated with sleep disorders, depression, and bipolar disorder.

Consisting of 19 items, the PSQI measures several different aspects of sleep, offering seven component scores and one composite score. The component scores consist of subjective sleep quality, sleep latency (i.e., how long it takes to fall asleep), sleep duration, habitual sleep efficiency (i.e., the percentage of time in bed that one is asleep), sleep disturbances, use of sleeping medication, and daytime dysfunction¹⁵. Each item is weighted on a 0–3 interval scale. The global PSQI score is then calculated by totaling the seven component scores, providing an overall score ranging from 0 to 21, where lower scores denote a healthier sleep quality¹⁵.

Traditionally, the items from the PSQI have been summed to create a total score to measure overall sleep quality. Statistical analyses also support looking at three factors, which include sleep efficiency (using sleep duration and sleep efficiency variables), perceived sleep quality (using subjective sleep quality, sleep latency, and sleep medication variables), and daily disturbances (using sleep disturbances and daytime dysfunctions variables)^{16,17}.

3. Statistical Analysis

Data obtained was compiled on a MS Office Excel Sheet (v 2010) and was subjected to statistical analysis using Statistical package for social sciences (SPSS v 21.0, IBM).

Inter group comparison of indices & Scores between both the groups has been done using t test.

Bivariate analysis was done in each of the 2 groups considering the indices as predictor variable & PSQI score as the outcome variable. Also linear regression estimates & plots have been depicted for each group & overall. Correlation between the 2 groups (considering numerical data as the outcome) has been done using Kendall's tau_b. Frequency & percentage of subjects with normal & abnormal sleep vs groups was compared using Phi coefficient. Odds ratio has been depicted using a 2x2 contingency table. Relationship of indices with scores has been established using Multiple linear regression For all the statistical tests, p<0.05 was considered to be statistically significant, keeping α error at 5% and β error at 20%, thus giving a power to the study as 80%.

4. Result

Results of the present investigation elucidated that mean PSQI was highest in the periodontitis group and lowest in healthy subjects and the difference among two groups was statistically significant. A positive correlation of PSQI with GI and PPD was observed in group B suggesting that PSQI scores commensurate with periodontal destruction. There was a statistically highly significant difference seen for the inter group comparison of the values for GI, PI, PPD & PSQI score (p<0.01) with higher values of indices & PSQI score in group B as compared to group A.

There was a low correlation & weak relationship between PSQI score & indices while a slight correlation & negligible relationship between PPD & score although statistically non significant (p>0.05).

There was a statistically highly significant difference seen in the number of subjects with good sleep vs abnormal sleep between both the groups, with higher no with good sleep in group A while higher no of subjects with abnormal sleep in group B. (p<0.01)

Table 1:- Bivariate Correlations for group A

		GINGIVAL INDEX	PI	PPD
PSQI SCORE	Pearson Correlation	.204	-.265	-.039
	Sig. (2-tailed)	.174	.076	.797
	N	46	46	46

Table 2:- Bivariate Correlations for group B

		Gingival Index	PI	PPD
PSQI SCORE	Pearson Correlation	.432**	.320*	.502**
	Sig. (2-tailed)	.003	.030	.000
	N	46	46	46

Table 3:- Correlation between the 2 groups (considering numerical data as the outcome) using Kendall's tau_b

		PSQI SCORE	
Kendall's tau_b	GROUPS	Correlation Coefficient	.684**
		Sig. (2-tailed)	.000
		N	92

There was a moderate correlation & substantial relationship between PSQI score & groups with statistically highly significant difference (p<0.01)

Interpretation of Kendall's tau_b

- 0 is no relationship,
- 1 is a perfect relationship

Table:- 4 Inter group comparison of indices & Scores

	GROUPS	N	Mean	Std. Deviation	Std. Error Mean	T value	p value
Gingival index	A	46	.50	.506	.075	-18.518	0.000**
	B	46	2.39	.493	.073		
PI	A	46	.46	.504	.074	-18.280	0.000**
	B	46	2.30	.465	.069		
PPD	A	46	1.50	.723	.107	-25.936	0.000**
	B	46	5.67	.818	.121		
PSQI Score	A	46	3.20	1.185	.175	-15.499	0.000**
	B	46	12.26	3.786	.558		

** = statistically highly significant difference (p<0.01)

= non significant difference (p>0.05) ... for all tables

* = statistically significant difference (p<0.05)

Comparison of Frequency of subjects with normal & abnormal sleep vs groups using Phi coefficient.

GROUP vs score categories				
Score categories			Total	
Abnormal sleep (>5)			Good sleep (<5)	
GROUP	A	5	41	46
	B	42	4	46
Total		47	45	92

Odds ratio = 0.0116, indicates lower risk of having abnormal sleep in group A as compared to group B.

		Value	p
Nominal by Nominal	Phi	-.805	.000

There was a statistically highly significant difference seen in the number of subjects with good sleep vs abnormal sleep between both the groups, with higher no with good sleep in group A while higher no of subjects with abnormal sleep in group B. (p<0.01)

Multiple linear regression

Model		Unstandardized Coefficients	Standardized Coefficients	t	Sig.
		B	Std. Error	Beta	
1	(Constant)	.416	.517		.805
	Gingival index	1.721	.517	.345	3.332
	PI	.044	.610	.009	.072
	PPD	1.328	.336	.555	3.956

a. Dependent Variable: PSQIScore

PSQI score = 0.416 + 1.721 x GI + 0.044 x PI + 1.328 x PPD.

While only GI & PPD have statistical significance.

5. Discussion

The present study showed that there is an association between sleep deprivation and periodontitis. Present study revealed that mean GI in group A and B were 0.50 and 2.39 respectively, whereas PPD were 1.50 and 5.67 respectively and PI were 0.46 and 2.30 respectively [Table 4]. Mean PSQI score in two groups was 3.20 and 12.26 respectively. The results of the present study are in agreement with previously conducted studies. In a study by Romandini et al 2017¹⁸ has highlighted an independent direct association between sleep duration and prevalence of periodontitis. Grover et al. 2015¹⁹ also revealed that mean PSQI was highest in the periodontitis group as compared to other two groups (clinically healthy, gingivitis groups) of 60 participants, ages 25 to 50 years living in Punjab. However, the present study results are in disagreement with Weiner et al 2016²⁰ which showed that association of sleep duration (<7 h/night), and periodontitis in individuals who

were aged 30 years and above failed to reach the association at a significant level in the adjusted analysis. The PSQI has 89.6% sensitivity and 86.5% specificity for identifying "good" and "bad" sleep using a cut-off global score of 5. It also has internal consistency and a reliability coefficient (Cronbach's alpha) of 0.83 for its seven components^{12,21}.

There are four possible mechanisms which have been proposed¹⁸. First, long sleep duration could potentially induce systemic inflammation Patel et al. 2009¹⁰ and this could have an effect in the development of periodontitis Pink et al. 2015²². Second, the immunity system impairment due to long sleep duration¹¹ could potentially increase the susceptibility to the periodontal disease which involves the interaction of bacterial, host and environmental factors (Seymour et al. 2015)²³. Third, some risk factors of periodontitis could be predisposed by a prolonged sleep duration (e.g. diabetes) and then work potentially as mediators in a hypothetical causal pathway between the same long sleep duration and periodontitis (Hall et al. 2008; Shan et al. 2015; Genco & Borgnakke 2013)^{24,25,26}. The fourth hypothetical mechanism is a "reverse causation": the systemic inflammation caused by periodontitis (Gocke et al. 2014; D'Aiuto et al. 2004)^{27,28} could potentially have an effect on sleep duration. In fact, some cytokines have shown to have a sleep-inducing effect, which may predispose people with elevated levels of inflammation to a prolonged sleep duration (Dowd et al 2011)²⁹. It has been found that in sleep-deprived individuals there is increased lymphocyte activation with over productions of IL-1, IL-6, IL-7, and TNF- α ³⁰. A study conducted by (Irwin et al 2006)³⁰ found that 4 hr of sleep restriction in one night led to increasing in monocyte production of IL-6 and TNF- α messenger RNA³¹. Many studies have found that there is a significant increase in IL-1 β and IL-1ra and a significant decrease in CRP and IL-6 in 40 h sleep-deprived individuals. Also it has been reported that racial differences, particularly elevation in CRP level, only in long sleepers among Asians, compared to other races, which have exhibited elevated levels of CRP even in short sleepers³⁰. More extensive research is required with larger sample size so as to confirm the association between less hours of sleep and periodontitis.

6. Conclusion

This study focuses on to assess if there is an association of sleeping less than 7 hours a night and chronic periodontitis in individuals of age 25 years and above. It was revealed that mean PSQI was highest in periodontitis group as compared to healthy group and the difference among the two groups was statistically significant. However further research needs to be conducted in this aspect so as to find the exact correlation and significance between sleep and periodontitis and that initial preventive measures like lifestyle modification and healthy sleep pattern could be inculcated.

7. Compliance with ethical standards

Ethical approval:- This study was conducted only after the approval of the ethical committee.

8. Conflict of interest

The authors certify that they have no commercial or associative interest that represents a conflict of interest in connection with the manuscript.

REFERENCES

- [1] <https://dictionary.cambridge.org/es-LA/dictionary/english/sleep>
- [2] AlDabal L., BaHammam A. S. (2011) Metabolic, endocrine, and immune consequences of sleep deprivation. *Open Respiratory Medicine Journal*;5(1):31-43.
- [3] Yaggi, H. K., Araujo, A. B. & McKinlay, J. B. (2006) Sleep duration as a risk factor for the development of type 2 diabetes. *Diabetes Care* 29, 657-661.
- [4] Kim, J. Y., Yadav, D., Ahn, S. V., Koh, S. B., Park, J. T., Yoon, J., Yoo, B. S. & Lee, S. H. (2015) A prospective study of total sleep duration and incident metabolic syndrome: the ARIRANG study. *Sleep Med* 16, 1511-1515.
- [5] Gottlieb, D. J., Redline, S., Nieto, F. J., Baldwin, C. M., Newman, A. B., Resnick, H. E. & Punjabi, N. M. (2006) Association of usual sleep duration with hypertension: the Sleep Heart Health Study. *Sleep* 29, 1009-1014.
- [6] Qureshi, A. I., Giles, W. H., Croft, J. B. & Blwise, D. L. (1997) Habitual sleep patterns and risk for stroke and coronary heart disease: a 10-year follow-up from NHANES I. *Neurology* 48, 904-911.
- [7] King, C. R., Knutson, K. L., Rathouz, P. J., Sidney, S., Liu, K. & Lauderdale, D. S. (2008) Short sleep duration and incident coronary artery calcification. *Jama* 300, 2859-2866.
- [8] Kanagasabai, T. & Arden, C. I. (2015) Contribution of Inflammation, Oxidative Stress, and Antioxidants to the Relationship between Sleep Duration and Cardiometabolic Health. *Sleep* 38, 1905-1912.
- [9] Nowjack-Raymer RE, Sheiham A. (2003) Association of edentulism and diet and nutrition in US adults. *J Dent Res*;82:123-6.
- [10] Patel, S. R., Zhu, X., Storf-Isner, A., Mehra, R., Jenny, N. S., Tracy, R. & Redline, S. (2009) Sleep duration and biomarkers of inflammation. *Sleep* 32, 200-204.
- [11] Patel, S. R., Malhotra, A., Gao, X., Hu, F. B., Neuman, M. I. & Fawzi, W. W. (2012) A prospective study of sleep duration and pneumonia risk in women. *Sleep* 35, 97-101.
- [12] Buysse, Daniel J.; Reynolds, Charles F.; Monk, Timothy H.; Berman, Susan R.; Kupfer, David J. (May 1989). "The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research". *Psychiatry Research*. 28 (2): 193-213.
- [13] Mollaveya, T; Thurairajah, P; Burton, K; Mollaveya, S; Shapiro, CM; Colantonio, A (2015). "The Pittsburgh sleep quality index as a screening tool for sleep dysfunction in clinical and non-clinical samples: A systematic review and meta-analysis". *Sleep Medicine Reviews*. 25: 52-73.
- [14] Currie, S.R. (2008). "Sleep Disorders". In Hunsley, John; Mash, Eric (eds.). *A Guide to Assessments that Work*. New York, NY: Oxford Press. pp. 535-550.
- [15] https://en.wikipedia.org/wiki/Pittsburgh_Sleep_Quality_Index
- [16] Tomfohr, LM; Schweizer, CA; Dimsdale, JE; Lored, JS (2013). "Psychometric characteristics of the Pittsburgh Sleep Quality Index in English speaking non-Hispanic whites and English and Spanish speaking Hispanics of Mexican descent". *Journal of Clinical Sleep Medicine*. 9 (1): 61-6.
- [17] Cole, J.C.; Motivala, S.J.; Buysse, D.J.; Oxman, M.N.; Levin, M.J.; Irwin, M.R. (2006). "Validation of a 3-factor scoring model for the Pittsburgh Sleep Quality Index in older adults". *Sleep-New York then Westchester*. 29 (1): 112-116.
- [18] Romandini M, Gioco G, Perletti G, Deli G, Staderini E, Lafori A *J Clin Periodontol*. 2017 May; 44(5):490-501.
- [19] Grover V., Malhotra R., Kaur H. Exploring association between sleep deprivation and chronic periodontitis: a pilot study. *Journal of Indian Society of Periodontology*. 2015;19(3):304-307.
- [20] Wiener RC. (2016) Relationship of routine inadequate sleep duration and periodontitis in a nationally representative sample. *Sleep Disord*.
- [21] Manzar MD, Zannat W, Hussain ME, Pandi-Perumal SR, Bahammam AS, Barakat D, et al. (2016) Dimensionality of the pittsburgh sleep quality index in the collegiate young adults. *Springerplus*.5:1550.
- [22] Pink, C., Kocher, T., Meisel, P., Dorr, M., Markus, M. R., Jablonowski, L., Grotevendt, A., Nauck, M. & Holtfreter, B. (2015) Longitudinal effects of systemic inflammation markers on periodontitis. *J Clin Periodontol* 42, 988-997.
- [23] Seymour, J. G., Berglundh, T. & Trombelli, L. (2015) Pathogenesis of Periodontitis. In *Clinical Periodontology and Implant Dentistry*, eds. Lang, N.P. & Lindhe, J., pp. 256-259. Oxford: Blackwell Munksgaard.
- [24] Hall, M. H., Muldoon, M. F., Jennings, J. R., Buysse, D. J., Flory, J. D. & Manuck, S. B. (2008) Self-reported sleep duration is associated with the metabolic syndrome in midlife adults. *Sleep* 31, 635-643.
- [25] Shan, Z., Ma, H., Xie, M., Yan, P., Guo, Y., Bao, W., Rong, Y., Jackson, C. L., Hu, F. B. & Liu, L. (2015) Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. *Diabetes Care* 38, 529-537.
- [26] Genco, R. J. & Borgnakke, W. S. (2013) Risk factors for periodontal disease. *Periodontol* 2000 62, 59-94.
- [27] Gocke, C., Holtfreter, B., Meisel, P., Grotevendt, A., Jablonowski, L., Nauck, M., Markus, M. R. & Kocher, T. (2014) Abdominal obesity modifies long-term associations between periodontitis and markers of systemic inflammation. *Atherosclerosis* 235, 351-357.
- [28] D'Aiuto, F., Parkar, M., Andreou, G., Suvan, J., Brett, P. M., Ready, D. & Tonetti, M. S. (2004) Periodontitis and systemic inflammation: control of the local infection is associated with a reduction in serum inflammatory markers. *J Dent Res* 83, 156-160.
- [29] Dowd, J. B., Goldman, N. & Weinstein, M. (2011) Sleep duration, sleep quality, and biomarkers of inflammation in a Taiwanese population. *Annals of epidemiology* 21, 799-806.
- [30] Irwin MR. (2012) Sleep and infectious disease risk. *Sleep*; 35:1025-6.
- [31] Frey DJ, Fleshner M, Wright KP, Jr. (2007). The effects of 40 hours of total sleep deprivation on inflammatory markers in healthy young adults. *Brain Behav Immun.*;21:1050-7.