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Case Report

Intraoperative Transesophageal Echocardiography for latrogenic Cardiac Tamponade Treatment in a Patient Undergoing Liver Transplantation

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Abstract

The current case report describes a case of Intraoperative progressive hemodynamic failure in a patient undergoing orthotropic liver transplantation. Rescue transesophageal echocardiography was used to facilitate rapid diagnosis of a iatrogenic acute pericardial tamponade, resulting from a suture penetrating the pericardium through the right hemi diaphragm. This potentially lethal complication required an immediate echo-assisted pericardiocenthesis.

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Keywords

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- Cardiac tamponade
- Transesophageal echocardiography
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ABBREVIATIONS

OLT- Orthotopic Liver Transplantation, IVC - Inferior Vena Cava , ESLD- End-Stage Liver Disease, TEE- Transesophageal Echocardiography, TIPSS- Transjugular Intra hepatic Porto systemic Shunt, PAP- Pulmonary Arterial Pressure ,PAC-Pulmonary Artery Catheter ,MAP- Mean Arterial Pressure BP, Blood Pressure, VVB- Veno-Venous Bypass, TEG-Thromboelastography ,CI- Cardiac Index ,CVP-Central Venous Pressure, ICU- Intensive Care Unit, RA/LA -Right/Left Atrium, RV/LV- Right/Left Ventricle, SVRI -Systemic Vascular Resistance Index, Svo, -Mixed Venous Oxygen Saturation

INTRODUCTION

Orthotropic liver transplantation (OLT) is a challenging task for the anesthetist, due to the hemodynamic alterations occurring during the procedure (surgical manipulation, clamping of the hepatic hilum, clamping of the IVC and graft reperfusion). Moreover, the cardiovascular system of patients with end-stage liver disease (ESLD) presents several abnormalities, including a hyper dynamic circulation, with reduced mean arterial pressure (MAP) and systemic vascular resistance, associated with an increased cardiac output. These abnormalities, if present, seem to be associated with adverse outcomes during the postoperative period.

For these reasons, hemodynamic monitoring to guide volume therapy and administration of isotropic or vasopressin drugs is

essential during OLT. The most recent trends in hemodynamic monitoring are focusing on the importance of acquiring data continuously, and in the least invasive way. Transesophageal echocardiography (TEE) is able to provide immediate and continuous visualization and assessment of global cardiac function and is increasingly being used as an intraoperative cardiovascular monitor during OLT.

We report a case of unexplained intraoperative progressive hypotension and tachycardia in a patient undergoing OLT. Rescue TEE was used to facilitate rapid diagnosis and treatment. TEE showed a pericardial tamponade, a condition that requires prompt recognition and immediate pericardial decompression to prevent a fatal outcome.

CASE PRESENTATION

A 41-year-old man with chronic hepatitis B and D was scheduled to undergo OLT. His preoperative neurologic status was consistent with advanced portosystemic encephalopathy rapidly developed after recurrent episodes of variceal bleeding that required the positioning of a transjugular intrahepatic portosystemic shunt (TIPSS). A preoperative transthoracic echocardiogram showed concentric left ventricular (LV) hypertrophy, normal right and LV function and normal pulmonary arterial pressures (PAP). No evidence of pericardial effusion was observed.

Anesthesia was induced with intravenous propofol (2 mg/kg), fentanyl (3 mcg/kg) and vecuronium (0.1 mg/kg) and, after

tracheal intubation, general anesthesia was maintained with desflurane and additional fentanyl and vecuronium boluses.

Before starting the transplant procedure, an 8.0-french pulmonary artery catheter (PAC) (Swan-Ganz Continuous Cardiac Output - CCOmbo Volumetric catheter; Edwards Life sciences, Irvine, CA, USA) was introduced into the right internal jugular vein using an 8.5-french introducer (AVA 3Xi 8.5-Fr Edwards Life sciences, Irvine, CA, USA) and connected to a Vigilance monitor (Edwards Life sciences, Irvine, CA, USA) to assess hemodynamic parameters and heart function. The procedure had no complications and revealed the normal waveforms sequence. A 20-gauge catheter was placed in the radial artery and connected to a pressure transducer to assess MAP.

A femoro-jugular veno-venous bypass (VVB) was used during the transplant. The jugular and femoral veins were cannulated with echo-assisted technique, the positioning of the guide wires and cannulae was confirmed using TEE [1].

Coagulation management was tailored using thromboelastography.

During the preanhepatic phase the skeletonization of the liver was difficult and complicated by large blood losses, requiring multiple transfusions (PRBC 25 units, FFP 2300 mL, fibrinogen 3g). Moreover, purulent fluid was found in the median suprahepatic vein, were a TIPSS had been previously placed.

Hemodynamics were characterized by a hyperdynamic circulation; after the placement of a PAC parameters found were: CI 6.4 L/min/m 2 , SVRI 425 dyne s/cm 5 /m 2 , MAP 50 mmHg, HR 90 bpm, CVP 16 mmHg and SvO $_2$ 76%.

Continuous infusion of furosemide was started because the patient was oligo-anuric.

Progressive hypotension was observed during hepatectomy so that a continuous infusion of norepinephrine was started at a dose of 0.28 mcg/kg/min, SVRI subsequently raised to 600 dyne s/cm 5 /m 2 , MAP to 60 mmHg, HR decreased to 70 bpm and CI remained between 6 and 7 L/min/m 2 , CVP decreased to 10-12 mmHg.

After portal vein anastomosis and declamping, post reperfusion syndrome was observed, hemodynamics were sustained with increased doses of Norepinephrine (up to 0,43 mcg/kg/min) and boluses of Epinephrine. Hepatic graft reperfusion appeared normal, with no signs of graft ischemia and intraoperative production of bile; cold ischemia time was 7 hours and 5 minutes while warm ischemia time was 1 hour and 35 minutes.

During the biliary anastomosis construction, progressive hypotension (MAP 35 mmHg) and tachycardia (HR 120 bpm) of undetermined etiology were observed; hemodynamic parameters alterations consisted in a profound reduction in CI from 8.0 to 1.8 L/min/m², SVRI were 840 dyne s/cm $^5/m²$, CVP raised to 16 mmHg and SvO $_2$ fell to 36%, arterial pressure waveform showed exaggerated respiratory variations. Continuous electrocardiographic monitoring showed sinus tachycardia with frequent premature supraventricular complexes. Arterial blood gas tensions, serum electrolytes and hematocrit remained

stable during these events. Airway pressures were unaffected and breath sounds appeared equal bilaterally. Positive end-expiratory pressure was not used during the current case. Initial attempts of resuscitation, including volume expansion with crystalloids, decrease of end-tidal desflurane concentration and administration of vasoactive drugs were unsuccessful.

TEE was used to assess cardiac function, and the mid esophageal long axis view revealed a large pericardial effusion (Figure 1). A compression of the cardiac chambers, with right atrial and ventricular diastolic collapse were observed in the 4 chambers view, confirming the diagnosis of cardiac tamponade. Pericardiocentesis was urgently performed by the surgeon and the hemopericardium quickly evacuated.

These maneuvers resulted in a rapid increase in systolic arterial BP, from 70 to 120 mmHg, and a decrease in heart rate, from 110 to 85 beats/min, with normalization of PAP and central venous pressures (CVP). No other injuries were observed.

Examination of cardiac structures after decompression of the pericardial tamponade and restoration of stable hemo dynamics, showed a suture penetrating the pericardium through the right hemi diaphragm.

The patient remained hemo dynamically stable throughout the rest of the transplant procedure and was transferred to the postoperative intensive care unit (ICU).

The Liver graft recovered its functions, but the patient developed postoperative acute renal failure requiring dialysis. The length of ICU stay and of ordinary ward stay was respectively 15 and 40 days. Finally the patient's neurological status did not show any significant improvement if compared to his pre transplant condition, hence he was transferred to a rehabilitation facility.

DISCUSSION

The physiopathology of cardiac tamponade involves a number of factors. These include the pericardial pressure/ volume relationship, rapidity of fluid accumulation, underlying

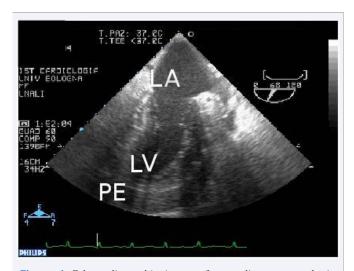


Figure 1 Echocardiographic image of a cardiac tamponade in midesophageal long axis view. LA left atrium, LV ventricle, PE pericardial effusion.



cardiac pathology (hypertrophy and shunts) and systemic volume status. It is well understood that small acute pericardial effusions, occurring most commonly post-operatively or traumatically, can lead to dramatic tamponade when fluid accumulates rapidly; whereas, moderate or even large pericardial effusions that accumulate slowly can be hemodynamically well tolerated [2,3]. Therefore, large pericardial effusions or the swinging motion of the heart are not always present in cardiac tamponade. On the contrary, a localized compressing effusion may lead to dramatic hemodynamic consequences without producing the classical echocardiographic signs of tamponade [4,5]. Iatrogenic cardiac injuries have been previously reported during liver transplantation [6] and during a number of procedures, including PAC insertion [7], laparoscopic Nissen fundoplication [8], transvenous pacing electrode insertion [9], sternotomy wire suture placement [10], defibrillator lead extraction [11], TIPSS insertion [12,13], subcutaneous implantation of a chemotherapeutic injection port [14], IVC filter implantation [15] and left lobe hepatectomy [16]. These injuries have also been reported during minor procedures including placement of central venous catheters [17,18] and chest tubes [19].

The vast majority of the previously described iatrogenic cardiac injuries during OLT, were accompanied by severe hemorrhage or life-threatening acute pericardial tamponade and the cardiovascular collapse appeared after abdominal closure in an apparently uncomplicated surgical procedure.

In our report, the patient developed acute pericardial tamponade during the biliary anastomosis phase.

The main differential diagnoses in this case were: pulmonary thromboembolism, air embolism and intracardiac thrombosis; considering only hemodynamic data, the etiological diagnosis would have been difficult because these conditions present with similar alterations. Moreover, hyperdynamic circulation related both to liver cirrhosis and probably to the SIRS related to the intravascular infection in the suprahepatic vein where the TIPSS had been positioned, and the contemporary use of vasoactive drugs, further complicated the interpretation of the hemodynamic parameters.

Prompt execution of TEE permitted immediate diagnosis and allowed for an immediate surgical approach.

latrogenic penetrating cardiac trauma may occur during hepatic surgery because the left lobe of the liver lies immediately adjacent to the diaphragmatic surface of the heart.

The resulting hemopericardium and subsequent tamponade may have occurred because the suture needle either perforated the right ventricular free wall or injured one of the pericardial vessels.

Multiple observations reported the usefulness of TEE during OLT to improve the monitoring of volume status and myocardial function and this technique is being increasingly used in transplant centers, a recent survey by Soong et al demonstrated that in 94.9% of the US transplantation centres, the usage of TEE is common practice [20,21]. Intraoperative TEE may provide additional critical information, such as identification of intracardiac thrombi [22] or complications related to TIPSS

[23]. Recently a report presented a case of postreperfusion graft congestion, TEE revealed hemodynamically significant thrombotic stenosis of the IVC [24].

The present report emphasizes the utility of TEE as a diagnostic tool in the setting of a life-threatening hemodynamic instability of unknown origin. The use of TEE, in these conditions, is well recognized as a category I indication (strong scientific evidence that TEE favorably affects clinical outcome) established by the joint American Society of Anesthesiologists - Society of Cardiovascular Anesthesiologists task force on TEE [25].

The most characteristic echocardiographic findings that suggest cardiac tamponade are the visualization of fluid filling the pericardial sac, diastolic collapse of the free walls of the right atrium (RA), and/or the RV. Any cardiac chamber can be collapsed, but this usually occurs on the right-sided chambers because both the RA and RV are low pressure chambers and more compliant. RA compression for more than one third of the cardiac cycle (late diastole) is a highly sensitive and specific sign of cardiac tamponade. RV compression in the early diastole is less sensitive for the presence of cardiac tamponade than RA diastolic collapse, but is very specific for cardiac tamponade. RV collapse may not occur when the RV is hypertrophied or when its diastolic pressure is greatly elevated. Left atrial (LA) collapse can be seen in about 25% of the patients with hemodynamic compromise and is very specific for cardiac tamponade. Left ventricular (LV) collapse is less common, since the wall of the LV is more muscular, but can be seen under special conditions, such as localized postsurgical tamponade or severe pulmonary hypertension [26,27]. Noteworthy, TEE is a relatively safe method for monitoring cardiac performance with a low incidence of major hemorrhagic complications in patients with documented esophagogastric varices undergoing OLT [28].

The routine use of TEE to improve the OLT outcomes is a debatable topic, however it has been proven that in cirrhotic patients with MELD scores < 15, the precision of the transesophageal echo-Doppler device was similar to PAC, therefore, in this subset of patients, it may represent a reliable alternative to PAC [29].

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J Liver Clin Res 1(1): 1005 (2014)



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