Case Report

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Open right hepatectomy in a patient with hepatocarcinoma and pulmonary hypertension

Abstract

Liver resection is the most effective treatment for primary and secondary malignancies. A major limitation of liver surgery is the high morbidity and mortality related to blood loss and liver function. The difficulty of the procedure and perioperative management may increase due to the association of comorbidities, such as the present case of a patient with hepatocarcinoma and pulmonary hypertension.

Keywords: hepatectomy, hepatocarcinoma, liver, central venous pressure, pulmonary hypertension

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Introduction

Liver cancer is the fifth most commonly diagnosed cancer in population in worldwide, and is the third most frequent cause of cancer death. Various factors may limit the possibility of surgical resection, such as tumor spread, having too small of a residual liver to provide adequate functional hepatic reserve, and involvement of the inferior vena cava or three main hepatic veins. Only 10-37% of patients with hepatocarcinoma could be offered the curative hepatic resection at the time of diagnosis.

Case report

A 64-year-old female with a diagnosis of hepatocarcinoma (segments V and VI) scheduled for right hepatectomy. She has a history of chronic obstructive pulmonary disease, Systemic arterial hypertension and Pulmonary Arterial Hypertension.

Echocardiogram shows preserved left ventricular ejection fraction (LVEF) 57%, right ventricle (RV) and right atrium (RA) with alterations. Moderately dilated right ventricle (RV) with significant pulmonary arterial hypertension (PAH) data severely dilated right atrium. Dilated inferior vena cava, color Doopler suggesting patent foramen ovale (PFO) with left-right shunt (Interatrial septal shunt). Liver and kidney function tests were normal, and the hemoglobin was 10 g/dL.

She was admitted to the operating room. Right hepatectomy is performed under balanced general anesthesia, with invasive monitoring, arterial line and central venous pressure (CVP) monitoring. Intravenous induction and orotracheal intubation were performed, and the maintenance was given with sevoflurane. Intravenous infusions of Fentanyl, dexmedetomidine and lidocaine. Tranexamic acid 1g was administered, and we lowered CVP to 5-6mmHg through fluid restriction, furosemide 20mg IV and an increase in the concentration of hypnotics. Norepinephrine and milrinone infusions are maintained from the beginning until the end of the procedure. Bolus of vasopressin was used during procedure.

4 PRINGLES were performed, with a total duration of 46 min. Bleeding of 1700 ml, thoracic ultrasound was used to search diffuse B lines of the lung, and data of pulmonary edema, without finding any of them. Resuscitation was performed with 1000 ml of crystalloids, 6 globular units, 2 fresh plasma and bicarbonate. Surgical duration of 5.5 hours. Post-resuscitation arterial blood gases are shown in a Table 1.

Table 1 Arterial gasometry

Intraoperative arterial gasometry		
Values	**During resuscitation**	**After resuscitation**
pH(7.35-7.45)	7.35	7.31
pCO_2 (35-45 mmHg)	27	40
HCO_3 (22-26 mEq/L)	14.9	20.5
Na (135-145 mmol/L)	147	137
K(3.5-5 mmol/L)	2.9	5
Glucose(70-140mg/dL)	127	234
Lactate (<2 mmol/L)	2.1	3.9
Hemoglobin (13-17g/dL)	7.4	9
Hematocrit (35-48%)	24	28.8
BE-b (+-2.5mmol/L)	-9.7	-6.2

She was extubated without complications and transferred to the Intensive care unit (ICU) with vasoactive drug support**:** norepinephrine that is withdrawn at 24 hours, ICU discharge at 48 hours, the patient has adequate evolution, laboratories without data of liver failure, discharged to her home after 10 days (Figure 1).

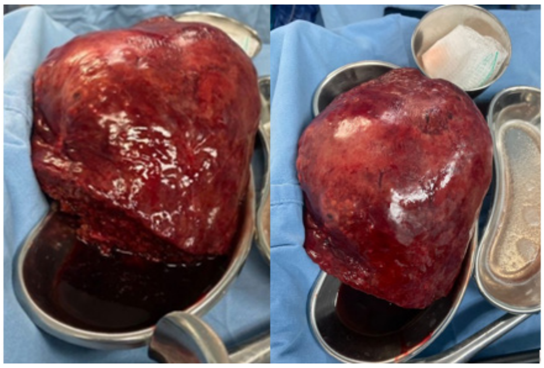

Figure 1 Piece of liver, right hepatectomy.

Discussion

Intraoperative bleeding includes hemorrhage not only from liver parenchymal division, but also from perihepatic ligament dissection, tumor separation from adjacent tissues, liver surface after being cut during the resection phase, and accidental rupture of the tumour.[1] Blood losses of >10 liters have been reported in major liver resections, however, modern multimodal perioperative techniques (advances in liver resection techniques, new bipolar coagulation and ultrasonic aspirator technologies, hemostatic agents, advanced anesthetic monitoring) have reduced losses to 300–900 mL.[2]

Strategies to reduce intraoperative bleeding include low Central Venous Pressure (CVP), use of antifibrinolytics such as tranexamic acid or aprotinin and some vascular occlusion techniques.[2] The use of intermittant Pringle's maneuver (Clamping of the portal triad) during liver transection is effective in reducing blood loss, but there is a limit to the duration that can be applied. Prolonged application of Pringle's maneuver for more than 120 minutes may have a deleterious effect on liver function.

The main source of bleeding comes from the hepatic veins, which lack valves, so it is important to control central venous pressure. It is well documented that CVP >5 mmHg significantly increases blood loss and morbidity and mortality associated with the hepatectomy procedure.[3] Justin T Huntington et al. in their literature review, examine prospective studies of the last 20 years and analyzed major hepatectomies, the strategies supported most convincingly to reduce blood loss, are particularly low CVP and vascular occlusion maneuvers (total hepatic inflow occlusion). It is recommended during the parenchymal section to maintain a pressure between 2 and 5mm Hg.[3]

Techniques used to maintain a low CVP may include: trendelenburg, avoidance of positive end-expiratory pressure (PEEP), minimal fluid administration, use of diuretics such as furosemide, vasodilators such as glyceryl trinitrate and recently milrinone has been described as a good option. Simply reducing intravenous fluids to about 75mL/hour can lower CVP in many patients without affecting renal function, while producing a statistically significant reduction in blood loss.[2] If intravenous fluid restriction does not allow the target CVP to be achieved, pharmacological methods would probably be necessary.

Peng Yang et al. analyzed 52 patients who underwent elective open hepatectomy were enrolled in the study and randomly divided into two groups: milrinone (M) group and nitroglycerin (NG) group. They found that the blood loss of group M, less than that of group NG meanwhile, time of hepatectomy and hepatic hilum occlusion were shorter in group M. Compared with the NG group, cardiac index and stroke volume index were higher in group M in the operation. The norepinephrine dosage necessary in the operation was of no difference in two groups.[4] Therefore, they conclude that milrinone can adequately maintain a low CVP with the benefit of better hemodynamics.[4]

Perioperative parenteral use of Tranexamic Acid (TXA) reduce the amount of operative blood loss and the need for blood transfusion in elective liver tumor resection.[5] In other recent studies, intraoperative TXA was associated with a 41% reduction in risk of 30-day receipt of Red Blood Cell Transfusions after hepatectomy for Colorectal Liver Metastases. This finding is important to potentially improve healthcare resource allocation and patient outcomes.[6] The first cases of Enhanced Recovery After Surgery (ERAS) protocols in liver surgery only appeared in the scientific journals in 2008. A recently systematic review and meta analyses shows that ERAS protocols in liver surgery reduces hospital cost and improve clinical outcomes, reduces length of hospital stay, readmission rates and mortality.[7]

Within the ERAS protocols, the following elements have a strong recommendation: 1. Preoperative elements: counseling, minimal fasting. 2. Intraoperative elements: antimicrobial prophylaxis (single dose) and skin preparation (2% chlorhexidine), maintain normothermia, intrathecal opioids instead of epidural analgesia, prevention of postoperative nausea and vomiting (PONV). 3. Surgery related: maintain low CVP during liver resection and use of balanced crystalloids, avoid Mercedes benz incision, minimally invasive surgery when is possible, avoidance of nasogastric tube and multimodal analgesia.[8,9]

The update to the hemodynamic definition of Pulmonary Hypertension (PH) is defined as a mean pulmonary artery pressure (mPAP) of >20mmHg, a decrease from the previous definition of mPAP ≥25mmHg.[10] The diagnosis of PH requires a clinical suspicion based on symptoms and physical examination and review of a comprehensive set of investigations to confirm that haemodynamic criterio are met and to describe the aetiology and the functional and haemodynamic severity of the condition.[11] The most useful diagnostic orientation method and non invasive evaluation of PH is echocardiography. The definitive diagnostic method is right cardiac catheterism.[11] Perioperative mortality remains significantly higher in patients with PH than in patients without PH, even when perioperative care is delivered in expert centers.[12]

Adequate coupling of the RV to the pulmonary circulation is essential for maintaining cardiac output in patients with PH. Most patients with severe PH exhibit significant RV maladptation or uncoupling to the pulmonary circulation, with a dilated impaired RV, and high RV filling pressure.[13] Load-dependent changes in RV function can also impact negatively on cardiac output. An excessive increase in RV preload can increase wall stress and endocardial ischaemia, and lead to severe tricuspid regurgitation and systolic RV dysfunction. A disproportionate decrease in preload to a hypertrophied and fibrotic RV with diastolic dysfunction can cause decompensation and a low cardiac output state. Cardiac output should therefore be optimized perioperatively by controlling RV preload and after load, supporting RV function to ensure adequate myocardial perfusion with inotropic support.[14]

Perioperative risk in patients with PH is influenced by patient factors, including severity of PH and RV dysfunction, exercise performance, and functional status; and surgical factors including the urgency, length, and type of surgery.[14] In this case, although the echocardiogram showed a severe dilated RV with preserved systolic function, and because of the low CVP goal in the surgery, Milrinone in low doses started along with Norepinephrine since induction, after arterial invasive line was placed. Invasive haemodynamic monitoring with measurement of CVPs and systemic arterial pressures is recommended, achieving the most haemodynamic stability.

Norepinephrine is a first-line treatment in most centers and improves coronary blood Flow in models of RV failure. Low-dose vasopressin may be a particularly useful selective systemic vasoconstrictor in this setting, as an infusion or even a bolus, which was our case, bolus of vasopressin was used during procedure. In terms of inotropes and inodilators can be use as pre-emptive, before and after anesthetic induction, to improve RV performance.

Conclusion

Morbidity and mortality in hepatectomies can increase due to patient comorbidities, perioperative management is complex, but there can be successful results if the patient is approached appropriately and multidisciplinary.

Acknowledgments

None.

Conflicts of interest

Authors declare that there is no conflict of interest.

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