Pediatric Stroke As The Presenting Symptom Of New Onset Diabetes Without DKA.

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Introduction:
Neurologic complications, such as cerebral edema, stroke, and extrapontine myelinolysis, are rare in pediatric patients with type 1 diabetes mellitus (T1DM) in the absence of severe diabetic ketoacidosis (DKA) or chronically poor glycemic control. Ischemic or hemorrhagic stroke may account for 10% of intracerebral complications of DKA.

DKA increases the risk for neurovascular compromise by several proposed mechanisms, including dehydration, hyperosmolarity, tissue hypoxia and acidosis.

Neurologic complications of hyperglycemia in adult patients without DKA are reported frequently; however, there are rare reports of stroke in pediatric patients with hyperglycemia without DKA.

Case Description:
We present the case of a previously healthy ten-year-old, premenarchal, thin, African-American female, who presented with a two day history of right facial droop and right hemiplegia.

On exam she exhibited subtle right lower facial weakness, 3/5 strength in the right upper and lower extremities with positive drift in the right upper and lower extremities. Neuroimaging demonstrated an acute left thalamic ischemic stroke without vascular defects.

A finding of hyperglycemia (initial blood glucose 217 mg/dl, 12 mmol/L) led to the further laboratory studies. A hemoglobin A1c of 8.4% and elevated diabetes autoantibodies confirmed the diagnosis of new-onset type 1 diabetes mellitus.

Studies:

<table>
<thead>
<tr>
<th>Diabetes Autoantibodies</th>
<th>Measured Level</th>
<th>Negative</th>
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</thead>
<tbody>
<tr>
<td>IAA</td>
<td>183 High</td>
<td>&lt;0.5 U/mL</td>
</tr>
<tr>
<td>GAD-65</td>
<td>2 High</td>
<td>&lt;5 U/mL</td>
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<tr>
<td>ICA 512 antibodies</td>
<td>23 High</td>
<td>&lt;1 U/mL</td>
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Pertinent labs included lack of ketosis (bicarbonate 24 mmol/L, negative urine ketones), normal lipid profile, ANA, C-Reactive Protein and CBC, and normal screening for prothrombotic conditions (Protein C activity, Factor V Leiden, Factor II 20210 mutation, Homocysteine, Cardiolipin IgG and IgM).

Her past medical history, the family history and social history were negative other than a brother with sickle cell trait.

She received diabetes management education and treatment with multiple daily injections of insulin. She gradually returned to a normal neurologic state over the subsequent year and has been without stroke recurrence for two years despite suboptimal glycemic control due to inconsistent diabetes care at home.

Discussion:
Stroke at presentation of T1DM is rare. It is especially rare in pediatric patients and in the absence of DKA. Hyperglycemia has been implicated in stroke risk but typically in association with concurrent comorbidities such as severe acidosis, metabolic syndrome, and dehydration.

There is only one report of stroke in a child at diagnosis of diabetes without concurrent DKA in the literature, and that child had thiamine-responsive megaloblastic anemia that may have contributed to her clinical presentation.

Conclusions:
Even though our case cannot prove causality, it raises questions about the impact of hyperglycemia on the risk of stroke in pediatric patients.

We propose that diabetes care providers should have a high index of suspicion for stroke symptoms in pediatric patients with new onset or established diabetes (including those without DKA), as prompt diagnosis and treatment of stroke decreases morbidity and mortality.

References: