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Renal Resistive Index and Myocardial Performance Index for Early Prediction of Acute Kidney Injury after Living Donor Liver Transplant

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ABSTRACT

Background: Acute kidney injury (AKI) frequently arises as a complication after living donor liver transplantation (LDLT), affecting patient outcomes. **Objective:** This research assessed how well renal resistive index (RRI) and myocardial performance index (MPI) predict early postoperative AKI in LDLT recipients.

Methods: A prospective observational study was conducted with 104 adult LDLT recipients at Mansoura University from September 2022 to June 2024. RRI and MPI were measured preoperatively and postoperatively using transabdominal sonography and transthoracic echocardiography respectively. AKI was determined according to the criteria set by the International Club of Ascites. The primary outcome was early postoperative AKI incidence, with secondary outcomes including RRI and MPI's predictive accuracy.

Results: Of 104 patients, 64 (61.5%) developed AKI. Postoperative RRI was significantly higher in the AKI group (0.744 ± 0.06 vs. 0.653 ± 0.05, $p < 0.001$), with a cutoff of ≥ 0.695 predicting AKI (sensitivity 82.8%, specificity 82.5%, AUC 0.891). Postoperative MPI was also higher (0.321 ± 0.04 vs. 0.282 ± 0.04, $p<0.001$), with a cutoff of ≥0.286 (sensitivity 85.9%, specificity 57.5%, AUC 0.745). Preoperative RRI was predictive (cutoff ≥0.655, AUC 0.714), but preoperative MPI was not. Risk factors included higher BMI, diabetes, and intraoperative hypotension.

Conclusion: RRI and MPI, particularly postoperative measurements, were effective predictors of early postoperative AKI in LDLT recipients, with RRI showing superior accuracy. These findings suggest that perioperative RRI and MPI monitoring could enhance AKI risk stratification and management, warranting further validation in larger studies.

Keywords: Liver Transplantation, RRI, MPI, AKI, LDLT, DDLT.

INTRODUCTION

Liver transplantation (LT) has been established as the sole effective therapeutic intervention for patients afflicted with end-stage liver disease (ESLD), irrespective of its diverse etiologies, which include viral hepatitis, various metabolic disorders, and specific hepatic malignancies. Globally, living donor liver transplantation (LDLT) has undergone rapid advancement and is now firmly recognized as a safe and ethically acceptable alternative to deceased donor liver transplantation (DDLT). In the Egyptian context, the establishment of a DDLT program remains pending due to persistent legal limitations, thereby positioning LDLT as the singular viable therapeutic avenue for patients contending with ESLD [(1)]. LT itself constitutes a highly extensive surgical undertaking, with numerous physiological stressors and procedural factors contributing to the heightened propensity for acute kidney injury (AKI) development during the perioperative period [(2)].

The occurrence of kidney injury carries a significant and detrimental impact on both the early post-operative course and the long-term outcomes following LT. Although there have been major advancements in recent decades that have improved graft-related outcomes and overall patient survival after LT, the morbidity and mortality linked to post-LT AKI, as well as its troubling incidence among these highly vulnerable patients, regrettably remain significantly high. Indeed, the reported incidence of post-LT AKI can be as high as 95% in certain documented series [(3)]. More specifically, a study undertaken at the National Liver Institute in Egypt, focusing on a cohort of 167 recipients of LDLT documented an incidence rate of renal impairment at 21% [(4)]. This localized epidemiological data provides a crucial perspective on the burden of kidney complications within this specific patient population, highlighting a significant proportion of recipients experiencing renal dysfunction in a prominent regional transplantation center [(4)].

The internationally recognized Kidney Disease Improving Global Outcomes (KDIGO) criteria, and foundational for Acute Kidney Injury (AKI) diagnosis, primarily rely on measurements of blood creatinine and urine volume. However, in the context of post-liver transplantation (LT), neither of these indicators consistently demonstrates sufficient sensitivity to promptly reflect a decrease in glomerular filtration rate. This inherent limitation frequently results in a significant lag in the timely recognition of post-LT AKI. Consequently, there is a pressing clinical necessity to develop and establish more sensitive early detection models for post-LT AKI, ideally incorporating additional, more responsive clinical risk factors [(5)].

A marked intrarenal vasoconstriction is one of the main pathophysiological features underlying AKI. This pathological process directly leads to substantial renal

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hypoperfusion, which in turn precipitates the typical clinical signs of AKI, which comprise oliguria and increased serum creatinine levels [6].

In this regard, the Renal Resistive Index (RRI), a readily measurable parameter obtained from renal artery Doppler ultrasonography, proves particularly valuable. RRI effectively demonstrates the intricate microvascular and macrovascular interactions between the systemic arterial system and the renal vasculature. Notably, elevated RRI values (typically defined as > 0.7) are consistently liked to a higher incidence of adverse cardiovascular events and accelerated progression of renal failure [7]. Beyond being directly influenced by intrarenal vascular resistance, RRI is also sensitive to broader systemic hemodynamic parameters, such as heart rate (HR) and the functional status of left ventricular systolic and diastolic mechanics [8].

The Myocardial Performance Index (MPI) represents a significant contribution to comprehensive cardiovascular risk assessment, primarily owing to its unique capability in detecting the early, subclinical stages of both diastolic and systolic dysfunctions. Distinct from isolated measurements, MPI offers a robust and unified metric that reflects overall global cardiac function [9]. Its utility has been extensively investigated across a spectrum of cardiac conditions, including but not limited to heart failure, myocardial infarction, and hypertension, where it has consistently demonstrated a strong association with adverse cardiovascular mortality outcomes [10].

AIM OF THE STUDY

Given the critical need for early AKI prediction in vulnerable patients, this study specifically intended to examine if a joint evaluation of Doppler Renal Resistive Index (RRI), evaluated via transabdominal sonography, and Myocardial Performance Index (MPI), measured through transthoracic echocardiography, may serve as effective predictors of early postoperative acute kidney injury in recipients undergoing living donor liver transplantation (LDLT).

MATERIALS AND METHODS

This prospective observational study included 104 cases who underwent LDLT at the Liver Transplantation Unit, Mansoura University, Mansoura, Egypt, between September 2022 and June 2024. We followed the Standards for Reporting Diagnostic Accuracy Studies (STARD) guidelines when preparing this manuscript [11].

Inclusion criteria: Adult recipients of either sex, aged 18-65. Patients with preoperative renal impairment (glomerular filtration rate [GFR] < 60 mL/min/1.73 m^2), renal artery stenosis, nephrectomy, coronary artery disease, severe valvular heart disease, and cardiac arrhythmia.

Patients care and management was conducted according to our institutional preoperative, intraoperative and postoperative protocols without any modification.

Preoperative assessment: The preoperative evaluation of potential liver transplant recipients is a meticulous process, systematically structured into four distinct phases to ensure comprehensive assessment of the patient's suitability for surgery and to mitigate risks:

- **Phase I** encompassed a detailed laboratory evaluation, which rigorously included the assessment of tumor markers to screen for potential hepatocellular carcinoma or other malignancies, alongside a thorough virological evaluation to ascertain the presence and activity of relevant infectious agents. This phase also involved a comprehensive radiological evaluation, typically utilizing cross-sectional imaging techniques, to precisely delineate the extent of liver disease, assess vascular anatomy, and identify any extrahepatic pathologies.
- **Phase II** focused on a detailed cardiopulmonary and neuro-psychiatric evaluation. The cardiopulmonary assessment is crucial to ascertain the patient's cardiac and pulmonary reserve, ensuring they can withstand the significant hemodynamic shifts and physiological stress associated with extensive transplantation surgery. The neuro-psychiatric evaluation aimed to identify any cognitive impairments, psychological conditions, or substance use disorders that could impact surgical candidacy, adherence to complex post-transplant regimens, and overall recovery.
- **Phase III** involved a thorough endoscopic evaluation, specifically including both upper and lower gastrointestinal tract endoscopy. This is performed to screen for and address potential complications of end-stage liver disease, such as esophageal or gastric varices, as well as to identify any other gastrointestinal pathologies that might require intervention or influence the surgical approach.
- **Phase IV** consisted of routine general consultations with various specialists. The primary objective of this phase was to meticulously exclude any possible hidden septic foci throughout the body, such as dental infections, urinary tract infections, or skin lesions. Identifying and treating these occult infections preoperatively is paramount to minimize the risk of severe post-transplant infectious complications, which can significantly impact patient morbidity and mortality.

Patients' preparation and anesthesia: Patients were subjected to a fasting regimen for solid foods for a minimum of 6 hours prior to the operative procedure, while being permitted to consume clear liquids freely up to 4 hours preoperatively. To mitigate the risk of dehydration during this fasting interval, an intravenous

infusion of 500 ml Ringer's acetate solution was administered.

For the induction of general anesthesia, a standardized intravenous protocol was employed, comprising fentanyl at a dose of 2 mcg/kg for analgesia, propofol at 1-2 mg/kg for induction of hypnosis, and rocuronium bromide at 0.8-1 mg/kg to facilitate neuromuscular blockade for intubation. Anesthesia maintenance was achieved through the administration of sevoflurane, delivered in a mixture of 40-60% oxygen, complemented by continuous intravenous infusions of fentanyl at 0.5 mcg/kg/h for sustained analgesia and rocuronium bromide at 200-400 mcg/kg/h to maintain muscle relaxation.

To ensure patient safety and optimize physiological conditions, core body temperature was meticulously maintained within normothermic ranges using forced-air warming blankets. Comprehensive standard physiological monitoring was continuously applied throughout the procedure, including oxygen saturation via pulse oximetry, continuous electrocardiogram (ECG) for cardiac rhythm assessment, body temperature measured by a nasopharyngeal probe, end-tidal carbon dioxide measured by capnography to assess ventilation, and the placement of an arterial catheter for continuous invasive arterial blood pressure monitoring and frequent arterial blood gas analysis. Furthermore, a central venous catheter was inserted for continuous assessment of central venous pressure (CVP), providing crucial insights into fluid status and right heart function.

Perioperative fluid management: Our center consistently employs a **goal-directed fluid protocol** during LDLT procedures, meticulously targeting a mean arterial pressure (MAP) of ≥ 65 mmHg to optimize organ perfusion. **Ringer's acetate** served as the primary maintenance intravenous solution throughout the perioperative period. Patients exhibiting a stroke volume variation (SVV) greater than 10% were systematically identified as fluid responders and promptly received boluses of 200 mL of 4% albumin in Ringer's acetate. The decision for **red blood cell (RBC) transfusion** was guided by a hemoglobin concentration threshold of 7–8 g/dL, applied in conjunction with dynamic clinical judgment. In cases of fluid non-responsiveness and persistent hypotension, norepinephrine infusion was initiated to support vascular tone. Meticulous control of random blood glucose levels was maintained between 110 mg/dL and 180 mg/dL through titrated intravenous insulin infusion or boluses of 10% or 25% glucose solution, as clinically appropriate. Furthermore, serum potassium (K^+) and ionized calcium (Ca^{2+}) levels were rigorously monitored and promptly corrected when necessary, particularly around the critical phase of graft reperfusion. **Hypotension** was stringently defined as a 20% reduction below the patient's basal MAP, while **Post-Reperfusion Syndrome** was characterized by a rapid 30% drop in MAP compared to the basal reading, sustained for at least 1 minute within 5 minutes after portal vein unclamping.

For acute hypotensive episodes in both designated patient groups, management involved the rapid infusion of 500 mL of 4% albumin or packed RBCs (Depending on the hemoglobin concentration during the anhepatic phase), alongside 20 mcg norepinephrine boluses, with subsequent continuous infusion if required. Incremental boluses of 10 mcg epinephrine were administered if the MAP remained below 65 mmHg after 1 minute of initial intervention. During the intensive care unit (ICU) stay, a daily **zero fluid balance** was targeted to prevent fluid overload, with fluid status predominantly maintained by Ringer's acetate and 10% glucose solution, alongside encouragement of early oral fluid intake from the first postoperative day. Albumin supplementation was strategically administered to ensure serum albumin levels were consistently maintained at ≥ 3.0 g/dL, supporting oncotic pressure and fluid distribution.

Intraoperative and ICU urine output: Urine output was monitored hourly during surgery. If urine output fell below 0.5 ml/kg/h and fluid status was adequate, furosemide 5 mg (IV) was given. In the ICU, if urine output remained below 0.5 ml/kg/h for two hours, furosemide 5-10 mg (IV) was administered and re-evaluated every 6 hours.

Postoperative care: All patients were transferred to the Intensive Care Unit (ICU) post-surgery. Early tracheal extubation in the ICU was performed once hemodynamic stability was confirmed (MAP > 65 mmHg, heart rate < 100 bpm, SpO_2 > 96% on 0.4 FiO_2), along with pH > 7.3, adequate consciousness, and muscle strength.

Immunosuppression: Intraoperatively: Patients received methylprednisolone 0.5 gm (IV) at the start of warm ischemia. Mycophenolate mofetil 500 mg via nasogastric tube and basiliximab 20 mg (IV) were given after hepatic artery anastomosis and declamping. In the ICU: Oral tacrolimus was started on postoperative day one, with doses adjusted to target serum levels of 5-10 ng/ml. Mycophenolate mofetil 500 mg was initiated four days post-operation. If acute kidney injury (AKI) was diagnosed, tacrolimus was temporarily replaced with methylprednisolone until kidney function normalized.

Diagnostic criteria for early post-liver transplant AKI: AKI was defined by the International Club of Ascites' revised classification for cirrhotic patients: a 0.3 mg/kg increase in serum creatinine within the first 48 postoperative hours. AKI severity was categorized into stages:

- Stage 1: Serum creatinine increase 1.5-1.9 times baseline, or increase more than 0.3 mg/dL.

- Stage 2: Serum creatinine increase 2-2.9 times baseline.
- Stage 3: Serum creatinine increase 3 times baseline, or increase to 4 mg/dL, or initiation of renal replacement therapy.

Imaging: Transthoracic echocardiography and transabdominal ultrasonography were performed at three time points: before anesthesia induction, after surgery completion (Before ICU transfer), and daily for the first seven postoperative days.

Transabdominal renal Doppler protocol: Doppler measurements were performed using a Portable Ultrasound Scanner (Toshiba Xario 200, 3.5 MHz abdominal probe). Three Doppler samples were taken from the kidney's upper, middle, and lower poles. Flow and Resistive Index (RRI) were noted at the interlobar or arcuate arteries. RRI was calculated using the formula: RRI = (peak systolic velocity − end diastolic velocity) / peak systolic velocity. The mean RRI was then derived from these three measurements. Finally, the heart rate at the time of RRI calculation was recorded to apply Mostbeck's heart rate correction: Corrected RRI = (observed RRI) − (0.0026 × (80 − heart rate)).

Transthoracic Echocardiography: Following American Heart Association guidelines, examinations were conducted with a Toshiba Xario 200 echocardiography device. Tissue Doppler images were recorded at 100 mm/s from the lateral mitral annulus. Isovolumetric contraction time (IVCT) was measured from the end of the A' wave to the beginning of the S' wave. Isovolumetric relaxation time (IVRT) was measured from the end of the S' wave to the beginning of the E' wave. Ejection time (ET) was determined by subtracting IVCT and IVRT from the total non-filling time. The Myocardial Performance Index (MPI) was calculated as: (isovolumetric contraction time + isovolumetric relaxation time) / ejection time. An MPI of ≥ 0.5 was considered abnormal, indicating subclinical left ventricular dysfunction.

Sample Size: Based on a previous study at our center indicating a 25% prevalence of acute kidney injury after living donor liver transplant, a sample size of 100 cases was determined. This sample size would achieve an Area Under the Receiver Operating Characteristic Curve (AUC) of 0.918, with a 95% confidence interval ranging from 0.850 to 0.986. The sample size calculation was performed using the UCSF website's calculators.

Ethical consideration: The research was approved by The Local Institutional Review Board (IRB) (MD.22.09.699) on September 28, 2022, and was subsequently registered on Clinical Trials (NCT05666232). All participants provided written informed consents. Confidentiality and personal privacy were respected in all levels of the study. Throughout its implementation, the study complied with the Helsinki Declaration.

Data collection

Preoperative data included patient demographics, primary diagnosis, disease-specific scores (Child-Turcotte-Pugh, MELD), comorbidities (Diabetes, hypertension, thyroid issues, cardiac diseases), 24-hour urine output, diuretic use, and blood/urine biochemistry. Intraoperative data encompassed ascetic fluid volume, transfused crystalloids and colloids, blood product transfusions, cell saver use, mean arterial pressure, central venous pressure, hypotensive episodes, urine output, diuretic use, anhepatic phase duration, cold and warm ischemia times, occurrence of post-reperfusion syndrome, and vasopressor/inotrope doses during surgery and at ICU transfer, as well as arterial blood gases and hemoglobin levels at each stage. Postoperative data included duration of mechanical ventilation, inotropic support, blood product transfusions, total urine output, six-hour urine output intervals, weekly tacrolimus blood levels for one-month, daily blood counts, liver and renal function tests, and arterial blood gas measurements. RRI and MPI were recorded preoperatively, before ICU transfer on the day of surgery, and daily for the first seven postoperative days. Outcome variables, including the necessity for renal replacement therapy, duration of ICU stay, and three-month hospital mortality were also recorded.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as numbers and percentages, while continuous variables were tested for normality using the Shapiro-Wilk test and then expressed as mean ± standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for non-normally distributed data.

Comparative analyses between the acute kidney injury (AKI) and non-AKI groups were performed using the independent samples t-test for normally distributed continuous variables, the Mann-Whitney U test for non-normally distributed variables, and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. The Monte Carlo correction was applied for Chi-square tests with expected frequencies <5.

Receiver Operating Characteristic (ROC) curve analysis was employed to evaluate the diagnostic accuracy of both the Renal Resistive Index (RRI) and Myocardial Performance Index (MPI) for predicting early postoperative AKI. The area under the curve (AUC) was calculated, and cut-off values were determined using the Youden index to maximize sensitivity and specificity.

Statistical significance was set at a p-value < 0.05 for all tests.

RESULTS

Among the 115 recipients screened for eligibility, 104 consecutive patients undergoing LDLT were included herein as shown in the flow diagram (Fig.1). A total number of 64 patients developed AKI. Table (1) showed the basic characteristics and demographic data.

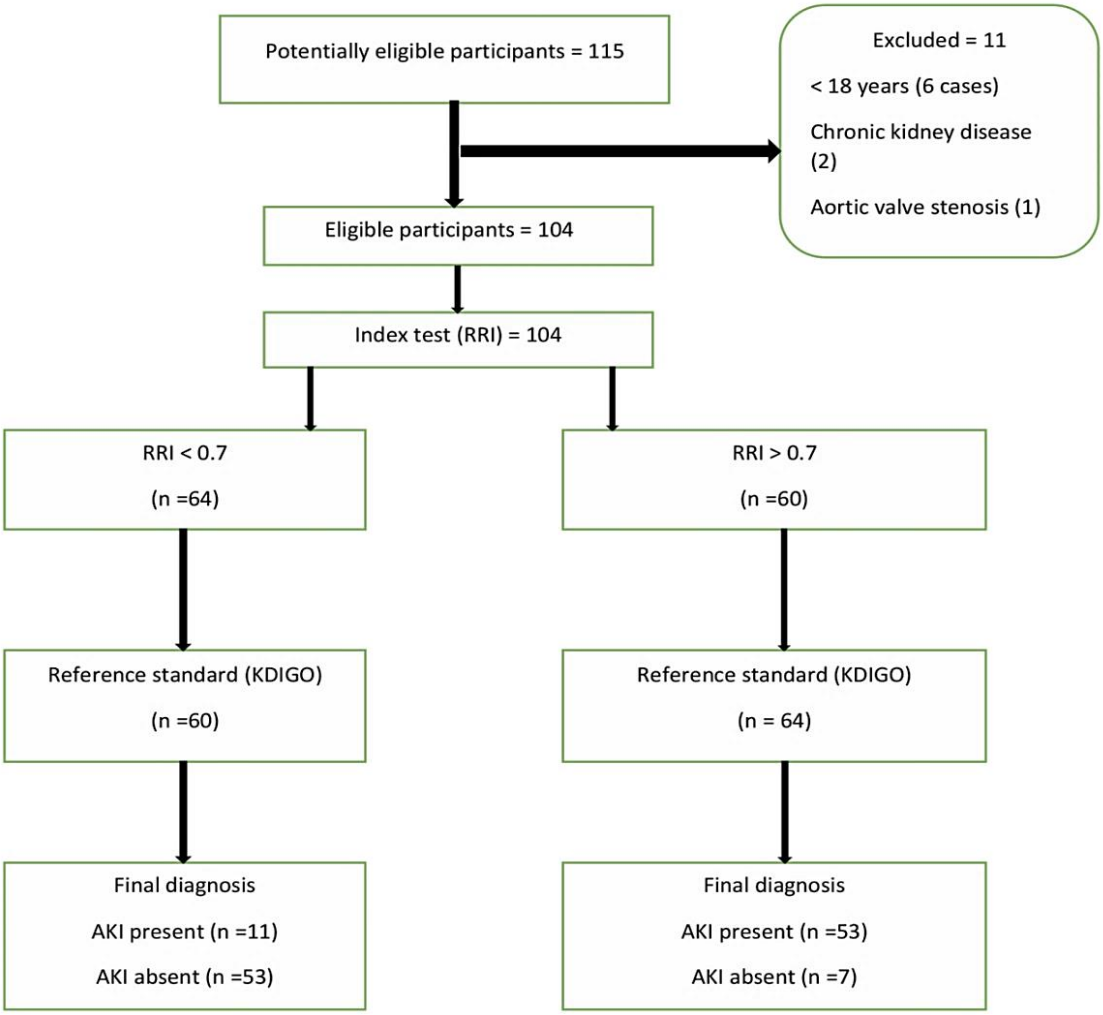

Figure (1): STARD flowchart diagram.

Demographic data and basic characteristics: The AKI group showed significant difference from non-AKI group as regarding weight (82.16 ± 15.77 kg vs76.25 ± 11.14 kg, p=0.04). Recipients' BMI was significantly higher in AKI group (29.03 ± 5.29 kg/m2 vs 26.55 ± 3.72 kg/min, p=0.01). Similarly, Donors' BMI was significantly higher in AKI group (27.03 ± 2.11 kg/m^2 vs 23.08 ± 2.77 kg/m^2, p<0.0001). Recipients with encephalopathy history were significantly higher in AKI group (54.7% vs 10%, p=0.001). Diabetes was more prevalent in AKI group (39% vs 10%, p=0.004). MELD Na score was significantly higher among AKI group (16 ± 4.8 vs 14 ± 4.7, p=0.05). The two groups were comparable as regarding age, sex, CTP score and the primary diagnosis as shown in table (1).

Table (1): Comparison of demographic data and history between studied groups

	No AKI group (n=40)	AKI group (n=64)	Test of significance
Age / years	47.62±13.82	51.92±9.77	t=1.86 p=0.07
Gender **Male** **Female**	 31(77.5) 9(22.5)	 50(78.1) 14(21.9)	 χ^2=0.006 P=0.940
Marital status **Single** **Married**	 6(15) 34(85)	 4(6.2) 60(93.8)	 χ^2=2.17 P=0.141
Weight (kg)	76.25±11.14	82.16±15.77	t=2.07 p=0.04*
Height (cm)	169.48±6.76	168.19±7.40	t=0.891 p=0.375
BMI(Kg/m^2)	26.55±3.72	29.03±5.29	t=2.58 p=0.01*
MELD score	12.2±4.19	12.88±4.24	t=0.793 p=0.429
MELD -Na score	14.60±4.72	15.55±4.95	t=0.967 p=0.336
CTP score **A** **B** **C**	 5(12.5) 15(37.5) 20(50.0)	 9(14.1) 23(35.9) 32(50.0)	 χ^2=0.061 P=0.970
Primary diagnosis **HCC** **Cirrhosis** **Caroli disease**	 8(20) 31(77.5) 1(2.5)	 20(31.2) 44(68.8) 0	 χ^2=3.02 P=0.221
Comorbidities **No** **Yes** **Diabetes** **Hypertension**	 27(67.5) 13(32.5) 6(10) 5(8.3)	 35(54.7) 29(45.3) 25(39) 11(17.2)	 χ^2=1.68 P=0.195 P=0.004* P=0.4
Donor BMI	23.08±2.77	27.03±2.11	t=8.21 p<0.0001*
Encephalopathy history	4 (10)	35 (54.7)	χ^2=20.97 P=0.001*
Ascites No Minimal /Mild Moderate Marked	 16(40) 12(30) 9(22.5) 3(7.5)	 17(26.6) 21(32.8) 16(25.0) 10(15.6)	 χ^2=2.82 P=0.419
Lower limb oedema **No** **Grade 2**	 36(90) 4(10)	 50(78.1) 14(21.9)	 χ^2=2.42 P=0.119

Data presented as mean and SD or numbers. t: Student t test, FET: Fisher exact test, χ^2=Chi-Square test *statistically significant.

Preoperative labs and sonographic parameters: RRI was significantly higher in AKI group (0.677 ± 0.07 vs 0.634 ± 0.04, p=0.001). Pulsatility index (PI) was significantly elevated in AKI group (1.44 ± 0.26 vs 1.25 ± 0.35, p=0.002). Myocardial performance index was higher but not significantly different from non-AKI group (0.267 ± 0.04 vs 0.266 ± 0.047, p=0.222). The lab values were comparable between the groups (Table 2).

Table (2): Preoperative sonographic and laboratory data between two groups

	No AKI group (n=40)	AKI group (n=64)	Test of significance
MPI	0.266±0.047	0.276±0.04	t=1.23 p=0.222
LVOT VP (cm/s)	100.30±15.06	100.98±11.44	t=0.261 p=0.795
LVOT VTI (cm)	21.89±3.81	21.878±3.78	t=0.036 p=0.971
LVOT PPG (mmHg)	4.12±1.32	4.22±0.988	t=0.429 p=0.669
LVOT MPG (mmHg)	2.03±0.78	2.07±0.607	t=0.290 p=0.772
LVOT Diam. (cm)	2.09±0.199	2.13±0.21	t=1.08 p=0.284
LVOT SV (ml)	75.83±18.35	78.59±17.92	t=0.757 p=0.451
LVOT CO (L/min)	5.71±1.32	5.91±1.52	t=0.687 p=0.494
IRVF Pattern **Biphasic** **Continuous** **Pulsatile**	 0 35(87.5) 5(12.5)	 4(6.2) 46(71.9) 14(21.9)	 Mc=4.46 P=0.108
RRI	0.634±0.04	0.677±0.07	t=3.43 p=0.001*
PI	1.25±0.35	1.44±0.26	t=3.17 p=0.002*
e.GFR	109.24±19.11	102.77±16.32	t=1.84 p=0.07
24h UOP (ml/kg)	0.802±0.156	0.771±0.19	t=0.877 p=0.383
MAP (mmHg)	84.95±5.31	86.31±4.47	t=1.41 p=0.163
Pulse pressure (mm/Hg)	33.92±3.05	36.86±5.03	t=3.32 p=0.001*
Heart rate (Beat/min)	76.75±11.14	75.45±8.99	t=0.652 p=0.516
HB(gm/dl)	10.37±2.06	9.92±1.97	t=1.11 p=0.269
WBCS ($\times 10^3/\mu L$)	3.78±0.89	4.08±0.86	t=0.591 p=0.556
Platelet ($\times 10^3/\mu L$)	75(52.3-103.5)	68(51.23-91)	z=0.431 p=0.666
Serum creatinine (mg/dl)	0.718±0.15	0.753±0.16	t=1.11 p=0.269
Uric acid (mg/dl)	4.22±1.18	4.90±1.97	t=1.98 p=0.05*
Albumin (g/dl)	3.24±0.70	3.01±0.57	t=1.83 p=0.071
AST (IU/L)	43(29-54.5)	40.5(32-54)	z=0.321 p=0.748
ALT (IU/L)	31(21-41.75)	18(21-46)	z=0.057 p=0.955
Total bilirubin (mg/dl)	1.5(1.05-2.73)	1.95(1.03-3.18)	z=1.33

	No AKI group (n=40)	AKI group (n=64)	Test of significance
			p=0.183
INR	1.42±0.35	1.43±0.29	t=0.189 p=0.850
APTT (seconds)	44.92±5.33	45.59±6.55	t=0.543 p=0.589
CRP level for positive cases (mg/L)	12.5(7-21.25)	13.5(11-21.75)	Z=0.695 P=0.487
LDH (IU/L)	133(108.75-151.75)	130.5(111.25-193.5)	Z=0.635 P=0.526
LACTATE (mmol/L)	1.2(1-1.48)	1.3(1-1.7)	Z=1.38 P=0.168
GGT (IU/L)	25.5(13-60.5)	27.5(18-55)	Z=0.658 P=0.510
ALP (IU/L)	5(5-6.75)	5(5-7)	Z=0.309 P=0.757
AMYLASE(IU/L)	21(18-27)	24 (19-34)	Z=1.92 P=0.06
Na (mEq/L)	135.82±3.18	134.33±4.09	t=1.97 p=0.051
K (mEq/L)	3.79±0.47	3.82±0.52	t=0.308 p=0.759
Ca (mg/dl)	8.13±0.48	8.09±0.29	t=0.383 p=0.702
Mg (mg/dl)	1.34±0.27	1.38±0.29	t=0.699 p=0.486
Ph (mg/dl)	3.44±0.84	3.51±0.84	t=0.418 p=0.677
Diuretic use (mg)	12 (30)	28 (43.8)	χ^2=1.97 P=0.214
RBCs units (No)	1.0 (0-2.75)	3.0 (1.0-5.0)	Z=3.19 P=0.001*

Data presented as mean and SD, median and range or numbers. est χ^2 =: Chi -Square test *statistically significant. Z: Mann Whitney U test, t: Student t test, MC: Monte Carlo test.

LVOT: Left Ventricular Outflow Tract**, VP:** Velocity Peak**, VTI:** Velocity Time Integral**, PPG:** Peak Pressure Gradient**, MPG:** Mean Pressure Gradient**, Diam.:** Diameter**, SV:** Stroke Volume**, CO:** Cardiac Output**, IRVF Pattern:** Intrarenal Venous Flow Pattern**, MELD score**: Model for End-Stage Liver Disease Score, **MELD -Na score: MELD** Score adjusted for Serum Sodium.
eGFR: estimated Glomerular Filtration Rate, **24h UOP**: 24-hour Urine Output, **MAP**: Mean Arterial Pressure, **HB**: Hemoglobin, **WBCs**: White Blood Cells, **Platelet**: Platelet Count, **Albumin**: Albumin, **AST**: Aspartate Aminotransferase, **ALT**: Alanine Aminotransferase, **Total bilirubin**: Total Bilirubin, **INR**: International Normalized Ratio, **APTT**: Activated Partial Thromboplastin Time, **CRP level for positive cases**: C-Reactive Protein, **LDH**: Lactate Dehydrogenase, **GGT**: Gamma-Glutamyl Transferase, **ALP**: Alkaline Phosphatase, **Na**: Sodium, **K**: Potassium, **Ca**: Calcium, **Mg**: Magnesium.

Intraoperative variables: Total operative time was significantly prolonged in AKI group (563.67 ± 96.26 min vs 512 ± 72.75 min, p=0.004). Durations of anhepatic phase, cold ischemia and warm ischemia time were higher but not significantly elevated in AKI group. There was a significant blood loss in AKI group (7242.19 ± 3209.41 ml vs 4867.5 ± 1458.72 ml, p=0.001). Therefore, number of RBCs units transported was significantly higher among AKI patients. Occurrence of post reperfusion syndrome (PRS) was more significant in AKI group (70.3% vs 30%, p=0.001). Serum lactate measured post-reperfusion was significantly higher in AKI group. Duration of hypotension was significantly prolonged and mean arterial blood pressure was significantly lower in AKI group. Total amount of vasopressor infused was significantly higher in AKI group (Table 3).

Table (3): Intraoperative data between the two groups

Intra-operative data	No AKI group (n=40)	AKI group (n=64)	Test of significance
Total operative time (min)	512±72.75	563.67±96.26	t=2.91 p=0.004*
Anhepatic phase (min)	71.55±16.39	76.2±14.69	t=1.50 p=0.136
Cold ischemia (min)	29.08±11.08	32.19±13.35	t=1.23 p=0.221
Warm ischemia (min)	38.68±10.35	38.8±9.43	t=0.062 p=0.951
Blood loss (ml)	4867.5±1458.72	7242.19±3209.41	t=4.39 p=0.001*
Ascites **No** **Minimal/mild** **Moderate** **Marked**	 16(40) 12(30) 9(22.5) 3(7.5)	 17(26.6) 21(32.8) 16(25) 10(15.6)	 χ^2=2.83 P=0.419
PRS (yes/no)	12(30)	45(70.3)	χ^2=16.15 P=0.001*
Lactate (PRS)(IU/L)	3.35(2.53-4.4)	4.6(3.5-6.1)	Z=3.49 P=0.001*
Lowest MAP (mmHg)	68(57.25-72)	55.5(52.25-66.75)	Z=3.95 P=0.0001*
Duration of Hypotension (min)	3.0(2.0-3.0)	5.0(4.0-6.0)	Z=7.23 P=0.001*
Vasopressor (ng/kg/min)	25(18-42)	53(32-99.5)	Z=3.85 P=0.001*
Purge amount (ml)	200(200-400)	300(200-400)	Z=1.27 P=0.205
Graft weight(mg)	931(862-986.75)	928(848-1006)	Z=0.047 P=0.963
GRWR (%)	1.2(1.07-1.33)	1.14(0.967-1.3)	Z=1.64 P=0.101
Thrombectomy	4(10)	12(18.8)	χ^2=1.45 P=0.229
HA number **1** **2**	 40(100) 0	 63(98.4) 1(1.6)	 FET=0.631 P=1.0
HA Anastmotic revision **Once** **Three times** **Twice**	 2(5) 0 2(5)	 6(9.4) 2(3.1) 8(12.5)	 MC=3.98 P=0.263

Data presented as mean and SD, median, minimum and maximum or numbers Z: Mann Whitney U test, t: Student t test, *statistically significant, χ^2= Chi -Square test, MC: Monte Carlo test , FET: Fisher exact test *statistically significant.

Post-operative labs and sonographic parameters: As shown in table (4), RRI was significantly higher in AKI group (0.744±0.06 vs 0.653±0.05, p=0.001). RRI cut off point that predicted AKI was 0.695 with a sensitivity of 82.8% and a specificity of 82.5% and AUC of 0.891. PI showed significant elevation in AKI (1.44±0.26 vs 1.25±0.35, p=0.001). MPI was significantly higher in AKI group (0.321±0.04 vs 0.282±0.04, p=0.001) MPI cut off point that predicted AKI was 0.286 with a sensitivity of 85.9% and a specificity of 57.5% and AUC of 0.745.

Table (4): postoperative sonographic and laboratory data between two groups

	NO AKI GROUP (N=40)	AKI GROUP (N=64)	TEST OF SIGNIFICANCE
MPI	0.282±0.04	0.321±0.04	t=4.72 p=0.001*
LVOT VP(CM/S)	105.04±11.18	110.89±13.21	t=2.33 p=0.02*
LVOT VTI(CM)	22.89±3.33	24.66±3.97	t=2.33 p=0.02*
LVOT PPG(MMHG)	4.49±1.0	5.07±1.21	t=2.53 p=0.013*
LVOT MPG(MMHG)	2.33±0.56	2.63±0.77	t=2.15 p=0.034*
LVOT DIAM.(CM)	2.15±0.19	2.19±0.22	t=0.980 p=0.329
LVOT SV(ML)	83.07±18.83	94.02±23.86	t=2.46 p=0.016*
LVOT CO(L/MIN)	7.99±2.06	9.40±2.37	t=3.07 p=0.003*
IRVF PATTERN **BIPHASIC** **CONTINUOUS** **MONOPHASIC** **PULSATILE**	 0 33(82.5) 0 7(17.5)	 8(12.5) 24(37.5) 1(1.6) 31(48.4)	 Mc=21.17 P=0.001*
RRI	0.653±0.05	0.744±0.06	t=8.06 p=0.001*
PI	1.28±0.23	1.68±0.40	t=5.69 p=0.001*
E.Gfr (ml/min/1.73m2)	108.48±19.78	82.42±26.27	t=5.39 p=0.001*
24h Uop (Ml/Kg)	1.29±0.13	1.10±0.37	t=3.11 p=0.002*
Intubation Duration (Min)	45(25-77.5)	75(41.25-485)	Z=3.09 P=0.002*
MAP (Mmhg)	88.12±8.48	86.8±5.26	t=0.987 p=0.326
PULSE PRESSURE (Mm/Hg)	35.50±2.91	44.34±5.15	t=9.90 p=0.001*
HEART RATE (Beat/Min)	97.38±8.58	100.06±8.63	t=1.55 p=0.124
Hb (Gm/Dl)	9.32±1.27	8.96±1.39	t=1.34 p=0.182
Wbcs (×10³/Ml)	13.27±4.9	14.72±3.24	t=1.11 p=0.268
Platelet (×10³/Ml)	94.5(62.5-147.5)	104.5(74-136)	Z=0.695 P=0.487
Serum Creatinine(Mg/Dl)	0.735±0.16	1.06±0.43	t=4.55 p=0.001*

	NO AKI GROUP (N=40)	AKI GROUP (N=64)	TEST OF SIGNIFICANCE
Uric Acid (Mg/Dl)	3.75±0.14	4.59±1.29	t=3.36 p=0.001*
Albumin (G/L)	3.46±0.48	3.20±0.57	t=2.38 p=0.019*
Ast (Iu/L)	158.0(100.5-211.25)	181(120.25-333.25)	Z=2.06 P=0.04*
Alt (Iu/L)	166.5(107.75-239.5)	162(97.75-396)	Z=0.952 P=0.341
Total Bilirubin (Mg/Dl)	3.7(2.45-5.1)	4.15(3-6.48)	Z=1.93 P=0.054
Inr	1.92±0.52	2.29±0.59	t=3.28 p=0.001*
Aptt (Seconds)	72.50±21.35	84.77±23.15	t=2.71 p=0.008*
Crp Level for Positive Cases (Mg/Dl)	12(4-38)	15(6.38-32.5)	Z=0.213 P=0.831
Ldh (Iu/L)	191(154-262)	237(179-343)	Z=2.23 P=0.026*
Lactate (Mmol/L)	3.5(2.42-4.68)	4.45(2.92-6.25)	Z=2.08 P=0.037*
Ggt (Iu/L)	31(16.5-46.75)	32(18.5-49.25)	Z=0.655 P=0.512
Alp (Iu/L)	5.0(5.0-5.0)	5.0(5.0-5.0)	Z=0.131 P=0.896
Amylase(U/L)	38.5(28.25-81.75)	56(35.25-99)	Z=1.86 P=0.063
Na (Meq/L)	137.72±2.21	136.41±3.97	t=1.92 p=0.06
K (Meq/L)	4.10±0.53	4.44±0.78	t=2.45 p=0.016*
Ca (Mg/ dl)	8.66±0.72	8.72±0.75	t=0.410 p=0.683
Mg (mg/dl)	1.45±0.42	1.29±0.20	t=2.45 p=0.01*
Ph (mg/dl)	3.84±0.12	4.26±1.05	t=1.92 p=0.06
Diuretic Use (MG)	2(5)	17(26.6)	χ^2=7.66 P=0.006*
Rbcs Units(no)	0	0	Z=1.35 P=0.176
Egfr 90 Days (ml/min/1.73m2)	97.71±17.62	81.98±16.77	t=4.19 p=0.001*

Data presented as mean and SD or numbers. Z: Mann Whitney U test χ^2=: Chi -Square test *statistically significant, MC: Monte Carlo test, t: Student t test.

Postoperative outcome, ICU stay was comparable between the two groups but mortality was significantly higher in AKI group as shown in table (5).

Table (5): Post-transplant outcomes

Outcomes	NOAKI group (n=40)	AKI group (n=64)	Significance
ICU stay (days)	6.19±1.838	7.25±4.24	0.2
Conversion to CKD (no/%)	0	2(3%)	0.188
Mortality(no/%)	0	9(14.5%)	**0.008***

Data presented as mean and SD or numbers, *statistically significant.

Diagnostic accuracy of RRI and MPI in predicting AKI

Based on the ROC curve (Figure 2) and its interpretation in Table (6):

- Preoperative MPI did not show statistical significance in predicting AKI, with an Area Under the Curve (AUC) of 0.582 and a P-value of 0.160.
- Preoperative RRI was statistically significant predictor of AKI, with an AUC of 0.759 and a P-value of 0.001∗. A cutoff of ≥ 0.655 yielded a sensitivity of 71.9% and a specificity of 62.5%.
- Postoperative MPI ('MPI after') was statistically significant predictor of AKI, with an AUC of 0.745 and a P-value of 0.001∗. Using a cutoff of ≥ 0.286, it achieved a sensitivity of 85.9% and a specificity of 57.5%.
- Postoperative RRI ('RRI after') demonstrated the highest diagnostic accuracy for predicting AKI, with an AUC of 0.891 and a statistically significant P-value of 0.001∗. At a cutoff of ≥ 0.695, its sensitivity was 82.8% and specificity was 82.5%. This indicated that postoperative RRI was the strongest individual predictor among the evaluated parameters.

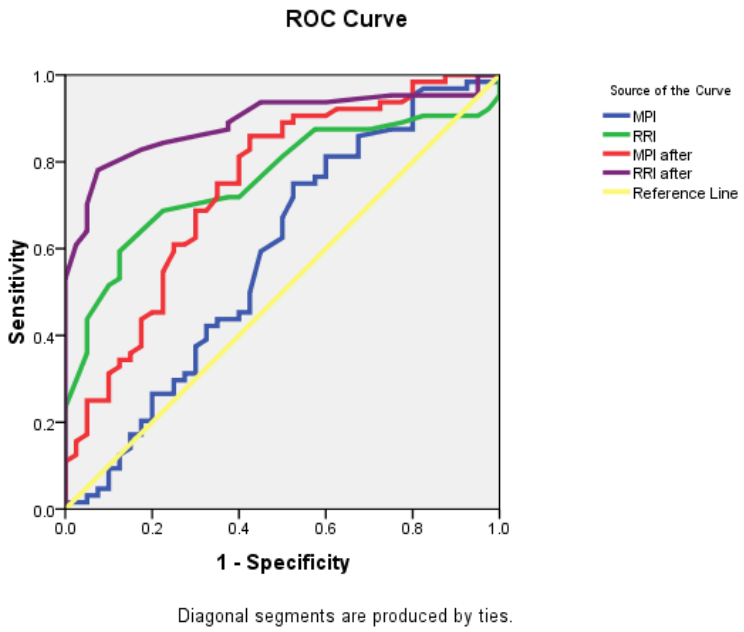

Fig (2): ROC curve of RRI and MPI preoperative and postoperative.

Table (6): Interpretation of ROC curve

Test Result Variable(s)	Area	P value	Asymptotic 95% Confidence Interval		Cut off point	Sensitivity %	Specificity %
			Lower Bound	Upper Bound			
MPI	0.582	0.160	0.464	0.700	≥0.2635	60.9	52.5
RRI	0.759	0.001*	0.667	0.851	≥0.655	71.9	62.5
MPI after	0.745	0.001*	0.645	0.844	≥0.286	85.9	57.5
RRI after	0.891	0.001*	0.828	0.954	≥0.695	82.8	82.5

DISCUSSION

Acute kidney injury (AKI) represents a highly frequent and significant complication following liver transplantation (LTx), with reported incidence rates demonstrating substantial variability, ranging widely from 5% to as high as 94% across different studies [12]. Critically, approximately 11% to 17% of these affected patients develop severe AKI, necessitating renal replacement therapy (RRT) in the early post-transplant period, underscoring the severity of this complication. Numerous independent investigations have consistently identified AKI as a prevalent and concerning sequela of LTx. Supporting this consensus, a comprehensive meta-analysis conducted in 2019 estimated an overall incidence rate of 40.7% for AKI after LTx, with a specific rate of 7.7% for severe AKI requiring RRT, further highlighting its considerable burden on patient outcomes [3].

Recent studies on AKI incidence in liver transplant (LTx) recipients show variability across countries: 37% in Sweden (141/386) [13], 50% in China (66/132) [14], 19.6% in the United States (100/511) [15], and 30.5% in Turkey (35/177) [16]. This variation likely stems from differing diagnostic criteria for AKI, such as RIFLE, AKIN, and KDIGO classifications [17], as well as differences in baseline patient characteristics, surgical techniques, and pre- and post-transplant management protocols. For instance, the Belgian study used KDIGO criteria and excluded patients with a preoperative serum creatinine above 1.5 mg/dL. The Chinese study analyzed 132 participants with less severe liver disease (MELD score=11.9) and excluded those who died within 48 hours post-LTx or had a documented glomerular filtration rate below 60 mL/min/1.73 m^2 for three months. Similarly, the Swedish study excluded re-transplant patients, those with acute liver failure, and those who died within 48 hours of LTx.

Shankar *et al.* [18] reported that 50% of their patient cohort experienced AKI post-LT within the initial seven postoperative days. Notably, the severity of renal injury observed in their study was confined to stages 1 or 2, and none of the affected patients required renal replacement therapy. Furthermore, their investigation reported no associated mortality. Their methodology was rigorous, meticulously excluding patients who presented preoperatively with hepatorenal syndrome, established baseline chronic kidney disease, or existing cardiac and circulatory disorders. Additionally, individuals experiencing perioperative circulatory shock secondary to severe hemorrhage, as well as those undergoing re-transplantation procedures, were also excluded from their study population. This stringent selection process likely aimed to isolate factors more directly related to the transplant procedure itself from pre-existing or severe intraoperative confounding variables.

A prior investigation conducted at the National Liver Institute in Egypt, which enrolled 167 patients, reported an incidence of AKI of 21% [4]. In contrast, the AKI incidence rate observed in our present study was 61.5%, which is significantly higher when compared to the findings of most previously published research. This substantial difference in reported incidence warrants further consideration, potentially attributable to variations in patient demographics, underlying comorbidities, diagnostic criteria for AKI, or perioperative management protocols employed across different centers and study periods.

In our study, approximately 64% of AKI episodes were stage 1 and 17% were stage 2, resolving without the need for renal replacement therapy (RRT). Around 17% progressed to stage 3, with only one patient requiring RRT. This aligns with reports indicating that most mild to moderate post-liver transplant (LTx) AKI cases are reversible.

While AKI can influence ICU length of stay, our analysis found no significant impact of mild or moderate AKI on 3-month or in-hospital survival. These findings are consistent with a study by **Wyssusek *et al.*** [19]. However, other research suggests a correlation between AKI (regardless of severity) and longer ICU stays, increased 90-day (and 30-day mortality, higher in-hospital mortality, and compromised long-term renal function and graft survival [20].

Two distinct studies have independently corroborated and further highlighted the critical clinical impact of AKI, demonstrating a significant association even between mild forms of AKI and reduced graft and patient survival rates following transplantation. These findings underscore that even subtle renal dysfunction post-transplant carries considerable prognostic implications, extending beyond the immediate postoperative period to influence long-term outcomes for both the allograft and the recipient [17].

Identifying precise and modifiable risk factors for AKI following LT presents a significant challenge. This difficulty primarily stems from the inherent variability and often inconsistent application of diagnostic criteria for AKI, which frequently rely predominantly on measurements of serum creatinine (Scr) and urine output [21]. A key methodological hurdle in this area is that the majority of retrospective observational studies, while valuable, often utilize only Scr for AKI classification. This practice is largely necessitated by the frequent unavailability of detailed, continuous hourly urine output data in medical records, which hinders a more comprehensive and accurate assessment of renal function changes over time. Consequently, this data limitation can lead to an incomplete picture of AKI development and severity, complicating the identification of specific, actionable risk factors for clinical intervention. However,

Scr is merely a marker of renal function, not direct kidney injury, and its levels can be delayed and insensitive in certain situations [22]. In LT candidates, who often have reduced creatinine production due to liver disease and decreased muscle mass, Scr may overestimate preoperative renal function and underestimate postoperative AKI severity. Furthermore, the choice of baseline Scr in the perioperative period presents challenges. Using immediate Scr or Scr after fluid resuscitation as a baseline can lead to AKI over-diagnosis. Conversely, comparing postoperative Scr (after significant fluid administration) to a preoperative baseline can result in under-diagnosis [23]. Therefore, diagnosing AKI after LT remains complex, highlighting the need for new biomarkers and the adoption of standardized definitions [24].

The renal resistive index (RRI) presents a promising non-invasive tool for evaluating preclinical kidney dysfunction and for predicting the risk of acute kidney injury (AKI). It serves as a valuable complement to conventional serum biomarkers, such as creatinine and urine output, which often reflect renal injury only after significant functional decline. RRI assesses the state of renal circulation through the analysis of Doppler waveforms, typically obtained from the arcuate or interlobar arteries within the kidney parenchyma [25]. Physiologically, given the notably higher density of vasoconstrictor receptors present in renal vessels, these arterioles exhibit a more significant degree of constriction during systemic compensatory responses, thereby leading to a pronounced increase in intrarenal vascular resistance [26]. Consequently, fluctuations observed in RRI values reliably reflect changes in both systemic peripheral and localized renal perfusion. An elevated RRI, in particular, often serves as a sensitive indicator of inadequate renal perfusion, signaling compromised blood flow within the kidney's microvasculature [27].

A meta-analysis, encompassing nine distinct studies focused on specific patient populations such as those with severe sepsis or requiring mechanical ventilation, identified a notable association between an elevated RRI and an increased risk of persistent AKI. This analysis reported RRI demonstrating a sensitivity of 0.83 and a specificity of 0.83 in predicting AKI when compared against conventional markers like serum creatinine or oliguria. However, the interpretation of these findings is tempered by significant limitations inherent to the meta-analysis itself, primarily characterized by substantial heterogeneity across the included studies and a notable absence of rigorous consideration for the methodological quality of the individual investigations [28]. Conversely, a subsequent study by **Darmon *et al.*** [29] involving a larger cohort (n = 371) of unselected critically ill patients, found RRI to have poor predictive ability for persistent AKI, with sensitivity and specificity of 50% (95% CI 41%–58%) and 68% (62%–74%) at an optimal cutoff of RRI = 0.71 [29]. The inconsistencies in these patterns can, to some extent, be attributed to the diverse patient populations studied, ranging from highly specific groups to heterogeneous cohorts in a critical condition. Earlier investigations exploring RRI changes after fluid challenges yielded mixed results.

Schnell *et al.* [30] observed RRI stability in mechanically ventilated patients undergoing fluid challenge, whereas **Moussa *et al.*** [31] concluded a consistent decrease in RRI in patients experiencing acute circulatory failure (from 0.73 ± 0.09 to 0.71 ± 0.09, $p < 0.01$). **Wybraniec *et al.*** [32] conducted an investigation into the clinical utility of pre-procedural RRI as a predictor for the development of AKI following coronary angiography. Their findings revealed an optimal RRI cutoff value exceeding 0.69, which demonstrated a notable diagnostic performance characterized by a sensitivity of 78% and a specificity of 81%. This suggests that pre-procedural RRI assessment holds considerable promise as a non-invasive tool for identifying patients at elevated risk for post-angiography AKI, potentially facilitating earlier preventive strategies.

Similarly, **Shanker *et al.*** [18] studied RRI's predictive value for post-liver transplant AKI, reporting that an RRI ≥ 0.69 on postoperative day (POD) 2 predicted AKI with 88% sensitivity and 92% specificity. In our study, the optimal RRI cutoff on POD zero for predicting AKI was 0.695, yielding a sensitivity of 82.8% and specificity of 82.5%.

The Myocardial Performance Index (MPI) is a widely utilized measure of overall cardiac function, encompassing both systolic and diastolic performance [32]. Its application extends across various cardiovascular conditions, including heart failure, myocardial infarction, dilated cardiomyopathy, amyloidosis, and heart transplantation [33, 34]. MPI has been shown to not only reflect disease severity but also offer significant prognostic insights. Furthermore, MPI appears to be independent of changes in heart rate, afterload, and possibly preload [34].

Kuznetsova *et al.* [35] observed a statistically significant positive correlation between the RRI and the E velocity, which is a key component among Doppler indices of left ventricular blood flow. However, their investigation did not extend to exploring RRI's specific association with MPI. In contrast to studies primarily focusing on renal hemodynamics, **Papadopoulou *et al.*** [36] presented findings from a cohort of patients with End-Stage Renal Failure (ESRF) undergoing hemodialysis (HD). Their research revealed that MPI values were significantly elevated in patients who experienced episodes of intradialytic hypotension when compared to those maintaining normal intradialytic blood pressure. This observation strongly suggests that MPI may serve as

a more sensitive and potentially earlier predictor of intradialytic hypotension than conventional echocardiographic indicators, offering valuable insights for managing this common and often problematic complication in HD patients.

Asami *et al*. [37] identified a significant and independent association between MPI and poorer clinical outcomes, specifically at 30 days and extending up to one year following transcatheter aortic valve replacement. Their research demonstrated that pre-procedural MPI served as an independent predictor of all-cause mortality within 30 days. Furthermore, post-procedural MPI exhibited a strong correlation with several critical long-term adverse events, including all-cause death, cardiovascular death, and major adverse cardiovascular and cerebrovascular events occurring between 30 days and one year. Consistent with the prognostic utility of MPI observed in other cardiac contexts, our current study similarly revealed its predictive value in the transplant setting. Specifically, we observed that an MPI value exceeding 0.286 on postoperative day (POD) zero demonstrated the capacity to predict AKI post-liver transplant with a notable sensitivity of 85.9% and a specificity of 57.5%. This finding underscored the potential of early postoperative cardiac function assessment via MPI as an indicator of subsequent renal complications.

To our current knowledge, this study represents the first report evaluating the intricate relationship between renal hemodynamics, as robustly assessed through RRI, and global cardiac function, comprehensively evaluated through MPI. Furthermore, this investigation uniquely explored the combined utility of these two distinct physiological parameters in predicting the occurrence of AKI post liver transplantation, thereby addressing a notable gap in existing literature.

LIMITATIONS

While this study offers valuable insights, several limitations should be acknowledged. The sample size, though sufficient for preliminary analysis, may not fully capture the variability present in a broader population, which could affect the generalizability of the findings. Additionally, the absence of multivariate analysis limits the ability to explore complex relationships among variables and to control for potential confounders. Furthermore, the predictors identified in this study have not yet undergone external validation, which may restrict their applicability across different contexts or populations. Future research would benefit from larger and more diverse samples, the use of advanced analytical approaches, and efforts to validate these findings in independent cohorts.

CONCLUSION

The findings of this investigation indicated that both the RRI and the MPI, particularly when assessed with postoperative measurements, served as effective predictors of early postoperative AKI in recipients of living donor liver transplantation (LDLT). Notably, RRI demonstrated superior diagnostic accuracy in this predictive capacity. These compelling results suggest that the integration of perioperative RRI and MPI monitoring into clinical practice could significantly enhance AKI risk stratification and guide more proactive management strategies. Nevertheless, to firmly establish their routine clinical utility, these promising findings unequivocally warrant further comprehensive validation through larger, multicenter studies.

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