Pediatric Anesthesia and Critical Care Journal 2021;9(2):69-74
doi:10.14587/paccj.2021.12

PACCJ

Anesthetic care of a child with severe hypernatremia

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Keypoints

1. A normal serum sodium concentration is maintained through the thirst mechanism, free water and sodium intake, balanced by sodium and water excretion. Free water excretion and sodium reabsorption are regulated primarily by aldosterone and antidiuretic hormone (ADH) through their effects on the tubules and collecting ducts of the kidneys.
2. Physiologic manifestations of severe hypernatremia include extreme thirst, confusion, lethargy, coma, subdural hemorrhage due to rupture of bridging veins, and dural sinus thrombosis.
3. In the adult population, a preoperative serum sodium ≥ 150 mEq/L has been shown to be independently associated with mortality with an odds ratio of 3.4 while no difference was noted in those patients with hyponatremia.
4. In patients with hypernatremia, rapid intravenous fluid administration and correction of the serum sodium can lead to fluid shifts, causing cerebral edema, increased intracranial pressure, and death.

Abstract

Severe electrolyte derangements are routinely corrected prior to elective anesthetic care. However, in urgent and emergent situations, anesthetic care may be required even with electrolyte disorders. Derangements in free water and sodium intake and excretion can lead to hypernatremia. Clinical manifestations of hypernatremia include extreme thirst, confusion, lethargy, coma, subdural hemorrhage, and dural sinus thrombosis. We present a 9-year-old patient with severe hypernatremia who required urgent anesthetic care during radiologic imaging. The physiologic mechanisms of sodium and water homeostasis are discussed, the etiology of hypernatremia reviewed, and suggestions for clinical care presented.

Keywords

sodium, hypernatremia, anesthesia

Introduction

Sodium is an essential cation involved in cell membrane stability and the regulation of plasma osmolarity.[1] The serum sodium concentration is maintained within the range of 135-145 mEq/L through the thirst mechanism, free water and sodium intake, and balanced by free water and sodium excretion.[2] Free water excretion and sodium reabsorption are regulated primarily by antidiuretic hormone (ADH) and aldosterone through their effects on the tubules and collecting ducts of the kidneys.[3] Hypernatremia is defined as a serum sodium concentration exceeding 145 mEq/L.[4] In the adult population, a

preoperative serum sodium ≥ 150 mEq/L has been shown to be independently associated with mortality with an odds ratio of 3.4 while no difference was noted in those patients with hyponatremia.[5]

Physiologic manifestations of severe hypernatremia (≥160 mEq/L) include extreme thirst, confusion, lethargy, coma, intracranial bleeding.[1] The most serious complication of hypernatremia is subdural hemorrhage due to the rupture of bridging veins and dural sinus thrombosis.[1] Rapid intravenous fluid correction can cause cerebral edema, increased intracranial pressure, and death.[6] Identifying the etiology of hypernatremia while assessing volume status and laboratory studies are necessary to guide appropriate anesthetic care. Although in elective cases, anesthetic care can be delayed, allowing for correction of hypernatremia, urgent or emergent cases must proceed. We present a 9-year-old patient with severe hypernatremia who required urgent anesthetic care during radiologic imaging. The physiologic mechanisms of sodium and water homeostasis are discussed, the etiology of hypernatremia reviewed, and suggestions for clinical care presented.

Case report

Review of this case and presentation in this format followed the guidelines of the Institutional Review Board of Nationwide Children's Hospital (Columbus, Ohio). The patient was a 9-year old, 46.6 kilogram male, presenting for urgent magnetic resonance imaging of the abdomen and pelvis for evaluation of recurrent perianal abscesses. His past medical history was significant for autism, chronic diarrhea, asthma, hearing loss and the recent development of a perianal abscess. Ten days prior to admission, he developed difficulty swallowing and was diagnosed with streptococcal pharyngitis which was treated with a dose of intramuscular penicillin. He continued to have decreased oral intake and lethargy. Upon presentation to emergency department, he was tachycardic with a heart rate of 128 beats/minute, his mucosa membranes were dry, and there was a large right-sided perineal abscess. He was subsequently admitted to the Intensive Care Unit (ICU) due to acute renal insufficiency secondary to hypernatremic dehydration. The initial serum sodium level was 169 mEq/L. He was placed *nil per os* and rehydration was started with 0.9% saline. Plans were made for an urgent MRI of the pelvis. Antibiotic therapy was started with clindamycin and ampicillin-sulbactam. Laboratory evaluation 3-4 hours after admission to the Pediatric ICU and prior to anesthetic care revealed sodium 164 mEq/L, chloride 134 mEq/L, Blood Urean Nitrogen (BUN) 50 mg/dL and creatinine 1.37 mg/dL. Preoperative physical examination including the airway, heart and lungs was unremarkable except for dry mucous membranes. The blood pressure was 122/72 mmHg, heart rate was 107 beats/minute, respiratory rate was 18 breaths/minute, and his oxygen saturation was 99%. The patient was transported to the operating room, routine American Society of Anesthesiologists' monitors were placed. After pre-oxygenation, anesthesia was induced by the intravenous administration of propofol (3 mg/kg) and lidocaine (1 mg/kg). Neuromuscular blockade was provided by cis-atracurium (0.3 mg/kg). A modified rapid sequence induction was performed with cricoid pressure and bag-valve-mask ventilation. Direct laryngoscopy was performed followed by endotracheal intubation with a 6.0 mm cuffed endotracheal tube. Anesthesia was maintained with isoflurane (expired concentration 0.5-2%) in airway and oxygen. Vital signs remained stable during the imaging procedure. Total anesthesia time for the procedure was 212 minutes. Intraoperative fluids included a 500 mL of 0.9% saline. Estimated blood loss was minimal. At the completion of the procedure, residual neuromuscular blockade was reversed with neostigmine and glycopyrrolate. Ondansetron was administered to prevent postoperative nausea and vomiting. The patient's trachea was extubated in the operating room and he was transported to the post-anesthesia care unit (PACU). Postoperatively, the patient's sodium level remained elevated at 163 mEq/L. Postoperatively, intravenous fluids were provided at 1.5 times maintenance with lactated Ringers (LR) and 0.45% sodium chloride, the

rates of which were adjusted to ensure a gradual decrease in the serum sodium level which was measured every 2-4 hours. The MRI of the pelvis showed a rim-enhancing fluid collection involving the right gluteal cleft with a diameter of 5.5 cm, no anal canal involvement, and no fistula or perirectal abscess. The following day, the patient was returned to the operating room for an incision and drainage of the abscess. On the morning of the surgical procedure, the patient's serum sodium level had decreased to 156 mEq/L. Other laboratory values included serum chloride 122 mEq/L, BUN 23 mg/dL and creatinine 1.15 mg/dL. In the perioperative unit, his vitals were stable with a temperature of 37.4ºC, pulse 97 beats/minute, respirations 16 breaths/minute, blood pressure 120/72 mmHg and oxygen saturation at 99%. Physical examination including the airway, heart and lungs were unremarkable. The patient was transported to the operating room and routine American Society of Anesthesiologists' monitors were placed. Anesthesia was induced by the intravenous administration of propofol (3 mg/kg) and fentanyl (1 µg/kg). Bag-valve-mask ventilation was provided and the patient's trachea was intubated. Anesthesia was maintained with sevoflurane (expired concentration 0.4-4%) in air and oxygen. The right-sided perineal abscess was incised and drained. There were no intraoperative problems. Estimated blood loss was minimal. Intraoperative fluids included 90 mL of 0.45% saline and 50 mL of LR. Total anesthesia time was 94 minutes. The patient was transferred to the PACU and then the inpatient ward where intravenous hydration was continued. Postoperative pain was managed with oral acetaminophen and oxycodone as needed. The wound culture was positive for *Streptococcus anginosus* and antibiotic therapy was narrowed to intravenous ampicillin followed by oral amoxicillin. The patient's postoperative serum sodium was 155 mEq/L. Fluid replacement was continued with a combination of 0.45% sodium chloride and LR to ensure a continued gradual decline in the serum sodium value. Serum sodium values were measured every 2 hours. The patient was transitioned to a regular diet. The serum sodium value reached a normal range of 143 mEq/L on the 4th day of hospitalization. Fluid therapy was switched to 5% dextrose in 0.45% sodium chloride. On day 11 of hospitalization, the patient was discharged home with no further abnormalities of sodium regulation noted.

Discussion

Normal sodium homeostasis is maintained through intricate feedback loops that involve the central nervous system, endocrine organs (adrenal and pituitary), and the kidneys.[7,8] Plasma sodium concentrations are tightly maintained within a range of 135-145 mEq/L despite variation in water and salt intake. In general, serum sodium is regulated by control of the balance between water and sodium intake versus elimination and loss. Hypernatremia represents a deficit of water in relation to sodium. This can occur from either a gain of sodium or a net loss of water, with the latter being more common. In the pediatric patient, hypernatremia occurs most frequently in the setting of dehydration demonstrating that intravascular volume and serum sodium levels do not generally correlate. Dehydration and the resulting hypovolemia may occur with hypernatremia, hyponatremia, or a normal serum sodium. Sodium gain, an uncommon cause of hypernatremia, generally results from therapeutic interventions such as the administration of sodium bicarbonate to treat acidosis or the administration of hypertonic saline.

In general, increased thirst and intake of free water rapidly corrects the trend toward hypernatremia. Osmoreceptors located at the subfornical organ of the thalamus are sensitive to changes in the serum osmolality.[9,10] The subfornical organ is linked to the primary site of thirst regulation called the lamina terminalis of the forebrain. Hypernatremia activates the osmoreceptors and triggers the thirst mechanism in the lamina terminalis. Sustained hypernatremia occurs only when osmoregulation and mental status is altered causing a disruption in the thirst mechanism or when access to free water is limited. In these circumstances, the intake of free water may be

inadequate to replace losses from the GI tract, skin or kidneys resulting in hypernatremia. The groups at highest risk are patients with altered mental status, hospitalized patients receiving parenteral fluids and medications, infants, and the elderly. Hypernatremia in infants usually results from diarrhea or acute illnesses that limit the intake of fluid resulting in associated dehydration and hypernatremia. Rarely, excessive water loss occurs in the kidneys related to deficits of ADH or lack of renal response to its action (central or nephrogenic diabetes insipidus). Measurement of urine and plasma osmolality, urinary specific gravity, and the urine sodium concentration may be helpful in determining the etiology if the etiology is unclear from the patient's history.

Free water balance (reabsorption versus excretion) is governed primarily by ADH, also known as vasopressin.[11,12] ADH functions with the osmoreceptors and thirst centers to maintain water and sodium homeostasis. Even a slight increase in serum osmolality results in the release of ADH from the posterior pituitary gland. A decrease in mean arterial pressure also activates the osmoreceoptors which act through the vagus nerve to stimulate ADH release. ADH acts on the renal V2 receptors which are present on principal cells in the collecting ducts and tubules.[5] In response, aquaporin channels of the principle cells open to increase water reabsorption, thereby decreasing plasma osmolarity and serum sodium levels. As water is reabsorbed in the collecting ducts, urine becomes more concentrated and the blood pressure increases. The opposite occurs in hypervolemia or hyponatremic states. Rarely, hypernatremia results from defects in ADH secretion (central diabetes insipidus) or resistance to its effects on the renal tubules (nephrogenic diabetes insipidus). In these conditions, hypernatremia will occur if water losses are not corrected. These conditions can generally be identified through a thorough history and also an evaluation of serum and urine osmolarity and sodium values.

Sodium excretion and urine flow are also regulated by the glomerular filtration rate (GFR), which is controlled by complex neural and humoral mechanisms involving the kidneys, endocrine system, autonomic nervous system, and baroreceptors.[13] The kidneys are responsible for filtering large amounts of sodium and small changes in GFR can cause significant changes in plasma sodium. Sodium reabsorption occurs in the proximal tubules and counters the effects of sodium excretion. Sodium excretion rates are increased or decreased based on the serum sodium. A low extracellular fluid (ECF) volume or low serum sodium is detected by cardiopulmonary baroreceptors located at the atria, ventricles and pulmonary interstitial tissue.[8] In contrast, aortic and carotid baroreceptors are sensitive to high ECF volume. Intrarenal receptors in macula densa cells, located in the juxtaglomerular apparatus, are also sensitive to low ECF and sodium levels.[9] Together, these receptors respond to low serum sodium by increasing renin secretion which activates the renin-angiotensin-aldosterone system.[5] Next, angiotensinogen is converted to angiotensin II by a sequence of renin and angiotensin-converting enzyme (ACE). Angiotensin II induces vasoconstriction and signals to the adrenal cortex to secrete aldosterone. Aldosterone induces salt and water retention by increasing sodium reabsorption of the collecting tubules and thereby restoring serum sodium and ECF volume.

In patients presenting with hypernatremia, appropriate treatment depends on the degree of dehydration present, the underlying etiology, the initial serum sodium, and the rapidity with which it increases. Especially in patients requiring anesthetic care, the primary goal is to restore intravascular volume with isotonic fluids based on the clinical assessment of the degree of dehydration. The assessment of the degree of dehydration is based on clinical assessment and baseline laboratory parameters indicative of end-organ perfusion. The parameters include acid-base status such as base deficit (serum bicarbonate), lactate, BUN, creatinine, and urine specific gravity.[14,15] Our patient presented with several clinical signs of dehydration ranging from tachycardia, dry mucous membranes, cool extremities, and delayed capillary refill of 3-4 seconds.

Laboratory tests including serum sodium, serum bicarbonate, and urine specific gravity (1.029) confirmed the clinical impression. In the presence of hypernatremia, the body compensates by accumulating active substances, also called idiogenic osmoles, within the cells of the brain to balance the increased osmolarity of the plasma.[12,3] If the serum sodium decreases too rapidly, there will be a shift of fluid from the extracellular space intracellularly with the development of cerebral edema and increased intracranial pressure or the development of central pontine myelinolysis.[3] Therefore, correction of hypernatremia must be gradual to prevent sudden fluid movement in the brain cells.

Initial treatment includes resuscitation with isotonic fluids to restore intravascular volume followed by slow correction of the serum sodium over 48 hours at a rate of no greater than 0.5 mEq/L per hour or 10-12 mEq/day. The Edelman equation can be used to estimate the free water deficit or the amount of positive water balance required to return the serum sodium to 140 mEq/L.[16] Using this approach, 50% of the water deficit is replaced over the first 24 hours followed by the remaining over the next 48-72 hours. The choice of fluids for replacement is equally important. Normal saline (0.9% NS) is initially preferred for volume resuscitation over less hypotonic fluids. Alternative solutions including Normosol®-R or Plasmalyte® provide not only physiologic amounts of sodium (140 mEq/L), but have the added advantage of providing buffers (acetate and gluconate). These added buffers may limit the dilutional acidosis that is seen when large volumes of 0.9% normal saline are administered. Although lactated Ringer's is frequently used for volume resuscitation, its serum sodium concentration of 130 mEq/L is relatively hypotonic to the high serum sodium in hypernatremic dehydration and should be avoided for initial bolus administration. Regardless of the fluid chosen and its rate of administration, frequent monitoring of serum sodium concentration, neurologic status, and intake/output are necessary.

For our patient, initial intraoperative resuscitation and fluid administration included 0.9% normal saline as the initial serum sodium was 164 mEq/L. This was followed postoperatively by a combination of lactated Ringer's and 0.45% saline to provide slow repletion of the fluid deficit as well as ongoing maintenance fluids. The administration rates of these two solutions were adjusted based on the decrease in the serum sodium concentration. During the rehydration phase, serum sodium was followed frequently. During our patient' second procedure, intravascular volume had been repleted and the perioperative serums sodium level had decreased to 156 mEq/L. As third space losses were minimal during the surgical procedure, ongoing maintenance fluids were provided by the combination of LR and 0.45% saline.

In summary, we present the perioperative care of a child with severe hypernatremic dehydration. A preoperative serum sodium ≥ 150 mEq/L has been shown to be associated with an increased risk of perioperative mortality in adults. Physiologic manifestations of severe hypernatremia may include extreme thirst, confusion, lethargy, and coma. Life-threatening central nervous system complications include subdural hemorrhage due to rupture of bridging veins and dural sinus thrombosis. Perioperative care includes initial intravascular resuscitation with isotonic fluids (0.9% normal saline) followed by correction of the free water deficit with a goal of decreasing the serum sodium by no greater than 10-12 mEq/day. Rapid intravenous fluid administration and correction of the serum sodium can result in cerebral edema, increased intracranial pressure, and central pontine myelinolysis.

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