

Acute Kidney Injury after Gastric Bypass Surgery

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Gastric bypass surgery is a common treatment for morbid obesity. The presence of comorbid conditions and drugs that are used to treat them can adversely influence kidney function. Risk factors and outcomes of acute kidney injury (AKI) after gastric bypass surgery are not well understood, however. A total of 504 patients underwent gastric bypass between January 2003 and 2005. Primary outcome was AKI, defined as a $\geq 50\%$ increase in serum creatinine relative to baseline or requirement of dialysis. Secondary outcomes were duration of hospitalization, all-cause hospital mortality, and readmissions within 30 d after surgery. Demographic, comorbid, and laboratory variables and preoperative medication use were examined as potential risk factors for AKI. A total of 42 (8.5%) patients developed postoperative AKI. Hyperlipidemia, preoperative use of angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARB), intraoperative hypotension, and higher body mass index were associated with increased frequency of AKI. By multivariable analyses, the independent risk factors for AKI were body mass index (odds ratio [OR] 1.03; 95% confidence interval [CI] 1.00 to 1.06), hyperlipidemia (OR 2.53; 95% CI 1.21 to 5.28), and preoperative use of ACE-I or ARB (OR 2.06; 95% CI 1.05 to 4.04). The postoperative mortality was 0.45% ($n = 2$), both of whom had AKI. Duration of hospitalization was greater in patients with AKI *versus* no AKI (4.0 *versus* 2.7 d; $P = 0.0003$). Postoperative AKI is not infrequent after gastric bypass surgery. Certain comorbid conditions and their commonly prescribed treatments, ACE-I or ARB, are independently associated with increased risk for postoperative AKI.

Clin J Am Soc Nephrol 2: 426-430, 2007. doi: 10.2215/CJN.03961106

Gastric bypass surgery is an increasingly common treatment for morbid obesity. Compared with 16,000 gastric bypass surgeries in the 1990s, an estimated 103,000 were performed in the United States in 2003 (1). The prevalence of gastric bypass surgery in the United States, on the basis of data from the 1998 to 2002 National Hospital Discharge Survey, increased significantly from 7.0 to 38.6 per 100,000 adults (2). The risk factors for acute kidney injury (AKI) and consequences of AKI on patient outcomes in the setting of gastric bypass surgery are not well understood. Morbidities such as type 2 diabetes, hypertension, hyperlipidemia, and degenerative arthritis have an increased association with morbid obesity (3,4). Common therapies that aim to treat these conditions includes drug classes such as angiotensin-converting enzyme inhibitors (ACE-I), angiotensin receptor blockers (ARB), diuretics, antilipidemic agents, and nonsteroidal anti-inflammatory drugs (NSAID). The effect of comorbid conditions, or the common drugs that are used to treat these conditions, on renal function after gastric bypass is unknown.

The incidence of AKI during hospitalization varies depending on the criteria used to define it (5). According to a recent survey of the National Administrative Database, the incidence of AKI between 1992 and 2001 was approximately 24 cases per

1000 discharges (6). Of all of the cases of AKI, fewer than half occur in surgical settings, and only 10 to 15% of cases occur in the setting of noncardiovascular surgery. Nevertheless, perioperative AKI is associated with a striking increase in morbidity and mortality among hospitalized patients (7). Depending on the degree of renal injury and the type of surgery, acute renal failure increases hospital mortality (*e.g.*, in high-risk surgeries such as cardiac surgery, postoperative mortality in severe AKI can be $>50\%$) (8). It is widely recognized that severe AKI that requires dialysis is an independent risk factor for death, regardless of the clinical setting. However, the effect of milder degrees of renal dysfunction on hospital outcome is more heterogeneous and may vary depending on the particular clinical setting (*e.g.*, contrast-induced nephropathy *versus* cardiac or vascular surgery). Epidemiologic data unequivocally indicate that amelioration of renal injury could offer survival benefit, yet our treatment options remain limited. Therefore, integral to improving outcomes in AKI is the need to identify high-risk patients in a particular clinical setting to prevent renal injury.

We hypothesized that comorbid conditions or their treatment may influence the risk for postoperative AKI in morbidly obese patients who undergo gastric bypass surgery. In an observational cohort study, we aimed to (1) examine the incidence of AKI; (2) identify risk factors of AKI; and (3) examine the effects of AKI on short-term hospital outcomes, including hospital mortality, length of stay, and readmission to hospital. Given the plausibility that certain drugs that are administered during the preoperative period may have a deleterious effect on postoperative renal function, one of the objectives of the study was to identify potentially modifiable risk factors for AKI.

Received November 30, 2006. Accepted February 12, 2007.

Published online ahead of print. Publication date available at www.cjasn.org.

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Materials and Methods

Patient Population

We considered all consecutive patients who underwent gastric bypass at the University of Cincinnati Medical Center between January 2003 and January 2005. Surgical procedures included both Roux-en-Y and laparoscopic procedures. A total of 504 patients underwent gastric bypass during the study period. A historical cohort design was used by identifying all patients who underwent gastric bypass during the study period and by creating a data set with predefined characteristics, described next. Data that were required for the study were retrieved from electronic medical records. The study was approved by the Institutional Review Board at the University of Cincinnati.

Definitions

Primary outcome was AKI and was defined as a $\geq 50\%$ increase in serum creatinine relative to baseline during the first three postoperative days or requirement of dialysis during the postoperative period. Secondary outcomes were duration of hospital stay, all-cause hospital mortality, and readmission to hospital or an emergency department visit within up to 30 d after the date of surgery. We examined a number of variables for their association with AKI, including demographic variables such as age, gender, race, and body mass index (BMI). We also examined preoperative comorbid variables, such as presence of diabetes, hypertension, or hyperlipidemia (comorbidities were defined as physician documentation of medical diagnosis that required treatment), and laboratory variables, including serum creatinine level, hemoglobin, and hematocrit. Intraoperative variables such as duration of surgery and intraoperative hypotension, defined as at least one reading of mean arterial pressure of <60 mmHg on the anesthesia record, were also recorded. In addition, we recorded preoperative medication use for the following classes of medications: ACE-I or ARB, β blockers, calcium channel blockers, diuretics, NSAID, antidiabetic therapy including insulin, and antilipidemic therapy. These variables were chosen on the basis of clinical plausibility, availability of the data, and known risk factors for AKI in other clinical settings. The data regarding preoperative medication use were derived from preoperative anesthesia assessment records. Thirteen patients were excluded because of missing postoperative data, leaving 491 patients for analysis.

Statistical Analyses

P values for bivariate relationships between dichotomous variables were computed as two-tailed exact values. *P* values for bivariate relationships between dichotomous variables and continuous variables were two-tailed and derived from *t* test. Two-tailed $P \leq 0.05$ were considered significant. A multivariable logistic model was constructed to predict the occurrence of AKI. A variable was a candidate for this model when it had a bivariate association with AKI of $P = 0.10$ or stronger or when the *P* value was near 0.10 and the variable was of particular clinical interest. Twelve variables met these criteria and were forced into a model to predict AKI and then subjected to backward selection (removal of the weakest predictor) until all variables that remained in the model had $P \leq 0.05$, adjusted for the other variables that remained in the model. Some variables that were removed late in the selection process were re-inserted into the model to determine whether they were significant, once variables with which they shared some degree of collinearity were removed. All analyses were performed using SAS software (SAS Institute, Cary, NC).

Results

Risk Factors for AKI

A total of 491 patients were included in the final analysis. Of these, 83.5% were female, 90.4% were white, and the mean age

was 43.05 (SD 10.03). The frequencies of hypertension, hyperlipidemia, or diabetes among the patient population were 57, 33.2, and 26.2%, respectively; 37 (7.54%) patients had all three comorbidities. Regarding preoperative medication use, 184 (37.5%) patients were receiving ACE-I or ARB and 161 (32.8%) patients were on diuretics as their treatment for hypertension; 105 (21.4%) patients were taking both of these classes. Antihypertensive medications other than ACE-I/ARB or diuretics included calcium channel blockers (14.5%; $n = 71$), β blockers (16.9%; $n = 83$), and other hypertensive classes (4.5%; $n = 22$). A total of 201 patients (40.9%) were taking NSAID; 51 (10.4%) patients were taking a combination of ACE-I/ARB, diuretic, and an NSAID. Regarding the use of hepatic hydroxymethyl glutaryl-CoA (HMG-CoA) reductase inhibitors, 81% (62 of 76) patients with hyperlipidemia were receiving these agents. Mean baseline serum creatinine was 0.75 mg/dl (SD 0.56), and mean preoperative hematocrit was 38.8%. Intraoperative hypotension, defined as mean arterial pressure of <60 mmHg, occurred in 20.4% ($n = 100$) patients.

Forty-two (8.5%) patients developed AKI during the postoperative period. Two of the 42 patients developed severe AKI that required dialysis. The distributions of risk factors that were associated with AKI are described in Tables 1 and 2. Presence of hyperlipidemia, preoperative use of ACE-I/ARB, intraoperative hypotension, and higher BMI were associated with increased frequency of AKI. Combination drug therapies did not influence the frequency of postoperative AKI. Treatment with HMG-CoA reductase inhibitors was also not associated with AKI. By multivariate analyses (Table 3), the independent risk factors for developing AKI included BMI (odds ratio [OR] 1.03 per unit increase; 95% confidence interval [CI] 1.00 to 1.06), hyperlipidemia (OR 2.74; 95% CI 1.33 to 5.66), and preoperative use of ACE-I or ARB (OR 2.17; 95% CI 1.11 to 4.24).

Outcomes

As shown in Table 4, the overall hospital mortality after gastric bypass was low (0.45%; $n = 2$). Both of the patients who died, however, had AKI. The peak serum creatinine levels in these two patients were 6.6 and 3.8 mg/dl, respectively. One of the two patients required continuous renal replacement therapy and died within 24 h of initiation of dialysis. Duration of hospitalization was significantly greater among patients with AKI as compared with those without AKI (4.0 *versus* 2.7 d; $P = 0.0003$). On the basis of the latest level of serum creatinine available to us before hospital discharge, 11 (27.5%) of the 40 patients returned to within 25% of their baseline renal function before hospital discharge. A total of 75 (15.3%) patients were either readmitted or needed an emergency department visit within 30 d of surgery; nine of 75 patients required more than one readmission. The probability of a patient's having at least one revisit to the hospital was not significantly different in patients with AKI *versus* without AKI. Of note, however, longer duration of initial hospital stay (3.3 *versus* 2.7 d; $P = 0.01$) was associated with higher frequency of readmission within 30 d.

Table 1. Risk factors of AKI after gastric bypass surgery^a

Risk Factor	AKI (<i>n</i> = 42; 8.6%; <i>n</i> [%])	No AKI (<i>n</i> = 449; 91.4%; <i>n</i> [%])	<i>P</i>
Gender			1.00
male (<i>n</i> = 81)	7 (16.6)	74 (16.5)	
female (<i>n</i> = 410)	35 (83.4)	375 (83.5)	
Race			0.86
white (<i>n</i> = 444)	38 (90.5)	406 (90.4)	
nonwhite (<i>n</i> = 47)	4 (9.5)	43 (9.6)	
Diabetes			0.10
yes (<i>n</i> = 129)	16 (38.1)	113 (25.2)	
no (<i>n</i> = 362)	26 (61.9)	336 (74.8)	
Hypertension			0.19
yes (<i>n</i> = 280)	28 (66.7)	252 (56.1)	
no (<i>n</i> = 211)	14 (33.4)	197 (44.9)	
Hyperlipidemia			0.003
yes (<i>n</i> = 76)	14 (33.3)	62 (13.8)	
no (<i>n</i> = 415)	28 (66.7)	387 (86.2)	
ACE-I or ARB use			0.002
yes (<i>n</i> = 184)	25 (59.5)	159 (35.4)	
no (<i>n</i> = 307)	17 (40.5)	290 (64.6)	
Calcium channel blocker use			0.81
yes (<i>n</i> = 71)	5 (11.9)	66 (14.7)	
no (<i>n</i> = 420)	37 (88.1)	383 (85.3)	
Diuretic use			0.73
yes (<i>n</i> = 161)	15 (35.7)	146 (32.5)	
no (<i>n</i> = 330)	27 (64.3)	303 (67.5)	
β blocker use			0.20
yes (<i>n</i> = 83)	10 (23.8)	73 (16.2)	
no (<i>n</i> = 408)	32 (76.2)	376 (83.8)	
NSAID use			0.51
yes (<i>n</i> = 201)	15 (35.7)	186 (41.4)	
no (<i>n</i> = 290)	27 (64.3)	263 (58.6)	
Preoperative renal function			0.41
Cr ≥1.2 mg/dl (<i>n</i> = 21)	3 (7.1)	18 (4)	
Cr <1.2 mg/dl (<i>n</i> = 470)	39 (92.9)	431 (96)	
Intraoperative hypotension ^b			0.01
yes (<i>n</i> = 100)	15 (35.7)	85 (18.9)	
no (<i>n</i> = 390)	27 (64.3)	363 (81.1)	

^aACE-I/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; AKI, acute kidney injury; NSAID, nonsteroidal anti-inflammatory drug; Cr, serum creatinine.

^bOne patient was missing data on intraoperative hypotension status.

Table 2. Risk factors for AKI after gastric bypass surgery^a

Risk Factor	AKI (Mean [SD])	No AKI (Mean [SD])	<i>P</i>
Age (yr)	45.6 (10.4)	42.8 (9.9)	0.08
BMI	54.1 (9.3)	51.0 (9.3)	0.04
Baseline creatinine (mg/dl)	0.75 (0.56)	0.77 (0.18)	0.08
Preoperative hematocrit (%)	38.8 (4.8)	39.2 (3.8)	0.53
% drop in hematocrit (postoperative versus preoperative)	12.9 (12.0)	11.8 (8.3)	0.44

^aBMI, body mass index.

Discussion

The present study assesses the incidence, risk factors, and outcomes of AKI after gastric bypass surgery. Hyperlipidemia, higher BMI, and preoperative use of ACE-I or ARB were independent risk factors for development of AKI. Both the incidence of AKI and the overall mortality after gastric bypass surgery were relatively low. AKI was significantly associated with increased duration of hospitalization but did not influence readmission episodes within 30 d of surgery.

Gastric bypass surgery is an increasingly common treatment for morbid obesity. Studies regarding outcomes of gastric bypass have described (1) postoperative mortality, (2) long-term outcomes including weight reduction and impact of weight loss on diabetes or hypertension, and (3) short-term adverse events (4,9–13). Flum *et al.* (9) reported early mortality among Medicare beneficiaries who underwent bariatric surgical procedures (1997 to 2002) and examined postoperative mortality at 30 d,

90 d, and 1 yr. The rates of 30-d, 90-d, and 1-yr mortality were 2, 2.8, and 4.6%, respectively. After adjustment for age, gender, and comorbidity index, the odds for death within 90 d were five-fold greater for those who were older than 75 yr as compared with 65 to 74 yr. Santry *et al.* (11) examined the trends in bariatric surgical procedures and associated outcomes as derived from the Nationwide Inpatient Sample, based on *International Classification of Diseases, Ninth Revision* (ICD-9) diagnosis codes. The number of bariatric surgical procedures increased from 13,365 surgeries in 1998 to 72,177 surgeries in 2002. A cross-sectional inquiry for the occurrence of complications and the procedure of gastric bypass surgery allowed testing of their association, albeit with lack of exact temporality. Genitourinary complications included ICD-9 codes for urinary tract complications, acute renal failure, acute dialysis, or insertion of short-term dialysis catheter during the same hospitalization as gastric bypass. The study reported that the postoperative mortality rates were low (1% or less) and that the mean duration of hospitalization reduced from 4.9 d in 1998 to 3.3 d in 2002 ($P < 0.001$). Occurrence of genitourinary complications also indicated a decreasing trend over time (2.1% in 1998 to 0.4% in 2002; $P = 0.003$). Both of these studies reported that postoperative mortality in gastric bypass is low. The information, however, is derived from administrative databases in which comorbid disease estimates are based on ICD-9 diagnoses codes and lack patient-level laboratory data. Most important, the studies provide a very limited insight regarding frequency and determinants of postoperative AKI. Our study indicates that the frequency of AKI after gastric bypass was almost 9%. Multivariable analysis indicated that the presence of hyperlipidemia, higher BMI, and preoperative use of ACE-I or ARB were independent predictors for postoperative AKI.

Several additional studies have also examined short-term or long-term outcomes after gastric bypass by using patient-level data (14–17). Two recent meta-analyses and systematic reviews examined outcomes in gastric bypass, based on the published literature between 1990 and 2003 (18,19). These meta-analyses confirm that the mortality after gastric bypass is consistently low and that there are unequivocal long-term benefits of the procedure in terms of weight reduction and control of associated comorbidities. Although the studies have reported a variety of short-term adverse events, the information regarding postoperative AKI or the impact of postoperative AKI on hospital outcomes is notably absent. AKI, in its most severe form, is an independent risk factor for mortality in hospitalized patients (7,20). It is increasingly evident that even a mild degree of renal dysfunction is associated with an increased risk for morbidity and mortality and resource utilization (21). However, whether similar thresholds of renal

Table 3. Risk factors for AKI: Multivariable analysis

Risk Factor	OR	95% CI	P
BMI (per unit increase)	1.03	1.00 to 1.06	0.05
Hyperlipidemia	2.74	1.33 to 5.67	0.006
ACE-I/ARB	2.17	1.11 to 4.24	0.02

^aCI, confidence interval; OR, odds ratio.

Table 4. Outcomes measures in AKI after gastric bypass surgery

Outcome	AKI	No AKI	P
Hospital stay (mean [SD])	4.0 (2.1)	2.7 (1.15)	0.0003
Readmission	9/40 (22.5%)	66/383 (14.7%)	0.24
Hospital mortality	2/42 (4.8%)	0/449 (0%)	0.007

dysfunction are associated with similar outcomes across different clinical settings remains unclear. Although the overall mortality after gastric bypass surgery was low, it should be noted that both of the patients who died had developed AKI. In addition, duration of hospitalization, a widely recognized surrogate for costs of care, was significantly greater in the AKI group compared with those without AKI.

This study confirms a high prevalence of conditions such as hypertension, diabetes, and hyperlipidemia in morbidly obese patients (3,4). Effects of drugs that are used to treat these conditions and may influence acute changes in renal function are not well studied in clinical settings. This study analyzed the risk for AKI that is associated with commonly prescribed medications for comorbid conditions in patients who undergo gastric bypass (as a standard of care, patients who undergo gastric bypass are routinely advised to take their prescribed medications on the morning of surgery). The data indicate preoperative use of ACE-I/ARB was associated with increased odds for development of AKI and that the association was independent of other major comorbid factors. The use of HMG-CoA reductase inhibitors was not associated with AKI in our cohort. Although complications such as perioperative rhabdomyolysis have been reported (22,23), we found no association among use of these agents, rhabdomyolysis, and occurrence of AKI.

The mechanisms behind the association between the use of ACE-I/ARB agents and AKI are not completely clear. It is possible that their preoperative use impairs the ability of kidneys to autoregulate sudden changes in renal hemodynamics and that the effect of these agents in the presence of other risk factors makes the kidneys vulnerable to sustaining ischemic injury. Whether the use of renin-angiotensin system blockers serves as a surrogate for presence of proteinuria could not be examined in our cohort because of the lack of data regarding protein excretion. It should be noted, however, that the number of patients with preoperative elevation of creatinine was very low. We interpret these results to suggest that avoiding drugs such as ACE-I or ARB during the preoperative period may reduce the risk for AKI. Whether this association can be applicable in other settings of AKI remains uncertain.

There are other limitations to this study. The report involves observations from a single institution. However, we examined more than 500 patients with demographic distribution and the overall hospital outcomes (mortality or duration of stay) that were similar to other larger multicenter observations, thereby providing generalizability and external validity for our findings. The event rate of AKI was relatively low, which may have limited our ability to test for a large number of risk factors in a

multivariable model. We did, however, examine major risk factors for AKI, based on literature review. Most important, evaluating modifiable risk factors for AKI may lead to strategies for ameliorating perioperative AKI.

Conclusion

Postoperative AKI is not infrequent after gastric bypass surgery. Although the overall postoperative mortality is low, AKI is associated with increased duration of hospital stay. Certain comorbid conditions that are unique to this patient population influence the risk for postoperative AKI. In addition, commonly prescribed medications, such as ACE-I and ARB, for treatment of comorbid conditions are independently associated with increased risk for postoperative AKI. This suggests that simple strategies, such as avoidance of these classes of drugs, may provide short-term benefits in reducing morbidity and costs of care in patients who undergo gastric bypass surgery.

Acknowledgments

The study was supported by internal revenue support from the Division of Nephrology.

The preliminary findings of this study were presented at the spring 2006 meetings of The National Kidney Foundation (April 19 to 23, 2006; Chicago, IL).

We acknowledge the help of personnel from the Department of Surgery at the University of Cincinnati in providing us the necessary access to create the data set for proposed analyses.

Disclosures

None.

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