

Evaluation of Post-Operative AKI in Major Open vs Laparoscopic Surgeries: A Prospective Comparative Study

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Abstract: ***Background:** Postoperative acute kidney injury (AKI) is a common and under-recognised complication of major abdominal surgery. Laparoscopy reduces tissue trauma and blood loss but introduces pneumoperitoneum, which can compromise renal perfusion. Whether the surgical approach itself independently determines early renal outcome remains unsettled, and prospective Indian data are sparse. **Methods:** A prospective comparative observational study was conducted in the Department of General Surgery, GSVM Medical College and LLR Hospital, Kanpur. One hundred consecutive adults with normal baseline renal function undergoing major elective abdominal surgery were grouped by surgical approach: open (Group A, n=48) and laparoscopic (Group B, n=52). Serum creatinine and cystatin-C were measured preoperatively and on postoperative days 1 and 2; estimated glomerular filtration rate (eGFR) was derived using the race-free 2021 CKD-EPI creatinine-cystatin C equation. AKI was classified by RIFLE criteria. Perioperative variables were analysed as candidate risk factors. **Results:** Postoperative AKI occurred in 12 of 100 patients (12.0%) - 7 of 48 (14.6%) after open and 5 of 52 (9.6%) after laparoscopic surgery (p=0.544; OR 1.60, 95% CI 0.47–5.45). All events were confined to RIFLE stage Risk; none required dialysis. Intraoperative hypotension was the dominant determinant: 11 of 11 patients with a mean arterial pressure below 65 mmHg developed AKI, compared with 1 of 89 without (p<0.001). Blood transfusion (OR 5.85, 95% CI 1.65–20.69; p=0.007) and blood loss exceeding 250 mL (OR 5.65, 95% CI 1.55–20.55; p=0.008) were also significant. Blood loss was higher after open surgery (276.6±98.7 vs 175.8±70.0 mL; p<0.001), but the absolute fall in eGFR did not differ between groups (−7.7±11.1 vs −6.0±9.6 mL/min/1.73 m²; p=0.327). **Conclusions:** Early postoperative AKI after major elective abdominal surgery was driven by intraoperative haemodynamic and haemorrhagic insult rather than by the surgical approach. Concern about pneumoperitoneum should not deter the use of laparoscopy, and perioperative effort is better directed at maintaining mean arterial pressure and limiting blood loss.*

Keywords: Acute kidney injury; Laparoscopic surgery; Intraoperative hypotension; RIFLE criteria; Cystatin C; CKD-EPI

1. Introduction

Postoperative acute kidney injury is among the most frequent and least appreciated complications of major surgery. Even transient, mild elevations in serum creatinine are associated with prolonged hospitalisation, progression to chronic kidney disease and increased short- and long-term mortality.¹ The EPIS-AKI study, the largest prospective international investigation of surgery-associated AKI, studied 10,568 patients across 30 countries and found that 18.4% developed AKI within 72 hours of major surgery, of whom a third had persistent injury and 8.7% required renal replacement therapy.² A pooled analysis of abdominal surgical cohorts has reported a comparable incidence of approximately 16%.

The choice between open and laparoscopic approaches poses a physiological paradox. Open surgery carries greater tissue trauma, inflammatory activation, haemodynamic instability and blood loss — all established antecedents of renal injury. Laparoscopy reduces each of these, yet introduces a distinct insult in the form of carbon dioxide pneumoperitoneum. Insufflation to 12–15 mmHg compresses the renal parenchyma and renal veins, reduces renal blood flow and glomerular filtration, and activates the renin-angiotensin-

aldosterone and sympathetic axes; desufflation adds an ischaemia-reperfusion component. When Trendelenburg positioning is used, the haemodynamic burden may be amplified further.³

Observational evidence has largely failed to confirm that these mechanisms translate into clinical harm. In a propensity-matched analysis of 3,692 colorectal patients, laparoscopy in the Trendelenburg position did not worsen postoperative eGFR and was in fact associated with lower odds of AKI (OR 0.70, 95% CI 0.55–0.90).³ Similar neutral or protective findings have been reported in rectal cancer surgery and hepatic resection.⁴

What is less often examined is whether the surgical approach matters at all once intraoperative haemodynamics are accounted for. Intraoperative hypotension has a well-described dose-duration relationship with renal injury,⁵ and blood loss and transfusion are themselves nephrotoxic through hypoperfusion, free haemoglobin and inflammatory mediators. The present study was designed to compare the incidence and severity of early postoperative AKI between major open and laparoscopic abdominal surgery in an Indian tertiary-care setting, and to identify which perioperative variables independently predict it.

2. Materials and Methods

Study design and setting

This prospective comparative observational study was conducted in the Department of General Surgery, GSVM Medical College and Lala Lajpat Rai (LLR) Hospital, Kanpur, a tertiary-care teaching institution in Uttar Pradesh, India. Patients were enrolled consecutively between August 2025 and March 2026. Allocation was non-randomised: the surgical approach was determined by the operating surgeon on clinical grounds, since randomisation of approach would be neither ethical nor practicable in routine care.

Ethical approval

The protocol was reviewed and approved by the Ethics Committee (Biomedical Health and Research), GSVM Medical College, Kanpur (Ref. No. EC/BMHR/2025/41; approved at the meeting of 16 July 2025) prior to the commencement of enrolment. The study conforms to the Declaration of Helsinki (2013 revision). Written informed consent, in Hindi or English as the participant preferred, was obtained from every patient.

Participants

Adults aged 18 years and above scheduled for major elective abdominal surgery- defined as an intra-abdominal procedure expected to exceed two hours under general or combined regional-general anaesthesia- were eligible, provided baseline renal function was normal (serum creatinine within the age- and sex-adjusted reference range and eGFR ≥ 60 mL/min/1.73 m²). Patients were excluded for pre-existing chronic kidney disease of any stage, emergency surgery, preoperative sepsis or shock, baseline serum creatinine above 1.6 mg/dL, renal replacement therapy or a functioning renal allograft, or inability to consent.

Group A comprised patients undergoing conventional open laparotomy- including open cholecystectomy, bowel resection and anastomosis, colectomy, gastrectomy, gastrojejunostomy, stoma formation or closure, and open hernia repair. Group B comprised patients undergoing laparoscopic procedures with carbon dioxide pneumoperitoneum maintained at 12–14 mmHg, including laparoscopic cholecystectomy, laparoscopic hernia repair, laparoscopic colectomy and fundoplication. Patients converted to open surgery were analysed in the laparoscopic group on an intention-to-treat basis.

Sample size

Assuming AKI incidences of 15% after open and 5% after laparoscopic surgery, a two-sided alpha of 0.05 and 80% power, 127 patients per group would have been required. Given the constraints of a single-centre study within a defined academic period, a convenience sample of 100 patients was enrolled. The study is therefore appropriately regarded as a pilot comparative analysis, and this limitation is addressed in the Discussion.

Measurements and outcome definitions

Serum creatinine was measured by an IDMS-traceable enzymatic method and serum cystatin-C by particle-enhanced nephelometric immunoassay standardised to ERM-DA471/IFCC, both in the institutional central

laboratory. Baseline samples were drawn within 24 hours before surgery and postoperative samples on days 1 and 2. Estimated GFR was calculated with the race-free 2021 CKD-EPI creatinine–cystatin C equation,⁶ selected for its superior accuracy (P30 >90%) and because cystatin-C is unaffected by the fluid shifts and altered muscle turnover that confound creatinine in surgical patients.

Intraoperative data comprised duration of surgery, estimated blood loss, intravenous fluid volume, transfusion, vasopressor requirement and intraoperative hypotension, the last defined as any recorded mean arterial pressure below 65 mmHg.

The primary outcome was the incidence of postoperative AKI within 48 hours, classified by the RIFLE criteria: Risk (creatinine $\times 1.5$ or eGFR fall >25%), Injury (creatinine $\times 2$ or eGFR fall >50%) and Failure (creatinine $\times 3$ or eGFR fall >75%).⁷ Secondary outcomes were AKI severity stage, absolute and percentage change in eGFR, hospital length of stay, need for renal replacement therapy and discharge status.

Statistical analysis

Data were analysed in SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Normality was assessed by the Shapiro–Wilk test. Continuous variables are presented as mean $\pm$ standard deviation and compared by Student's t-test or the Mann–Whitney U test as appropriate; categorical variables are presented as frequencies with percentages and compared by the chi-square or Fisher's exact test. Unadjusted odds ratios with 95% confidence intervals were calculated for candidate risk factors. Where an exposure showed complete separation from the outcome, the odds ratio is not estimable and the association is reported as an exact test result with the corresponding risk difference. Spearman's rank correlation was used for continuous associations. A two-sided p-value below 0.05 was taken as significant.

3. Results

Baseline characteristics

All 100 enrolled patients completed the protocol with no loss to follow-up. Forty-eight (48%) underwent open and 52 (52%) laparoscopic surgery. Patients in the open group were older (44.9 $\pm$ 12.5 vs 39.5 $\pm$ 14.5 years; $p=0.040$). Sex distribution, body mass index and the prevalence of diabetes, hypertension, cardiovascular disease and smoking were comparable (Table 1). Baseline serum creatinine, cystatin-C and blood urea nitrogen were well matched, but baseline eGFR was higher in the laparoscopic group (100.4 $\pm$ 18.5 vs 93.1 $\pm$ 13.3 mL/min/1.73 m²; $p=0.028$), consistent with its younger age profile.

Intraoperative course

Operative duration was similar between groups (3.2 $\pm$ 1.2 vs 3.3 $\pm$ 1.1 hours; $p=0.438$). Intraoperative blood loss was substantially greater with open surgery (276.6 $\pm$ 98.7 vs 175.8 $\pm$ 70.0 mL; $p<0.001$), as was the transfusion rate (37.5% vs 11.5%; $p=0.005$). Intravenous fluid volumes were comparable. Intraoperative hypotension occurred in 14.6% of open and 7.7% of laparoscopic patients and vasopressors

were used in 14.6% and 5.8% respectively; neither difference was statistically significant (Table 2).

Primary outcome

Postoperative AKI occurred in 12 of 100 patients (12.0%); 7 of 48 (14.6%) in the open group and 5 of 52 (9.6%) in the laparoscopic group. The difference was not significant (Fisher's exact $p=0.544$; OR 1.60, 95% CI 0.47–5.45; RR 1.52, 95% CI 0.52–4.46). Every event was confined to RIFLE stage Risk; no patient progressed to Injury, Failure, Loss or End-stage disease, and none required dialysis. Ordinal comparison of RIFLE severity showed no between-group difference (Mann–Whitney $U=1310.0$; $p=0.451$).

Renal function trajectory

Serum creatinine rose modestly from baseline through day 2 in both groups, with no significant between-group difference on day 1 ($p=0.305$) or day 2 ($p=0.530$). Postoperative eGFR was lower after open surgery (85.4 ± 18.9 vs 94.4 ± 20.5 mL/min/1.73 m²; $p=0.046$), but this is confounded by the higher baseline in the laparoscopic group. The more informative measure- the absolute fall from each patient's own baseline- did not differ (-7.7 ± 11.1 vs -6.0 ± 9.6 mL/min/1.73 m²; $p=0.327$), nor did the percentage decline ($8.8\pm12.8\%$ vs $6.1\pm9.4\%$; $p=0.065$) (Table 3).

Risk factors

Intraoperative hypotension showed a near-absolute association with renal injury: all 11 patients in whom mean arterial pressure fell below 65 mmHg developed AKI, against 1 of the remaining 89 ($p<0.001$). Because the exposure separates the outcome completely, no finite odds ratio can be estimated; the risk difference is 98.9 percentage points. Blood transfusion (OR 5.85, 95% CI 1.65–20.69; $p=0.007$) and blood loss exceeding 250 mL (OR 5.65, 95% CI 1.55–20.55; $p=0.008$) were both significantly associated with AKI. Age above 50 years approached significance (OR 3.34, 95% CI 0.97–11.49; $p=0.057$). Surgical approach was not associated with AKI (OR 1.60; $p=0.544$) (Table 4). Patients who developed AKI had greater blood loss (304.6 ± 111.7 vs 213.2 ± 92.0 mL; $p=0.007$), higher transfusion rates (58.3% vs 19.3%; $p=0.007$) and a far higher frequency of intraoperative hypotension (91.7% vs 0%; $p<0.001$) than those who did not. Postoperative eGFR (58.8 ± 10.7 vs 94.4 ± 17.2 mL/min/1.73 m²) and the fall from baseline (-33.3 ± 7.9 vs -3.2 ± 1.7 mL/min/1.73 m²) differed markedly (both $p<0.001$). Age, body mass index, baseline creatinine, baseline eGFR, diabetes and hypertension did not differ significantly between the two, although the comparison of 12 against 88 patients was underpowered for moderate effects.

Baseline creatinine correlated inversely with the postoperative creatinine ratio ($r=-0.784$; $p<0.001$), indicating that patients starting from a lower creatinine sustained proportionally larger relative rises. Duration of surgery, blood loss and fluid volume did not correlate significantly with the creatinine ratio.

Clinical outcomes

Hospital stay was equivalent (4.4 ± 1.0 vs 4.5 ± 1.3 days; $p=0.943$). No patient in either group required renal replacement therapy or developed RIFLE Loss or End-stage

disease. Fluid overload was recorded in one laparoscopic patient (1.9%). At discharge, 83.3% of open and 90.4% of laparoscopic patients were classified as recovered, the remainder as stable.

4. Discussion

The central finding of this study is that the surgical approach did not determine early renal outcome, whereas intraoperative haemodynamics did. Although AKI was numerically more frequent after open surgery, the confidence interval around the odds ratio comfortably included unity, and the fall in eGFR from each patient's own baseline was statistically indistinguishable between groups. By contrast, every patient who experienced a mean arterial pressure below 65 mmHg developed AKI, and only one patient who did not experience hypotension did so.

This pattern is consistent with the larger literature. Essber and colleagues, comparing 1,846 propensity-matched pairs undergoing colorectal surgery, found no association between the laparoscopic approach and postoperative eGFR and a protective effect on AKI odds, concluding that laparoscopy should not be avoided out of concern for renal injury.³ Neutral or favourable renal outcomes for the minimally invasive approach have also been reported in rectal cancer surgery.⁴ The theoretical hazard of pneumoperitoneum is real at the level of renal physiology- transient oliguria during insufflation is well documented- but in patients with normal baseline function undergoing elective procedures of ordinary duration and pressure, it appears to be offset by the reduction in blood loss and systemic inflammatory insult.

The strength of the association with intraoperative hypotension in this cohort warrants comment. Salmasi and colleagues established a dose–duration relationship between intraoperative mean arterial pressure below 65 mmHg and postoperative renal and myocardial injury in a large non-cardiac surgical population.⁵ Our finding is directionally identical but far more extreme, and the completeness of the separation almost certainly reflects the small number of events and the homogeneity of an elective cohort with normal baseline renal function, in which hypotension was the principal deviation from an otherwise uncomplicated perioperative course. The practical inference is nonetheless secure: the modifiable determinant of early renal injury in this setting sits with the anaesthetic and haemodynamic management, not with the choice of incision.

Blood loss and transfusion behaved as interlinked risk factors, each roughly quintupling the odds of AKI. Their relationship to the surgical approach is instructive: open surgery produced significantly more blood loss and transfusion, yet did not produce significantly more AKI. The approach influences renal outcome only indirectly, through the intraoperative insult it generates, and where that insult is contained the approach itself ceases to matter.

The restriction of every event to RIFLE stage Risk, with no dialysis and no difference in hospital stay, should not be read as clinical irrelevance. Mild AKI is associated with adverse long-term renal and mortality outcomes,¹ and in a population with normal preoperative renal reserve

undergoing elective surgery, stage 1 injury is precisely what would be expected. Detecting it at all required systematic biochemical surveillance; in routine practice, with creatinine checked only when clinically prompted, most of these 12 patients would have gone unrecognised. The use of the 2021 CKD-EPI creatinine–cystatin C equation strengthens the renal assessment. Creatinine alone is an unreliable index in the perioperative period, where haemodilution, reduced muscle turnover and altered volume of distribution all distort it; the combined equation, which removed the race coefficient and achieves P30 values above 90%, is better suited to this population.⁶ Reporting change from each patient's own baseline rather than absolute postoperative values also avoids the confounding introduced by the between-group difference in baseline eGFR.

These findings have direct relevance to Indian surgical practice, where laparoscopic capability is expanding rapidly but perioperative renal surveillance is inconsistent and dialysis access limited outside tertiary centres. The message for practice is that renal concern is not a reason to prefer open surgery, and that a targeted effort to maintain mean arterial pressure above 65 mmHg and to minimise blood loss is likely to yield more renal benefit than any decision about approach.

5. Limitations

Several limitations qualify these conclusions. The study was single-centre and non-randomised, so allocation reflected surgeon judgement and the groups differed in age and baseline eGFR. The enrolled sample of 100 was well below the 254 patients the power calculation required, and the study was consequently underpowered to detect a true difference of the magnitude hypothesised; the negative primary finding should be interpreted as an absence of demonstrated difference rather than as demonstrated equivalence. The very small number of events (12) limits any multivariable modelling, and the complete separation between hypotension and AKI precludes a stable adjusted estimate; the univariate associations reported here should be regarded as hypothesis-generating. Renal function was assessed only to postoperative day 2, so late or persistent AKI would not have been captured. Intraoperative hypotension was recorded as a binary variable rather than as area under a pressure–time curve, which discards information about depth and duration. Open and laparoscopic groups contained heterogeneous procedures of differing magnitude. Finally, novel tubular injury biomarkers such as NGAL and [TIMP-2]×[IGFBP7], which detect injury hours earlier than creatinine,⁸ were unavailable.

6. Conclusion

In this prospective comparative study of 100 patients undergoing major elective abdominal surgery, early postoperative acute kidney injury occurred in 12% and was confined entirely to RIFLE stage Risk. The surgical approach did not significantly influence either the incidence or the magnitude of renal functional decline. Intraoperative hypotension, blood loss above 250 mL and blood transfusion were the factors associated with injury. Concern about pneumoperitoneum is not a sound basis for preferring open

surgery, and perioperative renal protection is better pursued through haemodynamic vigilance and meticulous haemostasis than through the choice of operative approach. Adequately powered multicentre studies incorporating continuous haemodynamic monitoring and early tubular biomarkers are needed to confirm these observations.

Declarations

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Conflict of interest: None declared.

Ethical approval: Obtained from the Ethics Committee (Biomedical Health and Research), GSVM Medical College, Kanpur (Ref. No. EC/BMHR/2025/41).

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Table 1: Baseline demographic and clinical characteristics

Variable	Open (n=48)	Laparoscopic (n=52)	p-value
Age (years)	44.9 ± 12.5	39.5 ± 14.5	0.04
Male sex, n (%)	22 (45.8)	29 (55.8)	0.428
Body mass index (kg/m ²)	26.9 ± 6.4	27.1 ± 5.6	0.85
Diabetes mellitus, n (%)	28 (58.3)	27 (51.9)	0.658
Hypertension, n (%)	6 (12.5)	7 (13.5)	1
Baseline serum creatinine (mg/dL)	0.9 ± 0.2	0.9 ± 0.2	0.627
Baseline serum cystatin-C (mg/L)	0.9 ± 0.2	0.9 ± 0.2	0.343
Blood urea nitrogen (mg/dL)	14.6 ± 5.6	15.7 ± 5.7	0.309
Baseline eGFR (mL/min/1.73 m ²)	93.1 ± 13.3	100.4 ± 18.5	0.028

Values are mean ± SD unless stated otherwise. eGFR by the 2021 CKD-EPI creatinine–cystatin C equation

Table 2: Intraoperative characteristics

Variable	Open (n=48)	Laparoscopic (n=52)	p-value
Duration of surgery (hours)	3.2 ± 1.2	3.3 ± 1.1	0.438
Estimated blood loss (mL)	276.6 ± 98.7	175.8 ± 70.0	<0.001
Intravenous fluid (mL)	2122.9 ± 481.2	2220.2 ± 566.2	0.237
Blood transfusion, n (%)	18 (37.5)	6 (11.5)	0.005
Intraoperative hypotension, n (%)	7 (14.6)	4 (7.7)	0.435
Vasopressor requirement, n (%)	7 (14.6)	3 (5.8)	0.188

Intraoperative hypotension defined as any recorded mean arterial pressure below 65 mmHg.

Table 3: Renal outcomes by surgical approach

Outcome	Open (n=48)	Laparoscopic (n=52)	p-value
Postoperative AKI, n (%)	7 (14.6)	5 (9.6)	0.544
RIFLE stage Risk, n (%)	7 (14.6)	5 (9.6)	0.451
RIFLE Injury / Failure, n (%)	0 (0)	0 (0)	—
Day 1 creatinine (mg/dL)	1.0 ± 0.2	0.9 ± 0.2	0.305
Day 2 creatinine (mg/dL)	1.0 ± 0.3	1.0 ± 0.2	0.53
Postoperative eGFR (mL/min/1.73 m ²)	85.4 ± 18.9	94.4 ± 20.5	0.046
Absolute ΔeGFR (mL/min/1.73 m ²)	−7.7 ± 11.1	−6.0 ± 9.6	0.327
Percentage eGFR decline (%)	8.8 ± 12.8	6.1 ± 9.4	0.065
Hospital stay (days)	4.4 ± 1.0	4.5 ± 1.3	0.943
Renal replacement therapy, n	0	0	—

AKI: acute kidney injury. Δ eGFR: postoperative minus preoperative eGFR

Table 4: Unadjusted associations with postoperative acute kidney injury

Risk factor	Odds ratio (95% CI)	p-value
Intraoperative hypotension	Not estimable*	<0.001
Blood transfusion	5.85 (1.65–20.69)	0.007
Blood loss >250 mL	5.65 (1.55–20.55)	0.008
Age >50 years	3.34 (0.97–11.49)	0.057
Duration of surgery >3 hours	1.93 (0.57–6.56)	0.291
Diabetes mellitus	1.74 (0.49–6.22)	0.392
Baseline eGFR <90 mL/min/1.73 m ²	1.67 (0.50–5.60)	0.405
Open surgical approach	1.60 (0.47–5.45)	0.544
Male sex	1.40 (0.41–4.75)	0.765
Hypertension	1.40 (0.27–7.25)	0.686

*All 11 patients with intraoperative hypotension developed AKI versus 1 of 89 without (Fisher's exact $p < 0.001$); complete separation precludes a finite odds ratio. Risk difference 98.9 percentage points.

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