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The impact of orthostatic hypotension on non-motor symptoms of Vascular Parkinsonism

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Introduction: Vascular Parkinsonism (VP) is a secondary parkinsonism typically associated with arteriosclerotic encephalopathy. Several clinical features characterized VP including motor and non-motor symptoms such as impairment of attention, executive function, and verbal memory, depressive disorder, apathy, sleep disorders, gastrointestinal dysfunction and orthostatic hypotension (OH). Although OH occurs in up to 26% patients with VP, its impact on other non-motor symptoms in patients with VP is still unknown.

Objective: The aim of our study was to assess the impact of OH on other non-motor symptoms in patients with VP with OH (OH+) and in patients with VP without OH (OH-).

Methods: The study included 11 patients with VP (OH+) and 11 patients without VP (OH-). All subjects underwent a complete clinical, neuropsychiatric and neuropsychological assessment. Clinical evaluation included full neurological examination, the Non-Motor Symptoms Scale (NMSS), to assess non-motor symptoms, and a standard Tilt-test protocol. Neuropsychiatric evaluation assessed: depression, anxiety, apathy, anhedonia and alexithymia. Neuropsychological battery included evaluation of: global index of cognitive impairment, short- and long-term verbal memory, long-term visual-spatial memory, immediate visual memory, language abilities, complex constructional praxis, attention and executive functions.

Results: The result of NMSS indicated that patients with VP (OH+) experienced more "excessive sweating" (Domain 9, item 30) than VP (OH-) (36,40% vs. 0,0%; p=0.027). No significant neuropsychiatric and neuropsychological differences were found between groups.

Conclusion: Our results suggest that patients with VP (OH+) have excessive sweating. If this relationship is causative or associative remains unclear. Possible explanations are that excessive sweating is driven by hypovolemia, one cause of OH, or that OH and excessive sweating are both consequences of autonomic nervous system dysfunction. We hypothesize that neuropsychiatric symptoms and neuropsychological deficits may not emerge in patients with VP (OH+) because compensatory mechanisms of cerebral vasoregulation and homeostatic autoregulation in VP may be already impaired.

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