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Relationship between neurocognitive impairment and obstructive sleep apnoea hypopnoea syndrome (OSAHS)

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Abstract
Introduction: Obstructive Sleep apnoea is by far the most common form of sleep disordered breathing and is defined by frequent episodes of obstructed breathing during sleep. Excessive daytime sleepiness (EDS) is the most common complaint in patients with OSAHS. Symptoms other than EDS which greatly impact daytime functioning are neuropsychological symptoms such as irritability, difficulty concentrating, cognitive impairment, depressive symptoms, and other psychological disturbances. Sleep fragmentation and disruption and nocturnal hypoxemia have both been proposed to be related to daytime sleepiness and neurocognitive impairment. Many patients with OSAHS may have mild neurocognitive deficits and this may affect their quality of life. In this study, we intended to find out neurocognitive impairment in Indian patients with varying severity of disease (OSAHS).
Aim and objective: The aim of this is to find out neurocognitive impairment in Indian patients with varying severity of OSAHS.
Methodology: Diagnosed OSAHS patients were divided into three groups according to the severity of condition on the basis of polysomnography and Montreal cognitive assessment tool was administered to assess neurocognition in all the three groups of patients. **Result:** Statistically, there was significant difference in MoCA score (p<o.o5) between the three groups and it is related to AHI and nocturnal oxygen saturation.
Conclusion: OSAHS affects cognitive ability of patients significantly in severe group as compared to mild group and it is related to disease severity.

Keywords: obstructive sleep apnoea-hypopnoea syndrome (OSAHS), montreal cognitive assessment, apnoea-hypoapnoea index (AHI), lowest oxygen saturation

Introduction

OSA is by far the most common form of sleep disordered breathing characterized by frequent episodes of obstructed breathing during sleep. Specifically, it is characterized by sleep-related decreases (hypopnoea) or pauses (apnoeas) in respiration. An obstructive apnoea is defined as at least 10 seconds interruption OSA is by far the most common form of sleep disordered breathing and is defined by frequent episodes of obstructed breathing during sleep. Specifically, it is characterized by sleep-related decreases (hypopnoea) or pauses (apnoeas) in respiration. An obstructive apnoea is defined as at least 10 seconds interruption of oronasal airflow corresponding to complete obstruction of the upper airways, despite continuous chest and abdominal movements, and associated with a decrease in oxygen saturation and/or arousals from sleep. An obstructive hypopnoea is defined as at least 10 seconds of partial obstruction of the upper airways, resulting in an at least 50% decrease in oronasal airflow [1].
Patients with OSAHS might have neurocognitive dysfunction. Sleep fragmentation and nocturnal hypoxemia both may contribute to excessive daytime sleepiness and neurocognitive impairments [2]. In India, overall prevalence of OSA in males is 13.6% and in females is 5.6% [3].
In this study, we intended to find out neurocognitive impairment in Indian patients with varying severity of disease (OSAHS).

Methodology

A sample of 75 Indian subjects who met the inclusion criteria was selected from Kure medical system and Army hospital (Research and Referral). Entire procedure was explained and informed consent was obtained from them. Apnoea hypopnoea index (AHI) was used as screening tool. Eligible Patients were categorized in three groups based on assessment of AHI (Apnoea-Hypopnoea index) by overnight Polysomnography (PSG).

Group A - Mild OSAHS group (AHI > 5-15).
Group B - Moderate OSAHS group (AHI > 15-30), and
Group C - Severe OSAHS group (AHI > 30).

Montreal Cognitive Assessment (MoCA) was administered to these patients to assess their neurocognitive function.

Outcome Measure

Montreal cognitive assessment (MoCA) was used to assess neurocognitive functions.

Results

Total MoCA scores decreased progressively as the severity of OSAHS increased, with significantly lower scores for the moderate and severe OSAHS patients ($P < 0.05$) compared with mild OSAHS groups. Importantly, mild neurocognitive impairment, defined as a MoCA score < 26, was more prevalent in the moderate (44%) and severe (56%) OSAHS patients as compared with the mild OSAHS (28.0%) group. Table 1 shows the baseline characteristic of all the subjects and suggests that there was no significant difference among

the three groups ($p<0.05$).

Table 1: Age of patients in mild, moderate and severe OSAHS groups.

Groups		Age
Mild OSAHS	Mean	48.40
	Standard deviation	8.94
Moderate OSAHS	Mean	48.16
	Standard deviation	9.12
Severe OSAHS	Mean	48.00
	Standard deviation	12.38
p-value	0.990^{NS}	

(NS= NON Significant)

Table 2: Significance between Three Groups.

MoCA score	Between groups	Within groups	Total score
Sum of square	25.920	222.80	284.00
df	2	72	74
Mean square	12.960	3.084	
F	4.20		
p-value	0.19*		

*Significant ($p<0.05$)

There is significant difference in MoCA score between three groups (table 2). A significant reduction in subdomain scores for memory/delayed recall occurs in severe OSAHS group as compared to mild OSAHS groups ($P < 0.05$).

The results of the MoCA test for all the three groups are presented as mean and standard deviation in Table 3, 4, 5 respectively.

Polysomnography Results

Analysis of sleep structure by overnight polysomnography (PSG) revealed increases in respiratory arousal index and decrease in oxygen saturation as severity of the disease increases.

Table 3: MoCA score in mild OSAHS.

Group A	Mild OSAHS	
	Mean	Standard deviation
MoCA score	26.72	1.59
MoCA subdomain score		
Visuospatial and executive	4.36	0.75
Naming	2.68	0.47
Memory and delayed recall	4.28	0.54
Attention	5.28	0.45
Language	2.44	1.76
Abstraction	1.76	0.43
Orientation	5.84	0.37

Table 4: MoCA score in moderate OSAHS.

Group B	Moderate Osahs	
	Mean	Standard deviation
MoCA Score	26.00	1.70
MoCA subdomain score		
Visuospatial and executive	4.08	0.70
Naming	2.72	0.45
Memory	4.04	0.61
Attention	5.28	0.67
Language	2.40	0.57
Abstraction	1.68	0.27
Orientation	5.92	0.27

Table 5: MoCA score in severe OSAHS.

Group C	Severe Osahs	
	Mean	Standard deviation
MoCA Score	25.28	1.94
MoCA subdomain score		
Visuospatial and executive	4.04	0.78
Naming	2.80	0.40
Memory and delayed recall	3.80	0.70
Attention	5.00	0.64
Language	2.28	0.73
Abstraction	1.60	0.50
Orientation	5.76	0.43

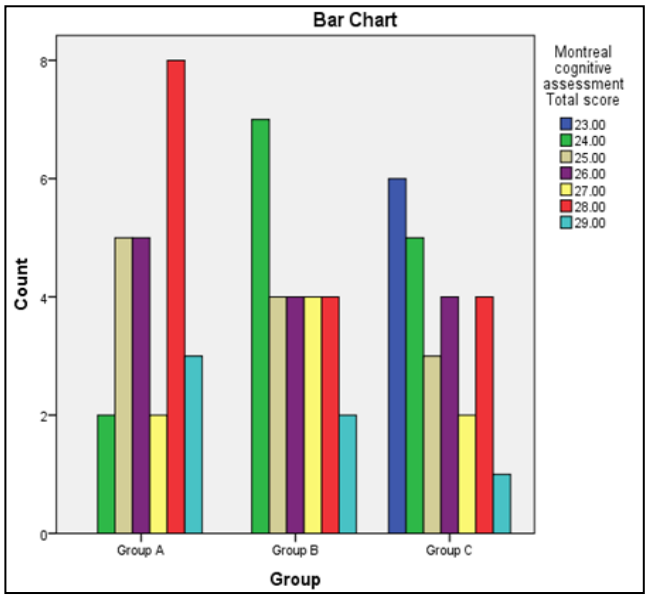

Fig 1: MoCA Score in all the three groups of OSAHS severity

Correlation between MoCA scores and polysomnography variables

Pearson correlation coefficient was performed to evaluate the correlations among MoCA score and PSG (AHI and (SaO2<90%), L-SaO2) variables. Negative correlation between AHI and MoCA and positive correlation between SPO_2 and MoCA was found.

Data analysis

Data are presented as mean and SD, and statistical analyses were performed using SPSS 20 software for

1. One-way analysis of variance comparing the MoCA and Polysomnographic variables among the groups of patients with different severities of OSAHS. Statistical significance was defined as P < 0.05.
2. Pearson correlation coefficient was performed (r) to evaluate the correlations between individual clinical and polysomnographic variables and MoCA and MMSE scores.
3. Chi square was performed to analyse qualitative data.

Discussion

In this study, 75 Indian patients with various severities of OSA were evaluated using MoCA questionnaire. Mild cognitive impairment as defined by a MoCA score <26 was more frequent in patients with moderate and severe OSAHS

than in the mild groups. To further explore the impact of OSAHS on various aspects of cognitive functioning, the MoCA subdomain scores were analysed. In the severe OSAHS patients, significant reductions were most evident in the subdomains for memory/delayed recall, whereas there were no significant differences among the three patient groups in the subdomains for language, and orientation. In the present study, 75 Indian patients with diagnosed OSAHS were selected with varying severity of the disease. Sleep disruption was more prominent in patients with severe and moderate group as compared with mild group. In addition, nocturnal hypoxemia was also increasingly evident as severity of the disease increases

The exact nature of the relationship between OSAHS and cognition remains unclear and has been debated. Bonnet *et al*. identifies that arousal and sleep fragmentation as factor contributing to impairment in cognition in OSAHS patients [4], whereas other studies have suggested that intermittent hypoxemia promotes oxidative stress by increased production of reactive oxygen species and angiogenesis, increased sympathetic activation with BP elevation, and systemic and vascular inflammation with endothelial dysfunction that contributes to diverse multiorgan chronic morbidity and mortality affecting cardiovascular disease, metabolic dysfunction, cognitive decline [5].

In the present study, the Pearson correlation coefficient analysis revealed that MoCA score was positively correlated with lowest oxygen saturation (L-SPO_2), which suggest nocturnal hypoxemia may be the contributing factor to neurocognitive impairments in OSAHS. The Pearson correlation coefficient analysis also revealed that MoCA score was negatively and significantly correlated with apnoea-hypoapnoea index (AHI). In addition, nocturnal hypoxemia was also increasingly evident as severity of the disease increases. Hence, both factors are considered as related to decreased performance on cognitive test.

In India, a study was conducted on pattern and correlates of neural dysfunction in obstructive sleep apnoea. This study suggested that patients with severe OSAHS has delayed information processing resulting in reduced short term memory. In this study, no significant correlation was present between test scores and apnoea hypopnoea index and also there is weak correlation between lowest oxygen saturation and scores of test. Our study suggests that neurocognitive impairments exists in all the three groups of OSAHS and is weekly but significantly correlated (0.01) with the polysomnography parameters (apnoea hypopnoea index and lowest oxygen saturation) [6].

The cellular and molecular mechanisms underlying the neurocognitive impairment associated with OSAHS remain poorly defined and warrant further investigation. Multiple pathophysiological processes or pathways, including oxidative stress, inflammatory responses, reduced synaptic plasticity and neuronal apoptosis, have all been suggested to contribute neuropsychological dysfunction [2].

Clinical relevance

As OSAHS is associated with cognitive deficits, we can give cognitive rehabilitation to the patient. The goals of cognitive rehabilitation are to enhance the person's capacity to process and interpret information. Restorative training focuses on improving a specific cognitive function (cognitive exercises includes computer assisted strategies), whereas compensatory training focuses on adapting to the presence of a deficit (memory books and electronic paging system.

We can make the patient aware about weight reduction as weight reduction leads to improvement in condition of patient and also reduces the severity of sleep apnoea.

We can prescribe physical exercise prescription to patients as physical exercise can be directly protective against mild cognitive impairments.

Conclusion

From this study, was concluded that Indian patients with OSAHS have cognitive deficits in all the three stages of severity, though the degree of impairments depends on the severity of the disease. Hence, we accept experimental hypothesis. The result of this study suggested that neurocognitive impairments in OSAHS patients is related to both the nocturnal hypoxemia.

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