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Effect of alcohol in traumatic brain injury: is it really protective?

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ABSTRACT

Background: Studies have proposed a neuroprotective role for alcohol (ETOH) in traumatic brain injury (TBI). We hypothesized that ETOH intoxication is associated with mortality in patients with severe TBI.

Methods: Version 7.2 of the National Trauma Data Bank (2007–2010) was queried for all patients with isolated blunt severe TBI (Head Abbreviated Injury Score ≥4) and blood ETOH levels recorded on admission. Primary outcome measure was mortality. Multivariate logistic regression analysis was performed to assess factors predicting mortality and in-hospital complications.

Results: A total of 23,983 patients with severe TBI were evaluated of which 22.8% ($n = 5461$) patients tested positive for ETOH intoxication. ETOH-positive patients were more likely to have in-hospital complications ($P = 0.001$) and have a higher mortality rate ($P = 0.01$). ETOH intoxication was an independent predictor for mortality (odds ratio: 1.2, 95% confidence interval: 1.1–2.1, $P = 0.01$) and development of in-hospital complications (odds ratio: 1.3, 95% confidence interval: 1.1–2.8, $P = 0.009$) in patients with isolated severe TBI.

Conclusions: ETOH intoxication is an independent predictor for mortality in patients with severe TBI patients and is associated with higher complication rates. Our results from the National Trauma Data Standards differ from those previously reported. The proposed neuroprotective role of ETOH needs further clarification.

1. Introduction

Each year approximately 1.7 million individuals sustain traumatic brain injury (TBI), which accounts for one third of all injury-related deaths in the United States [1]. Alcohol (ETOH) intoxication is a known risk factor for injury and is frequently associated with development of head injury [2,3]. Studies have highlighted that 25%–50% of patients with head injury are under the influence of ETOH at the time of injury [4,5]. ETOH intoxication is known to be associated with worse outcomes

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after injury however [6–8], its impact on mortality in patients with TBI still remains debatable.

The neuroprotective effects of ETOH after TBI were initially highlighted in animal and laboratory studies [9,10]. In recent years, there have been several clinical studies, which have demonstrated similar beneficial effects of ETOH in patients with moderate and severe TBI [11–18]. Studies have also shown a dose dependent effect of blood ETOH level on survival rates after intracranial injury [17]. However, all studies demonstrating the beneficial effects of ETOH have combined patients with varying severity (moderate and severe) and mechanism (blunt *versus* penetrating) of head injury into a single category. Given the higher mortality rate in patients with penetrating head injury and severe TBI, the true effect of ETOH on patients with TBI still remains unclear. Additionally, the contrasting results from studies demonstrating no association between ETOH intoxication and survival have further raised questions about the protective effect of ETOH in patients with head injury.

The aim of our study was to evaluate the effects of ETOH intoxication on outcomes in patients with blunt isolated severe TBI. We hypothesized that ETOH intoxication is associated with mortality in patients with severe blunt TBI.

2. Methods

This was a retrospective analysis of all trauma patients included in the National Trauma Data Bank (NTDB) version 7.2, which includes patient records from the years 2007–2010. The NTDB is the largest collection of trauma patients in the United States, which comprises of patient records collected from >700 trauma centers. The NTDB is managed and maintained by the American College of Surgeons. This study was reviewed by the University of Arizona, Institutional Review Board and was determined to be exempt from approval.

We included patients aged ≥18 y with isolated severe blunt TBI and measured serum ETOH level on admission. We defined isolated severe TBI as head Abbreviated Injury Scale (AIS) score of ≥4 and with other body region (abdomen, thorax, and extremity) AIS score <3. The measurement of ETOH was qualitative (Yes or No). Patients who were transferred from other institutions, patients with burns, patients who died in the emergency department or within 24 h of hospital admission, and patients with missing variables such as demographics (age, sex), injury severity parameter (head-AIS), ETOH measurement, and discharge disposition were excluded. Additionally, we also excluded patients with chronic history of ETOH using comorbidity code of "alcoholism" as recorded in the NTDB.

The following data points were abstracted from the NTDB: demographics (age and sex) mechanism of injury, vitals on presentation (systolic blood pressure, heart rate, and temperature), Glasgow Coma Scale score on presentation, injury severity parameters (head–AIS, Injury Severity Score), hospital and intensive care unit length of stay (LOS), in-hospital complications, discharge disposition (home, rehabilitation center, and skilled nursing facility), and in-hospital mortality.

The primary outcome measure was in-hospital mortality. The secondary outcome measures were in-hospital complications and hospital LOS. We defined in-hospital complications as infectious complications (pneumonia, urinary tract infections, and sepsis), hematological complications (deep venous thrombosis, pulmonary embolism), and acute respiratory distress syndrome (ARDS).

Data is reported as mean ± standard deviation (SD) for continuous variables, as frequency and proportions for categorical variables, and as median [range] for ordinal variables. The study population was divided into two groups, patients who tested positive for serum ETOH (ETOH-positive) and patients who tested negative for serum ETOH (ETOH-negative). We used Mann–Whitney *U* and Student *t*-tests to identify the differences between the two groups for continuous and ordinal variables. Chi-square test was used to record the differences in the two groups for categorical variables. We then performed a univariate analysis to identify factors associated with mortality and in-hospital complications in patients with isolated severe TBI. Factors with *P* value ≤0.2 were considered to be significant and were used in a multivariate regression model to assess the factors associated with mortality and in-hospital complications. *P* value ≤0.2 on univariate analysis was chosen as significant to include maximum number of factors that could possibly affect our outcome (mortality) and to strengthen our multivariate regression model. In the multivariate regression model, *P* value ≤0.05 was considered significant. Odds ratios and 95% confidence intervals were calculated for each variable in the univariate and multivariate models. All statistical analyses were performed using Statistical Package for Social Sciences (SPSS, Version 20; SPSS, Inc, Chicago, IL).

3. Results

A total of 172,089 patients with TBI were reviewed of which 23,983 patients were included in the analysis. The mean age was 46.3 ± 21.6 y, 66.9% (n = 16,045) were male, median Glasgow Coma Scale (GCS) was 13 [3–15], and median head AIS was 4 [4–5]. A total of 5461 (22.8%) patients tested positive for ETOH intoxication on presentation. ETOH-positive patients were younger (P = 0.001) males (P = 0.01) who were more severely injured (P = 0.01) compared with ETOH-negative patients. There was no difference in GCS score (P = 0.1), mechanism of injury (P = 0.08), and severity of head injury (P = 0.1) between the two groups. A total of 6861 (28.6%) patients were screened for drug toxicology of which 47.1% (n = 3233) had positive drug toxicology. Patients with ETOH positive were more likely to have a positive drug toxicology (P = 0.01). Table 1 demonstrates the demographics of the study population.

ETOH-positive patients were more likely to develop in-hospital complications (P = 0.01) compared with ETOH-negative patients. Pneumonia (5.6%) followed by ARDS (2.8%) was the most common complications in ETOH-positive patients. Table 2 highlights the complications in patients of the study population.

Table 3 demonstrates the outcomes in ETOH-positive and ETOH-negative patients. ETOH-positive patients had a longer hospital (P = 0.02) and intensive care unit (P = 0.04) LOS compared with ETOH-negative patients. Patients with positive ETOH were less likely to discharge home (P = 0.01) compared