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White matter lesions, clinical parameters and neuropsychological deficits in patients with OSAS



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Objectives

Obstructive Sleep Apnea Syndrome (OSAS) related cognitive dysfunctions have already been reported. However, no significant correlations were found between cognitive dysfunctions and clinical and polysomnographic parameters. Nevertheless, silent cerebrovascular lesions have been found in these patients, suggesting that a subcortical damage may be the cause of the cognitive alterations found in these patients. Aim of the study was to analyze the correlations between neuroradiological findings, clinical parameters and cognitive deficits of patients with OSAS.

Patients and Methods

Patients with diagnosis of OSAS made according to ICSD-2 criteria were consecutively



enrolled. They completed a neuropsychological battery, including measures of attention, working memory, verbal memory, executive functions and visuospatial skills. OSAS was assessed by polysomnography. The patients underwent MRI acquisitions with T1 and FLAIR sequences. An automatic measurement of white matter (WM) lesions volume was carried out through the use of LST SPM8 software (Fig.1).

Figure 1 FLAIR MR images (on the left) and the same slice with automated segmented lesions (on the right).

Results

Demographics and MRI characteristics of our 27 patients are shown in table 1 and table 2. The volumes of gray matter, white matter, cerebrospinal fluid and intracranial lesion load were calculated. Significant correlations between WM lesions and neuropsychological tests were found and are shown in table 3. No significant correlations were found between the indices of severity of apnea and neuropsychological tests nor neuroimaging parameters.

Table 1: demographic characteristics		Table 2: MRI parameters		Table 3: correlations of neuropsychological tests with WM lesions.		
Variable	Mean ± SD	MRI Parameter	Volume (Mean ± SD)	Test	Score (Mean ± SD)	Correlation with WM lesions
Age (years)	56.81 ±12.38*	GM	470.4 ± 85.4	MMSE	26.56 ± 2.30	ρ = -0.420; p = 0.029
Sex (M) (%)	78	WM	591.1 ± 99.5	FAB	14.40 ± 3.65	NS
Education (years)	7.96 ± 3.71			Hamilton	7 ± 4.83	NS
BMI (kg/m2)	31.91 ± 4.48	CSF	224.2 ± 49.3	RAVLT		
Diabetes (%)	33	ICV	1239.8 ±270.2	Immediate recall	30.69 ± 5.56	NS
Hypertension (%)	67	Lesions	1.8 ± 4.3	Delaved recall	5.36 ± 1.58	NS
Dyslipidemia (%)	56			Words recognition	10 78 + 2 90	$\rho = -0.473 \ \rho = 0.013$
ESS	14.5 ± 4.68	GM=Grey Matter; WM=V	Vhite Matter;	False recognition	1.56 + 1.45	NS
AHI	36.31 ± 30.63	CSF=Cerebrospinal Flui Data are shown in ml.	d; ICV=Intracranial volume	Digit Span	1.00 - 1.10	
ODI	36.62 ± 31.08			Span forward		a = 0.462 = 0.017
*ρ = 0.582; p = 0.001;				Span lorward	5.15 ± 1.40	$\rho = -0.463, \rho = 0.017$
3MI=Body mass index; M=n	nale; ESS=Epworth			Span backward	2.92 ± 1.26	NS
ODI=Oxygen Desaturation I	ndex			MFTCT		
Conclucion				Accuracy	0.93 ± 0.09	NS
		fermed betw		Time (sec.)	126.81 ± 47.09	NS
A significant	correlation was	tound betw	een some	False	0.91 ± 2.08	NS
neuropsychological performances and WM lesions load,				Stroop		
ndependently from vascular risk factors and severity of apnea				Time (sec.)	22.84 ± 10.32	NS
ndices. Our results point out the role of age related WM lesions in			Errors	1.22 ± 1.62	ρ = 0.464; p = 0.017	
the pathogenesis of cognitive deficits found in patients with OSAS.				COWAT	24.86 ± 8.37	$\rho = -0.484; p = 0.012$



M. L. Ho, S. D. Brass. Obstructive sleep apnea. Neurology International 2011; volume 3:e15; 60-68.



