Case report

Pediatr Emerg Med J 2024;11(2):88-90

https://doi.org/10.22470/pemj.2023.00913

pISSN: 2383-4897 / eISSN: 2508-5506

Pediatric post-concussion bradypnea and hypopnea treated with hypertonic saline: a case report

Utkarsh Fichadia[1], Spartak Mednikov[2]

[1]Emergency Department, Mary Bridge Children's Hospital & Health Network, Tacoma, WA, United States
[2]Pharmacy Department, Mary Bridge Children's Hospital & Health Network, Tacoma, WA, United States

Abstract

Mild traumatic brain injury (TBI) is defined as a Glasgow Coma Scale score of 13-15, and includes concussion, a broad constellation of clinical findings. The clinical findings can include headaches, changes in cognitive function, emotional lability, and cardiovascular, oculomotor, and vestibular dysfunction. While hyperosmolar therapy is a mainstay in medical therapy for moderate to severe TBI, limited evidence exists for its use in mild TBI.

Key words: Apnea; Brain Concussion; Brain Injuries, Traumatic; Craniocerebral Trauma; Saline Solution, Hypertonic

Introduction

Respiratory disturbances as a result of traumatic brain injury (TBI) have been reported in medical literature and in animal studies. This ranges from bradypnea, hypopnea, or obstructive sleep apnea to impact brain apnea following various severities of TBI (1). Intravenous (IV) hypertonic saline (HTS) is an effective therapeutic measure against increased intracranial pressure (ICP) in the setting of moderate to severe TBI (2). Minimal literature exists to support the use of IV HTS in the setting of mild TBI in the absence of space-occupying injuries or lesions (3). To the authors' best knowledge, this case report describes the first documented use of IV HTS solution for the treatment of persistent bradypnea and hypopnea in a concussed adolescent. We obtained written consent for publication from the patient's legal guardian prior to the writing of this case report.

Received: Dec 2, 2023 **Revised:** Jan 11, 2024
Accepted: Jan 11, 2024

Corresponding author
Spartak Mednikov
Pharmacy Department, Mary Bridge Children's Hospital & Health Network, 315 Martin Luther King Jr. Way, Tacoma, WA 98405, United States
Tel: +1-253-403-3755
E-mail: samednikov@multicare.org

Case

A previously healthy, 11-year-old, 38.4 kg girl presented to the pediatric emergency department for evaluation following a head injury. The girl had sustained a fall after she was caught in a dog leash striking the back of her head on the ground. Her family reported a temporary loss of consciousness, subsequent memory deficit, and emesis.

The initial vital signs were as follows: blood pressure, 122/77 mmHg; heart rate, 106 beats/minute; respiratory rate, 26 breaths/minute; temperature, 36.4 ˚C; oxygen saturation, 98% on room air; and a Glasgow Coma Scale (GCS) score of 14. The physical examination was significant for swelling and tenderness of the left occipitoparietal region and superficial abrasions below the left eye and forehead. Given the GCS score and other signs of altered mental status, a computed tomography (CT) scan was obtained, showing a small to moderate-sized soft tissue contusion to the left parietal scalp with

otherwise no intracranial abnormalities.

Immediately after performing the CT scan, the girl developed hypoxia with oxygen saturation rapidly decreasing to 73% on room air and end-tidal carbon dioxide of 20 mmHg secondary to hypopnea and bradypnea (5-6 breaths/minute) when asleep. She responded to verbal and physical stimuli with immediate spontaneous return of intrinsic respiratory drive and resolution of hypoxia. As a precautionary measure, she was placed on supplemental oxygen via nasal cannula at a rate of 2 L/minute. Episodes of bradypnea and hypopnea recurred twice while sleeping, and failed to resolve spontaneously.

Even though the CT did not show any evidence of obvious intracranial abnormalities, there was concern that the girl had subclinical cerebral edema and concussion. A decision was made to try a bolus dose of HTS. She had a sodium concentration of 143 mmol/L drawn 3 days prior during an unrelated outpatient encounter, and a sodium concentration of 141 mmol/L drawn just prior to the administration of HTS. She had a 22-gauge IV catheter in the left antecubital vein not posing a concern for IV infiltration. She received a 115 mL (3 mL/kg) HTS bolus infused over 5 minutes. Within minutes of completing the infusion, the girl awoke spontaneously and became alert with a GCS of 15.

The girl was observed in the emergency department for 2 hours after the administration of HTS and remained stable on room air with no further recurrence of bradypnea or hypopnea. Secondary to her concussive symptoms, she was hospitalized and then discharged home in stable condition after a 13-hour inpatient observation.

Discussion

A concussion is a form of mild TBI that is the result of multiple pathophysiological processes, including but not limited to hypoxia, ischemia, vasogenic edema, excitotoxicity, and increased oxidative stress following the compression and deformation of brain tissue after mechanical injury (4).

The primary mechanisms of HTS in lowering ICP are improved tissue perfusion and oxygenation (5,6). This is due to alterations in blood flow, i.e., reduced viscosity, and physical properties of red blood cells such as a reduction in cell size of approximately 15%. Administration of HTS also reduces the size of endothelial cells leading to vasodilation of small capillaries and reflex arterial vasoconstriction. Furthermore, HTS reduces intracerebral fluid volume by creating an increased osmotic gradient, which causes a fluid shift from the parenchyma across the blood-brain barrier. In an adult study, an ICP lowering effect was observed in as little as 5 minutes after the HTS administration (7). This finding explains the rapid change in clinical picture, which was observed in this current case.

The secondary and more hypothetical mechanisms that have been reported with HTS are neurochemical and immunomodulatory. It is presumed that a rise in extracellular sodium concentration raises the resting membrane potential, reducing depolarization and preventing cell death. In addition, administration of HTS is postulated to reduce neutrophil cytotoxicity further preventing cell death.

The primary risks of HTS administration are associated with its high osmolarity, and warrant reliable IV access to prevent tissue injury in the event of infiltration. Sudden shifts in sodium and serum osmolarity are well tolerated by pediatric patients. If possible, it is prudent to confirm patients' serum sodium concentrations within normal limits prior to administration to avoid the risk of osmotic demyelination associated with chronic hyponatremia.

This case report provides an example of favorable outcome of hyperosmolar therapy with HTS in an adolescent girl with respiratory depression due to mild TBI. It should be noted that the use of HTS may be considered an off-label indication given the currently scant evidence. This highlights a need for further study to support the therapeutic option.

ORCIDs

Utkarsh Fichadia
(https://orcid.org/0009-0007-1629-3193)
Spartak Mednikov
(https://orcid.org/0009-0008-1035-5688)

Conflicts of interest

No potential conflicts of interest relevant to this article were reported.

Funding sources

No funding source relevant to this article was reported.

Author contributions

Conceptualization: UF
Data curation, Investigation, Methodology, Project administration, and Resources: SM
Formal analysis, Supervision, Validation, and Visualization: all authors
Writing-original draft: SM
Writing-review and editing: all authors
All authors read and approved the final manuscript.

Data availability

All data presented in this manuscript are available from the corresponding author upon reasonable request.

References

1. Gelineau-Morel RN, Zinkus TP, Le Pichon JB. Pediatric head trauma: a review and update. Pediatr Rev 2019;40:468-81.
2. Kochanek PM, Tasker RC, Bell MJ, Adelson PD, Carney N, Vavilala MS, et al. Management of pediatric severe traumatic brain injury: 2019 consensus and guidelines-based algorithm for first and second tier therapies. Pediatr Crit Care Med 2019;20:269-79.
3. Lumba-Brown A, Harley J, Lucio S, Vaida F, Hilfiker M. Hypertonic saline as a therapy for pediatric concussive pain: a randomized controlled trial of symptom treatment in the emergency department. Pediatr Emerg Care 2014;30:139-45.
4. Javaid S, Farooq T, Rehman Z, Afzal A, Ashraf W, Rasool MF, et al. Dynamics of choline-containing phospholipids in traumatic brain injury and associated comorbidities. Int J Mol Sci 2021;22:11313.
5. Zipfel J, Engel J, Hockel K, Heimberg E, Schuhmann MU, Neunhoeffer F. Effects of hypertonic saline on intracranial pressure and cerebral autoregulation in pediatric traumatic brain injury. J Neurosurg Pediatr 2021;28:631-7.
6. Sekar A, Jagannath A, Shukla D. Current practice in neurosciences: sugar (mannitol) versus salt (hypertonic saline) - the ideal hyperosmolar agent in traumatic brain injury. Neurol India 2021;3:1-8.
7. Schwarz S, Schwab S, Bertram M, Aschoff A, Hacke W. Effects of hypertonic saline hydroxyethyl starch solution and mannitol in patients with increased intracranial pressure after stroke. Stroke 1998;29:1550-5.