

Association of perioperative acetate-buffered balanced crystalloid solution with postoperative myocardial injury after hip replacement: A retrospective cohort study

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Abstract

Background

Postoperative myocardial injury after non-cardiac surgery is associated with adverse short- and long-term outcomes. Patients undergoing hip replacement may be particularly vulnerable because of older age, comorbidity burden, and perioperative physiologic stress. We evaluated whether perioperative administration of sodium-potassium-magnesium-calcium-glucose injection (SPMCG), an acetate-buffered balanced crystalloid solution, was associated with a lower risk of postoperative myocardial injury.

Methods

This single-center retrospective cohort study included adults who underwent hip replacement between January 1, 2014 and November 30, 2023 with postoperative troponin I measurements. Exposure was perioperative SPMCG administration or not. The primary outcome was postoperative myocardial injury within 3 days, defined as peak cardiac troponin I > 0.04 ng/mL. Secondary outcomes included postoperative myocardial injury within 7 days, postoperative atrial fibrillation, heart failure, ICU admission, in-hospital mortality, and postoperative length of stay. Propensity score matching at a 1:3 ratio and multivariable regression were used to address confounding.

Results

A total of 865 patients were included, of whom 40 (4.6%) developed postoperative myocardial injury within 3 days. In the crude cohort, 764 patients received SPMCG, with 24 (3.1%) developing myocardial injury, compared to 16 among 101 patients who did not receive SPMCG (15.8%). Perioperative SPMCG use was associated with a lower risk of postoperative myocardial injury within 3 days (adjusted odds ratio [OR], 0.33; 95% confidence interval [CI], 0.14–0.78; $P = 0.011$), as well as a lower risk of postoperative myocardial injury within 7 days and ICU admission. Similar associations were identified in the matched cohort. No significant differences were observed in other outcomes.

Conclusions

Among patients undergoing hip replacement, perioperative SPMCG administration was associated with a lower risk of postoperative myocardial injury. This finding requires further validation in prospective multicenter studies.

Background

Postoperative myocardial injury is one of the most frequent cardiovascular complications after non-cardiac surgery, with reported incidences ranging from 6.1% to 36.1% depending on the population studied and the troponin assay used [1–4]. Even when clinically silent, postoperative troponin elevation is consistently associated with increased short- and long-term mortality, as well as adverse cardiovascular outcomes [3–6]. Previous studies have identified various risk factors, in addition to comorbidities, specific surgical procedures also increase the incidence of postoperative myocardial injury [7, 8]. Notably, hip replacement, including total hip arthroplasty and hemiarthroplasty, exhibits a particularly high incidence of postoperative myocardial injury due to a combination of factors such as advanced age, significant comorbidity burden, extensive surgical trauma, and perioperative pain. A

study including replacement surgery found that 42% of patients had elevated postoperative troponin, with a 62% incidence of myocardial injury after hip replacement for femoral neck fracture, substantially higher than other surgical types [9]. Although interventions such as statins [10] and dabigatran [11] have been shown to reduce cardiovascular risk, there is limited data available on both safety and efficacy in the prevention of postoperative myocardial injury.

Balanced crystalloid solutions, characterized by the substitution of chloride ions with organic anions and supplementation of essential electrolytes (e.g., Mg^{2+} , K^+ , Ca^{2+}), demonstrate superior efficacy over normal saline in large-volume resuscitation by mitigating risks of hyperchloremia, metabolic acidosis, and acute kidney injury [12, 13]. Evidence suggests that acetate-containing solutions may reduce postoperative arrhythmias in cardiac surgery [14], indicating potential cardioprotective properties. Sodium-Potassium-Magnesium-Calcium-Glucose Injection (SPMCG), a generic formulation, contains 2.052 g/L sodium acetate alongside physiological electrolytes [15]. Notably, the electrolyte composition of SPMCG adheres to modern principles of balanced fluid therapy, potentially offering cardioprotective advantages.

Nevertheless, whether perioperative SPMCG confers cardioprotection against postoperative myocardial injury after hip replacement remains unclear. We therefore conducted this retrospective cohort study to examine its association with postoperative myocardial injury in patients undergoing hip replacement.

Methods

Study design

This single-center retrospective cohort study was conducted at Shenzhen Second People's Hospital, China. The study was approved by the Clinical Research Ethics Committee of Shenzhen Second People's Hospital (approval number: 2024-267-02PJ; approved on August 21, 2024), and the requirement for written informed consent was waived because of the retrospective design and use of de-identified data. The study was registered at the Chinese Clinical Trial Registry (ChiCTR2400091471; October 29, 2024) before formal data analysis and manuscript preparation. This study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Participants

The source cohort included all adult patients aged ≥ 18 years who underwent hip replacement, including total hip arthroplasty and hemiarthroplasty, between January 1, 2014 and November 30, 2023, in the institutional Research Center System Database. Patients were excluded if they did not undergo the planned surgery or had incomplete key medical records.

Because postoperative cardiac troponin I (cTnI) surveillance was not universally performed in routine clinical practice, the primary analytic cohort was restricted to patients with at least one available cTnI measurement during postoperative days 1–3 (POD1–3). Patients without cTnI measurements during POD1–3 were excluded from the primary analysis rather than being classified as free of myocardial injury. To assess the potential influence of incomplete postoperative troponin surveillance, we performed sensitivity analyses in the source cohort by classifying patients without postoperative cTnI measurements as not having troponin-defined myocardial injury.

Data collection

Data were extracted from the institutional Research Center System Database. Demographics included age, sex, body mass index (BMI), American Society of Anesthesiologists (ASA) physical status, and surgery type (emergency or elective). Comorbidities included hypertension, atrial fibrillation, heart failure, coronary artery disease, valvular disease, renal failure, diabetes, stroke, chronic obstructive pulmonary disease (COPD), peripheral vascular disease, and active cancer. Baseline medications included angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs), beta-blockers, calcium channel blockers, diuretics, and statins. Preoperative laboratory tests closest to surgery included cTnI, myoglobin, N-terminal pro-B-type natriuretic peptide (NT-proBNP), complete blood count, hypersensitive C-reactive protein (hsCRP), procalcitonin, creatinine, electrolytes, liver enzymes, and glucose. Postoperative data included the peak cTnI values within 3 and 7 days, peak myoglobin and NT-proBNP within 7 days, the first postoperative electrolyte and glucose measurements within 3 days, postoperative atrial fibrillation, heart failure, intensive care unit (ICU) admission, in-hospital death, and postoperative length of stay. The use of SPMCG during the perioperative period was also documented. Complications were diagnosed clinically and adjudicated via cardiology consultation and review of source records.

Exposure and outcomes

The exposure of interest was perioperative administration of SPMCG. The perioperative exposure window was defined a priori as the interval from 1 day before surgery through the day of surgery. In both groups, fluid therapy followed routine clinical practice and consisted of mixed fluids rather than a protocolized single-solution strategy. Patients in the control group received fluids such as lactated Ringer's solution, 0.9% saline, hydroxyethyl starch, compound sodium chloride solutions, and dextrose-containing fluids. Patients in the SPMCG group could receive the same concomitant fluids, with the addition of SPMCG. Fluid selection was at the discretion of the treating clinicians and followed the hospital's standard perioperative management procedures.

The primary outcome was the incidence of postoperative myocardial injury, defined as a peak cTnI concentration > 0.04 ng/mL during postoperative days 1–3 [16, 17]. We did not distinguish whether the increased cTnI was caused by an ischemic etiology, as elevated troponin often predicts a poor clinical outcome. Since postoperative troponin elevation is clinically relevant regardless of mechanism, the primary outcome should be interpreted as troponin-defined postoperative myocardial injury rather than strictly adjudicated myocardial injury after non-cardiac surgery (MINS).

Secondary outcomes included postoperative myocardial injury within 7 days, postoperative atrial fibrillation, postoperative heart failure, ICU admission, in-hospital mortality, and postoperative length of stay. Exploratory outcomes included cTnI concentrations within 3 and 7 days postoperatively, changes from preoperative cTnI to postoperative peak values, and postoperative myoglobin and NT-proBNP concentrations.

Statistical analysis

Continuous variables are presented as mean (standard deviation) or median (interquartile range) and were compared with the t-test or Mann-Whitney U test as appropriate. Categorical variables are presented as numbers (%) and were compared with the chi-squared test or Fisher's exact test. The sample size was determined by the number of eligible patients available in the study period.

To mitigate confounding, propensity scores matching was performed using demographic, comorbidities, baseline medications, and preoperative laboratory variables. The primary analysis was performed in patients with available cTnI measurements during POD1–3. Multivariable logistic regression was used to estimate the association between perioperative SPMCG use and postoperative troponin-defined myocardial injury within 3 days. Covariates were selected a priori based on clinical relevance and potential confounding and included age, sex, BMI, ASA physical status, emergency surgery, major cardiovascular comorbidities, renal failure, diabetes, preoperative cTnI, hemoglobin, platelet count, and hsCRP. The same modeling strategy was used for binary secondary outcomes.

To further address confounding by indication, propensity-score methods were used as complementary analyses. The propensity score was estimated using demographic characteristics, comorbidities, baseline medications and preoperative laboratory variables. A 1:3 nearest-neighbor greedy algorithm with a caliper of 0.01 was used. Balance was assessed with standardized difference, and values ≥ 0.207 (1.96) indicating imbalance [18]. We converted missing covariate values (e.g., preoperative cTnI, hemoglobin, platelet count, hsCRP) into binary categorical variables and included them in matching to reduce bias.

Sensitivity analyses were performed to test the robustness of the primary association. First, analyses were repeated after excluding patients with missing covariate values. Second, patients with preoperative cTnI > 0.04 ng/mL were excluded to reduce the influence of pre-existing myocardial injury. Third, the analysis was repeated in the source cohort by classifying patients without postoperative cTnI measurements as not having troponin-defined myocardial injury, to explore the potential impact of incomplete troponin surveillance. Fourth, analyses were restricted to patients who received SPMCG specifically during surgery to assess the influence of timing on postoperative myocardial injury. Finally, the E-value was calculated to estimate the potential impact of unmeasured confounders. All sensitivity analyses were adjusted for the matching covariates.

All analyses were performed with R software (version 3.4.3, R Foundation for Statistical Computing, Vienna, Austria) and Empower Stats (X&Y Solutions Inc., Boston, MA, USA). All tests were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Participant characteristics

Among 3,543 eligible patients, 865 met the inclusion criteria. The main reason for exclusion was the absence of cTnI measurements during postoperative days 1-3. Baseline characteristics of patients with missing values are shown in Table S1. Overall, 40 of 865 patients (4.6%) developed postoperative myocardial injury within 3 days. Of the included patients, 764 received SPMCG and 101 did not. After propensity score matching, 216 patients in the SPMCG group were matched to 72 controls (Figure 1).

Before matching, patients who received SPMCG were younger, had lower ASA physical status, underwent fewer emergency procedures, and had fewer comorbidities and baseline cardiovascular medications, whereas control patients had a lower BMI. After matching, the groups were relatively balanced, with the standardized difference of all variables less than 0.207 (Table 1). Preoperative laboratory variables were broadly similar after matching (Table S3).

Primary outcome

In the crude cohort, postoperative myocardial injury within 3 days occurred in 24 of 764 patients (3.1%) in the SPMCG group and 16 of 101 patients (15.8%) in the control group. In the matched cohort, the corresponding incidences were 8 of 216 (3.7%) and 10 of 72 (13.9%), respectively. After multivariable adjustment, perioperative SPMCG use was associated with a lower risk of postoperative myocardial injury within 3 days (adjusted odds ratio [OR], 0.33; 95% confidence interval [CI], 0.14-0.78; $P=0.011$), and the association remained directionally consistent in the matched cohort (Table 2).

Secondary outcomes

In the crude cohort, postoperative myocardial injury within 7 days occurred in 30 of 764 patients (3.9%) in the SPMCG group and 17 of 101 patients (16.8%) in the control group. SPMCG use was associated with a lower risk of postoperative myocardial injury within 7 days (adjusted OR, 0.39; 95% CI, 0.17-0.88; $P=0.023$) and ICU admission (adjusted OR, 0.26; 95% CI, 0.11-0.60; $P=0.002$). These associations remained directionally similar after matching. No statistically significant associations were observed for postoperative atrial fibrillation, heart failure, in-hospital mortality, or postoperative length of stay (Table 2).

Exploratory analyses

The peak cTnI values at postoperative day 3 and day 7, changes from preoperative cTnI to postoperative peak values, postoperative myoglobin, and postoperative NT-proBNP concentrations did not differ significantly between groups after matching (Table 3).

Postoperative electrolytes were generally comparable between groups after matching, whereas preoperative glucose lower in the SPMCG group (Tables S4 and S5). These exploratory findings should be interpreted cautiously.

Subgroup analyses

All subgroup analyses showed a decreased incidence of postoperative myocardial injury in patients receiving SPMCG, except for stroke. The OR for almost all subgroups was less than 1, indicating robustness of the main analysis (Figure 2 and Table S6).

For patients with preoperative cTnI >0.04 ng/mL, 50.0% (4/8) who received SPMCG had postoperative myocardial injury, compared with 85.7% (6/7) who did not receive SPMCG ($P=0.282$). For patients with preoperative cTnI ≤ 0.04 ng/mL, 2.7% (15/554) in the SPMCG group and 11.1% (8/72) in the control group had postoperative myocardial injury ($P<0.001$) (Figure S1).

Sensitivity analyses

The direction of the association between perioperative SPMCG use and postoperative myocardial injury was consistent across all sensitivity analyses (Table 4).

Multivariable regression, including not considered missing covariate values (OR, 0.14; 95%CI, 0.02-0.93; $P=0.042$), excluded patients with preoperative myocardial injury (OR, 0.32; 95%CI, 0.08-1.20; $P=0.091$), included all patients with unmeasured cTnI (OR, 0.26; 95%CI, 0.10-0.67; $P=0.006$) and those treated with SPMCG intraoperatively (OR, 0.23; 95%CI, 0.08-0.63; $P=0.004$), yielded a similar effect estimate to the primary analysis. The E-values for the relative risk point estimate and the lower bound of the 95% CI were 5.51 and 3.64, respectively, indicating that moderately strong unmeasured confounding would be required to fully explain the observed association.

Discussion

In this retrospective cohort of adults undergoing hip replacement, perioperative administration of acetate-buffered balanced crystalloid solution was associated with a lower risk of postoperative myocardial injury within 3 days. The association remained directionally consistent across subgroup and sensitivity analyses and was accompanied by a lower risk of postoperative myocardial injury within 7 days and ICU admission.

The incidence of postoperative myocardial injury in our cohort (4.6%) was lower than that reported in several previous orthopedic studies [9, 19]. Variations may reflect differences in population heterogeneity, troponin assay sensitivity, and diagnostic criteria. A study included elderly patients with hip fractures and multiple comorbidities, selecting a more sensitive cutoff of postoperative high-sensitivity troponin I peak ≥ 5 ng/L [19]. Another study focusing exclusively on arthroplasty patients reported a 42.5% myocardial injury incidence, but the population consisted of patients with various cardiac risk factors [9]. Our more diverse cohort includes patients aged ≥ 18 years with conditions such as hip fractures, tumors, osteoarthritis, dysplasia, and femoral head necrosis, which likely reduced overall myocardial injury incidence.

Several biologically plausible mechanisms may explain the observed association. Balanced crystalloids replace chloride with organic anions and incorporate essential electrolytes such as Mg^{2+} , K^+ , and Ca^{2+} , which have been well-documented for their clinical benefits. Compared to normal saline, the use of compound electrolyte solutions in major abdominal surgery has been shown to significantly reduce the incidence of dialysis-requiring acute kidney injury [20]. Recent perioperative fluid research has further emphasized that, in routine mixed-fluid practice, the actual electrolyte burden of administered fluids, such as weighted average chloride concentration, may provide more clinically relevant information than a simple binary classification of fluid type [21]. Based on their composition and buffering systems, balanced crystalloids are further categorized into types such as lactate- and acetate-buffered solutions. Lactate is primarily metabolized in the liver, while acetate undergoes peripheral metabolism, primarily in skeletal muscle, kidneys, and myocardium, where it feeds directly into the tricarboxylic acid cycle as acetyl-CoA. This metabolic flexibility theoretically positions acetate as an advantageous buffer. Although research comparing acetate- and lactate-buffered crystalloids has yielded mixed outcomes, acetate-buffered solutions have demonstrated reduced postoperative arrhythmias and transient hyperlactatemia [14, 22]. However, more robust evidence is needed to evaluate the perioperative benefits of acetate-buffered crystalloids.

SPMCG also contains magnesium and a relatively low calcium concentration, both of which may theoretically attenuate calcium overload and electrical instability during ischemia-reperfusion [23, 24]. In this context, compared with lactated Ringer's solution, the lower calcium concentration of SPMCG may be advantageous under conditions of myocardial stress. However, this interpretation remains indirect, and the underlying mechanisms still require further clarification. In addition to its lower calcium content, the presence of Mg^{2+} may represent another component of the protective profile of SPMCG. Observational studies have shown inverse associations between serum magnesium levels and cardiovascular events, vascular stiffness, and endothelial dysfunction [25, 26]. Randomized controlled trials in cardiac surgery have further reported that magnesium sulfate reduced cTnI and creatine kinase-MB levels, as well as the incidence of myocardial infarction and arrhythmia [27]. Since Mg^{2+} levels are often reduced in patients undergoing hip arthroplasty [28], intraoperative infusion of SPMCG may also help maintain perioperative magnesium balance and reduce the risk of hypomagnesemia. In fact, except for lactate Ringer's solution, the addition of Mg^{2+} to most balanced crystalloids highlights the growing recognition of its importance. Nevertheless, the specific contribution of Mg^{2+} to the overall effects of SPMCG cannot be quantified in the present study, and this potential mechanism should therefore be interpreted as hypothesis-generating rather than conclusive.

The low-concentration glucose component of SPMCG may be another potential factor. Perioperative fasting, surgical stress, and prolonged surgery may contribute to metabolic instability. Previous studies have shown that glucose-insulin administration can shift ischemic myocardium from fatty acid toward glucose metabolism, thereby reducing oxygen consumption and prolonging cardiomyocyte survival [29]. Although SPMCG does not contain insulin and therefore cannot be considered equivalent to glucose-insulin-potassium therapy, low-concentration glucose infusion may still help maintain perioperative glucose availability, attenuate catabolism, and partially support metabolic homeostasis [30, 31]. In the present study, preoperative hypoglycemia was observed in a proportion of patients undergoing hip replacement, whereas the use of SPMCG was not associated with marked perioperative hyperglycemia, suggesting that this formulation may help maintain glycemic balance with a limited glucose load [32]. However, because patients received mixed fluids, these mechanistic considerations should be interpreted as hypothesis-generating rather than confirmatory.

The study has several limitations. First, despite adjustment, our observational study may have been affected by unmeasured confounders. We used the E-value to estimate the bias and found that unmeasured confounders were unlikely to explain the entirety of the treatment effect. Second, most patients were excluded due to missing POD3 cTnI measurements, resulting in a possible selection and detection bias. A sensitivity analysis classified patients who did not receive cTnI measurement as normal and yielded similar results to the primary analysis. Third, a mixture of fluids may be received in the SPMCG group, which weakens the relationship between SPMCG and postoperative myocardial injury within 3 days. If the infusion of a single fluid had been strictly implemented, then the result seen for SPMCG users might have been even more pronounced. Finally, we included all eligible patients and therefore did not estimate the sample size a priori. However, based on a myocardial injury incidence of 3.1% in the SPMCG group and 15.8% in the control group, our sample size provided 93% power to detect a relative difference at a significance level of 0.05.

Conclusions

Perioperative SPMCG administration was associated with a lower risk of postoperative myocardial injury after hip replacement surgery. Given the observational design and potential for residual confounding, these findings should be interpreted cautiously and confirmed in prospective, multicenter studies with standardized troponin surveillance and protocolized fluid exposure.

Abbreviations

ASA
American Society of Anesthesiologists
BMI
Body mass index
CI
Confidence interval
cTnI
Cardiac troponin I
ICU
Intensive care unit
IQR
Interquartile range

MINS

Myocardial injury after non-cardiac surgery

NT-proBNP

N-terminal pro-B-type natriuretic peptide

OR

Odds ratio

POD

Postoperative day

SD

Standard deviation

SPMCG

Sodium-Potassium-Magnesium-Calcium-Glucose Injection

STROBE

Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate

The study was approved by the Clinical Research Ethics Committee of Shenzhen Second People's Hospital (2024-267-02PJ). The requirement for written informed consent was waived because of the retrospective design and use of de-identified data.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request. corresponding author: liuzhiheng@szseyy.com.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

NL and NS conceived and designed the study. YZ and JC collected the data. ZZ and XY analyzed and interpreted the data. XH and WY contributed to the methodology. QX and BY drafted the manuscript. YL and ZL acquired funding, supervised the project, and critically revised the manuscript. All authors read and approved the final manuscript.

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Tables

Table 1 Baseline characteristics of patients undergoing hip arthroplasty before and after propensity score matching.

Variables	Before propensity score matching			After propensity score matching		
	SPMCG (N=764)	No SPMCG (N=101)	Standardized Difference	SPMCG (N=216)	No SPMCG (N=72)	Standardized Difference
Demographics						
Age (yr), mean (SD)	66.3 (15.0)	73.5 (14.4)	0.487	70.0 (14.1)	70.0 (14.7)	0.005
Gender			0.034			0.019
Female	464 (60.7)	63 (62.4)		124 (57.4)	42 (58.3)	
Male	300 (39.3)	38 (37.6)		92 (42.6)	30 (41.7)	
BMI (kg m ⁻²), mean (SD)	23.1 (3.4)	21.9 (3.2)	0.364	22.5 (3.4)	22.7 (2.9)	0.032
ASA physical status			0.454			0.019
1-2	449 (58.8)	37 (36.6)		101 (46.8)	33 (45.8)	
3-5	315 (41.2)	64 (63.4)		115 (53.2)	39 (54.2)	
Surgery type			0.116			0.026
Emergency surgery	104 (13.6)	18 (17.8)		32 (14.8)	10 (13.9)	
Elective surgery	660 (86.4)	83 (82.2)		184 (85.2)	62 (86.1)	
Preoperative comorbidities						
Hypertension	278 (36.4)	51 (50.5)	0.288	101 (46.8)	30 (41.7)	0.103
Atrial fibrillation	31 (4.1)	6 (5.9)	0.086	7 (3.2)	3 (4.2)	0.049
Congestive heart failure	7 (0.9)	3 (3.0)	0.149	3 (1.4)	1 (1.4)	0.000
Coronary artery disease	75 (9.8)	11 (10.9)	0.035	19 (8.8)	6 (8.3)	0.017
Valvular disease	42 (5.5)	9 (8.9)	0.132	13 (6.0)	4 (5.6)	0.020
Renal failure	20 (2.6)	7 (6.9)	0.203	12 (5.6)	3 (4.2)	0.065
Diabetes	119 (15.6)	30 (29.7)	0.342	52 (24.1)	14 (19.4)	0.112
Stroke	87 (11.4)	15 (14.9)	0.103	34 (15.7)	11 (15.3)	0.013
COPD	7 (0.9)	0 (0.0)	0.136	-	-	

Peripheral vascular disease	75 (9.8)	12 (11.9)	0.066	25 (11.6)	8 (11.1)	0.015
Active cancer	51 (6.7)	2 (2.0)	0.232	5 (2.3)	2 (2.8)	0.029
Medications at baseline						
ACEI/ARB	78 (10.2)	11 (10.9)	0.022	23 (10.6)	8 (11.1)	0.015
Beta blocker	35 (4.6)	10 (9.9)	0.206	12 (5.6)	2 (2.8)	0.139
Calcium channel blocker	116 (15.2)	21 (20.8)	0.146	41 (19.0)	14 (19.4)	0.012
Diuretic	16 (2.1)	6 (5.9)	0.197	4 (1.9)	0 (0.0)	0.194
Statin	44 (5.8)	9 (8.9)	0.121	18 (8.3)	6 (8.3)	0.000
Preoperative laboratory data						
cTnI (ng/mL)			0.211			0.066
Low	241 (31.5)	26 (25.7)		54 (25.0)	20 (27.8)	
High	321 (42.0)	53 (52.5)		120 (55.6)	38 (52.8)	
Missing	202 (26.4)	22 (21.8)		42 (19.4)	14 (19.4)	
Haemoglobin (g/L)			0.061			0.162
Low	355 (46.5)	47 (46.5)		98 (45.4)	35 (48.6)	
High	360 (47.1)	46 (45.5)		105 (48.6)	35 (48.6)	
Missing	49 (6.4)	8 (7.9)		13 (6.0)	2 (2.8)	
Platelet count (cells 10 ⁹ /L)			0.066			0.181
Low	351 (45.9)	48 (47.5)		105 (48.6)	35 (48.6)	
High	362 (47.4)	45 (44.6)		97 (44.9)	35 (48.6)	
Missing	51 (6.7)	8 (7.9)		14 (6.5)	2 (2.8)	
hsCRP (mg/L)			0.422			0.097
Low	338 (44.2)	25 (24.8)		75 (34.7)	23 (31.9)	
High	307 (40.2)	57 (56.4)		112 (51.9)	37 (51.4)	
Missing	119 (15.6)	19 (18.8)		29 (13.4)	12 (16.7)	

Data are presented as n (%) unless noted otherwise. SD, standard deviation; SPMCG, Sodium-Potassium-Magnesium-Calcium-Glucose Injection; BMI, body mass index; ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor

blocker; cTnI, cardiac troponin I; hsCRP, hypersensitive C-reactive protein. The low and high cTnI, hemoglobin, platelet count, and hsCRP categories were dichotomized at the median value.

Table 2 Associations between SPMCG and primary and secondary outcomes before and after propensity score matching.

Variables	Before propensity score matching				After propensity score matching			
	SPMCG (N=764)	No SPMCG (N=101)	Adjusted OR/ β (95%CI) ^a	P- value	SPMCG (N=216)	No SPMCG (N=72)	Adjusted OR/ β (95%CI) ^a	P- value
Primary outcome								
POD3 postoperative myocardial injury	24 (3.1)	16 (15.8)	0.33 (0.14, 0.78)	0.011	8 (3.7)	10 (13.9)	0.04 (0.01, 0.24)	<0.001
Secondary outcomes								
POD7 postoperative myocardial injury	30 (3.9)	17 (16.8)	0.39 (0.17, 0.88)	0.023	10 (4.6)	10 (13.9)	0.08 (0.02, 0.36)	0.001
Postoperative atrial fibrillation	9 (1.2)	0 (0.0)	-	-	2 (0.9)	0 (0.0)	-	-
Postoperative heart failure	6 (0.8)	0 (0.0)	-	-	1 (0.5)	0 (0.0)	-	-
Admission to ICU	28 (3.7)	15 (14.9)	0.26 (0.11, 0.60)	0.002	11 (5.1)	10 (13.9)	0.16 (0.04, 0.60)	0.006
Death in hospital	5 (0.7)	0 (0.0)	-	-	2 (0.9)	0 (0.0)	-	-
Length of postoperative stay (days), median (IQR)	8.0 (6.0-11.0)	9.0 (6.0-13.0)	-0.28 (-1.47, 0.92)	0.650	8.0 (6.0-11.0)	8.0 (6.0-11.3)	-0.16 (-2.59, 0.48)	0.178

Data are presented as n (%) unless noted otherwise. IQR, interquartile range; OR, odds ratio; CI, confidence interval; SPMCG, Sodium-Potassium-Magnesium-Calcium-Glucose Injection; POD, postoperative day; ICU, intensive care unit.

^a Adjustment was performed for age, gender, BMI, surgery type, ASA physical status, hypertension, atrial fibrillation, congestive heart failure, coronary artery disease, valvular disease, renal failure, diabetes, stroke, COPD, peripheral vascular disease, active cancer, ACEI/ARB, beta blocker, calcium channel blocker, diuretic, statin, cTnI, hemoglobin, platelet count, hsCRP.

Table 3 Associations between SPMCG and exploratory outcomes before and after propensity score matching.

Variables	Before propensity score matching			After propensity score matching		
	SPMCG (N=764)	No SPMCG (N=101)	P- value	SPMCG (N=216)	No SPMCG (N=72)	P- value
Exploratory outcomes						
POD3 cTnI (ng/mL)×10 ²	1.0 (0.3-1.0)	1.0 (0.3-1.4)	0.023	1.0 (0.5-1.0)	1.0 (0.3-1.0)	0.533
POD7 cTnI (ng/mL)×10 ²	1.0 (0.4-1.0)	1.0 (0.6-1.5)	0.014	1.0 (0.6-1.0)	1.0 (0.4-1.0)	0.691
Difference between POD3 and preoperative cTnI (ng/mL)×10 ²	0.0 (0.0-0.1)	0.0 (0.0-0.4)	0.103	0.0 (0.0-0.1)	0.0 (-0.1-0.4)	0.352
Difference between POD7 and preoperative cTnI (ng/mL)×10 ²	0.0 (0.0-0.1)	0.0 (0.0-0.4)	0.086	0.0 (0.0-0.1)	0.0 (0.0-0.4)	0.504
Postoperative myohemoglobin (ng/mL)	235.7 (142.9-415.3)	272.9 (147.9-531.5)	0.168	275.5 (155.7-545.3)	272.0 (158.3-441.3)	0.596
Postoperative NTproBNP (pg/mL)	239.0 (116.0-592.0)	432.0 (148.0-1365.0)	0.001	279.0 (133.5-877.5)	318.0 (134.3-1060.0)	0.886

Data are presented as median (IQR). SPMCG, Sodium-Potassium-Magnesium-Calcium-Glucose Injection; POD, postoperative day; cTnI, cardiac troponin I; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Table 4 Sensitivity analyses of the association between SPMCG and postoperative myocardial injury within 3 days.

	No. (%) of patients		Non-adjusted OR (95%CI)	P-value	Adjust OR (95%CI) ^a	P-value
	SPMCG	No SPMCG				
Matched cohort excluded patients with missing covariate values	5/138 (3.6)	5/46 (10.9)	0.31 (0.09, 1.12)	0.073	0.14 (0.02, 0.93)	0.042
Excluding patients with preoperative myocardial injury	20/756 (2.6)	10/94 (10.6)	0.23 (0.10, 0.50)	<0.001	0.32 (0.08, 1.20)	0.091
Categorizing patients with missing cTnI as not having myocardial injury	24/2729 (0.9)	16/359 (4.5)	0.19 (0.10, 0.36)	<0.001	0.26 (0.10, 0.67)	0.006
Account for patients with intraoperative use of SPMCG	22/727 (3.0)	18/138 (13.0)	0.21 (0.11, 0.40)	<0.001	0.23 (0.08, 0.63)	0.004

SPMCG, Sodium-Potassium-Magnesium-Calcium-Glucose Injection; OR, odds ratio; CI, confidence interval.

^a Adjustment was performed for age, gender, BMI, surgery type, ASA physical status, hypertension, atrial fibrillation, congestive heart failure, coronary artery disease, valvular disease, renal failure, diabetes, stroke, COPD, peripheral vascular disease, active cancer, ACEI/ARB, beta blocker, calcium channel blocker, diuretic, statin, cTnl, hemoglobin, platelet count, hsCRP. All adjusted covariates do not include missing data.

Figures

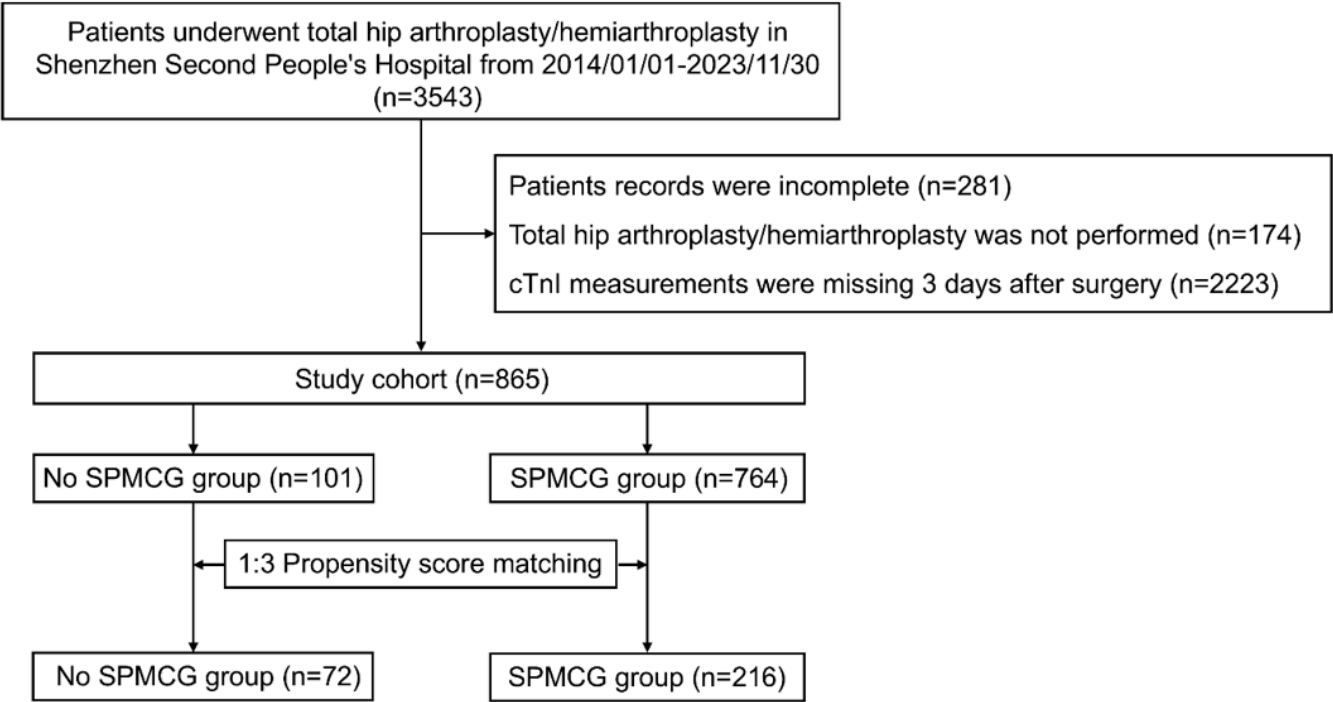


Figure 1

Flowchart of the study cohort

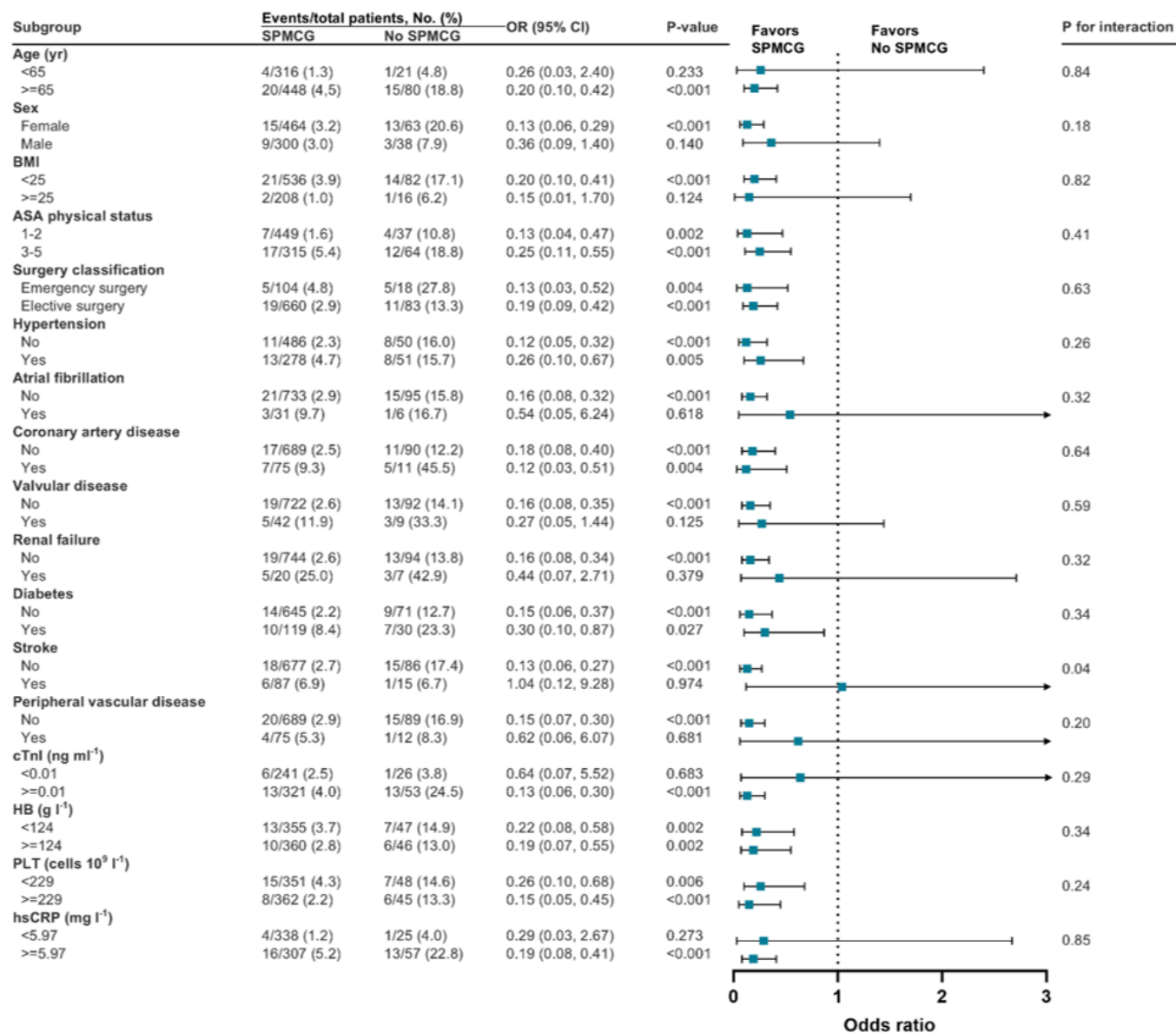


Figure 2

Subgroup analyses of the association between SPMCG and postoperative myocardial injury within 3 days.

Supplementary Files

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