Hypertonic Saline for ICP Reduction in Traumatic Brain Injury Patients: An Evolving Practice

Kirsten Busey, PharmD, BCCCP ■ Kathryn Samai, PharmD, BCPS

The use of hypertonic saline (HTS) for intracranial pressure (ICP) reduction in patients with traumatic brain injury (TBI) has become common in clinical practice despite insufficient evidence to support a recommendation for its use in the Brain Trauma Foundation’s latest Guidelines for the Management of Severe Traumatic Brain Injury (Hays et al., 2011; Ullman et al., 2016). Hypertonic saline has been compared with mannitol, the historical agent of choice, and has been found to be effective in cases of mannitol failure with the potential for a longer duration of ICP-lowering effect (Ware et al., 2005). Unlike mannitol, HTS does not cause volume wasting and functions as an effective volume expander due to its osmotic properties. Unfortunately, the lack of data regarding the use of HTS has resulted in a large variation in how trauma centers utilize and administer this therapy.

Hypertonic saline has a reflection coefficient of 1.0, meaning that it does not readily cross the blood–brain barrier (Rockswold et al., 2009). A bolus of HTS causes an acute rise in serum osmolality, which, in turn, causes the mobilization of free water from the cranial vault into the central vasculature, thus lowering ICP. Hypertonic saline is commercially available in multiple concentrations (3%; 7.5%; 15%; 23.4%) and can be administered by either bolus dosing or continuous infusion. Bolus dosing is the most common method of administration with the most accessible literature. Doses of 120 mEq (30 ml of 23.4% HTS or 250 ml of 3% HTS) are usually given in response to the detection of elevated ICP in patients with an ICP monitor in place or in patients with signs of impending brain stem herniation (Kerwin et al., 2009; Surani, Lockwood, Macias, Guntupalli, & Varon, 2015). Bolus dosing offers the advantage of quickly delivering a large osmolar load, creating the greatest gradient for fluid shift across the blood–brain barrier. The goal of bolus therapy is to reduce ICP to less than 22 mmHg and maintain a goal cerebral perfusion pressure of 60 mmHg (Ullman et al., 2016).

Continuous infusions of more dilute concentrations of HTS such as 3% have also been used on the basis of the concept of providing a consistent osmolar gradient between the cranial vault and the central vasculature, thereby providing continuous ICP control (Qureshi et al., 1998). Continuous infusion of 3% HTS is administered at a low infusion rate with the goal of achieving a serum sodium of 145–155 mEq/L. This practice has been shown to continually reduce ICP by 4–6 mmHg; however, continuous infusion of HTS has not been shown to be effective beyond 48 hr due to the ability of cerebral tissue to adapt to an elevated serum osmolality.

Hypertonic saline therapy must be closely monitored regardless of method of administration. Serum sodium and osmolality should be checked every 4–6 hr. A serum sodium of more than 155 mEq/L or an osmolality of more than 320 mOsm/L should be regarded as the upper limits at which point hyperosmolar therapy should be discontinued and alternative forms of ICP reduction should be explored. Hypernatremia and hyperosmolarity in TBI patients may have adverse effects such as renal failure and pulmonary edema, as well unknown effects on neurological outcomes (Kerwin et al., 2009). In addition, patients must be monitored for the development of fluid-wasting syndromes such as neurogenic diabetes insipidus, which could result in further elevation in serum sodium.

Considering there is a lack of clear guidance for the use of HTS for ICP reduction in TBI patients, trauma centers that utilize this therapy should establish their own guidelines and protocols to ensure patient safety and consistent prescriber practices. Future research should focus on equal comparisons between mannitol and HTS, optimal method of HTS administration, and HTS dosing. Furthermore, the use of HTS therapy for ICP reduction should be tied to functional patient outcomes, as well as mortality, to ensure that this treatment modality results in a better quality of life in the TBI patient population.

REFERENCES


