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Abstract

Objectives: Although traumatic injuries to the superior mesenteric vein (SMV), portal vein (PV), and hepatic vein (HV) are rare, their impact is significant. Small single center reports estimate mortality rates ranging from 29% to 100%. Our aim is to elucidate the incidence and outcomes associated with each injury due to unique anatomic positioning and varied tolerance of ligation. We hypothesize that SMV injury is associated with a lower risk of mortality compared to HV and PV injury in adult trauma patients. **Methods:** The Trauma Quality Improvement Program database (2010-2016) was queried for patients with injury to either the SMV, PV, or HV. A multivariable logistic regression model was used for analysis. **Results:** From 1,403,466 patients, 966 (0.07%) had a single major hepatoportal venous injury with 460 (47.6%) involving the SMV, 281 (29.1%) involving the PV, and 225 (23.3%) involving the HV. There was no difference in the percentage of patients undergoing repair or ligation between SMV, PV, and HV injuries ($P > .05$). Compared to those with PV and HV injuries, patients with SMV injury had a higher rate of concurrent bowel resection (38.5% vs 12.1% vs 7.6%, $P < .001$) and lower mortality (33.3% vs 45.9% vs 49.3%, $P < .01$). After controlling for covariates, traumatic SMV injury increased the risk of mortality (odds ratio [OR] 1.59, confidence interval [CI] = 1.00-2.54, $P = .05$) in adult trauma patients; however, this was less than PV injury (OR = 2.77, CI = 1.56-4.93, $P = .001$) and HV injury (OR = 2.70, CI = 1.46-4.99, $P = .002$). **Conclusion:** Traumatic SMV injury had a lower rate of mortality compared to injuries of the HV and PV. SMV injury increased the risk of mortality by 60% in adult trauma patients, whereas PV and HV injuries nearly tripled the risk of mortality.

Keywords

hepatoportal, portal vein, superior mesenteric vein, hepatic vein, trauma

Introduction

Traumatic superior mesenteric vein (SMV), portal vein (PV), and hepatic vein (HV) injuries represent rare yet devastating injuries. Mortality rates are high for major hepatoportal venous trauma, ranging from 29% to 83% for SMV,¹⁻¹⁴ 50% to 100% for PV,⁹⁻²¹ and 50% to 100% for HV injuries^{9,22-27} in mostly single institution studies. Most deaths are a result of exsanguination leading to hemorrhagic shock, with many patients dying prior to arrival to the hospital.²⁷ For patients who undergo operative exploration, survival is often difficult to achieve secondary to the complexity associated with access and control of these injuries with some series reporting 100% intraoperative mortality.^{11,13,21} Investigators have reported that mortality of hepatoportal venous injuries is associated with initial systolic blood pressure (SBP), injury severity score (ISS), transfusion requirements, and presence of additional injuries.^{3, 9,13,28}

SMV, PV, and HV have unique anatomic positioning and varied tolerance of ligation. As an example, while HVs are one of the most difficult abdominal veins to expose,

ligation is often well tolerated because of the venous drainage provided by the remaining two HVs.^{29,30} SMV injuries are often easier to locate by exposing the vessel at the root of the mesocolon, and prior studies have shown that SMV ligation leads to less bowel ischemia and portal hypertension compared to PV ligation.^{2,12,31} Sirinek et al⁵ reported that mortality of patients with SMV, PV, and HV injuries was 25%, 100%, and 60% in a single institution case series. We hypothesize that SMV injury is associated with lower risk of mortality compared to PV and HV injury in adult trauma patients.

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Methods

This work was deemed exempt by the institutional review board of the University of California, Irvine, as it involved a deidentified national database. This study was a retrospective analysis using the Trauma Quality Improvement Program (TQIP) database, which currently includes over 850 trauma centers from all 50 states. The TQIP database was queried from January 2010 to December 2016 to identify patients aged 18 years and older. Patients with a single major hepatoportal venous injury, defined as either a SMV, PV, or HV injury, were identified using the *International Classification of Diseases (ICD) Version-9 diagnosis codes*: 902.31 for SMV, 902.33 for PV, and 902.11 for HV. Patients with injuries to more than one major hepatoportal vein were excluded. These 3 groups were then compared.

Patient demographic information including age, gender, body mass index (BMI), and prehospital comorbidities was collected as well as injury profile including lowest SBP within 24 hours, severe (grade > 3) abbreviated injury scale (AIS) for body region, and ISS. Concomitant vascular and organ injuries associated with each vessel injury were also compared. The primary outcome was in-hospital mortality. Secondary outcomes included total hospital length of stay (LOS), intensive care unit LOS, ventilator days, requirement of exploratory laparotomy or bowel resection, blood product transfusion, and in-hospital complications. Additionally, percent of patients with each injury undergoing venous ligation versus surgical repair was classified using *ICD* procedure codes. Rates of mortality and bowel resection associated with venous ligation versus surgical repair were compared. Descriptive statistics were performed for all variables. Continuous data were reported as means with standard deviation or medians with interquartile range. The χ^2 and Fisher exact *t* test were used to compare categorical variables between patients with either SMV, PV, or HV injury. Kruskal-Wallis test was used for continuous variables between patients with either SMV, PV, or HV injury.

A univariable logistic regression model was first used to find the magnitude of association between predictor variables and mortality. A hierarchical multivariable logistic regression model was then used with covariates with statistical significance ($P \leq .20$), which included age > 65 years, male gender, hypotension (defined as SBP < 90) or tachycardia (defined as heart rate > 100) on admission, packed red blood cells (PRBCs) ≥ 6 units given in 4 hours, obesity (defined as BMI ≥ 30), peripheral vascular disease, chronic kidney disease, diabetes, smoker, hypertension, chronic obstructive pulmonary disease, cerebrovascular accident, ISS ≥ 25 , AIS > 3 in abdomen, thorax, head, or spine, concomitant injuries to superior mesenteric artery (SMA), inferior mesenteric artery, aorta, inferior vena cava (IVC), liver, spleen, pancreas, stomach, small intestine, colon/rectum, kidney, as well as development of acute kidney injury and acute respiratory distress syndrome. The adjusted risk for mortality was reported with an odds ratio (OR) and 95% confidence intervals (CIs). The reference group used in our analysis included all adult trauma patients to find the adjusted risk of mortality of patients with SMV, PV, or HV injury compared

to those without SMV, PV, or HV injury, respectively. All *P*-values were 2-sided, with a statistical significance level of < .05. All analyses were performed with IBM SPSS Statistics for Windows (version 24, IBM Corp, Armonk, New York).

Results

Demographics and Mortality in Adult Trauma Patients with Major Hepatoportal Venous Injury

From 1,403,466 patients, 966 (0.07%) had a single major hepatoportal venous injury with 460 (47.6%) involving the SMV, 281 (29.1%) involving the PV, and 225 (23.3%) involving the HV. Compared to patients with PV and HV injuries, those with SMV injury were older (39 vs 21 vs 26 years, $P < .001$), with a higher median BMI (29 vs 26 vs 26 kg/m², $P = .01$) and a higher rate of comorbidities such as diabetes mellitus (8.9% vs 3.6% vs 2.7%, $P = .001$) and hypertension (16.5% vs 6.8% vs 5.8%, $P = .001$) (Table 1). Patients with SMV injury had a lower mean ISS (22 vs 25 vs 29, $P < .001$), lower percentage of severe AIS for abdomen (55.2% vs 68.3% vs 76.0%, $P < .001$), and higher median lowest SBP within 24 hours (81 vs 73 vs 72 mm Hg, $P = .01$) compared to those with PV and HV injuries. A higher percentage of SMV injuries were from a blunt mechanism (61.3% vs 44.1% vs 55.1%, $P < .001$) compared to PV and HV injuries.

Mortality of major hepatoportal venous trauma was the following: 33.3% for SMV, 45.9% for PV, and 49.3% for HV. SMV injury was associated with a lower mortality rate compared to PV and HV injuries ($P < .001$).

Risk of Mortality Associated with Major Hepatoportal Venous Trauma

Multivariable analysis of adult trauma patients revealed traumatic SMV injury increased risk of mortality (OR = 1.59, CI = 1.00-2.54, $P = .05$), although less so than PV injury (OR = 2.77, CI = 1.56-4.93, $P = .001$) and HV injury (OR = 2.70, CI = 1.46-4.99, $P = .002$).

Secondary Outcomes in Adult Trauma Patients with Major Hepatoportal Venous Injury

Compared to patients with PV and HV injuries, patients with SMV injury required exploratory laparotomy more frequently (67.4% vs 63.0% vs 56.9%, $P = .03$) as well as bowel resection (38.5% vs 12.1% vs 7.6%, $P < .001$). There was no difference for in-hospital complications between SMV, PV, and HV injuries (Table 2). While patients with SMV injury had longer LOS compared to patients with PV and HV injuries (10 vs 7 vs 5 days, $P = .001$); when comparing only patients who survived hospitalization, the LOS was similar among patients with SMV, HV, and PV injuries ($P > .05$). Patients with PV injury required more median PRBC units within 24 hours compared to those with SMV and HV injuries (18 vs 11 vs 12 units, $P = .01$).

Vein ligation occurred at similar rates with 21.1% of SMV injuries, 18.9% of PV injuries, and 16.4% of HV injuries

Table 1. Demographics of Adult Trauma Patients With Major Hepatoportal Venous Injury.

Characteristics	SMV, n = 460	Portal Vein, n = 281	Hepatic Vein, n = 225	P Value
Age, year, median (IQR)	39 (29)	31 (25)	26 (18)	<.001
Male, n (%)	354 (77.0%)	207 (73.7%)	167 (74.2%)	.54
ISS, mean (SD)	22 (16)	25 (16)	29 (16)	<.001
Lowest SBP within 24 hours, in mm Hg, median (IQR)	81 (36)	73 (34)	72 (41)	.01
BMI, median (IQR)	29 (10)	26 (7)	26 (7)	.01
Comorbidities, n (%)				
Peripheral vascular disease	1 (0.2%)	0 (0.0%)	0 (0.0%)	.58
Congestive heart failure	7 (1.5%)	0 (0.0%)	0 (0.0%)	.02
End-stage renal disease	2 (0.4%)	0 (0.0%)	0 (0.0%)	.33
Smoker	58 (12.6%)	41 (14.6%)	33 (14.7%)	.66
Diabetes	41 (8.9%)	10 (3.6%)	6 (2.7%)	.001
Hypertension	76 (16.5%)	19 (6.8%)	13 (5.8%)	.001
COPD	22 (4.8%)	13 (4.6%)	4 (1.8%)	.14
Cerebrovascular accident	2 (0.4%)	1 (0.4%)	1 (0.4%)	.98
Mechanism of injury, n (%)				
Blunt	282 (61.3%)	124 (44.1%)	124 (55.1%)	<.001
Penetrating	175 (38.0%)	157 (55.9%)	99 (44.0%)	<.001
Others	3 (0.7%)	0 (0.0%)	2 (0.9%)	<.001
AIS (grade > 3), n (%)				
Spine	9 (2.0%)	10 (3.6%)	6 (2.7%)	.41
Thorax	70 (15.2%)	33 (11.7%)	48 (21.3%)	.01
Abdomen	254 (55.2%)	192 (68.3%)	171 (76.0%)	<.001
Lower extremity	12 (2.6%)	4 (1.4%)	7 (3.1%)	.42

Abbreviations: AIS, abbreviated injury scale; BMI, body mass index; COPD, chronic obstructive pulmonary disease; IQR, interquartile range; ISS, injury severity score; SBP, systolic blood pressure; SD, standard deviation; SMV, superior mesenteric vein.

Table 2. Clinical Outcomes in Adult Trauma Patients With Major Hepatoportal Venous Injury.

Outcome	SMV, n = 460	Portal Vein, n = 281	Hepatic Vein, n = 225	P Value
LOS overall, days, median (IQR)	10 (18)	7 (17)	5 (14)	.001
LOS, alive patients only, days, median (IQR)	16 (21)	16 (21)	12 (21)	.25
ICU, days, median (IQR)	6 (12)	5 (11)	5 (9)	.31
Ventilator, days, median (IQR)	3 (10)	3 (8)	3 (6)	.55
PRBC units within 24 hours, median (IQR)	11 (18)	18 (24)	12 (28)	.01
Required ex lap, n (%)	310 (67.4%)	177 (63.0%)	128 (56.9%)	.03
Required bowel resection, n (%)	177 (38.5%)	34 (12.1%)	17 (7.6%)	<.001
Complications, n (%)				
Acute kidney injury	40 (8.7%)	22 (7.8%)	14 (6.2%)	.53
Acute renal failure	1 (0.1%)	0 (0.0%)	0 (0.0%)	.58
Deep vein thrombosis	23 (5.0%)	18 (6.4%)	15 (6.7%)	.60
Pulmonary embolism	13 (2.8%)	9 (3.2%)	5 (2.2%)	.80
ARDS	28 (6.1%)	14 (5.0%)	7 (3.1%)	.25
Pneumonia/VAP	55 (12.0%)	23 (8.2%)	21 (9.3%)	.23
Mortality, n (%)	153 (33.3%)	129 (45.9%)	111 (49.3%)	<.001
Days to death, days, median (IQR)	1 (1)	1 (0)	1 (0)	.38

Abbreviations: ARDS, acute respiratory distress syndrome; ICU, intensive care unit; IQR, interquartile range; LOS, length of stay; PRBC, packed red blood cell; SMV, superior mesenteric vein; VAP, ventilator associated pneumonia.

($P > .05$). Repair was required in 13.7% of patients with SMV injury and was not significantly different than those with PV (12.5%) or HV (16.4%) injury ($P > .05$). For those with SMV or HV injury, repair versus ligation did not significantly change the rates of bowel resection or mortality ($P > .05$). For those with PV injury, ligation resulted in a higher mortality compared to repair (71.4% vs 45.3%, $P = .02$) but did not affect the rate of bowel resection ($P > .05$).

Concomitant Injuries in Adult Trauma Patients with Major Hepatoportal Venous Injury

Compared to patients with PV and HV injuries, patients with SMV injury had a higher incidence of concomitant injuries of the SMA (21.7% vs 3.6% vs 2.7%, $P < .001$), aorta (8.3% vs 3.6% vs 4.4%, $P = .02$), small intestine (52.0% vs 24.9% vs 13.3%, $P < .001$), and colon/rectum (36.5% vs 17.8% vs 12.9%,

Table 3. Concomitant Injuries Associated With Major Hepatoportal Venous Injury in Adult Trauma Patients.

Concomitant injury	SMV, n = 460	Portal Vein, n = 281	Hepatic Vein, n = 225	P Value
Vascular injury, n (%)				
Celiac artery	7 (1.5%)	1 (0.4%)	2 (0.9%)	.31
Superior mesenteric artery	100 (21.7%)	10 (3.6%)	6 (2.7%)	<.001
Inferior mesenteric artery	3 (0.7%)	0 (0.0%)	0 (0.0%)	.19
Aorta	38 (8.3%)	10 (3.6%)	10 (4.4%)	.02
Inferior vena cava	41 (8.9%)	55 (19.6%)	44 (19.6%)	<.001
Organ injury, n (%)				
Liver	143 (31.1%)	217 (77.2%)	192 (85.3%)	<.001
Spleen	103 (22.4%)	56 (19.9%)	52 (23.1%)	.64
Pancreas	68 (14.8%)	41 (14.6%)	11 (4.9%)	<.001
Stomach	59 (12.8%)	44 (15.7%)	14 (6.2%)	.004
Small intestine	239 (52.0%)	70 (24.9%)	30 (13.3%)	<.001
Colon/rectum	168 (36.5%)	50 (17.8%)	29 (12.9%)	<.001
Kidney	68 (14.8%)	48 (17.1%)	34 (15.1%)	.69

Abbreviation: SMV, superior mesenteric vein.

Table 4. Risk of Mortality Associated With Concomitant Injuries in Patients With Single Major Hepatoportal Venous Injury in Adult Trauma Patients.

Concomitant Injury	SMV, n = 460	Portal Vein, n = 281	Hepatic Vein, n = 225
Vascular injury, OR (CI)			
Celiac artery	1.17 (0.78-1.78)	1.16 (0.77-1.76)	1.17 (0.78-1.78)
Superior mesenteric artery	1.61 (1.16-2.24)	1.67 (1.20-2.31)	1.69 (1.22-2.34)
Inferior mesenteric artery	0.61 (0.24-1.59)	0.61 (0.24-1.59)	0.61 (0.24-1.58)
Aorta	1.31 (1.17-1.47)	1.31 (1.17-1.47)	1.31 (1.17-1.47)
Inferior vena cava	2.70 (2.14-3.41)	2.65 (2.10-3.35)	2.63 (2.08-3.32)
Organ injury, OR (CI)			
Liver	0.99 (0.93-1.05)	0.99 (0.93-1.05)	0.99 (0.93-1.05)
Spleen	0.81 (0.76-0.86)	0.81 (0.76-0.86)	0.81 (0.76-0.86)
Pancreas	1.50 (1.15-1.96)	1.49 (1.15-1.95)	1.52 (1.16-1.98)
Stomach	0.96 (0.80-1.15)	0.95 (0.79-1.15)	0.96 (0.80-1.16)
Small intestine	1.13 (1.02-1.26)	1.14 (1.02-1.27)	1.14 (1.03-1.27)
Colon/rectum	0.95 (0.85-1.07)	0.96 (0.86-1.07)	0.96 (0.86-1.07)
Kidney	0.70 (0.65-0.77)	0.70 (0.65-0.77)	0.70 (0.65-0.77)

Abbreviations: CI, confidence interval; OR, odds ratio; SMV, superior mesenteric vein.

$P < .001$), and lower incidence of injuries to the IVC (8.9% vs 19.6% vs 19.6%, $P < .001$) and liver (31.1% vs 77.2% vs 85.3%, $P < .001$). Compared to those with SMV and PV injuries, patients with HV injury had a lower incidence of injuries to the pancreas (4.9% vs 14.8% vs 14.6%, $P < .001$) and stomach (6.2% vs 12.8 vs 15.7%, $P = .004$) (Table 3).

Concomitant injuries were included in the multivariable regression model and the concomitant injuries that most significantly increased the risk of mortality was similar among SMV, HV, and PV injuries and included IVC injury, SMA injury, pancreatic injury, aortic injury, and small intestine injury (Table 4).

Discussion

This study is the first to report demographics and patient outcomes on individual major hepatoportal venous traumatic injuries in a large cohort of patients nationwide. The results of this study confirmed our hypothesis that SMV injury is associated

with a lower rate of mortality in adult trauma patients compared to PV and HV injuries. Traumatic SMV, PV, and HV injuries are all independent predictors of mortality. In support of our hypothesis, SMV traumatic injury is associated with a lower risk of mortality as it only increased risk of mortality by 60%, whereas HV and PV injuries nearly tripled the risk of mortality.

The results of our study are consistent with prior case series that have demonstrated traumatic SMV injury to have a lower mortality rate compared to PV and HV injuries.^{5,32} A possible explanation for this is that a lower percentage of patients with SMV injury had IVC injury compared to those with PV and HV injuries in our study and we found IVC injuries to nearly triple the associated risk of mortality. This is further supported by Asensio et al, who demonstrated that of all abdominal venous injuries, IVC injury was associated with the highest mortality (75%-100%).¹⁰ Another possible explanation is that SMV ligation is better tolerated than PV ligation. We found that a similar

proportion of patients with SMV and PV injuries undergo ligation, and prior studies have suggested that SMV ligation is better tolerated. While there is a concern for possible bowel necrosis, Donahue and Strauch³³ found that SMV ligation had less morbidity and mortality compared to PV ligation due to continued venous drainage of the bowel via the inferior mesenteric vein and portosystemic collaterals. In further support, Stone et al¹² found that SMV ligation had a mortality of 67% while PV ligation had a mortality of 88%, and Coimbra et al¹⁴ found that SMV ligation had a mortality of 50% while PV ligation had a mortality of 100%. Our study supports these prior single institution series as we show SMV injury has a similar rate of ligation to PV injury yet lower overall mortality. Additionally, our study showed that the rates of mortality were not significantly different between SMV ligation and repair. This finding can be used to guide surgeons and we recommend consideration of ligation of the SMV as a potential means of salvage in a hemodynamically unstable and exsanguinating patient.

On the other hand, injury to the PV is especially troublesome as it supplies the majority of blood flow to the liver. We found that traumatic PV injury is associated with the highest risk for mortality among hepatoportal venous trauma. This can be expected as the PV has a blood flow rate of 1 L/min, leading to death secondary to exsanguination when injured.¹⁷ This is supported by our study, in which patients with PV injury required 40% more blood transfusions than those with SMV or HV injury. Another possible explanation is that ligation of the PV is less tolerated than ligation of SMV and HV injuries. This finding has been demonstrated in multiple other studies including some series, which demonstrated a mortality of 100% with PV ligation.^{11,18-21} However, Sabat et al recently performed an analysis of the National Trauma Data Bank and found PV ligation had a mortality rate of 59.2% and ligation of PV did not increase the risk of mortality compared to repair on multivariable analysis.¹⁷ Our study found that PV ligation increased the rate of mortality compared to PV repair; however, this was only in a bivariate comparison as the sample size was not large enough to support a multivariable regression model and we could not control for patient and intraoperative factors. Therefore, the higher rate of mortality associated with ligation versus repair of PV in our study may simply reflect selection bias. Currently, not enough evidence exists to support ligation of PV. Future studies are needed to determine what specific factors, such as time to operating room, type of vascular injury, and intraoperative physiology, lead to the ideal setting for repair versus ligation to help lower mortality of this highly lethal injury.

While many studies have reported that the majority of hepatoportal venous injuries are from penetrating mechanisms, we found that just over half of hepatoportal venous injuries were from blunt mechanisms.^{1,10} Coimbra et al's study from 1987 to 2006 showed that SMV and PV injuries occurred from a blunt mechanism only 27% of the time. Additionally, they showed mortality from blunt compared to penetrating trauma for SMV was 57.0% vs 37.5%, with cause of

mortality being hemorrhage in 92% of patients.¹⁴ One potential explanation for the increase in blunt hepatoportal venous trauma is the widespread adoption of massive transfusion protocols (MTPs), which allow for blunt trauma patients who may have previously died from exsanguination prior to reaching the operating room to survive to diagnosis of the hepatoportal venous injury. Most prior reports on hepatoportal venous trauma are before the widespread implementation of MTPs.³⁴ Furthermore, Dente et al³⁵ showed that the implementation of a MTP at their institution increased the survival of blunt trauma patients from 44% to 55% but had no effect on survival of penetrating trauma patients. Additionally, improved trauma systems including shorter prehospital transport times and expeditious access to an operating room may allow for the subset of blunt hepatoportal venous trauma patients who previously would have died at the scene to survive until hospital diagnosis. This is supported by Ball et al³⁶ who demonstrated that shorter hospital time was associated with a 34% increase in total number of abdominal vascular injuries received by their trauma center. Future research is needed focusing on management techniques specific for blunt hepatoportal venous trauma as the incidence is rising and brings unique challenges not present in penetrating vascular trauma.

Limitations to this analysis include those inherent to a retrospective database such as reporting bias, coding error, and missing data. In addition, we are restricted to data fields available in the TQIP database and were unable to assess the exact injury characteristics (ie, American Association for the Surgery of Trauma grade), as well as the specific location of injury (ie, intra versus extra parenchymal HV and retropancreatic SMV, etc) and the patients intraoperative hemodynamic status, which would impact treatment and mortality. Furthermore, information regarding the use of intravascular shunting and concomitant assistance of a vascular and/or hepatobiliary surgeon is not available within TQIP. Finally, TQIP is confined to index hospitalization outcomes, thus no information regarding long-term outcomes are available. Despite these limitations, our study provides a large contemporary experience from trauma centers across the country making the results more generalizable.

Conclusion

Compared to injuries of the PV and HV, traumatic SMV injury is associated with nearly 12% decreased absolute rate of mortality. Furthermore, traumatic PV and HV injuries are associated with a nearly tripled increase in mortality in adult trauma patients, while SMV injury only increased the risk of mortality by 60%. Mortality rates did not differ between SMV ligation and SMV repair, which suggest that SMV ligation can be tolerated in exsanguinating patients where no other options exist. The significant morbidity and mortality we found to be associated with these injuries can help guide clinicians when speaking to patients and their families about treatment strategies and prognosis.


Declaration of Conflicting Interests

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