THE EFFECTS OF ALCOHOL ON HEAD INJURY IN THE MOTOR VEHICLE CRASH VICTIM

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(Received 19 January 2001; in revised form 17 August 2001; accepted 24 September 2001)

Abstract — The objective of this study was to determine if alcohol potentiates the severity of traumatic brain injury (TBI) in motor vehicle crash (MVC) victims, controlling for crash severity characteristics. Prior studies evaluating effects of alcohol on TBI have not accounted for severity of crash. We evaluated severity of head injury by Marshall score [a classification scale of intracranial pathology on head computed axial tomogram (CT)], and blood-alcohol concentration (BAC), while controlling for crash characteristics [traffic accident deformity score (TAD) and belt use]. Marshall scores were determined from initial CT or autopsy reports, by a neurosurgeon, and were categorized into less severe injury (<3) and more severe (≥3). Logistic regression using this variable as the outcome parameter and crash characteristics, age and BAC as predictors was done, and the odds ratio (OR) and 95% confidence interval (0.95 CI) calculated. Fifty-eight patients were analysed: 41% were BAC positive, 30% had a modified Marshall score of ≥3. Patients with positive BAC were 2.1-fold more likely to have a more severe head injury as measured on CT scan by the Marshall scores. We suggest that alcohol potentiates severity of TBI as determined from head CT among MVC victims. Further research will be needed to substantiate this finding as well as to determine its long-term effect on clinical outcome.

INTRODUCTION

Motor vehicle crash (MVC) injuries are the largest component of injury mortality in the USA. Injuries from motor vehicle crashes are the third leading cause of years of potential life lost, and are estimated to result in >523 000 hospitalizations in the USA each year (Rivera et al., 1997). Alcohol intoxication is found in >40% of individuals who are fatally injured in motor vehicle crashes (US Department of Transportation, 1994). The presence of alcohol increases the probability of involvement in a MVC (Ward et al., 1982). Furthermore, alcohol use has been associated with higher motor vehicle speeds and lower seatbelt use, resulting in overall more severe crashes (Hingson et al., 1996). Researchers have also reported that the presence of alcohol has a potentiating effect on overall injury in MVC (Waller et al., 1986; Pories et al., 1992). In one study, alcohol-involved drivers were 1.7–2.0 times more likely to be seriously or fatally injured (Waller et al., 1986). In another study, alcohol positive MVC victims had Injury Severity Scores (ISS) 86% higher than their alcohol-negative counterparts (Waller et al., 1999). A 1993 study also concluded that fatality risk was 2-fold higher in alcohol-intoxicated MVC patients (Evans and Frick, 1993).

Traumatic brain injury (TBI) accounts for much of the morbidity and mortality seen in both the MVC victims with and without alcohol present. The leading cause of traumatic brain injury is MVC. Investigators noted that alcohol intoxication was present in ~50% of patients who died attention for TBI (Edna, 1982; Brismar et al., 1983; Gurney et al., 1992; Zink et al., 1996). A number of laboratory studies examining alcohol and TBI, using a variety of animal models and species, support the concept that alcohol worsens TBI (Flamm et al., 1977; Albin and Bunegin, 1986; Franco et al., 1988; Zink et al., 1993, 1996, 1998; Zink and Feustel, 1995). While the laboratory data present a strong argument for the potentiating role of alcohol in TBI, clinical evidence has been less clear. Gurney et al. (1992) showed that intoxicated subjects were more likely to be intubated in the field. Support has also been found for a potentiating role of alcohol in a study of time to death in MVC patients with central nervous system injuries (Zink et al., 1996). In addition, alcohol has been found to be positively associated with an increased incidence of mass lesions (Ruff et al., 1990), prevalence of neurological impairment at discharge, and a poorer long-term outcome (Kraus et al., 1989; Marshall et al., 1992). Other clinical investigations have found negative results in regard to the potentiating role of alcohol (Bigler et al., 1996) or showed no adverse effects attributable to acute alcohol use on TBI (Nath et al., 1986; Jurkovich et al., 1993). One study of 1200 trauma patients found no increase in head injury as a cause of death in intoxicated vs non-intoxicated patients (Ward et al., 1982).

A limitation of alcohol-related brain injury studies is that the data have been collected only after hospital admission and crash characteristics or initial mechanisms of trauma have not been studied (Ward et al., 1982; Huth et al., 1983; Marshall et al., 1992; Jurkovich et al., 1993). In addition, studies often do not account for victims who died on scene. These considerations may make it more difficult to detect a potentiating effect of alcohol, if one truly exists. Those studies that have controlled for crash characteristics have not independently investigated brain injury. The purpose of the present study was therefore to determine, using a specific measurement of brain injury, if alcohol potentiated severity of TBI in the MVC victim after controlling for crash characteristics. Specifically, we hypothesized that, after controlling for crash severity, injured patients who have alcohol present will have evidence of a more severe anatomic brain injury based on a standardized head CT scoring system, than their alcohol-negative counterparts.

SUBJECTS AND METHODS

Study design

The sample population was a retrospective cohort group of 1450 MVC victims. The data used were collected prospectively in a previously conducted study of MVC victims (Waller et al., 1986). The MVC victims were categorized into less severe injury (<3) and more severe (≥3). Logistic regression using this variable as the outcome parameter and crash characteristics, age and BAC as predictors was done, and the odds ratio (OR) and 95% confidence interval (0.95 CI) calculated. Fifty-eight patients were analysed: 41% were BAC positive, 30% had a modified Marshall score of ≥3. Patients with positive BAC were 2.1-fold more likely to have a more severe head injury as measured on CT scan by the Marshall scores. We suggest that alcohol potentiates severity of TBI as determined from head CT among MVC victims. Further research will be needed to substantiate this finding as well as to determine its long-term effect on clinical outcome.

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et al., 1997). These data were supplemented by retrospectively reviewing head CT scans that were done as part of the initial trauma evaluation of the patients. In the prospectively conducted portion of the study, informed consent was obtained prior to BAC determination from subjects or next of kin. The institutional review boards of both the university hospital and the community hospital approved this study.

Patient population and setting

Subjects were patients ≥18 years old, who were occupants of automobiles or small trucks involved in an MVC and who presented directly from the crash scene to one of two university-affiliated Emergency Departments within 6 h of the crash. Pregnant patients, institutionalized patients, and transfer patients were excluded. Motorcyclists, occupants of trucks, pedestrians, or bicyclists were excluded. Site 1 is a large community teaching hospital. Both of these hospitals are located in Ann Arbor, MI, USA, which is a city of ~125,000 citizens situated within a metropolitan statistical area (MSA) of 330,000, and surrounded by similarly populated counties. The University Hospital morgue was also monitored for patients delivered directly from a MVC. Data collection occurred over a 29-month period at the university hospital (April 1992 to August 1994) and over a 15-month period at the community hospital (April 1993 to June 1994). The initial prospective study found 2148 subjects in the sampling frame; of these, 1450 patients provided consent and had valid measures of crash characteristics and injury obtained. These cases were reviewed, and, after inclusion and exclusion criteria were applied (see Results for details), 58 patients were available for analysis.

Study protocol

At both Emergency Departments, subjects were recruited throughout the evening hours (15.30–23.30), the time when most MVC subjects presented. Site 1 was also time-sampled during the day and midnight shifts. Data sources included ambulance reports, hospital records, autopsy records, crash reports, and head CT.

Measurements

Alcohol data. Blood-alcohol concentration (BAC) was determined by serum analysis (enzyme immunoassay), whole blood analysis (gas chromatography), or breath analyser (Alco-sensor III, Model #1020312; Intoximeters Inc., St Louis, MO, USA) conducted in the Emergency Departments. Blood or breath samples obtained within 6 h of the injury deaths at the scene were used to determine alcohol level. When blood samples were not available, post-mortem tests of vitreous humour were used. Gamma-glutamyltransferase (GGT) has been used by some investigators to identify chronic alcohol misuse with levels >85 used as indicators of chronic misuse (Wallner et al., 1999). GGT measurements (enzymatic assay) were obtained using Ektachem 700 XR analyser (Johnson and Johnson, Rochester NY, USA: institutional norms range: 1–35 IU/l).

Crash data. Data on the crashes were compiled from hard copies of crash reports, obtained from local enforcement agencies, within a few days of the crash event. Crash reports were abstracted by specially trained research assistants and provided occupant position, belt use, and TAD [traffic accident deformity score, which measures degree of vehicle deformation]. The TAD scale ranges from 1 to 7 and is a measure of vehicle crush that is highly correlated with occupant injury.

Injury severity. Information from medical records on injury was abstracted by certified nurses who had attended the Association for the Advancement of Automotive Medicine injury scoring course and had experience in injury scoring. Injury severity scores (ISS) (Baker et al., 1974), anatomic injury scores (AIS) (Committee on Injury Scaling, 1985) and anatomic profile (AP) (Copes et al., 1990) scores were calculated. The AIS is a consensus-derived, anatomically based system that classifies individual injuries by body region on a 6-point ordinal severity scale ranging from AIS 1 (minor) to AIS 6 (currently untreatable). The injury severity score was then calculated by using the sum of the squares of the highest AIS scores in the three different body regions and gives a better fit between overall severity and probability of survival. ISS scores range from 1 to 75. Scores were obtained using Tricode (Tricode Analytics, Bel Air, MD, USA), a computer software package for AIS (85) and ISS determination. AP scores were then calculated, using the three highest scoring major injuries within each of the three AP regions (or ISS components). The AP score takes into account the potentially confounding effects of multiple injuries occurring in any one component (Copes et al., 1990).

TBI severity. In order to identify patients with TBI from this prospectively collected database, APA scores were reviewed. Patients with APA scores of >0 (where A is brain, head, and spinal cord) were examined. These groups of patients were then examined by chart review and were excluded if they had an isolated spinal or facial injury. Patients who died on scene or in the Emergency Department and who had a completed autopsy were reviewed and included if they had APA scores >0.

The Marshall Classification was used to scale severity of head injury on the presenting head CT or autopsy report. A neurosurgeon, who was blinded to any clinical or ethanol data, read the head CT and assigned a Marshall score. The Marshall score is originally based on patients with Glasgow Coma Score (GCS) <8, and has been shown to be predictive of clinical outcome (Marshall et al., 1991). For the purpose of this study the score served as a standard measure of anatomical classification of severity and does not account for initial GCS, and is therefore not predictive of clinical outcome. The original Marshall classification ranges from 1 to 4, and categorizes separately any lesion that is surgically evacuated (Marshall et al., 1992). For the purpose of this study, scores were grouped into a modified Marshall classification. Small numbers precluded analysing differences in each Marshall category, therefore patients were divided into two Marshall groups (Table 1). Clinically, patients with modified Marshall 0–2 have a less severe head injury and may be without focal deficits, whereas Marshall ≥3 patients demonstrate significant intracranial abnormalities.

Statistical analysis. Univariate comparisons were made between the two modified Marshall groups on demographic and crash characteristics known to be related to injury severity. \( \chi^2 \)-Test was used for dichotomous measures and t-test for continuous measures, with significance level set at \( P < 0.05 \). Logistic regression was employed to measure the independent effect of alcohol on Marshall score, by controlling for other
variables known to affect injury from previous analyses of the full data (seatbelt use, age, TAD, the interaction of belts and TAD, and alcohol use) (Waller et al., 1999). Odds ratios for alcohol use were calculated both unadjusted and then adjusted for crash severity. Confidence intervals (0.95 CI) were also calculated.

RESULTS

The initial prospective study found 2148 subjects in the sampling frame; 1705 were approached and consented. Valid measures of crash characteristics and injury were obtained for 1450 patients. This initial database of 1450 patients was reviewed and 81 patients were identified with APA scores >0 or a completed autopsy. Of these, 20 were excluded for having isolated facial or isolated spinal injuries. Therefore, 61 patients were eligible for review of head CT or autopsy report. However, as three head CT could not be located, 58 patients were therefore available for analysis. A majority 46 (79%) of these cases were driving the vehicle.

Table 1. Distribution of computed axial tomogram findings assigned by Marshall score

<table>
<thead>
<tr>
<th>Marshall score</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1 no intracranial pathology</td>
<td>16 (27.6)</td>
</tr>
<tr>
<td>2 cisterns present, midline shift 0–5 mm, no mixed density lesion &gt;25 cc</td>
<td>25 (43)</td>
</tr>
<tr>
<td>3 compressed cisterns with midline shift 0–5 mm</td>
<td>9 (15.5)</td>
</tr>
<tr>
<td>4 midline shift &gt;5 mm; or surgically evacuated lesions, and those &gt;25 mm</td>
<td>8 (13.8)</td>
</tr>
</tbody>
</table>

Total number of subjects = 58.

Table 2. Marshall score: anatomical description

<table>
<thead>
<tr>
<th>Marshall &lt;3</th>
<th>Marshall ≥3</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: No visible intracranial pathology seen on CT in patients who were alert, appropriate and oriented</td>
<td>3: Cisterns compressed or absent with midline shift 0–5 mm; no high or mixed density lesion &gt;25 cc</td>
</tr>
<tr>
<td>1: No visible intracranial pathology seen on CT</td>
<td>4: midline shift &gt;5 mm; no high-mixed density lesion &gt;25 cc Any lesion surgically evacuated</td>
</tr>
<tr>
<td>2: Cisterns present with midline shift 0–5 mm; no high or mixed density lesion &gt;25 cc; may include bone fragments and foreign bodies</td>
<td>Any lesion &gt;25 cc not surgically evacuated</td>
</tr>
</tbody>
</table>

Table 3. Relationship of Marshall category to demographic, crash, and alcohol use characteristics

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Marshall &lt;3 (n = 41)</th>
<th>Marshall ≥3 (n = 17)</th>
<th>Total (n = 58)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>26 (63)</td>
<td>13 (76)</td>
<td>39 (67)</td>
</tr>
<tr>
<td>Age (years) (mean SD)</td>
<td>35 (16.8)</td>
<td>30 (11.83)</td>
<td>32 (15.1)</td>
</tr>
<tr>
<td>No belt use</td>
<td>20 (48)</td>
<td>8 (47)</td>
<td>28 (48)</td>
</tr>
<tr>
<td>TAD 26 (most severe)</td>
<td>21 (51)</td>
<td>13 (76)</td>
<td>34 (58)</td>
</tr>
<tr>
<td>Alcohol present</td>
<td>15 (37)</td>
<td>9 (53)</td>
<td>24 (41)</td>
</tr>
<tr>
<td>BAC (mean ± SD)</td>
<td>174.8 ± 78.5</td>
<td>143.2 ± 89</td>
<td>160.8 ± 76.69</td>
</tr>
<tr>
<td>GGT (mean ± SD)</td>
<td>37.6 ± 30</td>
<td>45.5 ± 35.0</td>
<td>47.08 ± 66.23</td>
</tr>
</tbody>
</table>

No measured difference between groups (P > 0.05).
TAD, traffic accident deformity score; BAC, blood-alcohol concentration; GGT, gamma-glutamyltranspeptidase (chronic alcohol misuse >85 U/l).

DISCUSSION

Previous clinical studies have suggested that alcohol potentiates overall mortality when injury forces are controlled for (Waller et al., 1986) and morbidity when summary measures
of injury severity are used (Waller et al., 1999). Data from this retrospective analysis suggest that alcohol may have a potentiating effect on TBI as shown by initial head CT in motor vehicle crash victims. This analysis controlled for variation in crash characteristics. In addition, MVC crash victims in this study who died on scene were included. This eliminated an important source of selection bias.

Alcohol may play a prominent role in the early response to TBI (Zink et al., 1996). The differences seen on initial head CT suggest that the effects of alcohol on TBI occur rapidly after the impact. These effects could be physiological, such as respiratory depression, or decreased cerebral perfusion (Zink et al., 1993, 1996; Zink and Feustel, 1995) or due to biomolecular changes in neuronal receptors and membrane function. Animal models suggest that alcohol exposure causes changes in cell membrane architecture and function (McCall et al., 1989). Alcohol may cause osmotic shifts that lead to cellular swelling (Siggins et al., 1996). Alterations in membrane-bound enzymes and cell membranes through abnormal free radical reactions in the presence of alcohol have been found (Flamm et al., 1977). These effects in turn may lead to increased susceptibility to cell membrane breakdown, increasing the effect of the initial kinetic impact (Marshall et al., 1991, 1992). Further laboratory evidence suggests alcohol may cause a potentiating effect on TBI by changes in cerebral perfusion (Zink et al., 1993).

While some previous clinical studies have found an association between alcohol and TBI, other studies, including an evaluation of severely head-injured patients in the Traumatic Coma Databank, have shown no effect on clinical course of alcohol on TBI (Ruff et al., 1990). In one study, alcohol-intoxicated patients hospitalized with TBI were more likely to require mechanical intubation (Gurney et al., 1992). Another study noted poorer neurological outcome and increased permanent disability in severe TBI patients who had high levels of alcohol or a history of chronic alcohol misuse (Kraus et al., 1989). However, it is important to note that no prior clinical study has accounted for crash characteristics. In addition, chronic alcohol use also has effects on haemostasis that may play a role in TBI, such as decreased platelet aggregation and function, as well as problems involving erythrocytes, leukocytes, and other haemostatic factors. In this study, the mean GGT (a measure of chronic alcohol use) was not significantly different between the two groups. Changes in haemostasis with acute ingestion are less well understood; however, current research supports the concept that coagulation is most likely less affected by acute alcohol ingestion than by chronic ingestion or sudden abstinence (Stein, 1999).

If alcohol has a potentiating effect on the severity of TBI, independently of the initial crash, this would have far-reaching clinical and public health consequences. These effects may suggest that while current public health messages are aimed at drunk drivers, an intoxicated passenger may also be at increased risk for a more severe head injury in a MVC. In addition, although the alcohol ‘legal limit’ in many states is 100 mg/dl, it is not yet clear at what BAC these potentiating effects may begin or end, calling into question the arbitrary nature of the ‘legal limit’.

Limitations of this study include a small sample size, hence a wide 0.95 CI. Post hoc power analysis, assuming the same proportion of patients with a Marshall score >5 found in this study (an alpha of 0.05 and power of 0.80), shows that 175 subjects per group would be needed to demonstrate a significant difference between the Marshall scores for the alcohol-positive and alcohol-negative groups. One further limitation in the use of initial head CT to describe TBI is that head CT represents a fixed point in time, whereas TBI is an evolving process. Initial CT does not fully describe the extent of pathology that may be present at 24–72 h post injury, or what the differences in CT will be associated with in terms of long-term outcome. In addition, it should be noted that GGT is only one measurement for haemostatic dysfunction in chronic alcohol use, and future trials should address in more detail the effects of coagulation disorders and platelet function in acute and chronic alcohol use in relation to TBI. Finally, it is unclear whether the current study’s findings apply to TBI resulting from mechanisms other than MVC, such as falls and assaults.

In conclusion, the present results suggest that alcohol may have a potentiating effect on TBI as shown by initial head CT in MVC victims. The outcome severity of the resulting brain injury is crucial not only to the individual physically, emotionally, and financially, but also to society as a whole. Further multi-centre studies are needed to elucidate the potentiating effect of alcohol in TBI. Results of such studies may aid in intervention both in the acute post-injury phase, to modulate the effects of alcohol on injury, as well as contribute to pre-injury/pre-drinking prevention efforts.

Acknowledgements — This study was funded in part by the NIAAA (R01-AA09110-01) and the University of Michigan Injury Research Center (UMIRC).

REFERENCES


Committee on Injury Scaling (1985) Abbreviated Injury Scale 1985 Revision. American Association for Automotive Medicine, Arlington Heights, IL.


Table 4. Percentage of patients with severe head injury

<table>
<thead>
<tr>
<th>(Marshall score &gt;3) in alcohol groups, controlling for TAD (traffic accident deformity score)</th>
<th>Alcohol +</th>
<th>Alcohol −</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAD &lt;6</td>
<td>2/10 (20.0)</td>
<td>2/14 (14.3)</td>
<td>n = 24</td>
</tr>
<tr>
<td>TAD &gt;6</td>
<td>7/14 (50)</td>
<td>6/20 (30)</td>
<td>n = 34</td>
</tr>
<tr>
<td>Total</td>
<td>n = 24</td>
<td>n = 34</td>
<td></td>
</tr>
</tbody>
</table>

Percentage of patients in severe Marshall category (severe head injury) increased with alcohol use, despite similar severity of crash (i.e. within TAD category).


