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Bilateral Ageusia in a Patient with a Left Ventroposteromedial Thalamic Infarct: Cortical Localization of Taste Sensation by Statistical Parametric Mapping Analysis of PET Images

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Unilateral taste loss is usually observed on the side contralateral to a thalamic infarction, despite gustatory function being represented bilaterally. We report a rare case of bilateral taste loss in a patient with an acute left unilateral thalamic infarction, with unilateral left insular hypometabolism demonstrated by statistical parametric map analysis of PET images. Our observations suggest that the left insular cortex and left ventroposteromedial thalamic nuclei are critical to bilateral gustatory sensation.
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Key Words : Unilateral thalamic stroke, Bilateral taste loss, PET

Unilateral taste disorders occur frequently after a unilateral thalamic stroke, but there have been few reported cases of acquired bilateral ageusia in patients with a unilateral thalamic lesion.[1] We experienced a rare case of acquired bilateral ageusia in a patient with an acute left thalamic infarction, and performed statistical parametric map (SPM) analysis of PET images to evaluate the cortical dysfunction.

CASE REPORT

A 62-year-old man developed sudden dysarthria and weakness of the right upper extremity. On admission, he was alert with intact orientation. The neurological examination revealed an impaired ability to perform rapidly alternating movements in the right upper extremity, but there was no other motor, sensory, or oculomotor abnormality. Brain MRI including diffusion-weighted imaging revealed an isolated lesion involving the left ventroposteromedial (VPM) thalamic nucleus (Fig. 1). The patient also complained of marked alteration togustatory perceptions. Gustatory function was assessed using the standardized taste-strips test, and the olfactory function was tested using the Korean version of the "Sniffin' Sticks" Test II (KVSST). The patient was unable to differentiate sweet, sour, salty, and bitter tastes on both sides of the tongue, and perceived all food as tasteless. His food intake stopped immediately after the stroke, but subsequently gradually returned to normal. The patient did not complain of memory or attention problems. KVSST

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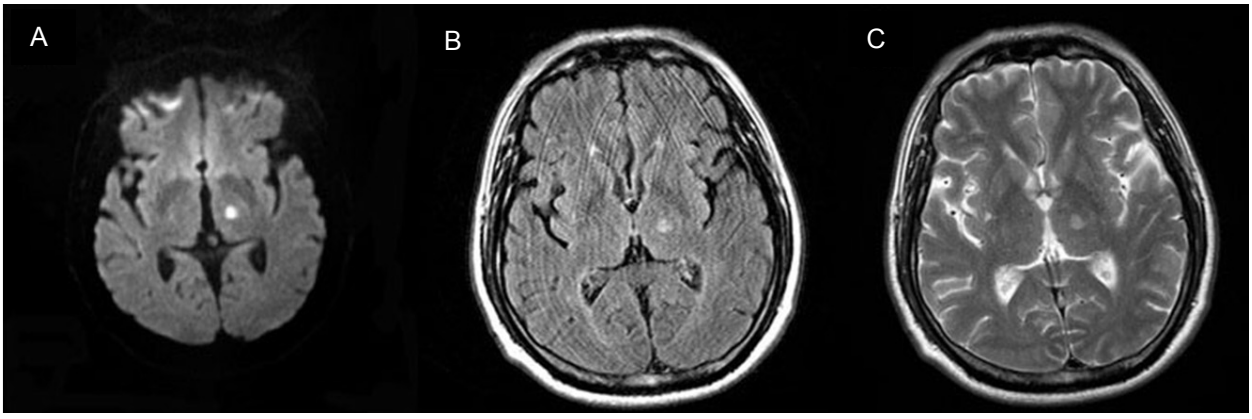

Figure 1. Diffusion-weighted (A), fast fluid-attenuated inversion-recovery (B), and T2-weighted (C) brain PET images all show an acute left thalamic infarction.

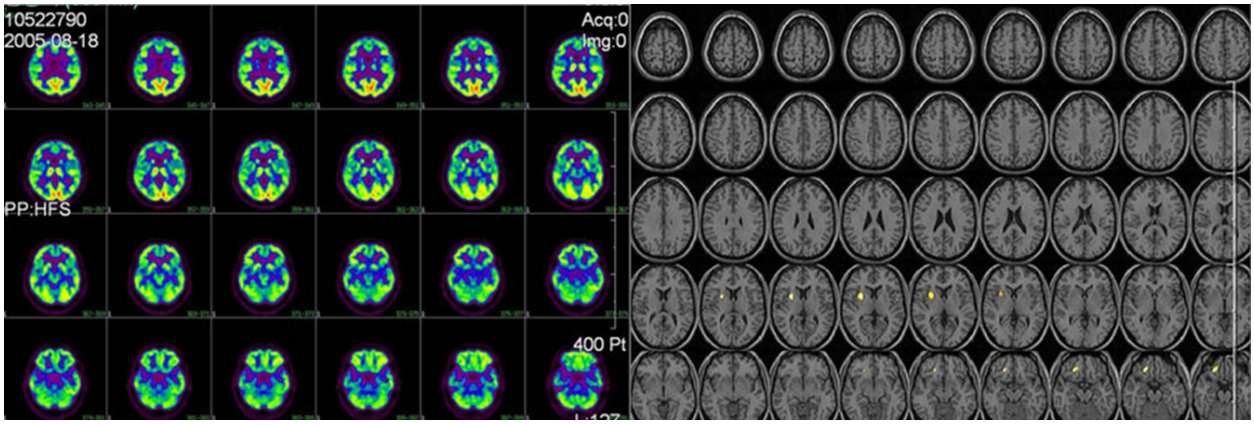

Figure 2. Brain ^{18}F-FDG PET (A) and SPM (B) images showing markedly decreased metabolism in the left frontal operculum and left insular cortex. SPM images are shown in a neurological orientation.

performed 6 months after the thalamic stroke confirmed that gustatory dysfunction was still present in the patient. ^{18}F-FDG PET imaging (ADVANCE PET scanner, GE, Milwaukee, WI, USA) data obtained from the patient and 12 age-matched normal controls was subjected to SPM analysis (Ultra-Sparc 10 workstation, Sun Microsystems, Silicon Valley, CA, USA; SPM99, Institute of Neurology, University College London, UK). All reconstructed PET images were spatially normalized into the MNI template (Montreal Neurological Institute, McGill University, Canada) using the affine transformation of the SPM99 program. The normalized images were smoothed by convolution with an isotropic Gaussian kernel with an FWHM of 8 mm to increase the signal-to-noise ratio. The images obtained from the patient were statistically compared with those of the 12 healthy volunteers on a voxel by-voxel basis using the *t*-test to detect significant decreases in the regional metabolism. Voxel thresholding at p=0.01 (uncorrected) and p=0.001 (uncorrected) was used. SPM analysis of brain PET images revealed hypometabolism in the left insular cortex (Fig. 2).

DISCUSSION

The solitary tract nucleus in the medulla is the first relay in taste sensation, which subsequently involves the central tegmental tract to the medial aspect of the VPM nucleus, immediately adjacent to the somatosensory area for the oral cavity and fingers.

Taste information is then relayed to the primary taste cortex in the frontal operculum and insular cortex, finally reaching the secondary cortical taste area in the orbitofrontal cortex.[1] From an examination of 15 cases of unilateral gustatory disturbance due to central lesions, Onoda and Ikeda suggested that the central gustatory pathways in humans ascend ipsilaterally from the solitary nucleus of the medulla oblongata to the pons, from where they cross to a higher position in the midbrain and reach the thalamus contralaterally.[2] Another clinical and MRI investigation also suggested that the central taste pathway in humans projects ipsilaterally from the solitary nucleus up to the level of the upper pontine or lower midbrain before crossing.[3] It is generally accepted that the taste pathway projects from the ipsilateral solitary nucleus in the medulla, and immediately crosses and traverses the contralateral medial lemniscus on its way to the thalamus. However, the cortical representation of gustatory fibers in the central pathway from the VPM thalamic nuclei in humans has not been elucidated. This is due to the paucity of functional imaging studies on taste disorders in patients with cortical or thalamic stroke, despite the insular cortex and thalamus having an important role in gustatory and lingual somatosensory function.

Recent investigations in stroke patients have indicated the presence of ageusia contralateral to a thalamic or corona radiata infarction, supporting that the gustatory fibers ascend contralaterally in the cerebral hemisphere and that the pathway ascends from the thalamus to the cerebral cortex via the posterior part of the corona radiata.[4] Functional imaging of the central gustatory pathway from the thalamus to the cortex has been mainly performed in nonhuman animals. An anterograde and retrograde tract-tracing study of the projections from the thalamic gustatory area in the rat showed that parvicellular VPM neurons projecting to the amygdaloid complex differed from those projecting to the insular cortex.[5] However, recent developments in functional brain mapping techniques using PET have revealed the specific structures in the human brain involved in taste sensation. A PET investigation of taste sensation in healthy humans showed that the thalamus, insular cortex, anterior cingulated gyrus, parahippocampal gyrus, lingual gyrus, caudate nucleus, and temporal gyri participated in the discrimination of a salty taste.[6] However, these studies have not elucidated the termination laterality of fibers from the thalamic gustatory relay.

The present report is one of only a few on bilateral taste loss associated with acute left unilateral thalamic infarction. Moreover, there have been few functional imaging studies on taste disorders in patients with a localized VPM thalamic infarct, which would facilitate identification of the connection between cortical and subcortical areas. We had expected our PET study to reveal bilateral cortical hypometabolism, and hence we cannot explain the exact reason for unilateral left insular hypometabolism manifesting as a bilateral taste loss. We postulate that the laterality of taste dominance is in the left hemisphere, with the right hemisphere only involved in the taste sensation of the right hemitongue. Hypometabolism in the left thalamus was evident from our PET investigation but not from the SPM analysis. We do not know the exact reason for this apparent discrepancy, but it might be attributable to the lesion being too small, even though there were definitely structural and functional impairments in that region. We believe that this case report provides useful information about the laterality of taste and the functional asymmetry of the cerebral hemisphere. Further gustatory functional imaging studies are necessary in acute stroke patients to elucidate the cortical representation and laterality of taste.

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