Cerebral Amyloid Angiopathy with a Varied Hemorrhage Pattern on T2*-Weighted Magnetic Resonance Image

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A 59-year-old dentist with diabetes mellitus, chronic hepatitis type C and hypertension developed cognitive impairment and consulted our hospital. Mini mental state examination score was 24, which seemed low for his age and educational background. Brain computed tomography (CT) showed multiple small hemorrhages in the cortical-subcortical area and a minor subarachnoid hemorrhage in the left occipital lobe. Magnetic resonance gradient-echo T2*-weighted imaging (T2*) demonstrated multiple microbleeds (MBs) in the subcortical area and cerebellum, subarachnoid hemorrhage and superficial hemosiderosis (Picture 1). Eight months later, he developed delirium, was arrogant to his clinic staff, and showed excessive eating despite dietary restrictions for diabetes. Voxel-based Specific Regional Analysis System for Alzheimer Disease (VSRAD) score was 2.68, indicating severe hippocampal atrophy. Tc-99m-ethyl cysteinate dimer (ECD)-single photon emission tomography showed decreased cerebral blood flow in the posterior cingulate gyrus and precuneus. Alzheimer disease (AD) was diagnosed based on these findings.

The patient did not develop major bleeding but T2*, which is a sensitive technique for detection of hemosiderin, showed a varied hemorrhage pattern, suggesting cerebral amyloid angiopathy (CAA). CAA is a well-known disease involving amyloid deposition in small vessels in the brain, particularly in elderly people, patients with AD, dementia with Lewy body, and hereditary amyloidosis. Amyloid deposits make the vessels fragile, resulting in MBs, lobar hemorrhage, multiple cortical infarctions, leukoencephalopathy or subarachnoid hemorrhage. Residues of bleeding spread in the subarachnoidal space can cause subpial hemosiderin deposition (1).

According to the Boston criteria for the diagnosis of CAA-related hemorrhage, postmortem autopsy or biopsy is necessary to define CAA (2). Although we did not perform biopsy in the present case, MBs in AD are known to be related to CAA and the varied pattern of hemorrhage on T2* confirms the diagnosis of probable CAA (1, 3). Because

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MBs are an important predictor of future hemorrhage, it is useful to evaluate hemosiderin deposits with T2* in AD patients (3).

Recent advances in imaging techniques have enabled amyloid deposition to be identified without biopsy (4). Susceptibility-weighted MRI is reportedly more sensitive than T2* for detecting MBs. The beta-amyloid burden is quantified with positron emission tomography (PET) assessed by Pittsburgh compound B. These techniques may be helpful in determining the imaging criteria for CAA.

References


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