Prevalence and risk factors of acute kidney injury in adults with obesity undergoing bariatric surgery: a systematic review and meta-analysis

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KEY WORDS

acute kidney injury, bariatric surgery, meta-analysis, obesity, risk factor

ABSTRACT

INTRODUCTION Acute kidney injury (AKI) is a potentially severe complication after bariatric surgery, associated with increased morbidity, prolonged hospitalization, and impaired long-term renal outcomes. However, the reported prevalence of postoperative AKI and its risk factors in adults with obesity remain inconsistent across studies.

AIM This systematic review and meta-analysis aimed to estimate the pooled prevalence of AKI after bariatric surgery and identify associated risk factors in adults with obesity.

MATERIALS AND METHODS A comprehensive literature search of PubMed, Embase, and the Cochrane Library databases was performed from inception to March 1, 2025, following the 2020 Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. Observational studies reporting AKI prevalence or risk factors in adults with obesity undergoing bariatric surgery were included. Two reviewers independently screened the studies, extracted data, and assessed their quality using the Newcastle–Ottawa scale. Random-effects models were applied to calculate pooled prevalence and odds ratios (ORs). Subgroup, sensitivity analyses, and publication bias assessments were also conducted.

RESULTS A total of 11 studies involving 242 159 patients were analyzed. The overall pooled prevalence of postoperative AKI was 2.5% (95% CI, 1.8–3.1), with substantial heterogeneity ($I^2 = 98.1\%$). Subgroup analyses showed higher AKI rates in case-control studies, small sample studies, and studies based on the AKI Network criteria. Significant risk factors for AKI included male sex (OR, 2.15; 95% CI, 1.54–3.01), pre-existing renal insufficiency (OR, 5.33; 95% CI, 1.4–20.26), hypertension (OR, 1.74; 95% CI, 1.37–2.21), hyperlipidemia (OR, 1.53; 95% CI, 1.04–2.24), and elevated body mass index (OR per unit increase, 1.05; 95% CI, 1.02–1.09). Diabetes and age were not significantly associated with AKI across the studies.

CONCLUSIONS AKI occurs in approximately in 2.5% of adults with obesity following bariatric surgery. Identifying

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Received: October 23, 2025.
Revision accepted: December 4, 2025.
Published online: January 9, 2026.
Wideochir Inne Tech Maloinwazyjne. 2026; 21 (1): 13-21
doi:10.20452/wiitm.2026.18007
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INTRODUCTION Obesity represents one of the most pressing global health concerns.[1,2] According to the World Health Organization, the global prevalence of obesity continues to escalate, particularly in younger populations and in developing countries.[3-5] Obesity is associated with a wide range of metabolic disturbances—including type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, and cardiovascular disease—and exerts a substantial impact on renal physiology.[6-8] Numerous studies have highlighted the direct nephrotoxic effects of obesity, such as glomerular hyperfiltration, increased renal tubular workload, insulin resistance, activation of pro-inflammatory cytokines, and the renin–angiotensin–aldosterone system, all of which contribute to an elevated risk of chronic kidney disease (CKD).[9-12]

In light of the limited and often unsustainable effectiveness of conservative lifestyle interventions and pharmacotherapy, bariatric surgery has become the most durable and effective

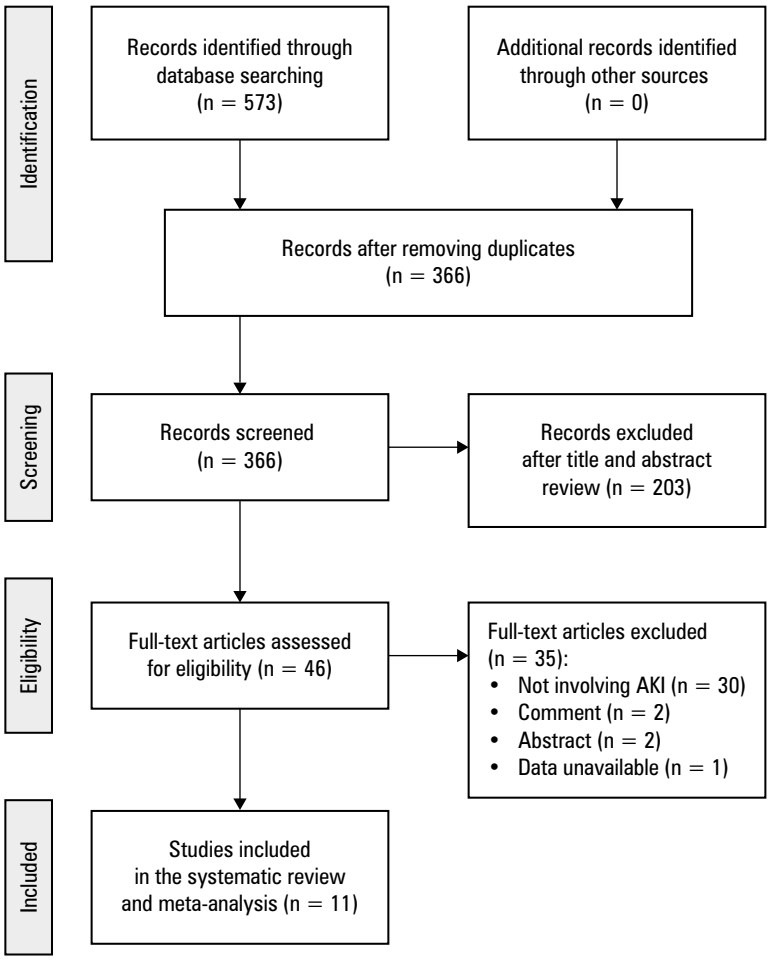

FIGURE 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow diagram of the study selection

treatment option for patients with severe obesity.[13,14] Procedures, such as Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG), are associated not only with sustained weight loss but also with significant improvements in glycemic control, remission of T2DM, resolution of hypertension, and even partial reversal of early-stage nephropathy.[15-17] Accordingly, bariatric surgery may be proposed as a renoprotective intervention, particularly in patients with obesity-related kidney disease.[18] However, the physiological stress associated with major surgery, coupled with considerable fluid shifts, changes in hemodynamics, and exposure to nephrotoxic agents, places patients at a risk of postoperative complications, among which acute kidney injury (AKI) has become an emerging concern.[19,20] AKI is defined as a rapid deterioration of renal function over hours to days, and it is typically diagnosed based on serum creatinine level increases and/or reduced urine output. In nonbariatric surgical populations, such as those undergoing cardiac, vascular, or orthopedic procedures, AKI is well known to be associated with longer hospital stays, greater costs, and worse long-term survival.

In the bariatric population, however, the true burden of AKI remains poorly characterized. Reported incidence rates vary widely from less than 1% to over 10%, partly due to inconsistent AKI definitions, heterogeneity in surgical technique, and variability in baseline renal function and comorbidity profiles.[19,21-24] Additionally, bariatric patients are often subject to perioperative factors that may predispose to AKI, including prolonged operative times, perioperative hypotension, rapid diuresis or dehydration, use of nonsteroidal anti-inflammatory drugs, iodinated contrast exposure, and baseline CKD or DM. Beyond these systemic and hemodynamic contributors, perioperative skeletal muscle injury and rhabdomyolysis have been increasingly recognized as important mechanisms of AKI in bariatric surgery. Patients with severe obesity undergoing lengthy procedures in fixed positions are at a particular risk of muscle compression, ischemia, and myonecrosis, with subsequent myoglobin release causing pigment-induced tubular toxicity and obstruction. In addition, anesthetic and intraoperative factors—including the depth and duration of neuromuscular blockade, pneumoperitoneum-related increases in intra-abdominal pressure, and inadequate hemodynamic support—may further amplify the risk of rhabdomyolysis and downstream kidney injury, whereas optimized positioning and the use of vacuum mattresses or pressure--redistribution systems can mitigate muscle ischemia and improve tissue perfusion. Importantly, even mild or transient postoperative AKI is increasingly recognized to confer a significantly elevated risk of progression to CKD, cardiovascular complications, and mortality, making early identification and prevention of AKI a critical perioperative priority. Despite the growing attention paid to renal outcomes after bariatric surgery, the current literature lacks a comprehensive synthesis of AKI prevalence and its predictors in this unique surgical population. While individual studies have identified potential risk factors, such as male sex, older age, DM, pre-existing CKD, and RYGB procedure, the results are often inconsistent and underpowered. No previous meta-analysis has, to our knowledge, specifically addressed the pooled prevalence of AKI in adults with obesity undergoing bariatric surgery, nor systematically evaluated risk factors associated with it.

AIM To bridge this knowledge gap, we conducted a systematic review and meta-analysis to estimate the pooled prevalence of postoperative AKI in adults with obesity undergoing bariatric surgery and identify clinical risk factors associated with increased AKI risk. Our findings aimed to inform preoperative risk stratification and support the development of targeted perioperative renal protection strategies in this rapidly growing surgical population.

MATERIALS AND METHODS This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[25] We aimed to identify all relevant studies

evaluating the prevalence and risk factors of AKI in adults with obesity undergoing bariatric surgery. The methodology was registered on the open science framework (https://osf.io/rz9ua/).

Data sources and search strategy We systematically searched PubMed, Embase, and the Cochrane Library databases from inception to March 1, 2025, using a comprehensive strategy that combined Medical Subject Headings and keywords related to bariatric surgery, obesity, and AKI. No language or geographic restrictions were applied. To ensure comprehensiveness, we also reviewed the reference lists of all included articles and relevant reviews, and searched gray literature and conference proceedings.

Study selection Studies were eligible if they reported the prevalence or predictors of AKI in adult patients (aged 18 years or older) with obesity, who underwent any form of bariatric surgery, including RYGB, SG, biliopancreatic diversion, or adjustable gastric banding. Eligible studies had to report postoperative AKI as a clinical outcome, defined by any standard classification. Observational studies (cohort or case-control) were included, while reviews, case series, editorials, and nonhuman studies were excluded. We also excluded studies that lacked sufficient data on AKI outcomes or focused on nonobese or pediatric populations.

Data extraction Two reviewers (WX and PJ) independently screened the titles and abstracts of all retrieved articles and conducted full-text assessments of potentially eligible studies. Any disagreements were resolved by consensus or by consultation with a third reviewer (PZ). From each eligible study, we extracted data on the study design, country, sample size, patient demographics, type of bariatric procedure, definition and timing of AKI, prevalence of AKI, and reported risk factors. Where studies reported multiple risk estimates, the most fully adjusted model was extracted. The authors were contacted when necessary to clarify unclear or missing data.

Quality assessment The methodological quality of the included studies was assessed using the Newcastle–Ottawa scale (NOS),[26] which evaluates selection of study groups, comparability, and ascertainment of outcomes. Studies scoring 7 or higher were considered high quality. Two reviewers (WX and PZ) independently completed the quality assessment, with discrepancies resolved through discussion.

Statistical analysis For quantitative synthesis, we used random-effects models based on the DerSimonian and Laird method to account for between-study heterogeneity. The pooled prevalence of AKI was calculated with 95% CIs using the Freeman–Tukey double arcsine transformation. We also performed meta-analyses of risk factors by pooling the adjusted odds ratios (ORs) or hazard ratios (HRs) reported across the studies. Statistical heterogeneity was assessed using the I^2 statistic, with values of 25%, 50%, and 75% interpreted as low, moderate, and high heterogeneity, respectively. Prespecified subgroup analyses were conducted based on the study design, sample size, NOS scores, single- or multicenter format, and AKI criteria. Sensitivity analyses were conducted using the leave-one-out testing method. Publication bias was assessed by visual inspection of funnel plots and the Egger regression test, with a P value below 0.05 indicating potential asymmetry. All analyses were performed using Stata software, version 14.0 (StataCorp, College Station, Texas, United States), and significance was set at a 2-sided P value below 0.05.

Ethics The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

RESULTS

Study selection A total of 573 studies were identified through systematic database searching, with no additional records retrieved from other sources. After removing duplicates, 366 records remained for title and abstract screening. Of these, 320 were excluded based on irrelevance to AKI or bariatric surgery. Forty-six full-text articles were reviewed in detail, and 35 were excluded due to a lack of AKI outcomes (n = 30), commentary, review (n = 2), or abstract-only report format (n = 2), and data unavailability (n = 1). Finally, a total of 11 studies were included in the meta-analysis.[19,21,22,24,27-33] The detailed process of study selection is illustrated in FIGURE 1.

Study characteristics The 11 included studies were published between 2011 and 2025 and involved a total of 242159 adults with obesity undergoing bariatric surgery across various geographic regions, including the United States, Canada, the United Kingdom, Australia, and Greece. The majority were retrospective cohort studies, 1 was a prospective, and 1 a case-control study (TABLE 1). Sample sizes ranged from 23 to 747926. Mean age of the participants ranged from 38.9 to 53 years. Most studies investigated RYGB, SG, or a mix of surgical types. AKI was most commonly defined using the AKI Network (AKIN) or Kidney Disease, Improving Global Outcomes (KDIGO) criteria. The prevalence of AKI varied widely, from 0.06% to 17.5%, depending on surgical type, setting, and diagnostic criteria. Quality appraisal using the NOS (TABLE 2) showed that 9 of the 11 studies were of high methodological quality (≥7 points), and 2 were rated as moderate quality (5 points). Common strengths included adequate selection and representativeness of cohorts, and consistent outcome ascertainment. Some studies lacked adjustment for additional confounders or had short follow-ups. Importantly, none

TABLE 1 Baseline characteristics of the studies on acute kidney injury after bariatric surgery

Author, year	Type of study	Study design	Country	Enrollment period	Age, y, mean (SD) or range	Type of bariatric surgery	AKI definition criteria	Prevalence of AKI, n/N (%)
Weingarten et al,[33] 2011	Single-center	Retrospective cohort study	United States	2003–2009	47.4 (8)	Laparoscopic gastric bypass	AKIN	21/340 (6.2)
Koukoulaki et al,[29] 2013	Single-center	Prospective cohort study	Greece	January–September 2010	39 (9)	Biliopancreatic diversion	AKIN	2/23 (8.7)
Weingarten et al,[32] 2013	Single-center	Case control study	United States	2005–2011	51.6 (12.7)	Gastric bypass	AKIN	71/1227 (5.8)
Morgan et al,[30] 2015	Multicenter	Retrospective cohort study	Australia	2007–2011	49.2 (11.9)	Mixed: SG, LAGB, RYGB, and revisions	AKIN	103/590 (17.5)
Abdullah et al,[21] 2016	Single-center	Retrospective cohort study	Canada	2009–2014	44.2 (10.9)	Laparoscopic RYGB and SG	KDIGO	35/1230 (2.9)
Koppe et al,[22] 2018	Multicenter	Retrospective cohort study	United Kingdom	1997–2015	45.2 (10.7)	Gastric band (45.1%), bypass (40.7%), and SG (13.8%)	ICD-10	39/2643 (1.5)
Nor Hanipah et al,[31] 2018	Single-center	Retrospective cohort study	United States	2008–2015	53 (43–61)	RYGB (69%), SG (14%), revision (14%), and plication (3%)	KDIGO	42/4722 (0.9)
Valera et al,[24] 2023	Multicenter	Retrospective cohort study	United States and Canada	2015–2019	44.4 (11.9)	Laparoscopic SG (73.1%) and laparoscopic RYGB (26.8%)	NA	446/747926 (0.06)
Khalid et al,[28] 2022	Multicenter	Retrospective cohort study	United States	2010–2018	44.4 (11.9)	SG	ICD-9/ ICD-10 diagnosis	176/16736 (1.05)
Abi Mosleh et al,[19] 2025	Single-center	Retrospective cohort study	United States	2008–2022	48.9 (11.6)	RYGB (69.4%), SG (25%), and DS (5.6%)	AKIN	51/1697 (3)
Cooper et al,[27] 2024	Single-center	Retrospective cohort study	United States	2019–2023	44.6 (17.4–85.3)	Laparoscopic or robotic SG and RYGB	NA	3/1224 (0.25)

Abbreviations: AKI, acute kidney injury; AKIN, Acute Kidney Injury Network; DS, duodenal switch; ICD-9, *International Classification of Diseases, Ninth Revision*; ICD-10, *International Classification of Diseases, Tenth Revision*; KDIGO, Kidney Disease: Improving Global Outcomes; LAGB, laparoscopic adjustable gastric banding; NA, not available; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy

of the included studies prespecified postoperative rhabdomyolysis as an end point, or systematically reported muscle injury biomarkers, such as serum myoglobin or creatine phosphokinase, despite the clinical relevance of these mechanisms.

Pooled prevalence of acute kidney injury The overall pooled prevalence of AKI following bariatric surgery was 2.5% (95% CI, 1.8–3.1), with substantial between-study heterogeneity (I^2 = 98.1%; **FIGURE 2**). To identify the potential sources of heterogeneity, we conducted a subgroup analysis. The subgroup analysis (**TABLE 3**) showed that the pooled AKI prevalence was higher in case-control studies (5.8%; 95% CI, 4.5–7.1) than in cohort studies (2.1%; 95% CI, 1.4–2.7). Studies with smaller sample sizes (<1000) reported significantly higher AKI rates (11%; 95% CI, 1.9–20.1) than those with larger cohorts (1.6%; 95% CI, 1–2.2). High-quality studies showed a pooled AKI rate of 3.3% (95% CI, 2.5–4.2), as compared with 0.6% (95% CI, −0.1 to 1.2) in moderate-quality studies. Single-center studies yielded slightly higher estimates than multicenter studies (0.029 [0.017–0.04]). AKI prevalence also varied by diagnostic criteria, with studies using the AKIN reporting higher rates (7.9%; 95% CI, 3.9–12) than those using other definitions (1%; 95% CI, 0.4–1.6). Furthermore, the leave-one-out sensitivity analysis (**TABLE 4**) demonstrated that the pooled prevalence remained robust when each study was sequentially excluded. Estimates ranged from 1.9% to 3%, with no single study disproportionately influencing the results.

Risk factors for acute kidney injury Risk factor analysis was conducted to identify the potential predictors of AKI in adults with obesity undergoing bariatric surgery (**TABLE 5**).

Male sex was significantly associated with an increased risk of AKI, with a pooled OR of 2.15 (95% CI, 1.54–3.01) across 4 studies with significant heterogeneity. Renal insufficiency, defined variably as baseline CKD or an elevated creatinine level, was the strongest individual predictor, with a pooled OR of 5.33 (95% CI, 1.4–20.26). This suggests that patients with impaired baseline renal function are at a markedly elevated risk of further

TABLE 2 Quality of the included studies based the Newcastle–Ottawa scale criteria[26] (points)

Study, year	Representativeness of the exposed cohort	Selection methods for the non-exposed cohorts	Ascertainment of exposure	Outcomes of interest not present at the study beginning	Study controlled for confounders	Study controlled for additional factors	Assessment of exposure	Follow-up long enough for outcomes to occur	Adequacy of follow-up of cohorts	Score	Overall quality[a]
Weingarten et al,[33] 2011	1	1	1	1	0	0	1	1	1	7	High
Koukoulaki et al,[29] 2013	1	1	1	1	0	0	1	1	1	7	High
Weingarten et al,[32] 2013	1	1	1	1	1	1	1	1	1	9	High
Morgan et al,[30] 2015	1	1	1	1	1	1	1	1	1	9	High
Abdullah et al,[21] 2016	1	1	1	1	1	1	1	0	1	8	High
Koppe et al,[22] 2018	1	1	0	1	1	1	0	1	1	7	High
Nor Hanipah et al,[31] 2018	1	0	1	1	0	0	1	1	0	5	Moderate
Valera et al,[24] 2023	1	1	0	1	1	1	0	1	1	7	High
Khalid et al,[28] 2022	1	1	0	1	1	1	0	1	1	7	High
Abi Mosleh et al,[19] 2025	1	1	1	1	1	1	1	1	1	9	High
Cooper et al,[27] 2024	1	1	0	1	0	0	0	1	1	5	Moderate

a Overall quality based on the total score (maximum 9 points)

kidney injury following surgery. However, this estimate was derived only from 2 studies and showed high heterogeneity (I^2 = 93.3%). Hypertension was also found to be a significant risk factor, with a pooled OR of 1.74 (95% CI, 1.37–2.21) and low heterogeneity (I^2 = 24.3%). Hyperlipidemia, examined in 2 studies, was significantly associated with AKI (OR, 1.53; 95% CI, 1.04–2.24). Higher body mass index (BMI) was also modestly but significantly associated with AKI risk (OR, 1.05 per unit increase; 95% CI, 1.02–1.09), indicating that more severe obesity confers a greater renal risk.

In contrast, several factors did not demonstrate significant associations with AKI. These included age (OR, 1; 95% CI, 0.91–1.11), DM (OR, 1.47; 95% CI, 0.95–2.28), cardiovascular disease (OR, 1.1; 95% CI, 0.78–1.68), respiratory disease (OR, 1.15; 95% CI, 0.74–1.79), and obstructive sleep (OR, 1.03; 95% CI, 0.83–1.28). For age and DM, the direction of effect was consistent with clinical expectations, but the difference was insignificant (I^2 >85%), suggesting inadequate power or confounding across the studies. Similarly, procedure duration (OR, 1.09; 95% CI, 0.96–1.24) did not reach significance, although longer surgeries could plausibly increase the AKI risk through prolonged hypotension or fluid shifts. These nonsignificant findings should be interpreted cautiously, as they may reflect underreporting or inadequate adjustment rather than true null effects.

In addition, no study provided sufficient data to analyze rhabdomyolysis-related variables or biochemical markers as potential predictors of AKI, which limits the ability to disentangle muscular versus intrinsic renal mechanisms underlying the observed events.

Publication bias The funnel plot assessing publication bias showed visual asymmetry (**FIGURE 3**), and the Egger test suggested evidence of small-study effects (P = 0.004). The trim-and-fill analysis added 3 hypothetical studies to correct asymmetry, but the adjusted pooled prevalence (0.017; 95% CI, 0.01–0.024) basically remained consistent, supporting the stability of the findings.

DISCUSSION In this systematic review and meta-analysis of 11 studies comprising over 240 000 adults with obesity, the pooled prevalence of postoperative AKI following bariatric surgery was estimated at 2.5%. More importantly, several significant and potentially modifiable risk factors for postoperative AKI were identified, including male sex, pre-existing renal insufficiency, hypertension, hyperlipidemia, and elevated BMI.

The prevalence of AKI identified in our study is consistent with the lower range of estimates reported in previous individual cohorts, which have varied widely from less than 1% to over 15%. This variability is partly attributable to differences in study design, sample size, and AKI definitions. Studies using strict KDIGO or AKIN biochemical criteria, rather than administrative code-based definitions, consistently reported higher

TABLE 3 Subgroup analysis of the prevalence of acute kidney injury after bariatric surgery

Outcomes		Number of studies	OR (95% CI)	I^2, %
Pooled results		11	0.025 (0.018–0.031)	98.1
Subgroup analyses based on the study design	Cohort study	10	0.021 (0.014–0.027)	98.1
	Case control study	1	0.058 (0.045–0.071)	NA
Subgroup analyses based on the sample size	<1000	3	0.11 (0.019–0.201)	93.5
	>1000	8	0.016 (0.01–0.022)	98.2
Subgroup analyses based on NOS quality scores	High quality	9	0.033 (0.025–0.042)	98.4
	Moderate quality	2	0.006 (–0.001–0.012)	90.7
Subgroup analyses based on the number of study centers	Single-center	7	0.029 (0.017–0.04)	95.6
	Multicenter	4	0.022 (0.012–0.033)	99.1
Subgroup analyses based on AKI criteria	AKIN	5	0.079 (0.039–0.12)	95.4
	Other	6	0.01 (0.004–0.016)	98.1

Abbreviations: NOS, Newcastle–Ottawa scale; OR, odds ratio; others, see **TABLE 1**

TABLE 4 Leave-one-out sensitivity analysis by sequential exclusion of individual studies

Study excluded	Estimate	95% CI
Weingarten et al,[33] 2011	0.0227332	0.01607082–0.02939557
Koukoulaki et al,[29] 2013	0.02442906	0.01779834–0.03105979
Weingarten et al,[32] 2013	0.02073672	0.01422301–0.02725043
Morgan et al,[30] 2015	0.01854915	0.01254139–0.02455692
Abdullah et al,[21] 2016	0.02387951	0.01703827–0.03072076
Koppe et al,[22] 2018	0.02595589	0.0189089–0.03300288
Nor Hanipah et al,[31] 2018	0.02773828	0.02025359–0.03522297
Valera et al,[24] 2023	0.03042848	0.02216252–0.03869433
Khalid et al,[28] 2022	0.0295613	0.02098872–0.03804355
Abi Mosleh et al,[19] 2025	0.0234546	0.0166774–0.03023181
Cooper et al,[27] 2024	0.02898698	0.02132772–0.03664624

TABLE 5 Risk factors for acute kidney injury after bariatric surgery

Risk factors	Number of trials	Pooled OR (95% CI)	I^2, %
Age, y	3	1.004 (0.908–1.11)	88.6
Body mass index	4	1.054 (1.024–1.085)	78.5
Male sex	4	2.152 (1.539–3.009)	84.5
Renal insufficiency	2	5.331 (1.403–20.26)	93.3
Hypertension	4	1.741 (1.371–2.212)	24.3
Diabetes mellitus	3	1.47 (0.95–2.275)	88.5
Procedure duration	3	1.093 (0.963–1.239)	94.4
Cardiovascular disease	2	1.145 (0.782–1.676)	69.1
Obstructive sleep apnea	2	1.029 (0.828–1.28)	15.3
Respiratory disease	2	1.151 (0.74–1.791)	85.5
Hyperlipidemia	2	1.526 (1.039–2.24)	88.6

Abbreviations: see **TABLE 3**

AKI prevalence, emphasizing the importance of standardized diagnostic criteria. Our findings also show that smaller and single-center studies tend to report higher rates of AKI, likely due to more granular clinical data capture, closer postoperative surveillance, or selection of higher-risk surgical candidates. These observations underscore the challenge of synthesizing epidemiologic data from a heterogeneous and rapidly evolving field, and they call for a consensus in defining and reporting renal outcomes in bariatric surgery.

A comparison with other surgical populations also yields important insights. While the 2.5% prevalence of AKI in bariatric surgery is lower than in cardiac or general abdominal surgery,[34,35] the elective nature of bariatric procedures and the relatively younger, comorbid population make even a modest AKI risk clinically meaningful. Prior studies have demonstrated that even transient AKI episodes are independently associated with prolonged hospitalization, higher readmission rates, and increased long-term risk of CKD and cardiovascular events.[36,37] In this context, the need for preventive strategies is amplified by the fact that many bariatric patients already carry a substantial burden of subclinical renal impairment and vascular dysfunction at baseline.

The identification of specific risk factors provides further clarity. Male sex was associated with more than a 2-fold risk of AKI. This may reflect sex differences in renal physiology, including lower baseline estimated glomerular filtration rate (eGFR) reserve in men, higher muscle mass (which may mask early creatinine-based changes), and differences in intraoperative hemodynamic responses. Renal insufficiency—arguably the most intuitive risk factor—was associated with a 5-fold AKI risk, reinforcing previous findings that pre--existing renal dysfunction compromises adaptive capacity during surgical stress. Importantly, even mild renal impairment may be sufficient to confer increased postoperative vulnerability. Hypertension and hyperlipidemia were also consistently associated with AKI.[38] These conditions, often present in patients with obesity as part of a metabolic syndrome, may synergistically impair endothelial function, promote renal microvascular disease, and reduce renal perfusion reserve during periods of intraoperative hypotension or volume shifts.[39,40]

Higher BMI, while previously considered only a technical surgical challenge, emerged as a biological predictor of AKI, likely reflecting the systemic inflammatory and hemodynamic burden of morbid obesity.[41] Interestingly, DM and older age, commonly implicated in surgical AKI risk models, were not significant in the pooled analysis. This may be due to inconsistent reporting, inadequate power, or insufficient control of confounders, such as glycemic control or DM duration.

Beyond traditional cardiovascular and renal risk factors, emerging clinical and mechanistic evidence indicates that rhabdomyolysis is a predominant and often underrecognized pathway

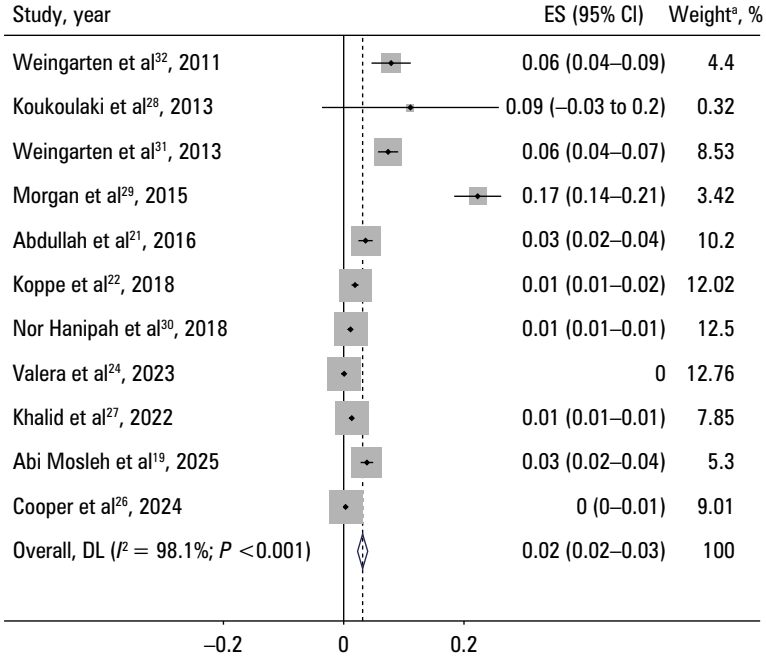

FIGURE 2 Forest plot of the prevalence of acute kidney injury in adults with obesity undergoing bariatric surgery

a Weights are derived from the random effects model.

Abbreviations: DL, DerSimonian–Laird; ES, effect size

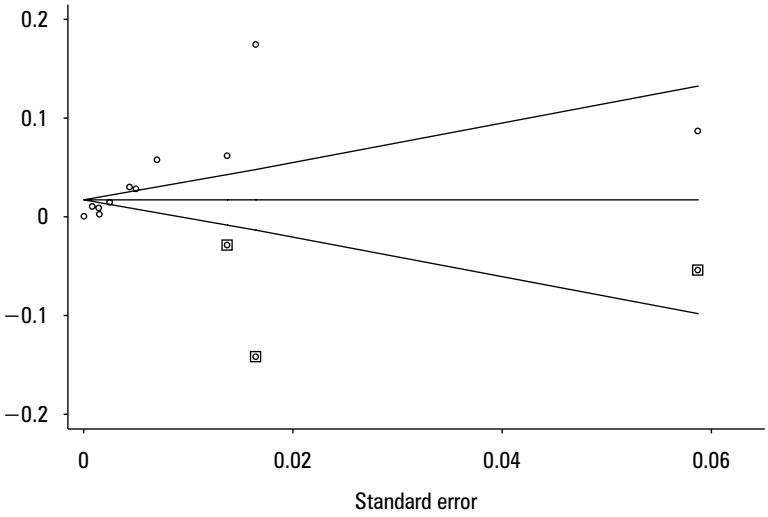

FIGURE 3 The "trim-and-fill" funnel plot with pseudo 95% CIs for assessing publication bias. The plot illustrates the relationship between the effect size and its standard error. Open circles represent the observed studies, while framed circles indicate the studies imputed using the trim-and-fill method to adjust for potential publication bias.

leading to AKI after bariatric surgery.[42,43] Patients with severe obesity undergoing lengthy laparoscopic procedures are particularly vulnerable to skeletal muscle compression due to high body mass in fixed positions, restricted repositioning, and pneumoperitoneum-related increases in intra-abdominal pressure.[44] Prolonged pressure on dependent muscle groups can precipitate ischemic muscle injury, myonecrosis, and massive myoglobin release.[45] Filtered myoglobin may induce tubular obstruction, oxidative tubular damage, and intrarenal vasoconstriction, thereby triggering pigment-induced AKI. In anesthetic and bariatric practice, a substantial proportion of postoperative AKI episodes are thought to be secondary to rhabdomyolysis rather than primary intrinsic renal disease or comorbidity-driven nephropathy.[41] Importantly, the depth and duration of neuromuscular blockade, adequacy of intraoperative hemodynamic support, vasopressor use, and temperature management all influence the risk of muscle injury and, consequently, AKI. Recent work in bariatric cohorts has specifically highlighted the contribution of prolonged pressure, suboptimal positioning, and impaired muscle perfusion to postoperative rhabdomyolysis and kidney injury, emphasizing the importance of preventive strategies, such as vacuum mattresses and pressure-relief positioning systems.[44,46]

These mechanistic considerations have direct implications for perioperative management. Preoperative risk stratification should move beyond a simple inventory of comorbidities, and incorporate both renal and muscular risk factors, including baseline kidney function, blood pressure control, BMI, anticipated operative duration, and the need for complex or revisional procedures.[47] Intraoperatively, optimizing patient positioning, using vacuum mattresses or other pressure-redistribution devices, padding vulnerable areas, and periodically reassessing pressure points during long operations may mitigate muscle compression and improve tissue perfusion. Anesthetic management should aim to maintain stable hemodynamics, avoid prolonged periods of hypotension or low cardiac output, ensure adequate intravascular volume, and carefully titrate neuromuscular blockade. In high-risk patients, early postoperative monitoring for muscle injury and AKI—including serial creatinine, urine output and, where possible, creatine phosphokinase, or myoglobin—may facilitate timely intervention and prevent progression to more severe injuries.[44,46] Integrating these measures into standardized bariatric pathways could represent an important opportunity to reduce the burden of postoperative AKI.

While the observed associations are biologically plausible, the findings should be interpreted in the context of important limitations. Firstly, the heterogeneity across the studies was high, even within subgroups, and may reflect differences in surgical technique, perioperative protocols, fluid management strategies, and nephrotoxic exposures. Secondly, most studies were retrospective in design, limiting the control for confounding variables, and none provided granular data on intraoperative or postoperative fluid balance—a key determinant of AKI. Thirdly, while the meta-analysis evaluated AKI prevalence, long-term renal outcomes, such as progression to CKD, dialysis, or decline in eGFR, were not reported, precluding an assessment of clinical sequelae. Finally, only 2 studies specifically evaluated severe or

stage 2–3 AKI, limiting insights into the distribution of AKI severity and its implications.

CONCLUSIONS In this systematic review and meta-analysis, the pooled prevalence of AKI following bariatric surgery in adults with obesity was 2.5%, with considerable variability across the study populations and methodologies. Several clinical characteristics—including male sex, pre--existing renal insufficiency, hypertension, hyperlipidemia, and higher BMI—were independently associated with an increased AKI risk. These findings underscore the importance of individualized perioperative risk stratification, and highlight opportunities for targeted preventive strategies in high-risk patients undergoing bariatric procedures.

ARTICLE INFORMATION

ACKNOWLEDGMENTS None.

FUNDING The study is sponsored by the Gansu Provincial Natural Science Foundation (22JR5RA988; to JB).

CONTRIBUTION STATEMENT WXC, PZK: funding acquisition, methodology, project administration, and writing of the original draft. PZK, PJB: conceptualization, data curation, formal analysis, and writing of the original draft. WXC, PZK, PJB: data curation, methodology, supervision, writing, review, and editing. All authors read and approved the final version of the manuscript.

CONFLICT OF INTEREST None declared.

AI STATEMENT Artificial intelligence was not used in the preparation of this manuscript.

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HOW TO CITE Wang X, Peng Z, Pan J. Prevalence and risk factors of acute kidney injury in adults with obesity undergoing bariatric surgery: a systematic review and meta-analysis. Wideochir Inne Tech Maloinwazyjne. 2026; 21: 13-21. doi:10.20452/wiitm.2026.18007

JOURNAL INFORMATION

Videosurgery and Other Miniinvasive Techniques is an official journal of the Videosurgery Foundation.

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