## G41 Fatal Tube Feeding Syndrome in a Young Child

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The goals of this presentation are to: (1) list the types of dehydration based on the relative losses of salt and water and on the composition and volume of intake; (2) list causes of hypertonic dehydration; and, (3) estimate free water deficit based on a child's weight and serum (or vitreous) sodium.

This presentation will impact the forensic science community by increasing awareness of the potential fatal consequences of enteral feedings with elemental hyperosmolar solutions in young children.

Tube feeding syndrome was first described in the 1950s as a cause of hypernatremia and hyperosmolar dehydration in adults, especially those with head injury or who were otherwise unable to communicate. Subsequently, it was recognized that decreasing the osmolarity of the solutions introduced via enteral tube feeding, in conjunction with increasing free water via parenteral or enteral methods, was successful in correcting or avoiding most electrolyte abnormalities. There has also been discussion concerning the importance of hyperosmolar dehydration causing a significant increase in morbidity and mortality in the pediatric population, especially if not corrected.

This study presents the case of a 2-year-old child born at 25 weeks estimated gestational age who developed cerebral palsy, chronic lung disease of prematurity with associated pulmonary hypertension, and retinopathy of prematurity. She exhibited developmental delay and feeding intolerance secondary to oral aversion for which she had received a gastrostomy tube.

She was admitted for feeding intolerance, vomiting after feeding, and failure to thrive secondary to complications of prematurity and for evaluation of gastro-jejunostomy tube placement. Initial evaluation revealed a 7.7kg female with a body length of 78.4cm, placing her below fifth percentile growth for both weight and length corrected for age. Admissions laboratory results included serum sodium 137mmol/L, chloride 103mmol/L, urea nitrogen 8mg/dL, and creatinine 0.33mg/dL. Nutrition and gastroenterology consultations recommended against gastro-jejunostomy placement and feedings were titrated over the first day with her home mixture of hyperosmolar (850mOsm/kg water) essential amino acid solution with high-caloric powder supplement. Multiple attempts at intravenous access were unsuccessful. Additional free water, either parenterally or enterally was never provided, and with oral aversion and developmental delay, the patient was unable to communicate thirst.

She had gone to sleep uneventfully the evening prior to death (five days after admission) and nursing notes at 4:00 a.m. on the date of death (six days after admission) cited no abnormality, with mother and child sleeping peacefully in the room. At 8:30 a.m. on the sixth day after-admission vital check, a nurse noted that the child had a pale ashen color, was cool to the touch, and was stiff without pulse or respiration. She was emergently coded for asystole and eventually pronounced at 9:06 a.m.

At autopsy, the ocular conjunctival membranes along with lips, anterior tongue, and buccal mucosa were dry and pale. Her peritoneal and pleural cavities were dry and tacky. The trachea was dry and the lungs demonstrated atelectatic areas comprising approximately 25% of the right lung's lower lobe, 10% of the right lung's upper lobe, and 50% of the left lung's lower lobe. The gastrostomy tube was in place and well seated with residual enteral feeding solution in the stomach. Minimal urine volume was observed within the bladder. Encephalopathy of prematurity was confirmed on neuropathology review with periventricular leukomalacia, periventricular hemosiderin deposition, cerebellar hypoplasia, and mild hydrocephalus.

Due to the appearance of dehydration at autopsy vitreous electrolyte analysis was completed demonstrating sodium 168mmol/L, chloride 151mmol/L, urea nitrogen 49mg/dL, and creatinine 1.33mg/dL. Urine specific gravity was 1.024. Testing of the enteral feeding solution exhibited osmolality 925mOsm/kg with sodium <100mmol/L and chloride <50mmol/L.

Hyperosmolar dehydration is a well-documented cause of morbidity and mortality in pediatric populations, especially when combined with other conditions. With the rise in survival rate of premature neonates and associated neurologic complications, it is important to recognize the danger of hyperosmolar feeding solutions and how they contribute to feeding tube syndrome in the growing pediatric population that requires them. The medicolegal investigator must be able to recognize the sequelae of dehydration at the time of autopsy and correlate with appropriate antemortem and postmortem electrolyte studies to detect such derangements.

**Forensic Science, Tube Feeding Syndrome, Hyperosmolar Dehydration**

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