Hyperpotassemia during Major Vascular Surgery: a Possible Indicator of Visceral Infarction

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During major vascular surgery, visceral blood perfusion may be disturbed temporarily, but occasionally lasts long enough to cause hypoxic insult. Recently we experienced acute hyperpotassemia during an aortic aneurysmal repair. We report a case of hyperpotassemia caused by visceral infarction in order to alert physicians to the accurate cause of unexplained hyperpotassemia which developed during and following major vascular surgery.

Report of a Case

A 63-year-old male, 159-cm in height and 62-kg in weight underwent elective repair of the descending aortic aneurysm, just above the diaphragm. Preoperative laboratory data were as follows: Na 142 mEq·l^{-1}, K 4.1 mEq·l^{-1}, Cl 109 mEq·l^{-1}, Ca 4.1 mEq·l^{-1}, GOT 13U, GPT 6U, LDH 241U, and alkaline phosphatase 141 IU. To perfuse the distal part of the aneurysm, a temporary bypass was instituted between the thoracic aorta and the left external iliac artery prior to cross-clamping the aorta. During the proximal anastomosis with a teflon graft being performed, the adequacy of blood flow via the bypass cannula was ascertained by arterial pressure wave monitoring of the right dorsal pedal artery. Following the proximal anastomosis, the anatomosis between the teflon graft and the abdominal aorta was started. At this time, bleeding from the proximal end of the abdominal aorta began to increase and the pedal arterial pressure waves gradually disappeared. This was caused by malpositioning of the distal end of the bypass cannula. A large amount of blood began to leak retrograde out of the dissected, false, intimal lumen at the free end of the abdominal aorta. To avoid further bleeding the temporary bypass was clamped and removed. This stopped blood perfusion below the celiac artery. Radial arterial pressure was maintained about 80/60 mmHg by rapid blood transfusions. Infusion of dopamine (5 μg·kg^{-1}·min^{-1}) and epinephrine (0.2–0.4 μg·kg^{-1}·min^{-1}) did not increase arterial blood pressure effectively. Blood pressure increased by about 20 mmHg following the administration of 20 ml of 2w/v% Ca-gluconate. At this time, laboratory data were as follows: Na 145 mEq·l^{-1}, K 6.1 mEq·l^{-1}, Ca 4.5

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$mEq \cdot l^{-1}$, PH 7.25, Pa_{CO_2} 32 mmHg, Pa_{O_2} 323 mmHg (F_{IO_2} 1.0), and BE-11.9 $mEq \cdot l^{-1}$. Arterial blood pressure was increased by about 25 mmHg temporarily following administrations of 36 mEq of sodium bicarbonate and 10g of glucose i.v. The distal anastomosis was completed and the aorta was declamped about 45 minutes later. The patient remained in shock state with systolic arterial pressure of about 80 mmHg and pulmonary artery pressure of 50/24 mmHg. About 2000 ml of packed red cell were transfused within 1 hour while the abdominal aorta was clamped. During this procedure pulmonary hemorrhage developed. At this time, laboratory data were as follows: Na 136 $mEq \cdot l^{-1}$, K 7.3 $mEq \cdot l^{-1}$, PH 7.17, Pa_{CO_2} 59 mmHg, Pa_{O_2} 198 mmHg, and BE-9.0 $mEq \cdot l^{-1}$. Peaked T-waves were observed on the electrocardiogram. Twenty ml of 50% glucose solution and 4 units insulin were infused. The blood glucose level decreased from 370 $mg \cdot dl^{-1}$ to 198 $mg \cdot dl^{-1}$. Diuresis resumed following declamping of the aorta and administration of furosemide 400 mg i.v. bolus. Data at this time were as follows: PH 7.20, and K 7.0 $mEq \cdot l^{-1}$. About 150 minutes after declamping, the potassium level decreased to 4.3 $mEq \cdot l^{-1}$. Circulatory status improved before the patient left the operating room. Pulsation of the dorsal pedal arteries was well-palpable in the ICU but the patient became oliguric. Immediate postoperative laboratory data were as follows: Na 137 $mEq \cdot l^{-1}$, K 5.1 $mEq \cdot l^{-1}$, CL 84 $mEq \cdot l^{-1}$, GOT 136U, GPT 33U, LDH 772 IU, and CPK 493 $U \cdot ml^{-1}$. Twelve hours after the surgery, laboratory data were as follows: Na 126 $mEq \cdot l^{-1}$, K 5.3 $mEq \cdot l^{-1}$, CL 84 $mEq \cdot l^{-1}$, GOT 5028U, GPT 1499U, LDH 9214 IU and blood sugar 377 $mg \cdot dl^{-1}$. An abdominal distention due to ileus gradually developed. An emergency laparotomy performed 30 hrs after the aneurysmal repair revealed multiple patchy necrosis on the liver surface and impending necrosis of the whole intestine except the descending colon. The celiac artery was partially occluded by an embolus of atheromatous flake and the superior mesenteric artery was also almost completely occluded. These emboli were surgically removed, but the patient ultimately died 57 hours after the aneurysmal surgery.

Discussion

Metabolic and/or respiratory acidosis, and massive blood transfusion are the major causes of hyperpotassemia[1,2]. In addition, massive and/or multiple visceral infarctions release intracellular potassium into blood circulation. However, visceral infarction has rarely been considered the cause of hyperpotassemia during the same clinical situation as the present case, because it is practically difficult to quantify to what extent hypoxia of the lower extremities would contribute to the development of systemic acidosis and subsequent hyperpotassemia after a high aortic cross-clamp was removed. Severer acidosis was followed by the suprarenal high aortic cross clamp than by the lower one in the pelvis[3].

In this case, it was speculated that the tip of the temporary bypass cannula peeled off the atheromatous clefts, which obstructed the hepatic and the celiac arteries.

The liver cells are rich in potassium[4] and take part in extrarenal potassium metabolism[5]. When the liver and the intestines sustain hypoxic insult, intracellular potassium ions are released into blood circulation leading to hyperpotassemia. Mizuyama has reported a stepwise increase in serum potassium concentration everytime a clamp of the intraabdominal major artery was removed[6]. Bercov-

itch reported hyperpotassemia in hepatic necrosis[7]. Laboratory study has substantiated that potassium ions are released from the intestine following hypoxic distress[8]. However, whether massive hepatic necrosis is associated with a concomitant hyperpotassemia remains controversial[9]. Hepatic necrosis with renall failure produces severer hyperpotassemia than that without one[10].

There is usually a time-lag of several hours or more between the hypoxic insult and the hepatic enzymatic manifestation. In this respect, the present case was in accordance with the earlier report[9].

Another potential mechanism of the hyperpotassemia is the embolism of the superior mesenteric artery which is the only source of blood supply to the pancreas. As the result of the embolism, basal secretion of insulin is disturbed, and further development of hyperpotassemia is potentiated by the failure of transcellular shift of potassium[11], even though the serum level of insulin was not measured in the present case.

According to Simmons[12], the serum potassium level increases or decreases by 0.6 $mEq \cdot l^{-1}$ every change in pH of 0.1. The serum potassium 7.3 $mEq \cdot l^{-1}$ in this case was incompatible with pH 7.17, therefore, neither metabolic nor respiratory acidosis was not considered the major determinant of the hyperpotassemia.

Development of pulmonary edema was due probably to compromized myocardial contractility induced by hyperpotassemia, acidosis, and myocardial depressant factors released from the anoxic viscera. The relative overtransfusion during the period of high aortic clamp also contributed to the development of pulmonary edema.

Physicians should investigate the possible causes of visceral infarction as soon as possible, when hyperpotassemia is persistent in spite of routine treatments including correction of acidosis. Cyanosis of an individual organ, palpation of feeding arteries, venous distention, serum insulin level and/or response to insulin administration may suggest the possibility of visceral infarction. Recent development of diagnostic aids such as echography has made diagnosis of serious events in the parenchymal organs much easier.

In conclusion, there is an possibility of visceral infarction and/or necrosis when an unexplained hyperpotassemia develops perioperatively. The physician should take measures to investigate the possible causes of visceral infarction.

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