Assessment of self- reported sleep duration and quality in relation to the cognitive status of older adults: A case- controlled study.

1.INTRODUCTION

1.1 BACKGROUND

Sleep is essential for the amalgamation of procedural memory. Sleep related consolidation of memory involves an interaction of different memory systems. (1,2) Sleep can affect many sub-domains of cognition, such as complex attention, memory, reasoning, language and executive functioning. Sleep disturbance is common in older adults because of age related increase in prevalence of multimorbidity, polypharmacy, psychosocial factors, and certain primary sleep disorders. (3, 4) Almost 40-70% of older adults are estimated to have chronic sleep related problems of whom nearly 50% remain undiagnosed. (5) While much is known regarding age related changes in cognitive functioning very little is known on the impact of sleep on late-life cognitive function.

Advanced age has been found related to normative declines in sleep and cognitive function. Enough evidence in literature points to an inherent connection between sleep and cognition. Data from observational studies support the part of sleep disturbances (mainly sleep duration, sleep fragmentation, and sleep-disordered breathing) in the development of cognitive decline. (2, 6) Sleep difficulties combined with age-related cognitive decline, can contribute to poorer performance in daily activities necessary for independence, quality of life, and successful ageing. Longer day time sleep is associated with a steeper decline in visuospatial reasoning and processing speed. (7) However, very few studies have correlated the association of duration and sleep patterns with different cognition domains in older adults.

The proportion of older people has increased exponentially in recent decades. This proportion is estimated to be more than 20% of population by 2050. (8) Therefore, the number of older adults suffering from cognitive decline and dementia has rapidly increased over the last few decades. (9)

Dementia is a common late life serious disorder. It is a very important contributor to a large proportion of disability and mortality in older people. Long term care of these people has become burdensome for particularly the families and the society at large. (10)

Till date there is no effective treatment available to treat dementia ; thus, developing dementia prevention strategies is a priority. (11,12) Decline in cognition during the long preclinical phase of dementia, is regarded as a cardinal marker. Thus, identifying risk factors for cognitive decline, including poor duration and quality of sleep is important for dementia prevention.

1.2. OBJECTIVE

This study primarily compared the duration and quality of sleep- in relation to cognitive function in various domains in patients of ≥ 65 years of age. The secondary Objective was to compare the Pittsburgh sleep quality index outcome in patients diagnosed with Mild cognitive impairment (MCI) with healthy older adults. To identify specific blood biomarkers (Lipid profile, Vitamin D, Vitamin B 12, Folic acid) in cognitively impaired older adults that may be linked to poor quality or duration of sleep.

2.MATERIALS AND METHODS

2.1. SUBJECT CHARACTERISTICS

This case control study was conducted at a tertiary care hospital in New Delhi, India, in the outpatient department and memory clinic of Geriatric Medicine. Enrolment for the study occurred between October 2021 and June 2023. Institute Ethics Committee, AIIMS, N. Delhi approved the study and we

followed ethical guidelines. Cases aged ≥ 65 years satisfying inclusion criteria with least 1 primary caregiver, presenting in the AIIMS outpatient department or memory clinic were enrolled in the study with informed consent.

Patients suffering from MNCD (Major Neurocognitive Disorder) (CDR> 0.5) at the time of evaluation, Patients with a GDS > 8/15, GAD-7 score > 4, critically ill patients and people with head injury, major neuropsychiatric illness or physical illness causing cognitive decline were excluded from the study. Individuals with a CDR (clinical dementia rating score) of 0.5 (suggestive of mild cognitive impairment) were selected as cases and CDR 0 selected as controls. Matching was done for major comorbidities (T2DM, HTN, CAD, COPD), following which 78 participants were selected from each group.

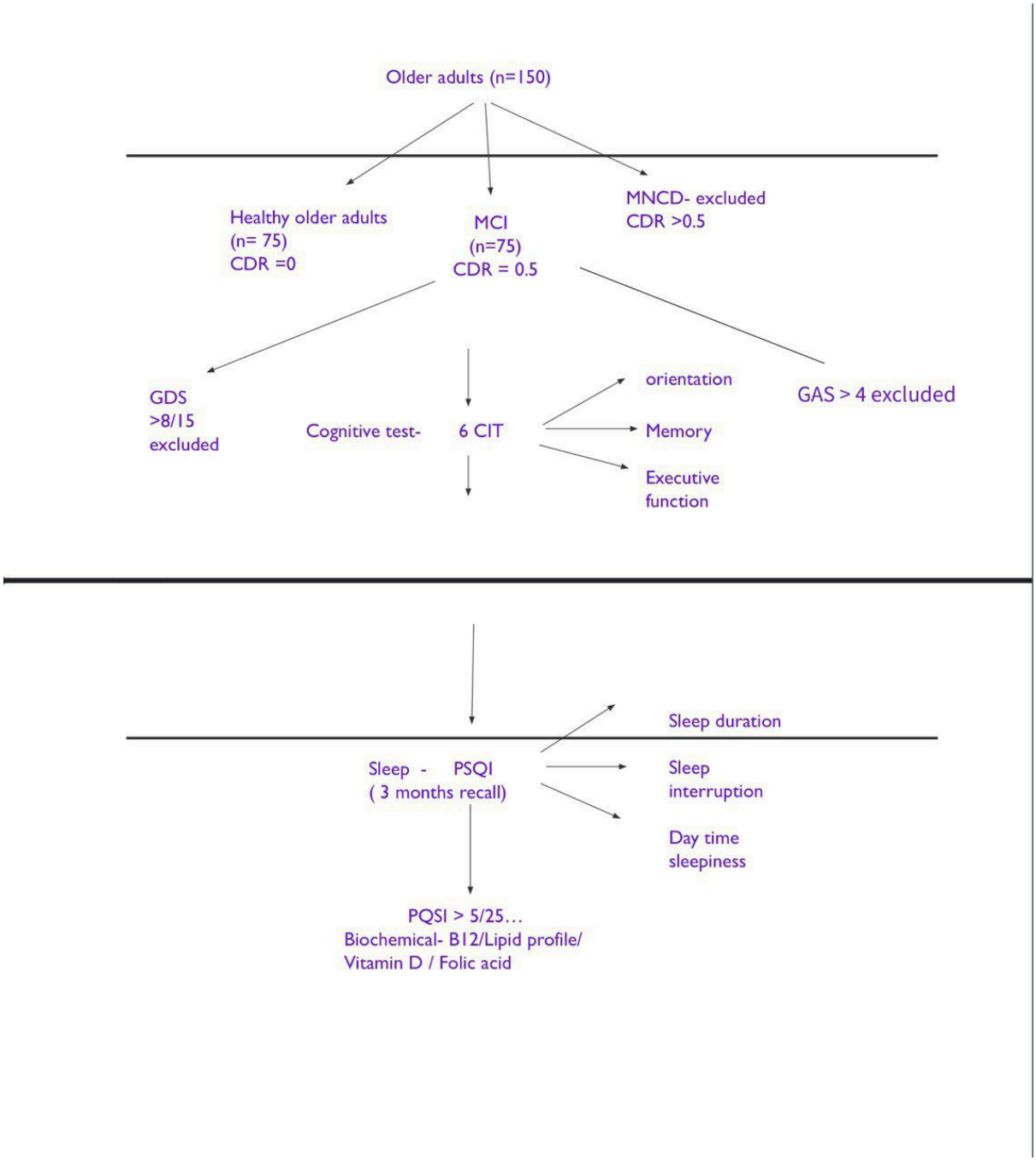
2.2. SAMPLE SIZE

Statistical data was collected based on article: Depressive Symptoms are the Main Predictor for Subjective Sleep Quality in Patients with Mild Cognitive Impairment—A Controlled Study. Based on this study a sample size of 78 patients with cognitive impairment and 78 patients who are cognitively healthy were selected for the study.

2.3. ASESSMENT

The assessment included demographic data, socioeconomic status, and evaluation of comorbidities. Patients were screened with Geriatric depression scale 15 (GDS -15), Generalised anxiety disorder scale-7(GAD -7) and 6 cognitive impairment test (6 CIT). Patients with a GDS > 8/15 or GAD-7 > 4, or 6 CIT >10 (CDR >0.5) were excluded from the study. Sleep duration, Interruption, Day time sleep per day were recorded from recall memory for the last 3 months from the date of the interview was documented. Any

change in sleep duration / quality and cognitive status was recorded.



2.4. BIOCHEMICAL ANALYSIS

Biomarkers for cognition in relation to decline in sleep ,were tested in group 2 (cases) (Fasting Lipid profile, serum Vitamin B12, serum Folic and serum 25(OH) Vitamin D.

2.5. STATISTICAL ANALYSIS

Consecutive cases satisfying the inclusion criteria were recruited until each group's sample size was achieved. Data was recorded in computer database (excel sheet) using statistical software SPSS 24.0 or STATA 14 descriptive analysis of the data was done. Qualitative data was stated in percentage and in numbers while, Quantitative data was expressed as Mean ± SD if normality exist, otherwise data was expressed as Median with minimum and maximum. After testing for normal distribution; quantitative data between the groups was done by unpaired t- test. If data failed normality; Mann Whitney test was used. Statistical significance was set at a p-value < .05.

3. RESULTS

156 consecutive patients were recruited from geriatric medicine OPD, with informed consent to participate. A baseline cognitive assessment and assessment of global PQSI and its individual components were done.

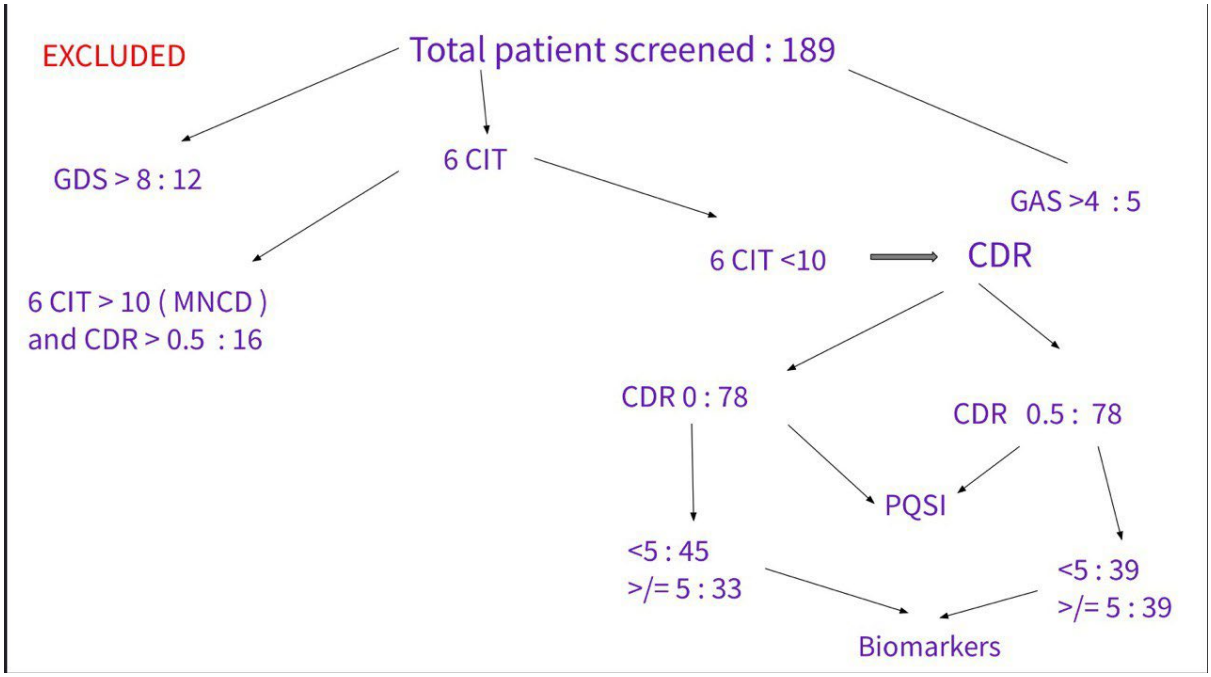

EXCLUDED
Total patient screened : 189
GDS > 8 : 12
6 CIT
GAS >4 : 5
6 CIT > 10 (MNCD)
and CDR > 0.5 : 16
6 CIT <10
CDR
CDR 0 : 78
CDR 0.5 : 78
PQSI
<5 : 45
>/= 5 : 33
<5 : 39
>/= 5 : 39
Biomarkers

Table 1 Baseline characteristics of the study population (n=156)

Variables	**CDR 0 (n=78) (control)**	**CDR 0.5 (n=78) (case)**	**p-value**
Age (IQ1, IQ3)	70.00 (66,73)	71.00 (67,77)	0.084
Sex			
Male	55 (49.11)	57 (50.89)	0.722
Female	23 (52.27)	21 (47.73)	
Education			
0 (no formal education)	2 (2.56)	0 (0.00)	**0.034**
1(pre-primary)	7 (8.97)	10 (12.82)	
2(primary)	14 (17.95)	5 (6.41)	
3(upper primary)	31 (39.74)	25 (32.05)	
4(secondary)	10 (12.82)	14 (17.95)	
5(senior secondary)	1 (1.28)	8 (10.26)	
6(undergraduate)	10 (12.82)	11 (14.10)	
7(postgraduate)	2 (2.56)	5 (6.41)	
8(diploma)	1 (1.28)	0 (0.00	
Comorbidities			
DM	33 (42.31)	30 (38.46)	0.624
HTN	17 (21.79)	19 (24.36)	0.704
CAD	11 (14.10)	6 (7.69)	0.199
COPD	5 (6.41)	4 (5.13)	0.731
Scales			
GDS 15	0 (0-3)	3 (1-6)	**<0.001**
CIT 6	0 (0-2)	7 (2-9)	**<0.001**
GAD-7 (n=82)	0 (0-2)	2 (0-3)	0.056
Blood markers			
LDL	100.5 (71-120)	89 (66-109)	0.145
Triglycerides	111 (91-152)	122.5 (92-156)	0.548
Total Cholesterol	133 (120-174)	145.5 (125-170)	0.575
HDL	45 (34-50)	45 (35-51)	0.662
Vitamin D	29.1 (18.4-37.3)	26.3 (18-34)	0.159
Vitamin B12	128 (81.8-225)	128 (109-259)	0.483
Folic acid	8.3 (5.9-14)	8.22 (5.5-13)	0.990

3.1. DEMOGRAPAHIC DETAILS OF THE STUDY PARTICIPANTS

156 participants were included in the study, of which 78 older adults had cognitive impairment- CDR 0.5 (cases) and 78 participants had normal cognition- CDR 0 (controls). Amongst the cases 57 were male (73%) and 21 were female (26%) whereas in the control group 55 were male (70%) and 23 were female (29%). The mean age for the cases was 71 years (66,73) and 70 years (67,77) for the controls. (Table: 1)

Demographic details of the case and control group were compared, there was a significant difference in the number of participants with no formal education with patients in control group having a larger group with no formal education (p=0.034). (TABLE 1)

The most common comorbidity in cases was type 2 Diabetes mellitus(T2DM) (38%), followed by hypertension (HTN) (24%), coronary artery disease (CAD) (6%), chronic obstructive pulmonary disease (COPD) (4%). The most common comorbidity in the control group was T2DM (42%), followed by HTN (17%), CAD (11%), COPD (5%), There was no significant difference in comorbidities between the 2 groups. (TABLE 1)

3.1.1. GERIATRIC DEPRESSION SCALE- 15 ITEM (GDS-15)

There was a significant difference in the GDS 15 score, between the participants with impaired cognition compared to those with normal cognition. ($p<0.001$). Participants of the control group had average GDS 15 score (0-3) while cases had a higher average score (1-6).

3.1.2. 6 ITEM COGNITIVE IMPAIRMENT TEST (6 CIT)

There was a significant difference in the total 6 CIT score between participants with impaired cognition compared to those with normal cognition. ($p<0.001$). Participants of the control group had average 6 CIT score (0-2) while cases had a higher average score (2-9).

3.1.3. GENRALISED ANXIETY DISORDER SCALE -7 ITEM (GAD 7)

There was no difference in terms of the GAD-7 score between the group with impaired cognition compared to the group with intact cognition.

3.1.4. BIOCHEMICAL ANALYSIS

The serum levels of low - density lipoprotein (LDL) ($p=0.145$), total cholesterol (TC) ($p=0.575$), triglyceride (TG) ($p=548$), high density lipoprotein (HDL) ($p=0.662$) was compared in the 2 groups, there was no significant difference in the group with impaired cognition compared to the group with normal cognition. The serum levels of vitamin D, vitamin B12 and Folic acid were compared and no significant difference was found between the 2 groups ($p=0.159$, $p=0.483$, $p=0.990$) respectively.

Table 2. Association of Sleep parameters with cognitive impairment

Variables	CDR 0 (n=78)	CDR 0.5 (n=78)	p-value
PQSI	2 (0-6)	6 (3-8)	**<0.001**
Total sleep duration	6 (6-7)	6 (5-7)	**0.018**
Sleep latency	10 (10-20)	12.5 (10-20)	**0.045**
TDS score			
0	28 (35.90)	21 (26.92)	**0.013**
1	33 (42.31)	21 (26.92)	
2	11 (14.10)	20 (25.64)	
3	6 (7.69)	16 (20.51)	
Abnormal sleep			
0	26 (33.33)	18 (23.08)	0.155
1	52 (66.67)	60 (76.92)	
Daytime sleepiness			
0	74 (94.87)	47 (60.26)	**<0.001**
1	4 (5.13)	31 (39.74)	
Sleep Latency score			
0	49 (62.82)	39 (50.00)	0.263
1	18 (23.08)	20 (25.64)	
2	7 (8.97)	9 (11.54)	
3	4 (5.13)	10 (12.82)	
Sleep disturbance			
0	47 (60.26)	21 (26.92)	**<0.001**
1	11 (14.10)	22 (28.21)	
2	13 (16.67)	19 (24.36)	
3	7 (8.97)	16 (20.51)	
Self-reported quality			
0	47 (60.26)	33 (42.31)	0.171
1	13 (16.67)	18 (23.08)	
2	14 (17.95)	20 (25.64)	
3	4 (5.13)	7 (8.97)	
Use of sleep medication			
0	74 (94.87)	70 (89.74)	0.250
1	4 (5.13)	4 (5.13)	
2	0 (0.00)	3 (3.85)	
3	0 (0.00)	1 (1.28)	

3.2. ASSOCIATION OF SLEEP PARAMETERS WITH COGNITION

The Pittsburgh sleep quality index score was compared between the groups with intact cognition and impaired cognition. There was a significant difference in terms of the global Pittsburgh sleep quality index, with P value <0.001, indicating a higher incidence of impaired sleep duration and quality in the group with impaired cognition than in the group with intact cognition. There was a t difference in terms of total sleep duration, sleep latency, increased daytime sleepiness and sleep disturbances, with reduced total duration, increased latency, increased disturbances and increased daytime sleepiness in the group with impaired cognition. (p value: 0.018,0.045, <0.001, <0.001 respectively) (TABLE 2)

Table 3. Association between domains of cognition and sleep quality, duration and latency

Variables	PQSI	Total duration of sleep	Sleep latency
Memory			
CDR 0	2 (0-6)	6.15±1.32	10 (10-20)
CDR 0.5	6 (3-8)	5.88±1.63	17.5 (10-20
p-value	**<0.001**	0.238	0.023
Orientation			
CDR 0	4 (0-8)	6.03±1.43	10 (10-20)
CDR 0.5	11 (9-14)	5.80±2.68	20 (15-30)
p-value	**0.028**	0.729	0.114
Judgement and problem solving			
CDR 0	4 (0-8)	6.02±1.39	10 (10-20)
CDR 0.5	6 (3-8)	6.03±1.80	10 (10-20)
p-value	**0.048**	0.972	0.839
Community affairs			
CDR 0	4 (0-8)	6.03±1.39	10 (10-20)
CDR 0.5	7 (3-9)	6.00±2.14	20 (10-20)
p-value	0.113	0.944	0.198
Home and hobbies			
CDR 0	4 (0-8)	6.04±1.40	10 (10-20)
CDR 0.5	6 (3-10)	5.95±1.91	15 (10-30)
p-value	**0.017**	0.808	0.148

3.3. ASSOCIATION OF SLEEP PARAMETERS WITH VARIOUS DOMAINS OF COGNITION

The impact duration and quality of sleep on various subdomains of Clinical dementia rating scale were evaluated by comparing global PQSI scores to scores in individual subdomains of CDR. These would indirectly represent subdomains of cognition like memory, perceptuomotor activity, language, attention, executive function and social cognition. There was a significant association of higher global PQSI scores with poor performance in the subdomains like memory, orientation, judgement and problem solving, home and hobbies.

4. DISCUSSION

The objective of this study was to examine the association of sleep duration and quality with cognition. There were 2 groups in this study population. Group -1 (control) comprised individuals with normal cognition (with a CDR – clinical dementia rating of 0), and Group-2 (cases) comprised individuals with mild cognitive impairment (MCI) (with a CDR of 0.5). The mean age of the participants with CDR 0 (clinical dementia rating) was 70 years (66-73), Median (IQ1-IQ3), while the mean age of the patients with CDR 0.5 was 71 years (67-77). There was no significant difference in age between the 2 groups (p=0.084). However, it was noted that participants in Group 2 were slightly older than those in Group 1. There was no significant difference in sex amongst participants of the 2 groups (P= 0.772), although most were male.

There was a significant difference in educational status between people in group -1 with normal cognition and those with mild cognitive impairment GROUP- 2 (p =0.034). There was no significant variation in the major comorbidities between the groups.

On assessing the groups by applying the geriatric depression scale (GDS), there was a significant difference in the absolute value of the geriatric depression scale among the groups. Patients in the group with MCI (mild cognitive impairment had higher scores than the patients with normal cognition (GROUP-1) ($p < 0.001$). Similar findings were observed in studies performed by Morimoto, Preuss, Gutzmann and Koboyashi suggestive of an association of cognitive impairment with depression. (13,14,15,16)

However, these values were not high enough to label as depression. If the score (GDS) was > 8, these were recorded as cases of depression and were excluded from the study.

This was significant because some older adults with late-life depression could present with cognitive impairment mimicking dementia, i.e., pseudodementia syndrome. (13)

There was a significant difference in the absolute value of 6 CIT (6-item cognitive impairment test) with patients having MCI with consistently higher values in 6 CIT as compared to participants with normal cognition among both groups ($p < 0.001$). This is consistent with other similar studies by Dr. Brooke and

Dr. Sheehan, reported in the literature. (17,18). The tool used in this study (6 CIT, a screening tool) has a sensitivity and specificity of 78% and 100 %, respectively. It was used to rule out MNCD (major neurocognitive disorder). (17) This was further confirmed by performing CDR on these participants.

Anxiety among the participants could be a confounder leading to poor sleep duration and quality of sleep. Hence GAD -7 (Generalised anxiety disorder scale) was applied to participants of both groups, and the participants with GAD > 4 (suggestive of anxiety) were removed. There was no significant difference in the absolute value of GAD-7 among the groups. The reliability coefficient Cronbach's α for GAD-7 scale is 0.895, greater than the recommended value of 0.80, suggesting reliability for application in this study. (19)

There was no association established between fasting lipid profile and cognitive status in this study, for LDL, TG, TC, HDL respectively. This result was inconsistent with findings from studies like one by Iqbal et al which found lowering LDL, TG, TC level could prevent cognitive impairment. However, while higher HDL levels have been shown associated with improved cognitive performance, there is a dearth of studies establishing a negative association between lipid profile and cognition in the Indian population. More studies are thus, required to analyse findings in this regard. The groups (CDR 0 and CDR 0.5) also showed no significant difference in serum lipid profile between the participants who had impaired sleep for LDL, TG, TC, HDL respectively. This result was inconsistent with previous studies, like one by Merlino which has shown an inverse U relationship between blood HDL cholesterol levels and sleep duration i.e., individuals with both less and excess sleep duration had low serum HDL. (20) Another study by Kaneita reported that high triglyceride levels and low HDL levels were associated with short and prolonged sleep duration. (21) These results could be explained based on the presence of confounders like high BMI and OSA in the participants of these studies and hence further studies are required to establish this association.

The average value of serum vitamin B12 in all the recruited patients was 230 pg /ml. In comparison, the average value in MCI patients was 260 pg/ml (within the normal range but lower than the optimum 300 pg/ml). There was no significant association established between serum levels of vitamin B12 and cognitive status established in this study. (p=0.483) There was no difference of serum vitamin B12 levels between participants with normal (CDR 0) and impaired cognition (CDR 0.5). (p=0.847) The findings were consistent with other studies that haven't been able to establish a steady association between serum

vitamin B12 levels, cognition and sleep duration or quality. A study by Paayal et al has shown an inverse relationship between serum vitamin B12 and sleep quality. (22) Another study by Al-Musharaf found that higher levels of serum B12 were related to better sleep quality. These findings, however, could not be extended to global PQSI scores or total serum B12 levels. (23). Our observation in this study was in line with these findings in literature.

There are controversies regarding the association of vitamin D deficiency with poor sleep quality or sleep disorder risk and cognition. Metanalysis and systematic reviews by Meehan et al, suggests an increased risk of sleep disorders and unhealthy sleep related to vitamin D deficiency. (24) Yet another study Larsen et al suggested no consistent relationship of vitamin D supplementation on sleep even in vitamin deficient population. (25) A similar lack of association was established between individuals of both groups with high PQSI score (>5) in our study. (p=0.837) The average serum 25OH vitamin D value in MCI patients recruited in our study was normal (30.36 ng/ml). The average value in all participants was 30.93 ng/ml (normal range in older adults 20-30 ng/ ml). There was no significant relation established between vitamin D and cognitive status in our study(p=0.159)

It is hypothesised in studies that folic acid supplementation could reduce oxidative stress and telomere shortening induced by insomnia, improving sleep quality (26). However, a similar association could not be established in our study. (p=0.356) The average value of folic acid in all participants was 10.0 ng/ml, while the average value in MCI patients was 9.9 ng/ml (normal range 3-17.5 ng/ml). There was no association between folic acid and cognitive status in our study(p=0.990)

There are inconsistencies with regard to association of biomarkers with sleep and cognitive impairment, while some suggest a positive association yet others refute such claims. A larger multicentric study with a more uniform method of assessment is thus required to ascertain these findings.

With regards to the association of sleep parameters, there was a significant association between global PQSI scores and poor cognition (<0.001), wherein patients with mild cognitive impairment had higher PQSI scores (>5) as compared to those with normal cognition. Similar observations were reported in other studies by Potvin and Tworoger on older adults in the community, comparing global sleep quality to cognitive status. (27,28). The variation between the 2 groups regarding total sleep duration was statistically significant.

(p = 0.018). The total period of sleep score is a scale used to quantify the total sleep duration as categorical variables. The total number of participants with more than 7 hours of sleep (i.e., low TDS SCORE) was found to be significantly different in the 2 groups (1= 35.9%) vs (2=26.92%) of participants. (p = 0.013). GROUP 1 had a significantly longer duration of sleep. (More than 7 hours) Similar findings were reported in other studies by Potvin, Tworoger and Gildner with both a lack of sleep (<5 hrs) and excess sleep (> 9 hrs) associated with incidents of dementia. (27,28,29)

Variation in sleep latency between the 2 groups was statistically significant (p = 0.045). Sleep latency was greater in participants of GROUP 2. However, there was no statistical difference in the individual sleep latency scores. Similar outcomes were reported in other studies comparing cognitive impairment in older adults to sleep latency. (28) A significant difference was reported in the total number of sleep disturbances among the groups. (p <0.001). This finding was similar to other studies, like one by Tworoger that compared women in the age group of 70-80 years, belonging to the nurse's cohort, who showed a decline in cognitive test scores associated with sleep interruptions (snoring) and less sleep duration. (28) In another study by Potvin examining the impact of sleep in cognitively healthy older adults between 60-70 years of age from Quebec City, Canada, followed up over 12 months, higher sleep disturbance scores and poor overall PQSI scores were related to incident amnestic and non-amnestic MCI. (27)

There was no difference in the number of sleep medications used by the 2 groups of participants. There was a significant difference in the incidence of daytime sleepiness among participants of the 2 groups. (p <0.001). There was variation in this observation in different studies. In a study by Merlino, that recruited 750 Italian older adults with dementia, daytime somnolence was found in up to 30% of the participants. (30) In another study by Tworoger involving women from the nurse's cohort, no significant association was found between cognitive scores and daytime sleepiness. (28) There was no statistical difference in self-reported sleep quality (reported as per the Likert scale) by participants to their overall cognitive status in this study. However, other studies, like one by Gildner including a longitudinal sample of older adults from 6 different countries as a part of the SAGE study, found that self-reported sleep quality had a positive correlation with cognitive Z scores. (29)

Regression bivariate analysis of PQSI against cognitive status was statistically significant ($p < 0.001$). Higher global PQSI scores were related to poor cognition (MCI). Several studies observed similar outcomes, including the previously discussed study in older women of the nurse's cohort. Multivariate analysis of cognitive function across various sleep parameters showed that shorter sleep duration and sleep interruptions were linked to poor cognition. Similarly, in a prospective study by Potvin and Tworoger examining Canada's community-dwelling older adults, higher global PQSI scores were linked to cognitive impairment. (27,28).

There was a difference in sleep latency in participants of both groups. ($p= 0.038$). There was a statistical difference in the regression analysis between daytime sleepiness and participants' cognitive status. ($P \leq 0.001$) A significant difference was reported in the regression analysis of sleep disturbance against cognitive status ($p= 0.001$). These results were once again comparable to findings reported in several studies, including data from the SAGE study, which found that individuals with shorter and poorer self-reported sleep quality had worse cognitive scores. (29)

There was no significant variation in the total duration of sleep, TDS Score, abnormal sleep quality, use of sleep medication and sleep latency score between the groups.

In comparing various subdomains of CDR to different components of the Pittsburgh sleep quality index, there was a significant association between deficit in the cognitive subdomain of memory and higher global PQSI scores. ($p < 0.01$), in cognitively impaired older adults (CDR 0.5/MCI).

No similar outcome was observed in terms of overall PQSI score in other studies. However, a statistically significant association($p<0.05$) was reported on comparing poor working /verbal memory to sleep duration: short sleep (less than 5 hours) and long sleep (> 9 hours) ($p<0.05$) in a study by Groeger et al. (31)

There was a statistically important relation between poor orientation and reduced sleep duration and quality. ($p= 0.028$). Although not specific to orientation, multiple studies by Potvin, Tworoger, Gildner and Groeger, did report a significant association between poor sleep duration and quality in multiple cognitive domains. (27,28,29,31) There was a statistically significant relation between impaired judgement and problem-solving and poor sleep duration and quality. ($p= 0.048$). Other studies established A similar association

between excess or lack of sleep and executive function and processing speed. ($p<0.05$) (32). In a study by Tai Xin You et al., confirmatory factor analysis of 5 cognitive tasks was used to analyse executive function (EF) and found that 7 hrs or more sleep was related to high EF scores. (32) A significant relation was observed between low scores in the subdomain home and hobbies and poor sleep duration and quality. ($p= 0.017$), this could be substantiated by the overall poor cognitive function scores, especially in areas of executive function and processing speed associated with abnormal sleep duration. Similar findings were observed in a systematic review and meta-analysis by Groeger et al, involving 35 publications (11 cross-sectional and 7 cohorts) that showed extreme sleep durations were linked to poor multidomain performance, including executive function, working and verbal memory. (31) Although the primary objective of establishing an relationship between self-reported sleep duration and quality with cognitive status in older adults was satisfied in this study, large multicentric studies are required in the future to confirm these results.

5. CONCLUSION

This study found that individuals with mild cognitive impairment had shorter duration and poor overall quality of sleep compared to older adults with healthy cognition. Participants with mild cognitive impairment had significantly higher global PQSI (Pittsburgh sleep quality index) scores than participants with normal cognition. There was a statistically significant association of poor performance in subdomains of memory, orientation, judgement and problem-solving, home and hobbies with higher global PQSI scores. This was suggestive of a significant association between poor performance in these subdomains of CDR (clinical dementia rating) and poor sleep duration and quality. No statistically significant association was found between serum fasting lipid profiles, serum 25OH (D), serum vitamin B12, and serum folic acid levels within cognitively impaired adults with poor sleep duration or quality.

Limitations

The study included a sample size of 156 participants, leading to the low power of the study as a consequence of the COVID pandemic. Although consecutive participants were taken from OPD, most participants belonged to the Delhi NCR region, making the study's outcomes less generalizable to a larger Indian population. There was a discrepancy in the self-reported quality of sleep and global PQSI scores of some of the participants. This could be due to the recall bias. Inconsistency in the association of blood biomarkers to sleep quality could also be due to variations in the estimation technique amongst the laboratories.

6. REFERENCES

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