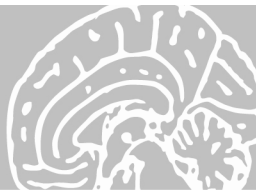
혈관성 인지장애 환자에서 아밀로이드와 뇌혈관병변의 임상적 영향



서상원·김희진·나덕렬
성균관대학교 삼성서울병원 신경과

Clinical Impact of Amyloid and Cerebrovascular Disease in SVCI Patients: Korean Studies

Sang Won Seo, Hee Jin Kim, Duk L. Na
Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Subcortical vascular cognitive impairment (SVCI), consisting of vascular dementia and MCI of subcortical types, is characterized by extensive cerebral small vessel disease (CSVD) such as white matter hyperintensities (WMH) and lacunes in white matter regions. Alzheimer's disease (AD) and CSVD are the two major causes of cognitive impairment in the elderly. Recent pathological and in vivo amyloid PET studies showed that many patients had both Aβ and CSVD burdens. However, the underlying mechanisms regarding how Aβ and CSVD burdens affect cognitive impairments remain unsettled. In our initial studies, we found there were differences in the neuropsychological results, cortical thinning, hippocampal shape, structural and functional connectivities between pure SVCI patients and pure AD patients. Direct comparison of two groups showed that AD had more temporoparietal abnormalities and memory dysfunction while SVaD had more frontal abnormalities and dysfunctions. Compared to SVCI patients with amyloid burdens (mixed dementia), pure SVCI patients revealed younger onset age, more lacunes, and less hippocampal atrophy. Several cross-sectional neuroimaging studies suggested that there are downstream changes for Aβ or CSVD burdens. That is, Aβ and CSVD burdens, respectively, affected thinning in the medial temporal and frontal regions, which were further associated with corresponding cognitive impairments. Also, a previous study from our group reported that structural network is disrupted by CSVD, but not Aβ, which can further influence frontal-executive dysfunction. Previous studies have suggested that AD and SVaD may lie on opposite ends of a single disease spectrum. One of the main reasons is that they have common risk factors, such as age, hypertension, diabetes etc. Another reason is that preclinical studies suggested that CSVD and Aβ burden have a strong association. This association leads to the possibilities of CSVD causing or accelerating AD through decreased clearance of amyloid, or cerebral amyloid angiopathy possibly accelerating CSVD. In particular cases, there were correlations between Aβ & CSVD & their synergistic effects on cognition or lobar microbleeds.

Key Words: Cerebrovascular disease, Amyloid, Clinical impact

Sang Won Seo, Hee Jin Kim, Duk L. Na
Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea
TEL: +82-2-3410-1233
E-mail: sangwonseo@empal.com