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Case Report

Severe Acute Hyperkalemia during Pre-Anhepatic Stage in Cadaveric Orthotopic Liver Transplantation

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Abstract

A serious hazard to patients during orthotopic liver transplantation is hyperkalemia. Although the most frequent and hazardous hyperkalemia occurs immediately after reperfusion of the newly transplanted liver, morbid hyperkalemia could happen in the other phases during orthotopic liver transplantation. However, pre-anhepatic hyperkalemia during orthotopic liver transplantation is rare. This report describes one such patient, who without transfusion, developed severe hyperkalemia during pre-anhepatic phase. The variations in serum potassium concentration of the present case indicate that it is necessary to take care of the changes of serum potassium concentration not only during reperfusion but also during the other phases of the liver transplantation.

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Keywords ● Hyperkalemia ● liver transplantation ● orthotopic

Introduction

Severe hyperkalemia during orthotopic liver transplantation, is very dangerous, and needs vigilant monitoring of serum potassium and acute management of the hyperkalemia.[1,2] The causes of hyperkalemia during different stages are; 1) extracellular shift in exchange for H^+ during severe metabolic acidosis in an-hepatic phase and reperfusion of the graft liver, and 2) exogenous potassium due to blood transfusion or entry of the preservative fluid University of Wisconsin (UW) solution into systemic circulation during reperfusion of the graft liver.[2] However, this morbid hyperkalemia is more common in the early reperfusion phase than at other times during liver transplantation.[3]

Although hyperkalemic episodes occurring immediately after reperfusion of new transplanted liver are most frequent and substantial, hyperkalemia in other phases during orthotopic liver transplantation is also hazardous and serious.[2,3] For a short duration (about 3-5 min) after reperfusion of the graft liver, patients usually develop hyperkalemia. The main sources of this hyperkalemia are preservative fluid (UW solution), which contains high concentration of potassium, and severe acidosis following reperfusion, which can mobilize intracellular potassium from all of the tissues.[2] However, hyperkalemia before reperfusion during liver transplantation anesthesia is not common. The two independent risk factors for pre-reperfusion hyperkalemia during liver transplantation are high baseline potassium concentration and red blood cell (RBC) transfusion.[3] Baseline potassium is the first potassium level in operation room. An insulin protocol, in which separated doses

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of the regular insulin is administered together with blood transfusion in patients with high baseline K, has been used to prevent hyperkalemia due to blood transfusion.[4] Herein in we present a case that developed hyperkalemia without blood transfusion during pre-anhepatic phase of liver transplantation.

Case Description

The patient was a 49-year-old male who underwent liver transplantation for a Meld score 22 cryptogenic cirrhosis, which had been complicated by bleeding from esophageal varices, ascites, and spontaneous bacterial peritonitis. The patient's baseline lab and paraclinic data prior to anesthesia are shown in table 1.

Anaesthesia was induced using thiopental, morphine, fentanyl and midazolam. Pancuroium was used for neuromuscular blockade. Ventilation was maintained with a mixture of air-oxygen plus isoflurane 1%. Normal saline and albumen 25% were administrated to maintain a central venous pressure of about 10 cm H_2O. Calcium gluconate and sodium bicarbonate were used to correct low Ca^{2+} levels and metabolic acidosis (base excess ≤-4), respectively. We monitored cardiovascular functions using electrocardiogram, arterial pressure via a radial artery catheter, and central venous pressure via a catheter inserted into the right internal jugular vein. The transplantation of the graft was performed using the piggy-back technique. Baseline potassium was 4 mmol/L. Urine output was more than 500 ml during 3 hours of hepatectomy, and surgical bleeding was less than 400 ml; therefore, no blood transfusion was performed. Metabolic parameters including serum potassium was checked by serial measurements of arterial blood gases (ABG) as needed (table 2). The first serum potassium was 4 mmol/L, and after 40 minutes of anesthesia and surgery it was in normal range. One hour after the start of surgery, hepatic artery was ligated. Thirty minutes after the ligation a tall T wave and bradycardia were noted on EKG monitoring. Therefore, serum potassium was checked by measuring ABG. The serum potassium was 6.5 mmol/L. Hyperkalemia was managed by 10 ml calcium gluconate 10%, 50 ml dextrose 50%, and 25 U regular insulin. Near the end of hepatectomy phase, K increased to 7.8 mmol/L; therefore, the operation was stopped, and patient received 20 ml calcium gluconate 10%, 1150 ml $NaHCO_3$, and 20 U to 210 U regular insulin. This led to a decrease of serum potassium to 4.09 mmol/L without episode of hypoglycemia. The hepatectomy was then done, and the second phase of the operation was followed.

Table 1: Baseline laboratory and paraclinical data of a 49-year-old man, who presented severe hyperkalemia during liver transplantation

AST	135 IU/L	BUN	8 mg/dl
ALT	88 IU/L	Cr	0.9 mg/dl
Albumin	3 g/dl	Na	135 mmol/l
Alkaline -ph	890 IU/L	K	3.8 mmol/l
Bilirubin(Total/Direct)	5.6/1.5 mg/dl	FBS	126 mg/dl
PT(patient/control)	25/13 sec	Echocardiogrphy	NL
INR	2.5	Chest X-ray	NL
Hb	12.8 g/dl	Electerocardiography	NL
Platelet	30 1000/uL	Encephalopathy	None
WBC	3700 /uL	Ascites	Moderate

Table 2: Metabolic parameters during anesthesia of a 49-year-old male, who presented severe hyperkalemia during liver transplantation

	5 min anesthesia	40 min anesthesia	90 min anesthesia	100 min anesthesia	120 min anesthesia	130 min anesthesia
PH	7.52	7.41	7.37	7.33	7.39	7.41
PO_2	225	162.8	180.6	170.8	178.1	169.6
SPO_2	99.7%	99.4	99.5	99.5	99.6	99.3
PCO_2	29.1	28	27	29	36	33
HCO_3	23.7	19.1	18.9	18	26	23
Base excess	1.3	-1.9	-2.8	-4.6	2.1	1.9
Na (mmol/l)	138	137	139	138	140	140
K (mmol/l)	4	4.9	6.5	7.8	6.0	4.09
Hb (g/dl)	11.5	10.8	10.1	10.2	10.0	9.8

BE: base excess

Discussion

Severe hyperkalemia frequently occurs immediately after revascularization during orthotropic liver transplantation.[5,6] There is, however, only one report on severe pre-anhepatic hyperkalemia in living-related liver transplantation.[7]

The changes in metabolic and hemodynamic parameters in different phases of the liver transplantations surgery vary significantly. One of such parameters is serum potassium that may increase dramatically during any phase of the operation.[1] Identification of risk factors for hyperkalemia during phases (pre-reperfusion, early post-reperfusion and late post-reperfusion) of orthotropic liver transplantation are very important. Moreover, risk factors in one phase may not apply to others. Using multivariate logistic regression analyses, a previous study determined independent risk factors for development of hyperkalemia in three phases of orthotropic liver transplantation.[3] The study showed that the incidence of pre-reperfusion hyperkalemia was less than post-reperfusion one, and higher baseline serum potassium and red blood cell transfusion were independent risk factors for the development hyperkalemia in the pre-reperfusion phase.[3] The study suggested that since higher baseline potassium and red blood cell transfusion were two predictors of pre-anhepatic hyperkalemia, insulin should be administered intravenously as soon as the transfusion begins in patients with a baseline potassium above 4.0 mmol/L (1–2 IU of regular insulin for each unit of red blood cells).[7]

The amount of bleeding during hepatectomy was less than 400 cc in the present case; therefore, no blood transfusion or administration of regular insulin was performed. Although baseline potassium was 4 mmol/L and urine output was above 500 ml during three hours of hepatectomy, ligation of hepatic artery gradually increased potassium reaching 7.8 mmol/L near the end of hepatectomy. When subjected to stress, liver can release a large amount of intracellular potassium.[3] Therefore, ligation of hepatic artery may have acted as a stress causing ischemia, which resulted in the flow of potassium from liver into systemic circulation and subsequent hyperkalemia. Treatment of hyperkalemia is mandatory to prevent cardiac arrest during operation. The most powerful and rapid-acting agent to decrease serum potassium is insulin. The effect of insulin on serum potassium occurs within seconds after insulin administration.[3] In a person with normal liver, majority (70%) of potassium uptake by insulin occur in the liver tissue. But patients with end stage liver disease due to liver cirrhosis have potassium intolerance, which means that potassium uptake response to insulin is very sluggish and unusual dose of insulin is required.[1,3] Moreover, cirrhotic patients have abnormal cellular glucose uptake and metabolism due to marked insulin resistance. This might be the reason for hypoglycaemia in our case. The lack of hypoglycaemia might also be due to the administration of methylprednisolon (15 mg/kg) for immune suppression therapy.[3] The changes in serum potassium in the present case suggest that it is necessary to take care of the changes of serum potassium concentration not only in post-reperfusion but also in pre-anhepatic stage during liver transplantation.

Conflict of Interest: None declared.

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