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Hypernatremia and Subdural Hematoma in the Pediatric Age Group: Is There a Causal Relationship?*

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ABSTRACT: Several researchers in the 1950's proposed that hypernatremia causes water to leave brain cells, shrinking the brain, thus tearing the bridging veins and resulting in subdural hematomas. Although the old literature suggests mechanisms linking the two in a cause and effect relationship, there is controversy as to whether hypernatremia leads to subdural bleeding or whether the reverse is true. This issue is important for forensic pathologists who must distinguish natural disease from trauma. An etiologic link between hypernatremia and subdural hematomas was suggested recently, and was proposed originally before Kempe's 1962 paper "The Battered Child Syndrome" which widely disseminated the concepts of child physical abuse, and of subdural bleeding resulting from non-accidental injury. Our study is a multifaceted investigation of infants which includes: a literature review, retrospective chart reviews of both living and deceased hypernatremic infants, a retrospective review of infants hospitalized with subdural hematoma, and a prospective collection of head injured, hypernatremic children. We conclude that hypernatremia, if present in association with subdural hemorrhage, is most likely secondary to intracranial pathology, and that hypernatremia often develops in critically ill infants suffering from a variety of medical conditions.

KEYWORDS: forensic science, forensic pathology, hypernatremia, subdural hematoma, child abuse

The theory relating hypernatremia to the development of subdural hematomas was presented in the mid 1950's. Researchers theorized that dehydration associated with hypernatremia drew water out of cells of the central nervous system, causing shrinkage of the entire brain. A decrease in overall brain size was postulated to stretch the bridging veins, resulting in a subdural hematoma (1). Over the ensuing decades, the literature has vacillated regarding the cause and effect relationship between hypernatremia and subdural hematomas. Freundlich et al. (2) addressed the causal relationship in a paper published in 1956. He wrote, "In children with dehydration and malnutrition we think it is extremely difficult to decide whether these states are the cause or result of the subdural hematoma; the sequence of events in these conditions is still obscure. . . There is a possibility of bleeding during a diagnostic subdural tap." Another advocate of the theory that hypernatremia leads to subdural hematomas acknowledges that the opposite may also be true, that intracranial trauma results in hypernatremia. In 1955, Finberg (3) wrote: ". . . Hypernatremia or its necessary concomitant, intracellular dehydration, is responsible, at least in part, for the nervous system damage observed. On the other hand a number of reports have indicated that the serum sodium concentration may be elevated following brain lesions due to trauma, brain surgery, and a variety of encephalopathies."

The theory linking hypernatremia and subdural hematoma was formulated before the landmark article "The Battered Child Syndrome" by Kempe et al. (4) in 1962. Child abuse in association with inflicted head injury was not widely recognized as a means of developing subdural hematomas before Kempe's paper. Kempe acknowledged that subdural hematoma, with or without fracture of the skull, was a common finding in non-accidental injury cases involving children or infants. Many researchers before this time did not recognize the effect of intentional injury on a child. Further, this theory emerged before the advent of computed tomography (CT) and magnetic resonance imaging (MRI) scans, when subdural hemorrhages were diagnosed by a procedure known as a subdural tap. In this procedure a needle was inserted into the subdural space, and any excess fluid was aspirated.

To reexamine the issue of hypernatremia and subdural hematoma in light of current medical knowledge and availability of neuroradiologic imaging techniques, we undertook a multifaceted investigation of the issue. This investigation included: a small retrospective review of autopsy findings in deceased children with elevated vitreous sodium values; a larger retrospective chart review of hospitalized hypernatremic children; a complementary retrospective chart review of hospitalized youngsters with subdural hemorrhages; a review of cases of fatal head injury with hospitalized survival intervals, and post-injury hypernatremia; a critical review of the literature; and a review of recent anecdotal cases of severe hypernatremia.

Materials and Methods

Postmortem Examination at the KY OCME

Pediatric vitreous sodium values obtained at autopsy at the Kentucky Office of the Chief Medical Examiner (KY OCME) were re-

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viewed. Over the four year period from 1993 to 1996, 96 cases were identified.

Chart Review of Hospitalized Hypernatremic Children

A retrospective chart review was conducted of hypernatremic patients, aged two years or less, hospitalized at Kosair Children's Hospital from January 1, 1994 through April 30, 1996. This pediatric hospital in Louisville, Kentucky is a 254 bed, level I trauma center. During this time period, there were 21,852 evaluations of children aged two and under. Of these, 203 patients with at least one serum sodium value of 150 meq/L or greater were identified through hospital laboratory data. Of the 203 patients, sufficiently complete medical records were available for review in 134 patients.

Chart Review of Hospitalized Children with Subdural Hematomas

A retrospective chart review was conducted of children, aged two years or less, with subdural hematomas, hospitalized at Kosair Children's Hospital from January 1, 1994 through September 19, 1996. Twenty-three children were identified.

Hypernatremia Following Head Injury at the KY OCME

Cases of fatal head injury with hospitalized survival intervals and post-injury hypernatremia were prospectively gathered. Five children aged 10 and under were identified.

Results

Our study included a three-part retrospective chart review of children aged two and under as well as a prospective review of cases at the KY OCME. In this review, the authors defined hypernatremia as a serum or vitreous sodium value of greater than or equal to 150 meq/L.

Postmortem Examination at the KY OCME

Previous studies have shown that vitreous sodium values accurately reflect antemortem serum sodium levels in the early postmortem period, and gradually fall as vitreous potassium rises (5) (see Table 1). A retrospective review of pediatric vitreous sodium levels obtained at autopsies at the KY OCME revealed nine out of a total of 96 patients whose sodium values exceeded 150 meq/L. These nine children ranged in age from seven weeks to four years, with only one child being over two years of age. The sodium values ranged from 151 meq/L to 173 meq/L, with an average value of 156.2 meq/L. Causes of death in these cases included infection, dehydration, and bowel obstruction. None of the children displayed subdural bleeding.

Chart Review of Hospitalized Hypernatremic Children

A retrospective chart review was conducted at Kosair Children's Hospital of admissions between January 1, 1994 and April 30, 1996 of hypernatremic patients, aged two years or less, to identify the incidence of coexistent subdural hematomas. One hundred and thirty-four hypernatremic children with complete medical records were identified. Fifty-five of the 134 hypernatremic children underwent diagnostic neuroradiologic studies, autopsy examination, or both. Of the 134 charts reviewed, a subdural hematoma was identified in only one patient. In this patient, the subdural hematoma preceded the development of hypernatremia. The patient was a two-month-old who was admitted to the hospital following an acute respiratory arrest. There were no external signs of trauma. Serum sodium on admission was 134 meq/L. Chest X-ray on admission revealed a left acromial fracture. A CT scan on the day of admission revealed a parafalcine subdural hematoma. Ophthalmoscopic examination revealed retinal hemorrhages. Two days later, shortly before death, the serum sodium concentration had risen to 169 meq/L. Subsequent autopsy revealed multiple scalp contusions, subdural and subarachnoid hemorrhage, retinal and optic nerve sheath hemorrhages, and the acromial fracture. The death was ruled a homicide.

Examination of the admitting and discharge diagnoses of the 134 hypernatremic infants revealed two main diagnostic groups: infants suffering from gastroenteritis and dehydration with relatively short admissions and good outcomes (21 patients), and critically ill infants in whom hypernatremia arose as a consequence of severe medical problems including meningitis, trauma, and "multiple complications of prematurity" (113 patients). Twenty-six of the 113 critically ill children (23.0%) died. Causes of death in these cases included: complications of prematurity, congenital malformations, and blunt head trauma. Of the 134 patients in the alleged "risk factor" group of hypernatremia, no child exhibited a subdural hematoma that could be attributed to elevated sodium values (see Table 2).

TABLE 1—*Hypernatremia identified by vitreous study at autopsy.*

Case Number	Age	Sodium Concentration (meq/L)	Cause of Death
#1	8 months	160	GE/dehydration
#2	2 months	153	interstitial pneumonitis
#3	3 months	151	NAC, dilantin embryopathy
#4	6 months	163	GE/dehydration
#5	18 months	173	dehydration, congenital toxoplasmosis
#6	2 months	152	RSV pneumonia
#7	4 years	152	bowel obstruction, Angelman's syndrome
#8	1 year	151	sepsis
#9	7 weeks	151	pneumonia

GE = Gastroenteritis.
NAC = No Anatomic Cause.
RSV = Respiratory Syncytial Virus.

TABLE 2—*Chart review of hospitalized hypernatremic children.*

Diagnostic Group	Number of Patients	Mortality (%)	Number of Cases of Hypernatremia Preceding SDH
Gastroenteritis/ dehydration	21	0	0
Multiple severe medical problems	113	23	0

SDH = Subdural hematoma.

Chart Review of Hospitalized Children with Subdural Hematomas

This portion of our study focused on hospitalized children aged 2 and under with subdural hematomas. The children were hospitalized at Kosair Children's Hospital between January 1, 1994 to September 19, 1996. Twenty-three patients with subdural hematoma were identified during this time interval. In all 23 patients, the etiology of the subdural hemorrhage was ascribed to trauma. Causes included: birth trauma, motor vehicle collisions, and child abuse. In all cases, the subdural hematoma was diagnosed by CT, MRI, or autopsy. One of the 23 children became hypernatremic following head trauma as described in the previous portion of the study (see chart review of hospitalized hypernatremic children). There were two deaths in this group; in both, autopsies revealed findings consistent with inflicted head trauma.

Cases of Hypernatremia Following Head Injury at the KY OCME

During the time of data collection and manuscript preparation, five pediatric cases demonstrating hypernatremia after head injury came to autopsy (see Table 3). These children ranged in age from 21 days to 10 years, and were healthy until the occurrence of the traumatic event necessitating their admission. All became hypernatremic during their hospital course, following acute traumatic injuries or anoxia. Three of the individuals had documented normal sodium levels on admission to hospital; in one case, blood for chemistry studies was not obtained until after the patient received Mannitol; and in the fifth case, admission serum sodium at an initial outlying community hospital was unavailable to the authors. The causes of death in these cases included head injury sustained in a motor vehicle collision (two cases), inflicted closed head injury (two cases), and anoxic encephalopathy following an unexplained cardiopulmonary arrest. The survival time ranged from 1 day to 11 days. The hypernatremia was attributed to central nervous system dysfunction as well as vigorous attempts to decrease post-traumatic cerebral edema by fluid restriction and diuretics.

Discussion

Literature Review

The relationship between hypernatremia and subdural hematomas has been addressed frequently in the literature since the 1950's. Finberg (1,6) purported that hypernatremia is linked to the subsequent development of subdural hematomas. His case studies included children under the age of two diagnosed by subdural tap. In 1959, Finberg (6) reported on hypernatremia in the infant by reviewing the history of seven infants whose maximum sodium serum concentration values ranged from 154 meq/L to 193 meq/L. Subdural taps were performed, revealing only one out of seven infants with "grossly bloody fluid." The infant with "4 or 5 mL of grossly bloody fluid" on subdural tap was an eleven-month-old male whose underlying disease process was diarrhea with dehydration. His maximum serum sodium concentration was 181 meq/L. Neurosurgical exploration by burr holes revealed "no membrane or large hematoma," which raises the concern that the subdural hemorrhage resulted from the tap. Five out of seven infants in Finberg's study, including a case of salt poisoning with a serum sodium concentration of 193 meq/L, displayed "clear fluid" in the subdural space.

In 1955, Finberg (3) reported a study of 81 patients diagnosed with hypernatremia, with the majority of the patients presenting with diarrhea. Of the 274 infants who presented with diarrhea, 69 were hypernatremic upon admission. Eight of the 69 hypernatremic patients died (11.6%). Two of the deceased hypernatremic infants had postmortem examinations both revealing extensive subarachnoid hemorrhage. There were no documented subdural hemorrhages in any of the infants in this study.

In another paper, Finberg (1) used an animal model to study whether subdural hematomas would develop as a result of administering a hypertonic solution to kittens. A hypertonic solution containing 1500 meq/L sodium, 1500 meq/L chloride, and 500 meq/L bicarbonate was injected intraperitoneally. The extreme hypertonic nature of this fluid is realized by comparison with normal saline solution, which contains 154 meq/L sodium and 154 meq/L chloride. Each animal was injected with 35 mL of this solution per 100 g of body weight. The equivalent volume in a ten-pound (4.5 kg) human infant would be an intraperitoneal injection of 1575 mL of this solution, or in the case of a 27 pound (12.3 kg) two-year-old child, greater than 4 liters of this fluid. The average serum sodium level in the kittens following the injection was 196 meq/L, but the time interval between injection and sample collection was not reported. Subdural hematomas were present in 23 of 27 cases. Eight of the 27 kittens scheduled to be sacrificed after 24 h died before the time elapsed. All eight of these kittens had subdural hematomas, as well as 15 of the 19 kittens sacrificed at 24 h post injection.

Little correlation can be made between the animal model and the case series of human infants due to the different manner of achieving hypernatremia. The animals received massive amounts of ex-

TABLE 3—*Hypernatremia following head injury.*

Case #	Age	Sodium Concentration Hospital Admission Value/Highest Value (mmol/L)	Survival Time (days)	Cause of Death
#1	6 months	134/180	1	head injury sustained in motor vehicle collision
#2	21 days	143/155	8	anoxic encephalopathy
#3	4 months	146/153	11	inflicted closed head injury (Shaken Impact Syndrome)
#4	10 years	150*/166	1	blunt traumatic injuries sustained in motor vehicle collision
#5	22 months	N/A/179	1	inflicted closed head injury

N/A = Not Available.
* = Value obtained following the administration of Mannitol.

tremely hypertonic fluid by direct injection into the peritoneal cavity. Post injection, the kittens exhibited immediate profound metabolic alterations which, in some cases, led to sudden death. The infants described in the companion case series generally displayed lower serum sodium levels developing over a longer time interval (6).

Finberg (7), in 1963, also reported a study of mass accidental salt poisoning in which a canister of formula mistakenly contained salt instead of sugar. Of the 14 infants exposed to the formula, five died before the situation was corrected. The brains of these five infants revealed "damage characterized by capillary and venous congestion, subarachnoid bleeding, gross and microscopic parenchymatous hemorrhages, and major dural sinus occlusions." There was no evidence of subdural bleeding due to the hypernatremia in any of the infants, even though the highest serum sodium concentration value was 274 meq/L.

Herzberger's paper (8) in 1956 reported an incidence of subdural effusions in 33 infants predominantly presenting with meningitis or gastroenteritis with malnutrition. His study revealed 32 out of 33 patients with effusions with or without membranes while only one case was described as a subdural hematoma. Herzberger wrote that the majority of the infants had a "very small shrunken firm brain, [with] thrombosis of the superior longitudinal and other dural sinus and cortical venous thrombosis." Although serum electrolyte values were not documented in this paper, Herzberger's study supports the idea that subdural hematoma is rare in infants with atrophic brains, although subdural effusions may be more common.

The relationship between hypernatremia and subdural hematoma is further addressed in 1959 by Luttrell and Finberg (9) in a study of three infants who had elevated serum sodium concentration values. The infants, all under the age of eight months, presented with a history of diarrhea, vomiting, or both. The autopsy of the first case revealed superior sagittal sinus thrombosis with extensive subarachnoid bleeding. The second infant's pathologic examination displayed thrombi in subependymal vessels and intraventricular hemorrhage. The third patient's autopsy demonstrated interhemispheric subdural hematomas as well as small subarachnoid hemorrhages. This patient was a neglected one-month-old female who died within 15 min of arriving at the hospital. The postmortem serum sodium concentration was 180 meq/L. Since Luttrell and Finberg's study was conducted before Kempe's child abuse article was published, death of the infants in their study was immediately attributed to natural disease processes without regarding the possibility of inflicted trauma as would be considered today.

Mocharla's (10) 1997 paper supported the argument that hypernatremic dehydration leads to the development of subdural hematomas. This paper discussed a case of a five-week-old boy who presented to the emergency department with lethargy, fever, diarrhea, decreased appetite, and emesis. The serum sodium concentration was 214 meq/L. The CT scan on admission revealed a right tentorial subdural hematoma, cerebral edema, and intraventricular hemorrhage. Within 12 h of arrival, the baby died. The doctors overseeing the infant's care initially suspected that the intracranial hemorrhage and cerebral edema were due to non-accidental trauma but later attributed them to hypernatremic dehydration. No mention of an autopsy was included in the manuscript. Researchers have proposed that subdural bleeding is due to tearing of veins when the brain is shrunken from hypernatremic dehydration, but in Mocharla's case, cerebral edema was present at admission which is incompatible with the concept of hypernatremia leading to cerebral shrinking and thus causing the subdural hematoma.

Recent Anecdotal Cases

The literature also cites excessive exogenous sodium intake as a mode of developing hypernatremia (11,12). Salt poisoning causes an increase in serum sodium concentration as well as an increase in total body sodium level which differs from hypertonic dehydration (resulting from diarrhea or inadequate water intake), which is an increase in serum sodium concentration but a decrease in total body sodium concentration. Salt poisoning impairs the kidney's ability to excrete excess sodium, and the serum osmolality often exceeds 400 mOsm/kg (11). Excessive intake of sodium in the infant or young child may be inflicted (4,5,13). At the 1989 Annual Meeting of the AAFS, Spencer (14) presented a case of homicidal salt poisoning in an eight-month-old infant, with a serum sodium of 244 meq/L, shortly before the infant's death. At subsequent autopsy, subdural hematoma was not observed. In 1993, Coe (5) described the death of a three-year-old after forced massive salt ingestion. The vitreous sodium was 210 meq/L. The autopsy revealed "no internal injuries or natural disease."

Habbick et al. (15) studied the effect of the serum sodium concentration on brain tissue as analyzed by computed tomography. The CT scan revealed low-density lesions in the basal ganglia, especially the putamen, which they attributed to hypernatremia. Another study describes the postmortem examination of a man whose death was due to exogenous salt intake (16). He presented to the emergency room with a serum sodium concentration of 201 meq/L. The postmortem exam revealed "severe cerebral edema, hemorrhages in the midbrain and pons, and tonsillar herniation in conjunction with necrosis." Although the sodium serum concentration was greatly elevated above normal, no subdural hematoma was present.

Etiologies, Signs and Symptoms of Hypernatremia

Hypernatremia may be caused by severe dehydration, sodium gain, or failure to excrete sodium (17). Dehydration may be due to insensible losses from the skin and respiratory tract or the excretion of a hypoosmotic urine. The latter may be a result of decreased antidiuretic hormone (ADH) due to central diabetes insipidus or end-organ resistance to ADH as in nephrogenic diabetes insipidus. ADH is produced by the hypothalamus and released from the posterior pituitary gland in response to variation in plasma osmolarity. In ADH deficiency, excessive water loss in the urine causes an increase in the serum sodium concentration. This syndrome, called diabetes insipidus, may be associated with a variety of intracranial abnormalities including meningitis, head trauma, and pituitary tumors (18). Insensible losses of water may also be caused by fever, respiratory infection, burns, or exposure to high temperatures—all leading to increased serum sodium levels. Breast-fed infants are also susceptible to developing hypernatremia. Maternal lactation failure may cause hypernatremic dehydration (19), or increased sodium content in maternal breast milk may lead to hypernatremia in the infant (20).

Mild hypernatremia associated with mild dehydration may be asymptomatic. Infants with acute hypernatremia may exhibit irritability coupled with severe lethargy (20). They also may present with fever, emesis, or respiratory distress and have a shrill cry (21,22). The symptoms of severe hypernatremia include disturbance of consciousness, hyperreflexia, muscle twitches, skeletal muscle rigidity, seizures, coma, or death (22,23).

Results of This Study

In the present study, none of the nine children with increased vitreous sodium values, as identified by a retrospective autopsy re-

view, demonstrated subdural hemorrhage. All of the children displayed congenital abnormalities and/or acquired natural diseases. Vitreous fluid is not aspirated routinely in children with evidence of traumatic intracranial hemorrhage, since the globes are examined for the presence of retinal hemorrhages in these cases. Therefore we cannot conclude that all children presenting with subdural hemorrhage were normonatremic.

Extensive retrospective chart reviews of 156 hospitalized children, first those with hypernatremia and then those with subdural hemorrhages (with one infant falling into both categories), failed to demonstrate any case of hypernatremia leading to subdural hemorrhage. However, the converse (closed head injury with subdural hemorrhage leading to hypernatremia) was observed in one case.

Since initiating this study, the authors have prospectively collected five pediatric cases in which diffuse, severe brain injury led to hypernatremia during hospitalization. These cases were identified by careful review of laboratory values in the medical records. We suspect that many such cases exist but are not necessarily noticed by Medical Examiners/Coroners, as hypernatremia may develop as a late, relatively inconsequential event in profoundly ill or severely injured patients, with central nervous system dysfunction.

Conclusion

Contrary to the older literature which theorized a causal relationship between hypernatremia and subdural hematomas, our study could not identify a cause and effect relationship between hypernatremia and the subsequent development of subdural hematoma. The postmortem examination of children with elevated sodium vitreous levels in our experience failed to reveal subdural hemorrhage, and in retrospective chart reviews only one case of subdural hematoma was identified in the hypernatremic hospitalized children—the hematoma preceding the hypernatremia. Subsequent prospective gathering of patients with fatal head injury and post-injury hospitalization yielded five cases in which head trauma or anoxia preceded hypernatremia.

After examining the issue from multiple approaches, we can find no evidence that isolated hypernatremia causes subdural hemorrhage. If there is a cause and effect relationship, our data suggest that it must be extremely rare. We conclude that hypernatremia may occur as one of myriad complications that can arise in critically ill children, with or without intracranial pathology.

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