Liver Graft-To-Recipient Spleen Volume Ratio (GSVR) as A Preoperative Predictor of The Outcome Of Adult Living Donor Liver Transplantation (ALDLT)

Original Article

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ABSTRACT

Background: The portal haemodynamic state is reflected in the spleen volume of cirrhotic recipients. Apart from pre-liver transplantation (LT) circumstances, increased portal venous flow following reperfusion during LT is also linked to the spleen volume; a low graft-to-spleen volume ratio (GSVR) has been found to predict postreperfusion portal hypertension (PHT). In order to prevent PHT, Gyoten *et al.* recently proposed that preoperative evaluation of GSVR may imply the necessity of splenectomy (SPX) before to reperfusion.

Aim: The study aim is to validate the effect of splenectomy in recipients with low GSVR on the outcome of LDLT.

Methods: This is a prospective cohort study. In our department of surgery at the National Liver Institute at Menoufia University, we operated on 77 cases between January 2021 and February 2024, 27 of which were paediatric cases that were not enrolled in the study and 50 of which were adult cases. Our study included 50 adult patients underwent ALDLT with or without splenectomy, 4 cases were excluded from study, 2 cases with GSVR >0.7 but splenectomy was done for previous hypersplenism, portal hypertension and other 2 cases with GSVR <0.7 but splenectomy wasn't done because of weak intra-operative portal vein flow.

Results: Our study revealed that there is significant correlation between the two groups regarding the platelet count and portal vein flow during the follow up of studied cases but there is no significant correlation regarding bilirubin level, PC/INR or ascites amount.

Conclusions: In our results, the difference is not significant between the 2 groups regarding postoperative complication or mortality. Increased Platelet count is the only significant parameter for splenectomy group. Further studies on Egyptian patients should be performed which can lead to different results and cut-off of GSVR due to the differences in the pathology of ESLD.

Key Words: GSVR, liver transplantation, portal hypertension, small for size syndrome.

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INTRODUCTION

For some types of hepatocellular carcinoma and endstage liver disorders, liver transplantation is a curative treatment. For situations such as portal hypertension, hypersplenism and avoidance of small for size syndrome (SFSS), simultaneous splenectomy is essential during LT^[1]. The use of simultaneous splenectomy (SPX) during LT is still debatable and not often carried out, although it may have a detrimental effect on surgical outcome such as operation period, blood loss, the development of portal vein thrombosis, and infection problems^[2,3].

According to Cheng *et al.*, the spleen volume in cirrhotic liver transplant patients (LT) indicates the portal haemodynamic condition. Furthermore, increased portal

venous flow following reperfusion during LT is similarly linked to the spleen volume; a low graft-to-spleen volume ratio (GSVR) has been shown as a predictor of postreperfusion portal hypertension (PHT)^[1]. Gyoten and associates. In order to prevent PHT, it is also suggested that a preoperative evaluation of GSVR may reveal the necessity of splenectomy (SPX) before to reperfusion^[2].

Reduced hepatic vasculature and elevated portal venous pressure are associated with living related liver transplantation (LRLT), which improves gradually as the graft regenerates and splenomegaly improves^[3]. When a recipient's portal vein flow surpasses 250 mL/min/100 g graft liver weight, portal hyperperfusion takes place^[4].

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Poor graft function may result from this in recipients with an acceptable graft-to-recipient weight ratio (GRWR) >0.8. Another factor contributing to posttransplant portal hyperperfusion and even small-for-size syndrome (SFSS) is the size of recipient spleen and liver graft[1]. Hepatocyte functional insufficiency resulting from abnormally endothelial activation because of high portal vein flow, arterial vasoconstriction, sinusoidal shear stress, and hepatocyte over-regeneration appears to be the main mechanism in SFSS^[5].

Since low GSVR can result in a poor prognosis and has been linked to post-LT thrombocytopenia, hyperbilirubinemia, coagulopathy, massive ascites, and early graft loss (EGL) when the spleen was preserved, Yao *et al.* concluded that splenectomy was recommended for patients with a low GSVR (≤0.7 gm/ml) regardless of the intraoperative PVP measurement^[6].

The purpose of this research is to confirm how splenectomy affects LDLT outcomes in recipients with poor GSVR.

PATIENTS AND METHODS

Patients who had LDLT at our surgical department, National Liver Institute, Menoufia University, between January 2021 and February 2024, were the subject of a single-center prospective investigation. The National Liver Institute Research Ethics Committee gave its approval to the project (Approval Number: 2021-P2-409-01). After excluding the following cases, 46 of the 77 consecutive patients were enrolled: Of the 27 pediatric instances, two had a GSVR >0.7g/mL but underwent splenectomy due to hypersplenism, while the other two had a GSVR <0.7 but underwent no splenectomy due to poor intra-operative portal vein flow following explant (Figure 1).

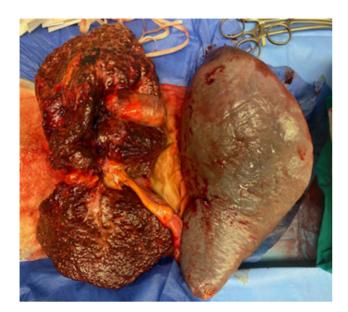


Figure 1: Native liver and spleen after hepatectomy and splenectomy.

The following criteria were gathered for this prospective investigation, which comprised adult patients over the age of 18 with end-stage liver disease from any etiology of liver cirrhosis and no prior splenectomy: age, gender, BMI, liver disease etiology, and laboratory and clinical evaluation of liver health using the Child-Paugh score, MELD-Na score, and portal hypertension criteria (encephalopathy, haematemesis, and ascites). In order to rule out the presence of a focal lesion, check for portal vein thrombosis (Figure 2), which is categorised by Yerdel et al.[7] if it is present, and check for portosystemic shunts such as lienorenal shunts and gastro-esophageal collaterals (varices), preoperative images (Multidetector Triphasic Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) were acquired during the two months prior to the liver transplant.

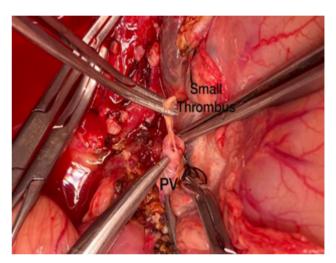


Figure 2: Portal vein thrombectomy.

Utilising three-dimensional CT scans of the recipient's spleen and the SYNAPS VINCENT program (Fujifilm Medical Co. Ltd. Tokyo, Japan), the spleen volume was measured. By dividing the estimated graft weight in grammes by the estimated spleen capacity in millilitres, the GSVR was evaluated. To estimate the graft weight preoperatively, we can take correlation coefficient of 0.91 from the expected graft volume^[2]. The Yao *et al.* study provided the GSVR threshold value of 0.7 g/mL^[6].

Our transplant team carried out all ALDLT operations on both donors and recipients in accordance with the collaborative protocol that our Institute and the Institute of Kyoto, Japan, developed in March 2003^[8].

Operative data was collected: Graft type (Rt or Lt grafts, graft with or without MHV), Actual graft weight and GRWR, Cold and warm ischemia times in minutes, Vascular venous reconstruction (major HV, segmental veins "RIV, V5, and V8", interposition vein grafts) and arterial reconstruction, Intraoperative Doppler US assessing the PV/HA patency (flow and velocities) and just after closure of the abdomen, and then twice daily until the 7th day after

surgery and once per day during the rest of hospital stay, Splenectomy or not, PVP measurement (Figure 3). PVP was recorded before hepatotomy, after hepatotomy with clamping of the PV, after splenectomy if done, after shunt or collaterals ligation if done and after reperfusion[9], Hepaticojejunostomy anastomosis or duct-to-duct biliary repair (Figure 4), plasma and blood transfusions per unit, and operating time in hours. A clinical condition known as primary graft non-function (PNF) causes liver necrosis or multisystemic malfunction and typically necessitates liver retransplantation (within the 90 days)^[10].



Figure 3: Portal vein pressure catheter insertion in a jejunal vein (white arrow), Caution not to injure marginal vessels (Blue dashed line)^[10].



Figure 4: Duct to duct biliary anastomosis.

Soejima *et al.*, provided the first objective definition of SFSS, where the presence of both prolonged functional cholestasis (total bilirubin level >5mg/dL on post-operative day [POD] 14 and intractable ascites (daily production of > 1L on POD 14 or > 500mL on POD 28) defined the Syndrome^[11]. The same group later revised their definition of prolonged functional cholestasis to reflect a total bilirubin level of >10mg/dL (instead of 5mg/dL) on POD 14^[12].

Acute rejection based on clinical, laboratory and histopathological outcomes. The National Healthcare Safety Network monitoring criteria from the Centres for Disease Control and Prevention will be used to identify bacterial infections (bacteremia, septicaemia, cholangitis, pleural, and wound)^[13].

Early graft loss defined as mortality or retransplantation during the first 3 months after transplantation^[10].

Statistical Analyses

Interquartile ranges (IQRs) or medians with ranges are used to describe continuous data where suitable. Numbers and percentages are used to display categorical data. The Chi-square test for categorical data and the Mann-Whitney U-test for continuous variables were used to express comparisons, respectively. Variables significant at a p<0.05 in the univariate analysis were utilised in the multivariate logistic regression model, and the PNF was examined using the univariate analysis (15). The Kaplan-Meier technique was used to estimate graft survival, and the log-rank test was used to analyse the survival differences between the two groups.

The association between drainage, portal flow, and GSVR was ascertained using the Pearson correlation coefficient. All statistical analyses were conducted using SPSS21.0 (IBM, United States).

RESULTS

There are 46 adult patients underwent ALDLT, these patients were divided in two groups, low GSVR group (<0.7) that underwent splenectomy (20 cases) and high GSVR group (>0.7) without splenectomy (26 cases).

Both groups are homogenous with no statistical significance between both groups regarding age, sex and BMI. Most of cases under the study are negative for hepatic viruses (n=21). Liver cirrhosis with HCC is the commonest cause for liver transplantation (n=14) then HCV related DLC (n=10) (Table 1).

There is statistical significance of the PVT between both groups of the study (p= 0.033), most cases with PVT are of the low GSVR group and underwent splenectomy (n= 6). Also, there is statistical significance of high preoperative PC (p= 0.014), INR (p= 0.05) and low platelet count (p= 0.02) in low GSVR group (Table 1).

There is statistically significant difference for spleen volume and portosystemic shunts to be ligated (p= 0.01) in low GSVR group and there is no significant difference in operation period, cold ischemia time, warm ischemia time or blood transfusion between the two groups (Table 2).

There is statistical significance between both groups of the initial PVP (p= <0.001) and PVP post clamping (p= 0.09), they are significantly high in low GSVR group (Table 2).

Bile leak is statistical significance in low GSVR group (N=8). Regarding bacterial infection, the five recorded cases were Klebsiella pneumonia detected by blood cultures which mostly hospital acquired infection (Table 3).

Eight patients underwent re-exploration, 6 cases of low GSVR group; there are three cases with bile leak underwent abdominal lavage and external biliary diversion (one case with hepaticojejunostomy anastomosis and two cases with duct-to-duct anastomosis), two cases for control of splenic bed spurter and one with graft cut surface bleeding. Regarding the two cases of high GSVR group were explored for hepatic artery thrombosis for reconstruction (Table 3).

We had 4 mortalities among low GSVR group; three cases with pneumonia and respiratory failure (two of them were previously explored for bile leak) and one case with septic shock for infected intra-abdominal collection (after exploration for splenic bed spurter). Also, we had 6 mortalities in high GSVR group; three cases with liver failure after hepatic artery thrombosis (two cases were explored before), two cases with pneumonia and respiratory failure and one case with renal failure (Table 3).

There is no statistical significance between both study groups regarding postoperative ascites amount. There is statistical significance of improving portal vein flow in low GSVR group after the first week of liver transplantation (p= 0.049). In low GSVR group, the platelet count remained low until postoperative day 5 to 7 and statistically significant improvement during post liver transplantation weeks 1 to 4 (Table 3).

There is no statistical significance of post operative lab results (bilirubin, albumin, prothrombin concentration and INR) between both study groups (Table 3).

Over the follow-up period (90 days), there was no significant difference in average survival days between splenectomy (78.3 days) and non-splenectomy groups (71.5 days) (*p*-value= 0.688) (Table 4, Figure 5).

Table 1: Pre-operative data and clinical characteristics of recipients with splenectomy or not according to graft-to-spleen volume ratio:

| Variables | Total (n= 46) | Splenectomy | | <i>P</i> -value |
|-------------------------------|---------------|--------------|-------------|-----------------|
| | | Yes $(n=20)$ | No $(n=26)$ | |
| Recipient factors | | | | |
| Age (years) | 48.3(11) | 47(11.8) | 49.3(10.5) | 527 |
| BMI (Kg/m²) | 27.7(4.6) | 27.2(3.8) | 28.1(5.1) | 0.522 |
| Female | 11(23.9) | 6(30) | 5(19.2) | 0.494 |
| Male | 35(76.1) | 14(70) | 21(80.8) | |
| Blood group | | | | |
| A+ve | 16(34.8) | 7(35) | 9(34.6) | 0.987 |
| B+ve | 7(15.2) | 5(25) | 2(7.7) | 0.213 |
| AB +ve | 5(10.9) | 1(5) | 4(15.4) | 0.369 |
| O+ve | 18(39.1) | 7(35) | 11(42.3) | 0.615 |
| Comorbidity | | | | |
| None | 29(63) | 14(70) | 15(57.7) | 0.391 |
| DM | 15(32.6) | 5(25) | 10(38.5) | 0.334 |
| HTN | 1(2.2) | 0(0) | 1(3.8) | 1 |
| Cardiac | 1(2.2) | 1(5) | 0(0) | 0.435 |
| Virology | | | | |
| HCV-ve / HBV -ve | 21(45.7) | 12(60) | 9(34.6) | 0.087 |
| HCV +ve | 6(13) | 1(5) | 5(19.2) | 0.369 |
| HBV +ve | 2(4.3) | 0(0) | 2(7.7) | 0.498 |
| HCV-ve post-treatment | 17(37) | 7(35) | 10(38.5) | 0.809 |
| Diagnosis | | | | |
| Autoimmune hepatitis | 4(8.6) | 3(15) | 1 (3.8) | 0.303 |
| Autoimmune hepatitis with HCC | 1(2.2) | 0(0) | 1 (3.8) | 1 |
| Bilharziasis | 5(10.9) | 2(10) | 3 (11.5) | 1 |
| Caroli syndrome | 2(4.4) | 1(5) | 1 (3.8) | 1 |
| Caroli disease | 1(2.2) | 0(0) | 1 (3.8) | 1 |
| Cryptogenic liver cirrhosis | 5(10.9) | 4(20) | 1 (3.8) | 0.151 |

| Variables | Total (n= 46) | Splenectomy | | <i>P</i> -value |
|-------------------------------------|---------------|--------------|-------------|-----------------|
| | | Yes (n= 20) | No (n= 26) | |
| DLC of unknown etiology with PVT | 1(2.2) | 1(5) | 0(0) | 0.435 |
| HCV related DLC | 10(21.7) | 3(15) | 7(26.9) | 0.476 |
| HCV related DLC with HCC | 14(30.4) | 5(25) | 9(34.6) | 0.482 |
| HBV related DLC | 1(2.2) | 0(0) | 1(3.8) | 1 |
| Primary sclerosing cholangitis | 1(2.2) | 0(0) | 1(3.8) | 1 |
| Wilson disease | 1(2.2) | 1(5) | 0(0) | 0.435 |
| Degree of liver disease | | | | |
| Child score | 9(2.6) | 9.4(2.6) | 8.7(2.7) | 0.326 |
| MELD | 14.4(5.4) | 15.7(4.8) | 13.4(5.7) | 0.068 |
| MELD-Na | 17.87(6.12) | 18.8(5.37) | 17.15(6.66) | 0.372 |
| Donor factors | | | | |
| Donor Age (years) | 26.9(6.3) | 26.1(5.3) | 27.5(7) | 0.689 |
| Donor BMI (Kg/m²) | 24.6(3.4) | 24.08(3.1) | 25.1(3.7) | 0.335 |
| Sequelae of end stage liver disease | | | | |
| Encephalopathy | 2(4.3) | 1(5) | 1(3.8) | 1 |
| Hematemesis | 3(6.5) | 1(5) | 2(7.7) | 1 |
| Ascites | 32(69.6) | 16(80) | 16(61.5) | 0.177 |
| PVT | 7(15.2) | 6(30) | 1(3.8) | 0.033 |
| Preoperative laboratory variables | | | | |
| Total bilirubin (mg/dL) | 1.9(2.03) | 1.9(1.75) | 2(4.25) | 0.73 |
| Direct bilirubin (mg/dL) | 0.79(1.64) | 0.77(0.74) | 0.97(2.55) | 0.92 |
| Albumin (gm/dL) | 2.85(1.4) | 3(1.35) | 2.7(1.25) | 0.89 |
| PC (%) | 54.3(18.8) | 48.35(10.29) | 58.9(22.47) | 0.041 |
| INR | 1.4(0.43) | 1.59(0.34) | 1.36(0.51) | 0.05 |
| PLT ($\times 10^3/\mu$ L) | 87.5(104.3) | 54.5(99.5) | 110(92.5) | 0.02 |

 Table 2: Intra-operative data of the study groups:

| Variables | Total $(n=46)$ | Splenectomy | | P-value |
|---|----------------|---------------|--------------|---------|
| | | Yes (n= 20) | No (n= 26) | |
| Operative characteristics | | | | |
| Right lobe graft - N(%) | 37(80.4) | 17(85) | 20(76.9) | 0.71 |
| Left lobe graft - N(%) | 9(19.6) | 3(15) | 6(33.1) | 0.71 |
| Spleen volume (cc) - Mean (SD) | 1142.4(683.7) | 1770.8(514.9) | 659(284.4) | < 0.001 |
| Estimated graft weight (gm) - Mean (SD) | 810(185.6) | 805.9(176.8) | 813.2(195.5) | 0.898 |
| Estimated graft volume (cc) - Mean (SD) | 890.4(203.9) | 885.7(194.3) | 894.1(214.7) | 0.892 |
| GRWR - Mean (SD) | 1.04(0.21) | 1.08(0.3) | 1.05(0.31) | 0.697 |
| Actual graft weight (gm) - Mean (SD) | 750.1(149.7) | 747.5(134.9) | 752.1(162.8) | 0.919 |
| Actual GRWR - Mean (SD) | 1.08(0.7) | 0.98(0.18) | 1.1(0.9) | 0.431 |
| Operative time - Mean (SD) | 11(2.5) | 10.9(2.4) | 11.1(2.7) | 0.863 |
| Cold ischemia time (min) - Mean (SD) | 62.5(40) | 67.5(52.5) | 60 (36.3) | 0.839 |
| Warm ischemia time (min) - Mean (SD) | 40(13.9) | 40(13.75) | 42.5(15) | 0.389 |
| Shunt ligation - N (%) | 20(43.5) | 13(65) | 7(26.9) | 0.01 |
| Initial PVP (mmHg) - Mean (SD) | 21(14) | 25.08(4.52) | 15.53(5.89) | < 0.001 |
| Final PVP (mmHg) - Mean (SD) | 14.19(4.5) | 13.5(6.75) | 13(5) | 1 |

| Variables | Total (n= 46) | Splenectomy | | <i>P</i> -value |
|---|---------------|-------------|------------|-----------------|
| | | Yes (n= 20) | No (n= 26) | |
| Type of biliary anastomosis (Duct to Duct/Hepatico-Jejunostomy) | 46 | 20(16/4) | 26(23/3) | 0.68 |
| Cell saver transfusion (mL) Median (IQR) | 350(625) | 0(287.5) | 350(625) | 0.28 |
| Packed RBCs transfusion (units) Median (IQR) | 1.5(2) | 1.5(2) | 1.5(2) | 0.86 |
| Plasma transfusion (units) Median (IQR) | 2(3.75) | 1(3.75) | 2(3.75) | 0.98 |

Table 3: Post-operative data of the study groups and outcome:

| Variables | Total (n= 46) | Splenectomy | – <i>P</i> -value | | |
|---|---------------|-------------|-------------------|---------|--|
| variables | 10tai (n= 40) | Yes (n= 20) | No (n=26) | P-value | |
| Complications | | | | | |
| Rejection | 3(6.5) | 2(10) | 1(3.8) | 0.57 | |
| Bacterial infection | 5(10.9) | 4(20) | 1(3.8) | 0.15 | |
| Bleeding | 2(4.3) | 2(10) | 0 | 0.18 | |
| HA thrombosis | 2(4.3) | 0 | 2(7.7) | 0.5 | |
| Bile leak | 11(23.9) | 8(40) | 3(7.7) | 0.038 | |
| Reoperation | 8(17.4) | 6(30) | 2(7.7) | 0.06 | |
| In-patient mortality 10(21.7) | | 4(20) | 6(23.1) | 1 | |
| Causes of reoperation | | | | | |
| Bile leak | 3 | 3 | 0 | | |
| Iepatic artery thrombosis | 2 | 0 | 2 | | |
| Splenic bed spurter | 2 | 2 | 0 | | |
| Liver cut surface bleeding | 1 | 1 | 0 | | |
| Causes of in-patient mortality | | | | | |
| neumonia and respiratory failure | 5 | 3 | 2 | | |
| iver failure after HAT | 3 | 0 | 3 | | |
| eptic shock | 1 | 1 | 0 | | |
| Renal failure | 1 | 0 | 1 | | |
| ost-operative amount of ascites in | mL | | | | |
| OD 1 | 385(505) | 501(428) | 448(370) | 0.579 | |
| POD 3 | 800(950) | 1078(713) | 884(596) | 0.343 | |
| OD 5 | 1000(1600) | 1150(1500) | 810(1500) | 0.147 | |
| POD 7 | 1100(1500) | 1479(1102) | 1241(1075) | 0.472 | |
| POD 14 | 290(1000) | 842(1045) | 518(726) | 0.32 | |
| POD 28 | 0(0) | 0(13) | 0(0) | 0.826 | |
| Post-operative platelet count (×10 ³ / | μL) | | | | |
| POD 1 | 117(98) | 146(105) | 170(130) | 0.438 | |
| OD 3 | 93(107) | 169(109) | 80(62) | < 0.001 | |
| POD 5 90(108) | | 163(160) | 68(48) | < 0.001 | |
| POD 7 | 122(154) | 230(119) | 91(63) | < 0.001 | |
| POD 14 | 231(282) | 447(205) | 172(72) | < 0.001 | |
| POD 28 | 336(408) | 503(210) | 237(138) | < 0.001 | |
| POD 35 | 299(318) | 478(258) | 170(149) | < 0.001 | |

| | Variables | Total (v= 46) | | – D value | | |
|-----------|--------------------------------|-------------------|--------------|--------------|--------------|-----------|
| | Variables | Total (n= 46) | Ye | es (n= 20) | No (n= 26) | – P-value |
| POD 42 | | 292(312) | 5 | 39(240) | 228(97) | < 0.001 |
| ost-opera | tive PV flow (cm/s) by ultra | sound | | | | |
| POD 1 | | 42.8(15.5) | 4 | 1.5(15.6) | 43.7(15.7) | 0.648 |
| OD 3 | | 38(13) | 3 | 9.2(6.7) | 43.9(14.5) | 0.47 |
| OD 5 | | 36(14) | | 35(10) | 40(15.5) | 0.064 |
| OD 7 | | 35(15) | 3 | 2(11.25) | 40(13) | 0.049 |
| OD 14 | | 36.6(10.1) | 3 | 1.8(6.7) | 40.9(10.8) | 0.003 |
| OD 28 | | 31(7.3) | 2 | 8.2(5.8) | 33.4(7.7) | 0.025 |
| ost-opera | tive lab results of Bilirubin, | Albumin, PC and I | NR | | | |
| | Total bilirubin (mg/dL) | | 4.93(4.12) | 4.89(3.98) | 4.93(4.02) | 0.64 |
| | Direct bilirubin (mg/dL) | | 2.88(3.39) | 2.94(3.27) | 2.72(3.35) | 0.53 |
| POD1 | Albumin (gm/dL) | | 2.7(0.41) | 2.72(0.33) | 2.68(0.46) | 0.78 |
| ш. | PC (%) | | 31(10.5) | 27.35(10.1) | 33.6(10.1) | 0.13 |
| | INR | | 2.39(0.64) | 2.47(0.53) | 2.33(0.72) | 0.474 |
| | Total bilirubin (mg/dL) | | 2.1(2.5) | 2.11(3.02) | 2.1(2.02) | 0.93 |
| | Direct bilirubin (mg/dL) | | 1.18(1.83) | 1.3(1.96) | 1.16(1.84) | 0.76 |
| POD3 | Albumin (gm/dL) | | 2.9(0.35) | 3.01(0.31) | 2.89(0.38) | 0.27 |
| Ь | PC (%) | | 45.71(14.31) | 49(14.85) | 43.09(13.58) | 0.171 |
| | INR | | 1.63(0.6) | 1.58(0.61) | 1.65(0.78) | 0.37 |
| | Total bilirubin (mg/dL) | | 2.5(2.72) | 2.02(3.58) | 2.76(2.27) | 0.58 |
| | Direct bilirubin (mg/dL) | | 1.31(2.21) | 1.21(2.48) | 1.64(1.94) | 0.73 |
| POD5 | Albumin (gm/dL) | | 3.03(0.42) | 3.1(0.42) | 2.98(0.42) | 0.371 |
| П | PC (%) | | 56.25(14.66) | 59.64(13.92) | 53.43(14.94) | 0.164 |
| | INR | | 1.4(0.43) | 1.33(0.36) | 1.42(0.54) | 0.3 |
| | Total bilirubin (mg/dL) | | 2.2(3.58) | 2.4(3.21) | 2.2(4.3) | 0.535 |
| | Direct bilirubin (mg/dL) | | 1.68(3.07) | 1.71(3) | 1.65(3.09) | 0.77 |
| POD7 | Albumin (gm/dL) | | 3.1(0.6) | 3.25(0.58) | 3(0.7) | 0.678 |
| ш. | PC (%) | | 64.63(17.39) | 65.44(16.46) | 66.38(15.72) | 0.733 |
| | INR | | 1.28(0.21) | 1.26(0.23) | 1.3(0.21) | 0.864 |
| | Total bilirubin (mg/dL) | | 1.28(2.65) | 1.35(2.62) | 1.22(3.17) | 0.93 |
| 4 | Direct bilirubin (mg/dL) | | 0.59(1.86) | 0.65(1.84) | 0.59(2.23) | 0.92 |
| POD14 | Albumin (gm/dL) | | 3.35(0.63) | 3.35(0.9) | 3.3(0.73) | 0.487 |
| PC | PC (%) | | 79.38(21.98) | 79.36(21.51) | 79.34(21.54) | 0.995 |
| | INR | | 1.11(0.24) | 1.11(0.31) | 1.13(0.23) | 0.94 |
| | Total bilirubin (mg/dL) | | 0.79(1.36) | 0.64(1.02) | 0.95(1.47) | 0.219 |
| ∞ | Direct bilirubin (mg/dL) | | 0.42(0.79) | 0.33(0.84) | 0.44(1.04) | 0.327 |
| POD28 | Albumin (gm/dL) | | 3.62(0.65) | 3.59(0.63) | 3.55(0.63) | 0.737 |
| ď | PC (%) | | 81.91(23.99) | 82.31(20.54) | 82.76(16.58) | 0.9 |
| | INR | | 1.08(0.19) | 1.07(0.2) | 1.11(0.18) | 0.976 |

Table 4: Analysis of the Kaplan-Meier Survival curve for all cases under the study for 90 days after surgery:

| | Mean | | | | |
|---------------------|---------------------|--------------|-------------|-----------------|-------|
| Splenectomy | E-4 | | 95% Confid | <i>P</i> -value | |
| | Estimate Std. Error | Std. Error — | Lower Bound | Upper Bound | _ |
| Yes (n= 20) | 78.250 | 5.499 | 67.472 | 89.028 | |
| No (<i>n</i> = 26) | 71.538 | 6.667 | 58.471 | 84.606 | 0.688 |
| Overall $(n=46)$ | 74.457 | 4.490 | 65.657 | 83.256 | |

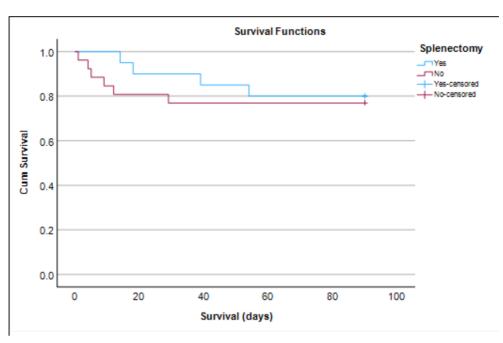


Figure 5: Kaplan-Meier survival curve for all cases under the study for 90 days after surgery.

DISCUSSION

In the context of graft insufficiency and the development of SFSS, it was traditionally believed that graft weight was the sole factor influencing graft outcome^[14]. found that using SFSGs (grafts with GRWR <1%) resulted in worse graft survival, most likely due to increased damage to parenchymal cells and decreased ability for metabolism and synthesis. According to Tanaka and Ogura (2004), early graft survival was considerably worse at Kyoto University when the GRWR was less than 0.8%^[15]. Additionally, our department found that the usage of SFSG (GRWR < 0.8%) is the primary cause of SFSS following ALDLT, which results in bad outcomes. Splenectomy was recommended to adjust portal inflow and avoid catastrophic outcomes^[16]. However, the idea of SFSS has changed from being solely reliant on the graft weight to being a multifactorial process that involves recipient factors as well as graft factors. Portal hyperperfusion, persistent portal hypertension, and shear stress are thought to be the main potential contributors, and this idea has become very popular^[15].

Simultaneous splenectomy with liver transplantation has been a matter of debate ever since the early beginning of LT as the indications for splenectomy have been changing with time and with development of new advances in treatments. Nowadays the only common indication for splenectomy is for PVP modulation and prevention of SFSS^[6]

In our study, we considered the cut-off value of GSVR is <0.7 gm/ml according to Yao et al., study in 2019 as it was the most recent study when we started our study and it was done over a large number of cases (349 LDLT). The relationship between graft size and spleen volume has been increasingly studied lately. The association between graft and spleen sizes was explained by Cheng et al., who also pointed out that GRWR alone might not be a full measure because it did not account for the recipient portal circulation's haemodynamic state. Thus, they included graft-to-recipient spleen size ratio, which, if less than 0.6, would indicate post-transplant hyper-perfusion[1]. In the same context, Gyoten et al. found a strong correlation between the association between graft and spleen volumes and PVP upon reperfusion. Additionally, they said that portal hypertension above 20mmHg was predicted by a spleen volume-to-graft volume ratio greater than 0.95^[2]. According to Macshut et al., a GSVR of less than 0.64gm/ml was a risk factor for elevated PVP during graft reperfusion^[10]. Based on each ratio, splenectomy was advised prior to reperfusion in all of these investigations. Low GSVR (<1.03gm/ml) was a significant predictor of portal hypertension and poor graft function following LDLT, according to a recent Chinese research. They did, however, suggest that in certain situations, partial splenectomy be considered rather than splenectomy^[17].

In our study, there is higher incidence of PVT before surgery in low GSVR group with statistical significance between both groups (p=0.033). Also, in Yao *et al.* study, here is higher incidence of PVT in low GSVR group but without statistical significance (p=0.524)^[6].

In our study, there is higher incidence of porto-systemic shunts to be ligated with statistical significance between both groups (p= 0.01).

Cheng *et al.* reported that there was no statistically significant relationship between the spleen size and the presence of portosystemic shunts $(p=0.149)^{[1]}$.

Also, in Yao *et al.* study, there is no statistically significant difference between the low GSVR and the presence of portosystemic shunts that need to be ligated $(p=0.768)^{[6]}$.

Our study found that while there was a significant difference in the pre-operative PC and INR levels between the two groups, there was no significant difference in the postoperative bilirubin level, PC, or INR between the two groups. According to Marubashi *et al.*, (2007), PT-INR >1.6 on POD 5, hyperbilirubinemia >20mg/dL for >7 days after POD 7, and a peak TB level >27mg/dL within 28 days have all been found to be significant predictors of death^[18]. Nevertheless, little is known about the processes behind these anomalies. According to a different Kyushu University study, portal hyper-perfusion, persistent thrombocytopenia, and hyperbilirubinemia > 30 days following LDLT were predicted by a graft-to-spleen volume ratio <0.88^[6].

In their study, Xiao *et al.* found no discernible changes between the normal GSVR and low GSVR groups in terms of post-operative ALB, INR, TB, and portal vein flow (cm/sec)^[17]. The low GSVR group that received splenectomy mostly from POD 7 had a substantial drop in portal vein flow, with a statistically significant difference in post-operative portal vein flow (cm/sec) (p= 0.049).

In our investigation, the two study groups' platelet counts differed significantly, and the group that had a splenectomy also showed a considerable improvement. From the first week onwards, there was a strong association between the two groups. Yao *et al.*, (2019) report that the platelet count grew quickly during postoperative weeks 1–4 and stayed low until (PODs) 5–7^[6].

In their research, Xiao *et al.* found that the platelet counts of the low GSVR and normal GSVR groups differed significantly. In the low GSVR group, it took more

than a week for it to normalise and stayed at a low median value[17].

According to our research, the low GSVR group that had a splenectomy had a high rate of ascites, which began to decline two weeks following liver transplantation. The two research groups do not, however, significantly correlate with one another. Additionally, Yao *et al.*, (2019) found that the low GSVR group did not recover for almost a month following liver transplantation, and the quantity of ascites remained high^[6]. According to Xiao *et al.*, the normal GSVR and low GSVR groups differed significantly in the quantity of ascites $(p=0.001)^{[17]}$.

The splenectomy group with low GSVR had a greater risk of infection and reoperation for postoperative hemorrhage, but there is no significant difference in operational time or blood loss between the two groups in our research. According to Ito *et al.* (2016), recipients who have a simultaneous splenectomy have a higher incidence of lethal infectious disease, postoperative hemorrhage within the first postoperative week, greater intraoperative blood loss and longer operation period than those who do not^[19]. Additionally, the low GSVR group in the Yao *et al.* trial saw a greater risk of infection^[6].

Although the incidence of rejection was greater in the low GSVR group (two instances) than in the other group (one case) in our investigation, the difference was not statistically significant (p= 0.57). The low GSVR group saw a greater frequency of rejection with statistical significance (p= 0.040), according Yao *et al.*, 2019^[6].

Eight bile leaks occurred in the low GSVR group that had splenectomy in our research, compared to three occurrences in the non-splenectomy group. This difference is statistically significant (p= 0.038). Bile leak incidence is considerable in low GSVR but not statistically significant (p= 0.126), per a 2022 research by Xiao $et\ al.$, [17].

Our study's survival analysis showed that, after 90 days, there was no significant difference in average survival days between splenectomy (78.3 days) and non-splenectomy groups (71.5 days) (p-value= 0.688). According to the Yao $et\ al.$, research, patients with splenectomy had a higher graft survival rate at 90 days (100% vs. 71.0% for the non-splenectomy group, p-value= 0.011) among patients with poor GSVR (n= 48)^[6].

CONCLUSION

In our results, the difference is not significant between the 2 groups regarding postoperative complication or mortality. Increased Platelet count is the only significant parameter for splenectomy group. Further studies on Egyptian patients should be performed which can lead to different results and cut-off of GSVR due to the differences in the pathology of ESLD.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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