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Perioperative Anesthesia Management of Liver Transplantation in a case with very severe hyponatremia - A Case Report

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Abstract

Hyponatremia is an important predictor of post-operative morbidity and mortality, especially central pontine myelinolysis,in liver transplant. We present a case of liver transplant in patient with serum sodium of 110 meq/dl.A case ofliver transplantation in a 58-year-old male with cryptogenic liver disease (model for end-stage liver disease31, Child-Turcotte-Pugh14) with refractory ascites and spontaneous bacterial peritonitis, alongwith severe chronic hyponatremia and mild nephropathy. Serum sodium level ranged around 120 meq/dl preoperatively,and conventional management strategies consisting of fluid restriction, albumin infusion, and diuretics were instituted, but hyponatremia persisted. Due to decompensation and rising bilirubin levels, the patient was takenfor transplantation with serum sodium seen to be 110meq/dl. Intraoperatively, management comprised monitoring plasma sodium levels and hourly arterial blood gas to detect acidosis and electrolyte levels also preventing rapid rise in sodium levels. 0.45% normal saline along with 37.5ml soda bicarbonate 7.5% to make an iso-osmolar solution was given. This helped in decreasing the sodium load and counteracts the metabolic acidosis. Blood products were given as required. Vasopressin infusion and albumin 20% infusion weregiven and continued in the initial post-operative days. At the end of surgery, the patient had a sodium level of 116meq/dl. In the intensive care unit, 0.45% dextrose and normal saline and 5% albumin were used. Daily serum sodium levels were monitored, and on discharge, on the 15th day, it had reached a level of 130meq/dl.Throughout thepost-operative period, the patient did not show any signs of any neurological sequelae and was fully conscious and oriented.We followed a restricted sodium load in our fluid infusions with our constitution of a combination of 0.45% normal saline with 37.5ml 7.5% soda bicarbonate to make an iso-osmolar fluid. This along with diuretics,vasopressin infusion, albumin, and stringent monitoring and slow rise of sodium levels most probably prevented any morbidity or mortality in our case.

Keywords: Liver transplantation,severe hyponatremia,bicarbonated saline,central pontine myelinolysis,sodium load.

Introduction

Hyponatremia is a common yet ominous sign in patients with end-stage liver disease (ESLD) for liver transplantation1. Nearly, 50% of cirrhotic patients have serum sodium concentrations below the lower limit of the normal range (135–145 mmol/L). Hyponatremia in cirrhosis has been defined as a serum sodium <130 mEq/L. Hyponatremia in cirrhosis usually results from antidiuretic hormone-mediated renal retention of free water despite increased total body sodium2. Major complications of hepatic decompensation have been associated with hyponatremia including bacterial infections, ascites, renal failure, encephalopathy, and reduced quality of life. It is now well-established that hyponatremic patients with ESLD have increased mortality independent of other indicators of severity of liver disease such as the model for end-stage liver disease (MELD) score3.

Case Report

We present a case of living donor-related liver transplantation in a male patient with severe and chronic hyponatremia. The 58 years male patient was diagnosed as a case of cryptogenic chronic liver disease, with a MELD score of 31 and Child-Turcotte-Pugh score of 14. There was a history of spontaneous bacterial peritonitis and massive ascites but no history of HE, HPS, or HRS. On routine pre-operative workup, the serum sodium level ranged around 120 meq/dl and conventional management strategies consisting of fluid restriction, albumin infusion,and diuretics, alongwith newer drugs like vaptans4were instituted, but the

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hyponatremia persisted. Due to decompensation and recurrent massive ascites, it was decided to take up the patient for transplantation with pre-operativeserum sodium at 114 meq/dl, which further dropped to110meq/dl on the morning of surgery. Intraoperativemanagement comprised monitoring plasma sodium levels and hourly arterial blood gasto detect acidosis and electrolyte levels, and alsopreventing rapid rise in sodium levels. 37.5ml of 7.5%soda bicarbonate was added to 500ml of 0.45% normal saline(to make an iso-osmolar solution), and this bicarbonated saline was used as i.v. fluid. This helped in decreasing the sodium load and in counteracting the metabolic acidosis. The maximum Na level intraoperatively was 118 meq/dl, which was immediately after reperfusion. In total, 8L of 0.45% bicarbonatednormal saline alongwith 12units of packed red blood cells, and other blood products were givenas required. Vasopressin infusion and albumin 20% infusion were started intraoperatively. The patient was shifted after surgery to the liver transplant intensive care unit (ICU) with minimum inotropic support and a Na level of 116 meq/dl.In the ICU, 0.45%dextrose and normal salineand 5% albumin were used. The next morning serum Na was found to be 119 meq/dl, and the patient was extubated when he became conscious and was responding to verbal commands. The patient was found to be fully conscious and oriented, and there were no signs of altered sensorium. Throughout his ICU stay of 7days, the Na levels kept on fluctuating and he was shifted to the step-down ICU unit with a Na of 122 meq/dl. Daily serum sodium levels were monitored, and on discharge, on post-operative day15th, it had reached a level of 130meq/dl.Throughout the post-operative period, the patient did not show signs of any neurological sequelae and was fully conscious and oriented.

Discussion

Hyponatremia is an important predictor of post-operative morbidity and mortality, especially central pontinemyelinolysis (CPM), in liver transplant5. Severe hyponatremia is generally considered a contraindication for surgery due to the risk of hepatic encephalopathy and CPM.Indeed, it has been shown that hepatic encephalopathy is independently associated with serum hyponatremia. Incirrhosis, ammonia and other neurotoxins induce a low-grade cerebral edema, possibly by inducing the synthesis of the osmotically active glutamine in the astrocytes. Moreover, with decreased levels of serum sodium, the osmolality of the extracellular fluid falls.This results in a chronic loss of the organic osmolytes, which may predispose the brain to demyelinative lesions on correction of the hyponatremia. Whereas excessively slow correction of hyponatremia is associated with increased morbidity and mortality, inordinately rapid treatment may cause CPM. CPM or osmotic demyelination syndrome is a disorder of pontine white matter as well as other areas of cerebral white matter that was first described in alcoholics. It produces insidious flaccid quadriplegia, mental status change, and cranial nerve abnormalities with the pseudobulbar palsy appearance. CPMhas been reported in up to 10% of patients after livertransplantation. The rate of correction of hyponatremia appears to be of importance in these patients, and a slow correction does not result in CPM. During the perioperative period, the aim of management should be to prevent major fluctuations in the [Na], rather than correct it, and therefore, extreme caution needs to be exercised while administering sodium-rich fluids (particularly blood products) and soda bicarbonate intraoperatively. However, in the context of high volume and blood product requirement, coagulopathy, acidosis in the anhepatic phase, alongwith hemodynamic fluctuations, maintaining static [Na] levels can be a challenging task in liver transplantation. We followed a restricted sodium load in our fluid infusions with our constitution of a combination of 0.45% normal saline with 37.5ml 7.5% soda bicarbonate to make an iso-osmolar fluid. Osmolarity of this bicarbonated normal saline is 264, which is almost similar to ringer lactate. This reconstituted fluid is freshly prepared using aseptic technique and immediately used. Bicarbonated normal saline has also been recommended by the American Association for the Study of Liver Diseases as a resuscitative fluid in acute liver failure patients with acidosis6. The use of bicarbonated normal saline along with diuretics, vasopressin infusion, albumin, and stringent monitoring, and slow gradual rise of sodium levels most probably prevented any morbidity or mortality in our case.

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