CEREBRAL ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

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Introduction: The association between obstructive sleep apnea (OSA) and stroke, with endothelial dysfunction (ED) as the main pathogenic mechanism involved, is increasingly recognized. Repeated cycles of hypoxia and re-oxygenation are supposed to be the main cause of ED in OSA but their exact influence is masked by high prevalence of arterial hypertension, diabetes and obesity in OSA patients, which all lead directly to ED. ED measured by functional mediated dilatation of brachial artery (FMD) has been clearly shown in patients with OSA when compared to healthy subjects. Furthermore the role of ED has also been determined in patients with lacunar stroke, the type of stroke particularly common in OSA patients. The cerebral endothelial function can be assessed by ultrasound measurement of cerebrovascular reactivity to L-arginine (CVR-arg). Cerebral endothelium might be specifically damaged in OSA patients.

Patients and Methods: 32 OSA patients were compared to 28 subjects with comparable risk factors for ED. CVR-arg was determined as a relative increase of the mean arterial velocity in both middle cerebral arteries measured by TCD during ten minute interval before and after L-arginine infusion.

Results: CVR-arg was significantly higher in the controls (10.6%±5.9%) when compared to OSA patients (7.9%± 5.1%) (p≤0.01), indicating that cerebral ED in OSA patients is more pronounced.

Conclusions: We found a significant cerebral ED in OSA patients compared to a risk factor matched control group. A direct association between OSA and ED probably dictates detection and treatment of OSA as an important independent risk factor of stroke.