Hyperintense Putamen on T1-Weighted MR Images in a Case of Chorea with Hyperglycemia

Chiyoko Nagai, Takeo Kato, Tadashi Katagiri, and Hideo Sasaki

Summary: A 77-year-old woman had an acute onset of chorea after hyperglycemic coma. She had no family history of neurologic disorders. Although brain CT showed no detectable lesions in the putamen, MR revealed a high intensity on T1-weighted images and a low intensity on T2-weighted images in both putamina.

Index terms: Chorea; Hyperglycemia; Brain, magnetic resonance

Basal ganglia lesions may show high signal intensity on T1-weighted magnetic resonance (MR) images (1–7). In some cases there are lesions only in the globus pallidus (5), but in others there are lesions in additional areas as well (1–4, 6, 7). In this paper, chorea after hyperglycemic coma in a patient who had high- and low-signal-intensity lesions on T1- and T2-weighted images, respectively, in both putamina is presented.

Case Report

The patient was a 77-year-old woman who had been treated for hypertension and renal dysfunction for 20 years. In January 1991, she had thirst and polyuria followed by coma with hyperglycemia (blood glucose, 633 mg/dL). There was also mild hyponatremia (126 mEq/L) at that time. Five days later when her coma, hyperglycemia, and hyponatremia had been corrected, choreic movements similar to those observed in Huntington disease appeared initially on her right arm and spread to the entire body. After drug therapy (tiapride, sulpiride, phenobarbital, and haloperidol) the choreic movements disappeared, but they reappeared when therapy was discontinued. Haloperidol was found to be most effective against her abnormal movements.

Brain MR examination (in April 1991) was performed on a 1.5-T unit (Magnetom, Siemens, Erlangen, Germany), and revealed an irregular high signal intensity on T1-weighted images (Fig 1A) and a low signal intensity on T2-weighted images (Fig 1B) in the putamina. There were ischemic-appearing lesions in the anterior cingulate gyrus and corpus callosum (Fig 1B). We observed an intense lesion in the central pons on T2-weighted images (Fig 1C). On brain computed tomography (CT) the putamen did not show any abnormal density (Fig 1D).

Blood examination showed anemia (hemoglobin, 9.5 g/dL), mild renal dysfunction (blood urea nitrogen, 28 mg/dL; creatinine, 1.3 mg/dL), hyperkalemia (5.9 mEq/L), hyperchloridemia (114 mEq/L), hypocalcemia (8.3 mg/dL), an increased serum level of parathormone (parathyroid hormone, 629.5 pg/mL), and a decreased serum level of manganese (0.3 μg/dL). Plasma amino acid analysis disclosed a marked decrease (trace) in monoethanolamine. The liver function tests and the serum level of creatinine phosphokinase were within normal limits. No acanthocytosis of erythrocytes was observed in the peripheral blood.

Seven months after onset, the patient was examined by positron emission tomography and found to have a normal glucose uptake in both putamina. Eleven months after onset, MR showed normal intensity of the putamina on T1-weighted images (Fig 2A), although the intensity remained low on T2-weighted images (Fig 2B). The pontine lesion was reduced in size (Fig 2C).

Discussion

The patient had no family history of neurologic diseases, including movement disorders. The choreic movements were of acute onset, and no personality changes were recognized. On CT and MR imaging, neither atrophy of the caudate nucleus nor dilatation of the lateral ventricles were observed. It is therefore unlikely that the patient had Huntington disease. It is also unlikely that she had chorea-acanthocytosis because she had no tongue-biting or peripheral neuropathy, a normal serum creatine phosphokinase, and no acanthocytosis of erythrocytes in the peripheral blood. Her manifestations are not suggestive of rheumatism-associated chorea because of the age of onset;

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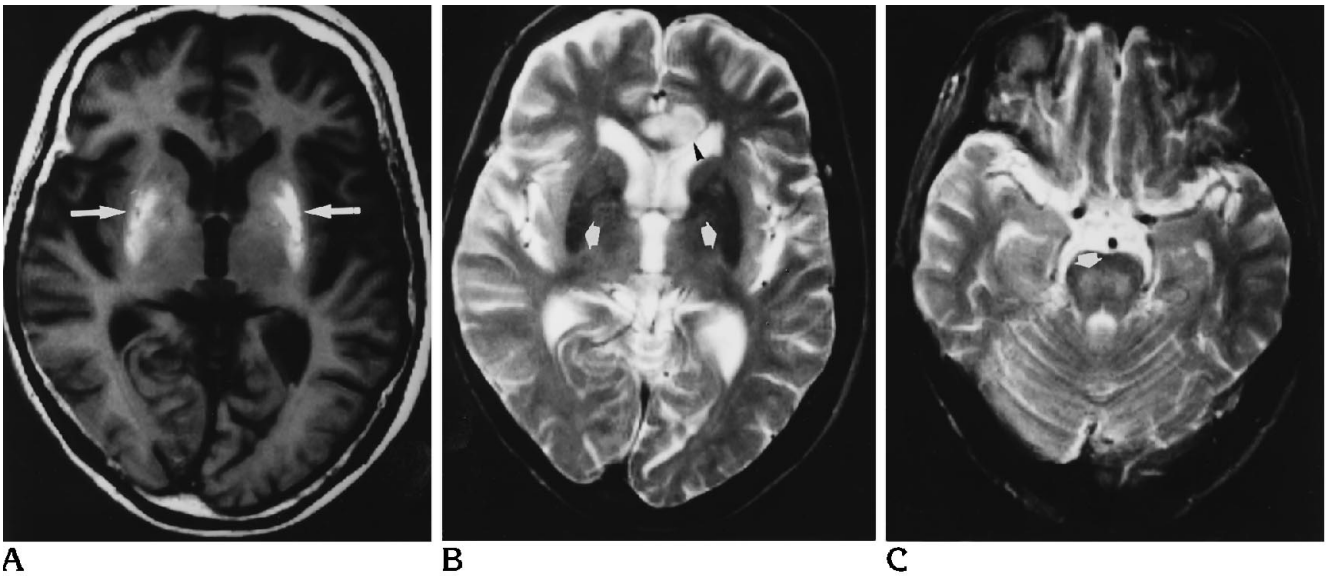


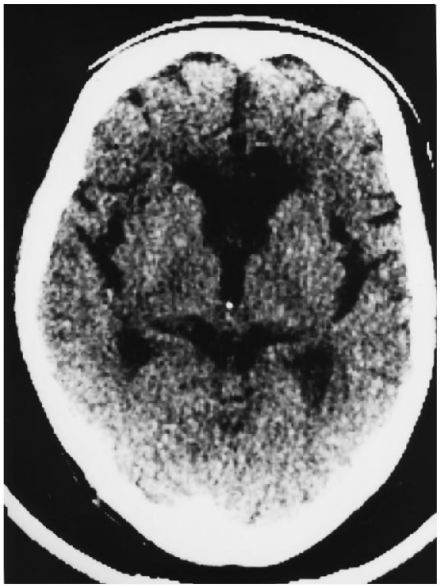
Fig 1. CT and MR images, 3 months after onset.

A, T1-weighted MR image (500/15/3 [repetition time/echo time/excitations]) reveals an irregular high signal intensity restricted to the putamina (*arrows*).

B, T2-weighted MR image (2500/90/1) shows a low signal intensity in the putamina (*arrows*). There are ischemic lesions in the anterior cingulate gyrus and corpus callosum (*arrowhead*).

C, T2-weighted MR image (2500/90/1) shows a high signal intensity lesion in the central pons (*arrow*).

D, On CT scan there is no detectable lesion including calcification in the putamina.



the absence of fever, arthralgia or cardiac problems; and the MR findings (8). Because the choreic movements appeared shortly after a bout of hyperglycemic coma, it seems most likely that the patient's symptoms developed secondary to hyperglycemia; ie, chorea caused by hyperglycemic encephalopathy (9).

We reviewed reported cases with high signal intensity in the basal ganglia on T1-weighted MR images (1–7). Cases reported by Dell et al (1) and Henkelman et al (7) showed a high intensity on T1-weighted images and a low intensity on T2-weighted images in the putamen. However, these cases apparently are different from the present case because (*a*) the distribution of their lesions was more widespread, not only in the putamen but also in the caudate nucleus, globus pallidus, thalamus, cerebral white matter, and so forth, (*b*) their lesions were of high-density on brain CT, indicating calcification, whereas our case showed a normal density in the putamen, and (*c*) their underlying diseases were pseudohypoparathyroidism, hypoparathyroidism, or ferrocalcinosis.

Because the present case had mild hyponatremia and it was corrected thereafter, the hyperintense lesion in the pons on T2-weighted images (Fig 1C) may be osmotic myelinolysis

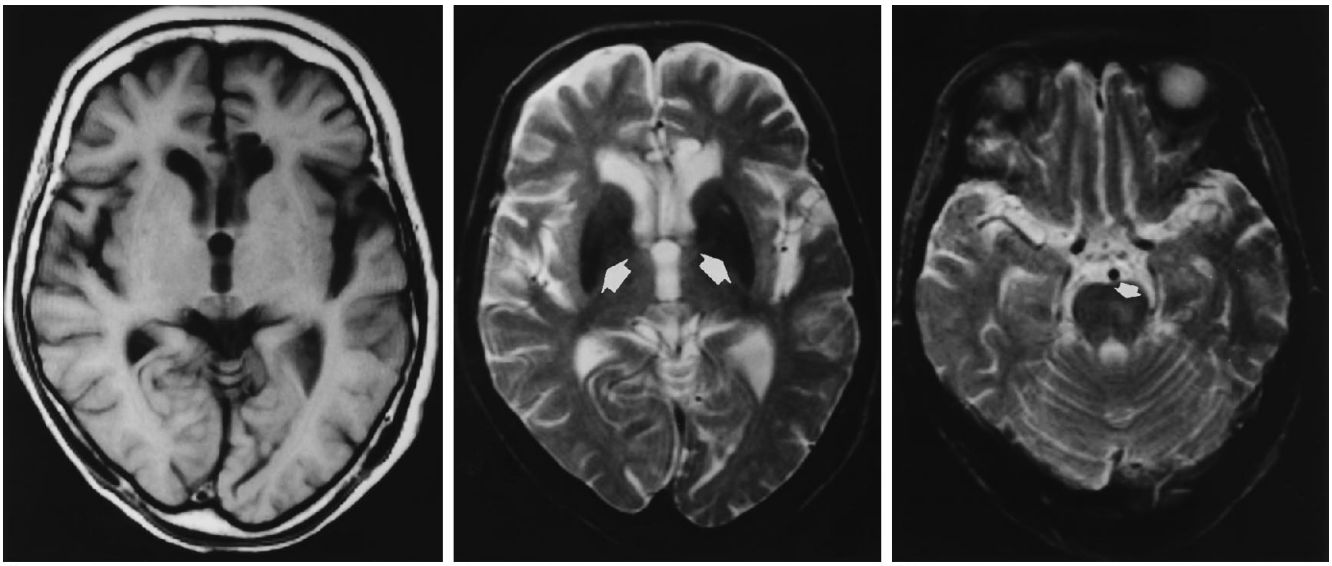


Fig 2. MR images, 11 months after onset.
A, T1-weighted MR image (500/15/2). The hyperintense putamen becomes normal in intensity.
B, T2-weighted MR image (2500/90/1). The signal intensity remains low in the putamina (*arrows*).
C, T2-weighted MR image (2500/90/1). The pontine lesion is reduced in size (*arrow*).

(central pontine myelinolysis). Ho et al reported a case of central pontine and extrapontine myelinolysis in which the basal ganglia showed a transient hyperintensity on T1-weighted images and a normal intensity on T2-weighted images during a recovery period of the extrapontine myelinolysis (10). The degree of the hyperintensity of the basal ganglia, however, was mild compared with that of our case. In addition, unlike in their case, the putamen of our case showed low intensity on T2-weighted images. Moreover, the basal gangliar lesion of their case included both the putamen and globus pallidus, whereas the hyperintense lesion on T1-weighted images was restricted to the putamen in the present case. Although we do incline toward the possibility that myelin destruction also occurred in the putamen in our case, the MR features are different from those of usual extrapontine myelinolysis.

Recently, three patients were reported with acute onset of hemichorea/hemiballism, who had a high signal intensity on T1-weighted images in the contralateral putamen (Nozaki U et al, "A Case of Hemichorea-Hemiballism with Hyperintense Putamina on T1-Weighted MR Images," *Rinsho Shinkeigaku* 1993;33:793 [abstract]; Yahikozawa H et al, 34th Annual Meeting of the Japanese Society of Neurology, 1993 [abstract]). All three patients had hyperglycemia at the onset of the abnormal involuntary movements. Hemorrhage and calcification in the putamen were ruled out by CT and MR examination. It seems, therefore, that such MR changes may occur in some hyperglycemic patients. The underlying mechanism for the bright signal in the putamen on T1-weighted images is still a matter of speculation. Because many patients with hyperglycemia show myelin destruction in the peripheral nerves (diabetic polyneuropathy), one possible explanation is that in the putamen "an unwrapping of the myelin sheath may occur that would preferentially admix the myelin-bound water with that of axonal free water and result in a shortened T1" (10).

With respect to the pathologic mechanism for choreic movements, we speculate that the patient was in a hyperactive dopaminergic state. The efficacy of haloperidol, which decreases the dopaminergic activity, on the patient's choreic movements supports this idea. Because γ-aminobutyric acid (GABA)ergic neurons in the striatonigral system inhibit dopaminergic neurons in the nigrostriatal system, the impairment of GABAergic neurons in the putamen may produce hyperactivation of dopaminergic neurons, resulting in choreic movements. A decrease in the activity of cholinergic neurons relative to the dopaminergic activity in the striatum also may cause choreic movements. However, if almost

all neurons are damaged in the striatum, choreic movements disappear and are replaced by dystonia and an akinetic rigid state, which are often observed at the advanced stage of Huntington disease. Therefore, in the present case, not all neurons are impaired in the putamen: GABAergic or cholinergic neurons, or both, may preferentially be affected in the putamen.

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