

2nd International Conference on

Respiratory and Pulmonary Medicine

October 17-18, 2016 Chicago, USA

Consequence of obstructive sleep apnea on functioning of nervous system

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Obstructive sleep apnea (OSA) is highly prevalent within the primary care community and yet it is frequently undiagnosed. OSA per se is not looked as disease by the masses and is thought as deviations from the usual sleep pattern. In fact, OSA is a major risk factor for cardiopulmonary insufficiency and other complications if not intervened appropriately. Until now no study has characterized the severity of OSA and its impact on functioning of nervous system in Indian subjects. The objective of this study was to evaluate the prevalence of OSA in a South Indian population, to assess the neurocognitive functioning in patients and also to correlate the changes in higher mental functions, nerve conduction velocities and brain natriuretic peptide (BNP) with the duration and severity of OSA. A retrospective analysis of data accrued in patients undergoing polysomnography (PSG) was undertaken. Cognitive functioning was assessed by mini-mental state examination (MMSE) and depression was evaluated using Zung self-report depression scale. Two hundred fifty-four patients of either sex in the age of 54 ± 11 years who tested positive for OSA were compared with control group. An apnea hypopnea index (AHI) of >5 in the presence of snoring and daytime somnolence was taken to define sleep apnea. The OSA patients were divided into three subgroups: mild, moderate and severe depending upon AHI. The mean AHI among the study group with OSA was 31.3 ± 18.6 as compared with 1.2 ± 0.5 in the control group. Among OSA patients (31%) were having mild OSA, moderate (33%) and severe (59%), respectively. The BMI among patients with OSA was 32 ± 6 as compared with 24 ± 4 in comparison group ($p < 0.001$). A significant decrease in cognitive impairment (by MMSE score) in OSA patients was observed when compared to control. Orientation, memory, registration, attention, calculation skills, language and constructive praxis were significantly low in OSA patients in contrast with controls. A higher decline in depression score was observed in severe OSA patients when compared to patients having moderate OSA. Autonomic dysfunctioning was observed with severe OSA, which may play a key factor in the causal link between OSA and cardiovascular disease. The risk of metabolic syndrome in OSA syndrome patients was almost three times more than that of the comparing group. Our study found a noticeable relation between the severity of the Mallampati score and a restrictive type of defect on spirometry in OSA patients. Our data also provided compelling evidence that OSA is associated with cognitive decline and depression. Although hypoxemic stress and sleep disruption are likely the key players in the pathogenetic mechanisms behind such derangements, the role of an underlying common denominator needs to be scrutinized. We are doing further studies to define the driving mechanisms through which sleep-disordered breathing promotes many of these consequences. Nevertheless, the present study was a novel approach to nervous system effects of OSA, which may help the clinicians to diagnosis and prognosis and the researchers to look into new era in OSA research.

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