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ORIGINAL SCIENTIFIC REPORT

The Impact of Obesity on Renal Trauma Outcome: An Analysis of the National Trauma Data Bank from 2013 to 2016

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Abstract

Background The obesity paradox has been recently demonstrated in trauma patients, where improved survival was associated with overweight and obese patients compared to patients with normal weight, despite increased morbidity. Little is known whether this effect is mediated by lower injury severity. We aim to explore the association between body mass index (BMI) and renal trauma injury grade, morbidity, and in-hospital mortality.

Methods A retrospective cohort of adults with renal trauma was conducted using 2013–2016 National Trauma Data Bank. Multiple regression analyses were used to assess outcomes of interest across BMI categories with normal weight as reference, while adjusting for relevant covariates including kidney injury grade.

Results We analyzed 15181 renal injuries. Increasing BMI above normal progressively decreased the risk of high-grade renal trauma (HGRT). Subgroup analysis showed that this relationship was maintained in blunt injury, but there was no association in penetrating injury. Overweight (OR 1.02, CI 0.83–1.25, $p = 0.841$), class I (OR 0.92, CI 0.71–1.19, $p = 0.524$), and class II (OR 1.38, CI 0.99–1.91, $p = 0.053$) obesity were not protective against mortality, whereas class III obesity (OR 1.46, CI 1.03–2.06, $p = 0.034$) increased mortality odds. Increasing BMI by category was associated with a stepwise increase in odds of acute kidney injury, cardiovascular events, total hospital length of stay (LOS), intensive care unit LOS, and ventilator days.

Conclusions Increasing BMI was associated with decreased risk of HGRT in blunt trauma. Overweight and obesity were associated with increased morbidity but not with a protective effect on mortality. The obesity paradox does not exist in kidney trauma when injury grade is accounted for.

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Abbreviations

AAST	American Association for the Surgery of Trauma
BMI	Body mass index
ED	Emergency department
GCS	Glasgow coma scale
HGRT	High-grade renal trauma
Intensive Care Unit	ICU
ISS	Injury severity score
LOS	Length of stay
LGRT	Low-grade renal trauma
NTDB	National Trauma Data Bank
RR	Risk ratio
US	United States
WHO	World Health Organization

Introduction

The kidney is the third most commonly injured organ in abdominal trauma and the second most commonly injured in penetrating trauma [1]. Traumatic renal injury is associated with in-hospital complications and mortality in 33% and 14% of cases, respectively [2]. Studying the mechanics of renal injury could lead to improved trauma therapeutics, safety equipment, and outcomes.

The obesity paradox is the apparent association of increased survival in overweight and class I obese patients compared to normal weight patients, despite higher morbidity [3]. Originally established in cancer [4–6], cardiovascular [7, 8], and other chronic diseases [9, 10], the obesity paradox has been recently observed in trauma patients in two large cohorts [3, 11]. Although several hypotheses have been proposed to explain the obesity paradox, it is unknown whether the protective effect of higher body mass index (BMI) is caused by less severe injuries or an increased injury tolerance. This analysis is limited, at least in part, by the difficulty in comparing trauma severity across multiple organs. The American Association for the Surgery of Trauma (AAST) has developed an anatomical-based severity grading system for providers to communicate renal injury which can objectively assess renal trauma severity across potential groups of interest [12].

We sought to examine the relationship between BMI categories as defined by the World Health Organization (WHO) and outcomes of injury grade and in-hospital mortality in patients with kidney trauma using a large dataset. Understanding the role of BMI in trauma morbidity and mortality, in addition to the mechanism by which its effects are mediated, aids risk stratification and clinical decision making. We hypothesize that being overweight and obese does not improve survival when controlling for AAST kidney injury grade.

Material and methods

Data source and study design

The National Trauma Data Bank® (NTDB) is a large US and Canadian trauma registry that includes comprehensive injury data [13]. We performed a retrospective cohort using NTDB data between 2013 and 2016, and following the STROBE guidelines for observational studies [14]. Institutional review board exemption was provided given that all data were de-identified.

Study population and measurements

We identified patients age 18 or older with renal trauma using ICD-9 codes (866.01; 866.02; 866.03; 866.11; 866.12; 866.13) or ICD-10 codes (S37.01-S37.019; S37.02-S37.029; S37.03-S37.039; S37.04-S37.049; S37.05-S37.059; S37.06-S37.069) (n = 43,166). We used methods described by Moore et al. to convert the Abbreviated Injury Scale to AAST grade [12, 15, 16]. We excluded patients that did not map to a specific AAST grade (n = 23,931) and patients who died in the field or in the emergency department (ED) (n = 846). BMI was classified according to the WHO categories (underweight <18.5; normal weight 18.5–24.9; overweight 25.0–29.9; class I obesity 30.0–34.9; class II obesity 35.0–39.9; class III obesity $\geq$ 40). We excluded patients with incomplete data (n = 2646) and outliers of weight and height using cutoff points from published literature (weight <30 kg or > 600 kg; height < 80 cm or > 250 cm, n = 562) [3]. For each case, we collected: age, sex, race, mechanism of injury (blunt vs penetrating), Injury Severity Score (ISS), pulse at arrival to ED, hypotension (systolic blood pressure < 90) at arrival to ED, initial Glasgow Coma Scale (GCS), trauma center level, intensive care unit (ICU) admission, ventilator use, blood transfusion, angioembolization use, length of stay (LOS), ICU LOS, ventilator days, comorbidities, complications, and hospital disposition. Low-grade renal trauma (LGRT) was defined as AAST grades I–II, whereas high-grade renal trauma (HGRT) was AAST grades III–V. To identify if a patient underwent surgery, we used ICD-9 codes (55.51; 55.52; 55.53; 55.54; 55.4) and ICD-10 codes (0TT00ZZ; 0TT04ZZ; 0TT10ZZ; 0TT14ZZ; 0TT20ZZ; 0TT24ZZ;

0TT30ZZ; 0TT40ZZ; 0TB10ZZ; 0TB00ZZ; 1PC89LB) for nephrectomy and ICD-9 codes (55.81; 55.86; 55.87; 55.89) and ICD-10 codes (0TQ00ZZ; 0TQ03ZZ; 0TQ04ZZ; 0TQ07ZZ; 0TQ08ZZ, 0TQ10ZZ; 0TQ13ZZ; 0TQ14ZZ; 0TQ17ZZ; 0TQ18ZZ; 0TQ30ZZ; 0TQ33ZZ; 0TQ34ZZ; 0TQ37ZZ; 0TQ38ZZ; 0TQ40ZZ; 0TQ43ZZ; 0TQ44ZZ; 0TQ47ZZ; 0TQ48ZZ) for nephrorrhaphy.

Statistical analysis

Descriptive statistics were reported as frequencies and percentages. Continuous variables were assessed for normality and reported as medians and interquartile ranges (IQR) or means and standard deviations (SD) accordingly. Mann–Whitney U or student-t test and Chi-square tests were used to compare continuous and categorical variables, respectively.

Multiple regression analysis was done to examine the association between BMI categories and HGRT with a priori adjustment for age, sex, mechanism of injury, and ISS. This was done using Poisson regression with robust SE reporting, since outcome (HGRT) was a common event (52%) and logistic regression would overestimate the risk ratio (RR) [17]. Subgroup analysis by mechanism of injury (blunt vs penetrating) was performed. Penetrating injuries were further classified as gunshot vs stab wounds. A sensitivity analysis performed by defining HGRT as AAST grades IV–V exclusively did not yield any material changes in our findings.

We used logistic regression to assess the adjusted odds ratios (OR) for inpatient mortality across BMI categories. Following published guidelines on studying mortality using NTDB, we controlled for age, sex, anatomical severity (ISS, AAST grade, initial GCS), physiological severity (hypotension, pulse, transfusion), mechanism of injury, presence of traumatic brain injury, and trauma center level [18]. The final model also controlled for comorbidities (bleeding disorder, cirrhosis, and disseminated cancer), and undergoing any intervention (nephrectomy, nephrorrhaphy, or angioembolization), chosen according to their significance levels ($p < 0.1$ in backward selection).

For our secondary analysis, we fit multiple logistic regression models using backward stepwise selection to assess the association between BMI categories and the odds of undergoing any intervention, nephrectomy, nephrorrhaphy, or angioembolization. We also used logistic regression to assess the adjusted OR of developing acute kidney injury (AKI) or cardiovascular events (composite outcome defined as myocardial infarction, cardiac arrest, pulmonary embolism, or cerebrovascular accident/stroke due to low outcome rate of individual components) across BMI categories. Covariates for AKI and cardiovascular events were chosen based on literature review and included age, ISS, GCS, hypotension, pulse, mechanism of injury, AAST grade, nephrectomy, sepsis, and comorbidities (alcohol abuse, hypertension requiring medication, congestive heart failure, diabetes mellitus, chronic kidney disease, previous myocardial infarction, angina within the past 30 days, history of stroke) [19–21]. Finally, we assessed the following continuous variables across BMI categories using multiple linear regression models for patients who survived to discharge: LOS, ICU LOS, and ventilator days. Models' fit was assessed using the Pearson goodness-of-fit test.

Due to limited ability to adjust for other organ injuries while assessing mortality and secondary outcomes, we performed a sensitivity analysis by considering only patients with isolated renal trauma. All statistical analysis was performed using Stata® version 16.1 and with a $p < 0.05$ considered significant.

Results

Our final cohort consisted of 15,181 renal trauma patients. Median age was 33 years (IQR 24–52), and 11,450 (75.4%) were males. The most common BMI category was normal weight (35.2%), followed by overweight (32.7%), class I obesity (17.1%), class II obesity (6.8%), class III obesity (5.6%), and underweight (2.4%) (Table 1).

Injury grade

Overweight and obese patients had significantly lower proportions of HGRT (between 40.5 and 52.3%) compared to normal weight (56.9%) (Table 1). In the adjusted analysis, increasing BMI above normal was associated with a progressive decrease in risk of HGRT (Fig. 1) compared to normal weight (overweight: RR 0.94, CI 0.90–0.97, $p < 0.001$; class I obesity: RR 0.86, CI 0.82–0.89, $p < 0.001$; class II obesity: RR 0.86, CI 0.81–0.92, $p < 0.001$; class III obesity: RR 0.75, CI 0.69–0.82, $p < 0.001$). Underweight was not associated with risk of HGRT (RR 1.02, CI 0.93–1.11, $p = 0.714$). In subgroup analysis by mechanism of injury, the relationship between BMI and HGRT remained in blunt trauma but not in penetrating trauma (Table 2). Further subclassification of penetrating trauma did not demonstrate any association in gunshot or stab wounds ("Appendix in Table 5").

In-hospital mortality

There were 817 (5.38%) in-hospital mortalities within the cohort. Stratified by BMI, mortality was lowest in individuals with normal weight (4.5%) and highest in class III obese (7%) (Table 1). After adjusting for covariates (including AAST grade), overweight, class I, and class II

Table 1 Renal trauma patient and injury characteristics

	Underweight	*p*	Normal Weight	Overweight	*p*	Class I obesity	*p*	Class II obesity	*p*	Class III obesity	*p*
N (%)	370 (2.4)		5343 (35.2)	4970 (32.7)		2600 (17.1)		1043 (6.8)		855 (5.6)	
Age	25 (21–39)	0.0004	28 (22–43)	35 (25–53)	<0.0001	41 (27–57)	<0.0001	42 (28–57)	<0.0001	38 (27–52)	<0.0001
Sex (Male)	218 (58.9)	<0.001	3964 (74.2)	3964 (79.8)	<0.001	1968 (75.8)	0.134	778 (74.6)	0.786	558 (65.3)	<0.001
Blunt	311 (84)	0.87	4498 (84.3)	4202 (84.7)	0.613	2234 (86.1)	0.041	897 (86.1)	0.142	744 (87)	0.046
AAST grade		0.712			<0.001		<0.001		<0.001		< 0.001
I	64 (17.3)		949 (17.7)	1112 (22.3)		747 (28.7)		304 (29.1)		264 (30.8)	
II	86 (23.2)		1351 (25.2)	1255 (25.2)		653 (25.1)		251 (24)		244 (28.5)	
III	120 (32.4)		1552 (29.1)	1391 (27.9)		657 (25.2)		285 (27.3)		201 (23.5)	
IV	75 (20.2)		1135 (21.2)	912 (18.3)		410 (15.7)		145 (13.9)		100 (11.7)	
V	25 (6.7)		356 (6.6)	300 (6)		133 (5.1)		58 (5.5)		46 (5.38)	
HGRT	220 (59.4)	0.346	3043 (56.9)	2603 (52.3)	<0.001	1200 (46.1)	<0.001	488 (46.7)	<0.001	347 (40.5)	<0.001
ISS	22 (14–33)	0.157	20.5 (13–29)	21 (14–29)	0.024	21 (13–29)	0.733	20 (13–29)	0.739	21 (13–29)	0.603
GCS	15 (14–15)	0.018	15 (14–15)	15 (14–15)	0.0015	15 (14–15)	0.934	15 (14–15)	0.188	15 (14–15)	0.908
Traumatic brain injury	102 (27.6)	0.045	1229 (23)	1253 (25.2)	0.009	590 (22.7)	0.758	217 (20.8)	0.121	198 (23.2)	0.92
Pulse rate	97.4 (23.1)	0.003	93.7 (23)	95.8 (23.2)	<0.0001	97.4 (23.7)	<0.0001	100.4 (24.1)	<0.0001	100.9 (23.7)	<0.0001
Hypotension	25 (6.7)	0.458	418 (7.8)	476 (9.6)	0.002	228 (8.8)	0.148	88 (8.4)	0.502	82 (9.6)	0.078
Transfusion	160 (43.2)	0.66	2248 (42.1)	2242 (45.1)	0.002	1164 (44.8)	0.023	465 (44.6)	0.134	389 (45.5)	0.06
Bleeding disorder	3 (0.8)	0.23	86 (1.6)	130 (2.6)	<0.001	90 (3.5)	<0.001	33 (3.2)	0.001	23 (2.7)	0.026
Diabetes Mellitus	6 (1.6)	0.313	131 (2.4)	263 (5.3)	<0.001	226 (8.7)	<0.001	154 (14.8)	<0.001	120 (14)	<0.001
Cirrhosis	0	0.99	14 (0.3)	11 (0.2)	0.675	9 (0.4)	0.513	6 (0.6)	0.122	6 (0.7)	0.047
Disseminated cancer	0	0.99	4 (0.07)	6 (0.1)	0.536	3 (0.1)	0.69	3 (0.3)	0.091	2 (0.23)	0.196
Chronic kidney disease	3 (0.8)	0.08	13 (0.2)	19 (0.4)	0.205	11 (0.4)	0.171	7 (0.7)	0.033	3 (0.4)	0.476
Congestive heart failure	1 (0.3)	0.99	17 (0.3)	39 (0.8)	0.002	27 (1.04)	<0.001	10 (0.9)	0.004	9 (1.1)	0.006
Previous cardiovascular event	3 (0.8)	0.743	37 (0.7)	66 (1.3)	0.001	26 (1)	0.147	11 (1.1)	0.215	9 (1.1)	0.255
Level I trauma center	158 (42.7)	0.482	2382 (44.6)	2247 (45.2)	0.521	1202 (46.2)	0.166	475 (45.5)	0.568	423 (49.5)	0.008
Intervention	33 (8.9)	0.455	541 (10.1)	511 (10.2)	0.793	210 (8.1)	0.003	80 (7.6)	0.014	59 (6.9)	0.003
Nephrectomy	22 (5.9)	0.448	373 (6.9)	332 (6.6)	0.545	166 (6.3)	0.321	49 (4.7)	0.007	40 (4.6)	0.012
Nephrorrhaphy	9 (2.4)	0.608	109 (2)	125 (2.5)	0.106	36 (1.3)	0.041	19 (1.8)	0.645	14 (1.6)	0.433
Angioembolization	2 (0.5)	0.184	72 (1.4)	72 (1.4)	0.662	16 (0.6)	0.003	14 (1.3)	0.989	5 (0.6)	0.062
Mortality	24 (6.5)	0.081	241 (4.5)	289 (5.8)	0.003	133 (5.1)	0.232	70 (6.7)	0.003	60 (7)	0.002

Reference category for all *p* values

All continuous variables are expressed as median (IQR) except for pulse rate as mean (standard deviation)

All categorical variables are expressed as frequency (%)

Including previous myocardial infarction, cerebrovascular accident/stroke

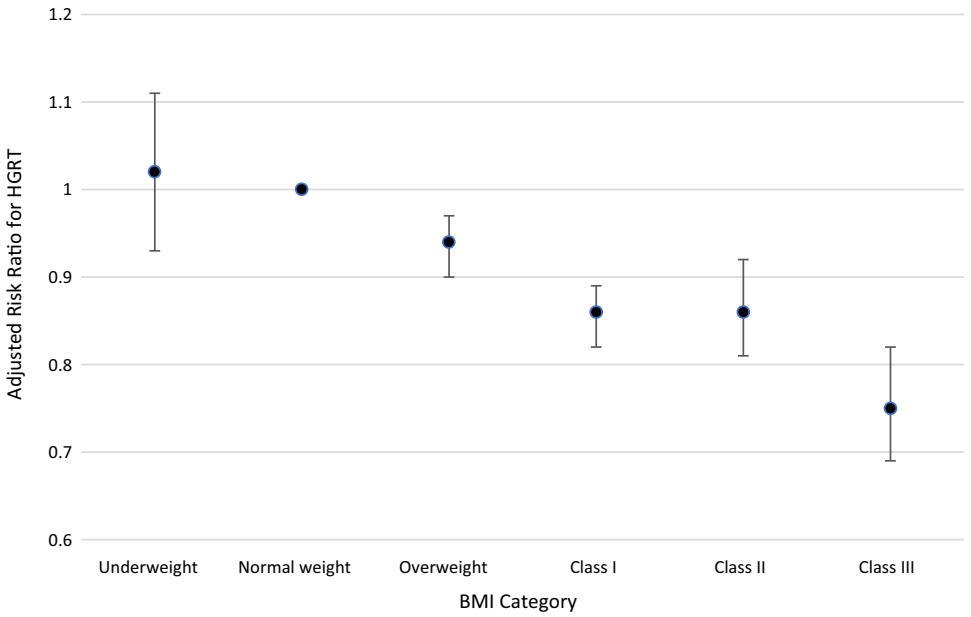

Fig. 1 Poisson regression analysis of high-grade renal trauma (HGRT) risk stratified by BMI category

Table 2 Subgroup analysis depicting the adjusted risk ratio (RR) for the association between BMI and HGRT by mechanism of injury

BMI category	Mechanism of injury					
	Overall		Blunt		Penetrating	
	RR (95% CI)	*p*	RR (95% CI)	*p*	RR (95% CI)	*p*
Underweight	1.02 (0.93–1.11)	0.714	1.01 (0.92–1.12)	0.7	0.99 (0.85–1.14)	0.915
Normal weight	Reference		Reference		Reference	
Overweight	0.94 (0.9–0.97)	<0.001	0.91 (0.88–0.95)	<0.001	0.99 (0.94–1.05)	0.989
Class I obesity	0.86 (0.82–0.89)	<0.001	0.82 (0.78–0.87)	<0.001	0.97 (0.90–1.04)	0.498
Class II obesity	0.87 (0.81–0.92)	<0.001	0.82 (0.75–0.89)	<0.001	1.04 (0.94–1.14)	0.387
Class III obesity	0.75 (0.69–0.82)	<0.001	0.71 (0.64–0.78)	<0.001	0.91 (0.81–1.04)	0.184

Table 3 Regression analysis of in-hospital mortality odds stratified by BMI categories

BMI category	Overall		HGRT		non-HGRT	
	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*
Underweight	1.67 (1.01–2.75)	0.047	0.89 (0.43–1.85)	0.76	4.4 (2.09–9.11)	< 0.001
Normal weight	Reference		Reference		Reference	
Overweight	1.02 (0.83–1.25)	0.841	0.99 (0.77–1.29)	0.976	1.02 (0.73–1.43)	0.885
Class I obesity	0.92 (0.71–1.19)	0.524	0.94 (0.67–1.31)	0.706	0.87 (0.58–1.29)	0.485
Class II obesity	1.38 (0.99–1.91)	0.053	1.54 (1.02–2.32)	0.041	1.15 (0.67–1.98)	0.613
Class III obesity	1.46 (1.03–2.06)	0.034	2.23 (1.43–3.48)	0.001	0.84 (0.48–1.47)	0.55

Table 4 Adjusted analysis of clinical outcomes of adult renal trauma patients stratified by BMI

	Normal weight	Underweight	Overweight	Class I obesity	Class II obesity	Class III obesity
Any intervention						
OR (95% CI)	Reference	0.86 (0.54–1.36)	1.07 (0.91–1.26)	0.86 (0.69–1.06)	0.84 (0.62–1.14)	0.77 (0.54–1.10)
p		0.528	0.367	0.174	0.287	0.165
Nephrectomy						
OR (95% CI)	Reference	0.88 (0.51–1.50)	1.05 (0.87–1.26)	1.20 (0.95–1.52)	0.87 (0.60–1.25)	0.94 (0.62–1.41)
p		0.645	0.603	0.119	0.465	0.769
Nephrorrhaphy						
OR (95% CI)	Reference	1.39 (0.69–2.79)	1.43 (1.09–1.87)	0.88 (0.59–1.30)	1.20 (0.72–1.99)	1.04 (0.58–1.88)
p		0.353	0.008	0.526	0.463	0.878
Angioembolization						
OR (95% CI)	Reference	0.41 (0.09–1.71)	0.97 (0.69–1.37)	0.39 (0.22–0.70)	0.99 (0.54–1.80)	0.46 (0.18–1.16)
p		0.223	0.88	0.002	0.975	0.104
AKI						
OR (95% CI)	Reference	1.17 (0.56–2.43)	1.64 (1.26–2.13)	1.94 (1.44–2.60)	2.14 (1.47–3.13)	3.10 (2.13–4.51)
p		0.661	<0.001	<0.001	< 0.001	<0.001
Cardiovascular events						
OR (95% CI)	Reference	1.07 (0.57–1.98)	1.14 (0.91–1.44)	2.01 (1.58–2.55)	1.64 (1.18–2.28)	2.26 (1.62–3.15)
p		0.827	0.227	<0.001	0.003	<0.001

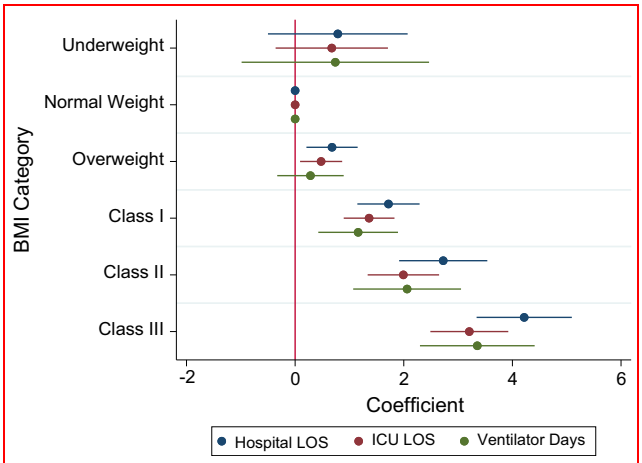

Fig. 2 Regression analysis comparing total hospital LOS, ICU LOS, and ventilator days across BMI categories

obesity were not protective against mortality, whereas underweight and class III obesity were associated with increased odds of mortality (Table 3).

Secondary outcomes

On adjusted analysis, BMI was not associated with the odds of receiving an intervention or undergoing nephrectomy (Table 4). There was a stepwise increase in the odds of developing AKI with increasing BMI categories, and all obese groups had elevated odds of developing cardiovascular events compared to normal weight patients (Table 4). Furthermore, there was a stepwise increase in total hospital LOS, ICU LOS, and ventilator days among patients who survived to discharge with each successive increase in BMI group above normal weight (Fig. 2). Compared to patients with normal weight, overweight, class I obese, class II obese, and class III obese patients spent 0.68 (CI 0.20–1.15, $p = 0.005$), 1.72 (CI 1.14–2.29, $p < 0.001$), 2.72 (CI 1.91–3.53, $p < 0.001$), and 4.21 (CI 3.34–5.09, $p < 0.001$) more days on average in total hospital stay, respectively (Fig. 2). They also spent 0.47 (CI 0.09–0.86, $p = 0.016$), 1.36 (CI 0.89–1.82, $p < 0.001$), 1.99 (CI 1.33–2.65, $p < 0.001$), and 3.2 (CI 2.48–3.92, $p < 0.001$) more days on average in ICU, and 0.28 (CI − 0.32–0.89, $p = 0.365$), 1.16 (CI 0.42–1.89, $p = 0.002$), 2.06 (1.06–3.05, $p < 0.001$), and 3.35 (CI 2.29–4.40, $p < 0.001$) more days on average on ventilator.

Sensitivity analysis

There were 1323 (8.7%) patients with isolated renal trauma. Sensitivity analysis for in-hospital mortality and secondary outcomes of morbidity was not qualitatively different from our main results when we only considered these patients ("Appendices in Table 6 and Figure 3"); however, the low number of patients and outcome events in

each BMI ("Appendix in Table 7") limited the power to obtain statistical significance in some instances.

Discussion

We report on the impact of BMI on renal trauma grade and outcome. We found increasing BMI above normal to progressively decrease the risk of HGRT. This relationship applied only in blunt trauma and not in penetrating trauma. When accounting for covariates, only extremes of BMI (underweight and class III obesity) were associated with increased in-hospital mortality. Increasing BMI was not associated with the risk of undergoing any intervention, or risk of nephrectomy, but was associated with increased AKI, cardiovascular events, longer total hospital LOS, ICU LOS, and ventilator days.

This study expands on the role of BMI in trauma outcomes reported in the literature. Arbabi et al. [22] studied crash injuries in relation to biomechanical factors and found that overweight patients had the least severe abdominal injury. The protection was attributed to increased insulating tissue by extra adiposity. Similar findings were demonstrated by Stein et al. where multivariate analysis showed higher BMI to be associated with lower risk of pelvic fracture in near-side lateral motor vehicle collisions [23]. The protective effect was assumed to be due to a cushion effect of increased soft tissue. Wang et al. [24] found increased subcutaneous fat depth to be associated with decreased injury severity to the abdominal region. In a population-based cohort by Schott et al., hip fracture risk was reduced by 40% for a standard deviation increase in fat mass, while changes in lean body mass did not affect fracture rates [25]. Furthermore, Fu et al. found that obese patients with blunt abdominal trauma sustained less gastrointestinal tract injuries and associated surgery [26]. These reports support the hypothesis that fat mass could aid better tolerating mechanical force incurred in trauma.

We postulate that the protective effect of higher BMI depicted in our study is due to extra adipose tissue acting as a shock absorber, thereby reducing damage to vital organs. An important distinction is that the risk of HGRT was not altered in cases of penetrating trauma. It has been hypothesized that acceleration/deceleration injuries from blunt trauma injure the kidney at natural weak points [27]. Thus, blunt trauma force could be less transmitted to those weak points if larger nearby fat surface area or thickness is present. On the other hand, injury in penetrating wounds results from an object's trajectory through renal parenchyma, and not related to diffuse pressure exerted around the kidney [27].

In this study, patients with class III obesity had increased adjusted mortality odds, which is in line with prior literature [28]. This could be explained by increased morbidity in these patients, who had the longest hospital LOS, ICU LOS, and ventilator days. Underweight was also associated with increased mortality. Similar findings were reported in general trauma patients [3]. ICU admission is associated with a state of malnutrition; thus, depleted energy stores and poor immune function probably contribute to mortality risk in this group [29].

Studies reporting the obesity paradox in trauma proposed several mechanisms to explain the counterintuitive relation between higher than normal BMI and improved survival. These include: 1. upregulation of cytokines produced by adipose tissue, especially leptin, which alter susceptibility to infection and toxicity of proinflammatory stimuli [30]; 2. visceral triglyceride saturation interferes with triglyceride interaction and lipolysis, reducing lipotoxic systemic injury and organ failure [31]; 3. presence of high nutritional reserves enable tolerance to malnutrition states associated with ICU admission [29, 32]; 4. shortcomings of BMI in differentiating adiposity from muscle mass, highlighted by studies that found that the obesity paradox disappeared when using alternative adiposity measures (like waist-to-hip ratio) [33, 34]. We hypothesize that studies reporting obesity paradox in trauma missed adjusting for an objective and accurate measure comparing injury grade. In our present study, this was mitigated by controlling for AAST renal injury grade.

Increasing BMI demonstrated a linear relationship with AKI, LOS, ICU LOS, and ventilator days. Obese patients were also at higher risk of cardiovascular events. These patients are exposed to a chronic inflammatory state and high level of baseline pro-inflammatory cytokines which could contribute to higher morbidity [35–37]. The findings are consistent with other studies of trauma patients [3, 28, 38]. Obese patients have several respiratory complications, including reduced lung volumes, compliance, and gas exchange, leading to difficulty weaning from mechanical ventilation [39].

Conservative management for renal trauma is increasingly popularized aiming to maximize renal salvage [40–42]. Our findings support this trend in patients with abnormal BMI, who are at greater operative risk due to the increased morbidity demonstrated. The increased mortality observed at BMI extremes does not necessarily warrant a more aggressive management approach, since the cause of death in these patients is not presumed to be directly due to their kidney trauma.

The study limitations merit mention. The NTDB has several inherent limitations as it is not a population based dataset, thus may not be representative of all trauma hospitals. Excluded cases with missing data (mostly due to

cases that did not map to any AAST grade) could have introduced some selection bias. Other limitations include missing data and possible coding errors. However, the large sample size of this cohort is likely resistant to these biases. Although we controlled for many covariates in our modeling adhering to published guidelines, there could be unknown or unmeasured confounders not accounted for. The absence of laboratory-specific data hindered our ability to analyze valuable factors such as serum inflammatory markers. There was limited granularity in several variables (such as renal function data and cause of death, and detailed circumstances of injury beyond blunt vs penetrating, such as crash severity if motor vehicle collision and velocity impact) which could have further informed our analysis. Finally, there was no information on the long-term outcomes of renal trauma patients.

Increasing BMI above normal decreases the risk of HGRT in blunt trauma. The obesity paradox does not exist in kidney trauma when injury grade is accounted for. Extremes of weight in both directions increase the risk of mortality in these patients. Further prospective studies are needed to confirm these findings across different types of trauma.

Appendix

See Fig. 3.

See Tables 5, 6, 7.

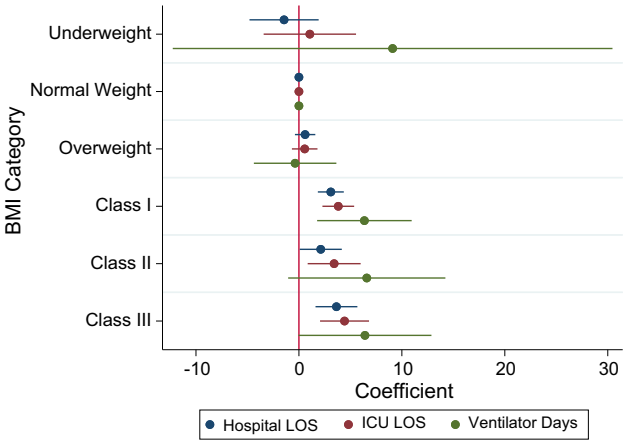

Fig. 3 Regression analysis comparing total hospital LOS, ICU LOS, and ventilator days across BMI categories in sub-population of patients with isolated kidney trauma

Table 5 Subgroup analysis depicting the adjusted risk ratio (RR) for the association between body mass index (BMI) and high-grade renal trauma in penetrating injury classified as stab versus gunshot wounds

BMI category	Penetrating injury			
	Stab wound		Gunshot wound	
	RR (95% CI)	*p*	RR (95% CI)	*p*
Underweight	1.37 (0.97–1.94)	0.067	0.98 (0.84–1.14)	0.839
Normal weight	Reference		Reference	
Overweight	1.06 (0.91–1.23)	0.411	0.98 (0.92–1.03)	0.528
Class I obesity	1.01 (0.79–1.29)	0.918	0.97 (0.90–1.05)	0.565
Class II obesity	1.14 (0.88–1.49)	0.306	0.98 (0.88–1.09)	0.782
Class III obesity	0.72 (0.44–1.17)	0.193	0.93 (0.81–1.05)	0.270

Table 6 Sensitivity analysis for clinical outcomes using sub-population of patients with isolated kidney trauma

BMI category	Mortality		AKI		Cardiovascular events	
	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*
Underweight	(no observations)		(no observations)		(no observations)	
Normal weight	Reference		Reference		Reference	
Overweight	0.71 (0.18–2.76)	0.623	2.34 (0.62–8.8)	0.207	0.91 (0.29–2.89)	0.882
Class I obesity	0.67 (0.11–4.06)	0.67	4.71 (1.14–19.3)	0.031	2.15 (0.68–6.78)	0.188
Class II obesity	2.43 (0.36–16.1)	0.358	19.4 (4.08–92.6)	<0.001	(no observations)	
Class III obesity	5.12 (1.10–23.7)	0.036	1.97 (0.26–14.7)	0.508	7.7 (2.01–29.4)	0.003

Table 7 BMI category and clinical outcomes event distribution in sub-population of patients with isolated kidney trauma

BMI category	Underweight	Normal weight	Overweight	Class I obesity	Class II obesity	Class III obesity
N (%)	20 (1.5)	551 (41.6)	417 (31.5)	206 (15.5)	64 (4.8)	65 (4.9)
Mortality	0	6 (1.1)	7 (1.6)	3 (1.4)	3 (4.6)	6 (9.2)
AKI	0	5 (0.9)	8 (1.9)	7 (3.4)	5 (7.8)	2 (3)
Cardiovascular events	0	7 (1.2)	8 (1.9)	9 (4.3)	0	6 (9.2)

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Declarations

Conflict of interest All authors declare no conflict of interests.

Informed consent Formal ethical approval was not individually sought as all data were de-identified.

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