Severe hyponatraemic encephalopathy after surgery: analysis of 7 cases and recommendations for effective treatment and prevention

Abstract:

Background: The use of hypotonic fluids in the course of paediatric surgery may cause life-threatening hyponatraemic encephalopathy which is preventable.

Clinical cases: 7 children aged 3 to 6 years developed encephalopathy about 11 hours post-operatively with a median Glasgow coma score of 7 (range 5-10), vomiting (n=5) and seizures or status epilepticus. They had been given hypotonic fluids, typically 5% glucose. Treatments included mechanical ventilation (n=3), anticonvulsants (n=7), fluid restriction (n=7), sodium chloride (n=5) and diuretics (n=5). The sodium was 135 12 hours later. Six children survived without sequelae while one child, who had a cardiorespiratory arrest, died with cerebral oedema.

Background

Hyponatraemic encephalopathy occured post-operatively in around 0.34% of 100,000 paediatric operations in Arieff's 1992 series and was fatal in 8.4% of those cases. Causes include adrenal failure, cerebral salt-wasting, the syndrome of inappropriate ADH secretion and the use of hypotonic fluids. Most paediatric cases are related to the use of hypotonic fluids related to difficulty in excreting free water. It is important to prevent acute hyponatraemia due to this cause in view of the neurological complications.

This study looked at a consecutive series and specifically examined the mechanism of hyponatraemia and the measures put in place to avoid fatal outcome.

Methods

This was a retrospective series over 7 years in 2 emergency care units which were part of a regional paediatric hospital system. The inclusion criteria were: acute hyponatraemia after routine surgery in previously healthy children or those who had no predisposing factor for hyponatraemia. Hyponatraemia in children with cardiac or neurological operations was excluded. Data collection involved completing forms from analysis of medical records, and the anaesthetic record. Key data included demographics, anaesthetic management, particularly intraoperative infusion, clinical signs at diagnosis, the laboratory evidence, treatment administered and the time to recovery. Account was taken of formulae used for fluid calculation, from biological data available, plasma and urine osmolalities and the corrected serum sodium.

Case 1. A three year old boy was operated for a tympanoplasty for congenital deafness. He received an infusion Intraoperatively of 375 mL of 5% glucose (G5%) and 1000 mL in the first 12 hours

postoperatively. In the night, he presented with seizures, associated with vomiting and coma but without motor deficit. His capillary blood glucose was 15 mmol/L. The brain scan performed as an emergency was interpreted as normal and the EEG showed diffuse slowing. The persistence of neurological signs mandated artificial ventilation and anticonvulsants. Serum sodium was 118 mmol/L. He was treated with mannitol (1.3 g/kg) which was associated with improvement in sodium levels. Infusion of Ringer lactate allowed normalization of serum sodium within 24 hours, extubation at 36 hours and correction of the hyponatraemia.

Case 2. A girl of six years, with psychomotor retardation and treated epilepsy, underwent osteotomy of the pelvis and femur. The child was infused with saline and then 5% Dextrose of uncertain volumes. Eight hours after waking, she presented with generalized convulsions, followed by coma with no motor deficit with slow breathing. She received 1.5 mg diazepam before being transferred to Pediatric intensive care. The EEG showed slowing. The usual treatment had not been given the morning of surgery. Serum sodium was 120 mmol/L,with normal blood sugar. Treatment included furosemide (0.5 mg/kg), fluid restriction, and phenobarbital. The serum sodium control after 12 hours was 137 mmol/L. The child had normal neurological status and was discharged from PICU the next day

Case 3. A five year old girl, with no previous medical history, was operated on for strabismus. She had received an infusion postoperatively of Dextrose 5% with a minimum volume of 1000 mL in less than 10 hours because the nurse looking after her did not record how many infusion bottles she used when she changed them systematically when they were empty. The child vomited and had two generalized seizures followed by postictal coma treated with Intravenous diazepam. Serum sodium was 118 mmol/L, with a glucose of 13 mmol/L. She collapsed with unresponsive coma and respiratory arrest requiring artificial ventilation. She was treated with clonazepam and fluid restriction, with intake of NaCl to 5.5 mmol/kg/d. The brain scan showed diffuse cerebral oedema with circulatory arrest on injection of contrast, and EEG showed no electrical activity. Serum sodium was normalized to 140 mmol/L in fifteen hours. Death brain was confirmed 36 hours after the start of resuscitation.

Case 4. A boy of two years with no previous medical history underwent circumcision. He was slow to wake and was vomiting, which prompted his hospitalization and continuing the infusion. He was infused with 1500 mL of 5% Dextrose within 18 hours after surgery. In the night he had a seizure treated with diazepam, followed by coma with pyramidal signs. The EEG showed paroxysmal discharges and the brain scans found cerebral oedema. Serum sodium was 114 mmol/L and glucose 9.1 mmol/L. The child received 0.5 g /kg mannitol and sodium supplements and fluids were restricted. Artificial ventilation was necessary, given the conscious level. Ten hours after the start of treatment, serum sodium was 144 mmol/L, conscious level was normal allowing extubation, and he fully recovered by the next day.

Case 5. A girl of three years with no previous medical history underwent tonsillectomy. She received a postoperative infusion of 5% Dextrose with the bottle being changed once or twice, so that she received 1 to 1.5 liters. Five hours after waking, the child vomited, followed within six hours by drowsiness, which was treated with the administration of 10% and then 30% Dextrose to treat hypoglycemia. A seizure occurred controlled with diazepam. Blood glucose was 16 mmol/L. The child was then perfused with an uncertain volume of Ringer's lactate, then transferred to intensive care. Upon

arrival, she had further convulsions and coma and received a further injection of diazepam. Serum sodium was 128 mmol/L and CT showed cerebral oedema. She received an infusion of 5 mL/kg of saline in four hours associated with an injection of furosemide and fluid restriction. Serum sodium went back to 136 mmol/L in eight hours. The EEG showed reactive changes. She made a full recovery.

Case 6. A boy of four years with no previous medical history underwent adenotonsillectomy. Post-operative recovery was marked by vomiting, which led to continued infusion for a total of 1500 mL of 5% Dextrose in six hours. Eight hours post-operatively, generalized seizures required diazepam, and he became comatose. The CT brain was interpreted as normal and serum sodium was 115 mmol/L. After resuscitation, blood glucose was 11.2 mmol/L. He was treated with fluid restriction, NaCl (6 mmol/kg in two hours) and phenobarbital. The EEG showed diffuse slowing. Within 12 hours, he regained consciousness and serum sodium was 138 mmol/L. The child was discharged after 48 hours.

Case 7. A child of four years with no previous medical history underwent surgery for strabismus. She was infused with 250 mL of saline intraoperatively. Postoperatively, she received a combination of nalbuphine, and paracetamol and metoclopamide. Dextrose 5% was perfused for a volume of at least 750 mL, The bottle was changed and renewed by the nurse without a prescription. About 10 hours after intervention she became delirious with a movement disorder. Serum sodium was 120 mmol/L and blood sugar was normal. Treatment was immediately started, involving a infusion of NaCl to 1 mmol/kg over one hour and then 6 mmol/kg/d with fluid restriction and 1 mg/kg furosemide. On arrival in ICU, the child was comatose. Treatment with mannitol (0.2 g/kg) and phenobarbital was commenced. The EEG was abnormal. ADH in plasma was 2.3 pg/ml (normal <4.7 pg/mL). Consciousness gradually improved and serum sodium was corrected seven hours after entry to 135 mmol/L. She made a full recovery.

Death of a child due to posttonsillectomy hyponatraemic encephalopathy

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Case

A four year old girl, weighing 15 kg, ASA 1, was hospitalized on October 18, 2000 in the ENT Department of a public hospital to undergo adenotonsillectomy because of repeated episodes of tonsillitis and snoring with significant adenoidal hypertrophy.

On October 19, surgery took place at 08:45. Anaesthesia lasted 15 minutes involving premedication with Atarax®, induction with Sevorane ® and the administration of Sufenta®. A Vasofix ® catheter No. 22 was placed at the beginning and a 250 ml infusion of Plasmolyte was commenced. Nasotracheal intubation was performed without difficulty. The surgery consisted of a tonsillectomy followed by Sluder adenoidectomy, in the usual time. There was little bleeding. After the anesthetic period of about 15 minutes, extubation was carried out on the table without problem.

The child was then transferred to recovery. On surgical intensive care postoperative sheet, signed by the anaesthetist with the child asleep, appeared the words:

- monitoring every quarter of an hour for one hour, then once an hour;
- Resuscitation treatment:

  Perf GV;

  Paracetamol 150 mg × 4/24 h;

  Nubin C (sic) IVL 2 mg × 4/24 pm if necessary;

  Nifluril, a suppository for children.

After one hour of monitoring in the PACU, the child was returned to the ENT service at 10:10, with 250 ml of Plasmolyte started at 08:30, still running. Until about 12:00, the ENT surgeon was examining the child, who appeared well, so he gave permission to resume feeding.

At 12:20, the child complained of pain and refused to drink. She vomited twice and refused to eat. When alerted the nursing staff indicated that this reaction was normal. Until 15:00, the nurse, informed by an auxiliary that the infusion started in the operating theater was over, replaced the empty bag with a bag of Plasmalyte 500 ml of 5% glucose. Indeed, for her, the prescription mentioned by the anesthesiologist: "Perf GV", involved the installation of a bag of 5% glucose. At 16:30, as the child had vomited blood, an ENT doctor who was with another patient, examined her. He noted no objective

signs but asked the caregiver to make sure she had an empty stomach. By 17:00 the child was agitated and vomited three to four times. By 18:00, agitation worsened and the child cried when touched. She continued to vomit and stopped passing urine. At 19:00, the child seemed calmer but vomiting persisted.

At 20:20, convulsions occurred. The nurse then rang the anesthetist and pediatrician on call. Blood pressure was 90 mmHg and temperature was 35 ° C. Shortly before this episode, at about 20:00, noting that the bag 5% glucose put up at 15:00 was almost empty, the nurse replaced the bag with an identical one of 500 ml of 5% glucose in order to keep the vein open. The paediatrician noted that the child was comatose with pauses in breathing and decided to transfer to the paediatric ward after the anesthetist had intubated the child for manual ventilation. At 22:00, given the results of additional tests requested, including hyponatremia at 115 mmol/L, the decision was made to transfer the child to the paediatric intensive care unit of the University Hospital. Lasix ® was injected, inducing a diuresis of 400 ml. On arrival at 00:15, the child received 10 ml of mannitol 20% as well as NaCl and Plasmalyte20%.

At ICU admission at the university hospital, about 01:50, the diagnosis of brain death was clinically confirmed and the electroencephalogram was isoelectric. The brain CT scan revealed an engorged cerebellum. The child died on October 20 at 19:00.

The hospital report concluded that death was due to severe metabolic encephalopathy with major cerebral oedema responsible for cerebral herniation. The diagnosis was "poisoning by water "postoperatively. The autopsy confirmed the conclusions.