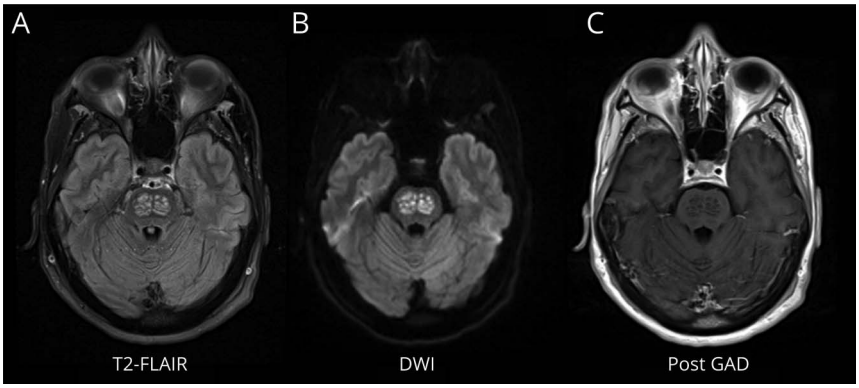
Teaching NeuroImage: Central Pontine Myelinolysis in Diabetic Ketoacidosis

Natalia Gonzalez Caldito, MD, Nurose Karim, MD, and Mehari Gebreyohanns, MD

Neurology® 2021;97:e1971-e1972. doi:10.1212/WNL.0000000000012301

Correspondence
Dr. Gebreyohanns
mehari.gebreyohanns@utsouthwestern.edu

Figure Central Pontine Myelinolysis in Diabetic Ketoacidosis



T2 fluid-attenuated inversion recovery (FLAIR) reveals symmetric hyperintensities centered in the pons (A) with restricted diffusion (B). T1 postcontrast with gadolinium demonstrated no enhancement (C). DWI = diffusion-weighted imaging; GAD = gadolinium.

A 38-year-old woman with uncontrolled type 1 diabetes (HbA1c 12.8%) was admitted for diabetic ketoacidosis (731 mg/dL blood glucose). Hyperglycemia was corrected within 24 hours to 129 mg/dL. Upon presentation, her sodium and potassium levels were 139 and 3.9 mmol/L, respectively, remaining stable until discharge. There was no history of malnutrition or alcohol abuse.

Four days later, the patient developed acute diffuse pyramidal weakness. Brain MRI revealed symmetric restricted diffusion in the pons with normal magnetic resonance angiography (figure). The patient remained stable and was discharged to a rehabilitation facility.

Central pontine myelinolysis (CPM) is a clinically heterogeneous neurologic disorder of demyelination in the pons, usually from rapid correction of hyponatremia.[1,2]

Diabetic ketoacidosis is an uncommon cause of CPM with uncertain physiopathology.[3] Here, it is plausible that a rapid drop in osmolality in a chronic state of high osmolality (uncontrolled diabetes) led to CPM. A slower correction of hyperglycemia possibly could have prevented it.

MORE ONLINE

Teaching slides
links.lww.com/WNL/B447

Study Funding
No targeted funding reported.

From the Department of Neurology & Neurotherapeutics, University of Texas Southwestern Medical Center, Dallas.

Go to Neurology.org/N for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

Disclosure

The authors report no disclosures relevant to the manuscript. Go to Neurology.org/N for full disclosures.

Appendix Authors

Name	Location	Contribution
Natalia Gonzalez Caldito, MD	University of Texas Southwestern Medical Center	Patient management, literature review, gathered data, drafted the manuscript for intellectual content
Nurose Karim, MD	University of Texas Southwestern Medical Center	Patient management and revised the manuscript for intellectual content
Mehari Gebreyohanns, MD	University of Texas Southwestern Medical Center	Patient management and revised the manuscript for intellectual content

References

1. Fitts W, Vogel AC, Mateen FJ. The changing face of osmotic demyelination syndrome: a retrospective, observational cohort study. *Neurol Clin Pract.* Epub 2020 Aug 26.
2. Rodríguez-Velver KV, Soto-Garcia AJ, Zapata-Rivera MA, et al. Osmotic demyelination syndrome as the initial manifestation of a hyperosmolar hyperglycemic state. *Case Rep Neurol Med.* 2014;2014:e652523.
3. Matías-Guiu JA, Molino ÁM, Jorquera M, et al. Pontine and extrapontine myelinolysis secondary to glycemic fluctuation. *Neurología.* 2016;31(5):345-347.

Teaching NeuroImage: Central Pontine Myelinolysis in Diabetic Ketoacidosis

Natalia Gonzalez Caldito, Nurose Karim and Mehari Gebreyohanns

Neurology 2021;97;e1971-e1972 Published Online before print June 2, 2021
DOI 10.1212/WNL.0000000000012301