Risk for arterial thrombosis after liver transplantation with hepatic artery reconstruction

Mohamed Ghazaly1,2, Pulkit Sethi1, Manikandan Kathirvel1, Navneet A. Tiwari1, Manoj Thillai1, Rohit Gaurav1, Veena Surendrakumar1, John O. O. Ayorinde1, Michael Allison1, Sara Upponi2, Christopher J. Watson1-6, Raj K. Praseedom1, Paul Gibbs1 and Kourosh Saeb-Parsy1,5,*

1Transplant Unit, Addenbrooke’s Hospital, Cambridge, UK
2Department of Surgery, Tanta University, Tanta, Gharbia, Egypt
3Department of Hepatology, Cambridge Biomedical Research Centre, Addenbrooke’s Hospital, Cambridge, UK
4Department of Radiology, Addenbrooke’s Hospital, Cambridge, UK
5Department of Surgery, University of Cambridge, and Cambridge NIHR Biomedical Centre, Cambridge, UK
6Correspondence to: Kourosh Saeb-Parsy, Department of Surgery, Addenbrooke’s Hospital, Hills Road, Box 202, Cambridge CB2 0QQ, UK (e-mail: ks10014@cam.ac.uk)

The paper was presented at the ILTS (International Liver Transplantation Society) 25th Annual International Congress in Toronto, Canada, 15–18 May 2019.

Dear Editor

Knowledge of anatomical variations is important during the procurement of deceased donor organs to avoid organ damage and to promote complex vascular reconstruction. Reconstruction of the hepatic artery is hindered by increased number of anatomical variants of the donor hepatic artery that could exist in up to 50 per cent of liver grafts. The liver’s arterial supply is very complex and each one of the eight segmental arteries can possibly derive separately from the aorta. Vascular complications following liver transplantation exacerbate postoperative morbidity. The aim of this study was to assess the impact of hepatic artery reconstruction (HAR) on hepatic artery thrombosis (HAT) after liver transplantation (LT) and subsequent recipient morbidity and mortality rates.

Some 244 LTs were performed at Addenbrooke’s Hospital, Cambridge, UK between 2014 and 2017 with a median follow-up of 30 months (range 12–48 months). HAT occurring following LT was ascertained with CT. Donor and recipient variables were outlined, and outcomes were compared between recipients with and without HAR (Supplementary material, Appendix S1).

In the case of donor aberrant arterial anatomy of the graft (Fig. S1), back-bench reconstruction was carried out (Fig. S2). All patients were discharged on aspirin 75 mg once daily orally long-term. Only patients with vascular reconstruction who were considered at higher risk of graft vessel thrombosis received long-term formal anticoagulation (initially consisting of therapeutic dose dalteparin, followed by oral anticoagulation).

Liver transplants with and without HAR were largely comparable in terms of donor and recipient characteristics (Table S1). Within the surgical/operative parameters, operative time, arterial anatomy, and Roux loop biliary reconstruction were all more frequent in the HAR group (P = 0.007, 0.001, and 0.008 respectively) (Table S2).

Table 1 Univariable and multivariable analysis of risk factors for HAT

| Risk factors                        | Odds ratio | P       |
|-------------------------------------|------------|---------|
| **Univariable analysis**            |            |         |
| HAR                                 | 3.88 (1.51, 10.00) | 0.001  |
| Recipient age                       | 0.69 (0.30, 9.63)  | 0.351  |
| Donor type                          | 1.18 (0.46, 3.00)  | 0.681  |
| Donor age                           | 0.85 (0.30, 4.33)  | 0.448  |
| Donor BMI                           | 2.41 (0.39, 7.66)  | 0.299  |
| HCC                                 | 0.84 (0.23, 3.00)  | 0.539  |
| Graft steatosis                     | 0.63 (0.37, 4.53)  | 0.497  |
| Cold ischaemia time                 | 1.39 (0.84, 3.62)  | 0.195  |
| Warm ischaemia time                 | 0.85 (0.27, 2.50)  | 0.237  |
| Operative time                      | 2.62 (1.60, 5.31)  | 0.042  |
| Abnormal arterial anatomy           | 2.97 (1.18, 7.51)  | 0.024  |
| Aortic conduit                      | 3.03 (0.91, 10.08) | 0.106  |
| Type of biliary reconstruction      | 0.79 (0.29, 2.13)  | 0.367  |
| PV conduit                          | 1.25 (0.15, 10.41) | 0.521  |
| Bile leak                           | 0.78 (0.20, 6.27)  | 0.415  |
| Isolated biliary stricture          | 1.90 (0.51, 7.05)  | 0.317  |
| Ischaemic cholangiopathy            | 4.86 (1.52, 15.61) | 0.002  |
| Re-exploration                      | 1.05 (0.34, 3.31)  | 0.729  |
| **Multivariable analysis**          |            |         |
| HAR                                 | 2.31 (1.16, 4.33)  | 0.015  |
| Operative time                      | 1.50 (0.06, 4.22)  | 0.223  |
| Abnormal arterial anatomy           | 1.68 (0.86, 3.26)  | 0.094  |
| Ischaemic cholangiopathy            | 2.64 (1.99, 6.93)  | 0.021  |

Values in parentheses are 95 per cent confidence intervals. HAR, hepatic artery reconstruction; HCC, hepatocellular carcinoma; PV, portal vein.
5.6 per cent; \( P = 0.0076 \), although HAR did not increase the incidence of morbidity or death (Fig. S3). Nine patients in the HAR group had HAT, and all six early HATs in the HAR group needed retransplantation with no 30-day deaths.

Univariable regression analysis showed HAR (\( P = 0.001 \)), abnormal arterial anatomy (\( P = 0.024 \)), operative time (\( P = 0.042 \)) and ischaemic cholangiopathy (\( P = 0.002 \)) as risk factors for HAT. Multivariable analysis revealed HAR (odds ratio 2.31 (95 per cent c.i. 1.16 to 4.33), \( P = 0.015 \)) and ischaemic cholangiopathy (odds ratio 2.64 (95 per cent c.i. 1.99 to 6.93), \( P = 0.021 \)) as risk factors for HAT (Table 1). There was no significant difference in patient survival (Fig. S3).

This study has some limitations including small size and probable confounding factors. Moreover, the authors did not analyse arterial resistive index on Doppler ultrasound. The need for bigger multicentre prospective studies is emphasized by these constraints. Nonetheless, the findings indicate that, although a higher risk of HAT is strongly related to bench arterial reconstruction for an aberrant donor arterial anatomy, patient survival can be comparable to that of the control group.

Acknowledgements

P.S., M.K., N.A.T., M.T., R.G., V.S. collected data. M.A., S.U., C.J.W., P.G., R.K.P. participated in study design. J.O.O. analysed data. M.G. collected data and wrote the manuscript. All co-authors reviewed and approved the manuscript. K.S.P. designed the study, reviewed data, critically revised the manuscript and finally approved the manuscript.

Disclosure. The authors declare no conflicts of interest.

Supplementary material

Supplementary material is available at BJS Open online.

References

1. Ozsoy M, Zeytlunlu M, Kilic M, Alper M, Sozbilen M. The results of vascular and biliary variations in Turks liver donors: comparison with others. ISRN Surg 2011;2011:36703683.
2. Michels NA. Newer anatomy of the liver and its variant blood supply and collateral circulation. Am J Surg 1966;112:337–346.
3. Watson CJE, Harper SJF. Anatomical variation and its management in transplantation. Am J Transplant 2015;15:1459–1471.
4. Mourad MM, Liossis C, Gunson BK, Mergental H, Isaac J, Muiesan P et al. Etiology and management of hepatic artery thrombosis after adult liver transplantation. Liver Transpl 2014;20:713–723.
5. Duffy JP, Hong JC, Farmer DG, Ghobrial RM, Yersiz H, Hiatt JR et al. Vascular complications of orthotopic liver transplantation: experience in more than 4,200 patients. J Am Coll Surg 2009;208:896–903.