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The correlation of intraoperative oliguria with acute kidney injury after noncardiac surgery: a systematic review and meta-analysis

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Background: Acute kidney injury (AKI) occurs commonly after major surgery and is correlated with increased in-hospital morbidity and mortality. There is no consensus on whether intraoperative oliguria affects postoperative AKI. We conducted a meta-analysis to systematically assess the correlation of intraoperative oliguria with postoperative AKI.

Methods: PubMed, Embase, Web of Science, and Cochrane Library databases were searched to identify reports on the relationship between intraoperative oliguria and postoperative AKI. Quality was assessed using the Newcastle–Ottawa Scale. The primary outcomes were the unadjusted and multivariate-adjusted odds ratios (ORs) for intraoperative oliguria to correlate with postoperative AKI. The secondary outcomes included intraoperative urine output in the AKI and non-AKI groups, the demand for postoperative renal replacement therapy (RRT), in-hospital mortality, and length of hospital stay in the oliguria and non-oliguria groups.

Results: Nine eligible studies with 18 473 patients were included. The meta-analysis revealed that patients with intraoperative oliguria had a considerably greater risk of postoperative AKI (unadjusted OR: 2.03, 95% CI: 1.60–2.58, $I^2 = 63\%$, $P < 0.00001$; multivariate-adjusted OR: 2.00, 95% CI: 1.64–2.44, $I^2 = 40\%$, $P < 0.00001$). Further subgroup analysis did not find differences between different oliguria criteria or surgical types. Furthermore, the AKI group's pooled intraoperative urine output was less (mean differences: – 0.16, 95% CI: – 0.26 to – 0.07, $P < 0.001$). Intraoperative oliguria was associated with increased demand for postoperative RRT (risk ratios: 4.71, 95% CI: 2.83–7.84, $P < 0.001$) and in-hospital mortality (risk ratios: 1.83, 95% CI: 1.24–2.69, $P = 0.002$), but not with prolonged length of hospital stay (mean differences: 0.55, 95% CI: – 0.27 to 1.38, $P = 0.19$).

Conclusions: Intraoperative oliguria was significantly associated with a higher incidence of postoperative AKI, as well as increased in-hospital mortality and demand for postoperative RRT, but not with prolonged hospitalization.

Keywords: acute kidney injury, complications, meta-analysis, oliguria, renal, urine output

Introduction

Postoperative acute kidney injury (AKI) occurs frequently and carries serious consequences, affecting 13.4% of patients undergoing major surgery and is associated with a sixfold increase in-hospital mortality[1–3], as well as an increased incidence of other complications, length of hospital stay (LOS), and hospitalization costs[4,5]. Unfortunately, although there are numerous beneficial treatments proven to reduce the risk of developing AKI in experimental animals, no widely available therapeutic strategies were found for patients in clinical practice[6,7].

The treatment of postoperative AKI remains supportive, making prevention of the AKI critical. Typically, strategies to prevent AKI include early recognition of individuals at risk for AKI after surgery and optimizing their clinical status[8]. Several baseline factors (e.g. hypertension, elderly patients, male sex, obesity, diabetes) and perioperative factors (e.g. hypotension, systemic inflammation, fluid overload, venous congestion, nephrotoxic drugs) for postoperative AKI have been identified, which may aid in the identification of high-risk individuals with postoperative AKI[9–12].

Urine output (UO) is an essential indicator of renal perfusion, intraoperative oliguria is generally caused by reduced intravascular flow or sustained general hypoperfusion[13,14]. Both conditions are thought to result in a reduction in renal perfusion and lead to decreased filtration load and UO, increasing the risk of postoperative AKI[15]. Meanwhile, intraoperative UO can be obtained efficiently and instantly at no additional cost to the patient, which can be considered a convenient and valuable indicator in forecasting the

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development of postoperative AKI[16]. However, contradictory results have been reported in studies examining the effect of predicting postoperative AKI of intraoperative oliguria. Some studies[16–22] indicated that intraoperative oliguria raised the risk of postoperative AKI. In contrast, the remaining studies[9,23–27] did not report this association. Each element cannot be clarified by a single study, and diverse research methodologies may lead to selection bias.

Therefore, the primary goal of this meta-analysis was to assess the association of intraoperative oliguria with postoperative AKI. In addition, we explored intraoperative UO in the AKI and non-AKI groups, the relationship between intraoperative oliguria and the demand for postoperative renal replacement therapy (RRT), in-hospital mortality, and LOS. This project hopes to raise consciousness about the role of intraoperative oliguria in developing AKI.

Materials and methods

This systematic review and meta-analysis was registered on the PROSPERO (Prospective Register of Systematic Reviews), protocol number CRD42022326281, and reported in accordance with PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses)[28]. A PRISMA checklist is available as a supplement (Supplementary Table 1, Supplemental Digital Content 1, http://links.lww.com/JS9/A96).

Data collection and retrieval strategies

The databases listed below were used to look for relevant research: PubMed, Embase, Web of Science, and Cochrane library, with a search time frame of database creation to June 2022. Medical Subject Headings (MeSH) or keywords 'acute kidney injury,' 'oliguria,' and 'intraoperative period' were used to search the literature without language restrictions. The specific search strategy is shown in the supplementary material (Supplementary Table 2, Supplemental Digital Content 1, http://links.lww.com/JS9/A96). In addition, we meticulously combed through the references of all included studies and pertinent review articles for possibly qualifying research.

Selection criteria

Studies that met the following criteria were included: observational studies; participants were adults who received noncardiac surgery; studies that used a clear definition of oliguria and AKI; studies that provided data on the correlation of intraoperative oliguria with postoperative AKI.

Studies with any of the following conditions were excluded: the type of study design was not described; participants underwent urological or transplantation procedures; literature that is duplicated or from which data cannot be extracted; the full text was not available.

Literature screening and data extraction

Two reviewers (Pang and Liang) screened all retrieved literature independently in strict adherence to predefined inclusion and exclusion criteria. First of all, an initial screening was conducted by reviewing the titles and abstracts of all retrieved studies. After excluding studies that were duplicates or did not match the inclusion criteria, the remaining studies were analyzed, and all potentially eligible studies were identified by reading the full text.

Additionally, the following details were retrieved from each eligible study: first author, country, publication year, study design, sample size, oliguria criteria, diagnostic criteria of AKI, the incidence of AKI, and outcome assessment. The primary outcomes were the unadjusted and multivariate-adjusted odds ratios (ORs) of intraoperative oliguria predicting the incidence of postoperative AKI. Besides, the secondary outcomes included intraoperative UO in the AKI and non-AKI groups, the demand for postoperative RRT, in-hospital mortality, and LOS in oliguria and non-oliguria groups. The extracted information was entered into a predesigned literature data extraction form for data generalization and analysis. All discrepancies were resolved through discussions with a third investigator.

Methodology and literature quality evaluation

To measure the risk of bias, we used the Newcastle–Ottawa Scale[29], which consists of three parts: study population selection, comparability between groups, and outcomes. Out of a total score of 9, we awarded scores of 0–3, 4–6, and 7–9 for studies of the low, medium, and high quality. If the score was less than 7, the literature was excluded. Two reviewers (Pang and Liang) performed the quality assessment independently. In addition, the above two reviewers followed the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach and independently used online software (https://gdt.gradepro.org/app) to evaluate the certainty of the evidence. Each outcome was assessed for the risk of bias, inconsistency, indirectness, imprecision, and other bias and the evidence was classified as very low, low, moderate, and high.

Statistical analysis

We used RevMan 5.4.1 software and STATA 14 software to process the data for the meta-analysis. The heterogeneity of the included studies was analyzed using the Q statistic for χ^2 test (test level: $\alpha = 0.1$), while the I^2 value was applied to identify the heterogeneity level. Suppose there was statistical heterogeneity ($P < 0.1$ or $I^2 \geq 40\%$), the random-effects model was adopted for pooled effect size analysis; otherwise ($P \geq 0.1$ and $I^2 < 40\%$), the fixed-effects model was adopted for pooled effect size analysis. For the primary outcome, the strength of the correlation between intraoperative oliguria and postoperative AKI was estimated primarily by combining unadjusted and adjusted OR values, respectively. It was reported as an OR value of 95% CI. In addition, for secondary outcomes (intraoperative urine volume, postoperative RRT demand rate, in-hospital mortality, and LOS), we calculated mean differences (MDs) with 95% CI for continuous variables and risk ratios (RRs) with 95% CI for dichotomous variables. Moreover, we converted the median and interquartile range (IQR) of continuous data to mean and SD using the formula provided by Luo *et al.*[30] and Wan *et al.*[31]. Publication bias was tested using a visual funnel plot and assessed by the Egger test. A significant statistical difference was considered as P less than 0.05.

Furthermore, the correlation of intraoperative oliguria with postoperative AKI was analyzed by subgroup analysis: different oliguria criteria (UO < 0.5 or 0.3 ml/kg/h); different types of surgery (abdominal or thoracic surgery).

Trial sequential analysis (TSA)

To assess the credibility of our primary outcome, we used TSA viewer (version 0.9.5.10 beta) to perform TSA. TSA based on the O'Brien–Fleming method to calculate α-spending boundaries. The required information size that estimated sample size was calculated based on a two-sided random-effects model, with type I and type II error set as 5 and 20%, respectively.

Results

Literature retrieval and screening

The study screening process is shown in Figure 1. Based on the above retrieval strategy for electronic database search, 205 studies were initially identified. In the initial screening stage, 70 studies remained after eliminating duplicates and studies that did not match the inclusion criteria by reviewing the titles and abstracts of all retrieved studies. Then full-text articles were read for re-screening to confirm all potentially eligible studies. Finally, our meta-analysis comprised nine studies[16–22,26,27].

Study characteristics and quality assessment

Table 1 summarizes the main characteristics of the included eligible studies. All nine studies were observational studies published between 2013 and 2021, with sample sizes ranging from 153 to 5894. In addition, two of the included study used more than one criterion to define intraoperative oliguria. Meng and Mu[20] defined oliguria as UO less than 0.8 or 0.5 ml/kg/h, respectively, while Zhao *et al.*[22] described oliguria as UO less than 0.5 or 0.3 ml/kg/h, respectively, each of these two studies yielded corresponding ORs for the different intraoperative oliguria definitions to predict the incidence of postoperative AKI. Therefore, 11 outcome effect quantities were extracted and combined for the nine included studies.

Supplementary Table 3, Supplemental Digital Content 1, http://links.lww.com/JS9/A96 summarizes the details of the quality assessment of each included eligible study using the Newcastle–Ottawa Scale, with an average score of 7.6. Each study scored at least 7, and one of the studies had a maximum score of 9, indicating that the quality of all included studies was high.

Association of intraoperative oliguria with the incidence of postoperative AKI

All 11 outcome effect quantities in the nine studies involved postoperative AKI, and the total number of included study subjects was 18 473. The pooled incidence of AKI in the oliguria and non-oliguria groups was 13% (95% CI: 8–19%) and 8% (95% CI: 5–10%), respectively (Fig. 2A). Meta-analysis results are shown in Figure 3. Because of the statistical heterogeneity among studies ($I^2 = 63\%$), a random-effects model was applied for analysis, which showed a combined unadjusted OR value was 2.03 (95% CI: 1.60–2.58, $P < 0.00001$), indicating that intraoperative oliguria significantly related to a higher risk of postoperative AKI. The funnel plot (Supplementary Figure 1, Supplemental Digital Content 2, http://links.lww.com/JS9/A97) and Egger test ($P = 0.925$) showed no significant publication bias.

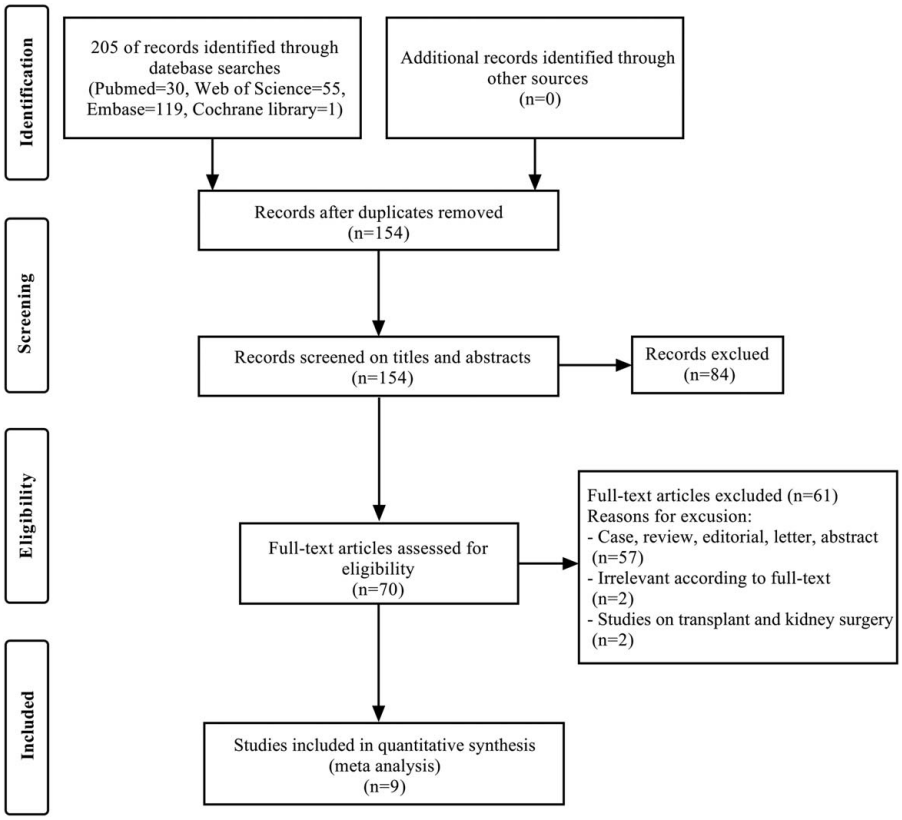

Figure 1. Flow chart of study screening.

Table 1
Main characteristics of the included studies.

References, year	Country	Study design	Sample size	Surgical type	Oliguria criteria	AKI criteria	Incidence of AKI, *N* (%)	ORs (95% CI)
Slankamenac *et al.*[17], 2013	Switzerland	Single-center, retrospective cohort study	549	Liver surgery	UO < 400 ml/24 h	RIFLE criteria	82 (14.9)	2.14 (1.29–3.55)
Goren *et al.*[26], 2017	Israel	Single-center, retrospective study of prospectively collected data	153	Open pancreatic surgery	UO < 0.5 ml/kg/h	AKIN criteria	15 (9.8)	1.90 (0.21–17.4)
Mizota *et al.*[18], 2017	Japan	Single-center, retrospective cohort study	3560	Major abdominal surgery	UO < 0.3 ml/kg/h	KDIGO criteria	224 (6.3)	1.82 (1.28–2.60)
Shiba *et al.*[16], 2018	Japan	Single-center, retrospective cohort study	5894	Noncardiac major surgery	UO < 0.5 ml/kg/h, lasting ≥ 120 min	RIFLE criteria	430 (7.3)	2.96 (2.38–3.69)
Myles *et al.*[19], 2019	International	Multicenter, post hoc analysis of prospectively collected data	2444	Major abdominal surgery	UO < 0.5 ml/kg/h, lasting ≥ 60 min	KDIGO criteria	513 (21.0)	1.52 (1.24–1.85)
Inacio *et al.*[27], 2021	Portugal	Single-center, retrospective cohort study	165	Major abdominal surgery	UO < 0.5 ml/kg/h	KDIGO criteria	32 (19.4)	0.87 (0.33–2.31)
Meng and Mu[20], 2020	China	Single-center, retrospective cohort study	1393	Lung surgery	UO < 0.8 ml/kg/h	KDIGO criteria	31 (2.2)	2.77 (1.36–5.67)
Meng and Mu[20], 2020	China	Single-center, retrospective cohort study	1393	Lung surgery	UO < 0.5 ml/kg/h	KDIGO criteria	31 (2.2)	1.51 (0.52–4.37)
Shim *et al.*[21], 2020	Korea	Single-center, retrospective matched cohort study	453	Laparoscopic colorectal resection	UO < 0.5 ml/kg/h	KDIGO criteria	79 (17.4)	2.84 (1.44–5.63)
Zhao *et al.*[22], 2021	China	Single-center, retrospective cohort study	3862	Pulmonary resection or esophagectomy	UO < 0.5 ml/kg/h	KDIGO criteria	205 (5.3)	1.60 (1.03–2.49)
Zhao *et al.*[22], 2021	China	Single-center, retrospective cohort study	3862	Pulmonary resection or esophagectomy	UO < 0.3 ml/kg/h	KDIGO criteria	205 (5.3)	2.85 (1.53–5.33)

AKI, acute kidney injury; AKIN, Acute Kidney Injury Network; KDIGO, Kidney Disease Improving Global Guidelines; ORs, odds ratios; RIFLE, Risk, Injury, Failure, Loss and End-stage kidney disease; UO, urine output.

Besides, the association remained significant in the sensitivity analysis of the combined adjusted nine outcome effect quantities, with a combined adjusted OR value of 2.00 (95% CI: 1.64–2.44, $P < 0.00001$) (Fig. 4). TSA showed that the cumulative *Z*-curve across the conventional boundary ($Z = 1.96$) and monitoring boundary, and reached the required information size. Therefore, current result was reliable and conclusive (Supplementary Figure 2, Supplemental Digital Content 2, http://links.lww.com/JS9/A97).

Subgroup analysis

In the subgroup analysis based on different oliguria criteria, there was significant heterogeneity between the two subgroups (UO < 0.5 or 0.3 ml/kg/h) (test for subgroup difference: $P = 0.04$ and $I^2 = 75.8\%$) (Fig. 5), implying that the choice of oliguria criteria significantly influenced the results of the meta-analysis. Taking UO less than 0.5 ml/kg/h as the oliguria standard, the pooled incidence of AKI in the oliguria group and non-oliguria group was 14% (95% CI: 8–22%) and 9% (95% CI: 5–14%), respectively (OR: 1.72, 95% CI: 1.39–2.14, $P < 0.00001$). Secondly, with UO less than 0.3 ml/kg/h as the oliguria standard, the pooled incidence of AKI was 11% (95% CI: 8–14%) and 5% (95% CI: 5–6%), respectively (OR: 2.64, 95% CI: 1.86–3.74, $P < 0.00001$) (Figs. 2B, 5).

The results of subgroup analysis based on different types of surgery showed, after abdominal surgery, the pooled incidence of AKI in the oliguria and non-oliguria groups was 19% (95% CI: 12–28%) and 12% (95% CI: 7–19%), respectively (OR: 1.67, 95% CI: 1.18–2.33, $P = 0.004$). After thoracic surgery, the pooled incidence of AKI was 6% (95% CI: 3–10%) and 3% (95% CI: 2–5%), respectively (OR: 1.94, 95% CI: 1.36–2.77, $P < 0.001$) (Figs. 2C, 5).

Intraoperative urine volume levels in the AKI and non-AKI groups

Five studies[18,20,22,26,27] reported differences in intraoperative urine volume between the AKI and non-AKI groups. In the AKI groups, the pooled postoperative UO level of 1.27 ml/kg/h (95% CI: 0.88–1.65) was substantially less than that of 1.50 ml/kg/h (95% CI: 1.18–1.83) in non-AKI groups (MD: – 0.16, 95% CI: – 0.26 to – 0.07, $P < 0.001$), and the difference showed statistical significance (Fig. 2D).

Association of intraoperative oliguria with in-hospital mortality, LOS, and postoperative RRT requirement

Three studies[16,19,27] reported postoperative RRT requirements of 8503 patients, with a higher value in the oliguria group compared to the non-oliguria group (RR: 4.71, 95% CI: 2.83–7.84, $P < 0.001$). Two studies[16,27] from the evaluation of 6059 patients showed that in-hospital mortality of the oliguria group was substantially greater than the non-oliguria group (RR: 1.83, 95% CI: 1.24–2.69, $P = 0.002$). A total of 8663 patients from four studies reported LOS (in days)[16,19,21,27], noticing that the time of the oliguria group was longer than the non-oliguria group. Still, the difference was slight and not statistically
significant (MD: 0.55, 95% CI: – 0.27 to 1.38, $P = 0.19$).

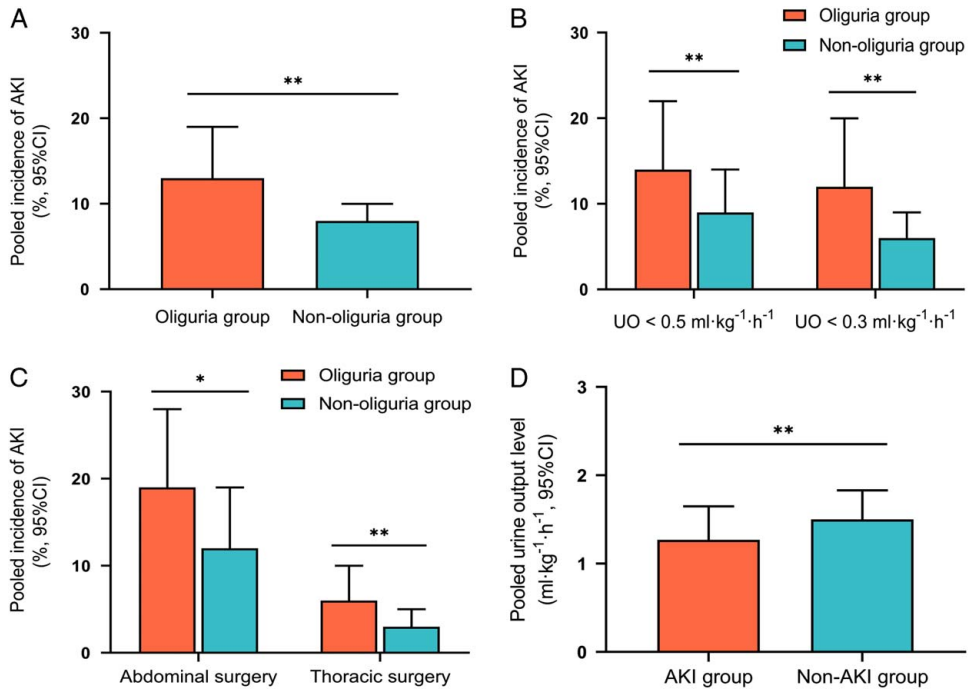

Figure 2. Intraoperative oliguria and postoperative acute kidney injury (AKI). (A) The pooled incidence of postoperative AKI in the oliguric and non-oliguric group; (B) subgroup analysis of different oliguria criteria; (C) subgroup analysis of different surgical types; (D) the pooled intraoperative urine output level in the AKI and non-AKI group; *$P < 0.01$ and **$P < 0.001$.

GRADE certainty of evidence

The GRADE evidence profile was presented in Supplementary Table 4, Supplemental Digital Content 1, http://links.lww.com/JS9/A96. The certainty of evidence was low for UO and LOS, moderate for the incidence of postoperative AKI, demand for postoperative RRT, and in-hospital mortality.

Discussion

In this systematic review and meta-analysis, we assessed the correlation of intraoperative oliguria with postoperative AKI, and the combined results suggested that intraoperative oliguria significantly related to a higher risk of postoperative AKI, but the strength of this association was weak. TSA analyses showed that there was enough information to confirm the results. Further subgroup analysis did not find differences between different oliguria criteria or surgical types. Moreover, the combined results showed that AKI individuals had considerably lower intraoperative urine volume. Patients with intraoperative oliguria were accompanied with increased in-hospital mortality and demand for postoperative RRT. Still, without prolonging LOS.

Our meta-analysis revealed that the occurrence of postoperative AKI for individuals who suffered intraoperative oliguria was substantially greater, consistent with previous research[16–22]. The underlying reasons are analyzed as follows. Firstly, UO is an important index to reflect organ perfusion. Intravascular hypovolemia and prolonged hypotension con-

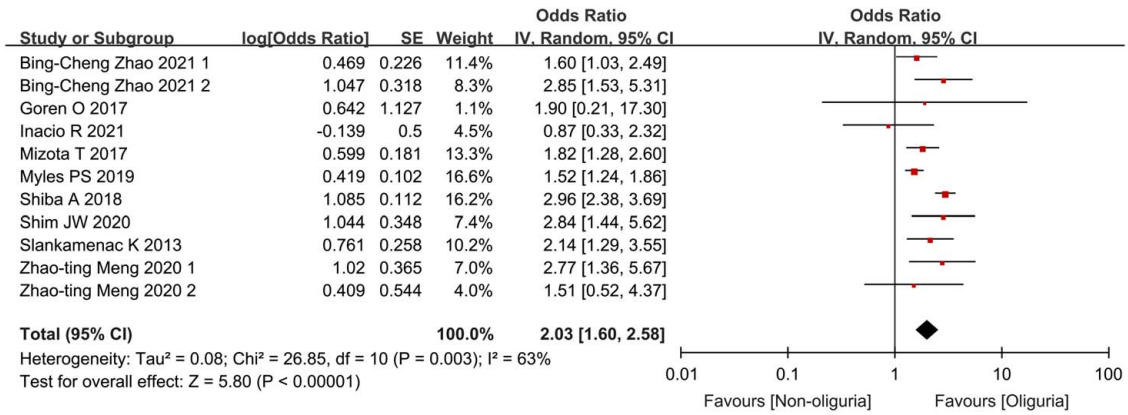

Figure 3. Forest plot showing combined unadjusted odds ratios for the association of intraoperative oliguria with postoperative acute kidney injury.

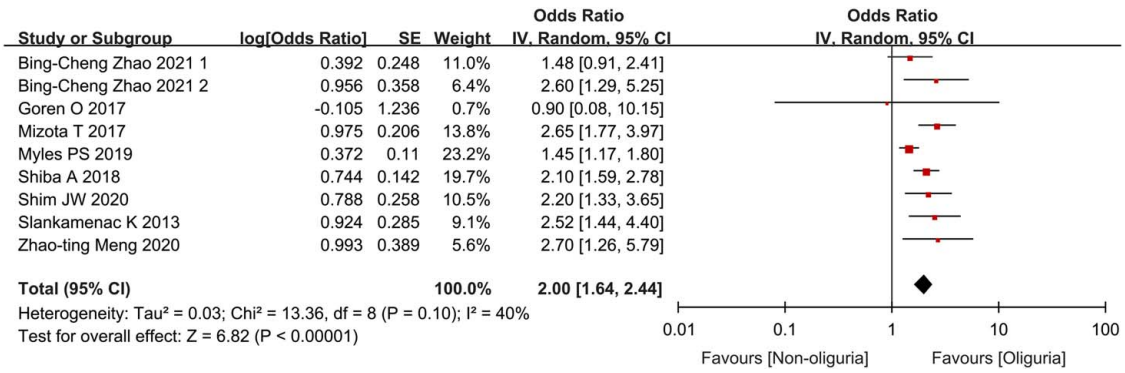

Figure 4. Forest plot showing combined multivariate-adjusted odds ratios for the association of intraoperative oliguria with postoperative acute kidney injury.

tribute to reduced perioperative renal perfusion and elevate the risk of postoperative AKI[15,32,33]. The compensatory response of the kidney at this time is the dilatation of the small afferent arteries and the vasoconstriction of the small efferent arteries to sustain glomerular filtration. Simultaneously, renin–angiotensin system mobilization leads to water and sodium ions storage, excretion of potassium ions, and eventually results in low UO[34–37]. Secondly, oliguria may indicate that early renal

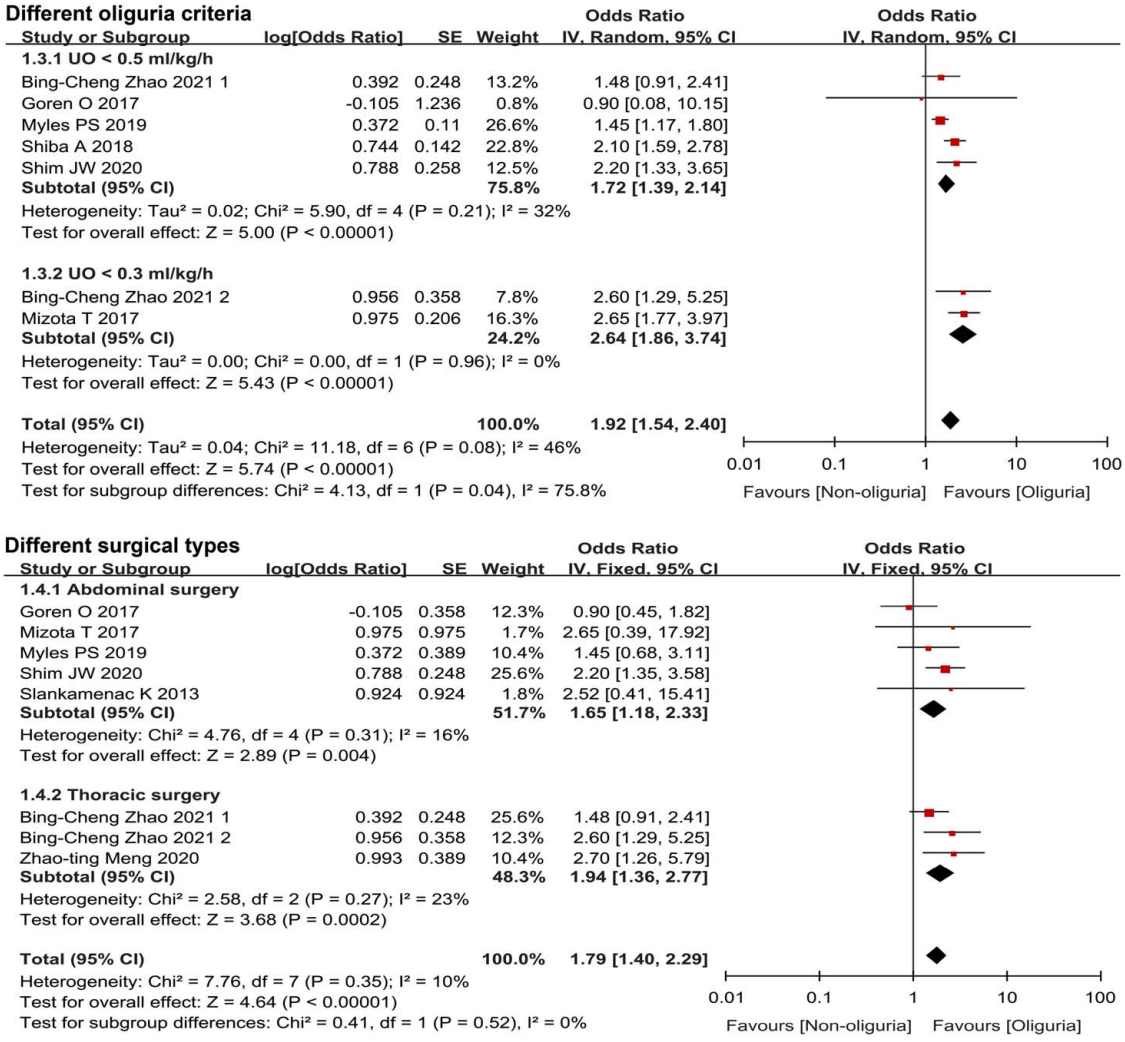

Figure 5. Forest plot of the effect of intraoperative oliguria on the incidence of postoperative acute kidney injury in the subgroup analysis of different oliguria criteria and surgical types.

function has been compromised, preceding the rise in creatinine[37–39]. In such cases, the kidney cannot provide adequate sodium urine, which may be caused by impaired glomerular filtration, primary urine caused by renal tubule leakage back to the interstitium of necrotic units, renal tubular obstruction, or renin–angiotensin system excitement[40]. Therefore, patients with intraoperative oliguria may have already developed subclinical renal dysfunction, making them more likely to progress to AKI postoperatively.

Additionally, the combined ORs of the meta-analysis showed a weak association (moderate heterogeneity) between intraoperative oliguria and postoperative AKI. The possible causes are analyzed as follows. Firstly, the results of the nine included original studies are inconsistent. Seven of the included studies showed a weak correlation[16–22], while the other two studies did not support that intraoperative oliguria was a risk factor for AKI[26,27], which further reduced the effective size of the combined meta-analysis and weakened the association. Secondly, the occurrence of oliguria does not always mean a decrease in renal perfusion pressure or glomerular perfusion. UO is also influenced by many nonrenal factors, including hemodynamics, stress, sympathetic tension, aldosterone, cardiac natriuretic hormone, antidiuretic hormone, etc[41,42]. Finally, intraoperative oliguria is only one of many perioperative risk factors for postoperative AKI, and the multifaceted etiology of AKI may undermine the role of oliguria. Several baseline risk factors (e.g. elderly, hypertension, diabetes) and perioperative factors contributing to postoperative AKI (e.g. systemic inflammation, hemodynamic changes, nephrotoxins) have been recognized, which can help identify patients at risk of postoperative AKI[9–12]. Overall, this weak association reminds us that intraoperative oliguria should not be used as a single predictor or screening indicator for the development of postoperative AKI, but intraoperative oliguria still has important clinical significance because it is an avoidable or modifiable risk factor for postoperative AKI.

However, two recent studies[26,27] suggested that intraoperative oliguria is not a reliable indicator for the development of postoperative AKI. We consider that this discrepancy may be attributable to the sample size. The sample sizes of both retrospective cohort studies (Goren's sample size is 153 and Inacio's sample size is 165) were too small to reliably assess the relationship between exposure and outcome with sufficient statistical power, which may have contributed to the false-negative result that oliguria was not identified as a risk factor for AKI. Additionally, we noticed that our findings differ from earlier studies showing no correlation of intraoperative oliguria with postoperative acute renal failure[9,23–25]. Possible reasons for these findings are that most of the sample sizes in the study are small, and acute renal failure is used to measure postoperative renal function changes, resulting in consideration of only advanced and serious renal insufficiency. Therefore, the relationship between intraoperative oliguria and mild postoperative renal damage may have been overlooked. However, mild renal damage is the leading cause of AKI after noncardiac surgery.

This study supported that intraoperative oliguria is the risk factor for postoperative AKI. Thus, we wondered whether rapid reversal of intraoperative oliguria by intravenous fluid volume expansion could help prevent AKI. Recently, two meta-analyses implemented by Egal *et al*.[43,44], examined an identical question in different populations of studied patients and yielded comparable findings. Their findings suggested that target treatment of oliguria reversal had no contribution to preventing AKI development. According to additional studies[45,46], extra intravenous fluids or diuretic regimens had no benefit in reducing the incidence of postoperative AKI in patients suffering oliguria. The possible causes are analyzed as follows. UO can be affected by many other factors besides renal perfusion, including hemodynamic changes, sympathetic tension, high intra-abdominal pressure, and hormonal influences such as aldosterone and antidiuretic hormone[41,42,47]. Oliguria may sometimes not indicate the onset of hypovolemia and inadequate renal perfusion. Thus, reversal of oliguria as a resuscitation goal and blind volume expansion may result in fluid overload, which substantially contributes to a greater risk of AKI[48], increased postoperative complications, prolonged LOS[49], and elevated mortality[50].

Regardless of the source of intraoperative oliguria, intraoperative oliguria is implicated in the development of AKI and therefore requires close attention. However, physicians should not use oliguria alone as a marker to trigger specific interventions (e.g. additional intravenous fluids or diuretic regimens). When intraoperative oliguria occurs, appropriate preventive interventions should be cautiously implemented to reduce the risk of postoperative AKI. The first clinical procedure to be initiated should carefully calculate and estimate the patient's intraoperative fluid intake and output, whether low blood volume or fluid excess is present, systemic hemodynamic changes, whether hypotension occurs, fluid reactivity, oliguria duration, and other information. Finding and identifying the potential source of oliguria may contribute to the development of appropriate renal protection and management programs, leading to improvements in volume and hemodynamic status (e.g., expansion of intravascular volume and combating hypotension)[22,51,52].

Besides, the result of our subgroup analysis suggested that intraoperative oliguria is significantly associated with postoperative AKI regardless of whether UO less than 0.5 or 0.3 ml/kg/h is the oliguria criterion. Thus, we recommend a clinical threshold of 0.5 ml/kg/h for intraoperative oliguria to avoid the risks involved with milder levels of oliguria. Intraoperative oliguria is linked to postoperative AKI in both surgery types (abdominal or thoracic surgery). It is most likely to benefit from the analysis of oliguria and the risk of AKI and requires our attention, as a significant incidence of AKI follows abdominal and thoracic surgery, second only to cardiac surgery[41]. Moreover, patients undergoing thoracic surgery generally receive restricted intravenous infusions to avoid postoperative pulmonary complications[53]. However, we should also be aware that overly restrictive fluid therapy may lead to oliguria, which is associated with a higher risk of AKI after surgery.

To the best of our knowledge, this systematic review and meta-analysis is the first to systematically analyze and comprehensively summarize data on the correlation of intraoperative oliguria with postoperative AKI, in-hospital mortality, the requirement for RRT, and LOS, especially after abdominal and thoracic surgery, and completed protocol registration on the PROSPERO. It included data from more than 18 500 patients from nine studies, which we analyzed in detail.

There are several limitations to this study that should be mentioned. Firstly, meta-analysis results based on retrospective and observational studies could not infer whether this association was causal or not. Secondly, the included studies differed in many elements, like surgical types and definitions of intraoperative oliguria or AKI. Therefore, the comparably high

clinical heterogeneity may have weakened the authenticity and accuracy of our conclusions. Finally, we excluded studies that did not include data on AKI as the incidence of AKI was our primary outcome and this study was affected by confounding factors. Thus, the applicability of our secondary outcome may be limited.

Conclusions

This meta-analysis found that a significant relationship was seen between intraoperative oliguria and subsequent postoperative acute kidney injury. Furthermore, it also demonstrated that patients with intraoperative oliguria were accompanied with increased in-hospital mortality and demand for postoperative RRT, but not with prolonged hospitalization. We recommend UO less than 0.5 ml/kg/h as the clinical standard of oliguria during operation, which has important clinical significance as a recognizable risk factor of postoperative AKI.

Ethics approval

The study was approved by the Ethics Committee of Xiangya Hospital.

Consent for publication

Not applicable.

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Author contribution

W.Z. and Z.P.: study conception and design; Z.P., S.L., and W. Z.: data analysis and interpretation; Z.P. and N.Z.: supervision of data analysis and interpretation; M.X.: data extraction; Z.P.: drafting of the manuscript; Z.P., S.L., Q.G., W.Z.: critical review and revision of the manuscript.

Conflicts of interest disclosure

The authors have no conflicts of interest.

Research registration unique identifying number (UIN)

1. Name of the registry: PROSPERO (Prospective Register of Systematic Reviews).
2. Unique identifying number or registration ID: CRD42022326281.
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): https://www.crd.york.ac.uk/PROSPERO/display_record.php?RecordID=326281

Guarantor

Wangyuan Zou.

Data availability

This is a meta-analysis article; data availability is not applicable; please contact the corresponding author if some data are needed.

Provenance and peer review

Not commissioned, externally peer-reviewed.

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