ejpmr, 2019,6(4), 522-525

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

SJIF Impact Factor 4.897

Research Article ISSN 2394-3211 EJPMR

Dr. V. Saravanan and Dr. D. Baby Chitra*

Assistant Professor, Kanyakumari Government Medical College, Asaripallam.

*Corresponding Author: Dr. D. Baby Chitra

Assistant Professor, Kanyakumari Government Medical College, Asaripallam.

Article Received on 07/02/2019

Article Revised on 27/02/2019

Article Accepted on 20/03/2019

ABSTRACT

Introduction: Sleep disturbances and chronic sleep shortage have turn out to be very common in current society. The quality and quantity of sleep patterns are influenced by cultural social, psychological, environmental, and genetic factors. In recent years, multiple evidence from epidemiological and laboratory research works have shown that disturbed or decreased sleep is linked with glucose intolerance, insulin resistance reduced acute insulin response to glucose and an augmented risk of developing type 2 diabetes. Moreover, decreased or disturbed sleep is associated with cardiovascular disease, decreased quality of life, and economic burden. Because sleep modulates glucose metabolism and homeostasis, and influences quality of life, hence identifying sleep problems may be an important factor in management of type 2 diabetes. However, most studies of sleep and type 2 diabetes have focused mainly on obstructive sleep apnea and restless leg syndromes. There are limited studies on various sleep disturbances among those with type 2 diabetes using polysomnography. We investigated the frequency of undiagnosed sleep disturbances not addressed previously and evaluated the association between sleep disturbances and glucoregulation in group of type 2 diabetic patients using polysomnography and HbA1c. Aim and Objcetives: To investigate the sleep pattern in type2 diabetes mellitus patients and its correlation to their glycemic levels. Materials and Methods: It is a cross sectional study. Thirty patients with type 2 diabetes with age group between 40-60 vrs participated in the study and thirty age matched non diabetic normal subjects were included in control group. After obtaining informed consent persons were subjected to polysomnography and biochemical analysis. **Results:** The Total Sleep Time (mins) and Sleep efficiency(%) shows a significant decrease in the diabetic patients when compared to the controls. There is a significant decrease in duration of N2& N3 stages of non REM sleep(in mins) along with decrease in REM sleep duration in the diabetic group when compared to the control group. The diabetic group shows a significant increase in sleep latency when compared with the controls. Pearson's correlation revealed as the duration of diabetes increases there is a decrease in duration and quality of sleep evidenced by decrease in total sleep time and sleep efficiency and increase in sleep latency, also when there is an increase in HbA1c there is a decrease in duration of sleep and sleep efficiency and increase in sleep latency. Conclussion: From this study we can conclude Type 2 diabetes patients have problems in sleep quality. These changes identified in the sleep study could lead to poor glycemic control in type 2 diabetes patients. Type 2 diabetes patients with poor glycemic control should be assessed for sleep disorders and if present it should be corrected to achieve optimum control of blood sugar levels.

KEYWORDS: Sleep, Diabetes mellitus, Polysomnography, HbA1c.

INTRODUCTION

The prevalence of Type 2 diabetes mellitus (T2DM) has been on rise steadily over the past 3 decades, and is mainly attributable to the remarkable increase in obesity rate.^[1] Over 300 million people around the world live with diabetes now, and if the current prevalence rate continues, over 550 million people will be living with diabetes by 2030.^[2] Hence diabetes represents a major health problem because of its high prevalence, morbidity and mortality, its influence on patient quality of life, and its impact on the health system.^[3] The quality of sleep is related to the regulation of energy and glucose homeostasis.^[4] Type 2 diabetes mellitus is a serious chronic disease whereby the body inefficiently use glucose as a fuel due to relative insulin deficiency caused by insulin resistance.^[5] Impairments in the daily sleep/wake cycle due to sleep disturbances, including shift working, obstructive sleep apnea, and insomnia, are known to increase the risk of T2DM.^[6] One study done by Tang and colleagues reported inadequate sleep quality and quantity as a risk factor of developing T2DM and poor glycemic control among sufferers.^[7] Sleep duration has also been connected with higher risk of developing T2DM as well representing a strong predictor of glycosylated hemoglobin (HbA1c), with sleep loss being associated with increased HbA1c.^[8]

The strong association between obesity and sleep disorders and disturbances is in line with past research regarding the negative impact of excess weight⁹. Obesity, a disease associated with its own pattern of health consequences and symptomatology, has been independently associated with an increased risk of sleep disorders, disruptions and poor sleep quality. For example, excess weight is a strong predictor of daytime sleepiness and sleep-disorders.^[9]

Multiple studies describe a connection between T2DM, and sleep disorders.^[10] Studies suggest that a high proportion of T2DM sufferers also manage comorbid sleep apnea, particularly males and those overweight. A large-scale survey study found that sleep problems were by up to 40% of individuals with T2DM, with sleep apnea, and restless legs symptoms the most likely among sufferers. Individuals with T2DM who are obese also frequently report sleep problems, with research suggesting that all three may represent a complex interwoven triad of conditions.^[11] There are limited studies on various sleep disturbances among those with type 2 diabetes using polysomnography. We investigated the frequency of undiagnosed sleep disturbances not addressed previously and evaluated the association between sleep disturbances and glucoregulation in group of type 2 diabetic patients using polysomnography and HbA1c. Based on this aim of our study is to investigate the sleep pattern in type2 diabetes mellitus patients and its correlation to their HbA1c. Our study objectives were to assess the polysomnographic parameters in type 2 diabetes mellitus patients, sleep pattern relation with HbA1c level also to assess the sleep pattern relation with duration of Type 2 diabetes mellitus.

MATERIALS AND METHODOLOGY

The place of the study was the Institute of Physiology and Experimental Medicine, Madras Medical College. The study duration was May 2016-April 2017. Approval to conduct the study was obtained from the Institutional Ethics Committee (IEC), Madras Medical College, Chennai.

Subjects for the study group were recruited from the Institute of Diabetology, RGGGH, Chennai. Study population consists of 30 patients of both genders in the age group of 40-60yrs diagnosed with and undergoing treatment for Type 2 diabetes mellitus. Thirty subjects matched for age and gender with normal blood sugar levels and HbA1c levels were taken as controls. Patients diagnosed with and on treatment for Type 2 Diabetes of any duration, both men and women in the age group of 40-60 years were included in the study. Subjects with the following conditions were excluded from the study-Type 1 diabetes mellitus, Patients regularly taking sleep medications, Psychiatric illness, Obstructive sleep

disorder, Pregnancy and post-partum period, Patients secondary infections, Neoplastic, hepatic. with respiratory and any cardiovascular disorders, Other concurrent medical illness like renal failure, cardiac failure etc., and subjects taking medications that influence sleep pattern. According to the above inclusion and exclusion criteria subjects were recruited for the study after obtaining informed consent both in the verbal and written form. After obtaining informed consent, the participants of the study were subjected blood glucose fasting and postprandial with Glycated levels-Hemoglobin and polysomnography. Digital polysomnography was done for the consented persons using the MEDICAID SC32 in the human experiments laboratory of Institute of Physiology and Experimental medicine.

RESULTS

The data obtained from the above said methods were statistically analysed using SPSS software version 17 statistical significance of the data collected were analysed using UNPAIRED T TEST. In total 30 patients participated in the study. Out of which 16 are males and 14 are females. The mean age of the participants was 50.83 ± 5.38 with mean value of BMI 27.1 \pm 2.99. The mean value of FBS was 154.73 ± 16.15 and PPBS was 237.93 ± 31.69 . Coming to comparison of sleep stages The sleep stages N2, N3 and REM of study group shows a significant decrease when compared to control group and N1 sleep stage shows a significant increase when compared to control group. (*p-value <0.05).

Table. I: Comparison of Sleep Stages Between StudyGroup and Control Group.

	GROUPING	N	Mean	Std. Deviation	Std. Error Mean	t	DF	p Value
N1(mina)	STUDY GROUP	30	25.93	4.068	.743		41.038	0.000*
N1(mins)	CONTROL GROUP	30	18.30	1.896	.346	9.315		
N2(mins)	STUDY GROUP	30	141.80	11.043	2.016		58	0.000*
N2(mins)	CONTROL GROUP	30	152.67	9.404	1.717	-4.103		
N2 (mins)	STUDY GROUP	30	47.20	10.810	1.974	0.075	42.961	0.028*
N3(mins)	CONTROL GROUP	30	52.23	5.475	1.000	-2.275		
REM(mins)	STUDY GROUP	30	64.53	8.016	1.464	-2.529	47 084	0.015*
KEN(mins)	CONTROL GROUP	30	68.83	4.742	.866	-2.329	+7.084	0.015*

The N2 %, N3% and REM % of study group shows a significant decrease when compared to control group and N1% shows a significant increase when compared to control group (p<0.05).

	GROUPIN G	N	Mean	Std. Deviation	Std. Error Mean	Т	DF	p Value
N1%	STUD Y GROUP	30	9.383	2.0707	.3780	7.562	37.548	0.000*
	CONTROL GROUP	30	6.317	.8039	.1468			
N2%	STUD Y GROUP	30	50.873	1.7426	.3181	-3.949	43.096	0.000*
	CONTROL GROUP	30	52.283	.8875	.1620			
N3%	STUD Y GROUP	30	16.697	2.6558	.4849	-2.255	35.461	0.030*
183.76	CONTROL GROUP	30	17.850	.8920	.1629			
REM%	STUD Y GROUP	30	23.047	1.1362	.2074	-2.077	47.693	0.043*
KEN176	CONTROL GROUP	30	23.550	.6867	.1254			

 Table. 2: Comparison of percentages of various stages

 of sleep between study group and control group.

The Total sleep time (TST), sleep efficiency % of study group shows a significant decrease when compared to control group and sleep latency of study group shows significant increase when compared to control group.(*p-value < 0.05).

Table: III: Comparison of Total sleep time, Sleep efficiency % and sleep latency between study group and control group.

	GROUPIN G	N	Mean	Std. Deviation	Std. Error Mean	t	DF	p Value
Total sleep	STUD Y GROU P	30	279.70	26.233	4.789		51.647	0.039*
time(mins)	CONTROL GROUP	30	292.03	18.188	3.321 -2.116	-2.110		
sleep efficency	STUD Y GROU P	30	74.300	7.1155	1.2991	-2.255	45.326	0.029*
%	CONTROL GROUP	30	77.650	3.9504	.7212			
Sleep latency (mins)	STUD Y GROU P	30	33.00	9.337	1.705	3,350	39.066	0.001*
	CONTROL GROUP	30	26.80	3.951	.721	5.550		

Correlation table of HbA1c with total sleep time, sleep efficiency and sleep latency. Total sleep time(r=-0.923) and sleep efficiency(r=-0.974) shows negative correlation whereas sleep latency (r=0.936) shows positive correlation. The correlation was highly significant with P<0.05 for all three parameters.

TABLE: IV Correlation OF HbA1c and sleep pattern in study group							
		Total sleep time	Sleep efficiency	Sleep latency			
		(mins)	%	(mins)			
	Pearson Correlation	923**	974**	.936**			
	Sig. (2-tailed)	.000	.000	.000			
	N	30	30	30			
		Negative	Negative	Positive			
		correlation	correlation	correlation			

Correlation table of Diabetes duration with total sleep time, sleep efficiency and sleep latency. Total sleep time(r=-0.926) and sleep efficiency(r=-0.991) shows negative correlation whereas sleep latency (r=0.929) shows positive correlation. The correlation was highly significant with P<0.05 for all three parameters.

TABLE: V Correlation of Diabetes duration and sleep pattern in study group								
		Total sleep time (mins)	Sleep efficiency %	Sleep latency (mins)				
Diabetes duration (Years)	Pearson Correlation	926**	991**	.929**				
	Sig. (2- tailed)	.000	.000	.000				
	N	30	30	30				
		Negative correlation	Negative correlation	Positive correlation				

DISCUSSION

Maria Pallayova et al^[12] analysed polysomnographic recordings of 22 diabetics and 22 non diabetics and found that there was a significant decrease in Slow Wave Sleep (SWS) duration in diabetics compared to control group. The authors have concluded that changes in sleep architecture could be used to predict diabetes at an early stage. In this present study also the duration of SWS is decreased in the diabetic group than the normal group.

Dorit Koren et al^[13] have done a study on 62 obese adolescents by measuring the HbA1c, oral glucose tolerance test, serial insulin and glucose level along with a overnight polysomnography. The authors found a U shaped association between SWS with both HbA1c and glucose levels. There was also a positive association between duration of SWS and insulin secretary measures. Thus the authors concluded that both insufficient and excessive sleep resulted in hyperglycemia. Decreased N3 was associated with insufficient insulin secretion. The present study also replicates the same findings like decreased duration of N2 and N3 stages. Also there was a significant association of HbA1c with duration and quality of sleep.

Similarly Esra Tasali et al^[14] did a study on 9 healthy young non obese individuals to establish the association between sleep architecture and insulin measures. They experimentally suppressed the SWS by using auditory stimuli without disturbing the total duration of sleep. The results showed a marked decrease in insulin sensitivity without any compensatory increase in insulin secretion denoting decreased glucose tolerance that shows an increased risk of diabetes. Toshia ki Ohkuma et al^[15] examined the associations of sleep duration and HbA1c levels in 4870 type 2 diabetes patients. They classified the participants according to the duration of sleep vizless than 4.5 h, 4.5–5.4 h, 5.5–6.4 h, 6.5–7.4 h, 7.5–8.4 h, and more than 8.5 h which was self reported. There was a quadratic association of sleep duration with HbA1c with 6.5-7 hr. being the normal comaparator. Both longer and shorter duration of sleep were associated with higher

HbA1c. our study also shows that shorter the duration of sleep higher the HbA1c.

Singh et al^[16] had done a detailed polysomnographic study on 33 diabetic patients in the age group of 40-80 yrs. they found an association with presence of various degrees of OSA in these subjects reflecting a poor quality of sleep. The sleep study parameters showed a decrease in sleep efficiency to approximately 52%. The present study also shows decrease in sleep efficiency with a mean of 74.30 \pm 7.11(normal should be atleast 85%). This sleep efficiency is also negatively correlating with HbA1c levels.

Gislason T et al^[17] did an epidemiological study on 3201 swedish men to investigate effects of somatic diseases on the sleep quality and pattern. The authors studied effect of COPD, Rheumatic disease, obesity, hypertension and diabetes on sleep. All subgroups had sleep complaints. Among them the diabetic group had problems like Difficulty Initiating Sleep, Difficulty Maintaining sleep and Excessive Daytime Sleepiness. This is in favour of the results obtained from the present study.

CONCLUSION

From the above discussions the following conclusions could be derived from the present study. Type 2 diabetes patients have problems in sleep quality. The Total sleep time is decreased than normal controls denoting a reduction in sleep duration in diabetics. There is a reduction in slow wave sleep which denotes that the sleep is not restorative and refreshing in these subjects. There is an increase in sleep latency and decrease in sleep efficiency that denotes that diabetics have difficulty in both initiation and maintenance of sleep. These changes identified in the sleep study could lead to poor glycemic control in type 2 diabetes patients. Thus patients reporting with sleep difficulties should be screened for diabetes. Type 2 diabetes patients with poor glycemic control should be assessed for sleep disorders and if present it should be corrected to achieve optimum control of blood sugar levels.

Limitation of The Study: However, the study has got its own limitations as the above findings need to be confirmed with a larger sample size.

REFERENCES

- 1. Finucane M, Stevens G, Cowan M, Danaei G, Lin JK, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet, 2011; 377(9765): 557-567.
- McCrimmon R, Ryan C, Frier B Diabetes and cognitive dysfunction. Lancet, 2012; 379(9833): 2291-2299.
- DePablos-Velasco P, Salguero-Chaves E, Mata-Poyo J, DeRivas-Otero B, García-Sánchez R, et al. Quality of life and satisfaction with treatment in

subjects with type 2 diabetes: Results in Spain of the PANORAMA study. Endocrinol Nutr, 2014; 61(1): 18-26.

- 4. Qian J, Scheer FA Circadian system and glucose metabolism: implications for physiology and disease. Trends Endocrinol Metab, 2016; 27(5): 282-293.
- 5. Defronzo R Banting Lecture. From the triumvirate to the ominous octet: A new paradigm for the treatment of type 2 diabetes mellitus. Diabetes, 2009; 58(4): 773-795.
- 6. Shan Z, Ma H, Xie M, Yan P, Guo Y, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care, 2015; 38(3): 529-537.
- Tang Y, Meng L, Li D, Yang M, Zhu Y, et al. Interaction of sleep quality and sleep duration on glycemic control in patients with type 2 diabetes mellitus. Chin Med J (Engl), 2014; 127(20): 3543-3547.
- 8. Knutson KL, Ryden AM, Mander BA, Van Cauter E Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. Arch Intern Med., 2006; 166(16): 1768-1774.
- Slater G, Pengo MF, Kosky C, Steier J Obesity as an independent predictor of subjective excessive daytime sleepiness. Respir Med., 2013; 107(2): 305-309.
- 10. Schmid SM, Hallschmid M, Schultes B The metabolic burden of sleep loss. Lancet Diabetes Endocrinol, 2015; 3(1): 52-62.
- 11. Martins RC, Andersen ML, Tufik S The reciprocal interaction between sleep and type 2 diabetes mellitus: facts and perspectives. Braz J Med Biol Res., 2008; 41(3): 180-187.
- Greco D, Gambina F, Pisciotta M, Abrignani M, Maggio F. Clinical characteristics and associated comorbidities in diabetic patients with restless legs syndrome. ExpClinEndocrinol Diabetes 2009; 117: 496-499.
- Cuellar NG, Ratcliffe SJ. A comparison of glycemic control, sleep, fatigue, and depression in type 2 diabetes with and without restless legs syndrome. J Clin Sleep Med., 2008; 4: 50-56.
- 14. Balachandran JS, Patel SR. In the clinic. Obstructive sleep apnea. Ann Intern Med 2014; 161: ITC1-I15; quiz ITC16.
- Drager LF, Li J, Reinke C, Bevans-Fonti S, Jun JC, Polotsky VY. Intermittent hypoxia exacerbates metabolic effects of diet-induced obesity. Obesity (Silver Spring), 2011; 19: 2167-2174.
- 16. Foster GD, Sanders MH, Millman R, Zammit G, Borradaile KE, Newman AB, Wadden TA, Kelley D, Wing RR, Sunyer FX, Darcey V, Kuna ST. Obstructive sleep apnea among obese patients with type 2 diabetes. Diabetes Care, 2009; 32: 1017-1019.
- 17. Redeker NS. Sleep disturbance in people with heart failure: implications for selfcare. J CardiovascNurs, 2008; 23: 231-238.