**Introduction**

- Osmotic demyelination syndrome (ODS) is well-known to occur in response to rapid correction of hyponatremia.
- Symptoms of ODS are usually delayed 2-6 days following the rapid serum sodium correction, and may include dysarthria, dysphagia, extremity weakness or paralysis, behavioral disturbances, confusion, seizures, lethargy, obtundation, and coma.
- Patients with ODS typically present with initial serum sodium concentrations less than 120 mEq/L. In extremely rare scenarios, ODS may result from other serum osmolar abnormalities.
- We present one such example - ODS in a Type 2 diabetic with chronic, severe hyperglycemia.

**Case Presentation**

- A 36-year-old male with poorly controlled type 2 diabetes mellitus (A1c of 15.7%) on metformin, insulin, and empagliflozin presented to the emergency department with unstable gait, dysarthric speech, anemia, confusion, and asymmetric weakness.
- Vital signs stable. Physical examination revealed left-sided weakness, slurred speech, and abnormal cerebellar testing.
- Head CT was negative for stroke.
- Lab workup demonstrated blood glucose 490, lactic acid 3.7, bicarbonate 24, and anion gap 14.
- Serum sodium, corrected for blood glucose 141.

**Imaging Findings**

Amorphous restricted diffusion and FLAIR signal hyperintensity within the central pons without significant pontine expansion or postcontrast enhancement. Pattern is suggestive of osmotic demyelination (central pontine myelinolysis). Other metabolic abnormalities such as hyperglycemia-related edema or acute hypertensive encephalopathy are not excluded, although brainstem predominant pattern is less common in these entities.

**Case Resolution**

- Throughout admission, his blood glucose remained variable, but was responsive to increasing doses of basal and rapid-acting insulin. Serum sodium remained unchanged.
- Neurology recommended high dose IV thiamine.
- Clinically, his mental status returned to baseline and his dysarthria and ataxia gradually and mildly improved over the course of his three-day admission.
- At discharge, he still could not walk independently and his speech remained slightly dysarthric.
- Patient attended two physical therapy sessions outpatient and appeared to be improving, but had two incidents of falls when not using his walker.

**Discussion**

- There are only a handful of documented cases reporting ODS in patients with stable serum sodium and hyperglycemia; nearly all cases are associated with Hyperosmolar Hyperglycemic State or blood glucose >600.
- The exact pathophysiologic mechanism of the relationship between high serum glucose and pontine demyelination is not clearly understood, but a possible explanation involves hypertonic pontine insult resulting in injured astrocytes, disruption of the astrocyte-oligodendrocyte network, and induced apoptosis.
- Management should involve frequent neurologic examinations, treatment of hyperglycemia, and physical rehabilitation. This case provides evidence that severe hyperglycemia can cause ODS.
- Recovery from hyperglycemia-induced ODS appears to be good overall. Ataxia may be a persistent feature and affect functional status.

**References**