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Cognitive impact of microvascular lesions and lacunes in the aging brain

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Most previous studies addressed the cognitive impact of microvascular pathology and lacunar infarcts using radiologic correlations which are known to correlate poorly with neuropathological data. Moreover, the absence of systematic bilateral assessment of vascular lesions as well as the masking effect of Alzheimer's disease related pathology and macrovascular lesions may explain discrepancies among previous reports. In order to define the relative contribution of silent lacunes and microvascular lesions in cognitive decline, we performed a detailed neuropathological analysis in both cortical and subcortical areas of 72 elderly individuals without significant neurofibrillary tangle pathology or macrovascular lesions. Cognitive status was assessed prospectively using the Clinical Dementia Rating (CDR) scale; neuropathological evaluation included Aß-protein deposition staging and bilateral assessment of microvascular ischemic pathology and lacunes; statistical analysis included multivariate models controlling for age, amyloid deposits and significant microvascular pathology. Thalamic and basal ganglia lacunes were negatively associated with CDR scores; cortical microinfarcts, periventricular and deep white matter demyelination also significantly affected cognition. In a multivariate model, cortical microinfarcts and thalamic and basal ganglia lacunes explained 22% of CDR variability; amyloid deposits and microvascular pathology explained 12% and the assessment of thalamic and basal ganglia lacunes added an extra 17%. Deep white matter lacunes were not related to cognitive status both in univariate and multivariate models. Our autopsy series provides important evidence that gray matter lacunes and cortical miroinfarcts are independent predictors of cognitive decline in elderly individuals without concomitant dementing processes such as Alzheimer's disease.