Cardiac Arrest During Liver Resection: two cases report

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Case Report

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Abstract

Background

Patients with liver diseases often require surgery, and liver resection is a common surgical procedure. Intermittent Pringle Maneuver (IPM) is always used to control blood loss during liver resection surgery, but ischemia-reperfusion injury reduced by IPM will lead rapid change of internal environment, which will increase patients’ perioperative risk.

Case presentation

We reported a 59-year-old male and a 44-year-old male who underwent liver resection surgery for liver cancer and hepatolithiasis respectively. Cardiac arrest occurred during the procedure. Luckily, with high quality of cardiopulmonary resuscitation and electrical debrillation, acidosis correction, as well as the treatment of lowering K\(^+\) and increasing Ca\(^{2+}\), they were rescued. The two patients recovered well without complication in 1-month followed-up.

Conclusion

In liver resection, the anesthesiologists need to pay close attention to the electrocardiography changes, especially at the moment of vascular opening during IPM. In addition, how to ensure the stability of patients’ internal environment during liver resection is an important clinical research work, which will provide evidence-based medical reference for anesthesiologists.

Introduction

Liver resection (LR) is one of the primary treatment strategies for all kinds of liver diseases, such as liver tumors[1], hepatolithiasis[2], and so on. Intermittent Pringle maneuver (IPM) is a common strategy to control of bleeding[3]. During IPM, the rapid change of internal environment will lead to fatal complication. We report two cases of cardiac arrest during LR, which used IPM to control blood loss.

Case Presentation

Patient A

A 59-year-old male (166.5cm, 66.5kg), scheduled for laparoscopic LR because of liver cancer. His results of pre-anesthetic evaluations were in Table 1 (patient A).
Table 1
Results of pre-anesthetic evaluation of two patients

<table>
<thead>
<tr>
<th>Assessment Item</th>
<th>Patient A</th>
<th>Patient B</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Society of Anesthesiologists (ASA)</td>
<td>II</td>
<td>II</td>
</tr>
<tr>
<td>Mallampati Score</td>
<td>I</td>
<td>I</td>
</tr>
<tr>
<td>Child-Pugh Classification</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Metabolic Equivalent (MET)</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>Surgical Risk Assessment</td>
<td>Medium-high risk</td>
<td>Medium risk</td>
</tr>
<tr>
<td>New York Heart Association (NYHA)</td>
<td>I</td>
<td>I</td>
</tr>
</tbody>
</table>

After careful preanesthetic preparation, general anesthesia was induced with proper dose of midazolam, etomidate, sufentanil and rocuronium. 3 minutes after the induction of anesthesia, a 7.5# tracheal catheter was intubated successfully. The patient was connected to a rebreathing anesthetic circuit with sevoflurane as the inhalant anesthetic and was mechanically ventilated throughout the procedure. Monitoring consisted of continuous electrocardiography (ECG), pulse oxygen saturation (SpO$_2$), continuous arterial blood pressure (ABP), central venous pressure (CVP), bispectral index (BIS), partial pressure of end-tidal carbon dioxide (PetCO$_2$), intermittent arterial blood gas analysis (BGA), urine volume.

IPM was used to control blood loss during the surgery, A 5 minutes of ischemic adaptation period was performed before the first IPM, subsequent IPM ischemia time was controlled within 15 minutes, and the intermediate perfusion period was no less than 10 minutes. IPM is performed by ligation of hepatic arteries and portal veins with aseptic elastic compression bands.

193 minutes after the operation began, ventricular fibrillation was appeared at the time of the third cycle of IPM, which was followed by cardiac arrest in several seconds. Monitoring results of K$^+$, Ca$^{2+}$ during this period are shown in Fig. 1.

Cardiopulmonary resuscitation (CPR) was initiated immediately, electrical defibrillation, and medication, sinus rhythm was resumed. The patient was successfully rescued after 37 minutes. The monitoring results of K$^+$, PH, lactic acid (Lac) during the 37 minutes shown in Fig. 2. The operation took 5 hours and 50 minutes, and the patient recovered well in 1-month postoperative follow-up.

Patient B

A 44-year-old male (160cm, 55kg), scheduled for laparoscopic LR because of hepatolithiasis. His results of pre-anesthetic evaluations were in Table 1 (patient B).

A same general anesthesia as patient A was conducted for him, and IPM was also used in this surgery.
183 minutes after the operation began, at the fourth time of opening vascular after IPM, ventricular fibrillation appeared, which was followed by cardiac arrest. The manifestations were similar with those of patient A. We did CPR, electrical defibrillation, and medication, and 24 minutes later, he resumed sinus rhythm. Intraoperative parameters were in Fig. 3–4. The operation took 4 hours and 50 minutes, and the patient recovered well in 1-month postoperative follow-up.

**Discussion**

Liver cancer is a prevalent public health problem, and it is the third (8.3%) most common cause of malignancy-related death globally[4]. Regardless of whether liver cancer is at an early or advanced stage, LR is regarded as the most effective treatment strategy. Hepatolithiasis is a prevalent disease in Asia, and LR is an effective and definitive treatment for patients with hepatolithiasis[2]. Above all, LR is a common surgery in clinical anesthesia work in China. Roughly 30% of cardiac output flows through the liver by a dual blood supply, so it's important to reduce intraoperative bleeding. Control of bleeding from LR consists of vascular inflow occlusion and control of hepatic venous backflow from the caval vein[5]. IPM, which is the common method of vascular inflow occlusion, involves clamping of the portal vein and the hepatic artery in the hepatic pedicle and causes hepatic ischemia. During IPM, liver ischemia leads to anaerobic glycolysis accelerated, and an increased release of lactic acid, an accumulation of oxygen radicals, and a disequilibrium in H+, Na+, and Ca^{2+} homeostasis[6]. On the moment of opening of vascular, internal environment disorder developed, contains hyperkalemia and acidosis. In both of our patients, there was an increase in blood potassium concentration (as Figs. 1 and 3) and a decrease in PH (as Figs. 1 and 3) when the blood vessels were opened.

Hyperkalemia affects the electrical activity of all cells, notably for cardiac muscle. In the ECG changes caused by hyperkalemia, at a serum potassium of approximately 6.5 mEq/L, the further decrease in the resting membrane potential causes a delay in the action potential, causing PR-segment prolongation, followed by QRS widening. And at potassium concentrations greater than 10 mEq/L, will ultimately results in ventricular fibrillation, pulseless electrical activity[7]. But in our two patients, a ventricular fibrillation was developed at a serum potassium of 7.0 mEq/L and 6.8 mEq/L, which we considered acidosis also played an important role. Ventricular fibrillation threshold is decreased by acute metabolic acidosis[8]. During LR, acidosis due to IPM is acute metabolic acidosis. In addition, Calcium ions (Ca^{2+}) play a major role in the cardiac excitation-contraction coupling, any imbalance in Ca^{2+} homeostasis of a cardiac myocyte can lead to electrical disturbances[9]. In our two patients, before cardiac arrest, Ca^{2+} maintain a relatively stable concentration level, but the concentration level is lower than physiological level. In the CPR and electrical defibrillation, with the treatment of lowering K+ and increasing Ca^{2+}, we finished a successful rescue.

Although there have been a report of short-term cardiotoxic blood potassium levels in the immediate aftermath of portal vein and hepatic artery occlusion[10], but IPM has been proved by many studies to be
effectively and safety in reducing bleeding during LR surgery[3, 11, 12].

To sum up, the rapid change of internal environment is an important cause of ventricular fibrillation and cardiac arrest in LR when IPM is used to control bleeding. Thus, in LR, the anesthesiologist needs to pay close attention to the ECG changes especially at the moment of vascular opening. In addition, how to ensure the stability of LR patients’ internal environment is an important clinical research work, which will provide evidence-based medical reference for anesthesiologists.

Declarations

Ethics approval and consent to participate:

Not applicable.

Consent for publication:

Informed written consent was obtained from the 2 patients after the nature of the study was fully explained to them. The 2 patients provided informed consent for publication of this cases report.

Availability of data and materials:

The datasets generated during the current study are not publicly available due to patients’ privacy and information security but are available from the corresponding author on reasonable request.

Competing interests:

The authors declare no competing interests.

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Authors’ contributions:

All the authors contributed to this manuscript. The manuscript was prepared and written by Yan Weng. Language corrections were made by Qing Zhong. Ziqi Shang and Yan Wang were the attending physician of the 2 patients and made the revision of the text. The supervision of prepared manuscript was made by Yan Weng. All authors have read and agreed to the published version of the manuscript.

References


**Figures**
Figure 1

Intraoperative $K^+$, $Ca^{2+}$ of patient A

Figure 1

Intraoperative $K^+$, $Ca^{2+}$ of patient A

3 $K^+$, PH, Lac during CPR of patient A

Before cardiac arrest | 13min after CPR | 23min after CPR | After successful of CPR
---|---|---|---
3 | 7.27 | 7.23 | 7.14
7 | 8.6 | 8.8 | 6.1
8.6 | 8.8 | 6.92 | 7.2
6.7 | 7.2 |
Figure 2

K$^+$ PH, Lac during CPR of patient A

![Graph showing Intraoperative K$^+$, Ca$^{2+}$ of patient B]

Figure 3

Intraoperative K$^+$ Ca$^{2+}$ of patient B
Figure 4

$K^+$, PH, Lac during CPR of patient B