JECT. 2015;47:228–230
The Journal of ExtraCorporeal Technology

Chronic Severe Hyponatremia and Cardiopulmonary Bypass: Avoiding Osmotic Demyelination Syndrome

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Abstract: Serum sodium concentration affects every cell in the body with respect to cellular tonicity. Hyponatremia is the most frequent electrolyte abnormality encountered, occurring at clinical admission in 22% of elderly patients. Any rapid correction of chronic severe hyponatremia can result in rapid cellular shrinking due to loss of intracellular free water. This is commonly associated with paralysis and severe brain damage due to osmotic demyelination syndrome (ODS). ODS occurs because the body has the ability to compensate for cellular fluid shifts due to chronic hyponatremia (by a decrease in brain concentration of several ions, amino acids, and organic osmolytes). Thus, the neurons are often at a functional state of fluid balance despite the sodium imbalance. The initiation of cardiopulmonary bypass (CPB) can introduce between 1 and 2 L of priming solution containing a normal sodium concentration creating a rapid rise in sodium concentration within the extracellular fluid. This abrupt change establishes a situation where intracellular free water can be lost resulting in cellular shrinking and ODS. In presenting this case study, we hope to add to the current literature with a specific isotonic approach to treating the chronically severe hyponatremic patient pre-CPB, during CPB, and post-CPB. **Keywords:** cardiopulmonary bypass, CPB, priming technique, chronic severe hyponatremia, osmotic demyelination syndrome, ODS, central pontine myelinolysis, open heart surgery. *JECT. 2015;47:228–230*

DESCRIPTION

Our patient was a 78-year-old male (178 cm, 82 kg) who presented to an outlying facility with sudden onset of chest pain during exertion. He was alert and oriented upon presentation and labs revealed a sodium concentration of 126 mmol/L. This concentration has been defined as moderately hyponatremic (1) (Table 1), and is only 2 mmol/L away from severe hyponatremia. It was noted by the attending physician that this was chronic hyponatremia of unknown etiology. The initial electrocardiogram showed ST depressions suggestive of a non-ST elevation myocardial infarction (MI) and the patient was transferred to our facility for further cardiac workup and treatment.

Received for publication October 5, 2015; accepted December 9, 2015.
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The senior author has stated that the authors have reported no material, financial, or other relationship with any health care–related business or other entity whose products or services are discussed in this paper.

Upon admittance to our medical center, further labs demonstrated the serum sodium concentration had dropped to 120 mmol/L; the patient was then severely hyponatremic. He had become disoriented and required intubation due to pulmonary edema. The patient was taken to the cardiac catheterization lab with clinical demonstration of an evolving MI. He was found to have severe multivessel coronary artery disease with decreased left ventricular systolic function and anterior apical akinesis. This included a left main (LM) lesion >90%, ostial left anterior descending (LAD) lesion >90%, largest diagonal 80%, left circumflex involving LM >90% and obtuse marginal 80%. It was also determined that he had a left dominant system. An intra-aortic balloon pump was placed. Due to the anatomy and severity of his disease, he was taken to the operating room for urgent coronary artery bypass grafting. Inadequate time existed for clinical correction of his hyponatremia and we were presented with the issue of initiation of cardiopulmonary bypass (CPB) with a patient diagnosed with chronic severe hyponatremia. The last sodium concentration before the patient presented to the operating room was 122 mmol/L.

Table 1. Definitions of different levels of hyponatremia (1,2).

Normal serum sodium concentration: 136–145 mmol/L
Mild hyponatremia: 130–135 mmol/L
Moderate hyponatremia: 125–129 mmol/L
Severe (profound) hyponatremia: 116–124 mmol/L
Life-threatening hyponatremia: less than 116 mmol/L

Our standard prime volume is approximately 1700 mL. It consists of 200 mL of 25% albumin (sodium concentration of 130–160 mmol/L) (Table 2), 1300 mL of PlasmalyteA (140 mmol/L), 50 mL sodium bicarbonate (1 mmol/mL), 100 mL of 25% mannitol (may contain sodium bicarbonate for pH balance), 10 g of Amicar (40 mL in 0.9 normal saline), and 10,000 units of heparin (10 mL in 0.9 normal saline). The sodium concentration of this solution was calculated to be approximately 153 mmol/L or 153 milliequivalents/L (mEq/L) (Table 3). A sample of the prime was sent to the lab to confirm this. The lab determined our prime to have a sodium concentration of 145 mEq/L. It was determined that if 1 L of 0.45% sodium chloride (sodium concentration of 77 mEq/L) was added to the current 1700 mL of prime (sodium concentration of 145 mEq/L), the resultant sodium concentration would be approximately 120 mmol/L (Table 4). This would adequately match our patient's serum sodium concentration. After the 1 L of 0.45% sodium chloride was added, 1 L of effluent (fluid lost from the circuit) was removed via hemoconcentration; effluent pulled from CPB solution leaves the remaining fluid near equal concentration of electrolytes to the previous concentration prior to the fluid removal. Only larger proteins such as red blood cells, mannitol and albumin, and other molecules too large to cross the membrane of the hemoconcentrator will increase in concentration within the circuit during hemoconcentration. After the effluent was removed, another sample of prime was sent to the lab and the sodium concentration of our prime was confirmed to be as calculated: 120 mmol/L.

Initiation of CPB began and a sample of the patient's first sodium concentration was determined to be 119 mmol/L. During CPB, it was carefully noted to take great caution in treating acid base deficiencies with sodium bicarbonate (Table 2) or to use .9 normal saline if additional volume or correction of a high potassium concentration was necessary. To perform zero-balance ultrafiltration during bypass, 1 L of 0.45% sodium chloride with 25 mEq of sodium bicarbonate (a combined sodium concentration of 77 + 25 mmol/L) as well as 500 mL of Lactated Ringers (130 mEq/mL) was added to the reservoir. Effluent was taken from the patient via hemoconcentration as possible (total of 3200 mL was removed before termination of CPB). This slightly lower sodium concentration of volume added accounted for the 50 mEq sodium bicarbonate present in the additive to cardioplegia given during cross clamp. The CPB time was 98 minutes and cross clamp was 84 minutes. Three saphenous vein grafts and the left internal mammary artery to LAD were successfully accomplished. Termination of CPB was achieved and there were no complications noted during CPB. The patient's first sodium concentration post pump was 123 mmol/L. Cerebral brain saturations (SrO2) were monitored throughout the entire case, pre-CPB, CPB, and post pump. The SrO2 values never dropped below baseline (taken upon entrance to the operating room, post intubation). The patient was extubated postoperative day (POD) 1 and remained alert and oriented until POD 2 where the patient exhibited some transient confusion. This resolved completely and the patient was successfully discharged to a subacute rehabilitation facility for physical therapy when a bed became available on POD 8.

Table 2. Sodium concentration of typical CPB drugs.

Plasmalyte: 140 mEq/L (140 mmol/L[1]*)
0.9 normal saline: 150 mEq/L
0.45 sodium chloride: 77 mEq/L
Lactated ringers: 130 mEq/L
5% dextrose: 0 mEq/L
Sterile water: 0 mEq/L
Sodium bicarbonate: 1 mEq/mL (1000 mEq/L)
Albumin 25%: 130–160 mEq/L
Mannitol 25%: may contain sodium bicarbonate to adjust for pH
Heparin sodium 1000 units/mL: generally in 0.9 normal saline

*mEq/L is equal to mmol/L for sodium (Na^+) as it is a monoatomic ion.

Table 3. Approximate estimation of sodium concentration of priming solution prior to adjustment.

1300mL Plasmalyte (140 mEq/L) + 200 mL albumin (∼145 mEq/L) + 50 mL Bicarb (1000 mEq/L) + 40 mL Amicar (negligible volume) + 100 mL mannitol (0 mEq/L) + 10 mL heparin (negligible volume) = approximately 1700 mL (*X* mEq/L)
X = 153 mEq/L
Using stoichiometry (less negligible additives)
[1.3 L Plasmalyte (140 mEq/L) + 0.2 L albumin (∼145 mEq/L) + 0.05 L Bicarb (1000 mEq/L)] = 1.7 L (*X* mEq/L)
X = 153 mEq/L

DISCUSSION

Generally, a patient with an acute drop in serum sodium concentration (within the previous 48 hours) will experience cellular swelling due to diffusion of free water into

Table 4. Calculation to determine sodium concentration to patient pre-CPB.

1.7 L prime (145 mEq/L determined by lab) + 1 L 0.45 saline (77 mEq/L) = 2.7 L prime (X)
New prime volume estimated to have sodium concentration of X = 120 mEq/L
This was determined by our lab to be the exact sodium concentration of our new prime after 1 L of effluent was removed via hemoconcentration. This was isotonic to our patient pre-CPB.

Table 5. Example calculation to determine volume needed to create an isotonic sodium concentration to patient pre-CPB.

1.7 L prime (145 mEq/L determined by lab) + XL 0.45 saline (77 mEq/L) = (1.7 L + XL) (120 mEq/L or the sodium concentration isotonic[1]* to patient pre-CPB)
Simplified: $(1.7)(145) + X(77) = (1.7 + X)(120)$
$(246.5) + 77X = 204 + 120X$
$246.5 - 204 = 120X - 77X$
$42.5 = 43X$
$X = 42.5/43$
X = 0.99 L of 0.45 saline to acquire a solution of 2.7 L of 120 mEq/L sodium concentration

*Any sodium concentration below 116–118 mEq/L has been classified as life threatening (5).

the cells (3). Many older patients, however, are chronically hyponatremic (4). For the urgent or emergent cardiothoracic surgical patient, it is on occasion not an option to admit them to the intensive care unit to address any preoperative issues. In the case of chronic severe hyponatremia and the emergent open-heart procedure, it is essential to note the catastrophic consequence of initiating CPB with a prime of normal sodium concentration. A rapid rise in sodium concentration could cause end-organ complication and result in osmotic demyelination syndrome (ODS). ODS (also known as central pontine myelinolysis) is associated with severe damage of the myelin sheath of nerve cells and can result in paralysis, irreversible brain damage, or death (5). By creating a prime volume pre-CPB that matches the patient's serum sodium concentration and tonicity (Table 5), the risk of ODS can be minimized. Considerations should be made during CPB to maintain the sodium concentration as close to the patient pre-CPB as possible. Mannitol should be used with extreme caution due to its hyperosmotic property (6). Also, sodium bicarbonate should be used cautiously to correct acidosis or hyperkalemia as it contains 1000 mEq/L of sodium. Frequent glucose monitoring should be considered as it has been noted that hyperglycemia can correlate to hyponatremia (7). Women's sodium concentration will be affected more by prime volume as they have a smaller plasma volume per kg (65 mL/kg vs. males who have an average of 75 mL/kg) (6). A patient with a higher preoperative hemoglobin will be affected more by the difference in sodium concentration of prime volume because they have a lower plasma volume percentage (and higher hematocrit) (6). Measures as described, here in, should be used to prevent rapid changes in sodium concentration and improve patient safety for those with chronic severe hyponatremia.

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