LETTERS TO THE EDITOR

To the Editor

Recurrence of Hyperglycemic-induced Chorea-ballismus after Haloperidol Withdrawal

Most cases of hyperglycemic-induced hemichorea-hemiballismus (HIHH) are completely resolved within several days by blood glucose control and/or small doses of dopamine receptor blocking agents although a few cases have persisted for several months.[1] The recurrence of HIHH has been rarely reported, however, the development with acute haloperidol withdrawal has not been previously reported.

Case

An 80-year-old man who had poorly controlled diabetes mellitus for 26 years was admitted because of involuntary movement on his left side. The movement had developed suddenly eight days earlier, and had transiently appeared in approximately 60-minute intervals recurring twenty or more times per day. The following morning, the movements continued throughout the entire day. The patient had been taking 100 mg aspirin due to angina pectoris for five years. He had no prior history of chorea or other movement disorders, and denied the use of any other drugs. There was no family history of neurological diseases.

The patient presented with choreo-ballistic movement involving the face and limbs on the left side. The involuntary movements were forceful, jerky, irregular, had large amplitudes, and caused a lurching gait. These movements were not present during sleep. His mental status was normal and the cranial, motor, and sensory nerve examinations showed no abnormalities.

The routine laboratory tests, including a complete blood count, liver enzymes, blood urea nitrogen, creatinine, electrolytes, thyroid function tests, and autoantibody screens were within the normal limits. The fasting blood glucose levels were 454 mg/dL and glycosylated hemoglobin A1c (HbA1c) was 15.0%. No ketones were detected in the urinalysis. MR imaging of the brain showed increased signal intensification in the area of the right putamen in the T1-wieghted images, no diffusion restriction in the diffusion weighted image, and no signal change in the gradient echo image of the same area.

The patient was diagnosed with HIHH based on his history, examinations, and classic radiologic features. The patient initially received subcutaneous insulin treatment, but the choreic movement persisted despite normal glucose levels. Seven days after admission, haloperidol was administered in an increasing dosage from 1.5 mg per day to 4.5 mg per day. Within the first three days, the patient reported mild improvement, and after seven days he reported almost complete resolution of his chorea. We continued to administer haloperidol over ten days after which the dosage was tapered out over one week. Three days after haloperidol withdrawal, the chorea recurred on the same side of the body despite the patient having normal blood glucose level range. The patient was re-prescribed the same dose of haloperidol, however, his symptoms persisted. The recurring chorea was resolved after administration of 12.5 mg of tetrabenazine.

Discussion

Recurrence of chorea-ballismus usually develops in patients with non-ketotic hyperglycemia, uremic encephalopathy, systemic lupus erythematosus, and Sydenham's chorea.[2] Although the mechanisms associated with the recurrence of chorea-ballismus remain unclear, this condition is believed to be associated with recurrent regional hypoperfusion in the basal ganglia.[2] In addition, neurotransmitters may contribute to hyperglycemia induced chorea-ballismus; that is, hyperglycemia can induce deficiencies in GABA and acetylcholine that may lead to chorea-ballismus.[1]

To our knowledge however, there are no reports of chorea-ballismus associated with haloperidol withdrawal. Chronic administration of haloperidol can result in the development of dopamine super-sensitivity, and subsequent withdrawal may result in biochemical alterations in the dopaminergic and GABAergic striatal systems.[3] Therefore, we believe that the recurrence of chorea may be primarily related to GABA and acetylcholine deficiencies in the basal ganglia and/or subsequent changes in the dopaminergic-GABAergic balance, which manifest following acute withdrawal of haloperidol.

In conclusion, this case suggests that recurrent chorea is not always due to hyperglycemia and may represent neurochemical changes due to treatment with dopamine receptor blocking agents. Although we could not define the relevant cause of the recurrent chorea after haloperidol withdrawal, tapering the dosage of dopamine receptor blocking agents associated with this condition should be done very slowly and carefully.

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