THE DEMENTIA OF PARKINSON'S DISEASE: VASCULAR LESIONS ARE IMPORTANT AND FREQUENT CONTRIBUTORS TO COGNITIVE DECLINE IN PD

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Numerous studies have demonstrated that lacunes and white matter lesions (WML), which are to a large extent ischemic in origin, correlate with impairment in frontal executive and psychomotor functions, memory decline, and depression. Strategic lacunes (such as in the head of the caudate or thalamus) may increase the risk of cognitive deficits (Reed BR et al. Arch Neurol 2004; 61:1545). Concomitant cortical and hippocampal atrophy may have additive or complementary effects. The main risk factors for subcortical ischaemic vascular dementia are age, hypertension, diabetes, smoking, hyperhomocysteinaemia, hyperfibrinogenaemia, and genetically determined vasculopathies, such as CADASIL or amyloid angiopathy (Román GC et al. Lancet Neurology 2002; 1:426).

Recent studies suggest that mixed brain pathologies account for the majority of dementia cases in community-dwelling older patients (Schneider JA et al. Neurology 2007; 69:2197). Interaction of degenerative and vascular pathology has been suggested in autopsy studies on Alzheimer dementia (AD) and Parkinson disease (PD) with dementia (Jellinger K. J Neural Transm 2002; 109:813; Jellinger KA, Attems J. Acta Neuropathol 2008; 115:427).

The risk of dementia in PD increases markedly with age and so does the risk of concomitant cerebrovascular disorder including small vessel disease. Vascular risk factors are risk factors for AD, which contributes to dementia in around 80 % of patients with dementia associated with PD. Elderly PD patients with history of minor stroke, ischaemic heart disease, diabetes and deep WML exhibit higher Hoehn and Yahr stages and lower Mini Mental State Examination scores than patients without vascular comorbidity (Papapetropoulos S et al. Eur J Neurol 2004; 11: 231; Beyer MK. Mov Disord 2006; 21:223). However, this might not be the case in younger PD patients (Derejko M et al. Neurol Neurochir Pol 2006;40:276) and extent and speed of progression of WML seem to be crucial for the prognosis, as demonstrated recently (Schmidt R et al. Stroke 2007;38:2619). Moreover, L-Dopa induced hyper-homocysteinaemia might increase the risk of cerebrovascular disease, but also atrophy and depression in PD (Rogers JD et al. Arch Neurol 2003; 60:59-64). Finally, the boundaries between vascular parkinsonism and PD may be vague, in particular in old subjects (Sibon I; Tison F. Curr Opin Neurol 2004; 17:49), and therefore the effect of vascular disease underestimated.

In summarizing, it is likely that vascular pathology, in particular if extensive, rapidly progressive, or located in strategic areas, contributes to cognitive and motor deterioration in PD.