Hemodynamic Changes during Liver Resection with Monitoring Right Ventricular Ejection Fraction (RVEF)

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Liver resection is one of the more hemorrhagic surgical procedures. We experienced the following case in which total blood loss was not great but the hemodynamic changes were severely harmful due to rapid bleeding along with surgical procedures. We observed that right ventricle ejection fraction (RVEF) was decreased as well as cardiac output (CO) during rapid bleeding with surgical procedure including pressure to inferior vena cava.

Case Report

Patient was 59-year-old, 62-kg woman with liver echinococcosis scheduled for a right lobectomy of the liver. She had undergone a abdominal hysterectomy in the past due to myoma uteri without problems. Past medical history was unremarkable except five years medication with anti-hypertensive drugs including angiotensin converting enzyme inhibitor. Blood pressure was well controlled at about 120/80 mmHg. Chest radiogram, ECG, and other laboratory data were within normal limits. Serology studies were positive for echinococcosis. Preoperative medications included atropine 0.5 mg and hydroxyzine 50 mg im.

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Before the induction of anesthesia, epidural catheterization was performed through T6 and T7. No local anesthetic was injected into epidural space until the operation was finished. A 7F three lumen catheter via the left external jugular vein, a right radial artery catheter and a right internal jugular pulmonary artery catheter were placed. Intraoperative monitoring included ECG, end-tidal CO_2, arterial O_2 saturation obtained by pulse oximeter, blood pressure, central venous pressure (CVP), pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP). Cardiac output (CO), and right ventricular ejection fraction (RVEF) were obtained by the thermodilution method (REF1, Edwards Laboratories). The right ventricular end-diastolic volume (RVEDV) and the right ventricular end-systolic volume (RVESV) were calculated from stroke volume and RVEF.

Anesthesia was induced with thiamylal 300 mg followed by succinylcholine 60 mg and maintained with enflurane and intermittent administration of pancuronium and fentanyl. Physiologic variables measured after the induction of anesthesia were within normal limits. Heart rate was 82 bpm, blood pressure 140/90 mmHg, PAP 32/19 mmHg, PCWP 17 mmHg, CVP 10 mmHg, CO 4.46 $l \cdot min^{-1}$, RVEF 0.49, RVEDV 110 ml during stable stage of the surgery (table 1).

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Table 1. Hemodynamic changes during liver resection

Variables		Stable state	During episode	After episode
Time		11:00	12:25	13:10
HR	beats·min^{-1}	82	76	82
BP	mmHg	140/90	60/40	120/80
PAP	mmHg	32/19	23/12	29/16
PCWP	mmHg	17	9	10
CVP	mmHg	10	5	8
CO	l·min^{-1}	4.46	1.81	3.75
CI	l·min^{-1}·m^2	2.69	1.09	2.26
SV	ml	54	24	46
RVEF		0.49	0.27	0.47
RVEDV	ml	110	89	98
RVESV	ml	56	65	52

Values are averages made from at least three times measurements. HR: heart rate, BP: blood pressure, PAP: pulmonary artery pressure, PCWP: pulmonary capillary wedge pressure, CVP: central venous pressure, CO: cardiac output, CI: cardiac index, SV: stroke volume, RVEF: right ventricular ejection fraction, RVEDV: right ventricular end-diastolic volume, RVESV: right ventricular end-systolic volume.

eased liver were uneventful and vital signs remained stable. By the time of resection of the right lobe of the liver, the patient had rapidly lost an estimated 400 or 500 ml blood. At this time the vena cava inferior was compressed due to surgical procedure. Hemodynamic changes occurred in spite of rapid blood transfusion including 600 ml packed red cells with saline and 500 ml 10% human albumin solution. No vasoconstrictors or catecholamines were used during the entire procedure. The blood pressure decreased to 60/40 mmHg, PCWP to 9 mmHg, CVP to 5 mmHg, CO to 1.81, and RVEF to 0.27, RVEDV to 89 ml, and heart rate to 76 bpm. Twenty minutes after the surgical dissection of the right lobe of the liver, the hemodynamic state became normal as indicated in the table.

Pulse, blood pressure, and temperature remained stable after this episode, and after 90 min observation at the end of the 4-hour procedure muscle relaxation was reversed with edrophonium 60 mg and atropine 1.5 mg. The patient could follow simple commands and her trachea was extubated. The estimated blood loss was 1000 ml and we transfused 800 ml packed red cells with saline, 600 ml fresh frozen plasma, 500 ml 10% human albumin solution and 3300 ml lactate Ringer solution.

Discussion

In this case, we experienced a very low cardiac output state for about 20 min although total blood loss was not great. Liver resection is one of the more hemorrhagic surgical procedures and it may be unavoidable to apply pressure to the inferior vena cava during the procedure. We previously reported the mixed venous oxygen saturation decreases severely during massive bleeding and suggested that the low cardiac output caused this decrement of mixed venous oxygen saturation (1). In this case, RVEF and RVEDV were measured as well as CO, and RVEF decreased 45% while CO decreased 59% when the right lobectomy was performed.

Right ventricular function is exquisitely dependent upon ventricular afterload (2). As Hurford, et al., depicted, if diastolic filling is maintained, as afterload increases, right ventricular ejection fraction decreases and the

ventricle dilates to maintain stroke volume (3). There are many reports demonstrating low RVEF due to pulmonary hypertension. In the present case, the rapid bleeding and the pressure to the inferior vena cava caused the factitious hypovolemia and low cardiac output but not pulmonary hypertension.

We interpreted the mechanism of the decrement of RVEF in this case possibly as [I] right ventricular end-systolic volume was not changed as RVEDV decreased within physiologic limits such that the Frank-Starling law worked [II] low coronary perfusion pressure caused decreased cardiac contractility, [III] sympathetic nervous system excitation by vasoreflexes caused a change of timing of right ventricular sinus (inflow) and conus (outflow) contraction. Armour, et al. and Raines, et al., demonstrated that during stellate stimulation, the conus contracts earlier, reducing or eliminating the delay of contraction between the sinus and conus[4,5].

Although ECG changes suggesting ischemia were not observed during and postoperative period, the coronary perfusion pressure during the episode was obviously lower than that of the stable state. In addition, heart rate was not changed during the hypotensive episode so that it was considered that sympathetic tone did not increase remarkably. Calculated right ventricular end-systolic volume increased 16% during the episode so that the right ventricular contractility was apparently decreased. These facts demonstrated that the decrement of the RVEF was caused by reasons [I] and [II] in this case.

In conclusion, during rapid bleeding along with surgical procedure including pressure to the inferior vena cava such as this case the cardiac function and the contractility can decrease severely. Monitoring RVEF as well as CO was efficiently useful to evaluate cardiac function.

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