ORIGINAL RESEARCH PAPER

Volume-7 | Issue-3 | March-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

RELATIONSHIP BETWEEN SLEEP APNEA AND VARIOUS PERIODONTAL DISEASE - A CLINICAL STUDY

Dental Science

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ABSTRACT

Periodontitis is associated with several cardio metabolic disorders that are co morbid with sleep disorders. A relationship between periodontitis and obstructive sleep apnea(OSA) is plausible, but has recieved little attention.This study investigated the strength of association between periodontitis and risk for OSA. In this study 80 non institutionalized participants were selected randomly and divided into 4 groups: 1. Healthy subjects (20) 2. Patients suffering from gingivitis (20) 3. Patients suffering from chronic periodontitis(20) 4. Patients suffering from chronic periodontitis(20). Data from the selected patients was collected by the department of periodontics by Hu Friedy Williams periodontal probes by placing it parellel to the tooth long axis and data collection for gingivitis was done using Loe and Sillness gingival Index. PSQI questionnaire was used to measure the quality and patterns of sleep. Results of the present study elucidated that mean PQSI was highest in the periodontitid group followed by gingivitis and lowest in the healthy subjects and the difference among the three groups was statistically significant.

KEYWORDS

Sleep Apnea, Periodontitis, Bacteria.

INTRODUCTION

Periodontal diseases are caused by pathogenic bacteria locally colonized in the dental biofilm creating infection and subsequent intricate inflammatory response in the periodontal tissues in response to challenge by dental biofilm[1].This inflammation can result in the destruction of the supporting structures, loss of periodontal attachment, pocket formation, loss of alveolar bone and eventual tooth loss. . The possible pathogenesis of periodontitis is an immune-inflammatory reaction provoked by the existence of plaque bacteria developing in the periodontal tissue. The sustained inflammatory mediators and host cellular components release proteolytic enzymes that damage soft and hard tissues. Its inception and evolution are modulated by a variety of risk factors, such as poor oral hygiene, genetics, tobacco and alcohol consumption, poor nutrition, stress, and impaired host immune responses. Various systemic diseases such as diabetes mellitus, cardiovascular diseases, hematologic diseases, and psychosomatic diseases, may play an imperative role in progression of periodontal diseases. Recognizing and eradicating these risk factors which influence inflammation can provide means by which to limit periodontal tissue destruction.

Sleep disorders (Sds), particularly sleep deprivation, may alter the immune system and induce systemic inflammation. It has been shown that sleep deprivation alone may be sufficient to induce low-grade systemic inflammation, a well-established risk factor for cardiovascular disease (CVD), and other conditions, such as diabetes, metabolic syndrome, and cancer. Similarly to SDs, periodontitis has been shown to alter immune and inflammatory pathways implicated in the pathogenesis of CVD. In this perspective, SDs may represent a low-grade systemic inflammatory status that also predisposes to local inflammatory conditions. However, the relationship between sleep and inflammation is highly complex and not fully understood. As previously suggested, the role of sleep in health outcomes may be at least partially driven by social and environmental factors, such as socioeconomic status, depression, stress, and obesity[1].

Sleep deprivation is becoming increasingly common in today's society. Compared to a few decades ago, major changes in sleep culture have been observed globally because there has been a trend toward adopting a 24/7 lifestyle, longer working hours and longer work shifts. Medical conditions and social and domestic responsibilities further contribute to sleep restriction. This has led to a significant reduction in total sleeping hours in both adults and children. Epidemiologic data indicate that sleep disturbance and short sleep duration adversely impact human physical health and mortality risk[2-3].

Inadequate sleep has 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 influences[1].

The purpose of this study is to determine if there is an association of periodontontal disease and sleep.

Material and Methods

The present study was conducted in the department of periodontology, himachal dental college and hospital sundernagar. Ethical clearance was taken from institutional Review Board. The patients were explained about the study and an informed consent was taken.

The data sources for the present study were patients attending OPD of himachal dental college and hospital sundernagar who were selected through simple random sampling.

The total of 80 participants who were non institutionalized and lived in sundernagar H.P were selected and divided into 4 groups

1. Healthy subjects(20)
2. Patients suffering from gingivitis(20)
3. Patient suffering from chronic periodontitis(20)
4. Patient suffering from aggressive periodontitis (20)

The inclusion criteria for the present study was healthy subjects of any age and gender. Subjects were excluded if they were edentulous, pregnant or lactating, smokers, suffering from known systemic diseases which could alter healing response of periodontium. The participants also responded to interview questions involving demographic information and questions regarding sleep using the Pittsburgh Sleep Quality Index.

Data from the selected patients was collected in Department of Periodontics by calibrated examiner using Hu Friedy Williams periodontal probes by placing it parallel to the tooth's long axis and data collection for gingivitis was done using Loe and Silness Gingival index[4-5]

This is a cross sectional study which was done during time interval of four months. The key outcome variable was periodontitis.

The presence of periodontitis was defined as at least 2 interproximal sites with an attachment loss of at least 3mm and at least 2 interproximal sites with probing depths of at least 4mm which are not on the same tooth or at least one site with a probing depth of at least 5mm.[6]

The key variable of interest was routine adequate sleep which was recorded using Pittsburgh Sleep Quality Index.[7]

PSQI is an effective instrument used to measure the quality and patterns of sleep in the older adult. It is brief, reliable, valid, and standardized self-reported measure of sleep quality. It differentiates "poor" from "good" sleep by measuring seven domains: Subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction over the last month. All subjects rated each of these seven areas of sleep. PSQI questionnaire was modified from the original in order to include the first 9 items only these items contribute to the total score.

Scoring of the answers was based on a 0 to 3 scale, whereby 3 reflected the negative extreme on the Likert scale.[8-9] The component scores were summed to produce a global score (range 0 to 21). A global sum of "5" or greater indicated a "poor" sleeper. Higher PSQI scores represented worse sleep quality.[10]

Participants were also asked during an interview to report age, sex, , socioeconomic status, and smoking status among other questions.

Statistical Analyses : SPSS version 21 was used to determine sample descriptions, bivariate associations of the variables of interest with periodontitis, and logistic regression of inadequate sleep on periodontitis in both unadjusted and adjusted analyses. The analyses accounted for stratification, eligibility, and sample weights.

Results

Table 1 : Demographical data

	Age	SES
Healthy	35	3
Gingivitis	37	3
Periodontitis	53	4
Aggressive periodontitis	26	3

Table 1 shows mean age which is found to be 37, 55, 26 and socioeconomic status of 3,3 , 4, 3 patients in all three groups that is gingivitis , periodontitis, aggressive periodontitis.

Table 2 : Mean parameters in 4 groups

Mean ± Standard Deviation			
	GI	PPD	PSQI
Healthy	0	2.04±.37	1.11±.99
Gingivitis	1.40±.41	2.110±.09	1.30±.79
Periodontitis	2.01±0.23	6.91 0±.72	5.04 ±.81
Aggressive periodontitis	1.89±0.17	5.36± 0.49	3.110±.68

GI – Gingival index; PPD – Pocket probing depth;
PSQI-Pittsburgh Sleep Quality Index

Table 2 results revealed that mean G.I in group 1, 2, 3,4 were 1.40+0.41, 2.01+0.2 and 1.89+0.17 respectively where as PDD were 2.11+0.09, 6.91+ 0.72 and 5.36+ 0.49 respectively.

Mean PSQI score in three groups was 1.30+0.79, 5.04 +1.81, 3.11+0.68.

Table 3 : Stepwise Regression Analysis Showing PPD

PPD	Standard Deviation	R-Value	p-Value	Significance
Age	10.058	0.133	0.005*	Significant
SES	1.075	0.098	0.068	Non- Significant
Healthy	5.43	0.11	0.075	Non-Significant
Gingivitis	3.519	0.089	0.081	Non - Significant
Periodontitis	8.451	0.102	0.023*	Significant
Aggressive periodontitis	7.281	0.462	0.005*	Significant

Table 3 shows value of PSQI found to be in significant relationship with age, gingivitis and periodontitis, aggressive periodontitis.

Table 4 : Stepwise Regression Analysis Showing GI

GI	Standard Deviation	R-Value	p-Value	Significance
Age	10.013	0.130	0.003*	Significant
SES	1.055	0.094	0.028*	Significant
Healthy	1.32	0.15	0.062	Non-Significant
Gingivitis	3.11	0.077	0.041*	Significant
Periodontitis	7.621	0.100	0.021*	Significant
Aggressive periodontitis	1.281	0.411	0.004*	Significant

Table 4 result shows a positive co relation of PSQI with G.I and PDD in gingivitis and periodontitis group

Table 5 : Stepwise Regression Analysis Showing PSQI

PSQI	Standard Deviation	R-Value	p-Value	Significance
Age	11.032	0.112	0.004*	Significant
SES	1.131	O.102	0.061	Non-Significant
Healthy	1.111	0.143	0.065	Non-Significant
Gingivitis	3.491	0.084	0.005*	Significant
Periodontitis	7.951	0.119	0.031*	Significant
Aggressive periodontitis	1.281	0.104	0.048*	Significant

Table 6: Multiple Logistic Regression Analysis intergroup comparison of PSQI

Dependant variable	Dental Diseases	Group	SD	p- Value	Significance
PSQI	Healthy	Gingivitis	0.35	<0.003	Significant
		Periodontitis	0.41		
		Aggressive periodontitis	0.37	<0.001	Significant
				<0.002	Significant
	Gingivitis	Healthy	0.33	< 0.004	Significant
					Significant
		Periodontitis	0.41	<0.001	
					Significant
		Aggressive periodontitis	0.37	< 0.002	
	Periodontitis	Healthy	0.33	<0.004	Significant
		Gingivitis	0.35	< 0.003	Significant
		Aggressive Periodontitis	0.37	< 0.002	
	Aggressive periodontitis	Healthy	0.33	<0.004	Significant
					Significant
		Gingivitis	0.35	< 0.003	
					Significant
		Periodontitis	0.41	<0.001	

The mean difference is significant at the 0.05 level. **Intergroup comparison is highly significant at $p \leq 0.001$. SD – Standard deviation; PSQI – Pittsburgh Sleep Quality Index

Discussion

This study was conducted to find whether there is a co- relation between sleep deprivation and periodontal diseases such as gingivitis , chronic periodontitis and aggressive periodontitis.

The variables taken in our study are age, socioeconomic status, gingival index , probing pocket depth and PSQI. It has been seen that PSQI is having a positive relationship with gingivitis, periodontitis and aggressive periodontitis. and age is having positive relationship with sleep deprivation.

The current investigation was aimed at assessing association of sleep deprivation with chronic periodontal disease. Results of the present investigation elucidated that mean PSQI was highest in the periodontitis group followed by gingivitis subjects and lowest in healthy subjects and the difference among three groups was statistically significant. A positive correlation of PSQI with GI and

PPD was observed in groups I and II suggesting that PSQI scores commensurate with periodontal destruction. The association was still significant after controlling for age, gender, and socioeconomic status.

Varieties of methods have been utilized for assessment of sleep quantity and quality like administration of the questionnaire, clinical interviews, sleep diaries, etc., In our study, we have selected PSQI for assessing sleep deprivation. Numerous studies using the PSQI in a variety of older adult population internationally have supported high validity and reliability.[10]

The study findings could not be subjected to direct comparison with any earlier similar investigation because to best of our knowledge this is a first investigation conducted for exploring association of sleep deprivation with aggressive periodontitis. But essentially there exists biological plausibility for such findings. Sleep deprivation leads to the development of unfavorable hormonal profile and modulation of host immune and inflammatory mechanisms.

Deprivation of sleep has been documented to increase lymphocyte activation with overproductions of interleukin-1 (IL-1), IL-6, IL-17, and tumor necrosis factor alpha (TNF-α). An important start in the efforts to find the source of sleep loss-associated inflammation is a work showing that one night of sleep restricted to 4 h led to increased monocyte production of IL-6 and TNF-alpha messenger RNA.[11]

During experimental sleep deprivation of healthy volunteers, cellular adhesion molecules have been found to increase which are pro-coagulatory and pro-inflammatory markers produced by stimulated vascular endothelium (e.g. E-selectin and intercellular adhesion molecule-1).[12]

Sleep-wake cycles have also emerged as prominent regulators of the immune system. Central nervous system regulation of immune responses is primarily driven by two effector signaling pathways: Activation of the hypothalamic pituitary adrenal (HPA) axis and the sympathetic nervous system (SNS). Sleep loss activates sympathetic activity with less robust evidence of effects on the HPA axis. Whereas activation of HPA axis inhibits both antiviral and pro-inflammatory genes, SNS activation suppresses antiviral responses (Th1-type gene expression such as interferon-γ and IL-12B) while stimulating pro-inflammatory genes (Th2-type cytokine genes such as IL-4 and IL-5), which together provides a plausible mechanism to connect sleep disturbance with various infectious and inflammatory diseases. Thus, sleep deprivation decreases immunity and ensues systemic inflammation.[13]

As many of cytokines have a significant role in the pathogenesis of chronic periodontal disease, there might occur potentiation of periodontal destruction in a sleep deprived individual. Further short sleep duration has been shown not only to increase pathogen susceptibility; but also to decrease the immunologic protection offered by standard vaccines.[14-15]

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. Due to multifactorial etiology of both sleep deprivation and periodontal disease other unknown confounding factors might explain this association too.[16]

Thus, the present study can be taken as hypothesis generating investigation and with its preliminary results suggestive of the association of sleep deprivation with severity of periodontal disease, definitely calls on for future studies with larger samples, studying the effects after controlling of other confounding factors and including both subjective and objective measures for assessment of sleep.

CONCLUSION

Within limits of the present investigation, there appears to be an association between periodontal diseases and sleep deprivation. Better understanding the interplay between sleep gdeprivation and periodontitis with more focused investigations may help in designing effective lifestyle intervention strategies for this multifactorial disease.

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