VOLUME-8, ISSUE-11, NOVEMBER-2019 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Dental Science

IS VARIATION IN SLEEP AN INDUCING FACTOR FOR CAUSING ORAL DISEASES- A CROSS-SECTIONAL ANALYSIS

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ABSTRACT

Background- Since inflammation is characteristic of both periodontitis and inadequate sleep, periodontal disease may have a role in inadequate sleep. Sleep deprivation is becoming increasingly common in today's society. The purpose of this study is to determine if there is an association of periodontontal disease and sleep.

Methodology: In this study 50 non institutionalized participants were selected randomly and divided into 2 groups: Patients suffering from gingivitis (25) . Patients suffering from chronic periodontitis(25). Data from the selected patients was collected by the department of public health dentistry 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: 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 Deprivation, Periodontitis, Gingivitis

INTRODUCTION

Periodontal diseases are caused by pathogenic bacteria locally colonized in the dental biofilm creating infection and subsequent inflammatory response in the supporting structures of the teeth and tissues at the gingiva[1].

Inflammatory and immune responses are key to the periodontal disease process; many studies have shown relationships between periodontal disease and conditions like diabetes , cardiovascular disease and sleep[2] . Since inflammation is characteristic of both periodontitis and inadequate sleep, periodontal disease may have a role in inadequate sleep.[3]

The word sleep means relaxation, rest or just a light term, but it is a complex and essential biological process that is required on a daily basis for all humans regardless of age, sex or ethnic origin. In addition to maintaining normal brain functioning, sleep has important roles in controlling the functions of many other body systems vital to health and well-being [4].

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[5-6].

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[4]. 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 and department of public health dentistry of 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 50 participants who were non institutionalized and lived in sundernagar H.P were selected and divided into 2 groups

1. Patients suffering from gingivitis(25)
2. Patient suffering from chronic periodontitis(25)

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 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.**[7-8]

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.[9]

The key variable of interest was routine adequate sleep which was recorded using Pittsburgh Sleep Quality Index.[10]

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.[11-12] 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.[13]

Participants were also asked during an interview to report age, sex, 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

	Total (n)	Age	Sex	
			Male	Female
Gingivitis	25	44	8	17
Periodontitis	25	52	5	20

Table 1 shows mean age which is found to be 44, and 52 for two groups that is gingivitis , periodontitis.

Table 2: Mean parameters in 2 groups

	Mean ± Standard Deviation		
	GI	PPD	PSQI
Gingivitis	1.40±.41	2.110±.09	1.30±.79
Periodontitis	2.01±0.23	6.91 0±.72	5.04 ±.81

Table 2 Dipicts the mean values and standard deviation of gingival index, periodontal index, Pittsburgh Sleep Quality Index of both gingival and periodontal cases

Table 3: Multiple Logistic Regression Analysis intergroup comparison of PSQI

Dependant variable	Dental Diseases	Group	SD	p-Value	Significance
PSQI	Gingivitis	Periodontitis	0.41	<0.001	Significant
	Periodontitis	Gingivitis	0.35	<0.003	Significant

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

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.[13]

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-). 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.[14]

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).[15]

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.[16]

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.[17-18]

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.[19]

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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