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Original Research Article

Study of correlation between perceived sleep disturbances in depressed patients with objective changes in sleep architecture using polysomnography before and after antidepressant therapy

Ganesh Ingole[1], Harpreet S. Dhillon[2]*, Bhupendra Yadav[3]

[1]Department of Psychiatry, Regional Mental Hospital, Yerwada, Pune, Maharashtra, India
[2]Department of Psychiatry, 166 Military Hospital, Jammu, Jammu and Kashmir, India
[3]Department of Psychiatry, 5 Air Force Hospital, Jorhat, Assam, India

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***Correspondence:**
Dr. Harpreet S. Dhillon,
E-mail: harpreet5467@yahoo.com

ABSTRACT

Background: A prospective cohort study to correlate perceived sleep disturbances in depressed patients with objective changes in sleep architecture using polysomnography (PSG) before and after antidepressant therapy.
Methods: Patients were recruited into the study after applying strict inclusion and exclusion criterion to rule out other comorbidities which could influence sleep. A diagnosis of Depressive episode was made based on ICD-10 DCR. Psychometry, in the form of Beck Depressive inventory (BDI) and HAMD (Hamilton depression rating scale) insomnia subscale was applied on Day 1 of admission. Patients were subjected to sleep study on Day 03 of admission with Polysomnography. Patients were started on antidepressant treatment post Polysomnography. An adequate trial of antidepressants for 08 weeks was administered and BDI score ≤09 was taken as remission. Polysomnography was repeated post remission. Statistical analysis was performed using Kruskal Wallis test and Pearson correlation coefficient.
Results: The results showed positive (improvement) polysomnographic findings in terms of total sleep time, sleep efficiency, wake after sleep onset, percentage wake time and these findings were statistically significant. HAM-D Insomnia subscale was found to correlate with total sleep time, sleep efficiency, wake after sleep onset, total wake time and N2 Stage percentage.
Conclusions: Antidepressant treatment effectively improves sleep architecture in Depressive disorder and HAM-D Insomnia subscale correlates with objective findings of total sleep time, sleep efficiency, wake after sleep onset, total wake time and duration of N2 stage of NREM.

Keywords: Depression, Perceived sleep quality, Polysomnography, Sleep architecture

INTRODUCTION

Sleep is a complex behaviour which continues to be an evolutionary enigma. Every animal engages in some form of sleep or sleep-like behavior.[1] Carskadon and Dement defined sleep as: Sleep is a recurring, reversible neuro-behavioral state of relative perceptual disengagement from and unresponsiveness to the environment. Sleep is typically accompanied (in humans) by postural recumbence, behavioural quiescence, and closed eyes.[2] Sleep repairs the physical body to improve and maintain general health, consolidate learning and memory, and recharge the psychological batteries to maintain emotional balance and well-being. Lack of sleep leads to

various mental and physical illnesses including an increased risk of hypertension, diabetes, obesity, heart attack, stroke and depression.[3] Sleep disturbances are widespread in psychiatric disorders and hence have been integrated in the official diagnostic criteria for many Psychiatric disorders, such as major Depression, Generalized Anxiety Disorder, Post-Traumatic Stress Disorder and Substance Related Disorder.[4] Depression and sleep disturbances have a very complex interaction with sleep disturbances presenting as the most commonly observed physical complaint in depressed patients with roughly 80% complaining of insomnia and the remaining 20% of hypersomnia.[5]

Furthermore, the prevalence of depressive symptoms has been shown to be high in patients with insomnia and breathing related sleep disorders.[6,7] A study in the general population revealed that 40% of subjects with insomnia presented with a mental illness within 6 months versus 16% of subjects without insomnia.[8] Taken collectively, these results suggests an increased incidence of major depression in those with sleep disturbances. Therefore, diagnosis and treatment of sleep disorders is very important for clinicians. Diagnosis of insomnia is based on the patient's perception of sleep quality while diagnosis of many other sleep related disorders eg. Restless legs syndrome (RLS), periodic limb movements (PLMs), REM sleep behavior disorder (RBD), obstructive sleep apnea (OSA), and excessive daytime sleepiness (EDS) are defined by polysomnographic (PSG) data.[9,10] However, these laboratory tests are highly demanding in terms of cost, human resources, and other logistics required; therefore, questionnaires and scales have been developed for screening patients. This study was planned with the objective of testing the utility of a quick, cost-effective and practical psychometry tool (HAM-D insomnia subscale) as an alternative to expensive and time consuming tests such as PSG to assess and monitor the sleep disturbances in depressed patients especially in OPD settings in a resourse constraint country.

METHODS

Inclusion criteria

- Patients admitted in psychiatry ward within the age range of 18 to 50 years.
- Meeting the ICD-10 Diagnostic criteria for research of depressive episode.

Exclusion criterion

- Those not consenting for the study.
- Patients having history of sleep disorder prior to onset of depression.
- Other psychiatric co-morbidities.
- Actively consuming alcohol, other psychoactive substances and psychotropic medications.
- Other co-morbid active medical and surgical illness.

The study was carried out in a tertiary care hospital over a period of 01 year and a total of 77 male patients were recruited, out of which, 09 met the exclusion criterion, 11 were not willing to participate. 57 patients were included in the study out of which 08 were lost to follow up and 06 did not improve with treatment. Remaining 43 patients completed the study. The subjects were evaluated by structured as well as unstructured clinical interview for diagnosis of depressive disorder as per ICD -10 Diagnostic Criteria Research.[11] On day 1 of admission, Psychiatric rating scales - The Beck's Depression Inventory-II (1996) (BDI) and The Hamilton Rating Scale for Depression (HAM-D) were applied.[12-15] The Hamilton Rating Scale for Depression (HAM-D) is one of the longest standing, most widely used measures of depression severity in research and clinical practice with good sensitivity and specificity. It is a clinician administered 21 items scale and takes approx. 10 minutes to complete the test. Items 4, 5, and 6 refer specifically to sleep, inquiring about insomnia prior to sleep onset, disturbed sleep in the middle of the night, and trouble falling back asleep in the early morning, respectively.[16] Other items may be peripherally involved with sleep difficulties as they refer to fatigue, retardation, and somatic symptoms in general. Patients were kept drug free till 3rd day of admission. Polysomnography was conducted on day 3 after allowing patient to get accustomed to ward environment. All patients who completed study were followed up till 8 weeks and post treatment. Polysomnography was conducted after achieving full remission as indicated by BDI score of 9 or less and HAMD insomnia subscale score of 0.

The sleep parametres studied were as follows[17]

- Total sleep time (TST)- The total time spent asleep during the sleep episode. This is equal to the time in bed less the awake time.
- Sleep efficiency (SE)- The ratio of total sleep time to time in bed expressed as a percentage of time spent asleep during the recording period. Normal values are typically above 90% in young and above 85% in elderly patients.
- Sleep Latency - Time from start of the recording ("lights out") to the onset of sleep. Normal values are typically below 30 min in young and below 45 min in elderly patients.
- Wake After Sleep Onset (WASO)- The total time scored as awake occurring after the sleep onset. Typically WASO should not exceed 30 min.
- N3 Latency- Total duration in minutes and as percentage relative to total sleep time of sleep stage N3. The amount of stage N3 decreases with older age, normal values are around 10% for elderly and 20-25% for young subjects.
- REM% - Total duration in minutes and as percentage relative to total sleep time of sleep stage REM. Normal values are 20-25%.
- REM Latency- The number of minutes from the onset of sleep to the onset of the first REM sleep

period. Reduced values are typically below 65 min in young and 50 min. in elderly patients.

Statistical analysis

Data analysis was done with the help of SPSS Software version 21. Considering 90% prevalence of sleep disturbances in Depression and 10% variation, sample size was calculated to be 43.[18,19] Application of Shapiro Wilk test showed that data was not normally distributed. Hence, paired comparison between before and after treatment for BDI score, HAM-D Insomnia subscale and Sleep architecture parameters was done with the help of Kruskal Wallis test. Correlation of HAM-D Insomnia Subscale with Polysomnography was done with Pearson Correlation Coefficient. p value less than 0.05 was taken as significant level.

RESULTS

The results of the study are discussed with the help of tables below. The range of the age group was 22 - 46 years with mean age of 31.28 years and standard deviation of 5.56.

As per ICD-10 DCR, 16.3% patients were of mild depression, 62.8% moderate and remaining i.e. 20.09% were of severe depression. Severity classification as per BDI score revealed, 76.74% moderate and remaining 23.26% falling in severe depression category. Median value for BDI score before treatment was 24 and after treatment was 5. This was statistically significant with a p value <0.001. Median value for HAM-D insomnia subscale score before treatment was 3 and after treatment was 0 (Table 1). This also was statistically significant with a p value <0.001.

Table 2 illustrates the median value for Total Sleep time before treatment was 278 minutes and after treatment was 320 minutes. This was statistically significant with a p value <0.001. Median value for Sleep Efficiency before treatment was 67.9% and after treatment was 76.5%. This was statistically significant with a p value <0.001. Median value for Sleep Latency before treatment was 26.5 minutes and after treatment was 20.0 minutes. This was statistically not significant as p value was 0.322. Median value for Wake After Sleep Onset before treatment was 114.25 minutes and after treatment was 89 minutes. This was statistically significant as p value <0.001. Median value for Total Wake Time before treatment was 130 minutes and after treatment was 100.50 minutes. This was statistically significant as p value <0.001. Median value for Percentage Wake Time before treatment was 32.1% and after treatment was 27.3%. This was statistically significant as p value <0.001.

Table 1: BDI and HAM-D Insomnia subscale Score.

	Before treatment			After treatment			Z	p value
	Mean	Median	SD	Mean	Median	SD		
BDI	26.08	24.00	7.33	4.98	5.00	2.44	-5.716	<0.001
HAM-D Insomnia Subscale	3.40	3.00	1.12	0.00	0.00	0.00	-5.768	<0.001

Table 2: Sleep Architecture changes before and after treatment.

	Before treatment		After treatment			
	Median	SD	Median	SD	Z	p value
Total Sleep Time	278.00	93.93	320.00	43.12	-4.372	<0.001
Sleep Efficiency	67.90	20.34	76.50	9.18	-4.735	<0.001
Sleep Latency	26.50	25.34	20.00	14.13	-0.991	0.322
Wake After Sleep Onset	114.25	93.18	89.00	48.28	-4.215	<0.001
Total Wake Time	130.00	94.08	100.50	46.26	-4.553	<0.001
% Wake Time	32.10	20.52	27.27	9.60	-4.662	<0.001
N1 %	6.40	14.91	6.00	6.05	-1.740	0.082
N2 %	31.90	16.48	28.50	10.59	-0.870	0.385
N3 %	39.00	19.97	42.50	12.96	-0.906	0.365
REM %	15.20	11.57	23.00	14.74	-2.731	0.006
N1 Latency	1.00	28.10	1.00	1.52	-2.633	0.008
N2 Latency	2.00	32.51	3.00	4.19	-1.006	0.314
N3 Latency	9.50	45.41	16.50	10.43	-0.036	0.971
REM Latency	82.00	86.13	120.00	50.67	-0.072	0.942

Table 2 also describes median value for Stage N1 sleep percentage before treatment was 6.4% and after treatment was 6% (p value 0.082). Median value for Stage N2% before treatment was 31.9% and after treatment was 28.5% (p value 0.385). Median value for Stage N3% before treatment was 39% and after treatment was 42.5% (p value 0.365). Median value for REM % before treatment was 15.2% and after treatment was 23%. This was statistically significant as p value was 0.006. Median value for N1 latency before treatment was 1 minute and after treatment was 1 minute. This was statistically significant as p value 0.008. Median value for N2 latency before treatment was 2 minute and after treatment was 3 minute (p value 0.314). Median value for N3 latency before treatment was 9.5 minute and after treatment was 16.5 minute (p value was 0.971). Median value for REM latency before treatment was 82 minute and after treatment was 120 minute (p value was 0.942) (Table 2).

Table 3 illustrates the correlation of HAM-D Insomnia Subscale with Polysomnography. HAM-D Insomnia subscale score showed a strong correlation with Total sleep time. A moderately strong correlation of perceived sleep difficulty was seen with sleep efficiency, wake after sleep onset, total wake time and N2%. A weak correlation was seen with Percentage Wake Time, N3% and N1 Latency.

Table 3: Correlation of HAM-D Insomnia Subscale with Polysomnography.

Before treatment	HAMD-Insomnia subscale	
	Pearson correlation	p value
HAM-D Insomnia Subscale	1	
Total sleep time	-0.700	0.000
Sleep efficiency	-0.699	0.000
Sleep latency	-0.008	0.959
Wake after sleep onset	0.695	0.000
Total wake time	0.673	0.000
% Wake time	0.468	0.002
N1%	-0.124	0.428
N2%	0.568	0.000
N3%	-0.401	0.008
REM%	0.010	0.950
N1 latency	0.494	0.001
N2 latency	0.115	0.465
N3 latency	0.115	0.465
REM latency	0.145	0.355

DISCUSSION

Patients with depression show abnormalities of almost all sleep parameters. Disrupted sleep continuity manifests as prolongation of sleep latency, increased number and duration of awakenings from sleep expressed as increased wake after sleep onset (WASO) time, decreased sleep efficiency and early morning awakenings. The distribution of deep sleep recorded in PSG as sleep stage N3, also called delta or slow wave sleep (SWS), is also altered in depressed patients.[17] REM sleep disturbances (REM sleep latency, increased REM sleep time and increased REM sleep density) are considered a biological marker of circadian rhythm disturbances in depression with melancholic features.[20] Antidepressant therapy in the long-term shows improvement in sleep parameters secondary to improvement of mood and daytime activity.[17]

The antidepressant medicines prescribed during our study were Escitalopram, Fluoxetine, Paroxetine, Sertraline and Mirtazapine (88.4% of the patients were prescribed SSRIs and 11.6% Mirtazapine). This prescription pattern was consistent with a recent multicentric study in India in which, 194 (62.2%) out of 312 patients were prescribed selective serotonin reuptake inhibitors (SSRIs).[21]

In table 1, BDI score before treatment had a median score of 24, which reduced to a median score of 5 post treatment (p<0.0001). Improvement in BDI to less than 9 was also a criterion of study, which indicated remission. Similarly, HAMD Insomnia subscale score before treatment had maximum value of 6 and minimum 2 with median 3, mean of 3.40, and SD 1.12. (Table 1). After treatment, all the values were 0 as it was one of the criteria for assessing sleep improvement after treatment (Table 1). This also was statistically significant with a p value <0.001. This statistically significant improvement in HAMD Insomnia subscale after treatment with antidepressants was consistent with multiple other studies on effect of antidepressants on sleep.[22-24]

Table 2 enumerates the sleep architecture before and after treatment. The median value of Total sleep time (TST) before treatment was 278 minutes, which increased to a median value of 320 minutes (p value of <0.0001). Pillai et al and Baglioni et al found similar findings with increase in total sleep time after treatment with antidepressants in two different meta-analyses.[4,25]

The median value of Sleep efficiency before treatment was 67.9 %, which after treatment improved to a median value of 76.5% (p<0.0001), thus signifying that treatment of depression had significantly improved sleep efficiency (Table 2). Wichniak and Wierzbicka found similar results with increase in sleep continuity (sleep efficiency and total sleep time) after treatment with antidepressants.[17]

Sleep Latency had improved after the treatment (Table 2), however the results were not statistically significant (p=0.322). Statistically significant improvement post treatment was found in Wake After Sleep Onset (p value <0.001), Total Wake Time (p value <0.001) and Percentage Wake Time (p value <0.001). Our study also observed reduction in N1 and N2 stages of sleep and increase in N3 stage of sleep, although these findings

were statistically not significant (Table 2). These findings were consistent with multiple other studies, which have shown that all antidepressants improve sleep parameters over long term despite the fact that some of them may impair sleep initially due to the activating effects.[17,26]

Effective treatment with antidepressants increases REM latency and suppresses REM sleep, however in our study, REM% before treatment had median value of 15.2% (Table 2) and after treatment had median value of 23%, (p value= 0.006) which could be attributed to activating effects of predominantly used SSRI's (88.4% of the patients were prescribed SSRIs and 11.6% Mirtazapine) over a short duration of time.[26] Median value for REM latency before treatment was 82 minute and after treatment was 120 minute. This finding of increased REM Latency was similar to most other sleep studies, however results in this study were not statistically significant (p =0.942).[17,25,26]

In table 3, objective findings of sleep disturbances are correlated with perceived sleep disturbances assessed by HAMD Insomnia subscale using Pearson correlation coefficient. A strong corelation was noted between perceived sleep disturbances and Total sleep time (Table 3). A moderately strong correlation between the two was seen with Sleep efficiency, Wake after sleep onset, Total wake time and N2%. A weak correlation was noted between perceived sleep disturbances and Percentage Wake Time, N3% and N1 Latency (Table 3). In a study by Vitiello and Larsen consisting of large group of healthy men (150) and women (95), to understand the relationship between self-reported subjective and objectively measured sleep quality, results showed a considerable correspondence between subjective and objective sleep quality for men but not for women, despite women more frequently endorsing the presence of significant sleep disturbance.[27] This finding coincides with our study, which could be attributed to all male patients in our study group. However, Some studies have reported significant differences between subjective and objective measures. A pilot study conducted by Farahani DM et al did not find any significant correlation between perceived sleep disturbances and PSG findings in depression patients.[28] In another study by Hebert and Fullum, it was found that self-reported sleep disturbances in a group of chronic tinnitus patients, complaints did not correlate with objective polysomnographic assessments of their sleep.[29] Majer et al, in a cohort of patients with chronic fatigue syndrome, reported sleep problems significantly more often than control subjects.[30] Yet, when measured objectively, these parameters and sleep architecture did not differ between the two groups.

In view of the dissenting findings, present evidence in this area is somewhat inconsistent. Moreover, such resemblances/differences can be largely disparate in various populations; therefore, the research results obtained with specific populations cannot be completely generalized. Hence, in an idealistic scenario, a concurrent use of objective and subjective sleep measures could provide more valid and reliable information of the patient's sleep health.

CONCLUSION

Antidepressant treatment effectively improves sleep architecture in Depressive disorder and HAM-D Insomnia subscale correlates with objective findings of Total sleep time, Sleep Efficiency, Wake After Sleep Onset, Total Wake Time and duration of N2 stage of NREM. Hence, HAM-D insomnia subscale being a simple and inexpensive tool can be a used as a screening tool, especially in the OPD setting.

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