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BPSD SEVERITY IN ALZHEIMER'S DISEASE IS INDEPENDENT FROM VASCULAR IMPAIRMENT AND WHITE MATTER LESIONS BURDEN

Bove R, Giuliani A^, Pavino V, Guglielmi V*, Rossi C*, Massimetti MC,
Iani C, Vaianella L°, Masullo C*, Bizzarro A*
S. Eugenio Hospital, Neurology Division; ^National Institute of Health, Environment and Health;
*Catholic University, Neuroscience Department;
°National Institute of Health, Cellular Biology and Neuroscience

Background: BPSD affected almost all AD patients during the disease history, decreasing quality of life for patients and caregiversb (1, 2). In Italy the family represents the main care-giving response to the needs of their demented relatives and time to spend in patient care-giving increases linearly with the disease progression. Caregiver's reports on patients ADLs are often influenced by the caregiver's burden (3). Several studies investigate on caregivers burden in order to determine a better evaluation of BPSD onset, severity and typology (4, 5). On the other hand, in literature there a lot of studies analyzing possible correlations between biological factors and BPSD (overall genetic risk factors), comorbidities (overall vascular impairment) and white matter lesions burden (6,7) showing conflicting results. Understanding pathogenetic background and risk factors is the primary step to a better BPSD management (1).

Aim of the study

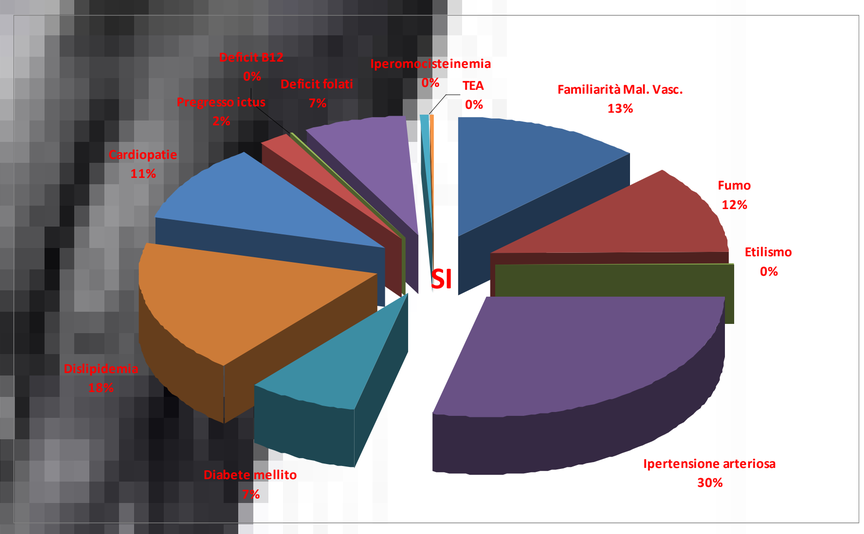
To analyze a possible relationship between BPSD severity and vascular, genetic and cognitive variables

Subject and methods: 135 patients diagnosed as Probable AD, according to NINCS-ADRDA criteria, were enrolled from two different Alzheimer Unit (Catholic University of Rome and Sant'Eugenio Hospital). Each subject underwent: clinical/instrumental examination (including brain MRI), neuropsychological evaluation (including MMSE), behavioural assessment (NPI).
Modified Fazekas Scale (FS) and Hachinski Ischemic Score (HIS) were applied to the whole sample in order to analyze the white matter lesions burden and the global vascular impairment. ApoE genotype was analyzed in 92 patients.
Sample characteristics are showed in Table 1. Vascular risk factors and comorbidities of the whole sample are showed in Graphic 1. ApoE genotype distribution is showed in graphic 2.
Data analysis was obtained by Spearman correlation coefficient and by Principal component analysis.

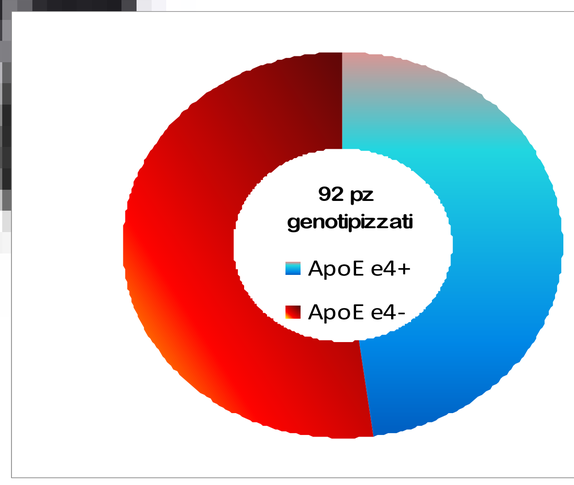
Table1

	Mean	StDv
Age	74,5	7,16
Education	8	4,14
MMSE	18,6	4,8
FS	1,14	0,86
HIS	2,82	2,08

Graphic 1



Graphic 2



Results

BPSD severity (estimated by NPI total score) was independent from cognitive impairment (MMSE), vascular impairment (HIS), white matter lesions (FS) and ApoE status.

Discussion and conclusions: Our data do not confirm a possible role for vascular impairment in BPSD pathogenesis as previously reported (2) even if the majority of studies considering vascular risk factors and white matter hyperintensities led to conflicting results (6,7). Although NPI represents the most diffused tool to investigate BPSD in dementia, our results, according to recent studies (8,9), underlie an emerging need to investigate bio psychosocial and environmental factors in BPSD pathogenesis. NPI is an helpful measure to asses BPSD in demented people but is a caregiver dependent measure. The caregiver individuality (such us age, educational level, personality, psychological disturbances…) might modify the caregiver reliability and determine a caregiver higher level of stress and burden (10). Therefore it's essential to asses also caregivers burden including measures of objective and subjective caregiver stress and analysis of environmental conditions. Furthermore, several studies show that grouping BPSD into "clusters of symptoms" (such as depression/anxiety; delirium/hallucinations…) a relationship with different variables might be established (10, 11) even if disagreeing results have been often reported. These data suggest a different role in BPSD pathogenesis for vascular, genetic and environmental factors and underlie the complex interactions between the different variables. The challenge for future studies might be to better understand this complex cooperation of variables in BPSD pathogenesis overall analyzing bio-psycho-social factors that are the most unidentified.

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