



PROLONGED CEREBELLAR ATAXIA AFTER –SEVERE HYPOGLYCEMIC ATTACK IN A PATIENT WITH T1DM : A RARE BUT DISTURBING SEQUELA



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BACKGROUND

- Hypoglycemia is common in people with diabetes who aim to achieve strict blood glucose control.
- The neurologic manifestations of hypoglycemia are mostly reversible and includes behavioral changes, difficulty in concentration, confusion, loss of fine motor functions and seizures.
- Herein we have presented a case who was pregnant and experienced severe hypoglycemia which led to permanent ataxia

CASE

- 26 years old female patient was brought to our emergency room by her husband because of being found unconscious. She was 10 weeks of pregnant and had history of type 1 diabetes mellitus (T1DM) for 13 years.
- At the time of admission her plasma glucose level was 23 mg/dl. Intravenous glucose infusion and glucagon were applied rapidly.
- In the initial physical examination she was lethargic, blood pressure was low with tachycardia while body temperature was normal. In the obstetric ultrasonography (USG), the fetus was alive. She had nonproliferative retinopathy in the eye examination and sensory neuropathy.
- In the laboratory examination, she had microalbuminuria and normal kidney and liver function tests, electrolytes, thyroid function tests and vit B12 levels. After her blood glucose returned to normal, complete neurologic examination was made. Her pupils were reactive, tendon reflexes were normal but she was dysarthric. On the second day, dysarthria continued. We realized she had gait disturbance and on coordination tests she had severe dysmetria in all 4 limbs (finger to nose and heel to shin).
- Cranial magnetic resonance imaging (MRI) and electroencephalography (EEG) didn't reveal any pathology

- With short acting analogue and NPH insulin, her blood glucose levels were within the target range but her dysarthria and ataxia persisted upon discharge on the 12 th day. One month after discharge, she still had moderate gait disturbance and slow speech in the pregnancy and the newborn control visit. She had an uneventful labor on the 39 th week of was healthy.

Table 1. Laboratory data of the patient

	Laboratory result	Normal range
Plasma Glucose	40 mg/dl	70-199
Urea	35 mg/dl	10-48
Creatine	0.8 mg/dl	0.1-1.2
Na	142 mg/dl	136-145
K	3.8 mmol/l	3.5-5
ALT	29 U/L	0-41
insulin	1.54 µU/ml	2.6-24.9
C-peptide	0.3 ng/ml	0.1-3.6
Anti -GAD	14 u/ml	0-1
Anti-islet	positive	negative
TSH	2.5 uIU/ml	0.4-4.5
Anti-TPO	149 IU/ml	0-34
Cortisol	30 µg/dl	6-19

CONCLUSION

- The neurologic manifestations of hypoglycemia include behavioral change, confusion, loss of consciousness, and seizures. Rarely, neuroglycopenia can present as ataxia, and prolonged and potentially irreversible deficits may occur with repeated episodes of hypoglycemia.
- Cerebellar dysfunction is a rare complication of hypoglycemia and may occur in patients with altered cerebellar glucose kinetics. In summary, glucose uptake and utilization in the cerebellum appear to be protective with regard to hypoglycemia.
- Hence, cerebellar dysfunction is a rare complication of hypoglycemia, this disorder may occur in patients with altered cerebellar glucose kinetics. It appears that the neuronal damage in our patient was severe enough to cause prolonged ataxia.
- Differential diagnosis involves hematoma, cerebellitis, drug or alcohol ingestion, Wernicke encephalopathy, vit B12 deficiency and ataxia telangiectasia. Reconsideration of therapy and glycemic targets and supportive therapy is warranted.