EDITORIAL VIEW

Low sodium; a high risk in perioperative pediatric patients

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Perioperative fluid therapy is aimed at providing maintenance fluid requirements, at correcting fluid deficit and at providing the volume of fluid needed to maintain adequate tissues perfusion. It gets more important in pediatric population as the little shift in the small total volume of intracellular and extracellular compartments in these patients is multiplied many folds in its effects. Perioperative fluid therapy has been suggested to be a medical prescription adapted to the patient status, the type of operation and the expected events in the postoperative period of which both the volume and the composition matter.

The landmark article in which Holliday and Segar[1] proposed the rate and composition of parenteral maintenance fluids for hospitalized children has been the mainstay of much of our practice of fluid administration in the perioperative period even to this day. However, the glucose, electrolyte, and intravascular volume requirements of the pediatric surgical patient may be quite different than the original population described, and consequently, use of traditional hypotonic fluids proposed by Holliday and Segar has been questioned, e.g. hyperglycemia and hyponatremia, in the postoperative surgical patient. There is significant controversy regarding the choice of isotonic versus hypotonic fluids in the postoperative period[2].

Holliday and Segar calculated maintenance electrolytes from the amount delivered by the same volume of human milk. Daily sodium and potassium requirements are 3 mmol/kg and 2 mmol/kg respectively in children. Thus, the combination of maintenance fluid requirements and electrolyte requirements results in a hypotonic electrolyte solution. Since the publication of this paper, the usual intravenous maintenance fluid given to children by pediatricians for decades has been one fourth-to one half-strength saline and usually 5% dextrose[3].

The dextrose is added to prevent assumed hypoglycemia in infants and smaller children. Although, very important in this group of patients, the risk of preoperative hypoglycemia has been demonstrated to be low in normal healthy infants and children (1-2%), despite prolonged fasting periods[4-6] as energy requirements during anesthesia are close to basal metabolic rate. Although neonates have a higher metabolic rate and an increased risk of perioperative hypoglycemia and lipolysis, but during anesthesia, even in neonates, both oxygen consumption and metabolic rate are decreased, and this may lead to reduced intraoperative glucose requirements.

Hyperglycemia, on the other hand, can induce osmotic diuresis and consequently dehydration and electrolyte disturbances. Several animal studies have also demonstrated that hyperglycemia will increase the risk of hypoxic-ischemic brain or spinal cord damage. Conversely, administering glucose containing solutions (to prevent hypoglycemia) has predisposed the pediatric patients to dangerously low levels of sodium. The fact is that dextrose containing solutions with low sodium is still administered as a perioperative fluid of choice in many parts of the world. This practice has already led to many cases of hyponatremia and brain injury or death[7]. For practical purposes, in the peri-operative environment, D5 0.45% solution is hypotonic. The sodium in such glucose containing solutions needs to be low to maintain isotonicity. These solutions become effectively hypotonic once the fluid enters the blood stream and the glucose becomes metabolized. This may occur when these solutions are utilized in the intraoperative or post-operative time period. Recent studies have focused attention on the incidence of postoperative hyponatremia and associated morbidity and mortality rates, generating debate on the advisability of perioperative fluid therapy and calling into question both the effectiveness of this strategy and the quantities used[8].

Improper fluid therapy has just compounded the problem of hyponatremia, that may have other causes as well,

including pituitary or adrenal insufficiency, brain injuries or brain tumors associated with salt losses, and inappropriate secretion of ADH. Plasma ADH is often increased in postoperative period as a result of hypovolemia, stress, pain, or traction of dura mater. The combination of ADH secretion and infusion of hypotonic fluids will produce dilutional hyponatremia. Normally, the kidneys are able to excrete in excess of 20l/d of electrolyte-free water. In water intoxication, dilutional and hypotonic hyponatremia ensues from a rapid intake of a large volume of parenteral electrolyte-free fluid in excess of renal excretion over a short period of time. As free water is retained, hyponatremia develops. The resultant hyponatremia causes osmotic movement of free water across cell membrane from extracellular to intra-celllular compartment and the brain is the most seriously damaged organ[9]. Some of the risk factors are postmenarchal female gender, and prepubescent children. In post menarchal women, estrogen seems to impair the ability of brain to adapt to hyponatremia. Children are more susceptible to brain edema then adults because of the ratio of brain size and intracranial capacity. By the age of six years, the brain size of a child is the same size as adult while the skull continues to grow until the age 16 to adult size. Hence the capacity of CSF to buffer the brain expansion is relatively less in children then adults.

In older infants the occurrence of iatrogenic hyponatremia in this way has led to a critical reappraisal of the validity of the Holliday-Segar method for not only calculating maintenance fluid requirements, but also the choice of solution, in the postoperative period. The emphasis needs to be laid, now, on prevention of hyponatremia, which is the most common electrolyte disorder in hospitalized patients, with an incidence of approximately 1%-4%[10-13]. In fact, excess total body water in the presence of a small serum sodium concentration can result in an increase of extracellular water, cerebral edema, and potential brain herniation. Cerebral edema can manifest as nausea, headache, confusion, lethargy, convulsions, seizures, or coma. Radiological diagnosis of cerebral edema is difficult, if not impossible. Other signs and symptoms may include hemiparesis, ataxia, nystagmus, tremor, rigidity, aphasia, muscle cramps, and fasciculations[12,13]. Severe hyponatremia is also associated with cardiopulmonary dysfunction, including arrhythmias, hypotension, hypoxemia, and pulmonary edema[12]. In the perioperative period, these signs may easily be confused with adverse effects of the anesthetic drugs and agents being used, thus delaying the proper and adequate treatment of the actual cause. Often the respiratory arrest is the first manifestation of such electrolyte imbalance because the hyponatremia progress unnoticed till it is too late. The mortality rate of hyponatremia in hospitalized patients is reported to be 7- to 60-fold more frequent compared with normonatremic controls[14].

Anesthesiologists should maintain an index of suspicion for hyponatremia from water intoxication in patients with neurologic symptoms during the perioperative period. Routine preoperative instructions regarding maximum perioperative water intake and inquiry into any concurrent alternative medical therapies may help to avoid this preventable complication. A careful intraoperative monitoring and adaptation of the infusion rate as needed is crucial because the glucose and fluid requirements may vary widely between subjects. Conceptionally, the distinction between maintenance requirements, deficits and ongoing loss is helpful. Although the pathophysiological basis for parenteral fluid therapy was clarified in the first half of the 20th century, some aspects still remain controversial.

Dextrose containing solutions are an inappropriate choice for perioperative fluid losses such as blood loss and insensible loss and urine output, and by all means, in infants and young children, 5% dextrose solutions should be avoided; 1% or 2% dextrose in lactated Ringer may be more appropriate[15]. Only children who are risk for hypoglycemia should receive dextrose containing solution. These children include neonates in the first few days of life, patients on total parenteral solutions, children with low body weight (less then 3rd percentile) or born to diabetic mothers among others.

It may be reasonable to choose a solution for fluid replacement which has a composition comparable to the composition of the fluid which must be replaced. In any case, only isotonic solutions should be used in clinical situations which are known to be associated with increases in antidiuretic hormone (ADH) secretion. In this context, it is important to realize that in contrast to lactated Ringer's solution, the use of normal saline can lead to hyperchloremic acidosis in a dose-dependent fashion[16].

In summary, administration of dextrose containing fluids in pediatric patients in the peri-operative environment should be strongly discouraged and should be reserved in patients at real risk of hypoglycemia. If in doubt blood glucose should be monitored and patient should be followed closely in the post operative period. The fluid therapy in pediatric patients, especially during the perioperative period, must be tailored to the individual patient and careful monitored. Prevention of iatrogenic hyponatremia is an easy to implement practice with a high dividend. "First of all, do no harm".

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