

Prior bariatric surgery and better short-term outcomes in acute pancreatitis: a multicenter propensity-matched analysis

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Abstract

Background Obesity is associated with an increased risk of severe acute pancreatitis (AP). Bariatric surgery is known to improve comorbidities and reduce systemic inflammatory burden. We aimed to evaluate whether a history of bariatric surgery is associated with differences in short-term clinical outcomes among patients hospitalized with AP.

Methods Retrospective study using the TriNetX research network, including adults aged 18 years or older who were hospitalized with AP. Patients with chronic pancreatitis, pancreatic malignancy or cystic pancreatic disease were excluded. Study cohorts were defined based on the presence or absence of prior bariatric surgery performed at least 1 year before the AP admission. Cohorts were matched 1:1 using propensity scores. The primary outcome was 30-day all-cause mortality. Secondary outcomes were acute kidney injury (AKI), need for hemodialysis, endotracheal intubation, vasopressor use, elevations in inflammatory markers, and pancreatitis-related procedures, such as endoscopic retrograde cholangiopancreatography (ERCP) with stent placement, cholecystectomy, and pancreatic debridement.

Results After matching, both cohorts included 4024 patients. Bariatric patients had significantly lower 30-day mortality (1.9% vs. 2.6%; relative risk [RR] 0.71, 95% confidence interval [CI] 0.53-0.95; P=0.021), less AKI (0.8% vs. 1.5%; RR 0.54, 95%CI 0.35-0.82; P=0.003), and less hemodialysis (0.5% vs. 0.9%; RR 0.54, 95%CI 0.31-0.93; P=0.025). ERCP with stent placement was less frequent in the bariatric group (1.0% vs. 1.6%; RR 0.60, 95%CI 0.40-0.89; P=0.011). Other secondary outcomes did not differ significantly between groups.

Conclusions Bariatric surgery was associated with better short-term outcomes in AP. This included lower mortality and renal complications, possibly via metabolic and inflammatory effects.

Keywords Acute pancreatitis, bariatric, obesity, mortality

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Introduction

Acute pancreatitis (AP) is a common cause of hospitalization related to gastrointestinal disease. In the United States, it

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results in approximately 200,000-275,000 hospital admissions each year [1,2]. Similar trends have been reported in Europe, Asia and North America, where its incidence continues to increase [3,4]. Although many patients have a mild and self-limited course, a clinically important subset, estimated at up to 20%, develop severe disease with systemic inflammation, organ failure and substantial mortality [5,6].

Obesity is associated with both the development of AP and worse clinical outcomes once the disease occurs. In the United States, roughly 40% of adults are obese, and close to 10% meet the criteria for severe obesity [7]. Prior epidemiologic and meta-analytic studies show that obesity contributes to more severe AP, probably related to systemic inflammation, gallstone disease and underlying metabolic dysfunction [8,9]. Patients with morbid obesity appear to be at high risk for morbidity, with organ failure, mortality and higher healthcare utilization during AP [10].

Bariatric surgery has proven to be effective in the management of severe obesity. Roux-en-Y gastric bypass and sleeve gastrectomy surgeries are associated with weight loss, improved control of type 2 diabetes and other metabolic disorders, and lower overall mortality [11,12]. Additionally, bariatric surgery induces reductions in proinflammatory cytokines, improvements in endothelial function, and favorable alterations in bile acid and gut microbiome signaling [13-15]. These changes may play a role in modifying the systemic inflammatory response that drives severe AP.

Data on AP outcomes in patients with a history of bariatric surgery are limited and inconsistent. Most of the studies, although small and single-center, reported better outcomes, which included lower mortality and organ failure in patients with a history of bariatric surgery [16,17]. Other studies reported higher rates of AP following bariatric surgery, thought to be secondary to rapid weight loss [18]. The smaller sample sizes emphasized the need for large-scale, multi-institutional analyses.

We utilized the TriNetX global health network to address this knowledge gap. This database allows for analysis of electronic health records from different healthcare systems, and enables comparisons of outcomes while adjusting for baseline characteristics and clinical factors. Therefore, we decided to utilize this database and determine whether prior bariatric surgery is associated with differences in short-term outcomes in patients admitted with AP. Our primary hypothesis was that a history of bariatric surgery would be associated with lower 30-day mortality and better complication profiles compared with matched controls who had no prior surgery.

Patients and methods

Data source

We utilized the TriNetX research network, a database that includes de-identified patient information from 68 healthcare systems in the United States and internationally. The study period was from 2005 to 2025. It has been validated as a platform that provides information on patient demographics, diagnosis, laboratory values and procedures, in both the inpatient and outpatient settings, that can be used for large-scale observational research [19].

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Study population

Adult patients aged 18 years or older who had a diagnosis of AP were extracted using ICD-10-CM codes, as in prior studies of AP using administrative datasets [2,3]. The first patient encounter with a diagnosis of AP was defined as the index hospitalization. Patients with chronic pancreatitis, pancreatic neoplasms or cystic pancreatic disease were excluded.

Two cohorts were constructed. The first comprised patients with a documented history of bariatric surgery at least 1 year before the AP episode. The second included patients without prior bariatric surgery. The 1-year threshold was chosen to minimize confounding from perioperative complications, and to ensure that patients had achieved at least partial stabilization of weight and metabolic changes post-surgery.

Covariates and propensity matching

Baseline covariates included demographics (age, sex, race, ethnicity) and comorbidities (hypertension, diabetes mellitus, ischemic heart disease, nicotine dependence, alcohol-related disorders, opioid use disorder, malnutrition, cholelithiasis). These comorbidities were selected for their known relevance to AP risk and outcomes [9,10]. Propensity score matching was performed in a 1:1 ratio using nearest-neighbor matching without replacement, balancing covariates between the 2 cohorts. Standardized mean differences <0.1 were considered indicative of acceptable balance [20].

Outcomes

All outcomes were assessed within 30 days of the index hospitalization, and patients were not followed over multiple years. The primary outcome was 30-day all-cause mortality following AP diagnosis. Secondary outcomes included acute kidney injury (AKI), need for hemodialysis, endotracheal intubation, vasopressor requirement, elevation of inflammatory markers (C-reactive protein [CRP] ≥ 10 mg/L; erythrocyte sedimentation rate [ESR] > 30 mm/h), and procedures including endoscopic retrograde cholangiopancreatography (ERCP) with stent placement, cholecystectomy, and pancreatic debridement. These outcomes were chosen to represent both clinical severity and healthcare utilization.

Statistical analysis

We compared categorical variables using chi-square tests, and continuous variables by Student's *t*-test. Relative risks (RR) with 95% confidence intervals (CI) were calculated for each outcome. A P-value of <0.05 was considered statistically significant. Analyses were conducted within the TriNetX platform.

Results

Baseline characteristics

We identified 4024 patients with prior bariatric surgery and 362,963 patients without prior surgery who were hospitalized with AP. Before propensity score matching, significant differences were observed between the groups. Bariatric patients were more likely to be younger, female and White, with higher rates of comorbidities such as hypertension, diabetes, malnutrition and gallstone disease. Additionally, before matching, patients with prior bariatric surgery had higher rates of alcohol-related disorders (15.2% vs. 8.3%), although this difference was eliminated post-matching (Table 1). After matching, both cohorts included 4024 patients. Baseline characteristics were well balanced, with no statistically significant differences in demographics or comorbidities. This balance supports the validity of subsequent outcome comparisons.

Primary outcome: mortality

Within 30 days of AP diagnosis, patients with a history of bariatric surgery had significantly lower all-cause mortality compared with matched controls (1.9% vs. 2.6%; P=0.021; RR 0.71, 95%CI 0.53-0.95). This difference represents a nearly 30% relative risk reduction, highlighting a meaningful survival advantage in the bariatric cohort.

Secondary outcomes: renal and critical care complications

The incidence of AKI was significantly lower in the bariatric cohort (0.8% vs. 1.5%; P=0.003; RR 0.54, 95%CI 0.35-0.82).

Similarly, this cohort had less need for hemodialysis (0.5% vs. 0.9%; P=0.025; RR 0.54, 95%CI 0.31-0.93). These findings suggest that bariatric surgery may confer renal protection during systemic inflammatory stress associated with AP.

In contrast, there were no statistically significant differences in the need for intubation (0.8% vs. 1.1%; P=0.135; RR 0.71, 95%CI 0.46-1.11) or vasopressor therapy (1.4% vs. 1.7%; P=0.396; RR 0.86, 95%CI 0.61-1.22).

Secondary outcomes: inflammatory markers and procedures

Rates of elevated inflammatory markers were similar between groups. CRP elevations occurred in 2.1% of bariatric patients compared with 2.8% of controls (P=0.083; RR 0.78, 95%CI 0.59-1.03), while ESR elevation was noted in 0.6% vs. 0.9% (P=0.122; RR 0.67, 95%CI 0.41-1.12). Procedural outcomes revealed notable differences. The need for ERCP with stent placement was significantly lower in the bariatric cohort (1.0% vs. 1.6%; P=0.011; RR 0.60, 95%CI 0.40-0.89). This may reflect differences in AP etiology, particularly gallstone-related disease, or altered endoscopic access after bariatric procedures. Cholecystectomy rates were similar across groups, while pancreatic debridement was rare and equally distributed (0.25% in both cohorts; P=0.99; RR 1.00, 95%CI 0.42-2.40) (Table 2).

Taken together, these findings demonstrate that prior bariatric surgery is associated with better short-term outcomes in AP. Lower mortality and fewer renal complications were the most striking benefits, while less use of ERCP with stent placement may reflect either different etiology or altered

Table 1 Baseline characteristics of the 2 cohorts before and after propensity score matching (PSM)

Variables	Before PSM			After PSM		
	Bariatric (n=4024)	Non-bariatric (n=362,963)	P-value	Bariatric (n=4024)	Non-bariatric (n=4024)	P-value
Number of patients	4024	362,936	0.001	4024	4024	0.345
Female	3087	170,730	<0.001	3087	3089	0.958
Male	764	169,305	<0.001	764	777	0.713
White	2714	222,998	<0.001	2714	2741	0.520
Black/African American	770	56,141	<0.001	770	787	0.631
Hispanic/Latino	315	38,979	<0.001	315	287	0.235
Hypertension	2759	122,691	<0.001	2759	2824	0.116
Ischemic heart disease	876	43,047	<0.001	876	876	>0.99
Diabetes mellitus	1543	67,109	<0.001	1543	1567	0.583
Nicotine dependence	873	51,577	<0.001	873	912	0.295
Alcohol-related disorders	613	30,283	<0.001	613	596	0.596
Opioid use disorder	304	8565	<0.001	304	279	0.282
Malnutrition	536	11,227	<0.001	536	502	0.258
Cholelithiasis	771	29,561	<0.001	771	736	0.317

Table 2 Outcomes between cohort 1, patients with a history of bariatric surgery at least 1 year prior to acute pancreatitis, and cohort 2, patients without a history of bariatric surgery, over an observation period of 30 days after an acute pancreatitis event

Outcome	Bariatric	Non-bariatric	P-value	RR (95%CI)
Number of patients	4024	4024	-	-
Mortality	75 (1.86%)	106 (2.63%)	0.021	0.71 (0.53-0.95)
AKI	33 (0.82%)	59 (1.49%)	0.003	0.54 (0.35-0.82)
HD	19 (0.47%)	35 (0.87%)	0.025	0.54 (0.31-0.93)
Intubation	34 (0.84%)	46 (1.14%)	0.135	0.71 (0.46-1.11)
Norepinephrine use	57 (1.42%)	67 (1.67%)	0.396	0.86 (0.61-1.22)
Elevated ESR (>30 mm/h)	25 (0.62)	37 (0.92%)	0.122	0.67 (0.41-1.12)
Elevated (CRP≥10 mg/L)	85 (2.11%)	111 (2.76%)	0.083	0.78 (0.59-1.03)
Pancreatic debridement	10 (0.25%)	10 (0.25%)	0.99	1.00 (0.42-2.40)
ERCP with stent insertion	38 (0.95%)	63 (1.57%)	0.011	0.60 (0.40-0.89)
Cholecystectomy	11 (0.27%)	14 (0.35%)	0.548	0.79 (0.36-1.73)

RR, relative risk; CI, confidence interval; AKI, acute kidney injury; HD, hemodialysis; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; ERCP, endoscopic retrograde cholangiopancreatography

anatomy or technical challenges. Other indicators of severe disease, including intubation, vasopressor support and inflammatory markers, showed no significant differences.

Discussion

This large, multicenter, propensity score-matched study, which used the TriNetX research network, found significantly better short-term outcomes in patients with a prior history of bariatric surgery who developed AP, compared to those without a history of bariatric surgery. These outcomes included lower 30-day mortality, lower rates of AKI and need for hemodialysis, as well as a lower frequency of ERCP with stent placement. There were no differences in the need for mechanical ventilation, vasopressor therapy, pancreatic debridement or inflammatory markers. Our results suggest that a history of bariatric surgery could ameliorate the clinical course of AP without changing the frequency of critical care interventions.

The relationship between obesity, bariatric surgery and AP is multifactorial. Obesity is associated with both an increased risk of developing AP and worse clinical outcomes once pancreatitis occurs. Data from a large individual patient-level meta-analysis showed that obesity independently increases the likelihood of severe disease, organ failure and mortality in AP [11]. Epidemiologic studies suggest that obesity not only raises the risk of gallstone formation, the most common cause of AP in Western populations, but also exacerbates the systemic inflammatory response following disease onset [4,5,17,18]. This inflammatory amplification is largely mediated by adipose tissue-derived cytokines, including tumor necrosis factor (TNF)- α and interleukin (IL)-6, which promote systemic inflammatory response syndrome and increase the risk of multiorgan dysfunction [13,14]. Additionally, greater peripancreatic and visceral fat volumes provide a substrate for

lipolysis. This could result in toxic free fatty acid release and local tissue necrosis, which in turn could further contribute to AP severity [21].

Bariatric surgery is associated with changes in several biologic pathways relevant to AP. Weight loss after bariatric procedures is accompanied by a reduction in visceral adiposity, which may contribute to lowering inflammatory cytokine levels, and improving endothelial function. Previous research reported lower levels of IL-6, TNF- α and leptin, along with higher levels of adiponectin, after bariatric surgery [15,16]. It has also been linked to improvements in insulin resistance, reduced hepatic steatosis, and more physiologic bile acid signaling, factors that may contribute to a metabolic environment less conducive to severe AP [22,23]. In this study, these systemic changes were accompanied by better survival and fewer renal complications in patients with a history of bariatric surgery. The observed benefits probably reflect both weight loss and metabolic effects, rather than weight reduction alone.

AKI is an important complication of AP. Several factors, including inflammation, volume depletion and microvascular injury, contribute to renal dysfunction during pancreatitis [6]. In patients with a history of bariatric surgery, we found lower rates of AKI and less need for hemodialysis. Similar associations have been described in long-term outcome studies following bariatric surgery, which report better renal outcomes and lower cardiovascular mortality [11,12,23]. Moreover, the absence of differences in intubation or vasopressor use suggests that bariatric surgery may preferentially influence renal and metabolic complications, rather than respiratory or hemodynamic severity.

Another key finding was the lower rate of ERCP with stent placement in the bariatric cohort. Gallstone disease is the leading cause of AP in obese patients, and bariatric surgery has been shown to alter gallstone risk in complex ways. While rapid postoperative weight loss transiently increases gallstone risk, long-term reductions in obesity may lower the lifetime gallstone burden [5,17,18]. Thus, the lower ERCP

with stent placement rates we observed could reflect a smaller contribution of gallstone-related pancreatitis in the bariatric group. Alternatively, this finding could represent an anatomic limitation, as procedures such as Roux-en-Y gastric bypass make conventional ERCP technically challenging. Advanced interventions, such as device-assisted enteroscopy ERCP or endoscopic ultrasound-directed transgastric ERCP (EDGE), are increasingly used in these patients, but these may not always be captured by coding data in TriNetX, which may affect the interpretation of the lower ERCP with stent placement rate [19,24]. Distinguishing between etiologic reduction and technical barriers requires a prospective study, ideally stratified by bariatric procedure type.

Although bariatric surgery has been reported in some series to increase the incidence of AP, particularly in the perioperative period or in association with rapid weight loss [10], most of these cases are mild. Our study excluded perioperative cases, by requiring at least 1 year between bariatric surgery and the AP episode. In this way, we captured outcomes among patients who had already derived metabolic benefits from surgery, thereby focusing on long-term effects rather than short-term postoperative complications.

The present study builds upon and extends prior work. Earlier database analyses and single-center cohorts have suggested better outcomes in post-bariatric AP, including lower rates of mortality and organ failure [8,9,16,17]. However, those studies were limited by smaller sample sizes and less robust control for comorbidities. The TriNetX database allowed a large multicenter analysis. An additional strength is the ability to perform propensity score matching. This step is important, as it improves the generalizability of the results. Importantly, these findings reflect an association only, as the underlying mechanisms are difficult to establish.

Moreover, we do not suggest that our results will directly affect the clinical management of AP. Instead, they suggest that patients with prior bariatric surgery might be less likely to develop severe complications. We think this may help in the risk stratification of those patients, as well as counseling. Future studies that focus on the influence of metabolic pathways after bariatric surgery could aid in the development of strategies that modulate the inflammatory response in patients with AP.

This study had several limitations. First, confounding bias would still be possible in the setting of a retrospective study design. Despite propensity matching, some factors that were not accounted for included the degree of obesity severity, duration from time of bariatric surgery, nutritional factors, or medications that may have influenced our outcomes. Secondly, this database relies on ICD-10 codes, which could be misclassified among different healthcare professionals and systems. Additionally, inflammatory markers that may suggest the degree of disease severity have not been validated for use in AP, including other known scores such as BISAP, APACHE II or Ranson's criteria [25]. Third, the type of bariatric surgery, whether sleeve gastrectomy or gastric bypass, was not differentiated in our study. This represents a limitation, as different bariatric procedures may have varying metabolic effects that possibly influence

gallstone disease and systemic inflammation [22]. Fourth, we did not assess long-term outcomes, such as recurrence of AP or development of chronic pancreatitis and exocrine pancreatic insufficiency. Fifth, we were unable to classify specific etiologies of AP, such as alcohol-related AP; this is an inherent limitation of the database. Instead, our results showed a higher prevalence of alcohol-related disorders in patients with a prior history of bariatric surgery (15.2% vs. 8.3%), which could be considered representative in this population. Importantly, this difference was eliminated after propensity score matching. Regardless, this inability to determine the etiologies of AP remains a key limitation. Sixth, the database we used collects information over years where changes in the management of AP may have occurred. However, the cohorts were analyzed from the same time frame, so these changes would be expected to affect both groups similarly. Nevertheless, this remains a limitation of retrospective. Finally, outcomes related to ERCP should be interpreted with caution. Patients with a history of bariatric surgery have an altered anatomy, which may affect the choice of procedure or coding, rather than reflecting actual differences in disease etiology.

Despite these limitations, the results highlight important clinical outcomes. As the prevalence of obesity is on the rise, along with bariatric surgery, understanding the effects of this on inflammatory conditions such as AP is crucial. Our results suggest that bariatric surgery not only positively impacts the cardiovascular and metabolic morbidities associated with obesity, but may also mitigate the systemic complications of AP. These results may inform counseling of obese patients who are considering bariatric surgery, and guide clinicians caring for post-bariatric patients with AP. Additionally, they provide a framework for translational research into the pathways via which bariatric surgery alters the inflammatory responses.

We suggest that further prospective research, in the form of randomized controlled trials or large registries, may precisely define the role of bariatric surgery in patients with AP. Studies specified by type of bariatric surgery with inclusion of known AP severity scores, and analysis of both short- and long-term outcomes should be pursued. Additionally, research involving the effects of bariatric surgery on AP at the cellular level (adipokine profiles, bile acid signaling) and gut microbiome could also add to the literature [22,23].

In conclusion, prior bariatric surgery was associated with better short-term outcomes in patients hospitalized with AP. These outcomes included better 30-day mortality, a lower incidence of AKI and need for hemodialysis, and less utilization of ERCP with stent placement. These benefits were not accompanied by increases in other measures of severe disease, suggesting that bariatric surgery may attenuate the adverse systemic effects of obesity in AP through favorable metabolic and inflammatory changes. As the prevalence of both obesity and bariatric surgery increases, clarifying these associations is essential. Future prospective and mechanistic studies are warranted to confirm these findings and to elucidate the pathways through which bariatric surgery modifies the course of AP.

Summary Box

What is already known:

- Obesity increases the risk and severity of acute pancreatitis (AP), while bariatric surgery improves metabolic and inflammatory profiles
- Prior smaller studies have suggested better AP outcomes in patients with a history of bariatric surgery, but large multicenter data are limited

What the new findings are:

- In a multicenter matched cohort, patients with prior bariatric surgery had lower 30-day mortality (1.9% vs. 2.6%) and fewer renal complications, including acute kidney injury and need for hemodialysis
- Rates of specialized procedures, such as endoscopic retrograde cholangiopancreatography with stent placement, were lower in post-bariatric patients, while critical care interventions (intubation, vasopressor use) were similar between groups

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