Intraoperative Management of a Patient Undergoing Extracorporeal Liver Surgery (Bench Surgery)

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Extracorporeal liver surgery is a relatively new surgical procedure that involves liver autotransplantation, and promises to increase the resectability rate in patients with advanced tumors as well as to improve the radicality of tumor resection[1]. Originally described by Pichlmayr in 1988[1], the term "bench surgery" was applied because surgeons were sitting on a bench while they performed this procedure. We describe here the intraoperative management of a patient undergoing extracorporeal liver surgery for cholangioma.

Case Report

A 59-year-old man (53 kg in weight and 164 cm in height) with a diagnosis of massive cholangioma in the left lobe of the liver was admitted to our hospital. The tumor was so large that it compressed the inferior vena cava and the right hepatic vein, making conventional liver resection impracticable. Thus, extracorporeal liver surgery was scheduled to radically resect the tumor, after informed consent was obtained from the patient following full explanation of this new procedure.

He had a history of mild hypertension plus a 20 pack-year history of cigarette smoking. Respiratory examination revealed bilateral dry rhonci sounds and prolongation of the expiratory phase of respiration. These pulmonary manifestations improved markedly following bronchodilator therapy.

Pulmonary function test indicated an obstructive pulmonary disturbance: the $FEV_{1.0\%}$ was 56% and the $RV_{\%}$ was 53%. Arterial blood gases while breathing room air were as follows: P_{O_2}, 80 mmHg; P_{CO_2}, 40 mmHg; and pH, 7.41. A chest radiograph showed changes consistent with mild emphysema but no effusions or infiltrates. Other laboratory data indicated mild liver dysfunction and mild anemia. The serum levels of electrolytes, creatinine, total bilirubin, and total protein as well as the prothrombin time and activated partial thromboplastin time were all within the normal range. The ECG showed premature atrial contractions at a rate of about 2 beats·min^{-1}.

In anticipation of a large blood requirement during surgery, a total of 30,000 ml of blood consisting of 150 units of packed

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red blood cells and 150 units of fresh frozen plasma was prepared. To deal with sudden massive intraoperative hemorrhage, we produced a rapid blood infusion device from a conventional infusion system for cardioplegic solution with a heat exchanger (MERA CP-II) and a conventional roller pump. With this system, blood transfusion was possible at rates of up to 600 $ml \cdot min^{-1}$. We prepared two such systems for this patient, thus allowing blood transfusion at a rate of up to 1,200 $ml \cdot min^{-1}$. In addition, 50 units of concentrated platelets and 2,000 units of factor VIII were prepared to treat postanhepatic coagulopathy. Thrombelastograpy was used for monitoring the coagulation system. Calcium chloride was provided to treat citrate intoxication, as well as sodium bicarbonate for metabolic acidosis, and vasopressors such as dopamine, ephedrine, epinephrine, and norepinephrine for hypotension. A defibrillator was also provided in case of ventricular fibrillation.

Atropine sulfate (0.5 mg, i.m.) and hydroxydine chloride (50 mg, i.m.) were administered 30 min before induction of anesthesia. Anesthesia was induced with thiamylal (300 mg, i.v.) and succinylcholine chloride (50 mg, i.v.), and was maintained with isoflurane in an air/oxygen mixture. Nitrous oxide was avoided for the maintenance of anesthesia because of its tendency to increase the bowel gas volume and to expand the intravascular gas volume of air embolism[2]. Muscle paralysis was accomplished with pancuronium bromide. As anesthetic supplements, 10 mg of diazepam and 1,000 μg of fentanyl were used to prevent intraoperative awareness.

After the induction of anesthesia, two 8.5 Fr. i.v. catheters were inserted into the right internal jugular vein; one was for a flow-directed pulmonary artery catheter and the other was for volume infusion. Another 8.5 Fr. i.v. catheter was placed in the left internal jugular vein for volume infusion. Both the catheters for volume infusion were directly connected to the rapid infusion devices as mentioned above. Moreover, a 14-G antecubital i.v. catheter for additional volume infusion and a 20-G radial artery catheter for pressure monitoring and blood collection were also inserted.

During surgery, about 10 $ml \cdot kg^{-1} \cdot min^{-1}$ of i.v. fluid without potassium and about 0.3 $g \cdot kg^{-1} \cdot hr^{-1}$ of glucose were administered. To maintain adequate renal and hepatic perfusion, 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of dopamine and 10 $ng \cdot kg^{-1} \cdot min^{-1}$ of prostaglandin E_1 were also administered continuously. Changes in hemodynamics, arterial blood gases, serum electrolytes, blood lactate, and the platelet count throughout surgery are summarized in table 1.

The duration of the preanhepatic phase was 9 hr and 10 min and the total amount of hemorrhage during this phase was about 1,000 ml. The mean urine output was 1.4 $ml \cdot kg^{-1} \cdot hr^{-1}$ without diuretics and the vital signs remained stable throughout this period.

During the anhepatic phase, we performed veno–venous (V–V) bypass from the portal and femoral veins to the axillary vein using heparin-bonded tubing circuits and a centrifugal pump. Before inserting the catheters for V–V bypass, 1,000 units of heparin was administered to mildly prolong the activated clotting time (ACT) from 109 to 160 seconds and to prevent clot formation in the circuit and pump. No additional heparin was administered thereafter. Just after the initiation of V–V bypass, blood from the femoral vein did not drain smoothly and the centrifugal pump showed frequent interruptions of movement over a period of several minutes. Severe hemoglobinuria was observed thereafter, which was treated with 4,000 units of haptoglobin and 10 mg of furosemide. Serum ionized Ca^{++} level which had been 0.93 $mmol \cdot l^{-1}$ just before the anhepatic phase decreased to 0.74 $mmol \cdot l^{-1}$ one hour after the start of the anhepatic phase, and this was treated with 800 mg of $CaCl_2$.

The duration of the anhepatic phase was 5 hr and 17 min and the total amount of hemorrhage during this phase was about 500 ml. Mean urine output

Table 1. Changes in hemodynamics, arterial blood gases, serum electrolytes, blood lactate, and platelet count during the preanhepatic, anhepatic, and postanhepatic phases

	Preanhepatic phase			Anhepatic phase			Postanhepatic phase			
	A	B	C	D	E	F	G	H	I	J
MAP (mmHg)	93	106	88	106	83	83	63	65	99	89
HR (beats·min^{-1})	69	90	94	87	88	80	109	91	89	91
CI (l·min^{-1}·M^{-1})	2.34	3.49	3.76	2.97	3.09	3.00	2.82	2.21	3.84	2.61
PAWP (mmHg)	15	13	14	15	9	10	11	9	11	9
CVP (mmHg)	11	9	8	7	6	6	6	4	9	7
pH	7.34	7.33	7.32	7.31	7.32	7.34	7.35	7.36	7.34	7.36
Pa_{O_2} (mmHg)	308	281	271	323	333	334	334	329	322	297
Pa_{CO_2} (mmHg)	41	43	45	41	44	39	42	37	39	43
BE (mEq·l^{-1})	−2.9	−2.1	−2.4	−4.7	−3.1	−3.4	−1.8	−2.9	−3.9	0.0
Na (mEq·l^{-1})	137	134	132	130	130	130	133	130	131	131
K (mEq·l^{-1})	4.6	3.9	4.0	4.0	3.9	3.8	3.9	3.7	3.7	3.6
Ca^{--} (mmol·l^{-1})	1.20	1.05	0.93	1.02	1.04	1.31	0.91	0.98	0.95	0.96
LA (mmol·l^{-1})	1.23	1.06	0.92	2.78	2.83	3.24	5.59	4.78	4.23	3.18
Plts ($\times 10^3 \cdot \mu l^{-1}$)	175	121	131	119	118	116	78	66	106	130

Phases: A, before the start of operation; B, 2 hr after the start of operation; C, just before the anhepatic phase; D, 2 hr after the start of the anhepatic phase; E, 4 hr after the start of the anhepatic phase; F, just before the end of the anhepatic phase; G, 5 min after reperfusion; H, 15 min after reperfusion; I, 1 hr after reperfusion; J, at the end of the operation.

Abbreviations: MAP, mean arterial pressure; HR, heart rate; CI, cardiac index; PAWP, pulmonary artery wedge pressure; CVP, central venous pressure; BE, base excess; Ca^{--}, ionized calcium; LA, blood lactate; Plts, platelet count.

was 3.8 ml·kg^{-1}·hr^{-1}. Although temporary hemoglobinuria and a decrease in ionized Ca^{++} were seen, vital signs remained stable and no significant hemodynamic changes were observed during this long anhepatic phase. Neither metabolic acidosis nor hypoglycemia developed, although mild hypothermia of 34.1°C was noted.

Just before reperfusion of the reimplanted liver, a total of 100 mEq of sodium bicarbonate and 2,000 mg of $CaCl_2$ were administered to prevent metabolic acidosis and hyperkalemia following revascularization of the liver. To treat abrupt massive hemorrhage, we filled the reservoirs of the 2 rapid blood infusion devices with the following reconstructed blood: 5 units of packed red blood cells, 5 units of fresh frozen plasma, and 500 ml of 0.9% sodium chloride. The hematocrit of this mixture was about 27%. Thus, although about 4,000 ml of hemorrhage occurred from the cut surface of the liver just after revascularization, this was easily treated by the two rapid blood infusion devices and the systolic blood pressure was maintained more than 90 mmHg. Coagulopathy was also not observed and the subsequent anesthetic course was uneventful. The duration of the postanhepatic phase was 3 hr and 50 min and the total amount of hemorrhage during this period was about 5,000 ml. Mean urine output was about 3.3 ml·kg^{-1}·hr^{-1} during the postanhepatic period.

Discussion

Extracorporeal liver surgery is still a fairly new surgical procedure. Similarly to conventional liver transplantation, this procedure carries the risk of serious intraoperative complications[3], including hypotension due to massive hemorrhage, citrate intoxication, hypothermia, hyperkalemia, thromboembolism, and air embolism. Moreover, the relatively long anhepatic phase may produce other problems such as hypo-

glycemia and metabolic acidosis due to absence of the liver.

In this case, we experienced temporary hemoglobinuria, hypothermia, and a decrease in ionized Ca^{++} during the anhepatic phase. The hemoglobinuria appeared to be caused by mechanical trauma to red blood cells at occurring the initiation of V–V bypass. Accordingly, to avoid hemolysis during V–V bypass, it should be thoroughly confirmed that blood drains smoothly from the femoral vein at the start of bypass.

Accidental hypothermia during the anhepatic phase has been reported previously[4]. This may be mainly due to the absence of the metabolic activity of the liver and heat loss from the widely opened abdominal cavity. A warming blanket, heated humidifier, and wrapping of the bowel with warm surgical sponges were used in this case. In addition, all the blood products transfused were warmed. To better maintain body heat, the frequent use of warm irrigation of the bowel may perhaps be recommended[4].

A fall of the platelet count and thrombus formation in the pump head may occur when a bypass is perfused without heparinaization[5,6]. Accordingly, to prevent clot formation in the circuit and the pump, a small dose of heparin was administered before V–V bypass in this case. No decrease in the platelet count was observed and no unmanageable bleeding related to the heparin was encountered either. However, a small amount of thrombus was observed in the pump head after surgery. This suggests that, as with heparin-bonded tubing circuits, there is a need to develop a heparin-bonded pump head in the future[5].

The decrease in ionized Ca^{++} during the anhepatic phase is caused by the disturbance of citrate metabolism. In addition to the absence of the liver, factors known to affect citrate metabolism include body temperature and the acid-base status. Decreases in ionized Ca^{++} prolong the Q–T interval and impair cardiac function[7]. Further more, a profound decrease in ionized Ca^{++} may produce abnormalities in the extrinsic and intrinsic clotting pathways. The frequent assessment of hemodynamic function and of serum ionized Ca^{++} levels were useful to prevent disarrangements secondary to a decrease in ionized Ca^{++} in this patient.

The most marked hemodynamic changes were seen just after revascularization, but the abrupt hemorrhage from the cut surface of the liver was treated well by our rapid blood infusion systems. Although mild decreases in ionized Ca^{++}, blood pressure, and cardiac output were observed, neither hyperkalemia, acidosis nor arrhythmias were observed. However, profound hyperkalemia may occur if the UW solution[8] (the cold storage solution for liver preservation including 120 $mEq \cdot l^{-1}$ of potassium) is not completely flushed out of the liver before revascularization. In addition, serious arrhythmias and profound hemodynamic changes may occur if the air in the vena cava is incompletely evacuated before revascularization[2].

In summary, we described the intraoperative management of a patient undergoing extracorporeal liver surgery for giant cholangioma. Similarly to the case in conventional liver transplantation, special consideration with regard to the possible complications of this new procedure is essential for achieving a successful outcome.

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