

SHORT REPORT

Thalamic experiential hallucinosis

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Abstract

Two patients with an infarct limited to the thalamus developed auditory and visual experiential hallucinations. Neuropathological studies in one patient showed a small cavity in the right intralaminar nuclei surrounded by focal spongiform change, partly involving the right dorsomedial nucleus. Neuro-radiological data in another patient indicated that the same nuclei in the left thalamus were also affected. It was concluded that a unilateral thalamic lesion could cause experiential hallucinations and the intralaminar and dorsomedial nuclei might be important structures to explain the phenomenon.

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Hallucinations related to memory were reported in temporal lobe epilepsy and were called experiential hallucinations by Penfield and Perot.¹ Hallucinations have been reported occasionally in association with thalamic infarction,^{2,3} haemorrhage,⁴ and degeneration,⁵ but experiential phenomena have been rarely described. We report data on two patients with a unilateral infarct confined to the thalamus who developed auditory and visual experiential hallucinations.

Case reports**CASE 1**

A 72-year-old right-handed man suffered sudden attacks of somnolence during daylight hours six months after the end of chemotherapy for lung cancer. He was treated with 60 mg morphine sulphate per day for four weeks to relieve pain in the chest. On examination, three days after onset, his pulse was 80 and regular, and his blood pressure was 120/70 mm Hg; he was afebrile. General examination was unremarkable. He was oriented to time, place, and persons, and was cooperative. When left alone, he soon lapsed into a somnolent state. He could be awakened quickly by light stimuli. Nocturnal insomnia was present. The nervous system was otherwise normal, including visual acuity and visual fields. The daytime somnolence and nocturnal insomnia continued for one week after onset. During this time, he reported a variety of auditory and visual hal-

lucinations that consisted of vivid recollections of his old jobs. He described transient episodes in which he saw and dyed clothes, talked of town affairs with his friends, and obeyed his employer's instructions. He had experienced similar events about 50 years ago in his first job. Other scenes were related to his last job. He felt that each episode lasted about five minutes and was convinced the hallucinations were real while they were happening. The hallucinations arose involuntarily in objective space. They were normally coloured and encompassed the whole visual field. There were no diurnal fluctuations in the appearance of the hallucinations.

The following laboratory investigations and analysis of CSF were normal: CBC, routine blood chemistry, antinuclear antibody, VDRL, triglycerides, cholesterol, vitamin B₁₂, T₄ and T₃ resin uptake, thyroid-stimulating hormone (TSH), and arterial blood gases. An EEG when the patient was hallucinating demonstrated diffuse slow waves at 5 to 7 Hz with bifrontal intermittent delta waves. An EEG when the spells were not occurring showed similar findings.

CT three days after onset showed a small, low density area in the central part of the right thalamus. CT 14 days after onset showed no lesion, but one month after onset the small, low density area reappeared in the same position, indicating a CT fogging effect.⁶

The hallucinations slowly resolved over a two week period, although morphine sulphate (60 mg/day) was continued. At the end of this time, hallucinations were shorter and simpler. He frequently reported that he saw a dish of sweet potatoes on the table near his bedside or a cigarette between his fingers: they disappeared in a few seconds. He had often eaten sweet potatoes one month before the occurrence of hallucinosis and was a heavy smoker. These hallucinations developed when he was sitting on a chair or resting in bed without falling asleep. He was left without memory impairment or any other neurological deficits.

Two months later, the patient died. Postmortem examination disclosed widespread carcinomatosis with involvement of both lungs, mediastinal nodes, liver, and left kidney. Small polypoidal growths were found on the mitral valve. The tumour histology revealed squamous carcinoma.

The brain showed mild atherosclerosis. It was fixed in formalin and sliced serially.

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Figure 1 Coronal section just posterior to massa intermedia shows a small and linear infarct in right thalamus (arrow). One division of scale = 5 mm.

An isolated macroscopic finding was a small cavity in the right thalamus (fig 1). Abnormal microscopic findings were also limited to the right thalamus. The cavity was situated in the intralaminar (IL) nuclei and was surrounded by spongiform change mainly involving the lateral part of the right dorso-medial (DM) nucleus. A small number of nerve cells were preserved and reactive gliosis was present in the focal spongy tissue. The classification of thalamic nuclei was based on that of Carpenter.⁷

CASE 2

A 46-year-old right-handed woman suddenly developed memory impairment. She was not taking any medications. On examination, three days after onset, her pulse was 72 and regular and blood pressure was 152/88 mm Hg; she was afebrile. General examination was unremarkable. She was alert and cooperative. Mild impairment of recent memory and mild weakness in the right upper extremity were present. The nervous system was otherwise normal, including visual acuity and visual fields. On admission, four days after onset, she reported a variety of vivid

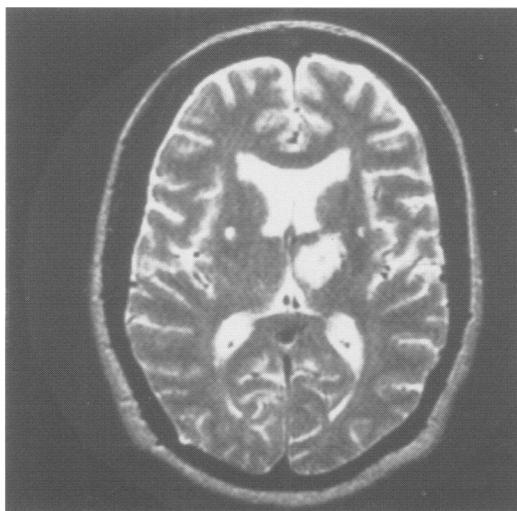


Figure 2 Transverse T2 weighted MRI shows a high signal intensity lesion in anterior and middle parts of left thalamus.

auditory and visual hallucinations. She described the events that had occurred at home a day before admission. When she was preparing for the hospital, her parents appeared and helped her, asking about her illness. They suddenly disappeared when she recognised that they had died 10 years ago. After admission, the occurrence of hallucinations continued. When she was sitting on a chair or resting in bed without falling asleep, she frequently experienced hallucinations. Three days after admission, her friends brought a box of sweet cakes, and she was advised not to eat sweets for control of her diabetes mellitus. About 30 minutes after hearing the advice, she experienced opening the box and eating the cakes but soon realised she had done no such thing when she heard a voice warning "don't eat." She felt that each hallucination lasted three to five minutes and was convinced the hallucinations were real while they were happening. The hallucinations arose involuntarily in objective space. They were normally coloured and encompassed the whole visual field. There were no diurnal fluctuations in their appearance.

The following laboratory investigations were normal except for mild hyperglycaemia: CBC, routine blood chemistry, antinuclear antibody, VDRL, triglycerides, cholesterol, vitamin B₁₂, T₄ and T₃ resin uptake, TSH, and arterial blood gases. An EEG taken when she was not hallucinating was normal.

CT, four days after onset, revealed an isolated lucency in the anterior part of the left thalamus. Contrast CT 14 days after onset showed enhancement in the same area. MRI scan using a 1.5 Tesla source demonstrated an area of hypodense signal intensity on the T₁ weighted phase in the anterior part of the left thalamus. A wider area of hyperdense signal intensity on the T₂ weighted phase was shown, involving the anterior and middle parts of the left thalamus (fig 2).

The hallucinations slowly resolved over a two-week period. At the end of the second week, the hallucinations were shorter and simpler. She often saw a teapot and several teacups on the table near her bedside, which disappeared in several seconds. They were the same teapot and teacups that she used at home. She was left with mild impairment of recent memory.

Discussion

The prominent clinical feature in our patients was the auditory and visual experiential hallucinations. Both neuroradiological findings, CT fogging effect in patient 1 and contrast enhancement in patient 2, were consistent with recent thalamic infarction.^{6,8} In patient 1, morphine sulphate might have had a role in causing hallucinations. However, it seems less likely because they disappeared although the drug was continued. Our data indicate that a unilateral thalamic lesion produces auditory and visual experiential hallucinations.

Feinberg and associates² reported data on a

patient with vivid visual hallucinations and a right paramedian thalamic infarct. Catafau and colleagues³ described data on one patient with complex visual hallucinations and a posterior thalamic infarct. Without giving anatomical details, Fisher⁴ reported data on five patients with unilateral thalamic haemorrhage who developed visual hallucinations. The rare disease of primary thalamic degeneration was frequently associated with hallucinations.⁵ Although these reports support the notion that thalamic lesions can evoke hallucinations, the authors did not document experiential phenomena.

Neuropathological studies in patient 1 demonstrated that the infarct involved the right IL nuclei. The DM nucleus was also affected by a focal spongiform change that seemed to be incomplete infarction. CT and T₁ weighted MRI of patient 2 showed that the infarction was in the region of the left tuberothalamic artery⁹ and based on the atlas of Daniels and coworkers,¹⁰ the left IL nuclei were involved by the infarct. In addition, the left DM nucleus was affected possibly by oedema because the high signal intensity lesion on T₂ weighted phase in the medial part of the left thalamus is not usually supplied by the tuberothalamic artery.⁹ These findings indicated that the IL and DM nuclei were involved in both patients.

Few pathoanatomical studies have focused on the development of thalamic hallucinosis. Catafau and colleagues³ suggested from MRI studies that thalamic hallucinations might be caused by a lesion of the thalamic reticular nucleus, which is related to the reticular activating system.¹¹ In our patients, the lesions of the IL nuclei may be responsible for the experiential hallucinations. The IL nuclei also receive rostral projections from the reticular activating system and project to wide regions of the cerebral cortex, including the temporal and limbic lobes, important in memory function.¹¹

Another possibility is that activation of the DM nucleus may account for the phenomena. Although it remains uncertain which thalamic structures are critical for memory, the DM nucleus still seems to be important.¹² In patient 1, the DM nucleus had focal spongiform change and in patient 2, the nucleus might be oedematous. A spongy state and oedema are rarely seen around a lacunar infarct.¹³ We speculate that these unusual histological changes in the DM nucleus may act as irritative foci, provoking the experiential phenomena, although epileptiform discharges on EEG were absent in our patients.

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