G116 A Case of Fatal Water Intoxication in a Toddler

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After attending this presentation, attendees will have reviewed the physical and chemical signs and symptoms of water intoxication, thereby broadening the differential diagnosis in cases of sudden death particularly in pediatric practice.

This presentation will impact the forensic community and/or humanity by demonstrating the clinical signs and symptom of water intoxication as well as the differential diagnosis of this condition so that it might be a consideration in cases of sudden unexpected death particularly when environmental conditions include exposure to water.

This presentation concerns a case of fatal water intoxication in a toddler after a day of swimming and a review of the literature on pediatric water intoxication.

A 2.5-year-old, 12 kg, previously healthy female presented to a community emergency department with profoundly altered mental status. Her caregiver reported the child had been at a home pool for several hours and on leaving had been "fussy" and may have fallen "onto her bottom" on the way to the car. She had one episode of copious clear emesis. Immediately after this she became progressively lethargic and unresponsive.

On presentation to the emergency department, her vital signs (HR 67, RR 35, O_2 saturation 64% on room air, BP 145/84) were consistent with Cushing's triad. She was emergently intubated and her vital signs normalized. Neurological exam revealed response only to painful stimuli. Her pupils were sluggishly reactive. Initial laboratories (prior to administration of any fluids) revealed a serum Na^+ 128, Cl^- 95, K 3.3, CO_2 15, BUN 15, Cr 0.2, and Glc 259. A bolus of 400cc of normal saline was ordered and transfer to a tertiary care pediatric hospital was arranged. Inadvertently, a bolus of D5W was reportedly administered instead of normal saline.

Initial labs at the tertiary care facility revealed a serum Na^+ of 120. Her pupils were fixed and dilated and she had no respiratory effort. Physical exam was normal with the exception of her neurological exam and specifically did not reveal any evidence of rashes or signs of trauma. Her neurological exam revealed she had no doll's eye reflex, corneal response, cough or gag. She did withdraw minimally to painful stimuli. An emergent head CT demonstrated diffuse cerebral edema with herniation and no radiological evidence of intracranial hemorrhage, although the differential diagnosis included trauma. She was admitted to the Pediatric Intensive Care Unit and fluid resuscitation was initiated. Within hours she developed central diabetes insipidus, with high urine output (8 cc/kg/hr), urine specific gravity or 1.000, and a rapidly increasing serum sodium (maximum Na^+ 155), despite changing intravenous fluids to 0.45 normal saline. Vasopressin therapy was initiated. Ophthalmologic examination ten hours after initial presentation showed splinter retinal hemorrhages in the posterior poles bilaterally. Over the next three days, her neurological exam deteriorated until she no longer withdrew to painful stimuli. At that time, her family elected to withdraw life support. A postmortem skeletal survey was negative for fractures.

The case fell under the coroner's jurisdiction and a forensic autopsy was ordered. At autopsy, no external signs of trauma were identified. Her thoracic and abdominal cavities were normal with the notable exception of cardiomegaly (76 g) and splenomegaly (90 g), neither of which were noted premortem. Cranial contents were consistent with diffuse cerebral edema and uncal herniation without intracranial hemorrhage. Cerebral spinal fluid collected using sterile technique was noted to be xanthochromic and somewhat gelatinous but not frankly purulent. Culture of the CSF grew *Klebsiella pneumoniae* in the broth only. No organisms were identified on gram stain of the CSF.

Local law enforcement officials conducted an investigation including interviews of all parties who had had contact with the child. Her adult caregivers on the day of her presentation reported that she had been in a floating toy in a backyard above ground pool for several hours during the day. Both adults present at the time did not recall the child becoming submerged at any time. One caregiver indicated that the adults and older children present at the pool had been jumping in from the edge creating "tidal waves" repeatedly. Repeated interviews with the caregivers revealed consistent histories of the events of the afternoon.

The cause of death was cerebral edema due to hyponatremia due to acute accidental water intoxication. The manner of death was determined to be accidental.

A review of the English language literature on accidental water intoxication reveals five cases in the

pediatric population with symptoms similar to this case. With timely, appropriate fluid resuscitation a full recovery is possible, even when the presentation includes a comatose state. It is most likely that this child swallowed a large volume of pool water, which acutely dropped her serum sodium that resulted in her symptoms, with subsequent irreversible cerebral edema and herniation.

Water, Intoxication, Pediatric

 ** Presenting Author*