学位論文審査結果の要旨

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（学位論文審査結果の要旨）

Maximum isotope accumulation in the retrosplenial cortex during amnesia attack and its temporal change suggest cortical spreading depression as a pathophysiology of patients with transient global amnesia

【主論文審査結果の要旨】

Transient global amnesia (TGA) is a clinical syndrome that is characterized by the sudden onset of anterograde amnesia and a less prominent impairment in retrograde memory lasting up to 24h. Although several etiologies have been proposed, including migraine, epilepsy, ischemia, venous congestion, and glutamate toxicity, none sufficiently explain the disorder in its entirety. We retrospectively investigated images obtained using single-photon emission computed tomography (SPECT), performed with ^{99m}technetium hexamethylpropyleneamine oxime, from 11 patients with TGA divided into the following groups based on the timing of SPECT: during TGA (n = 2, Group 1), at the start of the attenuation of memory impairments (n = 2, Group 2), and after TGA (n = 7, Group 3).. Regional isotope accumulation was examined using a three-dimensional stereotactic surface projection (3D-SSP) analysis. After calculating the mathematical product of the mean severity and extent ratio, unique comparable bar graphs were prepared with respect to Brodmann's areas (B). Increases in isotope accumulation were the largest in the retrosplenial cortex (B29, B30), posterior cingulate cortex (B23, B31), and precuneus (B7) in Groups 1 and 2, whereas decreases were noted in the same regions in Group 3. This is the first study to identify cerebral sites and describe changes consistent with the completion of amnesia as a symptom using an image analysis, such as a 3D-SSP analysis. We consider cortical spreading depression (CSD) to be the

etiology of TGA based on maximum isotope accumulation in the above sites and a literature review on CSD.

本論文は一過性全健忘（TGA）において、発作中および発作終了後の脳血流シンチを測定し解析することで、その発症機序として皮質拡延性抑制（CSD）が関与している可能性を示した論文であり、学術上極めて有益であり、学位論文として価値あるものと認めた。

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