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Severe hyponatremia in a hospitalized child: Munchausen by proxy

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Abstract

An 8-week old child presented to a referring institution with profuse diarrhea and infectious enteritis for one week. He was initially treated for suspected *Salmonella spp* sepsis and meningitis as it was found in the stool, but the child's illness progressed manifested by paroxysmal profuse diarrhea and increased urine output. After several weeks, he suffered a sagittal venous thrombosis and intracranial hemorrhage. Subsequently the child was transferred to our institution for intestinal evaluation. The patient's diarrhea and excessive diuresis resolved and his sodium normalized soon after transfer. After his mother arrived 4 days later however, he immediately developed severe hyponatremia (serum [Na⁺] 214 meq/L), with resumption of diarrhea and excessive diuresis. A gastric aspirate during the crisis demonstrated an extremely high sodium content ([Na⁺] = 1416 mEq/L), consistent with salt intoxication. Surveillance of mother revealed that she manipulated the indwelling nasogastric tube and she admitted to salt administration upon confrontation. This case describes one of the varieties of ways Munchausen Syndrome by proxy can manifest with profound neurological sequelae, and highlights the need for close observation and swift intervention when sufficient cause is present.

Introduction

Hyponatremic dehydration is common in children, especially during pathological conditions such as infectious gastroenteritis or inadequate fluid intake. The underlying pathophysiology involves losses of free water in excess of sodium, leading to profoundly decreased total body water but relatively conserved total body sodium. Slow rehydration to restore intravascular and intracellular volume and careful correction of hyperosmolality accomplished over several days leads to resolution of symptoms, as the enteritis resolves in the vast majority of cases without significant sequelae. Less commonly, complications attributed to excessively rapid rehydration and correction of serum osmolality lead to central nervous system consequences such as osmotic myelinolysis. In exceptional cases, an unremitting course of diarrheal illness and hyponatremia of unclear etiology can persist

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with protracted symptoms requiring more comprehensive evaluation. This case details the clinical course of a child initially managed for hypernatremic dehydration and enteritis, with profound episodic hypernatremia over several weeks.

Case Report

At 8-weeks of age a male child was admitted to an inpatient pediatric unit after a week of non-bloody emesis and diarrhea for intravenous rehydration. By report, he had an uneventful first several weeks of life following an uncomplicated pregnancy and birth and his family had pet reptiles. Within a day after admission, the patient demonstrated severe dehydration, changes in mental status, and altered serum electrolytes ($[Na^+] = 166$ meq/L, $[Cl^-] = 142$ meq/L, glucose = 713 mg/dl). This proved refractory to fluid and electrolyte management. Shortly thereafter, he developed seizures which necessitated intubation and several days of mechanical ventilation for which he was transferred to the pediatric ICU. A magnetic resonance venogram (MRV) one week after admission revealed sagittal sinus thrombosis and thromboses of the straight sinus, vein of Galen and medullary veins (Figure 1). For this he was treated with enoxaparin because of concerns that thrombus extension could worsen neurological outcome. His seizures were confirmed electrographically, and phenobarbital and levetiracetam were administered. Subsequently, *Salmonella sp.* was isolated from stool culture a week after admission and he was treated for presumed sepsis and meningitis from this pathogen based on his clinical picture of shock and altered level of consciousness at the time of presentation. An exposure to *Salmonella* may have been his family's pet reptiles. Concurrent blood cultures were sterile and cerebrospinal fluid was not obtained at that time secondary to technical difficulties. A total 6-week course of cefotaxime was administered and follow-up cultures (blood and stool) were negative for pathogens. Subsequent CSF analysis a month later was performed because of ongoing diarrhea, and revealed WBC 11 cells/mm³ (29% lymphocytes, 71% monocytes), RBC 9 cells/mm³, CSF glucose 63 mg/dl, serum glucose 100 mg/dl, and protein 96 mg/dl (consistent with either resolving meningitis and/or residual effects of sinus venous thrombosis). A repeat brain MRI approximately one month after admission demonstrated severe parenchymal injury from the initial injury, sagittal vein thrombosis and subsequent hemorrhagic transformation of the venous infarction (Figure 1). Additionally, wide fluctuations of serum sodium, (attributed to episodic profuse diarrhea (at times 17 stools per day followed by brisk diuresis) and the use of enoxaparin may have played some role in the evolution of the brain injury. Despite treatment for *Salmonella*, the diarrhea persisted, and required stool and urine replacement to maintain intravascular volume. Gastrointestinal endoscopy demonstrated only slight blunting of intestinal villi and grossly normal mucosa. After a two-month hospitalization, he was transferred to our institution for further evaluation of his diarrhea.

Upon arrival, brisk urine output (48 ml/kg/hr) was noted without significant stool output. Admission serum sodium was 135 mEq/L and other electrolytes were within the physiological range. Hourly urine and stool output was replaced with intravenous replacement fluids (requiring up to nine liters in the first days of admission), which was the referring institution's fluid replacement strategy. His neurologic exam demonstrated dysconjugate upward gaze, inability to fix on objects, and mild hypertonicity. His anticonvulsant drug regimen of phenobarbital, oxcarbazepine and levetiracetam was continued at standard doses, and he demonstrated no clinical seizure activity. A brain MRI obtained after transfer demonstrated intracerebral hemorrhage, significant loss of left parietal brain parenchyma with cysts and fluid collections in the area of the hypothalamus (Figure 2). The father arrived to our institution with the child but the mother was delayed in traveling until hospital day 4.

The therapeutic plan at our institution included consideration of (i) octreotide administration for reported secretory diarrhea and (ii) arginine vasopressin administration for possible central diabetes insipidus due to significant neurological injuries. During the first three days, minimal stool output was noted and octreotide was transiently required. Arginine vasopressin was administered secondary to his profuse, hypoosmolar urine (urine osmolality of 140 mosm/L while serum osmolality was 294 mosm/kg) decreasing urine output from 35 ml/kg/h to 2 ml/kg/hr. By the fourth hospital day after transfer, his clinical condition had stabilized considerably with normal urine output and $[\text{Na}^+] = 142 \text{ mEq/L}$.

The infant's mother arrived soon thereafter, and within 24 hours the child abruptly developed profuse diarrhea and worsening hypernatremia (Figure 3). Over 24 hours, all sodium was removed from intravenous fluids. Despite these measures, serum sodium level continued to rise sharply to a peak level of 214 mEq/L as his mother remained at the bedside. No exogenous sodium was being administered via IV fluids, and the infant's urine and stool output were being replaced hourly with equivalent amounts of free water precluding hypernatremic dehydration. We therefore suspected the enteral administration of a sodium-containing solution, and a gastric aspirate from the nasogastric tube was sent to the laboratory for evaluation of the $[\text{Na}^+]$ of the gastric fluid. The $[\text{Na}^+]$ concentration of the aspirate was 1416 mEq/L and the $[\text{Cl}^-]$ was 1338 mEq/L. Simultaneously, stool $[\text{Na}^+]$ was noted to be 896 mEq/L and serum aldosterone was decreased ($< 1.6 \text{ ng/ml}$). Over the next 24 hours, urine osmolality increased from 140 mOsm/kg to 980 mOsm/kg and urine $[\text{Na}^+]$ increased to 337 mEq/L. These laboratory findings led us to conclude that salt intoxication had occurred, with a high probability that it was caused by the enteral administration of sodium-containing solutions.

We urgently evaluated the sodium content of all medications the child had received in the preceding 24-hour period and found no abnormalities. Left only with the possibility of intentional salt poisoning perpetrated either by family or staff, we contacted child protective services and local law enforcement officials. A plan of surveillance of all visitors for the child was arranged. As a part of this plan, the infusion port of the nasogastric tube was securely closed, and supervising nurses and physicians closely monitored the patient. Within 12 hours of occluding the NG tube, the mother complained in writing about being prevented from manipulating the nasogastric tube. She was then observed leaning over the child with a blanket on her shoulders and was confronted by the ICU staff and hospital police. The infant's NG tube was found open, and in mother's possession was a fluid-filled syringe. The syringe was confiscated and later determined to contain an extremely high concentration salt solution. In addition, the mother's purse contained sodium chloride tablets, oral rehydration solution and syringes. She had no prior history of abusive behavior towards children. Law enforcement personnel subsequently took the mother into custody.

The patient's serum sodium gradually normalized through the remainder of his stay with ongoing replacement therapy, and arginine vasopressin was discontinued. Slow re-feeding was successful and he was transferred back to his referring institution for long-term neurological, neurosurgical and developmental interventions for his brain injuries.

Discussion

The differential diagnosis of profound hypernatremia and dehydration in an infant is long. Infectious (bacterial, parasitic or viral) enteritis can increase free water loss and result in hypernatremia. Hypernatremic dehydration can also occur from ingestion of complex sugars, inappropriately reconstituted infant formula and inadequate breast feeding [1]. In a large series, Finberg and colleagues described a catastrophic case where salt was mistakenly exchanged for sugar in the hospital's formula room [2]. The resulting formula had a $[\text{Na}^+]$ of

919 mEq/L. Of the 14 children affected, 6 died and all autopsies demonstrated intracranial hemorrhage. Of the survivors, one demonstrated a serum $[Na^+]$ of 274 mEq/L. Several case reports from the 1960s involve fatal salt intoxication at a time when table salt was recommended as an emetic in poisoning [3]. Additional causes of hypernatremia include diabetes insipidus (central or renal in origin), increased insensible water losses (burns, fever), and diuretic use among others.

Munchausen Syndrome by proxy (MBP) was a term coined by Meadow in 1977 [4] to describe illness-producing behavior of caregivers reminiscent of Munchausen syndrome. Since then, MBP has been defined as “the intentional production or feigning of physical or psychological signs or symptoms in another person who is under the individual’s care for the purpose of indirectly assuming the sick role” [5]. More recently, two forms of MBP have been defined specifically for children – pediatric condition falsification (PCF) and factitious disorder by proxy (FDP) [6–7]. The diagnosis of PCF is made when an adult falsifies physical or psychological symptoms and includes the adult causing medical conditions by their actions, over- or under-reporting of actual symptoms of the child or coaching the victim to alter the physical signs or symptoms. FDP entails an adult who falsely reports symptoms of nonexistent illnesses, and get secondary gain from “fooling” the medical caregiver as an authority figure.

Determining the incidence of MBP is difficult. In a comprehensive survey within the UK, Davis and colleagues [8] describe an incidence of 0.5 cases per 100,000 children (< 16 y). Of the 120 cases, 39 were the victims of poisoning by a caregiver and a majority of victims were under 5 y of age (92/120; 76.6%). Sheridan and colleagues [9] reviewed the literature and found 451 cases of MBP reported in over 154 journal articles. In this review, only about 2% presented with hypernatremia but approximately 20% had symptoms of diarrhea as our case did. They also found that the biological mother was the predominant perpetrator in cases with known outcomes (76.5%), a 8% mortality/severe disability rate, average duration of abuse was 21 months, and an average age of 48 months for victims. One can surmise MBP is neither uncommon nor benign and requires education and the vigilance of pediatric health care workers to make the diagnosis as early as possible to protect the health of the victim.

While MBP has been described in cases of disorders of sodium metabolism [7, 10–12], its occurrence in a closely monitored intensive care unit is unique. Meadow [11] described twelve cases of intentional salt poisoning which bear the closest association with our case. Of these, the median age was 2.5 months, 4 children achieved peak serum $[Na^+] > 200$ mEq/L, 7 had other fabricated injuries proven and 2 died. One child in this series had salt administered directly into a nasogastric feeding tube, while others were found to have extremely high concentrations of salt in prepared formula or fruit juices. It is unclear if any of these instances occurred while in a medical facility, and it appears clear that none occurred while in an intensive care unit for several weeks. Kamerling and colleagues [13] have also described MBP within a Pediatric Intensive Care Unit in a military hospital, as the mother was observed intentionally extubating the child. Though a perpetrator’s abusive activities are difficult in a highly monitored setting, this case and ours clearly illustrate it is not impossible for the motivated individual.

For our patient, the mother demonstrated some behaviors that are consistent with a MBP caregiver, particularly hyper-attentiveness to the child, claimed symptoms of a rare undiagnosable disorder, symptoms in presence of the parent only and symptoms refractory to usual treatments [14]. However, other characteristics not observed included (i) less parent worry about disease than medical staff, (ii) prehospital information discrepancies, (iii) history of unexplained infant death or similarly ill children in the home and (iv) caregiver

extensive prior medical experience [7]. Ultimately, as in many cases of MBP, confronting the perpetrator leading to confession of the alleged acts is the definitive action that can be taken by the medical care team.

In summary, our case illustrates that physicians should be alert that bizarre and unexplained complexes of symptoms may be a manifestation of MBP, and prompt diagnosis of this condition is the only hope toward improving outcome for the victims of this behavior.

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Figure 1.

MRI image of end-stage brain injury that resulted from (i) seizure at presentation, (ii) sagittal venous thrombosis and (iii) hemorrhagic transformation of stroke. It is possible that wide fluctuations of serum sodium observed at the outside institution and our own contributed to the evolution of the brain injury observed.

Figure 2. Temporal profile of serum sodium concentration (lower line graph) and serum osmolality (upper line graph) through 15 days of admission at our institution.