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Isolated traumatic brain injury in patients with cirrhosis: do different treatment paradigms result in increased mortality?

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Abstract

BACKGROUND: Cirrhosis is associated with increased mortality in trauma, yet its effects on outcomes after traumatic brain injury (TBI) are unclear. We hypothesized that cirrhosis adversely effects mortality and increases complications after TBI.

METHODS: Cirrhotic patients with isolated TBI were matched with noncirrhotic TBI patients in a 3:1 ratio based on age, sex, injury mechanism, and injury severity score at our academic, level 1 trauma center.

RESULTS: Of the 8,748 patients with isolated TBI, 65 patients had concurrent cirrhosis. Cirrhotic patients had increased mortality compared with matched controls (31% vs 17%, $P = .03$) and were less likely to undergo emergent neurosurgical operation (12% vs 25%, $P = .03$). There was no difference in admission Glasgow Coma Score, type of intracranial hemorrhage, length of stay, or complications between the groups.

CONCLUSIONS: Cirrhotic patients have increased mortality after TBI and were less likely to undergo operative intervention. New treatment paradigms may be needed to improve outcomes for cirrhotic patients suffering TBI.

The combination of trauma and liver cirrhosis has been termed a "deadly duo,[1]" with a nearly 3-fold increase in hospital mortality[2] and 5-fold increase in all-cause morbidity compared with patients with trauma alone.[3] In reported series, cirrhotic trauma patients receive more blood transfusions, spend more time on a ventilator and have longer ICU and total lengths of stay[4] compared with matched noncirrhotic trauma patients. Furthermore, the presence of ascites, hyperbilirubinemia, and elevated PT/international normalized ratio (INR) on admission are each independently associated with a more than 50% increase in mortality.[5]

Traumatic brain injury (TBI) is a leading cause of death and disability in the United States.[6] TBI accounts for 50,000 deaths per year, most of which are from older individuals.[7] Currently, cirrhotic patients account for

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approximately 1% of all the trauma admissions[4,8,9] and several factors place cirrhotic patients at high risk for TBI, including fall risk and coagulopathy. Mechanical falls are the most common cause of TBI for all ages[5] and the most common mechanism of injury among cirrhotic patients.[4,5] Coagulopathy of chronic liver disease may result in further progression of intracranial hemorrhage after initial injury.[10] Despite our growing knowledge of TBI, there is a paucity of literature addressing outcomes in cirrhotic patients after head injury. The single publication on this topic using the national trauma database found that mortality in 47 cirrhotic patients with isolated TBI was twice that of matched noncirrhotic patients. In this study, we aim to validate the mortality risk in a larger patient cohort, stratify risk based on cirrhosis severity, and evaluate the effect of cirrhosis on TBI management.

Methods

This study protocol was approved by our institutional review board. All cirrhotic trauma patients admitted to the University of California San Diego, a level I trauma center, from 2000 to 2013 with isolated TBI were identified from the trauma registry. Pre-existing cirrhosis was identified as comorbidity from the trauma registry and is captured if there is written documentation of cirrhosis or end-stage liver disease in the patient's medical record history. Isolated TBI, defined as the presence of intracranial hemorrhage, brain contusion, diffuse axonal injury, or cerebral edema with an Abbreviated Injury Scale (AIS) score ≤ 1 for all other body regions, was identified based on ICD-9 codes 800-801.99, 803.1-803.99, 850-854.19.

Patients with cirrhosis were matched with noncirrhotic TBI patients in a 3:1 ratio based on age (within 10 years), sex, head AIS, injury severity score (ISS) category (0 to 9, 10 to 15, 16 to 24, 25 to 75), and injury mechanism. Cases with alternate mechanisms were used if there were fewer than 3 available matches using all criteria. Data abstracted included demographics, admission vital signs, injury severity score, admission Glasgow Coma Score (GCS), type of intracranial hemorrhage, blood product transfusion, neurosurgical intervention, admission blood alcohol level, length of stay (LOS), complications, and mortality. Emergent neurosurgical operation was defined as craniotomy or craniectomy. Model for End-Stage Liver Disease (MELD) scores were calculated for cirrhotic patients based on admission laboratory values (INR, total bilirubin, creatinine) to determine cirrhosis severity as previously described.[11]

Statistical significance was calculated with chi-square for bivariate categorical variables, Student t test for continuous variables, and ANOVA for multivariate analysis. Statistical significance was defined as $P < .05$. Logistic regression was performed using IBM SPSS Statistics, version 21. Variables were considered for inclusion in the logistic regression model if their bivariate P value was less than .1. Model reduction was performed using forward stepwise regression with criteria for entry set a $P < .05$ and criteria to remove from the model at $P > .01$.

Table 1 Study population demographics

Demographics	n (%)
Total	260
Age (y), mean ± SD	56.05 ± 10.32
Male	208 (80.0)
Mechanism of injury	
Blunt	252 (96.9)
Falls	157 (60.4)
Found down	60 (23.1)
Assault	23 (8.8)
Penetrating	8 (3.1)
Injury severity, median (IQR)	
ISS	17 (16–25)
Head AIS	4 (4–5)
Admission GCS	14 (7–15)

AIS = abbreviated injury scale; GCS = Glasgow Coma Scale; IQR = interquartile range; ISS = injury severity score; SD = standard deviation.

Results

During the 13-year study period, 30,132 patients were admitted to the trauma center. Of those, 8,748 had isolated TBI, and 65 patients (1.4%) had isolated TBI and liver cirrhosis. One hundred ninety-five noncirrhotic TBI patients were matched for a total study population of 260 patients. Demographic data, injury mechanism, and injury severity for the study group are summarized in Table 1.

Characteristics of cirrhotic and matched noncirrhotic patients are shown in Table 2. There was no difference in admission GCS, Head AIS, or percentage of patients with severe head injury (Head AIS >3). In addition, there were no differences in types of intracranial hemorrhage between cirrhotic and noncirrhotic patients, however, cirrhotic patients were less likely to have an associated skull fracture. The coagulation profile of cirrhotic patients was more abnormal than noncirrhotic patients with higher admission INR (1.41 vs 1.06) and lower platelet counts (112 vs 217 $\times$ 10^3 cells/μL, respectively). Of note, blood alcohol levels were similar between groups.

Mortality was significantly higher in cirrhotic patients compared with noncirrhotic patients (31 vs 17%), yet cirrhotic patients were only half as likely to undergo emergent neurosurgical intervention (Table 3). Conversely, cirrhotic patients were more likely to receive blood products during their admission, predominantly consisting of fresh frozen plasma (FFP; 44%), followed by platelets (31%) and packed red blood cells (11%). For cirrhotic patient in which emergent neurosurgical intervention was not pursued, none had an advanced directive stating a