Is Portal Venous Pressure or Porto-systemic Gradient Really A Harbinger of Poor Outcomes After Living Donor Liver Transplantation?

Amol Vijay Kanetkar, Dinesh Balakrishnan, Sudhindran Sudhindran, Puneet Dhar, Unnikrishnan Gopalakrishnan, Ramachandran Menon, Othiyil Vayoth Sudheer

Department of Gastro Intestinal Surgery and Solid Organ Transplantation, Amrita School of Medicine, Amrita Institute of Medical Sciences and Research Centre, Kochi, Kerala, India

Background: **Portal hyperperfusion as a cause of small for size syndrome (SFSS) after living donor liver transplantation (LDLT) remains controversial. Portal venous pressure (PVP) is often measured indirectly and may be confounded by central venous pressure (CVP).** ***Methods:*** **In 42 adult cirrhotics undergoing elective LDLT, PVP was measured by direct canulation of portal vein and porto systemic gradient (PSG) was obtained after subtracting CVP from PVP. None underwent portal inflow modulation. SFSS was looked in 27 patients after excluding 15 with technical complications.** ***Results:*** **Clinical features of SFSS found in 6 patients, 5 with graft recipient weight ratio (GRWR) > 0.8% and PVP < 20 mm of Hg. One with GRWR < 0.8% could truly be labeled as SFSS. Incidence of SFSS was not higher in patients with elevated PVP > 20 mm of Hg (14.3% vs 0%, *P* = 0.259) or PSG > 13 mm of Hg (33.3% vs 0%, *P* = 0.111). Intensive care unit (ICU) stay was longer in patients with elevated PVP (14.55 vs 9.13 days, *P* = 0.007) and PSG (16.8 vs 9.72 days, *P* = 0.009). There was no difference in graft functions, post-operative complications and mortality in first month post-LDLT.** ***Conclusion:*** **Elevated PVP or PSG increased morbidity but neither predicted SFSS nor affected survival. (J Clin Exp Hepatol 2017;7:235–246)**

The demand for liver transplantation has continued to increase in the last three decades and has far outpaced the supply of organs from deceased donors. The entire evolution of living donor liver transplantation (LDLT) for adult recipients rests on two principles—donor safety and ensuring an adequately sized graft for the recipient. The size of the graft is most often limited by the concern for donor safety. Small for size syndrome (SFSS) refers to the clinical syndrome caused by a partial liver graft that is too small to sustain metabolic demands in the recipient. Portal hyperperfusion as measured by portal venous pressure (PVP) has been proposed to be a cause of SFSS after implantation of a reduced size liver graft and a potential contributor to poor outcomes after LDLT. Measurement of PVP is often indirect and not standardized. Moreover elevated central venous pressure (CVP) may confound the measured values of PVP. A porto systemic gradient (PSG) obtained by subtracting CVP from PVP may be more representative of graft injury occurring from portal hyperperfusion. This prospective study was performed to assess whether elevated PVP or PSG can predict poor recipient outcomes, especially SFSS.

MATERIALS AND METHODS

Patients

A prospective observational study was conducted at Amrita Institute of Medical Sciences, Kochi, Kerala, India from September 2014 to August 2015. Patients over 18 years of age undergoing elective LDLT during the period of study were included. Patients undergoing ABO incompatible LDLT, LDLT for fulminant hepatic failure, pediatric LDLT and patients with previous porto systemic shunt or splenic artery ligation or splenectomy, patients with portal vein thrombosis (PVT) or anomalies and patients with Budd Chiari syndrome were excluded from the study. Clearance from the Institutional Ethical Committee was taken prior to commencement of the study. Informed consent was taken from the patients prior to inclusion in the study. Disease severity in the recipients was assessed using Child Turcot Pugh (CTP) score and Model for End Stage Liver Disease (MELD) score.

Keywords: graft recipient weight ratio, cirrhosis, small for size syndrome
Received: 20.05.2016; *Accepted:* 29.01.2017; *Available online:* 6 February 2017
Address for correspondence: Kanetkar Amol Vijay, Resident, Department of Gastro Intestinal Surgery and Solid Organ Transplantation, Amrita School of Medicine, Amrita Institute of Medical Sciences and Research Centre, Ponekkara P.O., Kochi, Kerala 682041, India. Tel.: +91 9497641457.
E-mail: avkanetkar@gmail.com
Abbreviations: BAL: bronchoalveolar lavage; CTP: Child Turcot Pugh; CVP: central venous pressure; GRWR: graft to recipient weight ratio; HAT: hepatic artery thrombosis; INR: International Normalized Ratio; LDLT: living donor liver transplant; MELD: Model for End Stage Liver Disease; MHV: middle hepatic vein; *N*: total number; *P*: probability value; PNF: primary non-function; PSG: porto systemic gradient; PVP: portal venous pressure; PVT: portal vein thrombosis; ROC: receiver operating characteristics; SD: standard deviation; SGOT: serum glutamate oxaloacetate transaminase; SGPT: serum glutamate pyruvate transaminase; SFSS: small for size syndrome
http://dx.doi.org/10.1016/j.jceh.2017.01.114

© 2017 INASL.

Donors and Grafts

All patients included in the study received right lobe grafts. Graft steatosis was assessed using liver attenuation index obtained from the plain computed tomography (CT) scan of the donor and a value less than 5 was defined as graft steatosis.[1] A good outflow was ensured by taking the grafts with middle hepatic vein or reconstructing segment V and VIII veins.

Operation and Portal Venous Pressure Measurement

Main portal vein was cannulated using 24 gauge anti thrombotic canula before the native liver was dissected and was connected to pressure sensor. Three pressure readings were recorded and their mean was calculated. A PSG was obtained by subtracting CVP from PVP. At the end of bench work for the graft, graft recipient weight ratio (GRWR) was calculated after measurement of the graft weight. Second set of pressures were recorded after reperfusion and completion of all vascular graft anastomoses, again via canulation of main portal vein.

None of the patients underwent portal inflow modulations.

Post-Operative Care and Immunosuppression

A daily record of patient's vital parameters, Glasgow coma scale, urine output, and drain output (ascites) was maintained. The status of the graft vasculature was assessed using Doppler ultrasound scans done every 24 h for first 4 days, then on 7th, 14th, 21st and 30th day or earlier if clinically indicated. In cases where equivocal findings were noted on Doppler ultrasound, CT angiogram was performed. The standard peri-operative antibiotic prophylaxis consisted of piperacillin-tazobactam and fluconazole. In cases with preoperative positive culture reports, appropriate culture sensitive antibiotics were administered in the peri-operative period. Other drugs and supportive medications including N-acetyl cysteine and ursodeoxycholic acid were also administered. Blood investigations were performed according to the routine protocol followed for LDLT patients. Cultures (ascitic fluid/urine/blood/bronchoalveolar lavage [BAL] or sputum) were obtained on post-operative day 3 and 5 or as clinically indicated.

Triple immunosuppression was administered in the post-operative period consisting of steroids, tacrolimus and either azathioprine or mycophenolate mofetil. The target whole blood trough level of tacrolimus was 6–10 ng/ml in the first month following LDLT.

Primary End Point: Small for Size Syndrome

Dahm–Clavien Definition[2]

Presence of two of the following on three consecutive days in the first post-operative week in a small for size graft (GRWR < 0.8%) after exclusion of technical (arterial/portal occlusion/outflow congestion/bile leak), immunological (rejection), infectious (cholangitis, sepsis) factors

1. Bilirubin > 5.85 mg%
2. INR > 2
3. Encephalopathy grade 3 or 4

Secondary End Points

Graft functions measured using the peak, mean and median levels of bilirubin, transaminases (SGOT, SGPT), INR. Post-LDLT ascites, peak levels of creatinine, profound deterioration of renal functions, lactates in the first 30 post -operative days.

Profound deterioration of renal function was defined as post-operative renal dysfunction with rise in serum creatinine more than two times the baseline value, or urine output less than 0.5 ml/kg/h for 12 h,[3] or the need for renal replacement therapy was also assessed.

Other variables compared were complications like outflow occlusion (hepatic vein thrombosis), hepatic artery thrombosis (HAT), PVT, primary non-function (PNF), post-operative bleeding, requirement of transfusions, bile leak, positive blood cultures, time spent on ventilator, intensive care unit (ICU) and hospital and mortality in first 30 days of LDLT.

PNF was defined as irreversible loss of graft function requiring emergency re-transplantation during the first 10 days after the initial liver transplantation. It is characterized by AST $\geq$ 2000 UI/L, INR $\geq$ 3.0 or acidosis (pH $\leq$ 7.3 and/or lactate concentration $\geq$2$\times$ normal), in the absence of a vascular thrombosis.

Post-operative bleeding was defined as drop in hemoglobin by 2 g% during any 24-h period requiring blood transfusion or reoperation.

Statistical Analyses

For assessment of end points patients were divided in to two groups using their PVP (cut-off of 20 mm of Hg).[4] For assessment using PSG, no reference value was available to segregate patients in to two groups. A receiver operating characteristics (ROC) curve was plotted for PSG values for SFSS. A cut-off of 13 mm of Hg was obtained with sensitivity of 100%, specificity 92.3%, area under curve 0.98 (Figure 1). Statistical analysis was performed using Mann Whitney test for continuous data, Fischer's exact test for categorical data using IBM SPSS software version 20.

RESULTS

A total of 99 liver transplantations were performed during the period of study (September 2014 to August 2015). Fifty five patients met the inclusion criteria of which 45 consented for pressure measurements, however readings could be obtained in 42 of these as operating surgeons did not cannulate portal vein for concern for worsening of intra op

© 2017 INASL.

Download English Version:

https://daneshyari.com/en/article/5664848

Download Persian Version:

https://daneshyari.com/article/5664848

Daneshyari.com