Hemorrhagic Pituitary Adenoma Manifesting as Transient Global Amnesia

—Case Report—

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Abstract

A 68-year-old female presented with recurrent transient global amnesia due to hemorrhagic prolactin-producing pituitary adenoma. Magnetic resonance imaging clearly revealed the anatomical relationship between the hematoma, within the parasellar tumor, and the compressed medial temporal lobe of the right (non-dominant) hemisphere. Within 4 weeks after the start of bromocriptin administration, the adenoma had markedly regressed and the affected temporal lobe was decompressed. She has experienced no further episode of transient global amnesia. Magnetic resonance imaging is recommended to detect latent organic lesions responsible for transient global amnesia, although the disorder is considered to be benign and of functional origin.

Key words: transient global amnesia, pituitary adenoma, prolactinoma, pituitary apoplexy, hippocampus, magnetic resonance imaging

Introduction

Transient global amnesia (TGA) is a clinical entity characterized by an isolated, sudden, and transient episode of amnesia unaccompanied by seizure or other major neurological symptoms, with an uneventful follow-up course in the vast majority of cases.^{2,4)} Although the exact pathophysiology of TGA remains unclear, it may be a transitory syndrome of diverse etiology. Transient ischemia in the medial temporal lobe and diencephalic structures fed by the vertebrobasilar system, rather than epileptic discharges occurring within these deep structures,⁴⁾ seems to be the most probable underlying mechanism.⁹⁾ Studies of cerebral blood flow and energy metabolism using positron and single photon emission computed tomographies have disclosed decreased blood flow and hypometabolism in the uni- or bilateral temporal and frontal lobes,^{1,11,14)} whereas neuroimaging findings have rarely implicated organic lesions in the mechanism of TGA.^{3,7,10,13)}

We describe a patient who suffered TGA due to a hemorrhagic pituitary adenoma with suprasellar ex-

tension.

Case Report

A 68-year-old, exclusively right-handed female with a previous history of hypertension developed a transient amnesic state during her visit to a local orthopedics clinic for finger joint pain on December 18, 1993. She suddenly became disoriented as to place, time, and circumstances, and repeated the same simple questions concerning these matters. She was awake but looked confused, although she manifested no convulsion, motor weakness, aphasia, or conscious disturbance. She could remember her personal identity and could carry on apparently normal activities. The amnesic episode lasted about 30 minutes, after which she became oriented, but could not recall the events that had occurred during the episode. She said that she did not have any mental or somatic abnormalities after the episode, and she had been uneventful until she experienced a second episode of memory disturbance with the same features on December 28, 1993, when some of her friends visited her home as previously arranged. She met her friends at the door, but at that time she could not recall the reasons for their visit. She repeatedly

Fig. 1 T₁-weighted MR images demonstrating a hematoma (arrowhead) within a parasellar pituitary adenoma compressing the right medial temporal lobe, and displacing the optic chiasma (OC) and pituitary stalk (PS) (upper row). The tumor and the hematoma were markedly reduced 4 weeks after the start of bromocriptin administration, the medial temporal lobe was decompressed, and a space (arrow) had appeared between the tumor and the lobe (lower row). PG: pituitary gland.

asked them why they were there. Thirty minutes after the visitors left her home, she became aware, but did not remember that she had received the visitors or accepted their gift.

Two months later she was admitted to our hospital. She was oriented and no neurological abnormalities were noted. However, computed tomography (CT) disclosed a parasellar mass. Magnetic resonance (MR) imaging demonstrated a hematoma in the chronic stage, measuring $2 \times 3 \times 2$ cm, located within the mass, compressing the right medial temporal lobe, and extending from the sella turcica (Fig. 1 upper row). Hormone examination disclosed marked elevation of serum prolactin level (920 ng/ml). The lesion was diagnosed as a hemor-

rhagic prolactin-producing pituitary adenoma (prolactinoma) with supra- and parasellar extension. She had not experienced any symptoms suggesting pituitary apoplexy. The tumor had also displaced the optic chiasma superiorly, but the results of ophthalmological examination were nearly normal.

She refused surgical treatment, and bromocriptin (7.5 mg/day) was administered. Her prolactin level decreased rapidly and entered the normal range (5.3 ng/ml) 7 days after the initiation of the treatment. Hematoma absorption and tumor regression resulted in a space between the tumor and the medial temporal lobe appearing on the serial MR images (Fig. 1 lower row). She has since been free from TGA for almost 2 years.

Discussion

TGA is generally considered a benign and functional disorder and is seldom caused by or associated with intracranial mass lesions. The two amnesic episodes in our patient were characteristic of typical TGA, despite their relatively short duration, allowing easy differential diagnosis from temporal lobe seizure, hysterical fugue state, temporal lobe encephalitis, and migraine. Although organic lesions such as glioma, hemorrhage, and meningioma are occasionally described as possible etiologies of TGA,^{2,3,7,10,13} an anatomical relationship has very seldom been documented in detail using MR imaging.¹⁴ Dysfunction of the temporodiencephalic structures either on the dominant or both sides due to tumor involvement was usually presumed to be the mechanism of TGA. In the present case, MR imaging definitively disclosed that the tumor, which might have expanded rapidly due to spontaneous hemorrhage, had compressed the right (non-dominant) medial temporal lobe at the parahippocampal gyrus and uncus.

Recent MR imaging studies have disclosed that asymptomatic or subclinical pituitary apoplexy (more appropriately termed hemorrhagic pituitary adenoma) is not as uncommon as believed previously.^{5,6,12} The markedly high serum prolactin level and MR imaging findings in our patient showed the entity could only be prolactin-producing adenoma, although histological confirmation was not obtained. Involvement of the medial temporal lobe due to pituitary adenoma with parasellar extension is not particularly rare,⁸ but only one case of TGA accompanied by pituitary adenoma has previously been reported, in which the exact anatomical relationship was not confirmed using neuroimaging.⁷ TGA presenting with hemorrhagic pituitary adenoma or pituitary apoplexy has not previously been described. We

speculate that the acute compression due to bleeding occurring within the suprasellar component of the adenoma might have affected the neuronal pathway in the hippocampus related to the memory process. This idea is supported by the almost simultaneous occurrence of the TGA episodes with the estimated time of hemorrhage based on the MR images, and by the absence of TGA episodes after decompression of the temporal lobe following bromocriptin administration. However, it is hard to explain why TGA was associated with involvement of the non-dominant hippocampus.^{3,13)}

TGA is not always a benign disorder. We stress the necessity for neuroimaging examination including CT and MR imaging for the detection of latent organic lesions which may be related to the mechanism of TGA. In particular, coronal MR images may disclose the anatomical relationship between the deep temporal structures and the responsible lesion.

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