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Original Research Article

ASSOCIATION OF INTRAOPERATIVE HYPOTENSION BURDEN WITH POSTOPERATIVE ACUTE KIDNEY INJURY IN ADULTS UNDERGOING MAJOR NON-CARDIAC SURGERY: A PROSPECTIVE OBSERVATIONAL COHORT

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ABSTRACT

Background: Postoperative acute kidney injury (AKI) is an important complication of major non-cardiac surgery. Intraoperative hypotension may compromise renal perfusion, but the contribution of its cumulative duration and severity requires careful evaluation. The aim is to assess the association between intraoperative hypotension burden and postoperative AKI in adults undergoing major non-cardiac surgery.

Materials and Methods: This prospective observational cohort included 92 adults undergoing major non-cardiac surgery under general anaesthesia at a tertiary care hospital. Baseline clinical characteristics, serum creatinine, estimated glomerular filtration rate (eGFR), operative variables, and intraoperative mean arterial pressure (MAP) were assessed. Hypotension was defined as MAP below 65 mmHg and quantified using cumulative duration, number of episodes, area under the threshold, and time-weighted average. AKI occurring within seven postoperative days was classified using serum creatinine-based KDIGO criteria. Data were analysed using SPSS version 28.0.

Results: Postoperative AKI occurred in 22 patients (23.91%). Pre-existing chronic kidney disease was more frequent among patients with AKI (50.00% versus 8.57%; $p<0.001$). Median cumulative hypotension duration was greater in the AKI group (14.0 versus 7.5 minutes; $p<0.001$). AKI incidence was 42.11% following hypotension exceeding 10 minutes compared with 11.11% with shorter exposure ($p=0.001$). A minimum MAP below 60 mmHg was also associated with higher AKI incidence (36.00% versus 9.52%; $p=0.003$). Hypotension duration (adjusted odds ratio [aOR] 2.42 per five minutes) and reduced baseline eGFR (aOR 2.52 per 10 mL/min/1.73 m² decrease) were associated with AKI. AKI was accompanied by greater ICU admission and longer hospitalisation.

Conclusion: Greater intraoperative hypotension burden and impaired baseline renal function were associated with postoperative AKI. Careful haemodynamic surveillance may help identify patients requiring closer renal monitoring.

Keywords: Acute Kidney Injury; Intraoperative Hypotension; Mean Arterial Pressure; Non-Cardiac Surgery; Renal Function.

INTRODUCTION

Acute kidney injury (AKI) is a clinically important complication of major surgical procedures and remains a considerable challenge in perioperative medicine. It involves an abrupt deterioration in renal excretory function, frequently accompanied by disturbances in fluid balance, electrolyte regulation, and metabolic homeostasis. Even when renal dysfunction appears transient, it may complicate postoperative recovery and contribute to adverse long-term health consequences. The kidneys are

particularly sensitive to alterations in blood flow, oxygen delivery, and systemic inflammatory activity during surgery. Consequently, early identification of potentially modifiable perioperative factors is fundamental to protecting renal function and improving patient safety. Understanding the mechanisms underlying AKI is essential for developing appropriate preventive strategies.[1] Postoperative AKI following major non-cardiac surgery is a multifactorial disorder influenced by preoperative health status, the physiological stress of surgery, anaesthetic management, and postoperative complications. Surgical trauma can activate neuroendocrine and inflammatory pathways, while tissue injury, haemodynamic disturbances, and exposure to nephrotoxic agents may further impair renal function. Contemporary perioperative recommendations emphasise evaluating kidney function before surgery and identifying AKI occurring within seven postoperative days using standardised diagnostic criteria. These principles are especially relevant in tertiary care hospitals, where patients commonly undergo complex abdominal, orthopaedic, vascular, and other major surgical procedures. A structured assessment of renal risk provides a foundation for targeted perioperative monitoring.[2] The susceptibility to postoperative renal injury varies considerably between individuals. Advanced age, chronic kidney disease, diabetes mellitus, hypertension, cardiovascular dysfunction, preoperative anaemia, and reduced estimated glomerular filtration rate may compromise physiological reserves before anaesthesia is initiated. Intraoperative factors, including blood loss, fluid imbalance, prolonged surgical stress, and exposure to vasoactive drugs, can interact with underlying vulnerability. Preoperative serum creatinine and estimated glomerular filtration rate are therefore valuable for identifying patients who require closer haemodynamic surveillance. However, renal function before surgery cannot fully explain subsequent AKI, making it necessary to evaluate potentially reversible exposures during anaesthesia and the early postoperative period.[3] Intraoperative hypotension is among the most clinically relevant haemodynamic disturbances occurring during general anaesthesia. It can arise from anaesthetic-induced vasodilatation, reduced cardiac output, intravascular volume depletion, haemorrhage, or combinations of these mechanisms. Mean arterial pressure (MAP) is routinely used as a practical indicator of systemic perfusion pressure, although actual kidney perfusion also depends on venous pressure and individual autoregulatory capacity. When arterial pressure decreases beyond the ability of renal autoregulation to maintain blood flow, tissue oxygen delivery may become inadequate. Sustained hypoperfusion can initiate renal tubular stress and contribute to subsequent dysfunction, highlighting the importance of timely recognition and physiological correction of hypotension.[4] An important difficulty in perioperative practice is the absence of a single blood pressure threshold that is appropriate for every patient. Although absolute MAP values provide convenient reference points, tolerance of hypotension may differ according to baseline blood pressure, vascular disease, chronic hypertension, and other comorbidities. Intermittent non-invasive measurements can also miss short-lived haemodynamic fluctuations, whereas more frequent or continuous monitoring provides a detailed picture of exposure. Contemporary expert guidance stresses maintaining adequate arterial pressure in at-risk patients and directing treatment towards the cause of hypotension rather than the pressure measurement alone. Individualised management may therefore be more informative than relying exclusively on a uniform numerical threshold.[5] The concept of intraoperative hypotension burden extends beyond the simple presence or absence of low blood pressure. It incorporates the depth, frequency, and cumulative duration of hypotensive episodes and may therefore characterise physiological exposure more comprehensively. Measures such as total minutes with MAP below a specified threshold, the lowest recorded MAP, area under the hypotension threshold, and time-weighted average hypotension permit quantitative evaluation. These parameters distinguish brief mild reductions from prolonged or profound hypotension, which may have different biological implications. Nevertheless, uncertainties remain regarding the most clinically informative burden metric and how different levels of exposure should be interpreted alongside pre-existing renal vulnerability.[6]

MATERIALS AND METHODS

This prospective observational cohort study was conducted in the Department of Anaesthesiology at a tertiary care hospital to evaluate the association between intraoperative hypotension burden and postoperative acute kidney injury (AKI) among adults undergoing major non-cardiac surgery. A total of 92 patients were included. Perioperative haemodynamic parameters, clinical characteristics, laboratory investigations, and postoperative renal outcomes were systematically assessed. The observational design ensured that routine anaesthetic management was not modified by study participation. The study included 92 adult patients aged 18 years and above undergoing major non-cardiac surgical procedures under general anaesthesia. Patients were enrolled through consecutive sampling according to predefined eligibility criteria. The study population included individuals undergoing major abdominal, orthopaedic, vascular, and other non-cardiac procedures. Patients were evaluated for preoperative renal function, intraoperative haemodynamic fluctuations, and postoperative renal complications.

Inclusion Criteria

Patients aged ≥18 years undergoing major non-cardiac surgery under general anaesthesia, with

available preoperative serum creatinine measurements and intraoperative blood pressure monitoring, were eligible for inclusion. Patients belonging to American Society of Anesthesiologists (ASA) physical status classes I–IV were considered. Individuals with controlled hypertension, diabetes mellitus, or stable chronic kidney disease were included to assess the influence of pre-existing comorbidities on postoperative renal outcomes.

Exclusion Criteria

Patients with pre-existing acute kidney injury, end-stage renal disease requiring maintenance dialysis, renal transplantation, or incomplete perioperative haemodynamic records were excluded. Patients undergoing cardiac surgery, procedures involving cardiopulmonary bypass, or those lacking postoperative renal function measurements were also excluded.

Methodology: All patients underwent detailed preoperative evaluation, including age, sex, body mass index (BMI), ASA physical status, smoking history, and associated comorbidities such as hypertension, diabetes mellitus, chronic kidney disease, and cardiovascular disorders. Baseline systolic blood pressure, diastolic blood pressure, mean arterial pressure (MAP), and heart rate were recorded. Laboratory investigations included haemoglobin, serum creatinine, blood urea, serum electrolytes, and estimated glomerular filtration rate (eGFR). Preoperative anaemia, antihypertensive medication use, and baseline renal impairment were documented as potential risk factors.

Intraoperative Haemodynamic Monitoring: Standard intraoperative monitoring included electrocardiography, pulse oximetry, non-invasive or invasive blood pressure monitoring, end-tidal carbon dioxide, and temperature monitoring. MAP was recorded at intervals not exceeding five minutes throughout anaesthesia. Additional variables included heart rate, anaesthetic technique, surgical procedure, operative duration, estimated blood loss, intravenous fluid administration, blood transfusion, urine output, and vasopressor requirements. Haemodynamic management was performed by the attending anaesthesiologist according to institutional practice.

Assessment of Intraoperative Hypotension Burden: Intraoperative hypotension was primarily defined as MAP below 65 mmHg. Hypotension burden was evaluated using cumulative hypotension duration, number of hypotensive episodes, lowest recorded MAP, and area under the threshold (AUT). An episode was defined as a continuous period of MAP below 65 mmHg. Cumulative duration was expressed in minutes, while AUT was calculated by integrating the magnitude of MAP reduction below 65 mmHg over time and expressed as mmHg·minutes. Time-weighted average hypotension burden was calculated by dividing AUT by the total intraoperative monitoring time. Additional analyses considered MAP below 60 mmHg and cumulative hypotension exceeding five and ten minutes.

Assessment of Postoperative Acute Kidney Injury: The primary outcome was postoperative AKI within seven days following surgery, assessed using serum creatinine-based Kidney Disease: Improving Global Outcomes (KDIGO) criteria. AKI was defined as an increase in serum creatinine of ≥0.3 mg/dL within 48 hours or ≥1.5 times baseline within seven days. Serum creatinine measurements were assessed at 24 and 48 hours and subsequently up to postoperative day seven, according to available follow-up. AKI severity was classified into KDIGO stages 1, 2, and 3. Postoperative urine output, requirement for renal replacement therapy, intensive care admission, and hospital length of stay were also documented.

Outcome Measures: The primary outcome was the association between cumulative intraoperative hypotension burden and postoperative AKI. Secondary outcomes included the relationship between AKI and hypotension severity, duration, time-weighted burden, baseline renal function, pre-existing comorbidities, blood loss, fluid administration, and vasopressor use. Patients were categorised into AKI and non-AKI groups for comparative analysis.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent-samples t-tests or Mann–Whitney U tests were used for continuous variables, while chi-square or Fisher's exact tests were applied to categorical variables. Binary logistic regression was used to examine the association between hypotension burden and postoperative AKI, with adjustment for selected clinically relevant confounders according to the number of AKI events. Odds ratios with 95% confidence intervals were calculated. Receiver operating characteristic (ROC) curve analysis was performed to assess the discriminatory ability of hypotension burden for predicting AKI. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Baseline Demographic and Clinical Characteristics of the Study Population

[Table 1] presents the baseline demographic and clinical characteristics of 92 patients undergoing major non-cardiac surgery, of whom 22 (23.91%) developed postoperative acute kidney injury (AKI), while 70 (76.09%) did not. The mean age was higher in the AKI group (60.3 ± 11.4 years) than in the non-AKI group (56.2 ± 12.3 years), although the difference was statistically insignificant (p=0.156). Male patients constituted 55.43% of the total population, while BMI and the prevalence of diabetes mellitus, hypertension, and ASA physical status III–

IV showed no significant differences between groups (p>0.05). Pre-existing chronic kidney disease was significantly more prevalent among patients developing AKI (50.00% versus 8.57%; p<0.001). Baseline eGFR was significantly lower in the AKI group (65.2 ± 22.6 versus 87.2 ± 18.4 mL/min/1.73 m²; p<0.001). Similarly, baseline serum creatinine was significantly higher among patients developing AKI (1.59 ± 0.48 versus 1.10 ± 0.34 mg/dL; p<0.001), indicating a strong association between impaired preoperative renal function and postoperative AKI.

[Table 2] Comparison of Intraoperative Characteristics and Haemodynamic Parameters Between AKI and Non-AKI Groups

[Table 2] compares the intraoperative characteristics and haemodynamic parameters between patients with and without postoperative AKI. Major abdominal surgery was the most frequently performed procedure (41.30%), followed by orthopaedic (28.26%), vascular (16.30%), and other non-cardiac surgeries (14.13%), with no significant difference in surgical distribution between groups (p=0.870). Operative duration, estimated blood loss, intravenous crystalloid administration, blood transfusion, and vasopressor requirements were comparable between groups (p>0.05). However, cumulative hypotension duration was significantly greater in the AKI group, with a median of 14.0 minutes compared with 7.5 minutes in the non-AKI group (p<0.001). Patients developing AKI also experienced more hypotensive episodes and a significantly lower mean minimum intraoperative MAP (55.1 ± 4.9 versus 60.0 ± 5.6 mmHg; p<0.001). Furthermore, the median area under the MAP 65 mmHg threshold was substantially greater in the AKI group (94.8 versus 26.0 mmHg·min; p<0.001). Time-weighted average hypotension burden was also significantly elevated among AKI patients (0.49 versus 0.15 mmHg; p<0.001), demonstrating a significant association between greater intraoperative hypotension burden and postoperative AKI.

[Table 3] Association Between Intraoperative Hypotension Duration, Severity, and Postoperative Acute Kidney Injury

[Table 3] demonstrates a significant association between the duration and severity of intraoperative hypotension and postoperative AKI. Among the 10 patients who did not experience intraoperative hypotension, no postoperative AKI was observed. The incidence of AKI progressively increased with cumulative hypotension duration, from 10.00% among patients experiencing 1–5 minutes of hypotension to 16.67% among those experiencing 6–10 minutes, reaching 42.11% in patients with hypotension exceeding 10 minutes (p=0.005). When patients were categorised according to cumulative hypotension duration, AKI incidence was significantly higher among those experiencing more than 10 minutes of hypotension than among those with 10 minutes or less (42.11% versus 11.11%; p=0.001). Similarly, patients whose lowest intraoperative MAP decreased below 60 mmHg demonstrated a significantly higher AKI incidence than those maintaining MAP at or above 60 mmHg (36.00% versus 9.52%; p=0.003).

[Table 4] Postoperative Acute Kidney Injury Severity and Clinical Outcomes

[Table 4] presents the severity of postoperative AKI and its association with subsequent clinical outcomes among the 92 patients. Postoperative AKI developed in 22 patients (23.91%), with KDIGO stage 1 accounting for the majority of cases (14 patients; 63.64%), followed by stage 2 in 6 patients (27.27%) and stage 3 in 2 patients (9.09%). Intensive care unit admission was significantly more frequent among patients developing AKI than among those without AKI (50.00% versus 15.71%; p=0.001). The mean duration of hospitalisation was also significantly longer in the AKI group (8.4 ± 1.6 days) compared with the non-AKI group (5.4 ± 1.8 days; p<0.001). Renal replacement therapy was required in one patient (4.55%) with AKI, while no patient in the non-AKI group required renal replacement therapy (p=0.239). Similarly, one in-hospital death (4.55%) occurred in the AKI group compared with none in the non-AKI group (p=0.239).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (N=92)

Parameters	Total (n=92)	AKI (n=22)	Non-AKI (n=70)	p-value
Age (years), mean ± SD	57.2 ± 12.1	60.3 ± 11.4	56.2 ± 12.3	0.156
Male sex	51 (55.43%)	11 (50.00%)	40 (57.14%)	0.557
BMI (kg/m²), mean ± SD	26.2 ± 3.4	25.6 ± 3.3	26.5 ± 3.4	0.268
Diabetes mellitus	33 (35.87%)	9 (40.91%)	24 (34.29%)	0.572
Hypertension	42 (45.65%)	9 (40.91%)	33 (47.14%)	0.609
Pre-existing chronic kidney disease	17 (18.48%)	11 (50.00%)	6 (8.57%)	<0.001
ASA physical status III–IV	27 (29.35%)	7 (31.82%)	20 (28.57%)	0.771
Haemoglobin (g/dL), mean ± SD	12.09 ± 1.54	11.68 ± 1.28	12.21 ± 1.60	0.117
Baseline eGFR (mL/min/1.73 m²), mean ± SD	81.9 ± 21.6	65.2 ± 22.6	87.2 ± 18.4	<0.001
Baseline serum creatinine (mg/dL), mean ± SD	1.21 ± 0.43	1.59 ± 0.48	1.10 ± 0.34	<0.001

Table 2: Comparison of Intraoperative Characteristics and Haemodynamic Parameters Between AKI and Non-AKI Groups

Parameters	Total (n=92)	AKI (n=22)	Non-AKI (n=70)	p-value
Type of surgery				0.870
Major abdominal surgery	38 (41.30%)	10 (45.45%)	28 (40.00%)	—

Orthopaedic surgery	26 (28.26%)	6 (27.27%)	20 (28.57%)	—
Vascular surgery	15 (16.30%)	4 (18.18%)	11 (15.71%)	—
Other non-cardiac surgery	13 (14.13%)	2 (9.09%)	11 (15.71%)	—
Operative duration (minutes), mean ± SD	217.7 ± 52.9	220.6 ± 50.2	216.7 ± 54.1	0.759
Estimated blood loss (mL), median (IQR)	410 (260–590)	430 (252–610)	395 (260–582)	0.745
Intravenous crystalloid volume (mL), median (IQR)	2450 (2188–2850)	2400 (1962–2838)	2500 (2250–2838)	0.472
Blood transfusion required	11 (11.96%)	4 (18.18%)	7 (10.00%)	0.450
Vasopressor administration	37 (40.22%)	9 (40.91%)	28 (40.00%)	0.940
Cumulative MAP <65 mmHg duration (minutes), median (IQR)	9.0 (4.0–16.0)	14.0 (9.5–28.0)	7.5 (4.0–14.8)	<0.001
Hypotensive episodes, median (IQR)	2 (1–2)	2 (2–4)	1 (1–2)	<0.001
Lowest intraoperative MAP (mmHg), mean ± SD	58.8 ± 5.8	55.1 ± 4.9	60.0 ± 5.6	<0.001
Area under MAP 65 mmHg threshold (mmHg·min), median (IQR)	39.8 (9.0–104.5)	94.8 (55.3–222.0)	26.0 (6.9–83.3)	<0.001
Time-weighted average hypotension (mmHg), median (IQR)	0.19 (0.04–0.53)	0.49 (0.23–0.91)	0.15 (0.03–0.29)	<0.001

Table 3: Association Between Intraoperative Hypotension Duration, Severity, and Postoperative Acute Kidney Injury

Hypotension parameters	Total patients	AKI cases	Non-AKI cases	AKI incidence	p-value
Cumulative duration of MAP <65 mmHg					0.005
No hypotension	10	0	10	0.00%	—
1–5 minutes	20	2	18	10.00%	—
6–10 minutes	24	4	20	16.67%	—
>10 minutes	38	16	22	42.11%	—
Duration-based comparison					0.001
Hypotension >10 minutes	38	16	22	42.11%	—
Hypotension ≤10 minutes	54	6	48	11.11%	—
Severity-based comparison					0.003
Lowest MAP <60 mmHg	50	18	32	36.00%	—
Lowest MAP ≥60 mmHg	42	4	38	9.52%	—

Table 4: Postoperative Acute Kidney Injury Severity and Clinical Outcomes

Parameters	Total (n=92)	AKI (n=22)	Non-AKI (n=70)	p-value
KDIGO stage 1 AKI	14 (15.22%)	14 (63.64%)	—	—
KDIGO stage 2 AKI	6 (6.52%)	6 (27.27%)	—	—
KDIGO stage 3 AKI	2 (2.17%)	2 (9.09%)	—	—
ICU admission	22 (23.91%)	11 (50.00%)	11 (15.71%)	0.001
Hospital stay (days), mean ± SD	6.1 ± 2.2	8.4 ± 1.6	5.4 ± 1.8	<0.001
Renal replacement therapy	1 (1.09%)	1 (4.55%)	0 (0.00%)	0.239
In-hospital mortality	1 (1.09%)	1 (4.55%)	0 (0.00%)	0.239

Table 5. Multivariable Logistic Regression Analysis of Factors Associated With Postoperative Acute Kidney Injury

Variables	Adjusted odds ratio (aOR)	95% confidence interval	p-value
Cumulative hypotension duration (per 5-minute increase)	2.42	1.56–3.75	<0.001
Baseline eGFR (per 10 mL/min/1.73 m² decrease)	2.52	1.63–3.88	<0.001
Age (per 10-year increase)	1.73	0.92–3.27	0.091

[Table 5] Multivariable Logistic Regression Analysis of Factors Associated With Postoperative Acute Kidney Injury

[Table 5] presents the multivariable logistic regression analysis examining factors independently associated with postoperative AKI among patients undergoing major non-cardiac surgery. After adjustment for age and baseline renal function, cumulative intraoperative hypotension duration demonstrated a statistically significant association with postoperative AKI, with each additional five minutes of hypotension associated with increased odds of AKI (aOR=2.42; 95% CI: 1.56–3.75; p<0.001). Reduced baseline eGFR was also significantly associated with postoperative AKI, with every 10 mL/min/1.73 m² decrease corresponding to higher odds of renal injury (aOR=2.52; 95% CI: 1.63–3.88; p<0.001). Increasing age demonstrated a positive association with postoperative AKI, with an adjusted odds ratio of 1.73 per 10-year increase (95% CI: 0.92–3.27), although this association did not reach statistical significance (p=0.091). The findings suggest that prolonged intraoperative hypotension and impaired preoperative renal function were the principal independent factors associated with postoperative AKI in the fitted model.

DISCUSSION

The present study evaluated the association between intraoperative hypotension burden and postoperative acute kidney injury among 92 adults undergoing major non-cardiac surgery. Postoperative AKI was observed in 22 patients (23.91%), while 70 patients (76.09%) maintained stable postoperative renal function. The mean age of patients developing AKI was 60.3 ± 11.4 years compared with 56.2 ± 12.3 years among patients without AKI (p=0.156). These findings may be compared with Grams et al. (2016),

who conducted a large observational study involving 161,185 major surgical hospitalisations and reported postoperative AKI in 11.80% of cases. Their study demonstrated that AKI incidence differed according to surgical category, reaching 18.70% following cardiac surgery and 13.20% following general surgery. The incidence in the present dataset (23.91%) was higher than the overall incidence reported by Grams et al., potentially reflecting differences in baseline renal function, surgical case mix, patient selection, and perioperative clinical characteristics. Their findings also demonstrated that postoperative AKI represents an important complication across different surgical specialties, supporting the clinical relevance of evaluating perioperative renal outcomes in patients undergoing major non-cardiac surgery.[7] Preoperative renal impairment emerged as an important factor associated with postoperative AKI in the present study. Pre-existing chronic kidney disease was observed in 50.00% of patients developing AKI compared with only 8.57% of patients without AKI ($p<0.001$). Furthermore, baseline eGFR was significantly lower in the AKI group (65.2 ± 22.6 versus 87.2 ± 18.4 mL/min/1.73 m²; $p<0.001$), whereas serum creatinine was significantly higher (1.59 ± 0.48 versus 1.10 ± 0.34 mg/dL; $p<0.001$). Comparable findings were reported by Mathis et al. (2020) in a multicentre investigation involving 138,021 patients undergoing major non-cardiac surgery, among whom 12,431 (9.00%) developed postoperative AKI. Their analysis demonstrated that preoperative chronic kidney disease was an important predictor of postoperative renal complications, with adjusted odds ratios of 2.03 (95% CI: 1.82–2.26) for CKD stage 3b and 3.89 (95% CI: 3.47–4.38) for CKD stage 4. The consistency between these findings and the present observations suggests that reduced baseline renal reserve may increase susceptibility to postoperative renal dysfunction. These findings emphasise the importance of comprehensive preoperative renal assessment when evaluating perioperative AKI risk.[8] Intraoperative haemodynamic disturbances demonstrated a significant association with postoperative AKI in the present study. The median cumulative duration of MAP below 65 mmHg was significantly higher among patients developing AKI than among those without AKI (14.0 versus 7.5 minutes; $p<0.001$). Similarly, the median area under the MAP 65 mmHg threshold was substantially greater in the AKI group (94.8 versus 26.0 mmHg·min; $p<0.001$). In comparison, Maheshwari et al. (2018) evaluated 42,825 patients undergoing elective non-cardiac surgery and reported intraoperative hypotension, defined as MAP below 65 mmHg, in 30,423 patients (71.00%). Postoperative AKI developed in 2,328 patients, approximately 5.00% of their cohort. Their analysis demonstrated that each doubling of hypotension duration was associated with increased odds of postoperative AKI (OR=1.05; 95% CI: 1.02–1.07; $p<0.001$). They also reported significant associations between the combined magnitude and duration of hypotension and renal injury during both pre-incision and post-incision periods.[9] The severity of intraoperative hypotension was another important parameter associated with postoperative renal impairment. In the present study, the mean lowest intraoperative MAP was significantly lower among patients developing AKI (55.1 ± 4.9 mmHg) compared with those without AKI (60.0 ± 5.6 mmHg; $p<0.001$). Furthermore, AKI incidence was 36.00% among patients whose MAP decreased below 60 mmHg compared with 9.52% among patients maintaining MAP at or above 60 mmHg ($p=0.003$). These findings are comparable with those of Sun et al. (2015), who investigated 5,127 patients undergoing elective non-cardiac surgery and reported postoperative AKI in 324 patients (6.30%). Their study demonstrated that exposure to MAP below 55 mmHg for 11–20 minutes was associated with significantly increased odds of AKI (adjusted OR=2.34; 95% CI: 1.35–4.05), increasing to 3.53 (95% CI: 1.51–8.25) when hypotension persisted for more than 20 minutes. Similarly, MAP below 60 mmHg for 11–20 minutes was associated with an adjusted OR of 1.84 (95% CI: 1.11–3.06).[10] A progressive increase in postoperative AKI incidence was observed with increasing cumulative hypotension duration in the present study. No AKI occurred among the 10 patients without recorded hypotension, whereas the incidence increased to 10.00% among those experiencing 1–5 minutes, 16.67% among those experiencing 6–10 minutes, and 42.11% among those experiencing more than 10 minutes of hypotension ($p=0.005$). Patients exposed to MAP below 65 mmHg for more than 10 minutes had significantly higher AKI incidence than those with shorter exposure (42.11% versus 11.11%; $p=0.001$). Similar duration-dependent associations were demonstrated by Walsh et al. (2013) in a study involving 33,330 non-cardiac surgical procedures, in which postoperative AKI occurred in 2,478 cases (7.40%). Compared with patients without MAP below 55 mmHg, the adjusted odds ratios for AKI were 1.18, 1.19, 1.32, and 1.51 for hypotension durations of 1–5, 6–10, 11–20, and more than 20 minutes, respectively.[11] The present study also evaluated cumulative hypotension burden using the area under the threshold and time-weighted average hypotension. The median time-weighted average hypotension was significantly higher in patients developing AKI than in those without AKI (0.49 versus 0.15 mmHg; $p<0.001$). Similarly, the median area under the MAP 65 mmHg threshold was approximately 3.65 times higher in the AKI group (94.8 versus 26.0 mmHg·min). These findings may be compared with Salmasi et al. (2017), who analysed 57,315 non-cardiac surgical patients and evaluated hypotension using absolute blood pressure thresholds, relative reductions from baseline, cumulative exposure duration, and time-weighted measurements. Their analysis demonstrated progressively greater risks of kidney and myocardial

injury with MAP below 65 mmHg and relative reductions exceeding approximately 20%. Exposure to MAP below 65 mmHg for 13–28 minutes was associated with increased odds of AKI (OR=1.20; 97.5% CI: 1.02–1.40). These observations are consistent with the present findings, suggesting that integrating the depth and duration of hypotension may provide a more comprehensive assessment of haemodynamic burden than evaluating minimum MAP alone.[12] Postoperative AKI was associated with increased clinical complications and healthcare utilisation in the present study. Among the 22 patients developing AKI, KDIGO stage 1 accounted for 63.64% of cases, followed by stage 2 in 27.27% and stage 3 in 9.09%. ICU admission was significantly more frequent among AKI patients than non-AKI patients (50.00% versus 15.71%; p=0.001), while mean hospital stay was significantly longer (8.4 ± 1.6 versus 5.4 ± 1.8 days; p<0.001). These findings are comparable with those reported by Worrall et al. (2025), who investigated 13,790 elective major non-cardiac surgical episodes and identified postoperative AKI in 1,334 cases (9.70%). Their study demonstrated that stage 1 AKI accounted for 61.30% of AKI cases, closely resembling the proportion observed in the present dataset. ICU admission occurred in 41.50% of patients with AKI compared with 27.80% of those without AKI, while median postoperative hospital stay was 7 versus 4 days, respectively (p<0.001). They additionally reported an eightfold increase in the odds of in-hospital mortality among AKI patients (OR=8.00; 95% CI: 5.10–12.50).[13] Multivariable logistic regression analysis in the present study demonstrated that cumulative hypotension duration and impaired baseline renal function were independently associated with postoperative AKI. Each additional five minutes of intraoperative hypotension was associated with increased odds of AKI (aOR=2.42; 95% CI: 1.56–3.75; p<0.001), while every 10 mL/min/1.73 m² reduction in baseline eGFR was associated with higher odds of postoperative renal injury (aOR=2.52; 95% CI: 1.63–3.88; p<0.001). Increasing age demonstrated a positive but statistically insignificant association (aOR=1.73; 95% CI: 0.92–3.27; p=0.091). Comparable findings were reported by Hallqvist et al. (2018) in an observational study involving 470 patients undergoing non-cardiac surgery, among whom 127 (27.00%) developed perioperative AKI. Their multivariable analysis demonstrated that an intraoperative systolic blood pressure reduction exceeding 50% from baseline was associated with significantly increased odds of AKI (adjusted OR=2.27; 95% CI: 1.20–4.30; p=0.013). Although the hypotension definitions and effect estimates were not directly interchangeable, both studies demonstrated an independent association between intraoperative haemodynamic instability and postoperative renal injury. Taken together, these observations support careful assessment of intraoperative blood pressure exposure and baseline renal function, while recognising that observational associations do not establish causation and that estimates from a small cohort require cautious interpretation.[14]

CONCLUSION

The present study demonstrated a significant association between increased intraoperative hypotension burden and postoperative acute kidney injury in adults undergoing major non-cardiac surgery. Prolonged hypotension, particularly MAP below 65 mmHg for more than 10 minutes, was associated with a higher incidence of AKI. Reduced baseline eGFR and pre-existing renal impairment were additional important risk factors. Postoperative AKI was associated with increased ICU admissions and prolonged hospitalisation. These findings suggest that careful intraoperative haemodynamic monitoring and timely correction of hypotension may help reduce postoperative renal complications, although prospective validation is required.

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