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Original Research

Optic Nerve Sheath Diameter Ultrasonography in Pediatric Patients with Diabetic Ketoacidosis

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ABSTRACT

Objectives: Subclinical cerebral edema has been reported in pediatric patients with type 1 diabetes and diabetic ketoacidosis (DKA) through magnetic resonance imaging. Ultrasonography of the optic nerve sheath diameter (ONSD) has been used to evaluate intracranial pressure. The objective of this study was to examine the utility of ONSD ultrasonography to evaluate intracranial pressures in children with DKA.
Methods: This prospective cohort evaluated pediatric patients who presented to the emergency department of the Children's Hospital at the University of Manitoba with DKA within 3 hours of initial treatment. A pediatric bedside neurologic evaluation tool for cerebral edema was utilized 1) within the first hour of the intravenous fluid initiation (t=0 hr); 2) 8 hours after initiation of treatment (t=8 hr); and 3) at hours after presentation (t=24 hr). At each time interval, 3 images of the patients' ONSDs were scanned by an 11 MHz linear array transducer. Increased intracranial pressure was considered in all patients whose mean ONSDs were >4.5 mm.
Results: We evaluated 7 patients, aged 4 to 17 years. No patients were clinically assessed as having cerebral edema. Overall, no significant differences emerged among the 3 time points (t=0 vs. t=8 hr; t=0 vs. t=24 hr; t=8 vs. t=24 hr) (all p>.216). Effect sizes were small at 0.14 (t=0 vs. t=8 hr); 0.27 (t=8 vs. t=24 hr); and 0.07 (t=0 vs. t=24).
Conclusions: Although not statistically significant, subtle changes in intracranial pressure may have been detected with ONSD ultrasonography in pediatric patients with DKA.

Mots clés :
Œdème cérébral
Acidocétose diabétique
Diamètre de la gaine du nerf optique
Pédiatrique
Ultrasonographie

RÉSUMÉ

Objectifs : L'œdème cérébral subclinique a été rapporté chez les patients de pédiatrie souffrant du diabète de type 1 et d'acidocétose diabétique (ACD) par l'imagerie par résonance magnétique. L'ultrasonographie du diamètre de la gaine du nerf optique (DGNO) a été utilisée pour évaluer la pression intracrânienne. L'objectif de cette étude était d'examiner l'utilité de l'ultrasonographie du DGNO pour évaluer les pressions intracrâniennes chez les enfants souffrant d'ACD.
Méthodes : Cette cohorte prospective évaluait les patients de pédiatrie qui se présentaient au service des urgences de l'Hôpital pour enfants de l'Université du Manitoba en raison d'une ACD au cours des 3 heures suivant le traitement initial. Un outil d'évaluation neuropédiatrique de l'œdème cérébral au chevet du patient était utilisé 1) au cours de la première heure après l'administration d'un liquide par voie intraveineuse (t=0 h); 2) 8 heures après le début du traitement (t=8 h); 3) dans les heures après l'arrivée (t=24 h). À chaque intervalle de temps, 3 images des DGNO des patients étaient balayées par une sonde linéaire de 11 MHz. L'augmentation de la pression intracrânienne était considérée chez tous les patients dont la moyenne du DGNO était>4.5 mm.

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Résultats : Nous avons évalué 7 patients âgés de 4 à 17 ans. Aucun patient n'était cliniquement évalué comme souffrant d'un œdème cérébral. Dans l'ensemble, aucune différence significative n'est apparue entre les 3 moments temporels (t=0 *vs* t=8 h; t=0 *vs* t=24 h; t=8 *vs* t=24 h) (tous p>0.216). Les petites tailles de l'effet se situaient à 0.14 (t=0 *vs* t=8 h); 0,27 (t=8 *vs* t=24 h); 0,07 (t=0 *vs* t=24).
Conclusions : Bien que non significatifs sur le plan statistique, les changements subtils de la pression intracrânienne pouvaient être détectés à l'ultrasonographie du DGNO chez les patients de pédiatrie souffrant d'ACD.

Introduction

Cerebral edema is a clinically significant complication of diabetic ketoacidosis (DKA) in children and adolescents, but predicting that patients are at risk for cerebral edema is challenging. Historically, a rapid change in serum osmolality was considered the primary mechanism of cerebral edema (1–5). However, evidence that supports various fluid protocols has not been clearly elucidated, and poor correlations exist between rate or volume of fluid administration, osmotic fluctuations (6–8), timing of treatment (9,10) and cerebral edema.

Subclinical cerebral edema in pediatric patients with DKA may be common. With magnetic resonance imaging (MRI), narrowing of lateral ventricles (11) or changes in T2 relaxometry and apparent diffusion coefficient mapping (12,13) have been reported. This may represent a milder pathophysiologic process or an increased risk for developing catastrophic cerebral edema.

Ultrasonography of optic nerve sheath diameter (ONSD) is a safe and noninvasive method for evaluating intracranial pressure (ICP) and has low interobserver variability (14). Because the subarachnoid space surrounding the optic nerve communicates directly with the chiasmal cistern of the central nervous system, increases in cerebral spinal fluid pressure will enlarge the ONSD (14). A meta-analysis of ONSD ultrasonography demonstrated a high level of accuracy to detect raised ICP when compared to invasive monitoring (15). Unlike higher modality imaging, ultrasonography is easily accessible and readily allows for serial evaluations. Ultrasonography of the ONSD has been utilized in several pediatric clinical scenarios, including traumatic brain injury, hydrocephalus, ventriculoperitoneal shunt failure, craniosynostosis, intracranial hemorrhage and cerebral malaria.

The objective of this prospective cohort study was to examine the feasibility and utility of ONSD ultrasonography in evaluating intracranial pressures in children with DKA. We hypothesized that significant changes in ONSD would be detected during the course of DKA management.

Methods

Patients

The University of Manitoba Research ethics board approved this prospective cohort pilot. Children presenting to the emergency department between April and June 2014 were eligible for the study if they were 4 to 18 years of age and had been diagnosed with both type 1 diabetes and DKA (defined as venous pH<7.25, serum bicarbonate concentration <15 mmol/L, positive test result for urine ketones or serum ketones >3 mmol/L and serum glucose >11 mmol/L). Children newly diagnosed and previously diagnosed with type 1 diabetes were eligible. Children were excluded if they arrived >3 hours after initial treatment in another facility, were hemodynamically or neurologically unstable, or had positive histories of ocular (i.e. glaucoma, trauma, optic nerve drusen) or neurologic pathology (i.e. hydrocephalus, cerebral shunts, idiopathic intracranial hypertension).

Protocol for diabetic ketoacidosis

Pediatric endocrinologists were consulted in all cases. Protocolized emergency standing orders included fluid boluses only with shock or orthostatic hypotension. Consistent with the International Society of Pediatric and Adolescent Diabetes guidelines (16), the hourly intravenous fluid rate was calculated by estimating the fluid deficit. This deficit was then corrected over a 48-hour period. Insulin was infused at 0.1 units/kg/hr 1 hour after the initiation of IV normal saline. Vitals and neurovitals were evaluated every hour, and serum laboratory tests were obtained on arrival and every 2 hours thereafter. The insulin infusion rate was maintained, but intravenous potassium and glucose were adjusted to avoid hypokalemia or hypoglycemia. Liberalization of fluid and oral intake occurred once DKA resolved, and subcutaneous insulin management was initiated.

Clinical assessment

After obtaining written consent from patients' guardians, a pediatric bedside neurologic evaluation tool previously described (10) for cerebral edema in DKA was utilized at 3 discrete time points: 1) within the first hour of the intravenous fluid initiation in the emergency department (t=0 hr); 2) 8 hours after initiation of treatment (t=8 hr); and 3) 24 hours after presentation (t=24 hr), by which time there had been metabolic resolution of DKA. The cerebral edema assessment score was divided into 3 categories: diagnostic, major criteria and minor criteria (10). Diagnostic criteria included abnormal motor or verbal response to pain; decorticate or decerebrate posture; and cranial nerve palsy or abnormal breathing patterns. Major criteria included altered mentation or fluctuating levels of consciousness, sustained heart rate deceleration, and incontinence. Minor criteria included vomiting, headache, age younger than 5 years, diastolic blood pressure greater than 90 mm Hg, and not being easily aroused from sleep. An early diagnosis of cerebral edema was suggested by 1 diagnostic criterion, 2 major criteria, or 1 major and 2 minor criteria.

Ultrasound procedures

A single investigator conducted all ultrasonography of the ONSD following each bedside neurologic examination. As described in the literature (17–24), transorbital ultrasonography using a handheld sterile 11 MHz GE 8L-RS linear array transducer (12 to 4 MHz) and a GE Logiq e compact digital ultrasound system (General Electric Healthcare, Wauwatosa, Wisconsin, USA) were used, with an estimated resolution of 0.137 mm. With a thermal index of 0 and a mechanical index up to 0.22, the probe was gently placed on a closed right superior eyelid on the temporal side when the patient was in the prone position. This was repeated 3 times at each time interval (t=0, 8, and 24 hrs). The optic nerve diameter was measured 3 mm posterior to the scleral surface of the globe by using electronic