

# Ultrasound-assisted diagnosis of intraoperative cardiac tamponade during hemihepatectomy: a case report

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#### **Abstract**

Cardiac tamponade is a rare complication that occurs during hemihepatectomy. This particular complication has a high degree of mortality and morbidity. A 51-year-old woman was admitted to our hospital for surgical treatment of a malignant liver tumor. During surgery, she developed sudden hemodynamic instability and signs suggesting cardiac tamponade, which was confirmed via transthoracic echocardiogram. Cardiac compression and creation of a pericardial window resulted in immediate hemodynamic improvement. At completion of surgery, a repeated transthoracic echocardiogram showed no pericardial effusion. Early ultrasound-assisted diagnosis and treatment of cardiac tamponade are crucial. Although cardiac tamponade rarely occurs during hemihepatectomy, medics should be aware of this possibility to ensure prompt diagnosis. Our findings strongly support the use of early cardiac compression in cardiac arrest during surgery with echocardiography for prompt and accurate diagnosis of cardiac tamponade. Additionally, our findings will hopefully make anesthesiologists aware of the need to maintain a high index of suspicion for cardiac tamponade with sudden hypotension and a large reduction in differential pressure, and encourage early use of echocardiography and timely cardiac compression.

# **Keywords**

Ultrasound, intraoperative cardiac tamponade, hemihepatectomy, echocardiography, cardiac compression, liver tumor

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# Introduction

Cardiac tamponade is a rare complication that can occur during hemihepatectomy. This complication has a high degree of mortality and morbidity. Only a few reports of intraoperative cardiac tamponade have been previously published. We report here the first case of intraoperative cardiac tamponade during hemihepatectomy, with prompt diagnosis and treatment with the assistance of echocardiography.

# Case report

A 51-year-old woman was admitted to our hospital for treatment of a liver lesion, which was found on a routine exam. The patient did not have any medical history, except for hypertension, and she did not take any medicine routinely. Vital signs, laboratory values, a physical exam, and cardiovascular evaluation were normal. Liver function tests, the platelet count, and

coagulation studies were within normal limits. A preoperative electrocardiogram and chest X-ray were unremarkable. Echocardiography showed a normal heart size with mild dilation of the aortic sinus, ascending aorta, and pulmonary artery. The estimated ejection fraction was 70%.

After induction of general anesthesia, the patient was ventilated with 100% oxygen at a tidal volume of 450 mL. Cannulation of the right internal jugular vein showed that the central venous pressure ranged from 5 to 7 mmHg. Exploration showed that the tumor was primarily located in Couinaud's segment IV and involved the post-hepatic inferior vena cava (IVC). The IVC was partially occluded with a clamp. Left hemihepatectomy was performed, including the junction between the anterior wall of the IVC and the middle and left hepatic veins. The right hepatic vein was preserved. Shortly after the IVC was opened, the patient's blood pressure and differential pressure rapidly decreased (Figure 1), while

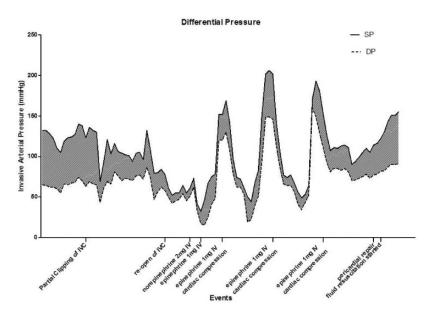


Figure 1. Graph of differential pressure. There was a large decrease in differential pressure during cardiac tamponade.

SP, systolic pressure; DP, diastolic pressure; IVC, inferior vena cava; IV, intravenously.

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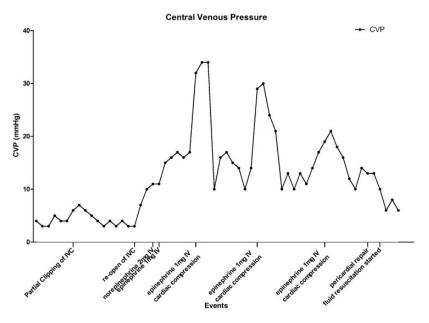
her central venous pressure markedly increased (Figure 2).

Continuous electrocardiographic monitoring showed sinus tachycardia. A total of 2 mg of norepinephrine was administered in separate doses. However, the patient's blood pressure did not respond to vasopressors continued decrease. Cardiopulmonary resuscitation was initiated with emergency cardiac compression and administration of 2 mg epinephrine for a total of three times. After hemodynamic improvement, a transthoracic echocardiogram showed an approximately normal ventricular size and motion (Figure 3). However, circulatory collapse recurred shortly thereafter. Echocardiography showed pericardial effusion (Figure 4). Incorrect suture placement between the pericardium and the IVC had caused cardiac tamponade, which was treated by creation of a pericardial window. The large hemopericardium was evacuated. After rechecking cardiac ultrasound (Figure 5), the patient was transferred to the intensive care unit, where she remained for 6 days.

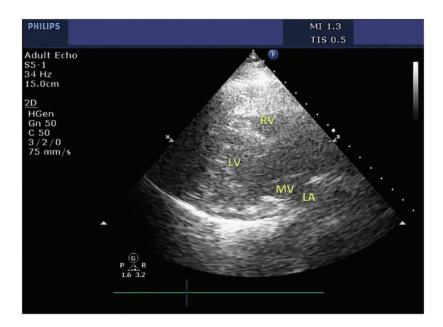
The patient continued to improve and was discharged home soon after leaving the intensive care unit. At a postoperative follow-up examination 1 month after discharge, a transthoracic echocardiogram showed good ventricular function and a stable hemodynamic status.

# **Discussion**

Cardiac tamponade is a medical emergency that results when sufficient fluid accumulates in the pericardial sac to compress the heart, resulting in decreased cardiac output and shock.<sup>3</sup> However, a recent study showed that iatrogenic complications were the most prevalent etiology for cardiac tamponade (36%).<sup>4</sup> Presentation may be rapid after chest wall trauma, aortic or cardiac rupture, or as a complication of cardiac procedures, necessitating prompt diagnosis and treatment.<sup>4</sup> Most reported cases of iatrogenic

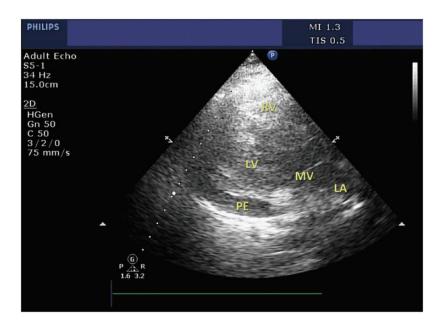


**Figure 2.** Graph of CVP. An abrupt increase in CVP occurred during cardiac tamponade. CVP, central venous pressure; IVC, inferior vena cava; IV, intravenously.



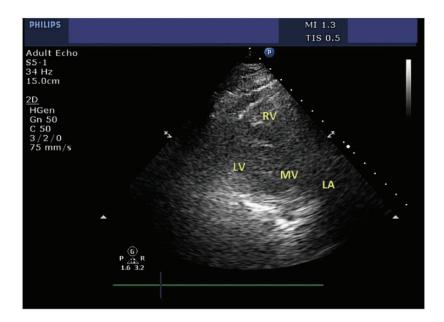
**Figure 3.** Transthoracic echocardiogram showing an approximately normal size and motion of the ventricle after the second cardiac compression. Mild pericardial effusion can be noted behind the inferior–lateral left ventricular wall.

RV, right ventricle; LV, left ventricle; MV, mitral valve; LA, left atrium.



**Figure 4.** Large hemorrhagic PE causing tamponade is seen on ultrasound with circulatory collapse. PE, pericardial effusion; RV, right ventricle; LV, left ventricle; LA, left atrium.

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**Figure 5.** Ultrasound shows that pericardial effusion has disappeared after creation of a pericardial window. RV, right ventricle; LV, left ventricle; MV, mitral valve; LA, left atrium.

cardiac tamponade resulted from interventional procedures (e.g., percutaneous coronary intervention, transcatheter aortic valve implantation, pacemaker/implantable cardioverter-defibrillator implantation, arrhythmia ablation, and endomyocardial biopsy). To the best of our knowledge, we report the first case of intraoperative cardiac tamponade during hemihepatectomy.

Cardiac tamponade in our case rapidly occurred Surgery in patients with hepatic tumors involving the intrapericardial IVC has an increased risk of cardiac tamponade because of the distorted anatomy and multiple severe tissue adhesions. Our patient's tumor was located near the proximal superior IVC and pericardium. Because of incomplete suture of the IVC after radical resection, the unsutured part slipped into the pericardium. This caused blood influx into the pericardial cavity.

The three classical signs of cardiac tamponade are known as Beck's triad as follows: low blood pressure resulting from decreased stroke volume, jugular vein distension resulting from impaired venous return, and muffled heart sounds resulting from fluid build-up inside the pericardium.<sup>3</sup> Other signs of tamponade include pulsus paradoxus (a drop of at least 10 mmHg in arterial blood pressure with inspiration) and ST segment changes on an electrocardiogram, which may also show low-voltage QRS complexes. General signs and symptoms of shock may also be present, such as tachycardia, shortness of breath, and decreased consciousness. The clinical diagnosis of cardiac tamponade is supported by the presence of breathlessness with clear lungs, tachycardia (>100 beats/minute), and pulsus paradoxus. However, these manifestations are difficult to recognize during general anesthesia. For surgical patients, the most common manifestations of cardiac tamponade are hypotension, tachycardia, a sudden increase in central venous pressure, and a rapid decrease in differential pressure.8

Echocardiography is essential for assisting the diagnosis of cardiac tamponade and can also guide treatment. Echocardiography performed by emergency physicians or anesthesiologists is reliable in diagnosing pericardial effusion and should be used routinely in critically ill patients with hemodynamic instability. Echocardiography can not only confirm the presence of pericardial effusion, but can determine its size and whether it compromises cardiac function (e.g., right ventricular diastolic collapse, right atrial systolic collapse, and a plethoric IVC).<sup>3,10</sup> The earliest echocardiographic indicator of hemodynamic compromise is diastolic compression of the right heart chambers. This compression appears on an echocardiogram as flattening of the normally anteriorly oriented curvature of the free wall or as curvature reversal as the compression becomes more severe. Collapse of the right atrium occurs in late diastole when this chamber is maximally emptied. As intrapericardial pressure increases further, the right heart chamber volume becomes markedly reduced. Ultimately, chamber volume in the left atrium and ventricle will also be reduced. In some cases, the compressible left atrium may show signs of collapse, but the left ventricle rarely does. The thicker and less compliant walls of the left ventricle show signs of preload reduction, but not free wall compression. The sudden accumulation of even a relatively modest volume of effusion may result in severe diastolic chamber compression and hemodynamic compromise. 11 IVC plethora with a blunted respiratory response is the most sensitive echocardiographic sign of cardiac tamponade.

Cardiac tamponade can lead to fatal hemodynamic collapse with hypotension and tachycardia. Other emergencies that may cause this drastic hemodynamic collapse, including acute myocardial infarction, pulmonary embolism, anaphylactic shock, and tension pneumothorax, should be ruled out without delay, as described below. The diagnosis of intraoperative acute myocardial infarction relies on an ST-T segment change and elevated serum myocardial enzymes. In our patient, an electrocardiogram and postoperative laboratory tests (normal troponin and creatine phosphokinase levels) ruled out acute myocardial ischemia. In patients with intraoperative pulmonary embolism, a oximeter usually stops detecting a signal at the fingertip, and end-tidal CO<sub>2</sub> rapidly decreases. Severe tachycardia (>120 beats/ minute), high central venous pressure, hypotension, and hypocapnia are usually observed during acute pulmonary embolism. Hypotension is usually difficult to correct with vasoactive drugs. Transesophageal and transthoracic echocardiography is important in diagnosing massive pulmonary thromboembolism. A close correlation has been demonstrated between the size of the right pulmonary artery and mean pulmonary artery pressure in patients with pulmonary thromboembolism who do not have prior cardiopulmonary disease. 12 Echocardiograms offer direct views of central thromboemboli. Echocardiograms also provide indirect evidence of pulmonary artery obstruction and right ventricular pressure overload, such as right ventricular dysfunction, tricuspid regurgitation, leftward bowing of the interatrial septum, and systolic flattening of the interventricular septum. 12 The early cutaneous signs of anaphylaxis are often unrecognized in anesthetized patients, meaning that bronchospasm and cardiovascular collapse are the first recognized signs. 13 Intraoperative anaphylaxis typically causes more than one of the following: rash, periorbital edema, throat or tongue swelling, high airway resishypotension, and tachycardia. tance. Epinephrine is the first-line medical therapy for anaphylaxis and is more effective than other vasopressors. The distance between the superior liver border and the diaphragm Wang and Chen 7

is only  $1.15\pm0.29\,\mathrm{cm}$ . Therefore, tension pneumothorax is possible. Progressive, otherwise unexplained hypoxemia and hemodynamic instability that are unresponsive to therapy and disproportionate to intraoperative blood loss are the main indicators of tension pneumothorax. Identification of the following four sonographic signs is required to diagnose pneumothorax: the absence of lung sliding, B-lines, and lung pulse, and the presence of lung point. <sup>14</sup>

Urgent drainage of pericardial effusion is usually the most effective management for iatrogenic cardiac tamponade, either by pericardiocentesis or surgical pericardiotomy. Autologous blood reinfusion from the pericardial space to a femoral vein can also be a life-saving procedure. <sup>16</sup>

# Conclusion

Findings in our case indicate the importance of early cardiac compression in intraoperative cardiac arrest and the usefulness of echocardiography in prompt and accurate diagnosis of cardiac tamponade. We hope that this case will raise awareness in anesthesiologists to maintain a high index of suspicion for cardiac tamponade when patients experience sudden hypotension and a large reduction in differential pressure.

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#### **Author contributions**

Jia-Wan Wang was the patient's anesthesiologist, reviewed the literature, and contributed to drafting the manuscript; Ying-Qi Chen was responsible for revision of the manuscript for important intellectual content; and all authors

issued final approval for the version to be submitted.

# **Declaration of conflicting interest**

The authors declare that there is no conflict of interest.

#### Ethics and informed consent

The study protocol was approved by the Ethics Committee of Beijing Chao-Yang Hospital. Informed written consent was obtained from the patient for publication of this report and accompanying images.

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