Postoperative Hyponatremic Convulsions in Two Malnourished Children

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The prevalence of hyponatremia in the postoperative period is about 4.4%(1). It is being increasingly recognized that postoperative hyponatremia can cause fluid shifts between the extracellular fluid and the brain cells, leading to cerebral edema and convulsions(2,3).

We report two children with postoperative hyponatremic convulsions with one death. Both children underwent elective surgery. They were both severely malnourished, being at or below the third percentile by weight for South Indian children. This led us to speculate that severely malnourished children may be especially susceptible to postoperative hyponatremia and its attendant complications. Previously published data regarding the deranged fluid and electrolyte metabolism in malnourished children tends to support our speculation(4). To our knowledge the following report is the first to draw attention to a possible correlation between the malnourished state and postoperative hyponatremia.

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Case Reports

Case 1: A 7-year-old boy weighing 12 kg (less than 3rd percentile) underwent right orchidopexy for intra-abdominal testis. Pre-operative packed cell volume (PCV) was 28%. No other preoperative investigations were done. Although malnourished, the child was alert and active and was accepted for anesthesia. There was no past or family history of convulsions. General anesthesia with endotracheal intubation and additional caudal block was given. The procedure lasted 2 hours and 35 minutes. During this time 300 ml of 1/5 Hartmann's Ringer Lactate in 5% dextrose was infused. The fluid regime was calculated according to the preoperative starvation time of 4 hours and fluid losses during the procedure. Intraoperative course was uneventful. The child was awake and alert following extubation. In the next 10 hours he received another 400 ml of 1/5 Hartmann's Ringer Lactate in 5% dextrose intravenously. During this period he had two episodes of vomiting. Ten hours following surgery he developed generalized convulsions. He was afebrile. Biochemistry at this point was as follows: serum sodium 117 mEq/L, serum potassium 4.8 mEq/L, serum calcium 8.3 mg/dl and random blood sugar 202 mg/dl, injection Valium 5 mg IV controlled the seizures temporarily and a 5% dextrose with 0.9% saline drip was started. However, the child developed repeated convulsions which could not be controlled with phenobarbitone and dilantin. Breathing was noted to

be shallow, hence he was intubated immediately and ventilated. The convulsions stopped but pupils started dilating. Mannitol and lasix were given along with IPPV as anticerebral edema measures. Pediatric and and neurological opinions were sought and it was felt that the child had raised intracranial tension. The child suffered a cardiac arrest half an hour after institution of IPPV from which he could not be resuscitated.

Case 2: A 3-year-old girl weighing 8.5 kg (3rd percentile) underwent cleft palate repair under general anesthesia. There was no past or family history of convulsions. Preoperative hemoglobin was 11.1 g/dl. This was the only investigation done. Child had been alert and active prior to anesthesia. The surgery lasted 1 hour during which period 200 ml of 1/5 Hartmann's Ringer Lactate in 5% dextrose was infused to allow a starvation period of 6 hours prior to surgery. Intraoperative course was uneventful. After extubation, the child was fully awake and crying. Postoperatively, 400 ml of 1/5 Hartmann's Ringer Lactate in 5% dextrose was infused intravenously over the next 12 hours. Six hours following surgery she had 4 episodes of vomiting and 12 hours later developed generalized convulsions lasting 30 seconds which stopped spontaneously. She was afebrile. The child received a bolus dose of IV dilantin 80 mg. Biochemistry at this point showed serum sodium 125 mEq/L, serum potassium 4 mEq/L, serum calcium 7.9 mEq/L, and random blood sugar 132 mg/dl. The child was started on full strength Hartmann's solution (FSHRL). Subsequently, serum sodium returned to normal over 12 hours and the child made an uneventful recovery. The low serum calcium level can be explained by a possible hypoproteinemia in this child. No calcium therapy was administered to this child for convulsions.

Discussion

Hyponatremia, whether caused by a pre-existing metabolic problem, excessive administration of sodium poor fluids or by inappropriate ADH secretion, is associated with an increase in body water relative to body solute. This is an important cause of non-febrile convulsions in children in the postoperative period(5-9). Shift of extracellular fluid into brain cells can result in cerebral edema, brain-stem herniation, permanent brain damage, respiratory arrest and death(10). Studies of fluid and electrolyte imbalance in malnourished children have shown that, the total body water, especially the extracellular water, is increased in protein calorie malnutrition, even in the absence of edema, with attendant hyponatremia(4). Whether these two children were hyponatremic preoperatively is not known.

The fluid regime followed in our hospital for children upto 10 kg weight is 4 ml/kg/hour of 1/5 HRL in 5% dextrose which covers the fluid requirement for the starvation period and intraoperative maintenence. Extra fluid as saline, blood or plasma is administered during surgery if tissue dissection is considerable. Postoperatively the child receives 100 ml/kg/24 hours of 1/5 HRL in 5% dextrose. Even though the volume and nature of intravenous fluids given to these children were appropriate according to their weight, the already existing hypotonicity of the extracellular space due to malnutrition might have resulted in further reduction of the tonicity. This could have resulted in sudden expansion of cell water causing water intoxication as suggested by Baskin and colleagues(11). Vomiting in the

postoperative period could also have compounded the relative sodium deficiency.

Treatment of hyponatremia, especially as regards how quickly it should be corrected, remains controversial. Rapid correction of hyponatremia is known to cause pontine myelinolysis and currently slow correction of hyponatremia, *i.e.,* raising the serum sodium by <12 mmol/lit/24 h is recommended. Three per cent saline infusion may be indicated; however, for patients with seizures or coma for an initial correction, a rate of 1-2 mmol/lit/hour should be used till a rise of 3-6 mmol/lit of serum sodium is achieved. This will stop the convulsions after which a slow correction should be done, *i.e.,* 3% saline should be discontinued(12,13). We have used normal saline and FSHRL in these cases. An initial correction with 3% saline may have been more appropriate in *Case 1.*

In conclusion it is possible that the malnourished state in these two children with postoperative hyponatremic convulsions is more than a chance association. We shall in future pay meticulous attention in monitoring and managing sodium levels in the malnourished child undergoing even minor surgery. Serum sodium estimation should be done preoperatively and postoperatively in all malnourished children undergoing elective surgery.

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