

## Case report

# Cerebellar Ataxia: An Unusual Complication of Hypoglycemia

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### Abstract:

A 58 year-old diabetic lady was found unconscious with a blood glucose of 42mg/dl ; the hypoglycemia was corrected , leading to gain of her consciousness . She had injected 20 U rather than her usual 15 U of regular insulin ,three times a day since last two days ,without advice of doctor. Later she developed gait ataxia and slurred speech about 10 hours after being found unconscious. She gave history of multiple recurrent episodes of unconsciousness after increasing the dose of insulin. She had no history of any gait disturbances in the past and she denied any history of alcohol or drug abuse. This case highlights a seldom-reported case of ataxia and reviews the likely mechanism for hypoglycemia-induced cerebellar dysfunction and why most patients do not manifest neuroglycopenia in this manner. In addition, the differential diagnosis of ataxia is reviewed and serves as a reminder that a history of hypoglycemia should be assessed in patients presenting with ataxia.

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### Case presentation

A 58 year-old diabetic lady was found unconscious with a blood glucose of 42mg/dl ; the hypoglycemia was corrected , leading to gain of her consciousness . She had injected 20 U rather than her usual 15 U of regular insulin ,three times a day since last two days ,without advice of doctor. Later she developed gait ataxia and slurred speech about 10 hours after being found unconscious. She gave history of multiple recurrent episodes of unconsciousness after increasing the dose of insulin. She had no history of any gait disturbances in the past and she denied any history of alcohol or drug abuse.

At presentation to our hospital ,she was unconscious, her blood sugar was 42mg/dl, blood pressure was 160/96mm hg, pulse 90/min ,oxygen saturation 98% and temperature 97.6°F.After giving 200 ml of 25% dextrose solution , she became conscious and oriented to time , place and person. Respiratory ,

cardiac and abdominal examination were normal. On neurological examination, she was dysarthric . Her pupil were reacting normally and she had normal extra ocular muscle movements with end point nystagmus bilaterally . All cranial nerves were intact; she had normal power and sensory examination, and had sluggish deep tendon reflexes in all four limbs. On testing of coordination, she had dysmetria in all 4 limbs (finger to nose and heel to shin), and a broad based unsteady gait that prevented ambulation .The repeat blood glucose was 146 mg/dL, and additional investigations revealed the following: Haemoglobin 10.2gm/dl, white blood cell count 8200/ $\mu$ l, hematocrit 32.8%, creatinine 0.8 mg/dL, sodium 133 mmol/L, potassium 4.2 mmol/L, bicarbonate 22mmol/L. Her glycosylated haemoglobin(Hb A1C) was 7.2% 1 month back. Lumbar puncture was not performed. Magnetic resonance imaging (MRI) of the brain did not show any haematoma or infarct.

The various possibilities causing symmetric cerebellar ataxia can be divided into acute, subacute, and chronic etiologies. As our patient had ataxia of acute onset, so the causes include cerebellar hematoma, drug or alcohol ingestion, vertebralbasilar ischemic attacks, viral cerebellitis, Wernicke's encephalopathy and post infectious syndrome.

As she was a diabetic and hypertensive, she was at high risk for intracerebral bleeding and cerebral ischemic events. Wernicke's encephalopathy was also considered as a possibility, as only one third of patients with Wernicke's actually have the classic triad of ataxia, ophthalmoplegia and confusion. However, the patient was not in a confusional state and 90% of patients with Wernicke's are thought to have disturbances of consciousness and mentation.<sup>4</sup> As the patient had been eating regularly severe thiamine deficiency was unlikely.

Her MRI brain did not reveal any hematoma or vertebralbasilar infarction and cerebellum was normal.

During hospitalization her blood sugar had been in range of 90-180mg/dl on insulin. Her ataxia persisted during hospitalization. At the time of discharge on 7<sup>th</sup> day, she had gait ataxia and persistently slurred speech and was discharged with the diagnosis of hypoglycemia induced cerebellar dysfunction. On follow up after 4 weeks her ataxia and slurred speech had slightly improved.

The patient signed herself out of the nursing facility 11 weeks later against medical advice. Two weeks later, her neurological examination was found to be normal.

### Discussion

Cerebellar dysfunction is a rare complication of hypoglycemia. The common neurological manifestations of hypoglycemia include confusion, altered

behavior, seizures and loss of consciousness.<sup>6</sup> Many studies in rats and humans have shown that metabolism of glucose is different in cerebellum as compared to cerebrum. This difference in glucose metabolism is responsible for protection of cerebellum from hypoglycaemia.<sup>3,8,9</sup> Few other cases have also been reported on hypoglycaemia induced cerebellar ataxia. Rajbhandari et al described a diabetic female who presented with slurred speech and cerebellar ataxia shortly before she had hypoglycaemia and was resuscitated in emergency with glucagon.<sup>1</sup> MRI brain revealed changes of central pontinomyelinosis. The patient gradually improved and after 6 months she had normal speech with residual impairment in walking.

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Kim et al reported a female diabetic who presented with hypoglycaemia and had transient cerebellar ataxia that resolved after 12 hrs.<sup>3</sup> Her MRI brain was normal. PET scan study of this patient was done to compare glucose uptake & metabolism of her cerebrum and cerebellum with that of normal controls. In normal controls, glucose uptake was higher in cerebellum relative to cerebrum, but the rate of metabolism of glucose was lower in the cerebellum. In the patient, by contrast, the rate of glucose uptake was lower in the cerebellum and metabolism was similar to that of cerebrum. This difference in glucose metabolism appeared to make this patient more susceptible to hypoglycemic cerebellar injury, whereas in the controls, the cerebrum was more likely to be adversely affected only by prolonged and severe hypoglycemia.

Schwaninger et al reported a case who presented with recurrent episodes of hypoglycaemia for 2 years and he had cognitive impairment, cerebellar ataxia and motor neuropathy.<sup>2</sup> On investigation, he was found to have insulinoma. MRI brain was suggestive of extra

pontine myelinosis .After removal of insulinoma, the gait ataxia had improved partially but lesion seen in MRI was still present.

This case highlights a seldom-reported case of ataxia and reviews the likely mechanism for hypoglycemia-induced cerebellar dysfunction and why most patients

do not manifest neuroglycopenia in this manner. In addition, the differential diagnosis of ataxia is reviewed and serves as a reminder that a history of hypoglycemia should be assessed in patients presenting with ataxia.

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