MICROVASCULAR PATHOLOGY HAS A CRUCIAL ROLE IN COGNITIVE DECLINE

G. Gold
Service de Médecine Interne de Réhabilitation Hôpital des Trois-Chêne Dpt de Réhabilitation et Gériatrie 3, Chemin du Pont Bochet, Thonex, France

Vascular dementia was described in the XIXth century and depictions dating back to the early 1900s have shown that dementia may be associated with many different types of acute and chronic vascular changes. However, the cognitive impact of each type of vascular lesion according to size and location has been much more difficult to define.

The relationship between macroinfarcts and cognitive changes is well known but the availability of new neuroimaging techniques in the late XXth century, has led to the realisation that chronic ischemic changes are much more commonly associated with impaired intellectual function. This has led to the definition of more homogeneous subtypes including sub cortical ischemic vascular dementia which is characterized clinically by a dysexecutive syndrome and memory deficits and neuropathologically by lacunes and white matter changes.

The morphological substrates of cognitive decline associated with cerebrovascular pathology have been the subject of several recent studies that have identified the crucial importance of cortical microinfarcts. Thalamic and grey matter lacunes are also associated with cognition but lacunes in deep frontal, parietal and temporal white matter seem to be clinically silent with regards to intellectual function. Demyelination, particularly when it is periventricular, also appears to play a measurable but more minor role.
Brain aging is associated with progressive development of both degenerative changes (amyloid plaques and neurofibrillary tangles) and vascular pathology in individuals with normal cognition. Mixed dementia is diagnosed when such lesions are described in older people with impaired intellectual function. However, this entity covers a wide clinical and neuropathological spectrum between pure Alzheimer's disease (AD) and isolated vascular dementia. Neuropathological studies over the past two decades have pointed out the key role of massive neurofibrillary tangle formation within adjacent components of the medial and inferior temporal cortex in clinically overt AD. Braak's hierarchical scheme of neurofibrillary tangle development has been shown to be a good correlate of clinical findings making it possible to identify, in part, the transition between normal aging and dementia. The recent development of relatively simple semiquantitative ischemic scores that are closely associated to cognitive function and the identification of thresholds for dementia of vascular origin have been applied successfully, along with Braak neurofibrillary tangle classifications of neurodegenerative pathology to neuropathologically define pure AD and vascular dementia and mixed cases. This classification has proven highly accurate in distinguishing dementia from cases associated with intact cognition.