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Research Article

Intraoperative Hypotension and Its Association with Postoperative Acute Kidney Injury

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ABSTRACT
Background: Intraoperative hypotension (IOH) is a common perioperative complication linked to inadequate organ perfusion. The kidney, with its high metabolic demand and unique microvascular architecture, is particularly vulnerable to ischemic injury. The precise thresholds of mean arterial pressure (MAP) and duration of hypotension that precipitate renal injury remain a critical area of investigation.
Objective: This study aimed to investigate the association between varying degrees of IOH and the incidence of postoperative acute kidney injury (AKI) in patients undergoing major non-cardiac surgery at a tertiary care center.
Methods: We conducted a prospective, observational cohort study of 100 adult patients undergoing elective major non-cardiac surgery under general anesthesia. Invasive or oscillometric blood pressure was recorded at 5-minute intervals intraoperatively. The primary exposure was IOH, defined as a mean arterial pressure (MAP) of <65 mmHg for a cumulative duration of ≥5 minutes. Patients were categorized into No IOH, Short IOH (<10 minutes), or Prolonged IOH (≥10 minutes). The primary outcome was the incidence of AKI, defined according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria, based on serum creatinine changes within the first 72 hours postoperatively.
Results: The overall incidence of AKI was 21%. The incidence was significantly higher in the Prolonged IOH group (44.4%) compared to the Short IOH group (16.7%) and the No IOH group (5.3%) ($p<0.001$). Multivariate logistic regression analysis identified prolonged IOH (MAP <65 mmHg for ≥10 minutes) as an independent predictor of AKI (Adjusted Odds Ratio [aOR] 9.43; 95% CI 2.18 – 40.75; $p=0.003$). Even a shorter duration of hypotension (≥5 minutes) showed a trend towards increased risk. Other significant risk factors included pre-existing hypertension and higher baseline serum creatinine.
Conclusion: Intraoperative hypotension, particularly when MAP falls below 65 mmHg for a cumulative duration of 10 minutes or more, is strongly and independently associated with an increased risk of postoperative acute kidney injury. This study underscores the importance of meticulous hemodynamic management and avoiding even modest periods of hypotension in the perioperative setting to safeguard renal function.

Keywords: Intraoperative Hypotension, Acute Kidney Injury, Postoperative Complications, Non-Cardiac Surgery, Mean Arterial Pressure, KDIGO Criteria.

INTRODUCTION

Postoperative acute kidney injury (PO-AKI) is a serious surgical complication that has received increasing attention in recent years. This condition refers to the sudden failure of a patient's kidneys to function normally after surgery, which brings a series of adverse outcomes to patients: not only is the risk of morbidity greatly increased, but hospital stays are also prolonged, medical expenditures surge, and the long-term risk of death is also elevated [1, 2]. Even a mild, transient decline in kidney function, without any obvious detectable clinical symptoms, can have a profound impact on a patient's subsequent long-term health. This situation increases the probability of newly developing chronic kidney disease, and if a patient already has chronic kidney disease, it will accelerate the rate of disease progression [3]. Why does postoperative acute kidney injury occur? Its underlying pathogenesis is complex, not caused by a single factor, but triggered by the confluence of multiple conditions: these factors include the patient's own inherent individual susceptibility to the disease, acute damage to body tissues caused by the surgical procedure, and various medical management plans before

and after the operation; the combination of these factors gives rise to this problem [4]. Among all the risk factors that may cause perioperative kidney injury, impaired blood perfusion to the kidneys is the core cause of ischemic acute tubular necrosis, which is precisely the most common type of perioperative kidney injury in clinical practice [5].

During the perioperative period, from before surgery, through surgery, to the early postoperative recovery phase, the human body has to bear a heavy physiological burden, and the original physiological state will experience severe disorders. The body's own regulatory mechanism that maintains the stability of the internal environment can be completely disrupted by anesthetic drugs, surgical trauma, and drastic fluctuations in the body's fluids. To ensure that all distal organs of the body receive sufficient oxygen, the core prerequisite is to continuously maintain adequate systemic arterial blood pressure. Intraoperative hypotension (IOH) is extremely common in operating rooms, and its general definition in the field is a mean arterial pressure (MAP) below a specific threshold, with this low-pressure state lasting for a specified period of time. The incidence of intraoperative hypotension calculated by different studies varies widely, mainly because of the different diagnostic criteria used and the different patient populations studied, but data from clinical reports show that among patients undergoing non-cardiac surgery, up to 30%-40% will experience intraoperative hypotension [6]. The kidney is far more sensitive to a drop in perfusion pressure than other organs, because the kidney's blood flow relies on its own regulatory mechanism to maintain stability, and the mean arterial pressure range in which this regulatory mechanism can function is very narrow, approximately only 75-160mmHg. Once the blood pressure falls below the lower limit of this range, the kidney's blood flow will decrease synchronously with the drop in arterial pressure, eventually leading to hypoxia in the renal medulla, which in turn triggers kidney cell damage [7]. Even in healthy, uninjured people, the living environment of the renal medulla itself is always in a hypoxic state, and its partial pressure of oxygen is already at a critical value; if intraoperative hypotension persists, it will suffer from hypoxia-induced damage earlier and more severely than other parts of the kidney [8].

Research on perioperative hemodynamic complications has been carried out for decades, but the clear association between the severity of intraoperative hypotension (IOH)specifically the magnitude of the blood pressure drop and the duration of the low-pressure stateand the subsequent development of acute kidney injury (AKI) has not been fully elucidated to this day. In simple terms, after years of research, the medical community still cannot accurately state what degree of blood pressure drop and how long its duration during surgery will definitely lead to subsequent kidney problems; this corresponding relationship has never been fully understood.

Past observational studies have consistently reached the same conclusion: if hypotension is severe and lasts for a long time, it will always be associated with poor renal prognosis, with no exceptions. However, the key question of what critical threshold of blood pressure starts to cause damage remains hotly debated in the medical community, with no unified consensus. Early landmark studies in the field once proposed that a mean arterial pressure (MAP) below 60 mmHg was the core threshold for organ damage [9], and this statement was once the basis for many clinical judgments.

However, in recent years, a large amount of new evidence has emerged, most of which comes from large retrospective cohort studies and a small number of prospectively designed analyses, and their conclusions differ from those of earlier studies: even milder hypotension, such as a mean arterial pressure below 65 mmHg that lasts for only a short period of time, may increase the risk of myocardial and kidney damage [10, 11]. This new conclusion has directly changed the inherent clinical thinking, and people have begun to realize that a situation of "relative hypotension" cannot be ignoredthat is, a patient's blood pressure is far lower than their usual baseline blood pressure, and this individualized low-pressure state may be as important as the general absolute blood pressure threshold [12]. Later, a more accurate measurement metric called "area under the threshold" was proposed, which refers to the cumulative total duration of blood pressure below the critical threshold; compared with only measuring a single hypotension value, it can more precisely measure how much burden hypotension imposes on the body, without omitting the key influencing factor of the duration of low pressure [13].

Returning to the practical problem that needs to be solved: the incidence of intraoperative hypotension is inherently high, and the consequences of postoperative acute kidney injury (PO-AKI) are particularly severe, so the demand facing us is clear: there is an urgent need for high-quality prospective research data to optimize perioperative hemodynamic management goals and provide reliable guidance for clinicians to make decisions. However, the vast majority of currently available research evidence comes from large retrospective database studies, that is, studies conducted by analyzing stock data of past patients. This type of research has inherent limitations: it cannot control for the interference of various confounding factors, that is, it cannot exclude other irrelevant variables that may affect the results, and it may not be able to accurately reflect the precise and complex association between intraoperative hypotension and acute kidney injury in various different surgical populations [14].

The institution where we conducted this study, Clearmedi Paridhi Hospital in Gwalior, serves a patient population where a large proportion of people have underlying comorbidities such as hypertension and diabetes. These pre-existing chronic diseases may further alter the kidney's susceptibility to damage, making these patients' kidneys more vulnerable than the general population and more likely to be injured by hypotension. Therefore, we specifically designed this prospective study to rigorously explore the specific association between the duration and severity of intraoperative hypotension and the incidence of postoperative acute kidney injury in patients undergoing major non-cardiac surgery. Unlike many previous studies, this study did not simply define intraoperative hypotension as a binary event of "either present or absent", that is, it did not only label patients as "having hypotension" or "not having hypotension", but instead took the duration of hypotension as a core distinguishing dimension, hoping to clarify the possible dose-response relationship between the bodily burden caused by hypotension and kidney injurythat is, whether the heavier the burden, the more severe the damage. Ultimately, we aim to produce actionable clinical reference information, practically improve patient safety, and enhance the treatment prognosis of all patients.

Objective

The primary objective of this study is to clarify the association between cumulative intraoperative hypotension and acute kidney injury that occurs within 72 hours after surgery. Hypotension is explicitly defined as mean arterial pressure (MAP) below 65mmHg, with this state lasting for at least 5 minutes; the postoperative diagnosis of acute kidney injury is fully determined in accordance with KDIGO criteria. The research team also put forward a preliminary hypothesis: the longer the cumulative duration of intraoperative hypotension, the higher the probability of postoperative acute kidney injury.

In addition to this core objective, this study has two secondary objectives. The first is to identify the critical duration of hypotension that can significantly increase the risk of acute kidney injury, that is to clarify after how long hypotension persists the risk of acute kidney injury will rise significantly. The second is to assess how other perioperative risk factors that may affect the onset of the condition, including age, preoperative baseline renal function, comorbidities, and type of surgery, impact the aforementioned association between intraoperative hypotension and postoperative acute kidney injury. By fulfilling these analytical objectives, we hope to generate more definitive evidence to provide references for perioperative hemodynamic management protocols, ultimately reducing the incidence of the preventable complication of postoperative acute kidney injury.

METHODOLOGY & MATERIALS

This is a prospective observational cohort study conducted at Clearmedi Paridhi Hospital, Gwalior, with a total duration of one full year, launched in March 2025 and continuing until February 2026.

Prior to formally enrolling patients, the research team first obtained the approval and consent from the Institutional Ethics Committee (IEC), then sequentially enrolled eligible participants, and ultimately registered a total of 100 patients into the study. All of these patients met the pre-set inclusion criteria of the study, and no cases were enrolled against the requirements.

All patients who joined the study signed a written informed consent form before their scheduled surgery, confirming that they understood the content related to the study and agreed to participate. All operations throughout the entire process of the study strictly followed the principles of the Declaration of Helsinki.

Inclusion Criteria

1. Adult patients aged 18 years or older.
2. Patients undergoing elective major non-cardiac surgery under general anesthesia with an expected duration of ≥2 hours. This included major abdominal, urological, orthopedic, and gynecological procedures.
3. American Society of Anesthesiologists (ASA) physical status I to III.

Exclusion Criteria

1. Patients with pre-existing chronic kidney disease, defined as an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73m² or documented renal transplant.
2. Patients undergoing emergency surgery, cardiac surgery, vascular surgery (excluding minor procedures), or renal/urological procedures directly involving the kidneys (e.g., nephrectomy).
3. Patients with a history of anaphylaxis or known allergy to anesthetic agents.
4. Pregnant or lactating women.
5. Patients who required significant intraoperative inotrope or vasopressor infusion before the onset of hypotension for hemodynamic support (e.g., cardiogenic shock).

Data Collection Procedure

All patients received the same standardized anesthesia protocol. After premedication, the induction of general anesthesia was performed using intravenous propofol (1.5-2.5 mg/kg), fentanyl (1-2 μg/kg) and a neuromuscular blocking agent (vecuronium or atracurium). Anesthesia was maintained with either sevoflurane or isoflurane in an oxygen/air mixture, along with supplemental opioids and muscle relaxants where needed. Mechanical ventilation was titrated to maintain normocapnia. Standard monitoring included electrocardiography, pulse oximetry, capnography and thermometer. Invasively (radial artery cannulation at ASA III or anticipated major blood loss) or non-invasively (automated oscillometric cuff on upper arm every 5 minutes for all others), blood pressure measurements were recorded.

IOH was defined as a MAP <65 mmHg (primary exposure). The cumulative time (in minutes) that the MAP was below 65 mmHg for each patient was derived from the continuous electronic anesthesia record. Patients were divided into three groups according to this cumulative time:

- **Group 1 (No IOH):** MAP <65 mmHg for<1min.
- **Group 2 (Brief IOH):** MAP <65 mmHg for a total of 1–9 minutes.
- **Group 3 (Prolonged IOH):** MAP <65 mmHg for a total duration ≥10 minutes.

Episodes of hypotension were treated at the discretion of the attending anesthesiologist, typically with intravenous fluid boluses, reduction of anesthetic depth, or administration of vasopressors (e.g., phenylephrine or ephedrine). The primary outcome, postoperative AKI, was determined by measuring serum creatinine levels preoperatively (baseline) and then at 24, 48, and 72 hours postoperatively. AKI was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria: an increase in serum creatinine by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours, or a ≥1.5 times increase from baseline within 72 hours [15]. Urine output was not used as a sole criterion for AKI due to the impracticality of hourly measurements in a general surgical ward post-discharge from the post-anesthesia care unit.

Statistical Data Analysis

Data were entered into a Microsoft Excel spreadsheet and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed data were presented as mean ± standard deviation (SD) and compared using one-way analysis of variance (ANOVA). Non-normally distributed data were presented as median and interquartile range (IQR) and compared using the Kruskal-Wallis test. Categorical variables were presented as frequencies and percentages and compared using the Chi-square test or Fisher's exact test, as appropriate. To identify independent predictors of AKI, variables with a p-value <0.10 in univariate analysis were entered into a multivariate logistic regression model. A two-tailed p-value of <0.05 was considered statistically significant.

RESULTS

A total of 100 patients were included in this study, and all of them fully cooperated to complete the entire research process. The baseline demographic information and clinical characteristics of these patients at the time of enrollment were recorded in detail in Table 1.

The average age of the participating patients was 54.8±13.2 years, and the proportion of

males in the overall population was slightly higher, reaching 58%. Among the comorbid chronic conditions these patients had, hypertension was the most common, accounting for 42% of the total number of patients, followed by diabetes, which accounted for 25%. For the ASA physical status classification used to assess the baseline physical condition of surgical patients, the distribution among this cohort of patients was: 30% were Class I, 45% were Class II, and 25% were Class III.

Researchers divided all 100 patients into three groups based on the cumulative total duration of hypotension that occurred during each patient's surgery. The specific number and proportion of patients in each group were recorded in Table 2: 19 patients (19% of the total) who never experienced hypotension throughout the surgery were assigned to the first group; 45 patients (45% of the total) who had a short duration of hypotension were assigned to the second group; the remaining 36 patients (36% of the total) who had a very long duration of persistent hypotension were assigned to the third group.

The baseline characteristics of the patients in these three groups were largely consistent at the time of study enrollment. Whether in terms of age, sex, BMI, or the prevalence of hypertension and diabetes, the differences between the groups were minimal, with no statistically significant differences, meaning the starting conditions of the three groups were comparable.

The primary outcome the incidence of postoperative AKI occurred in 21 of the 100 patients (21%). The incidence of AKI was markedly higher among patients with prolonged IOH. Specifically, AKI occurred in 1 patient (5.3%) in Group 1 (No IOH), 5 patients (11.1%) in Group 2 (Short IOH), and 16 patients (44.4%) in Group 3 (Prolonged IOH). This difference was highly statistically significant ($p < 0.001$). Figure 1 provides a bar chart showing the incidence of AKI across the three groups, and Figure 2 is a pie chart showing the distribution of AKI cases based on the duration of hypotension.

Table 3 presents the intraoperative hemodynamic parameters. While the lowest recorded MAP was, by definition, lowest in the prolonged IOH group, the mean baseline MAP and total intraoperative fluid administration were similar. However, the use of vasopressors was significantly higher in the prolonged IOH group ($p<0.001$).

In a univariate analysis (Table 4), age, male sex, a history of hypertension, and prolonged IOH were all associated with an increased risk of AKI. However, after adjusting for potential confounders in a multivariate logistic regression model (Table 5), prolonged IOH remained the strongest independent predictor of postoperative AKI (Adjusted Odds Ratio [aOR] = 9.43; 95% Confidence Interval [CI] = 2.18 – 40.75; $p = 0.003$). A history of hypertension also remained an independent predictor (aOR = 3.85; 95% CI = 1.22 – 12.17; $p = 0.022$).

Table 1: Baseline Demographic and Clinical Characteristics (N=100)

Characteristic	Value
Age (years), Mean ± SD	54.8 ± 13.2
Sex (Male), n (%)	58 (58.0%)
BMI (kg/m²), Mean ± SD	25.6 ± 4.1
Comorbidities, n (%)	
- Hypertension	42 (42.0%)
- Diabetes Mellitus	25 (25.0%)
- Coronary Artery Disease	8 (8.0%)
ASA Physical Status, n (%)	
- I	30 (30.0%)
- II	45 (45.0%)
- III	25 (25.0%)
Baseline Serum Creatinine (mg/dL), Mean ± SD	0.91 ± 0.18

Table 2: Comparison of Baseline Characteristics Between IOH Groups

Characteristic	Group 1 (No IOH) n=19	Group 2 (Short IOH) n=45	Group 3 (Prolonged IOH) n=36	p-value
Age (years), Mean ± SD	51.2 ± 13.5	54.1 ± 12.8	56.9 ± 13.4	0.32
Sex (Male), n (%)	10 (52.6%)	26 (57.8%)	22 (61.1%)	0.84

Hypertension, n (%)	6 (31.6%)	19 (42.2%)	17 (47.2%)	0.54
Diabetes Mellitus, n (%)	4 (21.1%)	11 (24.4%)	10 (27.8%)	0.86
Baseline Serum Creatinine (mg/dL), Mean ± SD	0.88 ± 0.15	0.91 ± 0.19	0.93 ± 0.17	0.62

Table 3: Intraoperative Hemodynamic Parameters and Management

Parameter	Group 1 (No IOH) n=19	Group 2 (Short IOH) n=45	Group 3 (Prolonged IOH) n=36	p-value
Lowest MAP (mmHg), Mean ± SD	68.2 ± 2.1	62.1 ± 3.5	56.4 ± 4.2	<0.001
Total Fluids (mL), Mean ± SD	1850 ± 450	2100 ± 550	2350 ± 680	0.08
Vasopressor Use, n (%)	1 (5.3%)	12 (26.7%)	25 (69.4%)	<0.001
Surgery Duration (min), Mean ± SD	188 ± 45	205 ± 52	212 ± 61	0.31

Table 4: Univariate Analysis of Factors Associated with Postoperative AKI

Variable	No AKI (n=79)	AKI (n=21)	Odds Ratio (95% CI)	p-value
Age (years), Mean ± SD	53.1 ± 12.9	61.3 ± 12.5	1.06 (1.02 – 1.10)	0.012
Sex (Male), n (%)	43 (54.4%)	15 (71.4%)	2.09 (0.74 – 5.89)	0.16
Hypertension, n (%)	29 (36.7%)	13 (61.9%)	3.08 (1.13 – 8.39)	0.028
Diabetes Mellitus, n (%)	19 (24.1%)	6 (28.6%)	1.26 (0.43 – 3.71)	0.67
IOH Group				
- Group 1 (No IOH)	18 (22.8%)	1 (4.8%)	1.0 (Reference)	-
- Group 2 (Short IOH)	40 (50.6%)	5 (23.8%)	2.25 (0.25 – 20.03)	0.46
- Group 3 (Prolonged IOH)	20 (25.3%)	16 (76.2%)	14.40 (1.80 – 115.01)	0.011

Table 5: Multivariate Logistic Regression Analysis for Predictors of AKI

Predictor	Adjusted Odds Ratio (aOR)	95% Confidence Interval	p-value
Age (per year)	1.04	0.99 – 1.09	0.11
Hypertension	3.85	1.22 – 12.17	0.022
IOH Group			
- Group 1 (No IOH)	1.0 (Reference)	-	-
- Group 2 (Short IOH)	3.12	0.31 – 31.44	0.33
- Group 3 (Prolonged IOH)	9.43	2.18 – 40.75	0.003

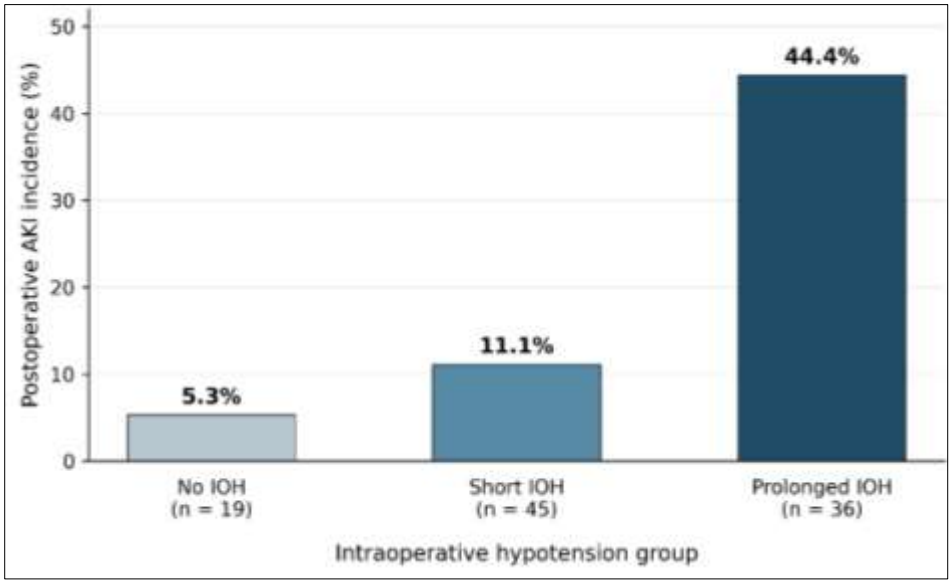

Figure 1: Incidence of AKI by Intraoperative Hypotension Group

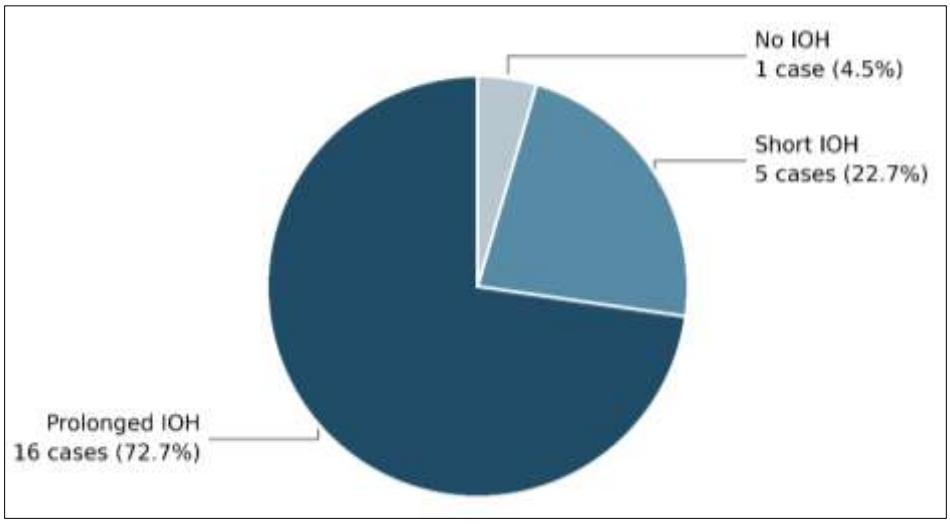

Figure 2: Distribution of 21 AKI Cases by Intraoperative Hypotension Group

DISCUSSION

This prospective cohort study provides compelling evidence that intraoperative hypotension (IOH) is a significant, independent, and modifiable risk factor for the development of postoperative acute kidney injury (AKI) in patients undergoing major non-cardiac surgery. Our findings demonstrate a clear dose-response relationship: the cumulative duration of time spent with a mean arterial pressure (MAP) below 65 mmHg directly correlates with an escalating risk of renal injury. The incidence of AKI was 5.3% in patients with negligible hypotension, rising to 11.1% for brief episodes, and sharply increasing to 44.4% for those with a cumulative hypotensive duration of 10 minutes or more. This association remained highly significant after adjusting for key confounders in a multivariate model (aOR 9.43, p=0.003), solidifying the role of hemodynamic stability as a cornerstone of perioperative renal protection.

Our conclusions align with the results of several large-scale observational studies in recent years, and on the basis of these studies, we add new findings that advance the understanding in this field. Salmasiet al. completed a landmark retrospective analysis widely recognized in the field, which included more than 57,000 patients and found that even a short duration of mean arterial pressure below 65 mmHgonly 1 to 5 minuteswould increase the risk of both myocardial injury and renal injury [10]. The key value of this study lies in overturning the overly lenient management principles previously adopted in clinical practice: the prevailing industrial convention at that time generally held that as long as the mean arterial pressure did not drop below 60 mmHg, it was safe, and such a blood pressure level was acceptable.

Later, another study by Mathis et al. reached a similar conclusion, echoing the earlier research. They found that compared with only relying on the single lowest recorded mean arterial pressure value during surgery, the duration of hypotension is a more core factor determining whether postoperative acute kidney injury will occur. This judgment logic is completely consistent with the approach of this study, which grouped patients by the cumulative duration of hypotension [16]. Our study can supplement more solid new evidence for this field relying on two core advantages: first, it adopts a prospective study design, eliminating the need to only retrieve existing past medical records as previous retrospective studies had to; second, it employs the strict KDIGO criteriacompared with administrative codes or the RIFLE criteria relied on by some early retrospective studies, KDIGO can more sensitively identify emerging, unapparent early renal injury, avoiding the omission of potential cases. In this study, the incidence of acute kidney injury in the prolonged IOH group reached as high as 44.4%, which also highlights the clinical vulnerability of such patients. This risk is particularly pronounced in scenarios where patients themselves have a heavy burden of underlying comorbidities, making them more prone to complications than patients in good health.

The pathophysiological basis supporting the conclusions of this study has long been repeatedly verified by the academic community and is a mature theory universally recognized in the field. The kidneys themselves have the

ability to autoregulate blood flow. As long as the mean arterial pressure is maintained within the range of 75-160 mmHg, the glomerular filtration rate can remain stable, sustaining the kidneys' normal function of filtering waste and maintaining the body's metabolism. Once the mean arterial pressure falls below this autoregulatory threshold, renal blood flow loses its ability to be autonomously regulated and changes completely with fluctuations in blood pressure, which in turn leads to insufficient oxygen supply to two types of cells in the kidneys with extremely high oxygen consumptionproximal tubule cells and thick ascending limb cells of the loop of Henlemaking them unable to maintain normal physiological activities. The physiological structure of the renal medulla makes it particularly sensitive to hypoxia: on one hand, transporting solutes in the body consumes a large amount of oxygen, and on the other hand, its inherent countercurrent oxygen exchange mechanism also amplifies the risk of hypoxia, making it more prone to problems than other parts of the kidney [8].

If the kidneys remain in an ischemic state for a long time, it will trigger a series of pathological reactions, including vascular endothelial dysfunction, systemic inflammation, and oxidative stress damage, eventually leading to acute tubular necrosis, which refers to the massive death of renal tubular cells in the kidneys, resulting in a complete loss of normal function [5]. In addition, many additional factors during the perioperative period can exacerbate this ischemic injury, such as inflammation caused by surgical trauma itself, and the inherent nephrotoxicity of some therapeutic drugs. The logic of the superimposed effect of these risk factors is the "multiple hits" hypothesis of acute kidney injury widely discussed in the academic community, where multiple damages acting together on the kidneys are far more destructive than a single damage [17]. This hypothesis can also explain why patients with underlying diseases that impair the kidneys' autoregulatory ability, such as chronic hypertension and diabetes, have a much higher risk of postoperative renal injury than the general population. This study observed that hypertension itself is an independent predictor of acute kidney injury, which is also completely consistent with this logiclong-term hypertension causes the kidneys' autoregulatory curve to shift to the right. The kidneys adapt to long-term elevated blood pressure, so a blood pressure that is within the normal range for ordinary people may be too low for these patients, failing to meet the kidneys' blood supply needs, and thereby impairing renal function [18].

The clinical significance of this study is extraordinary. It is by no means a paper-only academic conclusion, but a key finding that can directly change clinical treatment logic. In the past, many anesthesiologists may have thought that an occasional episode of hypotension in patients during surgery is an unavoidable minor issue during general anesthesia, at most an insignificant physiological side effect that does not require much attention. But this study explicitly overturns this perceptionintraoperative hypotension is a real dangerous event, and if left unaddressed, it will cause serious damage to vital organs such as the heart and kidneys. The weight of these conclusions is sufficient to support a complete adjustment of past hemodynamic management strategies in clinical practice, completely changing the previous response model of passively fighting fires only after problems arise into a new model of proactive prevention and active blood pressure control.

If we still follow the old method of only relying on intermittent blood pressure measurements taken every few hours or even longer, it is very likely to miss many obvious episodes of hypotension that have already occurred. By the time it is detected, the blood pressure has already been low for a long time, missing the optimal intervention window. This precisely illustrates that the use of full continuous blood pressure monitoring for patients with multiple underlying diseases and high surgical risks does have outstanding benefits, enabling every abnormal fluctuation in blood pressure to be captured. For all anesthesiologists, the threshold of mean arterial pressure below 65 mmHg must be established as a clear red line that requires immediate intervention. Once a patient's blood pressure drops below this line, no time can be wasted, and measures must be taken immediately to bring the blood pressure back up. The ultimate goal is only one: to compress the cumulative total duration of hypotension throughout the entire surgical procedure as close to 0 minutes as possible, avoiding any occurrence if possible.

To achieve the goal of minimizing the duration of hypotension as much as possible, several core strategies are indispensable, and they are also the top priorities repeatedly emphasized in the study: a one-size-fits-all blood pressure standard cannot be applied to all patients;

instead, exclusive blood pressure targets must be tailored according to each patient's own preoperative baseline blood pressure; we cannot wait for patients to have insufficient blood volume and their blood pressure to drop before administering fluids; proactive fluid management must be arranged in advance to maintain sufficient circulating blood volume; if medication is needed, vasopressors should also be initiated as early as possible, rather than waiting until blood pressure collapses to remedy the situation [19].

A thought-provoking phenomenon was also found in the statistical data of this study: the proportion of patients in the prolonged hypotension group who used vasopressors was actually very high. This contradictory situation most likely points to a problemthe attending physicians at that time still adopted the most traditional passive response approach: they only hurriedly started using vasopressors to remedy the situation after the blood pressure had dropped, or even had been low for a long time, which is equivalent to looking for a fire extinguisher only after the fire has already started. If the proactive prevention strategy advocated by the study is adopted, where low-dose vasopressors are administered to patients in advance before their blood pressure falls below the safety line, preventing the blood pressure from dropping below the dangerous threshold at the source, it may be more effective in reducing the total harm caused by hypotension and fundamentally lowering the risk of organ damage.

To truly translate these written research conclusions into better clinical diagnosis and treatment outcomes for patients, two types of tools can play a key role in implementation: one is special training programs for frontline medical staff, to help all clinicians fully understand the hazards of hypotension and integrate the new logic of active blood pressure control into their daily work; the other is a supporting clinical decision support system, which can proactively send reminders to the attending physician when the cumulative duration of a patient's blood pressure below the threshold gradually approaches the dangerous value, preventing the risk from being missed because the doctor is too busy to check the monitor [20].

Finally, this study once again clearly confirms that there is a strong, independent, and stepwise association between the cumulative duration of intraoperative hypotension and the risk of postoperative acute kidney injuryin simple terms, the longer the duration of hypotension, the higher the risk of postoperative renal injury in patients, and every additional minute increases the risk by one level. Even maintaining a mean arterial pressure below the 65 mmHg threshold for a short period of time is extremely dangerous, leaving no room for the idea that "short-term hypotension is harmless", and every minute of hypotension must be taken seriously. As long as anesthesiologists are willing to change their mindset, persist in closely monitoring patients' blood pressure, and proactively maintain blood pressure stability, there is a clear opportunity to reduce this common and costly postoperative complication, helping patients avoid the risk of postoperative renal injury [21].

LIMITATIONS OF THE STUDY

This study has several limitations, which must all be taken into account when interpreting its relevant conclusions; no definitive judgment should be reached solely based on the results of this study.

First, this study adopted an observational research design. This design only conducts research by collecting and analyzing observed clinical data, and can only confirm that there is a strong association between intraoperative hypotension IOH and acute kidney injury AKIthat is, the two conditions frequently co-occurbut it cannot fully determine that intraoperative hypotension directly causes acute kidney injury, nor can it establish a definite causal relationship between the two. Even though we performed a multivariate analysis, which means we incorporated all other conceivable confounding factors that might affect the results into the statistical adjustment, there are still some unmeasured variables that cannot be ruled out. These unaccounted-for factors include the specific complexity of the surgery, the actual amount of blood loss during the procedure, and subtle differences in the depth of anesthesia among different patients, all of which may interfere with the final conclusion, and their impacts cannot be completely eliminated.

Second, the study sample included only 100 patients. This size is sufficient to identify associations with large effects, that is, if an association has a particularly prominent impact, this sample size is enough to detect it statistically. However, it may not have sufficient statistical power to detect significant risks in the

subgroup of patients with "Short IOH". If a larger-scale, multi-center study can be conducted in the future, more reliable risk estimates can be calculated for all classifications of hypotension.

Third, the blood pressure of some patients was measured using an intermittent oscillometric cuff method that took readings every 5 minutes. This interval measurement method cannot continuously record every change in blood pressure, and may miss all sudden, transient episodes of hypotension, failing to fully capture these temporary blood pressure abnormalities. This may lead to misclassification of some patients: that is, patients who did experience transient hypotension may be incorrectly assigned to the group that did not experience such events.

Fourth, we only used the serum creatinine criterion to define AKI, but this biomarker for assessing kidney injury is not only laggingunable to reflect problems in the kidney in a timely manner when they first arisebut also insufficiently precise, making it an imperfect indicator for identifying kidney injury. If new urinary biomarkers such as NGAL and KIM-1 had been incorporated into the study, we could have detected milder cases of kidney injury much earlier.

Finally, we did not track the long-term kidney outcomes of patients, such as whether patients subsequently developed chronic kidney disease from acute kidney injury, or whether they required renal replacement therapy. These are all important patient-centered observation endpoints, key outcomes directly related to patients' long-term health and quality of life, which were not covered in this study.

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CONCLUSION

This study confirms that for patients undergoing major non-cardiac surgery, if intraoperative hypotension occursspecifically, when the mean arterial pressure (MAP) drops below 65 mmHg, and this state accumulates to 10 minutes or longerthis hypotension is a strong and independent predictive risk factor for postoperative acute kidney injury. What is an independent risk factor? It means this risk is not caused by other underlying health conditions the patient already has, but is a standalone threat directly brought by hypotension itself.

From our study results, a very clear correlation can also be identified: the longer hypotension persists, the risk of acute kidney injury rises sharply, which is the explicit dose-response relationship mentioned in the studythe longer a patient is exposed to the harmful state of hypotension, the greater the "cumulative damage", and the rate of risk increase will only accelerate. Even after accounting for and adjusting for the interference of preoperative demographic characteristics such as age and gender, as well as all underlying diseases including pre-existing hypertension, the correlation between the duration of hypotension and the risk of kidney injury still holds, and will not disappear simply because a patient has other pre-existing health problems. Many patients who experienced excessively

long periods of intraoperative hypotension developed acute kidney injury after surgery, and this extremely high incidence precisely highlights the vulnerability of the kidneyseven a short period of insufficient blood supply may damage them, and the kidneys' tolerance to reduced blood flow is far lower than most people assume.

When these conclusions are applied to clinical practice, the direction is very clear: this is not a vague conclusion confined to academic papers, but a practical requirement that can directly guide daily diagnosis and treatment. Anesthesiologists, as well as all medical staff providing perioperative care for patients, must proactively monitor and keep a close watch on patients' hemodynamic indicators at all times. They must not wait to remedy problems after they arise, but must keep patients' blood pressure and other blood supply-related indicators stable from the very start. The mean arterial pressure threshold of 65 mmHg must be treated as an uncrossable red line: once the value drops below this threshold, the patient enters a dangerous state, and clear, decisive response measures must be taken immediately, with no delays or waiting.

The core goal to achieve is also very clear: minimize the total time a patient's blood pressure stays below this threshold, to keep the risk of kidney damage as low as possible. How can this goal be achieved? Several specific directions must be prioritized: reasonably arrange the volume and timing of fluid infusion, avoiding inappropriate or delayed supplementation; proactively use vasopressors as early as possible, instead of administering medication long after blood pressure has dropped below the dangerous line; and set personalized blood pressure targets tailored to each patient's own baseline blood pressure, rather than applying the same rigid standard to all patients. Thoroughly shifting from the previous passive model of waiting for problems to occur before intervening to an active strategy of preemptive prevention is the core key to protecting patients' kidney function and helping them achieve better treatment outcomes, and this transformation must be implemented.

Future research will focus on advancing in two directions to refine this prevention and control logic: first, conduct intervention trials to verify the actual effect of specific hemodynamic management protocols, to confirm whether these specific control measures can truly reduce risks; second, explore new kidney biomarkers to detect early signs of kidney injury as soon as possible, instead of identifying the problem only after symptoms appear. Ultimately, these efforts will enhance our ability to prevent this serious complication, helping more patients avoid the danger of postoperative kidney injury.

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