

Liver Transplantation in the Face of Severe Electrolyte Abnormalities: A Discussion of Management Techniques

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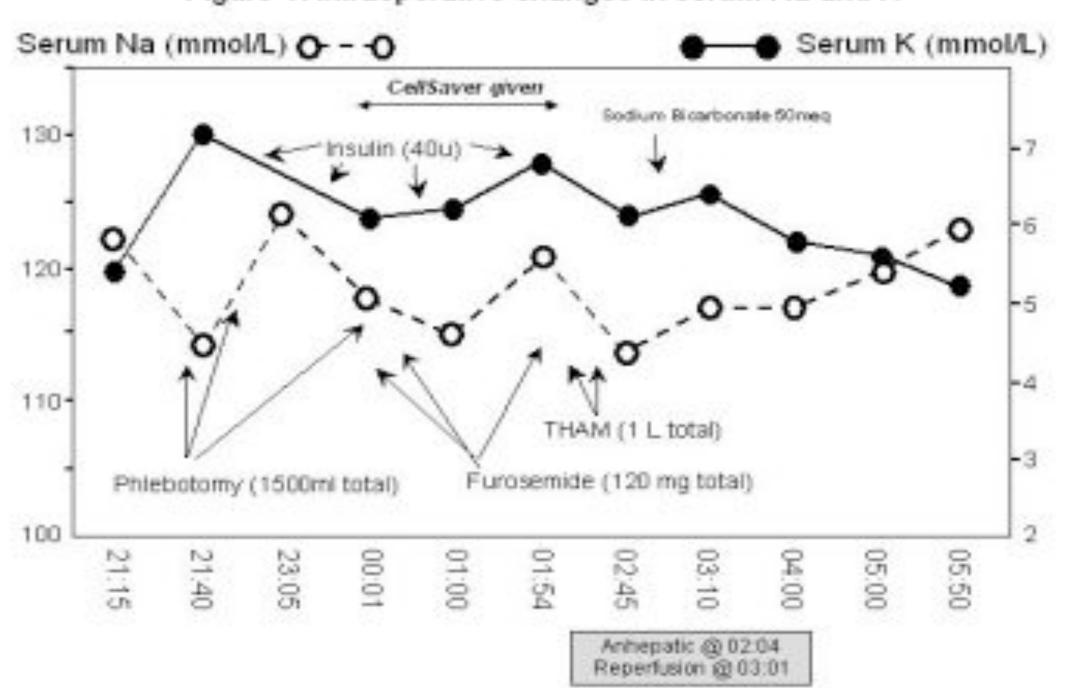
Introduction:

Electrolytes disorders are a common finding in patients with End stage liver disease (ESLD). Liver transplantation (OLT) is frequently associated with massive transfusion and fluid shifts in patients with renal dysfunction, and management of hyponatremla, hyperkalemia and hypervolemia are major concerns for anesthesiologists. This case describes a multimodal approach in treatment of combined severe hyponatremla, hyperkalemia and hypervolemia.

Case Description:

A patient for Liver Transplant was found to be hyponatremic (118 mM/L), hyperkalemic (6.9mM/L), and uremic (BUN/Cr = 95/2.6) preoperatively (Fig 1). OLT was proceeded not to loose a small window of opportunity, although potential complications were recognized (1).

Figure 1. Intraoperative changes in serum Na and K



To correct hyperkalemia, dextrose 50%(200 ml) and insulin (40 units) were given, his own phlebotomized blood (1.5L) and homologous blood (2units) were washed using an autotransfusion system before reinfusion, and continuous venovenous hemodialysis (CVVH) was instituted. To maintain a hyponatremic state, tromethamine sulfate (THAM, 1000 mL)) was used to treat metabolic acidosis instead of NaHCO³.

However, sodium bicarbonate was used once at reperfusion for rapid correction of acidosis. The treatment was successful. Sequelae of hyperkalemia were avoided even during reperfusion and Na level was not increased more than 10mM/L intraoperatively. He remains free of central pontine myelinolysis eight months postoperatively.

Discussion:

Hyponatremla is a strong predictor of wait-list mortality, and is associated with worse post-transplant outcomes. Treatment of hyponatremia is very difficult especially given the risk of central pontine myelinolysis. Table 1 shows the content of sodium in typical fluids administered during OLT.

Table 1: Sodium content in different solutions

Solution	Na+ mEq/L	<i>K+</i> mEq/L
THAM E®a	30	5
NaBicarb 8,4%	1000	
Saline 0,9%	154	
Plasmalyte	140	5
FreshFrozenPlasma	172	3.5
Albumin 5%	145-160	2

We chose THAM as our primary buffering agent for treating acidosis because of it's lack of sodium content. Correcting the base deficit by THAM before reperfusion of the graft liver may help to prevent the complications of reperfusion (hyperkalemia, metabolic acidosis, and release of myocardial depressant and vasoactive substances from the donor liver). These metabolic derangements may produce hypotension, bradycardia, arrhythmias, vascular collapse or cardiac arrest.

A possible advantage of THAM is that it promptly enters the hepatic intracellular space, where it exerts a buffering effect and may protect hepatocytes against hypoxia. Correction of acidosis with NaHCO³ in liver transplantation can limit acidosis, but this frequently leads to hypercarbia, hypernatremia and postoperative rebound alkalosis (4). Hypernatremia caused by the sodium load of NaHCO³ might burden the kidney and increase the risk of central pontine myelinolysis

An advantage of sodium bicarbonate is that it can be administered quickly.

In patients with renal impairment, hyperkalemia and hypervolemia, CVVH can be performed on the hemodynamically unstable patient, because infusion of a large amounts of blood products such as packed red cells, plasma and platelets are frequently required (5).

Intraoperative autotransfusion can reduce potassium load by washing the shed blood with 0.9% saline in a cell-saver device, but the washing process removes electrolytes, plasma, including clotting factors, pharmacologic drugs, pro- and anti-inflammatory factors, and platelets. Washing pRBCs before transfusion with a continuous autotransfusion device can significantly reduce the amount of potassium infused. One disadvantage of washing pRBCs is the increased sodium and chloride levels and decreased pH (6).

Conclusion:

A multimodal approach is required to treat electrolytes disorders in patients with end-stage liver disease and renal dysfunction. Unexpected severe hyponatremla was thought to be caused by acute excessive water consumption after repeated paracentesis, but it is possible he was developing hepatorenal syndrome. Because of his risk for multiorgan failure and sepsis without OLT we proceeded.

The combination of CVVH, THAM and cell saver was able to achieve a slow increase of sodium level (10 mg/h), avoiding CNS dysfunction by CPM, and to correct Hyperkalemia.

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