Refractory ascites is an uncommon complication that may develop postoperatively after liver transplantation. The diagnosis and treatment of this condition is a real challenge. We report two cases of patients who underwent a transplant due to cryptogenic cirrhosis and developed refractory ascites during the immediate postoperative period. This is a serious complication associated with decreased survival by up to one year and a reduced quality of life. After ruling out the main causes of ascites, a portal hyperflow was a potential etiology. This condition perpetuates itself with splenic circulation and brings about a reduction in the hepatic arterial flow. Therefore, if arterial blood flow to the spleen is diminished, venous return and portal circulation will be reduced and arterial blood flow will improve. Splenic artery embolization is a procedure introduced many years ago for the management of splenic artery steal syndrome and small-for-size living donor liver transplantation. This procedure is performed in order to reduce portal hyperflow and consequently, ascites. In conclusion, splenic artery embolization is a therapeutic option for the treatment of refractory ascites after liver transplantation.

Key words: Spleen embolization. Refractory ascites. Liver transplantation.

INTRODUCTION

Refractory ascites are a rare, uncommon complication that develops during the postoperative period after liver transplantation and are unrelated to the surgical technique (1). The causes may be systemic (not associated with portal hypertension) or related to portal hypertension (prehepatic, hepatic and posthepatic) (2). Among the systemic causes, bacterial or fungal peritonitis (70%) and renal failure (6%) are the most significant. Furthermore, portal vein obstruction, suprahepatic vein torsion and veno-occlusive disease are the predominant causes related with portal hypertension (3). Prehepatic causes include portal hyperflow, which represents a diagnosis of exclusion. Risk factors have also been identified that predispose to this phenomenon, including vessel size, microvascular changes during a rejection event, mismatch between donor and recipient liver size and postoperative complications such as chylous ascites, thrombosis and infection. Treatment options are based on diuretics, evacuating paracentesis, peritoneovenous shunts and retransplantation in extreme cases.

We report two cases of patients who were transplanted due to cirrhosis secondary to idiopathic portal hypertension (PHT) and presented with refractory ascites during the immediate postoperative period.

CASE REPORT 1

A 70-year-old male patient with a history of HBP was diagnosed with idiopathic portal hypertension of a prehepatic origin. This condition presented seven years previously as an upper GI bleed (UGIB) secondary to esophageal varices. The patient progressed to end-stage liver disease and underwent a liver transplant (LT) from a brain-dead living donor. During surgery, a large splenomegaly that reached the midline with significant collateral circulation was found. The patient was discharged following a favorable postoperative period only to be readmitted one month later due to refractory ascites that did not respond to ascites taps and intensive diuretic therapy. Renal causes were excluded (normal renal function), as were causes with an infectious (sterile fluid on culture) and cardiac (no constrictive pericarditis on heart catheterization) origin. The patient had a normal liver function and expected levels of immunosuppression; a transjugular biopsy was performed which was nonspecific. Hepatic pressures were also measured and a 13-mmHg gradient was found. An immunological and
A hematological study ruled out thrombophilia and changes in the bone marrow. Having excluded all important causes of refractory ascites, a portal hyperflow secondary to hypersplenism that manifested as giant splenomegaly (according to Poulin's classification) was thought to be the likely origin. Hence the patient underwent a complete splenic artery embolization via a femoral access route (Fig. 1). A follow-up computed tomography (CT) scan one week after the procedure identified necrosis that involved over 90% of the spleen. The patient gradually improved with paracentesis and diuretic therapy which was rendered unnecessary 40 days after embolization. No embolization-related complications developed and the patient remains ascites-free at six months after the LT.

**CASE REPORT 2**

A 65-year-old woman with a history of hypertension (HBP) received a diagnosis of liver disease which presented with UGIB from esophageal varices at age 37. Ten years later, she underwent a Warren splenorenal shunt that improved the ascites symptoms but worsened the hepatic encephalopathy events. Thirty years after the initial onset of symptoms, the patient eventually underwent a liver transplant with optimal characteristics from a brain-dead living donor, accompanied by shunt ligation to improve blood flow through the portal vein. The patient had a torpid postoperative course with seizures secondary to tacrolimus treatment, which was changed to cyclosporine with renal function impairment that required hemofiltration. One month after transplantation, the patient had progressively developed refractory ascites and hydrothorax, which required intensive diuretic therapy and daily ascites taps. A liver biopsy revealed grade-2 acute rejection, which was managed with corticosteroids and increased immunosuppression without improvement of the ascites. After multiple readmissions due to hydrothorax and ascites events, imaging tests revealed a splenomegaly that was not present at the time of surgery, as well as an increased splenic blood flow on the Doppler scan (Fig. 2). A splenic artery embolization was performed via the brachial artery. After three months, the patient remains asymptomatic with no further ascites episodes.

Liver transplantation was performed in both patients with vena cava preservation and a continuous suture was used for the suprahepatic vein-to-vena cava and portoportal anastomoses.

**DISCUSSION**

Refractory ascites is an uncommon, potentially serious complication that may develop postoperatively after liver transplantation and is associated with a survival reduction of up to one year. The International Ascites Club defines this condition as ascites that persists beyond four weeks after an uneventful liver transplant (4).

One of the pathophysiological mechanisms underlying this condition is portal hyperflow or hyperperfusion (5). This condition manifests clinically as ascites or hydrothorax and is refractory to treatment with diuretics and ascites taps. Portal hyperflow leads to decreased hepatic arterial flow via the so-called hepatic arterial buffer response. This is an intrinsic self-regulation mechanism that releases adenosine, a strong arterial vasodilator, to diminish arterial blood flow. According to this theory, adenosine accumulation in Mall's space would result in hepatic vasodilation in the presence of a decreased portal flow. Conversely, when portal flow increases, adenosine levels decrease, leading to arterial vasoconstriction (6). The exact place where adenosine is produced remains unknown. However, as long as this buffer system works appropriately, damage in the form of enzymatic impairment will not likely develop within the first few months after transplantation. However, continued portal hyperflow may trigger phenomena such as refractory

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**Fig. 1.** Case report 1. A. Arteriogram before embolization. B. Splenic artery embolization with 500-700-micron particles and 9-mm coils. C. Pre-embolization CT scan. D. Post-embolization CT scan.

**Fig. 2.** Case report 2. A. Arteriogram obtained via the brachial artery. B. Splenic artery embolization with 14-16-mm Amplatzer devices. C. Post-embolization CT scan.
ascites. Hyperflow feeds on splanchnic collaterals, mainly favored by splanchnic circulation. Therefore, if arterial flow to the spleen is decreased, venous return and portal circulation diminish. Thus, decreasing portal hyperflow and increasing adenosine release, which results in hepatic artery vasodilation and improved arterial flow (2). In this way, the problem is solved by targeting one of the links in the chain, i.e., by reducing splenic circulation with embolization.

Most post-embolization complications are usually minor and may be managed conservatively. The most common case is post-embolization syndrome, which manifests with abdominal pain.

Volume differences between the spleen and liver is a useful predictor of response after embolization; the procedure is more effective when the ratio is higher than 0.5. Some authors correlate embolization success with the infarction area after treatment in a directly proportional manner (7,8). Furthermore, the larger the infarction area, the greater the platelet count improvement. In addition, after partial embolization, residual splenic tissue may undergo hypertrophy in the long term, which requires a repeat procedure. Table 1 lists the refractory ascites cases reported in the literature that were treated with splenic embolization; our patients are amongst the few who received complete embolization.

In conclusion, splenic artery embolization for the management of refractory ascites after liver transplantation is a viable option when the usual causes have been excluded.

### REFERENCES


### Table 1. Characteristics of reported refractory ascites cases managed with splenic artery embolization

<table>
<thead>
<tr>
<th>Authors (site)</th>
<th>n</th>
<th>Mean age (years)</th>
<th>MELD</th>
<th>Embolization after transplantation (days)</th>
<th>Time to ascites resolution after embolization (months)</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quintini et al. 2011 (1) (Cleveland Clinic)</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>78 ± 40</td>
<td>-</td>
<td>No</td>
</tr>
<tr>
<td>Kim et al. 2012 (9) (Seoul National University)</td>
<td>5</td>
<td>40</td>
<td>-</td>
<td>6-28</td>
<td>Abdominal pain (4), fever (1)</td>
<td></td>
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<tr>
<td>Presser et al. 2015 (5) (Cleveland Clinic)</td>
<td>12</td>
<td>-</td>
<td>22</td>
<td>89 ± 12</td>
<td>1-9</td>
<td>No</td>
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<tr>
<td>Meighani et al. 2016 (10) (Detroit Hospital)</td>
<td>1</td>
<td>49</td>
<td>20</td>
<td>120</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Pravisani et al. 2016 (2) (University Hospital of Udine)</td>
<td>23</td>
<td>59</td>
<td>15</td>
<td>110 ± 61</td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>Nutu OA et al. 2017 (Hospital 12 de Octubre)</td>
<td>2</td>
<td>67</td>
<td>12</td>
<td>90</td>
<td>2</td>
<td>No</td>
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</table>