Emergency anesthesia for evacuating a traumatic acute subdural hemorrhage in a child overdosed with hypertonic saline

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ABSTRACT

A previously healthy 1-year-old child with a traumatic acute subdural hemorrhage received 10 times higher dose of hypertonic saline inadvertently immediately before surgery. This case report describes deviations in fluid management needed to alleviate salt toxicity and its adverse effects during surgery under anesthesia perioperatively. The child made an uneventful recovery with no evident residual damage at follow-up.

Key words: Electrolytes, hypertonic saline, pediatric, subdural hemorrhage

Introduction

Hypertonic saline (HS) temporarily mitigates the effects of raised intracranial pressure (ICP) by reducing brain volume through osmosis. In acute intracranial hemorrhage, this intervention borrows a short time to maintain the “status quo” and proceeds to definitive surgery. This is because the “space” created within the cranial cavity by “shrinking” the brain reduces ICP. However, this temporary “space” will soon be filled with the accumulating hematoma making a second dose virtually ineffective to reduce ICP. Therefore, if HS was used in the event of an acute intracranial bleed, there is an obligatory need to follow with a definitive procedure as soon as possible to accrue any long-term benefit.

The current principles of head trauma management advocate the use of HS for resuscitation and limit the use of crystalloids as it might worsen cerebral edema and raise ICP. On the other hand, salt overdose may induce cerebral demyelination and renal injury. In this case report, in a 1-year-old child with traumatic acute subdural hemorrhage, we describe the deviations in fluid management adopted to minimize the clinical effects of an inadvertent 10 times overdose of HS immediately prior to anesthesia for surgical evacuation of hematoma.

Case Report

A 1-year-old, previously healthy, 10 kg male child, presented to Accident and Emergency (A and E) department following a domestic accident. On admission, he was irritable and drowsy (Glasgow Coma Score = 7) with a mild right-sided weakness and unequal pupils (right 5+ and left 3+). There were no external injuries. As venous access was difficult, an intra-osseous (IO) needle was placed in the right tibia. He was...
On arrival in theater, his systolic blood pressure was 98 mmHg with good pulses. However, his peripheries were mottled, with a pale blue skin. Therefore, venous access was gained through a left femoral triple lumen central line. Arterial pressure monitoring was established through a right femoral 22G cannula. His tympanic membrane temperature was 36.0°C. At this time, it became evident that he had had received 30 ml/kg of (3%) HS instead of the 3 ml/kg dose prescribed in A and E department. Surgery commenced regardless and anesthesia was maintained with O₂/N₂O and sevoflurane and positive pressure ventilation.

He passed a large volume of urine on his bed at the induction of anesthesia. Based on clinical judgment, a total of 400 ml (40 ml/kg) of Hartmann’s solution was infused during surgery in 5 ml/kg boluses to maintain blood pressure during anesthesia. Blood loss was moderate, but a transfusion was not required. An arterial blood gas during surgery recorded a mixed respiratory acidosis (pH - 7.08, PCO₂ -9.2 KPa, PO₂ -31.6 KPa, Na⁺ - 154 mmol/L, K⁺ - 3.0 mmol/L, Cl⁻ - 129 mmol/L, Ca²⁺ -1.36 mmol/L, glucose - 14.1 mmol/L, lactate - 0.8 mmol/L, hemoglobin [Hb] - 10.0 g/L, base excess - 9.8). The highest serum sodium recorded during the surgery was 158 mmol/L. Hb was 8.0 g/dl at the end of the surgery. His right fronto-occipital hematoma was evacuated. He was transported to the pediatric Intensive Care Unit (PICU), ventilated, and sedated with midazolam 3 mcg/kg/min and morphine 20 mcg/kg/h infusions.

He was extubated 2 h after surgery in the PICU. His postoperative fluid intake was maintained at 2/3rd of daily requirement orally. He demonstrated a good urine output of 1-2 ml/kg/h. SaO₂ was 98%–100% self-ventilating room air, respiratory rate was 24–28 bpm, there was no respiratory distress, heart rate was 140/min, and blood pressure was 112/66 mmHg. Pupils were equal and reacted to light. He was awake and alert. His electrolytes showed a normal Na⁺ (139–141 mmol/L) but a lower and normalizing K⁺ (3.4, 3.6, 3.8 mmol/L) during the next 24 h. He developed a single temperature spike to 38.8°C that resolved with paracetamol. The drug error with regard to HS overdose was explained to parents in keeping with the duty of candor. He made an uneventful recovery with no detectable residual defects either due to subdural hematoma or due to salt overdose. There was no evidence of renal injury or residual brain damage during the immediate postoperative period or at subsequent follow-up after 1 month.

**Discussion**

In the event of a traumatic acute intracranial bleed, the highest priority is to evacuate the accumulating hematoma and achieve hemostasis while maintaining adequate cerebral perfusion. This is because rising ICP reduces cerebral blood flow and leads to irreversible brain damage and even death. Therefore, there was no option but to embark on surgery in this child. The HS overdose had to be managed concurrently during surgery. There was a need to match the diuresis with an unanticipated amount of isotonic fluid (400 ml of Hartmann’s in this case) to maintain a favorable cardiovascular physiology under anesthesia. Irrespective of the advocated reduced crystalloid use in head trauma,[31] the proactive use of Hartmann’s solution was prudent retrospectively in this child as it was isosmotic and countered the excessive loss of potassium expected with the subsequent diuresis. Isotonic Hartmann’s solution also prevented worsening of cerebral edema through a reverse osmotic process. This improved his fluid balance. Furthermore, the high chloride in blood was mimicking a nonanion gap acidosis. Its treatment includes bicarbonate. In this context too, Hartmann’s solution as a replacement fluid was more appropriate, as in the presence of normal liver function, its lactate would be converted to HCO₃⁻. This mitigated the effects of nonanion gap acidosis.

During the root-cause analysis, we noted that the overdose of HS occurred following infusion of a 300 ml 3% solution bag directly connected to the patient without an intervening mechanism such as a burette or a programed pump to limit it at 30 ml. Such salt poisoning can be fatal.[32] It is also known to cause acute renal injury. Reports of salt overdose are scarce.

A clinical status with blue-mottled skin and cold peripheries simulates septic shock even with normal blood pressure and core temperature. In this child, this clinical feature may have resulted from hyperosmolar circulation leading to reduced extravascular fluids and “shrunk” skin. The end result was a sluggish skin circulation leading to a pale, mottled, blue skin. This seems a clinical feature of HS overdose.

Osmotherapy constitutes the cornerstone of medical therapy of cerebral edema irrespective of the etiology of brain injury, and HS is superior to mannitol.[19] HS has now become the standard treatment in traumatic brain injury to manipulate ICP. HS, in combination with colloid, has been used in Europe for trauma resuscitation for many years. Hypertonic solutions restore disturbed macro- and micro-circulation in hypovolemic states. Even small amounts of HS induce a relevant fluid shift from the extravascular space into the intravascular space. This was the principle of “small-volume resuscitation” in acute hypovolemia and hypovolemic shock.[33] It is more effective than mannitol in reducing elevated ICP given as either a bolus or continuous infusion.[31] The use of HS is gaining acceptance in the neurosciences critical care based on its efficacy in reducing cerebral edema and its favorable hemodynamic profile.
Observations are similar in pediatrics. Hyperchloremic metabolic acidosis is an observed complication of its use. The treatment of hyperchloremic metabolic acidosis includes bicarbonate-containing solutions accompanied by potassium replacement to avoid severe hypokalemia. This avoids hypokalemia-associated cardiac arrhythmias and muscular paralysis due to the rapid introduction of potassium into the cells. In the presence of normal liver function, Hartmann’s solution seems ideal for this purpose as noted in our case. Furthermore, in hyperchloremic acidosis (also termed acute nonanion gap metabolic acidosis), there is no blood pH and/or serum bicarbonate level to guide the initiation of treatment.

The use of infusions of 3% HS (Na+ = 514 mEq/L) and sustained hypernatremia and hyperosmolality is safely tolerated in pediatric patients with traumatic brain injury. However, malignant edema formation late in the course of intracerebral hemorrhage after prolonged administration of HS may occur possibly due to a rebound phenomenon of hyperosmolar therapy. Other recognized adverse effects of supra-physiologic hyperosmolality include renal failure, pulmonary edema, or central pontine demyelination. However, we did not encounter any of these complications in our child.

Based on animal experiments, the use of 5 ml/kg of HS is considered safe. It maintains serum sodium concentration under 160 mmol/L. In our case report, the HS overdose was 30 ml/kg and its adapted management within the immediate 1-3 h (Hartmann’s solution 40 ml/kg) helped maintain serum Na+ below 160 mmol and curtailed the effects of acute hyperchloremic acidosis.

In head injury, following a single dose of HS, the ICP falls immediately after initiation of infusion with further significant decreases observed at 20 and 60 min. Rapid infusion of further daily single doses of HS is considered a safe treatment of elevated ICP in severe head injury. HS has clinically desirable physiological effects on cerebral blood flow, ICP, and inflammatory responses in models of neurotrauma. Our child did not require any further dose of HS postoperatively.

Hyperosmolality of plasma above 320 mosm/L is also recognized as a contributory factor for detrimental outcomes including death. In our case, more aggressive use of Hartmann’s solution prevented a rise of serum sodium above 158 mmol and this is important to keep plasma osmolality within acceptable limits as above.

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Conflicts of interest
There are no conflicts of interest.

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