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Introduction

Hallucination is defined as a strong subjective perception of any object when no such situation is present. As for visual hallucination, it is thought to arise from irritative foci analogous to epileptic discharges: when the focus is occipital, brief stereotypical flashes of light or color are described by the subject; when the focus is in the temporal lobe, the subject reports complex stereotypical formed images1). According to Bender and Kanzer2), visual hallucinations have been attributed to disturbances in the parieto-occipital zones of the nondominant cerebral hemisphere. However, it is clear from the papers of Weinberger and Grant3) and Cogan4) that there are types of visual hallucinations that do not have the precise localizing value in the cerebral cortex.

Peduncular hallucinosis is of the latter type without localizing value in the cerebral cortex, and is characterized by vivid, nonstereotypical, colorful imagery that is due to mesencephalic dysfunction. Such images are usually small, quiet, wandering animals or human beings, which are criticized by the patient's consciousness. Lhermitte5) reported a case in 1922 and van Bogaert6) published a postmortem study showing a lesion in the cerebral peduncles or adjacent midbrain structures. Alajouanine7) showed such phenomena were also caused by pontine dysfunction. Thus, the term "peduncular" has been used to indicate the mesencephalon in a wide sense.

Geller and Bellur reported8) the first clinico-radiological documentation using magnetic resonance imaging (MRI) of a mesencephalic infarct. However, there have been no reports of studies using positron emission tomography. In this report, we present a case of
peduncular hallucinosis using CT, MRI and PET and discuss a possible mechanism of visual hallucination.

Case Report

An 81-year-old woman was admitted to Miyama Hospital one and a half months after the onset of cerebral infarction of the right basal ganglia. In the acute phase she suffered from stupor and consciousness disturbance and was treated with conservative therapy. Four months later, she manifested delirium at night. She described her hallucinations as follows: "A foreigner with brown wavy hair wearing a black mourning dress stares at me. She stands to my left but says nothing. I try to drive the phantom away with a folded newspaper." "A black and white spotted cat sneaks into my room and wanders around"

An interview by a psychiatrist revealed that she was polite and cooperative, without major change of her personality. Her consciousness was clear. Mild cognitive disturbance, disorientation of time and space, and emotional incontinence were observed. The Mini Mental State Examination (MMSE) and Dementia Screening Test (DST) showed mild cognitive disturbance. Her IQ as measured by WAIS (Wechsler Adult Intelligence Scale) was 76. Dementia of cerebrovascular accident origin was suspected based on DSM-III R (American Psychiatric Association, 1987), on clinical course, and on CT findings. The Hachinski ischemic score was 7.

General medical examination including laboratory tests indicated that she suffered from hypertension, anemia, and cardiomegaly with ischemic ECG changes. Findings of neurological examination was unremarkable except for left hemiplegia with intact language function. As shown by a bedside ophthalmologic examination, she had 20/40 visual acuity in both eyes and showed left homonymous hemianopia. The fundus oculi were normal.

Computed tomographic (CT) scan revealed a periventricular lucency (PVL), a low density area in the right basal ganglia compatible with her left hemiplegia (Figure 1). An MRI with a 0.14-Tesla magnet demonstrated areas of high signal intensity in the right tegmentum and in the right side of the pons on the T2-weighted image (Figure 2). PET (PT 931, USA) and the F-18-fluoro-deoxy-glucose (FDG) technique at rest showed decreased glucose metabolism in the right frontal lobe, the right basal ganglia, the right temporo-parieto-occipital region, and the bilateral inferior temporal regions (Figure 3).

With the administration of Tiapride hydrochloride (75 mg in the evening), her complaints of hallucination disappeared within 2 weeks. However, delirium at night with vascular type dementia remained.

Discussion

Highly formed hallucinosis caused by a midbrain lesion detected by MRI was revealed in a vascular type dementia patient. The patient showed mental deterioration, not
only cognitive impairment but also emotional incontinence, and delirium at night. The high Hachinski score, the presence of hypertension, the onset of dementia after stroke, and the findings on CT scanning also suggested cerebrovascular origin. As far as the diagnosis of peduncular hallucinosis was concerned, her complaints of ontinuous hallucinations lasting for hours were compatible with those of previously reported cases\textsuperscript{5,8,10} and also she had a pontine lesion. Therefore, this is a case of peduncular hallucinosis with vascular type dementia. To our knowledge, this is the first clinicoradiological documentation of peduncular hallucinosis not only using MRI to detect a mesencephalic infarct during life but also using PET to reveal low metabolism in a widespread region.

As for the mechanism of visual hallucination, Cogan\textsuperscript{4} posits two separate systems, i.e., "irritation" and "release." The "irritative" type have the localizing value as described in the introduction. As for the "release" type, Cogan agrees with West's theory\textsuperscript{11} that the brain is constantly being stimulated by excessive sensory input that is normally excluded from the consciousness which selectively acts to keep out unwanted or unneeded input. When the usual information input level no longer suffices to inhibit this emergence, the "precepts or memory traces" may be released as hallucinations. Adapting this theory to the visual system, it would appear that removal of visual stimuli by cerebral lesions appears to lessen the normal inhibition for visual functions that then released. Mesencephalic dysfunction may be considered to lessen the inhibition system leading to a release of memory traces resulting in peduncular hallucinosis.

Hallucination and delirium are false recognition without true objects: the former is criticised by the patient's consciousness but the latter is not. As far as her clinical course is concerned, hallucinations completely disappeared after administration of Tiapride hydrochloride (D2-antagonist)\textsuperscript{12} while the delirium at night remained. What is a possible mechanism explaining this phenomena? Lhermitte emphasized the relationship between peduncular hallucinosis and the sleep mechanism. To explaining the relationship among sleep, delirium, and hallucinosis, two major systems must be considered. One is the system which clears the consciousness and the other is the system which induces sleep\textsuperscript{13}. The former is called the ascending activating system and contains at least two subsystems: the adrenergic system (the locus ceruleus) and the dopaminergic system. The latter is called the ascending inhibiting system (the dorsal raphe nuclei, serotonin). The dynamic balance between these two ascending systems may determine the clinical level of alertness and play a pivotal role in the relationships among sleep, delirium, and hallucination. Namely, in the alert state, the activating system is much more-dominant than the inhibiting system; in the sleep state, the inhibiting system is more dominant than the activating system; in the delirium state, these two systems are equally dominant; in delirium accompanying the hallucination state, the activating system is slightly dominant. Tiapride hydrochloride may repress the activating
system in the patient suffering delirium with hallucination, resulting in a state in which the patient suffers only delirium. However, it remains still controversial.

Summary

A patient suffering peduncular hallucinosis with vascular type dementia is described. Magnetic resonance imaging revealed an infarction in the region ranging from the right upper ventral part of the pons to the right midbrain tegmentum. Positron emission tomography and the 18-F-fluorodeoxyglucose method demonstrated decreased glucose metabolism in the right frontal lobe, the right basal ganglia, the right temporo-parieto-occipital region, and the bilateral inferior temporal regions. This is the first clinicoradiological documentation of peduncular hallucinosis using MRI and PET. The mechanism of hallucination and delirium is discussed.

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References

5) Lhermitte J.: Confin Neurol. 9 (1949) 43.