

CASE REPORT

Intracardiac thromboembolism during liver transplantation[☆]



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Abstract We describe a case of intraoperative cardiac thrombosis during orthotopic liver transplant surgery that resulted in intraoperative death. By using transoesophageal echocardiography, the cause of the decompensation of the patient could be determined and the mechanism of thrombus migration from thrombi from the venous circulation to the left heart was accurately observed.

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PALABRAS CLAVE

Ecocardiografía
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Trombosis;
Trasplante hepático

Tromboembolismo intracardíaco durante trasplante hepático

Resumen Describimos un caso de trombosis cardíaca intraoperatoria durante una cirugía de trasplante ortotópico hepático que derivó en muerte intraoperatoria. Mediante ecocardiografía transesofágica, colocada durante la descompensación del paciente, se pudo determinar la causa del problema y observar con precisión el mecanismo de migración de trombos desde la circulación venosa hacia el corazón izquierdo.

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Introduction

Cardiac embolism and intracardiac thrombi (ICT) are complications of liver transplantation (LT). They may occur during reperfusion of the organ, and are associated with a high rate of morbidity and mortality.

There is currently no standard treatment for ICT, but the diagnostic technique of choice is transoesophageal echocardiography (TOE) monitoring. TOE enables the anaesthesiologist to rule out differential diagnoses, including hypovolaemia and myocardial ischaemia, during the acute phases of transplantation, and it should be used all liver transplant patients.

Case report

We present the case of a 43-year-old man with terminal liver disease measured on the model for end-stage liver disease (MELD) scale, cirrhosis due to haemochromatosis, and portal vein thrombosis diagnosed preoperatively by 3D computed tomography, together with grade 1 portal vein thrombosis (less than 50% obstruction, according to the Yerdel and McMaster classification).¹

A few weeks prior to transplantation, the patient was hospitalised for episodes of abdominal infection and acute kidney failure secondary to hepatorenal syndrome, without the need for haemodialysis. He also presented grade 1 hepatic encephalopathy and hepatopulmonary syndrome with right pleural effusion, which was drained the day before surgery.

Although analytical tests had shown no significant metabolic alterations, during surgery the patient presented acute haemodynamic decompensation after declamping, with severe hypotension and extreme bradycardia. Advanced cardiopulmonary resuscitation manoeuvres were started, and adrenaline followed by isoproterenol were administered, after which nodal rhythm was recovered. However, due to the persistence of bradycardia, a transient percutaneous pacemaker was placed through a Swan-Ganz introducer. Given the acute and unexpected nature of the event, the anaesthesiologist requested placement of a diagnostic TOE probe, which showed massive intracardiac thrombosis. Annex, video 1, shows a thrombus from the inferior vena cava located in the right atrium, together with a pacemaker cable.

Fig. 1, and Annex, video 2 show a thrombus passing through the foramen ovale of the interatrial septum and thrombosis in the left atrium.

Annex, video 3, shows a thrombus at the level of the mitral valve ready to migrate to the left ventricle.

Despite our resuscitation attempts, the patient deteriorated rapidly and died. TOE monitoring was fundamental in the diagnosis and management of this severe complication.

Discussion

Reperfusion is one of the most critical moments during LT surgery, and is a risk factor for mortality.² The triad of reperfusion hypotension secondary to the release of blood from the hypothermic organ, which causes acidosis and hyperkalaemia induced by the preservation solution, end-stage

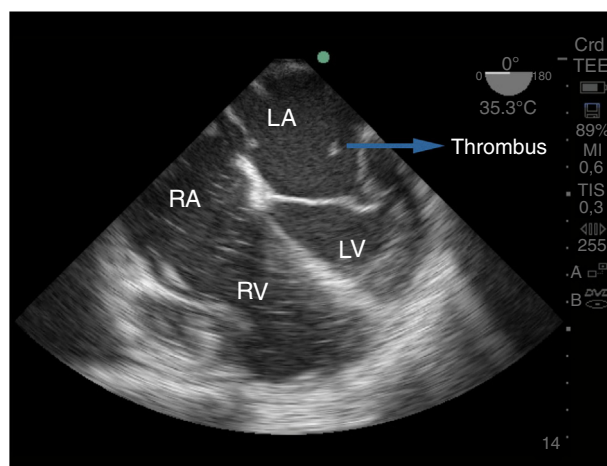


Figure 1 Four chamber view showing a thrombus in the left atrium and another one transiting from the right atrium through the patent foramen ovale. Note the enlargement of the right-sided cavities. RA: right atrium; LA: left atrium; RV: right ventricle; LV: left ventricle.

liver disease-induced coagulation disorders, and the presence of a pulmonary artery catheter as a source of clot formation,³ favour the formation of ICT at the time of reperfusion.

The cause of ICT in our patient may have been multifactorial. He was hospitalised for infectious symptoms weeks before the transplant, and multiple blood products were administered prior to organ reperfusion. He presented partial portal thrombosis, which was resolved with simple surgical thrombectomy. This procedure has been associated with a higher risk of bleeding, which in turn increases the complexity of the transplant and the risk of morbidity and mortality. Another cause could be rethrombosis. Although the cause of this is unclear, some have suggested it could be due to intimal damage caused during thrombectomy.

Coagulation in patients with end-stage liver disease (ELD) is extremely deficient due alterations in the delicate balance between procoagulants, anticoagulants and fibrinolytic proteins in the diseased liver. Based on this, we believe that it would have been very useful to use TOE monitoring from the beginning of surgery for early detection of thrombosis.

During LT surgery, the pulmonary artery is catheterised in order to monitor cardiac output using volumetric measuring devices (PiCCO/EV-1000). The appearance of ICT is heralded by sudden haemodynamic changes, hypoxia, increased central venous pressure, and an increase or decrease in pulmonary and arterial pressures; however, the most accurate, real-time diagnosis of ICT is achieved with TOE.

ICTs, though considered rare or possibly "underdiagnosed," are a life-threatening complication. TOE is not routinely used in Latin America in transplant surgery, although it is currently considered an essential monitoring tool, with a sensitivity of 80% and a specificity of 100% for the diagnosis of thrombi.⁴

The echocardiographic images obtained show the mechanism by which the thrombus reaches the left side of the heart. The normal pressure of the left atrium is 12–15 mmHg, and that of the right atrium ranges from 0 to 5 mmHg. When the pressure of the right atrium increases due

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