Refractory Diabetes Insipidus Leading to Diagnosis of Type 2 Diabetes Mellitus and Non-Ketotic Hyperglycemia in an Adolescent Male

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CASE REPORT

An obese 16-year-old male presented with chronic headaches and bitemporal visual field loss. Physical exam showed acanthosis nigricans and BMI of 39.8 kg/m.^{2} MRI of the brain revealed craniopharyngioma (**Figure 1**). He developed hypopituitarism after surgery, requiring Hydrocortisone, Levothyroxine, Testosterone enanthate, and desmopressin (DDAVP).

Two months later, he presented with uncontrolled polyuria despite increasing DDAVP doses up to 1.7 mg daily. A two-hour post prandial blood glucose was 400 mg/dL and hemoglobin A1C was 10.2%, consistent with diabetes mellitus (DM). Diabetes autoimmune panel was negative. He was started on insulin Glargine and Lispro. Two days after starting insulin, he had a seizure consisting of staring episodes, right upper extremity shaking, right-eye deviation and urinary incontinence. Laboratory exam showed venous pH 7.36, sodium 139 meq/L, bicarbonate 19.3 meq/L, glucose 569 mg/dL, serum osmolality 305 mOSM/kg and negative urinary ketones, consistent with nonketotic hyperglycemia. MRI of the brain showed nonenhancing T1-hyperintensity within the left basal ganglia (**Figure 2**). He returned to baseline neurologic status shortly after admission. EEG obtained prior to discharge was negative for epileptiform activity. One month later, his hemoglobin A1C was 8.3% on Glargine and Metformin. He had no further seizure episodes and repeat brain MRI was normal.

DISCUSSION

"Recalcitrant diabetes insipidus" in this obese adolescent was due to undiagnosed Type 2 DM. Nonketotic hyperglycemia with acute neurological changes is rare in adolescents with Type 2 DM. Acute neurological presentations associated with nonketotic hyperglycemia include focal

Figure 1. Coronal T1-weighted image demonstrates a complex cystic and solid suprasellar mass with rim enhancement, which is characteristic of craniopharyngioma.

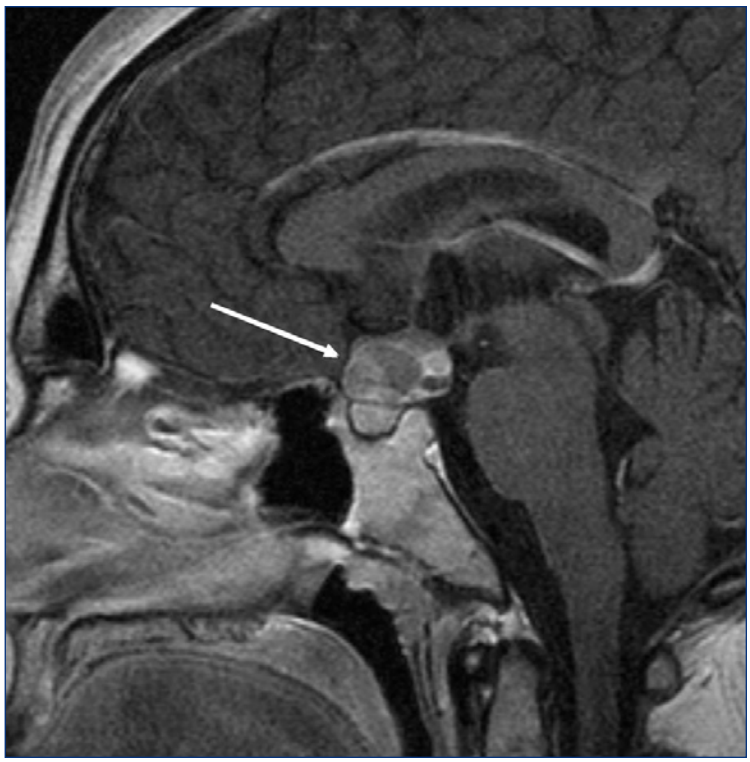
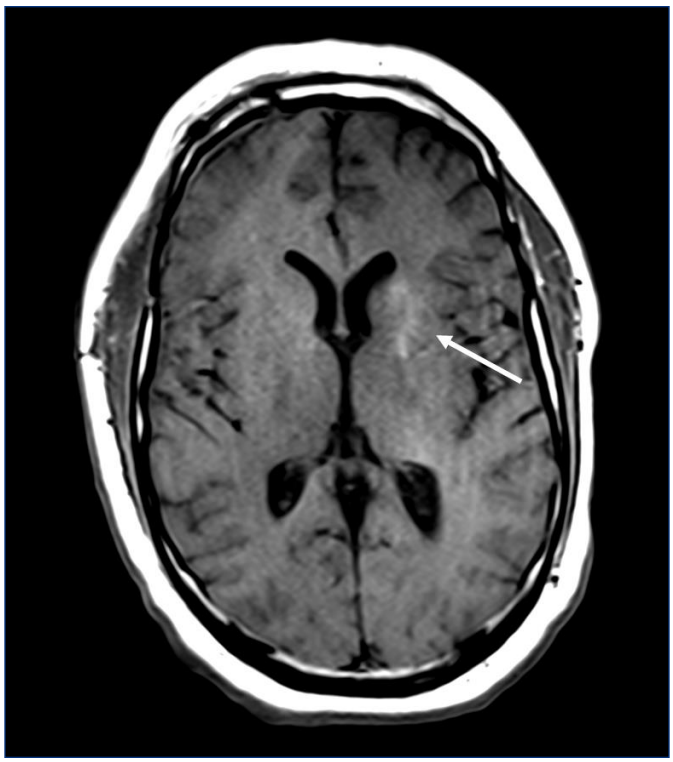


Figure 2. New non-enhancing T1-weighted hyperintensity within the left basal ganglia, consistent with non-ketotic hyperglycemia.



partial seizures, epilepsia partialis continua, chorea, and ballismus.[1-3] The etiology of these findings in nonketotic hyperglycemia is not compeltely understood. The proposed mechanism is increased neuronal excitability due to a relative increase in the metabolism of GABA, an inhibitory neurotransmitter, and the presence of neuronal K_{ATP} channels.[4] As in our patient, these neurological manifestations are reversible with the correction of hyperglycemia.

References

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