Hepatic artery thrombosis (HAT) following orthotopic liver transplantation (OLT) can be a devastating complication that places the patient at risk of sepsis, biliary problems and graft loss. In the early years of liver transplantation, retransplantation was considered the best treatment.1 The need for retransplantation puts additional stress on a limited resource.

In this article, Professor S.T. Fan and his group report an 11-year experience with HAT in 248 liver transplants, 140 of them from live donors (LDLTs). There were seven episodes of HAT in six patients. Two of the episodes occurred early (2 and 9 days postoperatively) and required reoperation for thrombectomy or retransplantation; the remaining four occurred later (21–105 days) and were treated with management of septic and biliary complications. This treatment was successful in three of the patients, while the fourth required retransplantation. The authors did not comment on the technical details of the original transplant procedure. In our experience, HAT complications occur in patients with problematic arterial reconstruction due to inadequate recipient inflow. A retrospective review of 680 adult liver cadaveric OLTs identified symptomatic HAT in 11 patients (1.6%).2 Of these, nine (82%) had inadequate recipient inflow requiring interposition of an allogeneic iliac graft in seven or anastomosis of an aberrant right hepatic artery in two.

Professor Fan’s experience with LDLT is noteworthy. LDLTs are technically demanding, as the arterial reconstruction more often involves small-calibre vessels, and the extensive procurement operation can lead to inadvertent intimal dissection of the hepatic arteries. The authors used a microvascular technique in LDLTs without anticoagulation. The low incidence of delayed HAT in these patients is a remarkable accomplishment that reflects both impressive technical ability and high-quality perioperative care.

Of note, delayed HAT was seen in two of eight (25%) LDLT patients who had undergone preoperative chemoembolization, compared with only one of 105 (1%) who had not. This difference was not seen in the cadaveric group. LDLT is notably different from cadaveric whole-organ transplantation in the relative amount of blood flow per gram of liver tissue. Preoperative chemoembolization probably causes either subclinical damage to the endothelium or increased vascular resistance in the graft that may then result in a higher risk of thrombosis, particularly in the face of increased relative blood flow. In this report, patients who developed collateralization fared better than those who did not. Interestingly, LDLT patients who develop delayed HAT all seem to also have developed arterial collateralization, which would perhaps suggest that LDLT grafting may stimulate neovascularization as part of the regenerative process.

The formation of collateral blood supply, which allows graft salvage with conservative treatment, is a process that continues to be both a mystery and an area of active research. One might speculate that delayed HAT develops in a gradual fashion, leading to increased expression of vascular endothelial growth factors. Were this the case, and with extensive current investigation and understanding of ischaemia-induced neovascularization, we may someday see the use of bioengineered endothelial growth factors or adjuvants in the prevention and treatment of HAT.3

References

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