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OBSTRUCTIVE SLEEP APNOEA IN PATIENTS WITH STROKE VERSES THOSE WITH REFRACTORY HYPERTENSION SEARCH FOR COMMON PATHOGENIC MECHANISM.

Anupama Gupta[1], Garima Shukla[2], Gautam Sharma[1], Ambuj Roy[3], Vinay Goyal[4], Vivekanandhan S[5], Balram Bhargava[6], Madhuri Behari[7]

[1] Center for Integrative Medicine and Research, All India Institute of Medical Sciences, New Delhi, [2] Division of Neurology, Department of Medicine, Queen's University, Kingston, ON, Canada., [3] Deparment of Cardiology, All India Institute of Medical Science, New Delhi, [4] Department of Neurology, All India Institute of Medical Scieces, New Delhi, [5] Department of Neuro-biochemistry, Neuroscience Centre, All India Institute of Medical Sciences, New Delhi, [6] Department of Cadiology, All India Institute of Medical Sciences, New Delhi, [7] Department of Neurology, All India Institute of Medical Sciences, New Delhi

Introduction: Obstructive sleep apnea [OSA] is a risk factor for stroke and refractory hypertension [RH]. OSA causes disrupted sleep, intermittent hypoxia, and hypoxia-induced systemic inflammation and sympathetic activation are potential pathogenic mechanisms for the first ever stroke and refractory hypertension. The aim of the present study was to evaluate sleep quality among OSA among patients with first ever stroke, as compared to RH and to assess markers of sympathetic activation and systemic inflammation in patients with either stroke or RH.

Methods: All patients with stroke and RH were recruited from the Departments of Neurology and Cardiology, respectively at the All India Institute of Medical Sciences, New Delhi. All patients were interviewed for demographic details, clinical history and were scheduled for polysomnography. Fasting blood samples were collected during morning hours, to estimate biochemical markers [Norepinephrine, CRP, TNF-∞, VGEF and IL6] . CRP, TNF-alpha, and IL-6], measured by the chemiluminescence method. Nor-adrenaline and VEGF were measured using ELISA techniques.

Results: A total of 114 stroke patients and 56 refractory hypertension patients were included. The prevalence of OSA among stroke and RH patients was found to be 86.85 and 81% respectively. The mean number of wake after sleep onset periods (WASOs) [45±19.22 vs 29.8±11.14, p< 0.01]was significantly higher in patients with stroke. REM AHI [240.04±24.65 vs 33.20±22.35, p< 0.01] was significantly higher in patients with RH.CRP and VEGF values were higher than normal, in both groups. The median value of early morning serum nor-adrenaline was higher in the RH group[407 (66.6-1000) vs 515 (240-1250), p = 0.05].

Conclusion: OSA is highly prevalent among the patients in both groups. Systemic inflammation is a common pathogenic mechanism linking OSA with RH and stroke, whereas early morning sympathetic activation appears significantly important in the pathogenesis of refractory hypertension.

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