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Effect of sevoflurane-based or propofol-based anesthesia on acute kidney injury after surgery for gastric cancer: a retrospective propensity score-matched analysis

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Abstract

Background Acute kidney injury (AKI) is a common complication of major abdominal surgery that is associated with adverse patient outcomes including death. The objective of this study was to investigate the incidence of postoperative AKI after gastric cancer surgery, comparing patients who received propofol-based TIVA with those who received sevoflurane-based INHA.

Methods We analyzed the medical records of all patients aged 19 years or older who underwent radical surgery for primary gastric cancer at the Harbin Medical University Cancer Hospital between January 2010 and September 2018. After propensity score matching, the incidence of AKI in the first 3 postoperative days was compared between patients who received propofol and those who received sevoflurane.

Results 3533 patients were included in the study. After propensity score matching, 1206 patients were assigned to each group. The logistic regression analysis showed that the incidence of AKI was not different in the two groups before (OR 1.05, 95% CI 0.80 to 1.38, $P=0.731$) and after propensity score matching (OR 1.02, 95% CI 0.71 to 1.47, $P=0.926$). Before propensity score matching, acute kidney injury occurred in 146 sevoflurane and 85 propofol patients. The overall incidence was 6.4% in the sevoflurane group and 6.7% in the propofol group. After propensity score matching, acute kidney injury occurred in 60 sevoflurane and 61 propofol patients. The overall incidence was 5.0% in the sevoflurane group and 5.1% in the propofol group.

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Conclusion In this retrospective study, no significant difference was found in the incidence of postoperative AKI after gastrectomy between patients who received propofol-based TIVA and those who received sevoflurane-based INHA in this retrospective study.

Keywords total intravenous anesthesia, inhalational anesthesia, acute kidney injury, gastric cancer, reoperative estimated glomerular filtration rate

Introduction

Acute kidney injury (AKI) is a heterogeneous disease, it refers to a clinical syndrome characterized by a rapid decrease in renal excretory function, with the accumulation of products of nitrogen metabolism such as creatinine and urea and other clinically unmeasured waste products [1–3]. AKI is a common complication of major abdominal surgery that is associated with adverse patient outcomes including death [4]. Perioperative AKI is usually caused by multiple injuries from complex causes, mainly due to the combined effect of renal hypoperfusion, oxidative damage, and inflammation [5]. And some evidence from laboratory and clinical studies suggests that inflammation and its associated molecules could be a key factor in AKI and cause dysfunction of renal cells [6, 7].

Propofol-based total intravenous anesthesia (TIVA) and sevoflurane-based inhalational anesthesia (INHA) are the two main general anesthesia techniques used during gastrectomy, have been shown to modulate the inflammatory responses to surgical stimulations in some clinical studies [5, 8–11].

As far as we know, no one has studied the relationship between AKI after gastric cancer surgery and the type of anesthetic used. Therefore, we aimed to investigate the incidence of postoperative AKI after gastric cancer surgery, comparing patients who received propofol-based TIVA with those who received sevoflurane-based INHA. We hypothesized that the incidence of AKI would be lower in the propofol group than in the sevoflurane group.

Methods

We analyzed the medical records of all patients aged 19 years or older who underwent radical surgery for primary gastric cancer at the Harbin Medical University Cancer Hospital between January 2010 and September 2018. Patients who required dialysis support, repeated surgery, anesthesia using sevoflurane in combination with propofol, unavailable preoperative or postoperative serum creatinine values, and incomplete or missing medical records were excluded from this study. Thus, 3533 patients were included in the final analysis.

According to the distinct anesthesia techniques, they were divided into total intravenous anesthesia group (TIVA) and inhalational anesthesia group (INHA). No premedication was administered before anesthesia induction. For anesthesia induction, spontaneous breathing with 100% oxygen was performed for 2 min for denitrogenation. In both groups, patients underwent anesthesia induction with midazolam 0.05 ~ 0.15 mg/kg, 0.5 ug/kg fentanyl, 0.15 ~ 0.2 mg/kg cisatracurium and 1 ~ 2.5 mg/kg propofol. For patients with total intravenous anesthesia, anesthesia was maintained with propofol and remifentanil infusion. For patients with inhalation anesthesia, anesthesia was maintained with sevoflurane inhalation and remifentanil infusion. Patients received patient-controlled intravenous analgesia (PCIA) at dosages of 3 ug/ml of fentanyl or 0.5 ug/ml of sufentanil for 72 h after surgery. In addition, in all patients on pre-operative treatment with antihypertensive drugs, medication was interrupted on the day of surgery. Postoperative AKI was diagnosed using Kidney Disease: Improving Global Outcomes (KDIGO) criteria [2]. We used plasma creatinine concentration as our primary marker for renal function because it has been validated as clinically important. The last serum creatinine concentration measured before surgery was used as the baseline serum creatinine concentration in this study. Serum creatinine values during the first 3 postoperative days were used to diagnose AKI. AKI stage 1 was defined as 1.5 to 1.9 times baseline or ≥ 0.3 mg/dl (≥ 26.5 μmol/l) increase. AKI stage 2 was defined as 2.0 to 2.9 times baseline. AKI stage 3 was defined as 3.0 times baseline or increase in serum creatinine to ≥ 4.0 mg/dl (≥ 353.6 μmol/l) or initiation of renal replacement therapy (RRT). AKI Incidence (%) = (Number of new AKI cases during the study period/Total at-risk population) ×100%. The denominator (at-risk population) excluded patients with pre-existing end-stage renal disease (ESRD) or missing baseline creatinine data. Hospital electronic medical records were retrospectively analyzed to obtain demographic and clinical data on all patients and their postoperative outcomes. Data included sex, age, BMI, Hb, ALB, method of anesthesia, surgery time, antibiotic or antiviral drug use (vancomycin, cephalosporin, aminoglycoside, rifampin, acyclovir or sulphonamide), colloid, urine output, crystalloid, hydroxyethyl starch use, transfusion of packed RBC, diuretic use, preoperative cerebrovascular disease, preoperative chronic kidney disease, preoperative chronic lung disease, preoperative ischaemic heart disease, preoperative diabetes mellitus, preoperative hypertension, ASA physical status, smoking, drinking preoperative anemia and serum uric

acid. The main measure of this study was the incidence of AKI after gastric resection.

Statistical analysis

Continuous variables are shown as mean ± SD and dichotomous variables as numbers (percentages). For continuous variables, Student's t-test is used if they follow a normal distribution. If they do not follow a normal distribution, the Mann-Whitney U test is used. Dichotomous variables were compared using χ^2 or Fisher's exact tests, as appropriate. To reduce the influence of confounding variables, propensity score matching (PSM) method was used to adjust intergroup differences between sevoflurane and propofol group [12]. Match using all the variables listed in the Table 1. The two groups of patients were matched using a 1:1 nearest neighbor matching algorithm without replacement, with a caliper of 0.25 of standard deviation of the propensity score on the logit scale. The balance of covariates between the TIVA and INHA groups was assessed by the standardized mean difference (SMD). An SMD < 0.1 indicated a good balance in the covariates between the two groups. we used the x^2 test to compare the incidence of postoperative AKI between the two groups. Next, we performed logistic regression analysis on the unmatched and matched cohort to investigate whether propofol-based TIVA was more associated with postoperative AKI than sevoflurane-based INHA. The results of logistic regression analysis were presented as odds ratio (OR) with 95% confidence intervals (CIs). Statistical analysis was performed with the SPSS 27.0 software (IBM Corporation, Armonk, NY, USA) and R statistical software version 3.6.3 (R Foundation for Statistical Computing, Vienna, Austria). $P < 0.05$ was considered statistically significant. G*Power 3.1 software was employed for statistical power analysis to assess the study's strength.

Table 1 Comparison between Sevoflurane group and Propofol group before and after propensity score matching

Variables	Unmatched cohort, *n* = 3533 Sevoflurane Propofol *n* = 2270 *n* = 1263		SMD	*P*	Matched cohort, *n* = 2412 Sevoflurane Propofol *n* = 1206 *n* = 1206		SMD	*P*
Age (years)	58.8 ± 10.2	58.8 ± 9.9	0.005	0.894	58.7 ± 10.2	58.8 ± 9.9	0.006	0.892
Sex: male	1647(72.6)	912(72.2)	0.008	0.856	869(72.1)	870(72.1)	0.002	1.000
BMI (kgm^{-2})	22.8 ± 3.2	22.7 ± 3.3	0.028	0.429	22.7 ± 3.3	22.6 ± 3.2	0.011	0.796
ASA physical status	30(1.3)	10(0.8)	0.076	0.098	11(0.9)	9(0.7)	0.019	0.898
1	2172(95.7)	1202(95.2)			1150(95.4)	1153(95.6)		
2	68(3.0)	51(4.0)			45(3.7)	44(3.6)		
≥ 3								
Alb (g/L)	40.7 ± 4.8	40.2 ± 5.1	0.090	0.010	40.3 ± 4.8	40.3 ± 5.0	< 0.001	1.000
Hb (g/L)	129.4 ± 25.0	127.8 ± 26.3	0.061	0.081	128.3 ± 25.4	128.2 ± 26.2	0.004	0.926
Serum uric acid (umol/L)	280.6 ± 94.5	270.9 ± 92.6	0.103	0.003	276.9 ± 93.1	272.9 ± 91.6	0.043	0.286
Surgery time (min)	175.7 ± 50.1	175.6 ± 46.7	0.002	0.946	175.6 ± 48.6	175.5 ± 46.3	0.002	0.968
Colloid (ml)	911.0 ± 390.4	900.8 ± 459.1	0.024	0.486	895.9 ± 373.6	892.8 ± 457.4	0.007	0.855
Crystalloid (ml)	1245.9 ± 396.3	1186.3 ± 405.4	0.149	< 0.001	1189.5 ± 385.1	1193.7 ± 400.9	0.011	0.794
Urine output (ml)	358.9 ± 189.1	131.6 ± 157.3	0.260	< 0.001	316.2 ± 150.5	314.8 ± 151.8	0.009	0.825
Antibiotic or antiviral drug use	5(0.2)	6(0.5)	0.043	0.323	2(0.2)	2(0.2)	< 0.001	1.000
Hydroxyethyl starch use	3(0.1)	1(0.1)	0.016	1.000	2(0.2)	1(0.1)	0.024	1.000
Transfusion of packed RBC	391(17.2)	210(16.6)	0.016	0.684	204(16.9)	201(16.7)	0.007	0.913
Diuretic use	1(0.1)	2(0.2)	0.036	0.606	1(0.1)	1(0.1)	< 0.001	1.000
Preoperative anaemia	317(14.0)	203(16.1)	0.059	0.100	180(14.9)	183(15.2)	0.007	0.909
Preoperative cerebrovascular disease	43(1.9)	29(2.3)	0.028	0.493	29(2.4)	24(2.0)	0.028	0.578
Preoperative chronic kidney disease	7(0.3)	12(1.0)	0.081	0.024	3(0.2)	3(0.2)	< 0.001	1.000
Preoperative chronic lung disease	10(0.4)	15(1.2)	0.083	0.02	4(0.3)	6(0.5)	0.026	0.751
Preoperative ischaemic heart disease	101(4.4)	61(4.8)	0.018	0.664	52(4.3)	58(4.8)	0.024	0.626
Preoperative diabetes mellitus	88(3.9)	50(4.0)	0.004	0.976	46(3.8)	47(3.9)	0.004	1.000
Preoperative hypertension	256(11.3)	140(11.1)	0.006	0.906	143(11.9)	135(11.2)	0.021	0.655
Smoking	1231(54.2)	659(52.2)	0.041	0.256	611(50.7)	638(52.9)	0.045	0.289
Drinking	875(38.5)	473(37.5)	0.023	0.544	435(36.1)	461(38.2)	0.045	0.292

Values are mean ± SD or number of patients (%). Antibiotic or antiviral drug includes vancomycin, cephalosporin, aminoglycoside, rifampin, acyclovir and sulphonamide

AKI acute kidney injury, *ASA* American Society of Anesthesiologists, *Alb* albumin, *BMI* body mass index, *Hb* hemoglobin, *RBC* red blood cells, *SMD* standardized mean difference

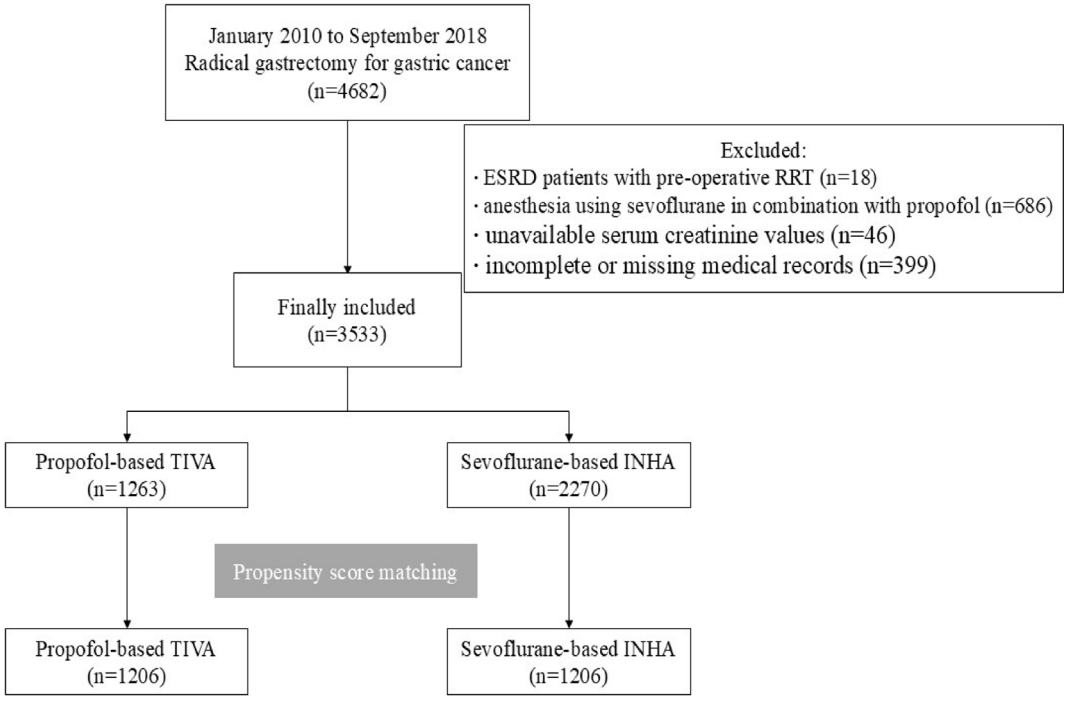


Fig. 1 Flow chart of study. *ESRD* end-stage renal disease, *RRT* renal replacement therapy, *TIVA* total intravenous anaesthesia, *INHA* inhalational anesthesia

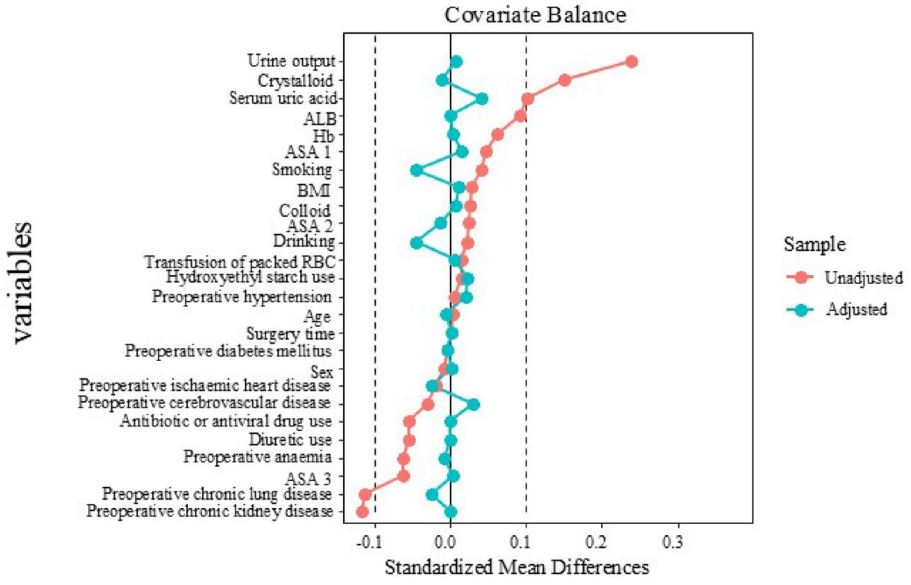


Fig. 2 The distribution of standardized mean difference for variables included before and after matching. *BMI* body mass index, *ASA* American Society of Anesthesiologists, *ALB* albumin, *Hb* hemoglobin, *RBC* red blood cell count

Results

A total of 4682 patients aged 19 years or older underwent radical gastrectomy for gastric cancer at the Harbin Medical University Cancer Hospital between January 2010 and September 2018. This sample size provided 83% power for the test. Eighteen patients were excluded due to end-stage renal disease (ESRD) defined as receiving RRT or preoperative estimated glomerular filtration rate (eGFR) less than 15mlmin^{-1} 1.73 m^{-2}, 686 patients were excluded due to propofol combined with sevoflurane anesthesia, 46 patients were excluded due to serum creatinine not available and 399 patients were excluded due to incomplete or missing medical records. Finally, 3533 patients were included in the study, including 1263 propofol-based intravenous anesthesia and 2270 sevoflurane-based inhalation anesthesia. After propensity score

Table 2 Incidence of postoperative acute kidney injury before and after propensity score matching

Variables	Sevoflurane	Propofol	95%CI of difference in incidence	*P*
Before propensity score matching				
Total acute kidney injury	146/2270 (6.4)	85/1263 (6.7)	-0.014 to 0.020	0.492
Acute kidney injury stage 1	114/2270 (5.0)	63/1263 (5.0)	-0.015 to 0.015	0.929
Acute kidney injury stage 2	24/2270 (1.1)	17/1263 (1.3)	-0.004 to 0.010	0.125
Acute kidney injury stage 3	8/2270 (0.3)	5/1263 (0.4)	-0.004 to 0.005	0.683
After propensity score matching				
Total acute kidney injury	60/1206 (5.0)	61/1206 (5.1)	-0.022 to 0.032	0.454
Acute kidney injury stage 1	43/1206 (3.6)	40/1206 (3.3)	-0.017 to 0.012	0.503
Acute kidney injury stage 2	13/1206 (1.1)	16/1206 (1.4)	-0.007 to 0.010	0.447
Acute kidney injury stage 3	4/1206 (0.3)	5/1206 (0.4)	-0.003 to 0.007	0.205

Values are number of patients (%)

CI confidence interval

Table 3 Logistic regression analysis for postoperative acute kidney injury according to type of anesthesia before and after propensity score matching

Variables	Odds ratio (95% confidence intervals)	*P*
Before propensity score matching		0
Total AKI		
Propofol (vs. sevoflurane)	1.05 (0.80–1.38)	0.731
AKI stage 1		
Propofol (vs. sevoflurane)	0.99 (0.72–1.36)	0.965
AKI stage 2		
Propofol (vs. sevoflurane)	1.27 (0.68–2.39)	0.444
AKI stage 3		
Propofol (vs. sevoflurane)	1.12 (0.37–3.44)	0.838
After propensity score matching		
Total AKI		
Propofol (vs. sevoflurane)	1.02 (0.71–1.47)	0.926
AKI stage 1		
Propofol (vs. sevoflurane)	0.93 (0.60–1.44)	0.738
AKI stage 2		
Propofol (vs. sevoflurane)	1.16 (0.55–2.44)	0.704
AKI stage 3		
Propofol (vs. sevoflurane)	1.50 (0.42–5.34)	0.529

AKI acute kidney injury

matching, 1206 patients were assigned to each group (Fig. 1). Table 1 shows the magnitude of the differences between two groups before and after propensity score matching. The inter-group differences between the two groups were well balanced, with all SMDs less than 0.1 (Fig. 2).

Table 2 shows the results of comparison of the incidences of postoperative AKI in the sevoflurane group and propofol groups before and after propensity score matching. Before propensity score matching, acute kidney injury occurred in 146 sevoflurane and 85 propofol patients. The overall incidence was 6.4% in the sevoflurane group and 6.7% in the propofol group. There was no difference in the incidence of postoperative AKI between propofol group and sevoflurane group (95%CI of difference in incidence: - 0.014 to 0.020, $P = 0.492$). The result obtained after propensity score matching is the same (95%CI of difference in incidence: -0.022 to 0.032, $P = 0.454$). Acute kidney injury occurred in 60 sevoflurane and 61 propofol patients. The overall incidence was 5.0% in the sevoflurane group and 5.1% in the propofol group. Figure 3 shows the incidence rates of each AKI

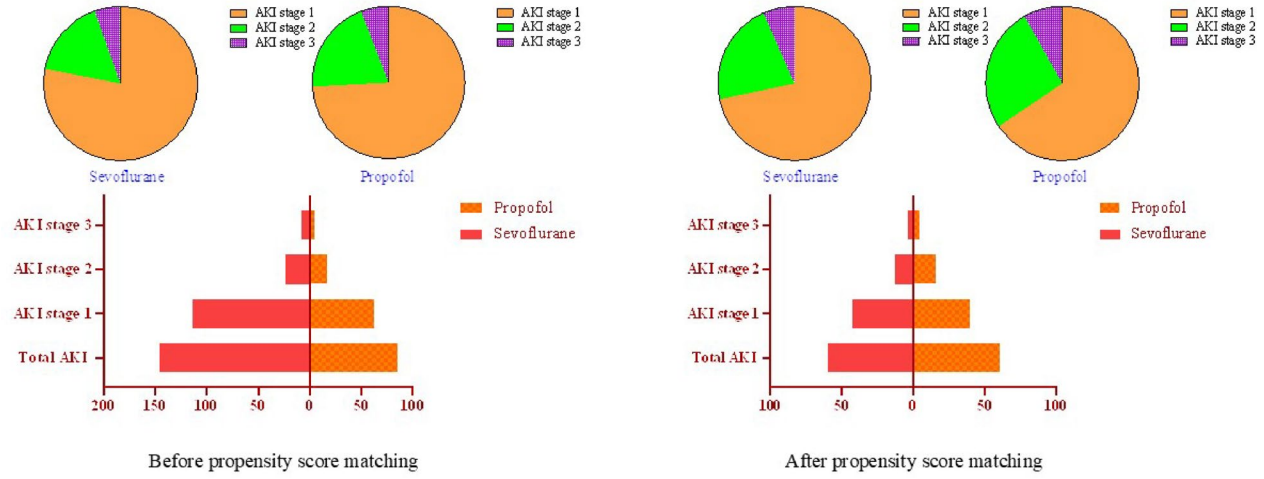
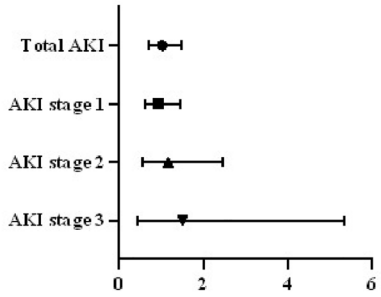


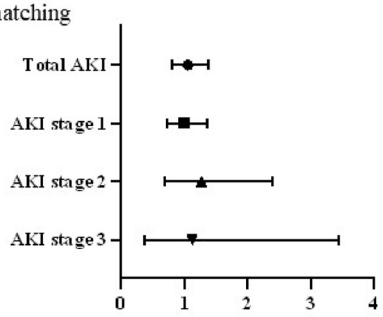
Fig. 3 The incidence rates of each AKI stage in the propofol group and sevoflurane group. The incidence rates of each AKI stage in the propofol group and sevoflurane group. *AKI* acute kidney injury

Variable	OR(95%CI)	*P*
Total AKI	1.02(0.71-1.47)	0.926
AKI stage 1	0.93(0.60-1.44)	0.738
AKI stage 2	1.16(0.55-2.44)	0.704
AKI stage 3	1.50(0.42-5.34)	0.529



Before propensity score matching

Variable	OR(95%CI)	*P*
Total AKI	1.05(0.80-1.38)	0.731
AKI stage 1	0.99(0.72-1.36)	0.965
AKI stage 2	1.27(0.68-2.39)	0.444
AKI stage 3	1.12(0.37-3.44)	0.838



After propensity score matching

Fig. 4 The forest plot between anaesthesia type and postoperative acute kidney injury. Logistic regression analysis of the forest plot between anesthesia type and postoperative acute kidney injury before and after propensity score matching. *AKI* acute kidney injury

stage in the propofol group and sevoflurane group before and after propensity score matching.

The logistic regression analysis (Table 3) showed that the incidence of AKI was not different in the two groups before (OR 1.05, 95% CI 0.80 to 1.38, $P=0.731$) and after propensity score matching (OR 1.02, 95% CI 0.71 to 1.47, $P=0.926$). There were no significant differences in the stage of AKI between the propofol and sevoflurane groups before propensity score matching (stage1, OR 0.99, 95%CI 0.72 to 1.36, $P=0.965$, stage2, OR 1.27, 95%CI 0.68 to 2.39, $P=0.444$, stage3, OR 1.12, 95%CI 0.37 to 3.44, $P=0.838$). There were also no significant differences in the stage of AKI between the propofol and sevoflurane groups after propensity score matching (stage1, OR 0.93, 95%CI 0.60 to 1.44, $P=0.738$, stage2, OR 1.16, 95%CI 0.55 to 2.44, $P=0.704$, stage3, OR 1.50, 95%CI 0.42 to 5.34, $P=0.529$), as shown in Table 3; Fig. 4.

Discussion

In this retrospective study, we investigated the relationship between anesthetic agent and the incidence of acute kidney injury after gastric cancer surgery. Our results showed that the incidence of postoperative AKI after gastrectomy did not differ between the sevoflurane group and the propofol group before or after propensity score matching.

Propofol, an ultra-fast-acting intravenous anesthetic with anti-inflammatory and antioxidant properties and few adverse effects [13], has been widely used in clinical applications. In some studies of rats, propofol reduced oxidative stress and AKI [14, 15]. The renal protective effect of propofol has been demonstrated in animal experiments using models of renal artery or abdominal aorta occlusion [16, 17]. Sevoflurane is a widely used volatile anesthetic with potent multiorgan protective effects during perioperative period [18–20]. In another randomized controlled trial, sevoflurane anesthesia increased the risk of kidney damage compared to propofol anesthesia [21]. Volatile anesthesia may reduce urine output to the degree that the AKI criteria are reached, but it is uncertain how that associates with structural long-term damage to the kidney [22]. However, multiple studies demonstrate the protective effect of volatile anesthetics against renal injury [23]. Anyway, due to the different evaluation criteria and the existence of experimental limitations, the effects of propofol and sevoflurane on AKI have been controversial.

Postoperative AKI is a frequent complication associated with increased medical expenses [6, 19, 24]. Thus, prevention of postoperative AKI is important. One meta-analysis revealed that propofol is associated with lower incidence of AKI compared with volatile anesthesia [25]. Another meta-analysis conducted on randomized controlled trials (RCTs) determined that sevoflurane reduced the risk of AKI compared to propofol [26]. In a clinical study, Yoo et al. demonstrated that propofol anesthesia significantly reduced the incidence and severity of acute kidney injury in patients undergoing valvular heart surgery with cardiopulmonary bypass compared with sevoflurane. The postoperative cystatin C was significantly

lower in the propofol group at 24 and 48 h. Serum interleukin-6 at 6 h after aorta cross-clamp removal, C-reactive protein at postoperative day 1, and segmented neutrophil counts at postoperative day 3 were also significantly lower in the propofol group [27]. This beneficial effect of propofol may be related to its ability to attenuate the perioperative increase in proinflammatory mediators. Li et al. found that the incidence of perioperative AKI was significantly lower in the sevoflurane group than in the propofol group. In this study, sevoflurane anesthesia was considered a method to reduce kidney damage by stabilizing hemodynamic changes, regulating oxidative stress and inflammation, etc [28]. Premuzic et al. found that ICU patients developed AKI and AKD more frequently at the end of ICU stay after neurosurgery with sevoflurane balanced anesthesia [29]. In a randomized controlled trial, Yoon et al. found that the type of anesthetic drug did not affect the incidence of acute kidney injury after nephrectomy [30]. In a propensity score matched retrospective study, Lee et al. found that propofol may be a better general anesthetic for nephrectomy than volatile drugs to reduce postoperative renal insufficiency [31]. In another retrospective observational study involving 2872 individuals, Oh et al. obtained the same results in patients who underwent curative lung resection surgery for non-small cell lung cancer [32]. However, Sondekoppam et al. did not find any association between the use of sevoflurane and postoperative renal impairment compared with other agents used for anesthesia maintenance [33]. Current research presents contradictory results. The published research on anesthetic effects on renal function in humans is inconclusive. Why some studies show lower AKI incidence after propofol anesthesia compared with volatile anesthesia and some show no difference at all is not readily explained.

A few reasons may have contributed to our failure to observe retrospective effects of propofol in this study. First, a continuous infusion of remifentanil, which may have retrospective effects in the peri-operative period, was used in both the propofol and sevoflurane groups [34, 35]. Thus, our use of a remifentanil infusion may have masked the effects of propofol and sevoflurane on postoperative kidney function. Another reason why we did not find propofol to have a nephroprotective effect is that propofol concentrations that produce antioxidant effects may vary from tissue to species specificity. The organ-protective effect of propofol was dose-dependent [36].

Another notable finding of the current study was that sevoflurane showed no renal toxicity. This may be because Compound A has important effects on the kidneys, and our study did not expose subjects to doses of Compound A that are toxic and may occur in some clinical settings.

The present study has several limitations. Firstly, because of its retrospective design, we could not control for all confounding parameters that might have affected our results. Although we performed PS analysis to control for selection bias, we could not entirely remove residual confounding. Secondly, although we included many covariates in propensity score matching to balance the sevoflurane-based inhalational anaesthesia and propofol-based TIVA groups, we did not consider intra-operative blood pressure and vasopressor use. As intra-operative vasopressor use or hypotension is associated with AKI after gastric cancer surgery, these may have biased the results of this study. Thirdly, due to the retrospective cohort design of this study, there may be selection bias. Fourth, this research was conducted in a single center, which might have limited its generalizability. Finally, Some antihypertensive drugs such as β receptor blocker drugs are advised continuous application on the morning of surgery, or it may increase the rate of circulatory system complications which lead to hypotension. But as this was a retrospective study, medication discontinuation reflected real-world practice variability. Future prospective studies are needed to standardize medication.

Conclusions

In gastric cancer surgery, there was no significant difference in the incidence of postoperative AKI between patients who received propofol intravenous anesthesia and those who received sevoflurane inhalation anesthesia.

Abbreviations

AKI	Acute kidney injury
TIVA	Total intravenous anesthesia
NIHA	Inhalational anesthesia
PCIA	Patient-controlled intravenous analgesia
RRT	Renal replacement therapy
PSM	Propensity score matching
OR	Odds ratio
CI	Confidence intervals
ESRD	End-stage renal disease
eGFR	Estimated glomerular filtration rate
ASA	American Society of Anesthesiologists
Alb	Albumin
BMI	Body mass index
RBC	Red blood cells
SMD	Standardized mean difference

Author contributions

Y.S., S.L. had contributions to study conception, design. Y.Y. and H.C. drafting the article and acquisition of data.L.W. had contributions to interpretation of data. H.C., L.W., Y.S. and M.W. had responsibility for the revision of important intellectual content and final approval of the version to be published.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was performed in accordance with the ethical principles of the Declaration of Helsinki. The study procedures were approved by the Ethics Committee of The Harbin Medical University Cancer Hospital. This is a retrospective study and individual informed consent to participate for this retrospective analysis with routine clinical data was waived by the Ethics Committee of The Harbin Medical University Cancer Hospital.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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