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Teaching NeuroImages: Central Pontine Myelinolysis in Diabetic Ketoacidosis

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Natalia Gonzalez Caldito, Nurose Karim, and Mehari Gebreyohanns report no disclosures relevant to the manuscript.

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DESCRIPTION

Central Pontine Myelinolysis (CPM) is a clinically heterogeneous neurological disorder of demyelination in the pons, usually from rapid correction of hyponatremia.^{1,2}

A 38-year-old woman with uncontrolled type 1 DM (hbA1c 12.8%) was admitted for diabetic ketoacidosis (731mg/dl blood glucose). Hyperglycemia was corrected within 24 hours to 129mg/dl. Upon presentation, the sodium and potassium levels were 139 and 3.9 mmol/L respectively, remaining stable until discharge. There was no history of malnutrition nor alcohol abuse.

Four days later, she developed acute diffuse pyramidal weakness. Brain MRI revealed symmetric restricted diffusion in the pons with a normal MR angiography (Figure 1). She remained stable and was discharged to a rehabilitation facility.

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 DM) lead to CPM. A slower correction of hyperglycemia could have possibly prevented it.

Appendix 1. Authors

Name	Location	Contribution
Natalia Gonzalez Caldito, MD	University of Texas Southwestern Medical Center	Patient management, literature review, gathered data and 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

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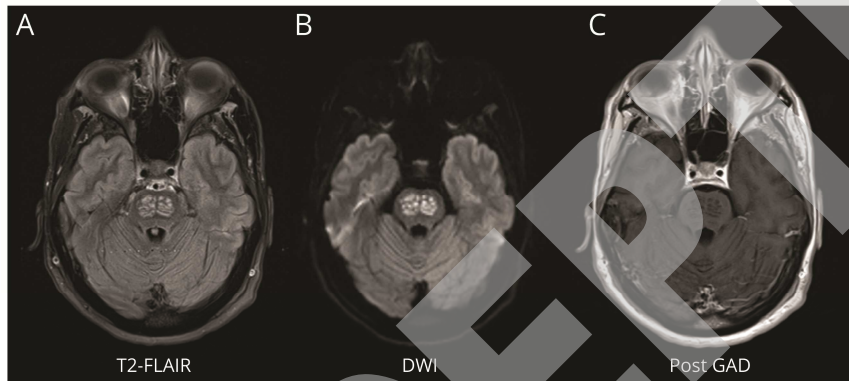
BIBLIOGRAPHY

1. Fitts W, Vogel AC, Mateen FJ. The changing face of osmotic demyelination syndrome: A retrospective, observational cohort study. *Neurology: Clinical Practice*. Epub ahead of print 26 August 2020. DOI: 10.1212/CPJ.0000000000000932.
2. Rodríguez-Velver KV, Soto-García AJ, Zapata-Rivera MA, et al. Osmotic Demyelination Syndrome as the Initial Manifestation of a Hyperosmolar Hyperglycemic State. *Case Reports in Neurological Medicine* 2014; 2014: e652523.
3. Matías-Guiu JA, Molino ÁM, Jorquera M, et al. Pontine and extrapontine myelinolysis secondary to glycemic fluctuation. *Neurología* 2016; 31: 345–347.

Figure 1: Central pontine myelinolysis in diabetic ketoacidosis

T2-FLAIR revealing symmetric hyperintensities centered in the pons (A) with restricted diffusion (B). T1 post contrast with gadolinium demonstrated no enhancement(C).

FLAIR: fluid-attenuated inversion recovery; DWI: diffusion-weighted magnetic resonance imaging; Post GAD: post gadolinium. MRI: Magnetic Resonance Imaging



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