

Research Article

Intraoperative hypotension associated with postoperative acute kidney injury in hypertension patients undergoing non-cardiac surgery: a retrospective cohort study

Jin Li^{1,†}, Yeshuo Ma^{2,†}, Yang Li^{1,†}, Wen Ouyang¹, Zongdao Liu², Xing Liu¹, Bo Li³, Jie Xiao⁴, Daqing Ma⁵, and Yongzhong Tang^{1,6,*}

¹Department of Anesthesiology, Third Xiangya Hospital, Central South University, 138 Tongzipo Road, Changsha, 410013, China, ²Department of Geriatrics, Third Xiangya Hospital, Central South University, 138 Tongzipo Road, Changsha, 410013, China, ³Operation Center, Third Xiangya Hospital, Central South University, 138 Tongzipo Road, Changsha, 410013, China, ⁴Department of Emergency, Third Xiangya Hospital, Central South University, 138 Tongzipo Road, Changsha, 410013, China, ⁵Division of Anesthetics, Pain Medicine & Intensive Care, Department of Surgery and Cancer, Chelsea and Westminster Hospital, Imperial College London, South Kensington Campus, London SW7 2AZ, UK and ⁶Clinical Research Center, Third Xiangya Hospital, Central South University, 138 Tongzipo Road, Changsha, 410013, China

*Correspondence. E-mail: tangyongzhong@csu.edu.cn

[†]Jin Li, Yeshuo Ma and Yang Li contributed equally to this work.

Received 25 July 2023; Revised 1 December 2023; Accepted 8 May 2024

Abstract

Background: Acute kidney injury (AKI) is a common surgical complication and is associated with intraoperative hypotension. However, the total duration and magnitude of intraoperative hypotension associated with AKI remains unknown. In this study, the causal relationship between the intraoperative arterial pressure and postoperative AKI was investigated among chronic hypertension patients undergoing non-cardiac surgery.

Methods: A retrospective cohort study of 6552 hypertension patients undergoing non-cardiac surgery (2011 to 2019) was conducted. The primary outcome was AKI as diagnosed with the Kidney Disease-Improving Global Outcomes criteria and the primary exposure was intraoperative hypotension. Patients' baseline demographics, pre- and post-operative data were harvested and then analyzed with multivariable logistic regression to assess the exposure–outcome relationship.

Results: Among 6552 hypertension patients, 579 (8.84%) had postoperative AKI after non-cardiac surgery. The proportions of patients admitted to ICU (3.97 vs. 1.24%, $p < 0.001$) and experiencing all-cause death (2.76 vs. 0.80%, $p < 0.001$) were higher in the patients with postoperative AKI. Moreover, the patients with postoperative AKI had longer hospital stays (13.50 vs. 12.00 days, $p < 0.001$). Intraoperative mean arterial pressure (MAP) < 60 mmHg for > 20 min was an independent risk factor of postoperative AKI. Furthermore, MAP < 60 mmHg for > 10 min was also an independent risk factor of postoperative AKI in patients whose MAP was measured invasively in the subgroup analysis.

Conclusions: Our work suggested that MAP < 60 mmHg for > 10 min measured invasively or 20 min measured non-invasively during non-cardiac surgery may be the threshold of postoperative AKI

development in hypertension patients. This work may serve as a perioperative management guide for chronic hypertension patients.

Trial registration: clinical trial number: ChiCTR2100050209 (8/22/2021).

<http://www.chictr.org.cn/showproj.aspx?proj=132277>.

Key words: Acute kidney injury, Intraoperative hypotension, Non-cardiac surgery, Chronic hypertension

Highlights

- We explored the appropriately perioperative management of intraoperative blood pressure to prevent against postoperative acute kidney injury (AKI) in the hypertensive patients undergoing a non-cardiac surgery.
- MAP less than 60 mmHg for more than 20 min was an independent risk factor of postoperative AKI, and for more than 10 min in the subgroup of invasive MAP.
- Our results provided the important evidences for the rational management of intraoperative MAP in the hypertensive patients. It also suggested that invasive measurement could reflect the true values of blood pressure more exactly.

Background

Acute kidney injury (AKI) is a common surgical complication. Previous studies have shown that surgery-related AKI occurs in ~18.3% of hospitalized patients [1], up to 74% of critically ill patients [2,3], 8.2–15.1% of patients undergoing noncardiac surgery [4,5] and 36–45% of patients undergoing cardiac surgery [6,7]. Importantly, postoperative AKI not only prolongs length of hospital stay but also leads to chronic kidney disease in some patients, which requires long-term renal replacement therapy and causes a heavy economic burden [8,9]. Perioperative hypotension was reported to be most prevalent during the intraoperative period. Patients who had hypertension or dyslipidemia were more likely to experience hypotension intraoperatively [10], and advanced age, high American Society of Anesthesiologists (ASA) score, anesthesia method and surgical position were also risk factors for intraoperative hypotension [11,12]. Thus, intraoperative hypotension may be an important cause of postoperative AKI [13]. The morbidity of postoperative AKI decreases with active management of intraoperative blood pressure [14,15]. Therefore, it is urgent to explore the appropriate perioperative management of intraoperative blood pressure to prevent postoperative AKI in patients undergoing noncardiac surgery.

Previous studies demonstrated that intraoperative hypotension was associated with postoperative AKI [14,16,17]. Indeed, an intraoperative mean arterial pressure (MAP) < 60 mmHg for 11–20 min and <55 mmHg for >10 min was associated with postoperative AKI in patients who underwent noncardiac surgery [14]. Our previous report also suggested that the risk of postoperative AKI increased significantly in patients who underwent noncardiac surgery when their intraoperative MAP was <55 mmHg for >10 min [15]. However, the threshold of intraoperative hypotension may not be useful for hypertensive patients whose autonomic regulation function curve of renal blood

flow often shifts to the right [18–20]. Chronic hypertension is one of the most common chronic diseases, affecting 1.4 billion patients worldwide, and a management guide for intraoperative MAP in these patients is not yet available [20,21]. Therefore, we conducted a retrospective cohort study to investigate the relationships between various levels of intraoperative MAP and total duration and postoperative AKI in hypertensive patients who underwent noncardiac surgery.

Methods

Study design and cohort selection

This retrospective cohort study of hypertensive patients who underwent noncardiac surgery from December 2011 to December 2019 was approved by the Ethics Committee (R21044) of Third Xiangya Hospital, Central South University, Hunan, China. The requirement for written informed consent was waived. The enrollment criteria included hypertensive patients who underwent noncardiac surgery at first admission and were aged ≥18 years. Patients with the following conditions were excluded: kidney transplantation, duration of surgery <60 min, loss of postoperative serum creatinine and loss of 30% of perioperative variables in a single patient (including loss of anesthesia and loss of intraoperative MAP). Ultimately, 6552 hypertensive patients were enrolled in the study, see Figure 1. Clinical trial number: ChiCTR2100050209 (8/22/2021). This manuscript adheres to the applicable STROBE guidelines.

Primary and secondary outcomes

The primary outcome was AKI diagnosed with the Kidney Disease-Improving Global Outcomes criteria [22], namely, a 0.3 mg/dl increase in creatinine over the preoperative value during the first two postoperative days or >1.5-fold increase during the first seven postoperative days. The secondary

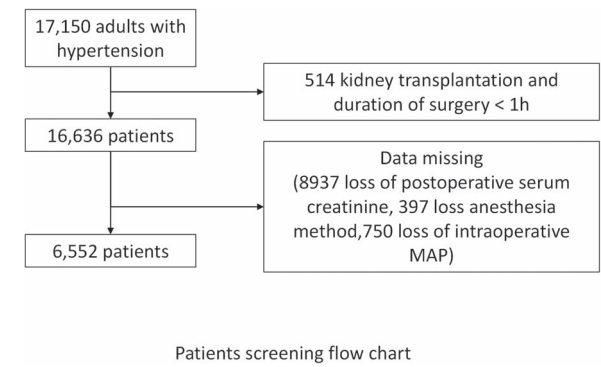


Figure 1. Flowchart of patients selection

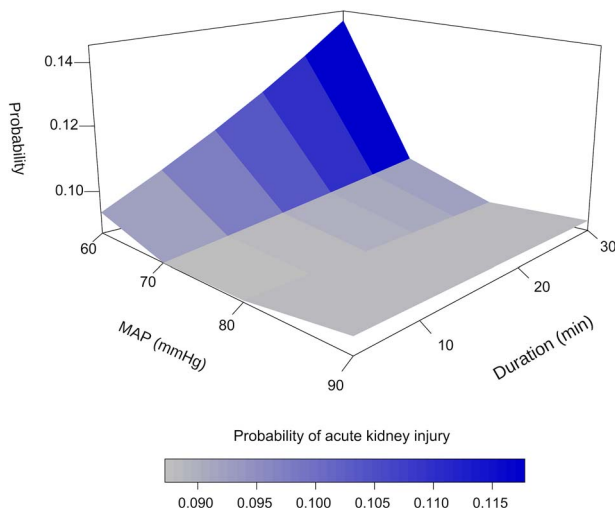


Figure 2. Correlation between the risk of postoperative AKI and the lower and long total duration of MAP. AKI acute kidney injury, MAP mean arterial pressure

outcomes included intensive care unit (ICU) admission, length of hospital stay and all-cause death.

Intraoperative blood pressure

The intraoperative MAP was extracted from the electronic medical records. When an arterial catheter was used, MAP was recorded at 30 s intervals. Without the arterial catheter line, noninvasive blood pressure was measured at 5 min intervals. In our study, blood pressure measured with an arterial catheter was recorded in 2787 patients (42.54%). Blood pressure values between measurements were linearly interpolated. According to our previous study [15], MAP ≤ 60 mmHg was defined as hypotension, and the total length of intraoperative hypotension was calculated.

Data sources

A formal sample size calculation was not performed because the effect sizes were unknown during the time of study design. Demographics and perioperative data, including diagnosis,

blood pressure, complications, laboratory examinations, pre-operative medicines, types of surgery, intraoperative conditions and postoperative outcomes, were extracted from the electronic medical records with Lex Clinical Research Data Warehouse Software (Le9 Healthcare Technology, Shanghai, China). All the data were also checked manually to ensure completeness and accuracy.

Statistical analysis

We performed multiple imputation to recover missing data (Table S1, see online supplementary material). Continuous variables are expressed as the mean $\pm$ SD if normally distributed; otherwise, non-normally distributed data are expressed as the median (interquartile range). The data was analyzed by Student's t test or Kruskal–Wallis rank-sum test followed by the least significant difference *post hoc* test wherever appropriate. Categorical variables are presented as numbers with proportions and were analyzed by the chi-square test. Bivariate analyses were used to compare the characteristics of the cohort with and without intraoperative hypotension for different MAP thresholds. Using line and curve fitting (restricted cubic spline), a preliminary analysis of the relationship between MAP exposure level and cumulative time during surgery and postoperative AKI was performed. Univariable logistic regression and collinearity analysis were conducted to select the variables that were significant at $p < 0.05$ and had a variance expansion factor < 4 . We then performed multivariate logistic regression analysis to calculate the odds ratio of postoperative AKI for different durations of operation (0, 1–5, 6–10, 11–20, > 20 min) with MAP ≤ 60 mmHg. To assess the robustness of our findings, a sensitivity verification was performed in five subgroups (general anesthesia, invasively measured MAP, without diabetes, estimated glomerular filtration rate (eGFR) > 60 ml/min/1.73 m², primary hypertension) and reported as adjusted odds ratios and associated 95% confidence intervals and p values. The data were analyzed by using SAS version 9.4 software (SAS Institute, Inc., Cary, NC, USA) and CRAN R (v3.4.1). A two-tailed p value < 0.05 was considered to indicate statistical significance.

Results

Primary and secondary outcomes

A total of 6552 surgeries were ultimately included in this study (Figure 1), and 579 (8.84%) patients developed postoperative AKI (Table 1). For the secondary outcome, the proportions of patients admitted to the ICU (3.97 *vs* 1.24%, $p < 0.001$) and experiencing all-cause death (2.76 *vs* 0.80%, $p < 0.001$) were greater among the patients with postoperative AKI (Table 2). Moreover, the patients with postoperative AKI had longer hospital stays (13.50 *vs* 12.00 days, $p < 0.001$).

The proportions of patients who experienced postoperative AKI increased when MAP was < 60 mmHg.

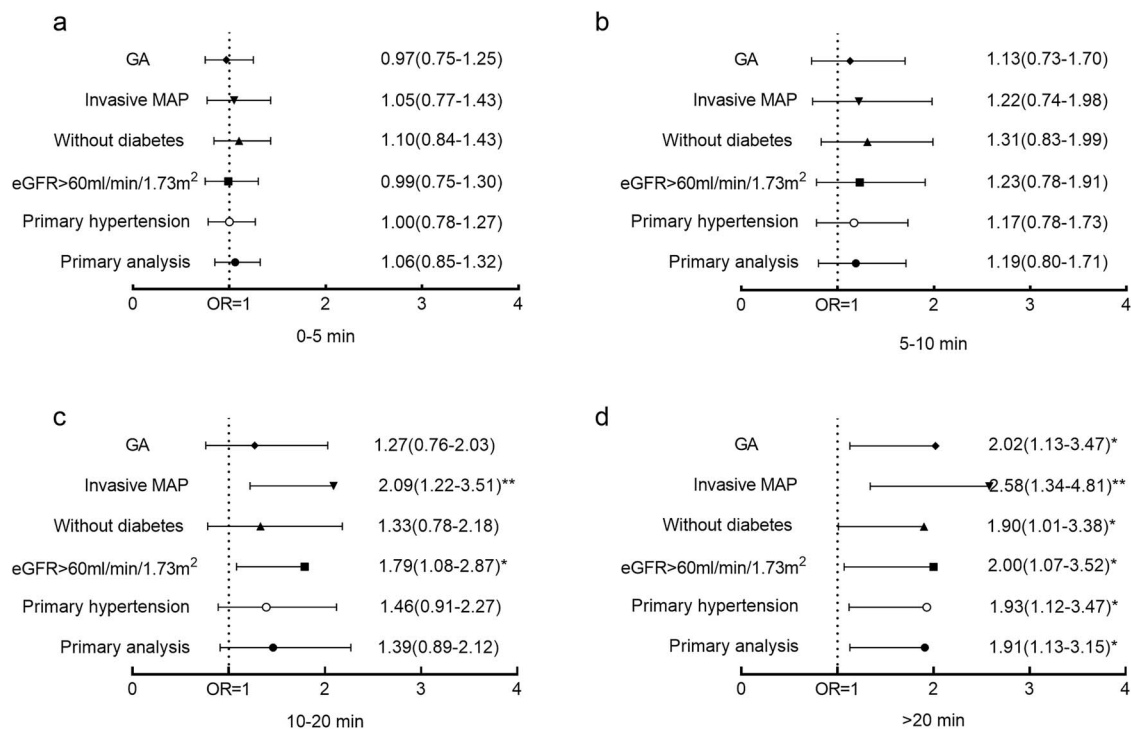


Figure 3. Sensitivity analyses between postoperative AKI and hypertensive patients with MAP ≤ 60 mmHg across different subgroups. (a) Total hypotension duration of 0-5 min. (b) Total hypotension duration of 5-10 min. (c) Total hypotension duration of 10-20 min. (d) Total hypotension duration of more than 20 min. AKI acute kidney injury, MAP mean arterial pressure, GA general anesthesia group

Table 1. Baseline characteristics of patients with and without postoperative AKI

Primary outcome	No AKI (n = 5973)	AKI (n = 579)	P value
Baseline			
Age (years)	61.95 ± 11.74	61.90 ± 13.26	0.654
Male, n (%)	2833 (47.43%)	326 (56.30%)	<0.001
Weight (kg)	63.90 ± 13.97	62.88 ± 12.54	0.294
Smoke	494 (8.28%)	54 (9.34%)	0.380
Alcohol	332 (5.57%)	34 (5.88%)	0.753
MAP (mmHg)	100.49 ± 9.58	101.20 ± 10.38	0.165
Hemoglobin (g/l)	123.61 ± 21.44	110.67 ± 26.38	<0.001
eGFR (ml/min/1.73 m ²)	79.78 ± 26.31	55.19 ± 35.69	<0.001
Albumin (g/l)	39.64 ± 5.09	36.52 ± 6.80	<0.001
Preoperative medications			
RASI	206 (3.45%)	19 (3.28%)	0.833
Calcium antagonists	27 (0.45%)	8 (1.38%)	0.003
Diuretics	174 (2.91%)	53 (9.15%)	<0.001
β-Blockers	66 (1.10%)	18 (3.11%)	<0.001
Lipid regulating drugs	23 (0.39%)	6 (1.04%)	0.331
Anticoagulant drugs	373 (6.24%)	77 (13.30%)	<0.001
Comorbidities			
Diabetes	1317 (22.05%)	165 (28.50%)	<0.001
Coronary heart disease	397 (11.72%)	47 (13.35%)	0.368
Respiratory diseases	565 (9.46%)	96 (16.58%)	<0.001
Emergency	675 (11.30%)	155 (26.77%)	<0.001
Cerebrovascular diseases	171 (5.05%)	26 (7.39%)	0.062
Primary hypertension	5540 (92.75%)	474 (81.87%)	<0.001
Tumor	1767 (29.58%)	186 (32.12%)	0.202

AKI acute kidney injury, MAP mean arterial pressure, RASI renin-angiotensin system inhibitors, eGFR estimated glomerular filtration rate

Table 2. Intraoperative characteristics of patients with and without postoperative AKI

Primary outcome	No AKI (n = 5973)	AKI (n = 579)	P value
ASA grade, n (%)			<0.001
I	74 (1.24%)	1 (0.17%)	
II	2455 (41.10%)	135 (23.32%)	
III	2945 (49.31%)	316 (54.58%)	
IV	357 (5.98%)	107 (18.48%)	
V	20 (0.33%)	7 (1.21%)	
Intraoperative			
Duration of surgery (min)	168.43 ± 91.75	170.78 ± 99.93	0.839
In fluids (ml)	2000 (1250–3000)	1740 (1000–2800)	<0.001
Out fluids (ml)	700 (400–1000)	600 (300–900)	<0.001
Hemorrhage (ml)	100 (50–300)	200 (50–500)	<0.001
Erythrocyte transfusions (ml)	0 (0–0)	0 (0–400)	<0.001
Vasoactive drugs	1143 (19.14%)	173 (29.88%)	<0.001
General anesthesia	4222 (70.68%)	433 (74.78%)	0.038
MAP ≤ 60, n (%)			<0.001
0 min	3832 (64.2%)	329 (56.8%)	
0–5 min	1521 (25.5%)	153 (26.4%)	
5–10 min	329 (5.5%)	39 (6.7%)	
10–20 min	186 (3.1%)	32 (5.5%)	
>20 min	103 (1.7%)	26 (4.5%)	
MAP ≤ 70, n (%)			0.03
0 min	2245 (37.6%)	211 (36.4%)	
0–5 min	1453 (24.3%)	125 (21.6%)	
5–10 min	630 (10.5%)	65 (11.2%)	
10–20 min	689 (11.5%)	58 (10%)	
>20 min	934 (15.6%)	118 (20.4%)	
Type of surgery			<0.001
General surgery	1764 (29.53%)	139 (24.01%)	
Urological	1047 (17.95%)	143 (24.70%)	
Gynecological	652 (11.37%)	29 (5.01%)	
Orthopedic	1100 (18.58%)	79 (13.65%)	
Neurosurgical	487 (8.16%)	35 (6.05%)	
Thoracic	379 (6.35%)	29 (5.01%)	
Others	701 (11.74%)	144 (24.87%)	
Secondary outcome			
Transferred to ICU (%)	74 (1.24%)	23 (3.97%)	<0.001
Hospital days (days)	12.00 (8.00–17.00)	13.50 (9.00–20.50)	<0.001
All-cause death (%)	48 (0.80%)	16 (2.76%)	<0.001

ASA American Society of Anesthesiologists, ICU intensive care unit, MAP mean arterial pressure, AKI acute kidney injury

As MAP decreased and the total duration increased, the mortality of postoperative AKI increased (Table S2, see online supplementary material).

There was a positive correlation between the risk of postoperative AKI and a shorter and longer total duration of MAP. The peak mortality of postoperative AKI was >14% when MAP was <60 mmHg for >20 min (Figure 2).

Logistic analysis

Single-variable logistic regression analysis revealed that MAP < 60 mmHg was associated with postoperative AKI (see supplementary Table S3 in the online supplementary material). In addition, many factors, such as age, sex, basic MAP, diabetes and secondary hypertension, were associated with postoperative AKI in patients who experienced MAP < 60 mmHg (Table S3, see online supplementary material).

After adjustment for age, sex, basic MAP, smoking status, alcohol consumption, emergency surgery, American Society of Anesthesiologists (ASA) grade, preoperative medication (diuretics, renin-angiotensin system inhibitors, calcium antagonists, β -blockers), hemoglobin, albumin, alanine aminotransferase, magnitude of surgery, type of anesthesia, total duration of surgery, in/out fluids, hemorrhage and vasoactive drugs, multivariate logistic regression analysis revealed that intraoperative hypotension with an intraoperative MAP < 60 mmHg for >20 min was a risk factor for postoperative AKI (Table 3). Furthermore, before the regression analysis, collinear analysis showed that there was no multicollinearity among these adjusted factors (kappa coefficient = 15.16).

Then, stepwise logistic regression was performed to further assess the data (Table 4). The logistic regression was

Table 3. Comparison of odds ratios of postoperative acute kidney injury in patients experiencing different total duration of intraoperative mean arterial pressure (MAP)

MAP, mmHg	Total duration of intraoperative hypotension (min)				
	0	0–5	5–10	10–20	>20
≤60	Reference	1.06 (0.85–1.32)	1.19 (0.80–1.71)	1.39 (0.89–2.12)	1.91 (1.13–3.15)*
≤70	Reference	0.81 (0.63–1.04)	1.05 (0.76–1.44)	0.79 (0.56–1.09)	1.03 (0.77–1.37)

Adjusted for age, sex, basic MAP, smoke, alcohol, emergency, ASA grade, preoperative medication (diuretics, renin–angiotensin system inhibitors, calcium antagonists, β -blockers), hemoglobin, albumin, alanine aminotransferase, magnitude of surgery, types of anesthesia, duration of surgery, in/out fluids amount, hemorrhage and vasoactive drugs

* $p < 0.05$

Table 4. Risk of postoperative AKI in patients experiencing different total duration of intraoperative hypotension

Total duration of intraoperative hypotension (min)	AKI OR (95% CI)		
	Model 1	Model 2	Model 3
0	Reference	Reference	Reference
0–5	1.17 (0.96–1.43)	1.27 (0.99–1.49)	1.06 (0.85–1.32)
5–10	1.38 (0.96–1.94)	1.44 (1.00–2.02)*	1.19 (0.80–1.71)
10–20	2.00 (1.33–2.92)***	2.10 (1.39–3.07)***	1.39 (0.89–2.12)
>20	2.94 (1.85–4.52)***	3.17 (2.00–4.90)***	1.91 (1.13–3.15)*

AKI acute kidney injury, MAP mean arterial pressure, OR odds ratio, CI confidence interval

Model 1: no adjustment. Model 2: adjusted for age, sex, basic MAP, smoke and alcohol. Model 3: adjusted for Model 2 plus diabetes, emergency, American Society of Anesthesiologists grade, preoperative medications (diuretics, renin–angiotensin system inhibitor, calcium antagonists, β -blockers), hemoglobin, albumin, alanine aminotransferase, magnitude of surgery, type of anaesthesia, duration of surgery, in/out fluids amount, erythrocyte transfusions and vasoactive drugs

* $p < 0.05$, *** $p < 0.001$

stable, and there was a strong association between intraoperative hypotension (MAP < 60 mmHg for > 20 min) and postoperative AKI.

Sensitivity analysis

To further confirm the relationship between intraoperative hypotension and postoperative AKI obtained from the above analysis, sensitivity analyses were performed in different subgroups. To eliminate the risks of comorbidities, type of anesthesia and the source of record, the subgroups were set up as primary hypertension, without diabetes, eGFR > 60 ml/min/1.73 m², general anesthesia and invasive MAP (Figure 3). Sensitivity analyses revealed that intraoperative hypotension (MAP < 60 mmHg for >20 min) was an independent risk factor for postoperative AKI in the subgroups of patients with primary hypertension, without diabetes and who were receiving general anesthesia. Interestingly, MAP < 60 mmHg for >10 min was an independent risk factor in the subgroups when the preoperative eGFR was >60 ml/min/1.73 m² and the MAP was invasively measured; this suggested that there was a strong and stable relationship between intraoperative hypotension and postoperative AKI in hypertension patients among the different subgroups.

Finally, variance inflation factor analysis was performed (Table S4, see online supplementary material), which further suggested nonmulticollinearity among the adjusted factors in the multivariate logistic regression.

Discussion

The results obtained in our study suggested that intraoperative hypotension with MAP < 60 mmHg for > 10 min when MAP was measured invasively and > 20 min when MAP was measured noninvasively was a significant independent risk factor for postoperative AKI in hypertensive patients who underwent noncardiac surgery. Patients who experienced postoperative AKI had an increased proportion of all-cause deaths with longer hospital stays.

Postoperative AKI is associated not only with an increase in short-term mortality [23,24] but also with a high risk of long-term complications [25]. Patients who experienced postoperative AKI were reported to be eight times more likely to die within 30 days of surgery [26]. Postoperative AKI is associated with intraoperative hypotension, which can be ameliorated with rational management of intraoperative blood pressure [14,15]. However, there is still no clear definition of the duration and severity of intraoperative hypotension that offers a rational management guide for intraoperative MAP to prevent postoperative AKI development in hypertensive patients [20], although a previous report showed that an intraoperative MAP < 60 mmHg for 11–20 min and <55 mmHg for >10 min was associated with postoperative AKI in patients who underwent noncardiac surgery [14]. Moreover, among patients who underwent noncardiac surgery and were <60 years old, our previous data suggested that the risk of postoperative AKI increased significantly when the intraoperative MAP was <55 mmHg for

>10 min [15]. Furthermore, our current work suggested that an intraoperative MAP < 60 mmHg for >20 min was a significant independent risk factor for postoperative AKI in hypertensive patients who underwent noncardiac surgery, which was consistent with the fact that the autonomic regulation function curve of renal blood flow shifted to the right in chronic hypertension patients [18,19]; this requires high perfusion pressure in hypertensive patients to effectively meet the high blood-flow requirements of the kidney cortex, while low blood pressure during surgery clearly compromises the blood supply [20]. Consequently, sublethal ischemia causes tubule cell injury and subsequently triggers an inflammatory response to promote postoperative AKI development [27]. Previous studies by our team and others revealed that toll-like receptors-4 (TLR4) was increased in renal tissue after ischemia–reperfusion injury, especially in renal tubular epithelial cells and vascular endothelial cells [28,29]. TLR4 activation not only promoted the release of proinflammatory mediators (including interleukin-6, interleukin-1, and tumor necrosis factor- α) and facilitated leukocyte migration and infiltration into the renal interstitium [30] but also sustained tubular necrosis and ultimately potentiated renal fibrosis [28]. More importantly, AKI, in turn, promoted significant systemic inflammatory responses in distant organs, including the lungs, heart, liver and brain, and resulted in multiorgan failure and death [31–33].

Previous studies have suggested that continuous blood pressure monitoring reflects real-time blood pressure stability better than noninvasive blood pressure monitoring during general anesthesia during orthopedic surgery [34]. Furthermore, invasive blood pressure measurements can detect hemodynamic perturbations more effectively and in a timely manner to facilitate treatment after surgery [35]. In addition, invasive blood pressure measurements and related management are related to lower mortality in critically ill patients with chronic hypertension [36,37]. Our data and those described above suggest that hypertensive patients may benefit more from invasive measurements of MAP than noninvasive measurements in terms of postoperative AKI, and invasive blood pressure measurements should be implemented in daily clinical practice for elderly hypertensive patients.

Our study has several limitations. First, the study is a retrospective observational study; therefore, our data may not accurately reflect this group of patients. Intraoperative blood pressure modulation in patients with chronic hypertension was lower than we expected, which may be related to the large range of our blood pressure segmentation and to the mild severity of preoperative hypertension in surgical patients. Second, although a large sample of patients was collected, the present study included data from only a single center; hence, the data reported herein may not represent patient generalizability. Further multicenter data analyses are urgently needed. Third, the effects of certain high-risk factors, such as high-grade ASA and kidney dysfunction, on the relationship between intraoperative MAP and postoperative AKI were not fully elucidated, and further research is needed.

Conclusions

In conclusion, we found that a definitive MAP of <60 mmHg lasted for >10 min when the MAP was measured invasively or >20 min when the MAP was measured noninvasively at the threshold for postoperative AKI development in hypertensive patients who underwent noncardiac surgery. Our work further suggested that invasive blood pressure monitoring should be implemented in our daily clinical practice in high-risk patients, e.g. those who are hypertensive and receive major surgery. Our work may indicate that rational intraoperative MAP management should be implemented to avoid MAPs < 60 min wherever necessary in hypertensive patients.

Abbreviations

AKI: Acute kidney injury; ASA: American Society of Anesthesiologists; eGFR: Estimated glomerular filtration rate; ICU: intensive care unit; MAP: Mean arterial pressure.

Supplementary data

Supplementary data is available at *Burns & Trauma Journal* online.

Funding

This work was supported by the Xiangya Bigdata foundation (to YZT), the Health Commission of Hunan Province Project D202303017033 (to YSM) and the Natural Science Foundation of Hunan Province 2021JJ31028 (to JX).

Authors' contributions

All authors designed the study and interpreted the data. JL, YL, JX and YM wrote the manuscript. WO, ZL and YT collected data. XL, BL and YT performed statistical analysis. DM and WO supervised the scientific process and critically revised the manuscript. YT approved the final version of the manuscript for publication.

Ethics approval and consent to participate

This research was approved by the Ethics Committee (R21044) of the Third Xiangya Hospital, Central South University, Hunan, China. Requirement for written informed consent was waived.

Conflict of interest

None declared.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

References

1. Zeng X, McMahon GM, Brunelli SM, Bates DW, Waikar SS. Incidence, outcomes, and comparisons across definitions of AKI in hospitalized individuals. *Clin J Am Soc Nephrol*. 2014;9:12–20.
2. Signe S, Marie SI, Isachsen MS, Nordhuus KM, Tveiten CK, Eken T, et al. Acute kidney injury in trauma patients admitted to the

- ICU: a systematic review and meta-analysis. *Intensive Care Med.* 2019;45:407–19.
3. Ulger F, Pehlivanlar Kucuk M, Kucuk AO, Ilkaya NK, Murat N, Bilgic B, *et al.* Evaluation of acute kidney injury (AKI) with RIFLE, AKIN, CK, and KDIGO in critically ill trauma patients. *Eur J Trauma Emerg Surg.* 2018;44:597–605.
 4. Tomozawa A, Ishikawa S, Shiota N, Cholvisudhi P, Makita K. Perioperative risk factors for acute kidney injury after liver resection surgery: an historical cohort study. *Can J Anaesth.* 2015;62:753–61.
 5. Slankamenac K, Breitenstein S, Held U, Beck-Schimmer B, Puhan MA, Clavien PA. Development and validation of a prediction score for postoperative acute renal failure following liver resection. *Ann Surg.* 2009;250:720–8.
 6. Graziani MP, Moser M, Bozzola CM, Galvez HM, Irman GJ, Alvarez PG, *et al.* Acute kidney injury in children after cardiac surgery: risk factors and outcomes. A retrospective, cohort study. *Arch Argent Pediatr.* 2019;117:e557–67.
 7. Xie X, Wan X, Ji X, Chen X, Liu J, Chen W, *et al.* Reassessment of acute kidney injury after cardiac surgery: a retrospective study. *Intern Med.* 2017;56:275–82.
 8. Moore PK, Hsu RK, Liu KD. Management of Acute Kidney Injury: Core curriculum 2018. *Am J Kidney Dis.* 2018;72: 136–48.
 9. Coca SG, Yusuf B, Shlipak MG, Garg AX, Parikh CR. Long-term risk of mortality and other adverse outcomes after acute kidney injury: a systematic review and meta-analysis. *Am J Kidney Dis.* 2009;53:961–73.
 10. Thanaboriboon C, Vanichvithya P, Jinaworn P. What is the risk of intraoperative cerebral oxygen desaturation in patients undergoing shoulder surgery in the beach chair position? *Clin Orthop Relat Res.* 2021;479:2677–87.
 11. Choi JW, Kim DK, Jeong HJ, Kim YR, Chung YJ, Son YH. Risk factors associated with hypotensive bradycardic events during open shoulder surgery in the beach chair position. *Korean J Anesthesiol.* 2021;74:38–44.
 12. Dai S, Li X, Yang Y, Cao Y, Wang E, Dong Z. A retrospective cohort analysis for the risk factors of intraoperative hypotension. *Int J Clin Pract.* 2020;74:e13521.
 13. Cao Y, Bass GA, Ahl R, Pourlofti A, Geijer H, Montgomery S, *et al.* The statistical importance of P-POSSUM scores for predicting mortality after emergency laparotomy in geriatric patients. *BMC Med Inform Decis Mak.* 2020;20:86.
 14. Sun LY, Wijesundera DN, Tait GA, Beattie WS. Association of intraoperative hypotension with acute kidney injury after elective noncardiac surgery. *Anesthesiology.* 2015;123:515–23.
 15. Tang YZ, Zhu CN, Liu JB, Wang A, Duan K, Li B, *et al.* Association of Intraoperative Hypotension with acute kidney injury after noncardiac surgery in patients younger than 60 years old. *Kidney Blood Press Res.* 2019;44:211–21.
 16. Rangasamy V, de Guerre L, Xu X, Schermerhorn ML, Novack V, Subramaniam B. Association between intraoperative hypotension and postoperative adverse outcomes in patients undergoing vascular surgery - a retrospective observational study. *J Cardiothorac Vasc Anesth.* 2020;S1053–0770: 31191–2.
 17. Agarwal A, Wei G, Boucher R, Ahmed F, Beddhu S. Modification of the effects of intensive systolic blood pressure control on kidney outcomes by baseline body mass index. *Nephrology (Carlton).* 2021;26:303–11.
 18. Wei J, Zhu J, Zhang J, Jiang S, Qu L, Wang L, *et al.* Aging impairs renal autoregulation in mice. *Hypertension.* 2020;75:405–12.
 19. Fan F, Geurts AM, Pabbidi MR, Ge Y, Zhang C, Wang S, *et al.* A mutation in gamma-Adducin impairs autoregulation of renal blood flow and promotes the development of kidney disease. *J Am Soc Nephrol.* 2020;31:687–700.
 20. Meng L, Wang Y, Zhang L, McDonagh DL. Heterogeneity and variability in pressure autoregulation of organ blood flow: lessons learned over 100+ years. *Crit Care Med.* 2019;47: 436–48.
 21. Meng L, Yu W, Wang T, Zhang L, Heerdt PM, Gelb AW. Blood pressure targets in perioperative care. *Hypertension.* 2018; 72:806–17.
 22. Tang Y-Z, Zeng P, Liao Y, Qin Z, Zhang H, Li B, *et al.* Correlation between perioperative parecoxib use and postoperative acute kidney injury in patients undergoing non-cardiac surgery: a retrospective cohort analysis. *BMJ Open.* 2021;11: e047840.
 23. Long TE, Helgason D, Helgadottir S, Palsson R, Gudbjartsson T, Sigurdsson GH, *et al.* Acute kidney injury after abdominal surgery: incidence, risk factors, and outcome. *Anesth Analg.* 2016;122:1912–20.
 24. Li JY, Yao YM, Tian Y. Early predictive value of high density lipoprotein cholesterol for secondary acute kidney injury in sepsis patients. *Zhonghua Shao Shang Yu Chuang Mian Xiu Fu Za Zhi.* 2022;38:130–6.
 25. Rewa O, Bagshaw SM. Acute kidney injury-epidemiology, outcomes and economics. *Nat Rev Nephrol.* 2014;10: 193–207.
 26. Kheterpal S, Tremper KK, Heung M, Rosenberg AL, Englesbe M, Shanks AM, *et al.* Development and validation of an acute kidney injury risk index for patients undergoing general surgery: results from a national data set. *Anesthesiology.* 2009;110:505–15.
 27. Huang WW, Zhu WZ, Mu DL, Ji XQ, Li XY, Ma D, *et al.* Intraoperative hypotension is associated with shortened overall survival after lung cancer surgery. *BMC Anesthesiol.* 2020; 20:160.
 28. Zhao H, Perez JS, Lu K, George AJ, Ma D. Role of toll-like receptor-4 in renal graft ischemia-reperfusion injury. *American journal of physiology Renal physiology.* 2014;306: F801–11.
 29. Gu J, Sun P, Zhao H, Watts HR, Sanders RD, Terrando N, *et al.* Dexmedetomidine provides renoprotection against ischemia-reperfusion injury in mice. *Crit Care.* 2011;15:R153.
 30. Xiang Y, Fu Y, Wu W, Tang C, Dong Z. Autophagy in acute kidney injury and maladaptive kidney repair. *Burns. Trauma.* 2023;11:tkac059.
 31. Ologunde R, Zhao H, Lu K, Ma D. Organ cross talk and remote organ damage following acute kidney injury. *Int Urol Nephrol.* 2014;46:2337–45.
 32. Tanaka S, Okusa MD. Crosstalk between the nervous system and the kidney. *Kidney Int.* 2020;97:466–76.
 33. Wang B, Xu J, Fu P, Ma L. MicroRNAs in septic acute kidney injury. *Burns Trauma.* 2023;11:tkad008.
 34. Meidert AS, Nold JS, Hornung R, Paulus AC, Zwissler B, Czerner S. The impact of continuous non-invasive arterial blood pressure monitoring on blood pressure stability during general anaesthesia in orthopaedic patients: a randomised trial. *Eur J Anaesthesiol.* 2017;34:716–22.

35. Turan A, Chang C, Cohen B, Saasouh W, Essber H, Yang D, *et al.* Incidence, severity, and detection of blood pressure perturbations after abdominal surgery: a prospective blinded observational study. *Anesthesiology*. 2019;130: 550–9.
36. Zhou B, Lin LY, Liu XA, Ling YS, Zhang YY, Luo AQ, *et al.* Invasive blood pressure measurement and In-hospital mortality in critically ill patients with hypertension. *Front Cardiovasc Med*. 2021;8:720605.
37. Kaufmann T, Cox EGM, Wiersema R, Hiemstra B, Eck RJ, Koster G, *et al.* Non-invasive oscillometric versus invasive arterial blood pressure measurements in critically ill patients: a post hoc analysis of a prospective observational study. *J Crit Care*. 2020;57:118–23.