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Case Report
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Guillain-Barré Syndrome (GBS) Mimicking Diabetic Ketoacidosis (DKA) with Dyselectrolytemia

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Abstract

Diabetic ketoacidosis (DKA) is a serious complication of diabetes characterized by high blood sugar, acidic pH levels, and increased ketone levels in the body, mostly observed in individuals with type 1 diabetes mellitus (T1DM) but can rarely occur in those with type 2 diabetes mellitus (T2DM). It is commonly triggered by factors like infection, new-onset diabetes, or non-compliance with treatment [1]. The most frequent cause of sudden flaccid paralysis is Guillain-Barré syndrome (GBS). It is due to an autoimmune reaction that destroys peripheral nervous system nerves, resulting in symptoms including tingling, weakness, and numbness that can eventually become paralysis [2].

***Case Presentation:** We report a case of a 14-year-old female with T1DM admitted with altered sensorium and vomiting. On evaluation, it was found to be DKA. The patient was managed according to the ISPAD guidelines for DKA, with hypotonic fluids, potassium chloride, and phosphorus replacement. After 24 h of treatment, DKA recovered, and the patient regained consciousness, but she still had weakness, was unable to sit without support, and had difficulty swallowing. Clinical examination showed muscle tone was decreased, power at the hip was 2/5, at the knee and ankle was 3/5, and at the shoulder and elbow was also 3/5. The neurologist's opinion was taken, cerebrospinal fluid (CSF) was normal, and the nerve conduction velocity showed axonal and segmental demyelination; GBS was diagnosed.*

***Conclusion:** Persistent weakness and dysphagia after DKA recovery and electrolytes are normalized, one should then suspect GBS. In early-onset GBS, CSF can be normal.*

Keywords: Guillain-Barré syndrome, diabetic ketoacidosis

Abbreviations: DKA: diabetic ketoacidosis; GBS: Guillain-Barré syndrome; T1DM: type 1 diabetes mellitus; T2DM: type 2 diabetes mellitus; CSF: cerebrospinal fluid; IVIG: intravenous immunoglobulin

Case Report

14-year-old female with a history of type 1 diabetes mellitus (T1DM) for 8 months, stopped insulin for one month. She was admitted to our hospital with altered sensorium and vomiting for 3 days, and a diagnosis of diabetic ketoacidosis (DKA) was made. She was admitted elsewhere for 2 days, but there was no improvement in her sensorium. The first ABG showed pH-7.0, bicarbonate 8.1 mEq/L, a high anion gap, and urine ketone 3+; on our hospital admission, ABG showed pH-7.3, bicarbonate 16.8 mEq/L, urine ketone 2+, potassium was low (2.0 mEq/L), sodium 155 mmol/L, phosphorus 1.42 mg/dL, and magnesium was normal (2.10 mg/dL). Due to an altered sensorium, a CT brain was done, which was normal. The patient was managed according to the ISPAD guidelines for DKA, with hypotonic fluids, potassium chloride, and phosphorus replacement. After 24 h of treatment, DKA recovered, and the patient regained consciousness, but she still had weakness, was unable to sit without support, and had difficulty swallowing. Clinical examination showed muscle tone was decreased, power at the hip was 2/5, at the knee and ankle was 3/5, and at the shoulder and elbow was also 3/5. The plantar was mute, and all reflexes were absent; a sluggish gag reflex, and sensation was normal. Initially, it was thought that this was due to hypokalemia and hypophosphatemia, and she was put on a Ryle tube. After vigorous replacement, potassium and phosphorus were normalized, with partial improvement in weakness, power 3/5 at the hip, knee, ankle, and shoulder, and at the elbow 4/5. Dysphagia persisted, but there was no respiratory difficulty; after that, Guillain-Barré syndrome (GBS) was suspected. A neurologist's opinion was taken, and cerebrospinal fluid (CSF) was normal. The nerve conduction velocity showed axonal and segmental demyelination. The patient was transferred to the neurology department, and intravenous immunoglobulin (IVIG) was administered for 4 days. The patient's weakness improved gradually; on day 3, she could walk without support and swallow food.

Discussion

Here we report a case of a 14-year-old girl with GBS with DKA. GBS encompasses a range of nerve-related disorders marked by gradual muscle weakness and absent reflexes. It is thought that the mechanism behind GBS is an inflammatory neuropathy caused by the cross-reactivity of antibodies with neural antigens brought on by particular infections. Similar to gangliosides, lipooligosaccharides are expressed in the bacterial walls of infectious organisms like *C. jejuni*. Because of this molecular mimicry, antiganglioside antibodies are produced that target nerves [3]. As GBS can lead to respiratory failure necessitating mechanical ventilation, early treatment is more beneficial for optimal results [4]. There are only 4 case reports in the literature, DKA with GBS [5–8], and in all these cases, CSF protein was high. Albuminocytologic dissociation in CSF is a diagnostic hallmark of GBS. In our case, an interesting point was that the CSF examination was normal. Protein levels in the CSF may appear normal during the initial stages of GBS, but approximately 90 percent of patients exhibit elevated protein levels by the conclusion of the second week of symptom onset [9]. Another interesting point in our case is that the electrolyte was deranged, which can mimic GBS, but after the electrolyte improved, persistent weakness and dysphagia can give clues for suspecting GBS.

Conclusion

In summary, we reported a rare case of DKA associated with GBS. Persistent weakness and dysphagia, after DKA recovers and electrolytes are normalized, one should then suspect GBS. In early-onset GBS, CSF can be normal.

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Conflicts of Interest

The author(s) do not have any conflict of interest.

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