Graft Portal Vein Thrombosis Before Liver Transplant

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Surgical injuries during organ procurement and vascular thrombosis may deteriorate liver graft quality and post–liver transplant (LT) outcome,[1] regardless of donor characteristics. Graft hepatic artery thrombosis before LT has been described. Conversely, there are no reports on graft portal vein thrombosis (gPVT) recognized during organ procurement. The aim of this letter is to describe 2 cases of gPVT recognized and treated within donor procedures.

In the first case, a 75-year-old woman died of an embolic stroke (brain death donor [DBD]) during arteriovenous extracorporeal membrane oxygenation after cardiocirculatory arrest. Once donor hepatectomy was completed, gPVT became evident and thrombectomy was performed. The histopathologic analyses showed 50% macrovesicular and 40% microvesicular steatosis, focal microthrombosis, and multifocal sinusoidal dilatation. Based on the presence of steatosis, portal vein thrombosis (PVT), and sinusoidal damage, the graft was discarded.

In the second case, a 62-year-old man DBD, who died for subarachnoid hemorrhage, was allocated to our Center as rescue allocation due to 40% macrovesicular steatosis. The procurement was performed by a separate transplant center team using in situ aortic cold perfusion and portal vein perfusion after hepatectomy. No abnormalities, except for steatosis and vasoactive drug infusion, were reported. The unexpected allocation (eg, urgent allocation) imposed to use a back-base dual hypothermic oxygenated machine perfusion (DHOPE) to extend cold ischemia time and recondition a steatotic graft (Table 1). Upon the beginning, suboptimal portal flow and resistances indicated poor graft viability (Figure 1). These hemodynamic data imposed an additional evaluation. The graft was then handled for a macroscopic inspection, and a gPVT was identified. A Doppler ultrasound (US) confirmed this diagnosis. We disconnected the graft and performed a thrombectomy (Figure 1). To evaluate the efficacy of thrombectomy and the possible presence of an intrahepatic PVT, we reconnected the graft to DHOPE. Portal and hepatic artery flow/resistances immediately reached normal values. After 345 minutes of DHOPE, the graft was successfully transplanted. Total cold ischemia time was 710 minutes. No liver (eg, early allograft dysfunction) or kidney complications occurred. Post-LT computed tomography and US showed no residual intraparenchymal PVT.

Posttransplant vascular complications are the most common causes of graft failure after LT. Among them, post-LT PVT was reported in 5% of the patients.[2] The most documented risk factors for post-LT PVT are related to primary liver disease and pre-LT medical history, while donor influence has not been exhaustively

TABLE 1.

Main characteristics of procurement and DHOPE

Procurement and DHOPE timing			
Duration procurement, min		210	
Procurement WIT, min[a]		47	
SCS before DHOPE, min		335	
Duration DHOPE, min		345	
Total cold ischemia time, min		710	
DHOPE parameters	Start	Pre-TR	Post-TR
PV flow, mL/min	80	130	250
PV resistance, Ω	0.05	0.03	0.01
HA flow, mL/min	35	67	75
HA resistance, Ω	0.79	0.53	0.43
AST, IU/L	146	186	193
ALT, IU/L	165	216	227
LDH, IU/L	304	372	394
Lactate, mmol/L	1.4	4.6	5.6
Potassium, meq/L	29	32	33
Glucose, mg/dL	187	246	254

[a]From aortic cross-clamp to the end of donor hepatectomy.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; DHOPE, dual hypothermic oxygenated machine perfusion; HA, hepatic artery; LDH, lactates dehydrogenase; pre-TR, pre-thrombectomy; post-TR, post-thrombectomy; PV, portal vein; SCS, static cold storage; WIT, warm ischemia time.

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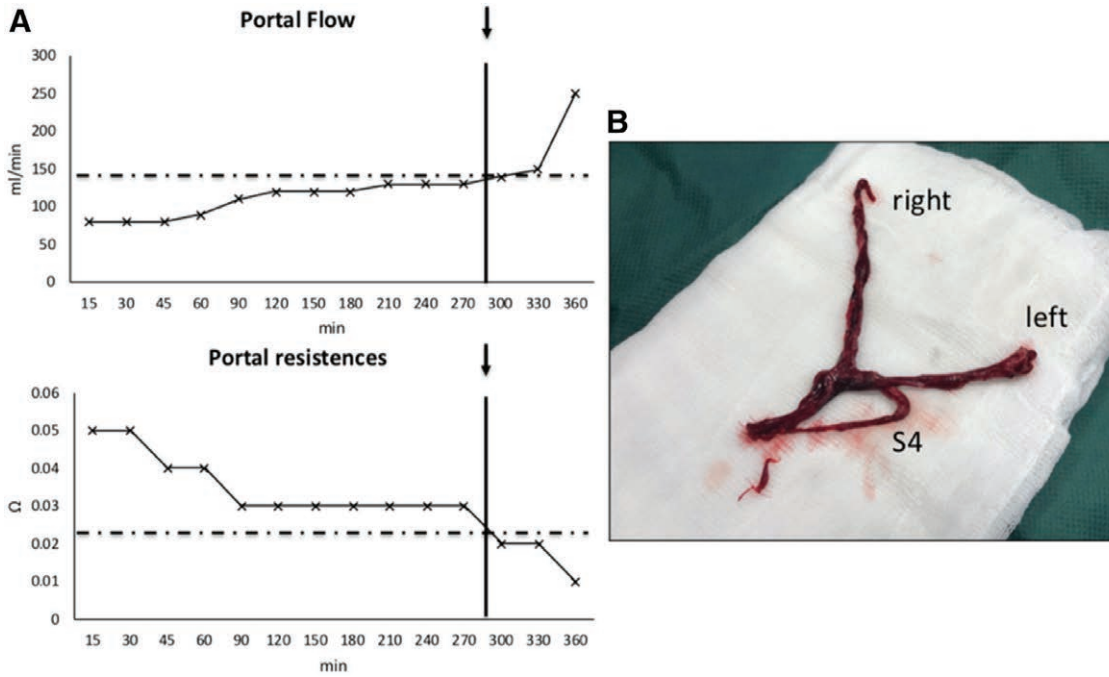

FIGURE 1. Main findings during DHOPE applied to the second case: (A) portal and arterial flows during dual hypothermic oxygenated machine perfusion (DHOPE) procedure. Dotted line: upper and lower limit of normal values of portal flow and resistances, respectively. Arrow: time of thrombectomy. B, Picture of portal vein thrombus after thrombectomy performed during DHOPE.

investigated. gPVT development could depend on the widely debated relation between ischemia-reperfusion injury and endothelial activation, both worsened by steatosis. This association results in the deposition of fibrin microthrombi and cellular debris in sinusoidal lumens that could lead to thrombus development.[3] DHOPE was recently investigated and found to have a potential role in steatotic graft reconditioning.[4] In our case, functional monitoring during DHOPE allowed evaluation, diagnosis, and treatment of the thrombosis-related graft complications. Indeed, portal and arterial flows were markedly reduced relative to the other cases in our series of DBD DHOPE grafts.[5] This anomaly imposed additional US evaluation that eventually allowed thrombus identification. Consistently, normalization of perfusion parameters enabled to confirm thrombectomy success. While intraoperative or post-LT US has a low detection rate of portal microcirculation dysfunctions,[6] DHOPE could be a valuable evaluation tool. In our opinion, application of this approach to the first case might have led to graft evaluation instead of abrupt discard.

In conclusion, we provide the first report of gPVT recognized and treated within donor and ex situ perfusion procedures. In this context, DHOPE not only represents an ideal platform for steatotic graft preservation and reconditioning, but it is also a valuable tool for graft evaluation.

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