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ORIGINAL CONTRIBUTIONS

Impact of Early Postbariatric Surgery Acute Kidney Injury on Long-Term Renal Function

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Abstract

Background Bariatric surgery can improve renal dysfunction associated with obesity and diabetes. However, acute kidney injury (AKI) can complicate the early postoperative course after bariatric surgery. The long-term consequences of early postoperative AKI on renal function are unknown.

Methods Patient undergoing bariatric surgery from 2008 to 2015 who developed AKI within 60 days after surgery were studied. Patients on dialysis before surgery were excluded.

Results Out of 4722 patients, 42 patients (0.9%) developed early postoperative AKI after bariatric surgery of whom five had chronic kidney disease (CKD) preoperatively including CKD stage 3 ($n = 2$), stage 4 ($n = 2$), and stage 5 ($n = 1$). Etiologies of AKI included prerenal in 37 and renal in 5 patients. Nine patients (21%) underwent hemodialysis in early postoperative period for AKI. The median duration of follow-up was 28 months (*interquartile range*, 4–59). Of the 40 patients eligible for follow-up, 36 patients (90%) returned to their baseline renal function. However, four patients (10%) had worsening of renal function at follow-up.

Conclusions The incidence of early postoperative AKI after bariatric surgery is about 1%. The most common causes of AKI after bariatric surgery are dehydration and infectious complications. In our series, 10% of patients who developed AKI in early postoperative period had worsening of renal function in long-term follow-up. In the absence of severe sepsis and severe underlying kidney dysfunction (CKD stages 4 and 5), full recovery is expected after postoperative AKI.

Keywords Acute kidney injury · Bariatric surgery · Obesity · Sleeve gastrectomy · Gastric bypass · Complication · Renal failure

Introduction

Obesity is associated with increased risk of diabetes, hypertension, and dyslipidemia. It is also associated with progression of chronic kidney disease (CKD) due to increased glomerular capillary pressure and glomerular hyperfiltration, leading to microalbuminuria and proteinuria [1]. Bariatric surgery can improve diabetes and hypertension, which are also risk factors

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for CKD. Recent studies have shown that bariatric surgery improves renal perfusion and glomerular function and reduces inflammatory markers which can lead to an improvement of microalbuminuria and proteinuria [2–6].

However, acute kidney injury (AKI) can complicate the early postoperative course after bariatric surgery. The etiology of early AKI after bariatric surgery can be multifactorial and complex in obese population [7, 8]. The preexisting factors such as diabetes, hypertension, and CKD in these patients can additionally worsen the renal insult during the early postoperative period [8, 9]. Perioperative AKI is associated with increase morbidity and mortality especially in cases of severe AKI that requires dialysis support [10, 11]. Identifying the high-risk patients for development of postoperative AKI would prevent renal injury. Our aim was to study the incidence and etiology of early postoperative AKI after bariatric surgery and to assess its long-term consequences on renal function.

Methodology

This retrospective study was conducted in an academic center after obtaining the Institutional Review Board approval. All patients who developed AKI within 60 days after bariatric surgery from 2008 to 2015 were included. Patients on dialysis before surgery were excluded. Data collected included baseline demographics, weight, body mass index (BMI), and presence of comorbidities including hypertension, dyslipidemia, diabetes mellitus, CKD, sleep apnea, and cardiovascular disease. Perioperative parameters, length of hospital stay, estimated blood loss, morbidity, and mortality rates were collected. Serum creatinine level, blood urea nitrogen (BUN) level, use of nephrotoxic medications, and intravenous contrast were retrieved from the electronic medical charts. Patients' creatinine and BUN levels were collected until the most recent follow-up.

Acute kidney injury was defined based on the Kidney Disease Improving Global Guidelines (KDIGO) [12, 13] as one of the following:

- An increase in serum creatinine by ≥ 0.3 mg within 48 h
- An increase in serum creatinine to ≥ 1.5 times baseline within previous 7 days
- Urine volume ≤ 0.5 ml/h for 6 h

All patients were managed by a multidisciplinary team of surgeons, internists, nephrologists, psychologists, and dieticians in perioperative period. Management for renal failure included maintaining fluid balance, blood transfusion (if needed), avoidance of nephrotoxic agents, and dialysis (if indicated).

The primary outcome of this study was to look at the long-term consequences of early postoperative AKI on renal function after bariatric surgery. The incidence, etiology, and management of the early postoperative AKI in bariatric surgical patients were also studied.

Data was summarized as the median and interquartile range (IQR) for continuous variables and as counts and frequency for categorical variables. The distribution of BUN and serum creatinine in different time points are shown using box-whisker plots.

Results

Demographics

Out of 4722 patients, 42 (0.9%) developed early postoperative AKI after bariatric surgery. Twenty-seven (64%) were female with median age was 53 years (IQR 43–61). The median preoperative BMI was 52 kg/m^2 (IQR 40–59). Associated pertinent comorbidities included hypertension ($n = 35$, 83%), diabetes mellitus ($n = 27$, 64%), sleep apnea ($n = 26$, 62%), dyslipidemia ($n = 21$, 50%), cardiovascular disease ($n = 12$, 29%), and CKD ($n = 5$, 12%). Underlying CKD included stage 3 ($n = 2$), stage 4 ($n = 2$), and stage 5 ($n = 1$). These five CKD patients did not require dialysis prior to bariatric surgery. Table 1 shows demographic data and preoperative comorbidities.

Perioperative and 30-Day Postoperative Details

All bariatric procedures were performed laparoscopically except one patient who underwent an open Roux-en-Y gastric bypass after previous jejunal-ileal bypass surgery. Bariatric procedures performed included Roux-en-Y gastric bypass ($n = 29$, 69%), sleeve gastrectomy ($n = 6$, 14%), revisional procedures ($n = 6$, 14%), and gastric plication ($n = 1$, 3%). Routinely, the

Table 1 Demographic data of patients with acute kidney injury after bariatric surgery ($n = 42$)

Age (years), median (IQR)	53 (43–61)
Female, *n* (%)	27 (64)
Preoperative BMI (kg/m^2), median (IQR)	52 (40–59)
Preoperative weight (kg), median (IQR)	175 (121–257)
Associated comorbidities, *n* (%)	
Hypertension	35 (83)
Diabetes mellitus	27 (64)
Sleep apnea	26 (62)
Dyslipidemia	21 (50)
Cardiovascular disease	12 (29)
Chronic kidney disease	
Stage 3	2 (5)
Stage 4	2 (5)
Stage 5	1 (2)

IQR interquartile range, *BMI* body mass index

intra-abdominal pressure during laparoscopy was set at 15 mmHg. The median operative time was 185 min (IQR 162–228), and the median length of stay was 7 days (IQR 4–19). The median estimated blood loss was 75 ml (IQR 50–150). The perioperative data is summarized in Table 2.

Early postoperative complications (< 30 days) were observed in 22 (52%) patients including anastomotic leak (n = 6), bleeding (n = 4), superficial and organ space surgical site infection (n = 3), perforated incarcerated umbilical hernia (n = 1), intestinal obstruction (n = 1), pneumonia with respiratory failure (n = 1), lung empyema (n = 1), respiratory depression secondary to narcotic overdose (n = 1), pancreatitis (n = 1), diabetic ketoacidosis (n = 1), heart failure (n = 1), and myocardial infarction (n = 1). Twelve patients (29%) had readmissions within 30 days of surgery.

Two patients died due to septic shock with multiple organ failure. One patient died on postoperative day 6 from gastrojejunostomy anastomotic leak, and another patient died 4 months after a revision of vertical band gastroplasty to Roux-en-Y gastric bypass due to abdominal wound infection and respiratory failure.

Table 2 Perioperative data patients with acute kidney injury after bariatric surgery (n = 42)

American Society of Anesthesiologist class, *n* (%)	
Class 1	0 (0)
Class 2	4 (9)
Class 3	26 (62)
Class 4	12 (29)
Class 5	0 (0)
Bariatric procedure, *n* (%)	
Roux-en-Y gastric bypass	29 (69)
Sleeve gastrectomy	6 (14)
Banded gastric plication	1 (3)
Revisonal surgery	6 (14)
Operative time (min), median (IQR)	185 (162–228)
Estimated blood loss (ml), median (IQR)	75 (50–150)
Postoperative length of stay (days), median (IQR)	7 (4–19)
30-day complications, *n*	
Leak	6
Intra-abdominal bleeding	4
Surgical site infections (superficial and organ space)	3
Myocardial infarction	1
Heart failure	1
Lung infections	2
Intestinal obstruction	1
Perforated incarcerated umbilical hernia	1
Narcotic induced respiratory depression	1
Pancreatitis	1
Diabetic ketoacidosis	1
30-day readmissions, *n*	12
Mortality, *n*	
30-day	1
1 year	1

IQR interquartile range

Acute Kidney Injury Details

The median interval between bariatric surgery and onset of AKI was 10 days (IQR 4–22). The median serum creatinine and BUN at baseline (before surgery) were 1.0 mg/dL (IQR 0.9–1.3) and 17 mg/dL (IQR 12–24), respectively. The median serum creatinine and BUN at diagnosis of AKI were 3.3 mg/dL (IQR 2.6–5.1) and 42 mg/dL (IQR 22–62), respectively. The median increase in serum creatinine at diagnosis was 2.2 mg/dL (IQR 1.5 to 3.5). In almost 90% of patients at the last follow-up, both the serum creatinine and BUN levels returned to the baseline; the median serum creatinine and BUN were 1.0 mg/dL (IQR 0.8–1.2) and 19 mg/dL (IQR 12–24), respectively. Figure 1 displays changes of BUN and serum creatinine in different time points from baseline to the last follow-up.

AKI occurred due to prerenal etiologies in 37 (88%) patients and renal causes in 5 (12%) patients. Prerenal causes included dehydration (n = 16), severe sepsis/septic shock (n = 11), hemorrhagic shock (n = 4), cardiogenic shock (n = 3), pancreatitis (n = 1), diabetic ketoacidosis (n = 1), and hypotension and respiratory depression secondary to narcotic overdose (n = 1). Renal causes included rhabdomyolysis (n = 2), contrast-induced nephropathy (n = 1), drug-induced nephropathy (n = 1), and preexisting CKD stage 5 (n = 1). Table 3 summarizes the etiology of postoperative AKI in bariatric surgical patients.

Both patients who had AKI secondary to rhabdomyolysis were females with BMI > 50 kg/m^2 and had undergone laparoscopic gastric bypass. Diagnosis of rhabdomyolysis was made based on the creatinine kinase (CK) level of 38,852 and 2137 U/L, respectively. One of the patients had severe back pain postoperatively, while the other patient was intubated for respiratory failure due to narcotic overdose at the time rhabdomyolysis was diagnosed. CT scan-guided drainage of abdominal collection postgastric bypass on day 7 caused contract-induced nephropathy. Drug-induced nephropathy was noticed on the 3rd day after administration of intravenous vancomycin for sepsis in the intensive care unit. These four patients were managed with aggressive fluid resuscitation and avoidance of nephrotoxic agents. All patients with renal etiology of AKI had complete resolution of renal failure prior to discharge except for the patient with stage 5 CKD, who went into end-stage renal disease and required permanent dialysis since then.

Majority of patients (n = 33, 79%) were managed conservatively based on underlying AKI etiologies without any dialysis support. Nine patients (21%) underwent hemodialysis

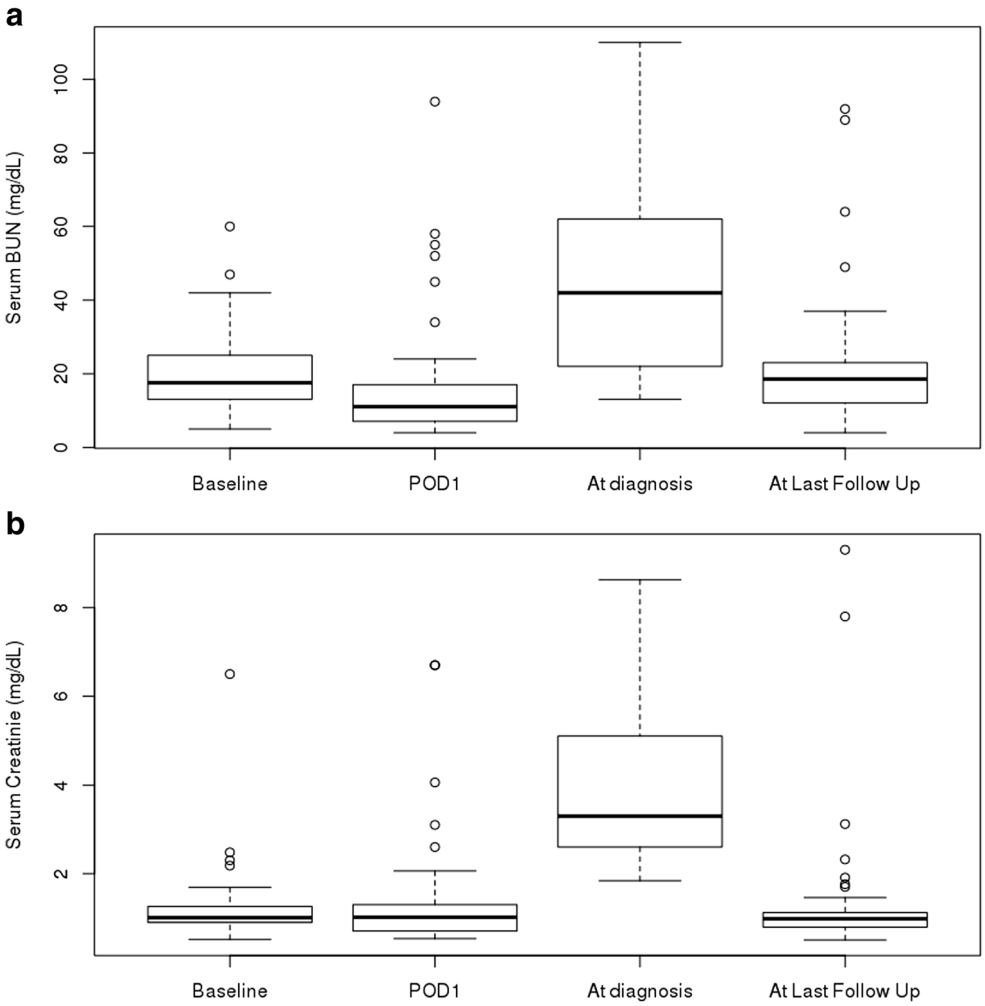

Fig. 1 Box-whisker plots showing the distribution of blood urea nitrogen (BUN) (Fig. 1a) and serum creatinine (Fig. 1b) in different time points from baseline to the last follow up. *POD1* postoperative day 1

in early postoperative period for AKI. Causes of AKI in these patients included septic shock ($n = 7$), bleeding ($n = 1$), and worsening of preexisting CKD stage 5 ($n = 1$).

The median duration of follow-up was 28 months (*IQR* 4–59). Of the 40 patients eligible for follow-up, 36 patients (90%) returned to their baseline renal function. However, four patients (10%) had worsening of renal function at follow-up; three patients who had CKD stages 4 and 5 before surgery required permanent renal replacement therapy. The fourth patient developed CKD stage 3 secondary to short gut syndrome due to multiple surgical procedures for management of intestinal leak and fistula. Table 4 summarizes the management and outcome of all AKI patients.

Discussion

In our study, the incidence of early postbariatric surgery AKI was about 1%. The most common causes of AKI after bariatric surgery were dehydration and infectious complications. With a median follow-up of 28 months, this series provides the longest follow-up data on clinical significance of postbariatric surgery AKI. We noted that most of these patients have a good outcome after acute postoperative renal insults with only 10% of patients having worsening of their renal function. In the absence of septic shock and severe underlying kidney dysfunction (CKD stages 4 and 5), full recovery would be expected after postoperative AKI.

Acute kidney injury is an increasing problem faced by nephrologists, general physicians, and surgeons [12]. AKI is recognized as a common cause for the development of CKD and is associated with a 6.5-fold increase in the risk of mortality. The recent UK government survey showed that 33% of cases of AKI were due to poor recognition of the risk factors such as sepsis and hypovolemia which resulted in inadequate management of AKI [14]. Hence, the KDIGO recommends the clinicians to effectively adopt the clinical practice guidelines for the management of AKI patients [12].

Many bariatric patients have preexisting medical comorbidities such as diabetes and hypertension, which predispose them to a higher risk of postoperative renal injury. The American College of Surgeons-National Surgical Quality

Table 3 Etiologies of postbariatric surgery acute kidney injury ($n = 42$)

Prerenal ($n = 37$)	
Dehydration	16
Hypotension secondary to cardiogenic shock	3
Hypotension secondary to bleeding	4
Hypotension secondary to pancreatitis	1
Sepsis due to	
Leak	6
Wound infection / intra-abdominal collection	3
Pneumonia with ARDS	1
Perforated incarcerated umbilical hernia	1
Diabetic ketoacidosis in type 1 diabetes	1
Hypotension and respiratory depression secondary to narcotic overdose	1
Renal ($n = 5$)	
Rhabdomyolysis	2
Contrast-induced nephropathy	1
Drug-induced nephropathy	1
Preexisting CKD stage 5	1

CKD chronic kidney disease, *ARDS* acute respiratory distress syndrome

Table 4 Management and outcomes of postbariatric surgery acute kidney injury ($n = 42$)

Management during AKI episode, *n*	
Intravenous fluids	27
Blood transfusion for acute blood loss	5
Discontinuation of nephrotoxic medications	1
Hemodialysis	9
Overall outcome, *n*	
Resolved prior to discharge	29
Resolved within 3 months after surgery	5
Improved to baseline CKD	2
Progressed to CKD (from normal baseline renal function)	1
Progressed into ESRF requiring dialysis during follow-up	3
Baseline CKD stage 4	2
Baseline CKD stage 5	1
Started on dialysis but died (no renal disease prior to surgery)	2

AKI acute kidney injury, *CKD* chronic kidney disease, *ESRF* end-stage renal failure

Improvement Program data on 300,000 patients who underwent noncardiac surgery noted that there was a 3- to 7-fold increase in postoperative AKI in patients with obesity compared to the normal population, with increasing risk with BMI [15]. Other authors have also assessed this risk in smaller studies. Weingarten et al. [16] reported that patients with higher BMI and diabetes have higher risk for postbariatric surgery AKI. Sharma et al. [9] showed that patients with BMI > 50 kg/m^2, previous CKD, and long operative times have higher risk of postoperative AKI.

The incidence of AKI has also been assessed by other studies in the past with variable results. A decade ago, Thakar et al. [17] noted an incidence of 8.5% for AKI among 491 gastric bypass patients. Weingarten et al. [16] showed that the incidence of AKI within 72 h after bariatric surgery was 5.8%, which was higher than the orthopedic and general surgeries (1%) [18, 19]. Sharma et al. [9] reported an AKI incidence of 2.3% among 1800 patients who had the laparoscopic gastric bypass. In our study, we reported a lower AKI incidence (0.9%) compared to other published studies.

Prerenal causes of AKI in bariatric surgical patients mainly occur due to poor oral intake after surgery or occurrence of surgical complications such as infection or metabolic complications [16]. In our cohort, 88% of patients had prerenal AKI; dehydration and infection were seen in 38 and 26%, respectively. Food intolerance or inadequate oral intake is not uncommon in early postbariatric surgical patients. Therefore, renal injury should be suspected in all patients with inadequate oral intake during the early postoperative period. In septicemia, kidney is almost always affected. Renal supportive therapy along with sepsis management is the mainstay of the treatment in these cases. Out of 11 septic patients in our cohort, 7 underwent dialysis for renal failure with 2 mortalities.

Male patients with BMI > 50 kg/m^2 who undergo longer operation are known to have a higher risk for rhabdomyolysis [20–23]. Lagandre et al. [21] showed that operating time over 4 h was associated with higher risk of rhabdomyolysis after bariatric surgery. Additionally, the risk of rhabdomyolysis is eight times higher in patients with diabetes. In our cohort, we had two patients with rhabdomyolysis after laparoscopic gastric bypass resulting in AKI. Both were females with BMI > 50 kg/m^2. One of them had diabetes.

Renal causes of postbariatric surgery AKI can be potentially preventable, especially drug and contrast-induced nephropathies. In our cohort, we had one patient with contrast-induced nephropathy and one patient with drug-induced nephropathy resulting in AKI. The patient with contrast-induced AKI had a CT-guided drainage of the intra-abdominal collection, which was necessary to control the infection. All the patients who had AKI secondary to renal etiologies had complete resolution of the renal failure prior to discharge except for the patient with CKD stage 5, who went into end-stage renal disease. Early detection together with a multidisciplinary team approach would be essential in these high-risk patients for a better overall outcome.

The present study has several limitations related to its small sample size and retrospective analysis from a single center. We did not study the intravenous fluid intake and urine output as these data were not consistently available among our patients. Some of our patients were possibly admitted to other hospitals during the initial AKI period and were managed

there. Therefore, this study may underestimate the true incidence of postbariatric surgery AKI. The follow-up period of patients varied as the care was transferred to nephrologists and the primary care physicians in some patients who could be outside of our health system. Finally, there were missing data on BUN and serum creatinine levels especially after resolution of AKI.

Conclusion

The incidence of early postoperative AKI after bariatric surgery is about 1%. The most common causes of AKI after bariatric surgery are dehydration and infectious complications. In our series, 10% of patients who developed AKI in early postoperative period had worsening of renal function in long-term follow-up. Patients with preexisting renal disease are at a greater risk. In the absence of severe sepsis and severe underlying kidney dysfunction (CKD stages 4 and 5), full recovery is expected after postoperative AKI.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Informed Consent Does not apply.

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