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Children with Diabetes Mellitus and Ketoacidosis: Cerebral Proton Magnetic Resonance Spectroscopy

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Abstract

Proton MR Spectroscopy measures the ratios of N-acetyl aspartate (NAA) to creatine (Cr) in the brain as a function of neuronal injury or dysfunction. We hypothesized that children with DKA might have lower brain NAA/Cr ratios, indicating subtle neuronal damage. During DKA treatment, 29 children underwent cerebral proton MR spectroscopy two to twelve hours after starting treatment and 72 hours or more after recovering from the episode. We measured the NAA, Cr, and choline peak heights in three brain regions: the basal ganglia, periaqueductal gray matter, and occipital gray matter. In DKA-related cerebral edema, these areas were found to be more susceptible to neurologic injury in previous studies. Throughout the acute illness and recovery periods, we compared the ratios of NAA/Cr and Cho/Cr.

Introduction

This complication is fatal and causes permanent neurologic morbidity in children. Children who present with greater hypomania and dehydration during DKA are at greater risk for cerebral edema, but the exact cause of this complication is unknown [1]. Although cerebral edema is only seen in a small percentage of children with DKA, several studies have suggested that cerebral edema may be present in some degree in most of these children. However, the connection between this "subclinical" cerebral edema and underlying brain injury remains a mystery. Proton MR spectroscopy is an imaging device that is exceptionally delicate for distinguishing cerebral injury.7,8 N-acetyl aspartate NAA is a putative neuronal marker9 and cerebral injury diminished neuronal feasibility, diminished neuronal capability, or neuronal misfortune is reflected by a reduction in the convergence of NAA comparative with other cerebral metabolites.7,8 Earlier work10 has shown a lessening in parietal NAA/creatine proportions in grown-ups with diabetes contrasted and typical controls [2]. Additionally, parietal NAA/Cr is lower in children11 with poorly controlled diabetes than in controls. Until this point in time, in any case, changes in the convergences of cerebral metabolites have not been assessed during DKA in youngsters [3]. Proton MR Spectroscopy was used in this study to look at brain injury and metabolism during DKA in children.

Materials and Methods

Over a one-year period, institutions enrolled patients in this study. All children who met the following requirements were eligible to take part in this study [4]. They had DKA (defined as serum glucose >300 mg/dL, venous pH 7.25 and/or serum bicarbonate 15 mEq/L, and a positive test for urine ketones or serum ketones >3 mmol/L), were younger than 18 years old, and had been diagnosed with type 1 diabetes mellitus. All children whose parents or guardians consented to participate were included in the study.

Imaging techniques

Using a 1.5T imaging system and a standard quadrature birdcage head coil (Signa Horizon, LX Version 9.1, GE Healthcare, Milwaukee, WI), the study's participants underwent MR imaging of the brain at two time points: 1) two to twelve hours after starting DKA treatment; and 2) 72 hours or more after recovering from the episode of DKA, after metabolic acidosis and ketosis had subsided [5]. Proton MR Spectroscopy was performed after axial T2-weighted fluid-attenuated inversion recovery images with a 24 cm FOV, 4.2 mm section thickness, and 0.8 mm section gap were obtained. The Probe-P sequence, a point-resolved spectroscopy sequence designed for the detection of resonances with long T2s, was used for multivoxel chemical shift imaging on the first 11 patients in the study. This succession was utilized at a solitary segment area that incorporated the occipital curves and basal ganglia by utilizing TR/TE 1000/144 and a part thickness of 20 mm. The area of interest for automatic shimming was a 0.5–1 cm-long rectangular area within the skull [6]. The distance between this district and air/tissue/bone connection points in the skull was expanded in the event that the line width was more noteworthy than 15 Hz, water concealment was under 97%, or flip points were outside a 135 ± 30° territory. The Functool package from GE Healthcare was used to show the spectra. Additionally, using the Probe-P sequence, single-voxel spectroscopy at the periaqueductal gray matter level was carried out on these 11 patients. One voxel of 8 cm3 was chosen, and programmed shimming on the voxel was performed and regularly delivered a line width of 4 Hz or less, water concealment of close to 100%, and a flip point. For a separate substudy12, the protocol was changed to allow for better resolution of lactate and ketone peaks [7]. As a result, only the right occipital gray matter and right basal ganglia were scanned with single-voxel MR spectroscopy on the 18 patients who were enrolled. On the basis of previously described regional patterns of brain injury seen in children with DKA and cerebral edema13–15 and on previously described diffusion-weighted imaging abnormalities in children with DKA, the periaqueductal gray matter, the occipital gray matter, and the basal ganglia were selected as regions of interest for spectroscopy investigation. When possible, pharmacologic sedation was avoided during the imaging procedures. However, when sedation was required, midazolam or sodium pentobarbital with a dose of 2 mg/kg or less was used [8].

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Discussion

In a similar vein, when compared to recovery, NAA/Cr ratios in the occipital and periaqueductal gray matter decreased during DKA. Cho/Cr ratios, on the other hand, did not change significantly during or after DKA treatment. During DKA, these alterations suggest that neuronal function, viability, or both are compromised. Children with type 1 diabetes frequently develop DKA [9]. DKA occurs in 25% or more of children with new-onset type 1 diabetes, and it occurs at rates as high as 0.2 events per patient year in children with established type 1 diabetes. The exact etiology of cerebral edema in DKA is unknown, but it is likely complex and multifactorial, and it occurs in approximately 1% of pediatric DKA episodes and has a mortality rate. Many survivors leave with permanent neurologic deficits. Hypoxia-ischemia, changes in cerebral blood flow, disruptions in cell membrane ion transport, the production of intracellular osmolytes, and elevated concentrations of various inflammatory mediators have all been examined as potential contributors by researchers [10]. DKA-related cerebral edema may have varying clinical presentations, ranging from completely asymptomatic to severe neurologic derangements and manifestations of increased intracranial pressure. Asymptomatic cerebral swelling may be present in the majority of DKA episodes in children. In many neurologic disorders, proton MR spectroscopy is a useful method for functional brain examination. NAA, choline compounds, and Cr/phosphocreatine are among the prominent neurotransmitters and metabolites found in the brain. NAA is a neuronal-axonal marker that is not found in blood, CSF, or mature glial cells. Neuronal loss, decreased neuronal viability, and ischemia and stroke have all been linked to decreases in NAA.19 Seizures, metabolic disorders of the brain, neurodegenerative processes, and ischemia and stroke have all been associated with decreased NAA.

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