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Original Research Article

INCIDENCE OF INTRAABDOMINAL HYPERTENSION (IAH) AND ITS IMPACT ON POSTOPERATIVE OUTCOMES IN ADULTS UNDERGOING LIVING DONOR LIVER TRANSPLANTATION (LDLT) – A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Intra-abdominal hypertension (IAH) is a recognized perioperative risk factor in critically ill and post-surgical patients, including liver transplant recipients. However, limited data exist regarding its prevalence and impact in adult living donor liver transplantation (LDLT), particularly in the Indian subcontinent. **Objectives:** To determine the incidence of IAH in adult LDLT recipients and assess its effect on postoperative renal, graft, hemodynamic, and survival outcomes. **Materials and Methods:** This prospective observational study included 37 adult LDLT recipients (18–70 years) over a defined study period at a tertiary care center. Patients were categorized into those with preoperative IAH (Group H, n=31) and without IAH (Group NH, n=6) based on urinary bladder pressure measurements. IAH was graded per WSACS definitions. Postoperative outcomes, including acute kidney injury (AKI), early allograft dysfunction (EAD), portal vein flow, vasoactive requirements, mechanical ventilation duration, ICU stay, and survival, were compared. **Result:** Preoperative IAH was present in 83.7% of patients, predominantly Grade I (64.5%) and Grade II (32.3%). Mean preoperative IAP was significantly higher in Group H compared to Group NH (14.8 ± 2.56 vs 10.33 ± 2.33 mmHg, p=0.03). IAP decreased to <10 mmHg by POD 3 in both groups. AKI incidence was higher in Group NH (66.7% vs 35.5%, p=0.15), and EAD occurred in 50% vs 35.5% of patients, respectively, though neither difference reached statistical significance. Portal vein flow, vasoactive support, mechanical ventilation duration, ICU stay, and survival rates were comparable between groups. **Conclusions**: IAH is highly prevalent in adult LDLT recipients, yet its presence did not significantly impact short-term postoperative outcomes in this cohort. The marked postoperative reduction in IAP underscores the role of intraoperative decompression and optimized perioperative management. Routine IAP monitoring and early intervention may help mitigate potential complications. Larger multicenter studies are needed to validate these findings.

INTRODUCTION

Monitoring the effects of Intra-abdominal Hypertension (IAH) has been in clinical practice in variety of clinical situation, including post-surgical and critically ill patients. Intra-abdominal Hypertension (IAH) as defined by the World Society of Abdominal Compartment Syndrome (WSACS) refers to Intra-abdominal pressure (IAP) greater than 12 mmHg and is categorized in four grades; Grade 1 (12 - 15 mmHg), Grade 2 (16 – 20mmHg), Grade 3 (21 – 25mmHg), Grade 4 (>25mmHg). When IAP exceeds 20 mmHg and is accompanied with new organ failure or dysfunction, it is termed as Abdominal Compartment Syndrome (ACS).[1]

Elevated IAP has detrimental effects on multiple organ systems, impairing respiratory mechanics, reducing cardiac output, decreasing renal perfusion, and compromising hepatic microcirculation.[2,3] These systemic effects of IAH are particularly relevant in surgical and critically ill patients, including those undergoing liver transplantation. Patients undergoing living donor liver transplantation (LDLT) are also at risk of IAH due to their preoperative chronic liver disease state, frequent association with tense ascites, the complex nature of the transplant procedure, including the risk of intraperitoneal haemorrhages (surgical bleeding or coagulopathy-related), the use of perihepatic or retroperitoneal packs to control bleeding, bowel congestion due to portal hypertension, massive fluid and blood product administration, Therefore, it is important to monitor IAP in post-LDLT patients.[4,5]

There are several important reasons why this study is necessary

1. While IAH has been studied in critically ill patients and those undergoing orthotopic liver transplantation (OLT), limited data exist regarding its incidence and impact in the setting of adult living donor liver transplantation (LDLT) - particularly in the Indian subcontinent.

2. Understudied in Adult LDLT:

While paediatric LDLT studies have reported IAH incidence as high as 85%, there is limited literature exploring IAH in adult LDLT patients, who may have different abdominal wall compliance, ascites burden, and postoperative recovery dynamics. Most available data pertain to OLT or mixed donor types.[6,7,8]

3. IAH Is Modifiable and Manageable

Unlike some perioperative risk factors, IAH is dynamic and potentially reversible through interventions such as ascitic drainage, optimization of fluid balance, or abdominal decompression.[1,8] Early identification can help prevent complications like AKI, delayed graft function, or ventilatory difficulties.[1,9]

4. Scarcity of Indian Data

LDLT constitutes a significant portion of transplant activity in India, yet there is a lack of region-specific evidence evaluating the burden and consequences of IAH in this population. Local data are critical for tailoring perioperative monitoring protocols and for contributing to international clinical guidelines.

Through this prospective observational study, we aimed to evaluate the incidence of Intra- abdominal Hypertension in adult patients undergoing Living Donor liver Transplant (LDLT) and its impact on postoperative outcome in adult LDLT patients. We also investigated the possible adverse effects of IAH on renal, respiratory, cardiac and graft function.

MATERIALS AND METHODS

This prospective observational study was conducted at a tertiary care hospital after approval from after approval from Institutional Ethical Committee (IEC approval number: IEC/2024107/MA12), the trial was registered under Clinical Trial Registry of India (CTRI registration number CTRI/2025/06/088527). Written informed consent was obtained from all the patients enrolled in the study, following the principles of Declaration of Helsinki. A total of 40 patients, aged 18 to 70 years of either sex, who underwent LDLT due to chronic liver disease were included in the study. Patients who underwent transplant for Acute liver failure (ALF), Acute on chronic liver failure (ACLF), patients with preoperative renal dysfunction (Acute kidney injury within 6 weeks), Chronic kidney disease (CKD), presence of cardiac comorbidity (coronary artery disease, severe valvular heart disease, cardiomyopathy, arrhythmias), reoperation or re-transplant and those who underwent Deceased Donor Liver Transplant (DDLT) were excluded from the study.

All the prospective liver recipients at our institute undergo a complete laboratory investigations and comprehensive cardiac, pulmonary, psychiatric, and anaesthetic evaluation prior to being posted for surgery / listed as a potential recipient. A brief anaesthetic check-up was done for all patients before shifting them to the operation theatre. All the standard ASA monitors (ECG, non-invasive blood pressure and pulse oximeter) were attached before induction of anaesthesia. A standard anaesthesia technique was followed for all the patients as per institutional protocol. The radial and femoral arteries were cannulated for invasive blood pressure monitoring and cardiac output monitoring. Internal jugular vein was cannulated with a four lumen central venous catheter for central venous pressure monitoring and administration of inotropes and continuous infusions of drugs. A 3rd generation Flo-Trac (Vigileo, Edwards) monitor attached to a femoral arterial catheter. Flo trac performs automatic arterial waveform analysis using the principle of pulse contour analysis, to yield stroke volume (SV), cardiac output (CO), systemic vascular resistance (SVR) and stroke volume variation (SVV). The above parameters were monitored continuously. All the operations during this study period were

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performed by a single surgical team and managed by single anaesthesia team.

Intra-abdominal pressure (IAP) measurement: After induction of anaesthesia and urinary catheterization, preoperative intraabdominal pressure (IAP) was measured by urinary bladder technique. After urinary catheterization, urine was drained from the bladder. A pressure transducer was attached to the foleys catheter through a PMO (pressure monitoring) line. The assembled system was then filled with saline solution and the transducer was positioned at point zero at mid-axillary level. 25mL of saline was injected and IAP readings was noted from the monitor. The urine drainage bag was the attached to the foleys catheter after detaching transducer assembly including PMO line. Similarly IAP was measured every 24 hours for three consecutive days in the postoperative period.

Postoperative monitoring and assessment: Possible deleterious effects of Intrabdominal hypertension (IAH) on postsurgical outcome were monitored and assessed as follows -

Renal function assessment

1. Daily urinary output/hour (ml/kg/hr)
2. Daily serum creatinine levels (mg/dl)

AKI was defined as per KDIGO guideline: Presence of any of the following-

- Increase in serum creatinine by ≥ 0.3 mg/dl within 48 hours or
- Increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days or
- Urine volume <0.5 ml/kg/h for 6 hours

AKI was considered to be potentially related to intraabdominal hypertension when AKI developed within the period of 48hr in postoperative period as adopted by Gianni et al in their study.

Early allograft dysfunction (EAD):

Determined by applying Olthoff criteria: Presence of one or more of the following

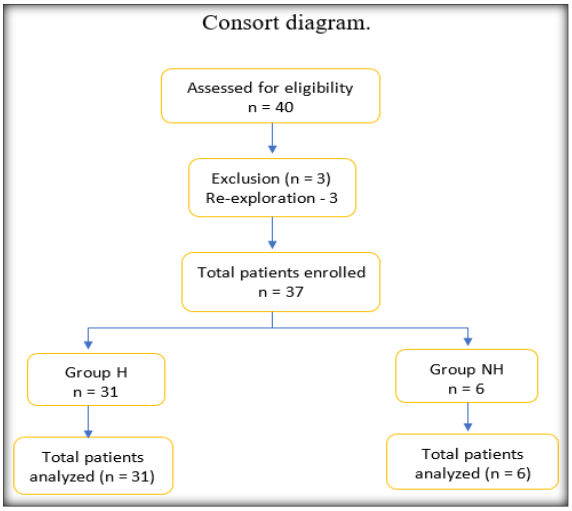
- Bilirubin ≥ 10 mg/dL on postoperative day (POD) 7
- INR ≥ 1.6 on POD 7
- ALT or AST > 2000 units/L within the first 7 postoperative days

Other postoperative outcome assessed were

- Total duration of mechanical ventilation
- Total vasoactive days and cumulative dose
- ICU length of stay
- ICU outcome (alive / dead)

Statistical Analysis

The data was entered into Microsoft excel format and continuous data were shown as Mean ± SD whereas categorical data were shown as numbers (%). The student's t test was used for unpaired numerical data analysis. The Pearson coefficient was used to test correlations between continuous variables. Univariate analysis of continuous variables was performed using the t-test or the Mann-Whitney U test as a non-parametric alternative where appropriate. Chi-square and Fisher's exact tests were used for categorical variables. Multivariable analysis in backward stepwise logistic regression was performed using variables with P value ≤0.20 in univariable analysis. All statistical tests were performed using SPSS version 22.



RESULTS

Table 1: Demographics and baseline parameters

Demographics	Group H (n=31)	Group NH (n=6)	P-value
Age (years, mean ± SD)	45.06 ± 9.24	48.17 ± 7.62	0.44
Gender - Male (%)	29 (93.5%)	5 (83.3%)	0.40
- Female (%)	2 (6.5%)	1 (16.7%)	
BMI (kg/m², mean ± SD)	23.22 ± 2.02	21.70 ± 2.43	0.10
Comorbidity - Present (%)	20 (64.5%)	3 (50%)	0.66
- Absent (%)	11 (35.5%)	3 (50%)	
Preoperative Parameters			
MELD-Na (mean ± SD)	21.94 ± 5.30	20.5 ± 5.92	0.50
Etiology of Chronic Liver Disease (%)			0.47
Alcoholic liver disease (Ethanol)	14 (45.2%)	4 (66.7%)	
Hepatitis B (HBV)	7 (22.6%)	0 (0%)	
Hepatitis C (HCV)	1 (3.2%)	1 (16.7%)	
Non-alcoholic steatohepatitis (NASH)	4 (12.9%)	1 (16.7%)	
Autoimmune hepatitis (AIH)	1 (3.2%)	0 (0%)	
Cryptogenic	4 (12.9%)	0 (0%)	
Decompensation (%)			
Jaundice	25 (80.6%)	4 (66.7%)	0.44

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Ascites	29 (93.5%)	5 (83.3%)	0.40
Hepatic encephalopathy (HE)	12 (38.7%)	2 (33.3%)	0.80
Acute variceal bleed (AVB)	7 (22.6%)	0 (0%)	0.19
Spontaneous bacterial peritonitis (SBP)	15 (48.4%)	0 (0%)	0.02
Hepatic hydrothorax (HH)	1 (3.2%)	2 (33.3%)	0.01

(BMI: Body mass index, SD: Standard Deviation)

There was no statistically significant differences in age, gender, BMI, comorbidities, MELD-Na scores, or etiologies of liver disease between Group H and Group NH. Group H showed significantly higher incidence of SBP (48.4% vs 0%, P=0.02) and lower incidence of hepatic hydrothorax (3.2% vs 33.3%, P=0.01). Other decompensation features (jaundice, ascites, HE, AVB) were comparable between both the groups. [Table 1]

Table 2: Preoperative incidence and grading of IAH

IAH Parameter	Category	n	Percentage (%)
Preoperative IAH (n = 37)	Present	31	83.7
	Absent	6	16.2
	Total	37	100
Grading of IAH (n = 31)	Grade I	20	64.5
	Grade II	10	32.3
	Grade III	1	3.2
	Grade IV	0	0
	Total	31	100

Preoperative IAH was present in 83.7% of patients. Among those with IAH, majority had Grade I (64.5%) and Grade II (32.3%); no cases of Grade IV IAH. [Table 2]

Table 3: Intraoperative parameters

Parameters	Group H (n= 31)	Group NH (n= 6)	P- value
Duration of surgery (hr)	11.34 ± 0.59	11.13 ± 0.95	0.48
Crystalloid (ml)	6322.57 ± 2640.87	6166.6 ± 1169.04	0.82
5% albumin (ml)	427.42 ± 206.09	375 ± 209.16	0.57
20% albumin (ml)	124.84 ± 46.96	115 ± 42.77	0.13
PRBC (units)	3.9 ± 3.01	3.33 ± 3.07	0.67
FFP (units)	2.65 ± 1.30	3.33 ± 2.65	0.33
Cryoprecipitate (units)	3.71 ± 2.86	3.33 ± 2.73	0.76
SDPC transfused (units)	0.03 ± 0.18	0.17 ± 0.40	0.19
Graft weight (grams)	619.93 ± 124.96	574.16 ± 170.27	0.56
Blood loss (ml)	2565.16 ± 1137.43	2991.6 ± 1665.42	0.44
Urine output (ml)	916.29 ± 297.95	948.3 ± 223.64	0.80
Positive fluid balance (ml)	1710.65 ± 2293.63	2188.3 ± 1516.6	0.62
Negative fluid balance (ml)	2331.77 ± 3109.15	538.33 ± 1318.64	0.17
Ascites drained (ml)	5538.71 ± 4282.965	2250.00 ± 3126.50	0.08
Pleural fluid drained (ml)	36.53 ± 36.02	18.26 ± 21.71	0.05
Noradrenaline maximum dose	0.35 ± 0.13	0.26 ± 0.12	0.16
Vasopressin maximum dose	0.02 ± 0.06	0.02 ± 0.06	0.73
Lactate on shifting	2.7 ± 0.69	3.7 ± 0.92	0.04

(PRBC: Packed red blood cell, FFP: Fresh frozen plasma, SDPC: Single donor plasma concentrate, SD: Standard Deviation)

All values are represented as mean ± SD

There was no significant differences between the groups with respect to surgical duration, fluid administration (crystalloids, albumin), blood product usage, graft weight, blood loss, or urine output. Group H had a significantly lower lactate level on shifting (2.7 vs 3.7 mmol/L, P=0.04). Pleural fluid drainage was higher in Group H (P=0.05), nearing significance. [Table 3]

Table 4: Comparison of preoperative and postoperative IAP

Parameters	Group H (n= 31)	Group NH (n= 6)	P- value
Preop IAP	14.8 ± 2.56	10.33 ± 2.33	0.03
IAP on POD 1	10.81 ± 2.25	10.17 ± 2.40	0.53
IAP on POD 2	10.19 ± 2.31	10 ± 2.60	0.85
IAP on POD 3	9.77 ± 2.14	9.67 ± 2.25	0.91

(IAP: Intra-abdominal pressure)

All values are represented as mean ± SD

Preoperative IAP was significantly higher in Group H (14.8 vs 10.33 mmHg, P=0.03). Postoperative IAP on POD 1–3 showed no significant intergroup differences. [Table 4]

Table 5: Postoperative Renal, graft function and ICU outcome parameters

Parameter	Group H (n=31)	Group NH (n=6)	p-value

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Serum Creatinine (mg/dL, mean ± SD)			
POD 1	0.71 ± 0.20	0.72 ± 0.27	0.94
POD 2	1.27 ± 0.86	0.68 ± 0.32	0.66
POD 3	0.64 ± 0.21	0.60 ± 0.27	0.67
Urine Output (mL/kg/h, mean ± SD)			
POD 1	0.98 ± 0.31	0.87 ± 0.13	0.41
POD 2	0.83 ± 0.16	0.83 ± 0.16	0.92
POD 3	0.74 ± 0.13	0.70 ± 0.11	0.49
Acute Kidney Injury (AKI), n (%)			0.15
Present	11 (35.5%)	4 (66.7%)	
Absent	20 (64.5%)	2 (33.3%)	
Early Allograft Dysfunction (EAD), n (%)			0.50
Present	11 (35.5%)	3 (50.0%)	
Absent	20 (64.5%)	3 (50.0%)	
Portal Vein Flow (L/min, mean ± SD)			
POD 1	2.23 ± 0.86	2.47 ± 0.80	0.54
POD 2	2.14 ± 0.57	2.51 ± 0.73	0.17
POD 3	2.15 ± 0.58	2.40 ± 0.85	0.38
POD 4	2.03 ± 0.50	2.01 ± 0.60	0.86
POD 5	2.04 ± 0.52	2.01 ± 0.32	0.91
POD 6	2.10 ± 0.36	2.03 ± 0.51	0.62
POD 7	2.02 ± 0.36	1.91 ± 0.35	0.52
Mechanical Ventilation & Vasopressors			
Duration of mechanical ventilation (hr)	12.06 ± 5.07	15.00 ± 10.82	0.29
Cumulative Noradrenaline dose (mcg)	37389 ± 35209.85	19441 ± 26617.70	0.27
Cumulative Vasopressin dose (U)	36.53 ± 36.02	18.26 ± 21.71	0.24
Vasoactive days	2.26 ± 1.60	1.50 ± 0.83	0.24
ICU Course			
ICU Length of Stay (days)	10.29 ± 5.58	9.33 ± 3.50	0.69
ICU Survival Outcome, n (%)			0.35
Alive	27 (87.1%)	6 (100%)	0.35
Dead	4 (12.9%)	0 (0%)	

(SD: Standard Deviation)

Serum creatinine and urine output values were comparable between groups on POD 1–3. Incidence of AKI and EAD was higher in Group NH, though not statistically significant. Portal vein flow was comparable throughout POD 1–7. No significant differences in mechanical ventilation duration, vasopressor use, or ICU stay. ICU survival was slightly higher in Group NH (100% vs 87.1%), but this was not statistically significant (p=0.35). [Table 5]

DISCUSSION

This prospective observational study provides valuable insights into the incidence and implications of intra-abdominal hypertension (IAH) in adult patients undergoing living donor liver transplantation (LDLT). Our findings reveal a high preoperative prevalence of IAH (83.7%) in this population, with the majority of patients falling into Grade I and II categories. Despite the high incidence, the postoperative outcomes between those with IAH (Group H) and without IAH (Group NH) showed limited statistically significant differences, highlighting the complex interplay between IAH and other perioperative factors.

The observed high preoperative IAH prevalence underscores the pathophysiological burden faced by end-stage liver disease patients. Factors such as massive ascites, bowel congestion due to portal hypertension, and reduced abdominal compliance contribute to elevated intra-abdominal pressures. This aligns with previous literature that reports similar findings in critically ill patients and paediatric transplant cohorts.[10,11]

Importantly, IAH has been recognized as a potentially modifiable risk factor in the perioperative period. In our study, the significant reduction in IAP from the preoperative to postoperative period (mean IAP from 14.8 mmHg to <10 mmHg by POD 3 in both groups) likely reflects intraoperative decompression strategies such as ascites evacuation, improved hemodynamic control, and postoperative volume management. This finding is consistent with prior studies where aggressive intraoperative fluid removal and careful volume resuscitation resulted in normalization of IAP after liver transplantation.[12]

While patients with preoperative IAH demonstrated trends toward higher postoperative serum creatinine and incidence of AKI (35.5% vs 66.7%), these differences did not reach statistical significance. This may be due to the small sample size in the non-IAH group (n = 6), limiting statistical power. Nonetheless, the clinical trend supports existing literature on the deleterious renal effects of elevated intra-abdominal pressure, which can reduce renal perfusion and glomerular filtration rate.[1] The role of IAH in the development of early allograft dysfunction (EAD) was less definitive, with similar rates in both groups (35.5% vs 50%), suggesting that other factors—such as graft quality, ischemia-reperfusion injury, and intraoperative hemodynamics—may play a more dominant role in determining graft performance.

Interestingly, despite slightly higher noradrenaline and vasopressin requirements, and a longer mean duration of mechanical ventilation in the IAH group,

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these differences were not statistically significant. This again may be attributed to small sample size or effective intraoperative and ICU management strategies that mitigated the impact of elevated IAP. The ICU length of stay and survival outcomes were comparable between the two groups, indicating that IAH, while prevalent, did not independently predict adverse outcomes in this cohort.

Of particular note is the lack of significant differences in portal vein flow postoperatively between the groups. This suggests that decompressive interventions and hemodynamic optimization were likely effective in maintaining graft perfusion, despite the presence of IAH in the preoperative period.

Our study adds to the limited body of evidence on IAH in adult LDLT, especially within the Indian subcontinent, where LDLT is the predominant transplant modality. The findings emphasize the importance of routine monitoring of intra-abdominal pressure in the perioperative period. Early identification and timely intervention may help prevent complications such as AKI, prolonged mechanical ventilation, or hemodynamic instability.

Limitations

The study is limited by its relatively small sample size, especially in the non-IAH group (n = 6), which restricts the generalizability of our findings and reduces the power to detect statistically significant differences. Additionally, the observational nature of the study precludes establishment of causality. The lack of long-term follow-up data prevents evaluation of the prolonged impact of IAH on graft survival and patient outcomes. Future studies with larger sample sizes and multicentric data are warranted.

CONCLUSION

Intra-abdominal hypertension is highly prevalent among adult patients undergoing living donor liver transplantation, with most cases presenting as Grade I or II. Although its presence was associated with trends toward higher rates of acute kidney injury, early allograft dysfunction, and increased vasoactive support, these differences were not statistically significant in our cohort, likely due to limited sample size. The significant postoperative reduction in intra-abdominal pressure highlights the benefit of intraoperative decompression and meticulous perioperative fluid management. Routine intra-abdominal pressure monitoring and timely interventions remain crucial in optimizing perioperative outcomes. Larger, multicentric studies are warranted to clarify the prognostic significance of IAH in this population and to guide standardized management protocols.

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Conflict of Interest Statement

The authors declare no conflicts of interest relevant to this work. No financial or non-financial support was received that could be perceived as influencing the study outcomes.

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