

Case Report

Hemichorea in a diabetes mellitus patient following acute ischemic stroke with changes in regional cerebral blood flow

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Abstract

It is not unusual to observe hemichorea in patients with diabetes mellitus, with origins attributable to recent ischemia. Our patient was a 66-year-old female with diabetes mellitus who suddenly developed right hemichorea, mild muscle weakness of the right upper extremity, ideational apraxia, and acalculia. Her blood glucose was 600 mg/dL, and HbA1c was 13.3%. After the patient underwent head magnetic resonance imaging (MRI), a new cerebral infarction was observed in the left frontal lobe, and treatment was started with edaravone and cilostazol. At the same time, insulin treatment was also started for hyperglycemia. The acalculia and ideational apraxia improved approximately 1 week after treatment initiated, and the hemichorea also decreased. ECD-SPECT was performed on admission, and it was observed that blood flow was decreased in the left frontal lobe and striatum, but increased in the thalamus; two weeks later on follow-up ECD-SPECT, blood flow had increased slightly in the left forebrain and striatum, while it had decreased slightly in the thalamus. This suggests that the cause of hemichorea was related to ischemia. When the activity of the pallidum is impaired, it is presumed that the inhibitory activity towards the thalamus weakens and the thalamic cells become over-excited, causing chorea.

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Keywords: diabetes mellitus; hemichorea; cerebral infarction; regional cerebral blood flow; ECD-SPECT

1. Introduction

It is well-known that involuntary movements can accompany diabetes mellitus (DM)^{1,2} and metabolic disorders of the subthalamic nucleus and/or striatum. Ischemia is generally considered responsible for this phenomenon.³ However, to the best of our knowledge, there have been no previous reports of diabetic patients exhibiting chorea accompanied by changes in regional cerebral blood flow (rCBF). We herein describe a case of DM in which hemichorea was triggered by cerebral infarction and remission was achieved with treatment. This

patient's course of disease and treatment supported the notion that ischemia was involved. In the present case, performance of single photon emission computed tomography using the ^{99m}Tc-ECD Patlak Plot method (ECD-SPECT)⁴ in the acute phase revealed decreased blood flow in the left striatum and increased blood flow in the left thalamus; a repeat ECD-SPECT study performed 2 weeks later revealed that both involved areas were smaller than they were on the first ECD-SPECT study. To the best of our knowledge, this is the first report of a diabetic patient exhibiting chorea with changes in rCBF in the acute and postacute phases.

2. Case report

The patient was a 66-year-old female who had no history of cigarette smoking or alcohol drinking. Her past medical history included hypertension, hyperlipidemia, and DM discovered several years before this presentation, all of which the

Conflicts of interest: The authors declare that there are no conflicts of interest related to the subject matter or materials discussed in this article.

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patient had ignored and which had therefore remained untreated. On September 11, 2010, abnormal behaviors were noted, including taking a bath with her undergarments on and becoming taciturn, and development of involuntary movements of the right hand and foot. A family member observed the aberrant behavior for a few days, but the symptoms did not improve. The patient and her husband consulted members of our department and she was hospitalized on September 14, 2010 (on day 4).

On physical examination, the patient was slightly obese, with a height of 154 cm and a weight of 66.2 kg. Her temperature was 35.8°C, blood pressure was 208/108 mmHg, and her pulse rate was 94/minute and regular. No abnormalities were noted on general physical examination. Her consciousness was clear, but detailed neurological examination revealed ideational apraxia and acalculia. Slight muscle weakness of the right upper extremity was noted. There were no abnormalities of tendon reflexes, coordinated movements, the sensory system, or ambulation. Hemichorea involving the right upper and lower extremities was observed. Blood cell counts were within the normal range. Total cholesterol was 231 mg/dL, the triglyceride level was 168 mg/dL, blood glucose was 600 mg/dL, and the HbA1c was 13.3%. Urinalysis results included protein (–), glucose (+++), and ketones (–). On head magnetic resonance imaging (MRI) on admission (on day 4), a recent cerebral infarction was observed in the left frontal lobe. A high signal intensity lesion was observed in the left frontal lobe on T2-weighted imaging (T2WI) (Fig. 1B), fluid-attenuated inversion recovery (FLAIR) (Fig. 1C), and diffusion-weighted imaging (Fig. 1D); a low signal intensity

lesion was seen on apparent diffusion coefficient (ADC)-map imaging (Fig. 1F). Moreover, the left striatum showed a high signal intensity on T1-weighted imaging (T1WI) (Fig. 1A) and a low signal intensity on T2WI (Fig. 1B), and there was a mild low signal intensity region on T2* (Fig. 1E). On magnetic resonance angiography (MRA) performed on day 4, an oozing-like intensity area from the left middle cerebral artery was observed. This was a region of medium intensity having an irregular border (Fig. 2). ECD-SPECT was performed on day 4 and showed decreased cerebral blood flow not only in the left frontal lobe but also in the left striatum (Fig. 3). Based on these results, a diagnosis of acute-phase cerebral infarction was made, and treatment with edaravone (60 mg per day, intravenous, from day 4 till day 14) and cilostazol (200 mg per day, oral administration) was started. At the same time, insulin treatment for hyperglycemia was also started. The acalculia and ideational apraxia improved approximately 1 week after treatment had begun, and the hemichorea also decreased. Two weeks after onset of symptoms (on day 18), when the hemichorea had almost disappeared, a second ECD-SPECT was performed; this revealed increased blood flow in the forebrain and striatum, as well as reduced blood flow in the thalamus as compared with the initial examination (Fig. 3).

3. Discussion

Suggested causes of chorea in DM patients include metabolic disorders caused by hyperglycemia,² as well as ischemia^{5,6} and bleeding⁷ in the striatum. There are several reports mentioning the involvement of cerebral ischemia.

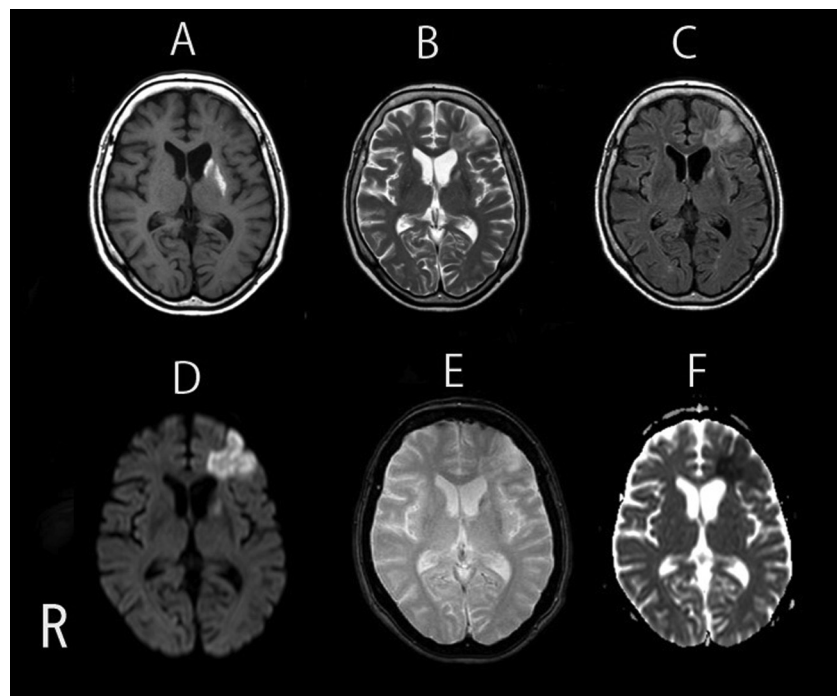


Fig. 1. Admission head MRI: (A) T1-weighted imaging (T1WI); (B) T2-weighted imaging (T2WI); (C) fluid-attenuated inversion recovery (FLAIR); (D) diffusion-weighted imaging; (E) T2* imaging; (F) apparent diffusion coefficient (ADC)-map imaging. The MRI findings are interpreted as: (a) old left striatal lesion with negative pressure effect; (b) hyperglycemic effect on the left striatum (high signal intensity in T1WI, nearly normal signal change in T2WI) (Figs. A and B); (c) acute infarction in the left frontal lobe (Figs. D and F).

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