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ORIGINAL ARTICLE

Alcohol exposure and outcomes in trauma patients

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Abstract
Objective To determine the injury patterns, complications, and mortality after alcohol consumption in trauma patients.
Methods The Trauma Registry at an American College of Surgeons (ACS) level I center was queried for all patients with a toxicology screen admitted between 1st January 2002 and 31st December 2005. Alcohol-positive (AP) patients were matched to control patients who had a completely negative screen (AN) using age, gender, mechanism, Injury Severity Score (ISS), head Abbreviated Injury Scale (AIS), chest AIS, abdominal AIS, and extremity AIS. Injuries and outcomes were compared between the groups.
Results As many as 5,317 patients had toxicology data, of which 471 (8.9%) had a positive alcohol screen (AP). A total of 386 AP patients were then matched to 386 control (AN) patients. The AP group had a significantly higher mortality than the AN group overall (23 vs. 13%; $p < 0.001$), and by ISS stratification: ISS < 16 (6 vs. 0.4%; $p < 0.001$), ISS 16–25 (53 vs. 28%; $p = 0.01$), and ISS > 25 (90 vs. 67%; $p = 0.01$). AP patients had a higher incidence of admission systolic blood pressure < 90 (18 vs. 10%; $p < 0.001$) and Glasgow Coma Scale (GCS) score ≤ 8 (25 vs. 17%; $p = 0.002$). AN patients had a significantly higher incidence of hemopneumothorax (11 vs. 7%; $p = 0.03$), while AP patients had a higher incidence of cardiac arrest (8 vs. 3%; $p = 0.004$). There was no difference in intensive care unit (ICU) and hospital length of stay.
Conclusion In a mixed population of trauma patients, an AP screen is associated with an increased incidence of admission hypotension and depressed GCS score. In this case-matched study, alcohol exposure appeared to increase mortality after injury.

Keywords Alcohol · Injury · Trauma · Complications · Mortality

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Introduction

Since the first documented use of alcohol over 10,000 years ago, it has been integral to many social interactions and even asepsis and analgesia. Despite the fact that alcohol may have some health benefits in reducing heart attacks, diabetes, and even Alzheimer's, overindulgence has become a very serious problem [1–3]. In the United States, alcohol misuse is now the leading risk factor for serious injury and the third leading cause of preventable death [4].

The clinical effect of alcohol is dependent on the size, gender, and metabolic state of the individual. Due to its depressive effects on the central nervous system, initial

effects can cause mild euphoria, relaxation, decreased inhibitions, and an impairment of judgment, as well as loss of efficiency in fine motor tasks. Later stages of intoxication can cause depressed respiration, hypotension, and even coma and death [5]. In trauma, both acute and chronic alcohol abuse is associated with a blunted physiologic response. Alcohol alters hemodynamic, metabolic, and inflammatory homeostasis after hemorrhage, blunts the catecholamine surge after injury, and reduces the electrical threshold for ventricular arrhythmias [6–8]. In the subacute phase of injury, immunosuppressive effects from alcohol increase the risk of secondary infections, caused by the attenuated neuroendocrine activation, and also by the suppression of proinflammatory cytokines [9, 10]. Coin-toxication with other drugs is a common finding and it has also been shown to increase complications [11].

Numerous studies have reported an increasing risk of non-fatal and intentional injuries with increasing alcohol consumption [12–16]. In the United States, it is estimated that half a million Emergency Department (ED) visits and a third of all ED drug abuse visits yearly involve alcohol [17]. In youths aged 16–20 years, alcohol is involved in 36% of traffic-related deaths [18]. Plurad et al. [19] have shown that over 40% of trauma deaths have a positive screen for alcohol, drugs, or both. The interaction of alcohol intoxication with injury pattern and outcomes is inconsistent in the literature. In two separate studies, Plurad et al. [20] and Phelan et al. [21] showed different effects of alcohol on mortality depending on the mechanism of injury and level of intoxication, with increased complications in pedestrians, but improved survival in victims of motor vehicle crashes.

The purpose of this study was to determine the injury patterns, complications, and mortality in matched cohorts of alcohol-positive (AP) and -negative (AN) trauma patients.

Methods

Patients who were admitted between 1st January 2002 and 31st December 2005 and who underwent a toxicology screen were eligible for inclusion in our study. Patients are routinely screened for toxicology by blood and urine when admitted through the resuscitation area of our ED. Patients dying within 24 h of admission, patients who failed to undergo toxicology screen, and patients who were positive for multiple drugs were excluded. Our Institutional Review Board approved this study.

Data items were extracted from the trauma registry at our urban, academic American College of Surgeons (ACS)-verified level I center. The variables studied included age, gender, mechanism (blunt vs. penetrating), injury diagnoses, operative procedures, admission vitals (heart rate, systolic blood pressure), admission Glasgow Coma Scale (GCS), Abbreviated Injury Scale (AIS), Injury Severity Score (ISS), results of alcohol screen, hospital and intensive care unit length of stay, and mortality. Patients who had a positive toxicology screen for alcohol only (AP) were compared to patients who had an entirely negative screen (AN).

Each AP patient was matched with a single AN patient on age ($\leq$18, 19–55, >55 years), gender, mechanism of injury (blunt, penetrating), ISS (<16, 16–25, >25), head AIS (<3, $\geq$3), chest AIS (<3, $\geq$3), abdominal AIS (<3, $\geq$3), and extremity AIS (<3, $\geq$3). Matched pairs of binary outcomes were analyzed using McNemar's test and matched pairs of continuous data were tested using the Wilcoxon signed-rank test. Differences were considered to be statistically significant at $p < 0.05$. Statistical analysis was performed using the SPSS software package, version 12.0 (SPSS Inc., Chicago, IL).

Results

There were 5,317 patients studied, where 471 (8.9%) were AP and 4,846 (91.1%) were AN patients. We matched 386 (81.9%) AP patients with a corresponding AN control. The matching criteria are shown in Table 1 and the admission characteristics are shown in Table 2. The AP group had a significantly greater incidence of admission hypotension (systolic blood pressure <90 mmHg) and GCS $\leq$ 8. AN patients had a significantly higher incidence of hemo-pneumothorax (10.6 vs. 7.0%; $p = 0.03$; Table 3), but the injury patterns were otherwise similar. AP patients had a significantly higher incidence of in-house cardiac arrest (7.5 vs. 2.8%; $p = 0.004$) in comparison to AN patients, but the incidence of surgical procedures was similar (Table 4).

The comparison of admission characteristics and outcomes for the entire study populations stratified by ISS is shown in Table 5. The overall mortality in the AP group was increased (23.1 vs. 12.7%, $p < 0.001$) compared to the AN group, which was maintained when stratified by the severity of injury. There was no difference in ICU and hospital length of stay.

Discussion

Studies have shown that 40 to 50% of trauma patients are injured while under the influence of alcohol [22–24]. An ED study reported that moderate alcohol consumption is associated with a ten-fold increased risk of injuries, and there are suggestions that this happens in a dose-dependent

Table 1 Characteristics of the study population by matching criteria

Variables	Alcohol ($n = 386$) % (n)	Non-intoxicated ($n = 386$) % (n)	p-value
Age, years			
≤18	5.4 (21/386)	5.4 (21/386)	1.00
19–55	82.1 (317/386)	82.1 (317/386)	1.00
>55	12.4 (48/386)	12.4 (48/386)	1.00
Gender			
Male	87.0 (336/386)	87.0 (336/386)	1.00
Female	13.0 (50/386)	13.0 (50/386)	1.00
Mechanism of injury			
Blunt	77.7 (300/386)	77.7 (300/386)	1.00
Penetrating	22.3 (86/386)	22.3 (86/386)	1.00
ISS			
≤15	73.6 (284/386)	73.6 (284/386)	1.00
16–25	13.7 (53/386)	13.7 (53/386)	1.00
>25	12.7 (49/386)	12.7 (49/386)	1.00
AIS			
Head			
<3	83.2 (321/386)	83.2 (321/386)	1.00
≥3	16.8 (65/386)	16.8 (65/386)	1.00
Chest			
<3	82.1 (317/386)	82.1 (317/386)	1.00
≥3	17.9 (69/386)	17.9 (69/386)	1.00
Abdomen			
<3	92.2 (356/386)	92.2 (356/386)	1.00
≥3	7.8 (30/386)	7.8 (30/386)	1.00
Extremity			
<3	93.3 (360/386)	93.3 (360/386)	1.00
≥3	6.7 (26/386)	6.7 (26/386)	1.00

Equality in proportions is tested by McNemar's test; paired difference in age and ISS by Wilcoxon signed-rank test

Equality is tested by the Kappa test

fashion [14, 25]. Alcohol alters judgment, frequently leading to increasing aggression and risk-taking behavior predisposing to interpersonal violence and subsequent injury [5, 19, 22, 26–28].

The interaction of alcohol exposure and cardiovascular response to injury is variable in the literature. Experimental studies show that alcohol blunts the physiologic response to hemorrhage by inhibiting the release of vasopressin and by a depressive effect upon the myocardium [8, 29, 30]. Clinical data shows that alcohol ingestion is associated with a significantly lower systolic blood pressure on admission [31]. Alcohol intoxication can be associated with life-threatening arrhythmias, such as prolonged P-wave and QTc interval, conduction disturbances, non-specific T-wave changes, and shortening of the action potential [32–35]. Our study supports an interaction between alcohol ingestion and cardiovascular response after injury as AP patients have a higher incidence of hypotension on presentation and cardiac arrest. AP patients may need more intensive monitoring for these cardiovascular complications.

Alcohol is a potent central nervous system depressant, reducing the level of consciousness, which can lead to stupor, coma, or even death [5, 22]. Data relating to the effects of alcohol ingestion on the GCS in trauma patients is also variable [36–38]. In a study of assault victims, there was a significant association between the degree of intoxication and the GCS [36]. In contrast, alcohol intoxication does not result in clinically significant changes in the GCS score in a study of patients with blunt traumatic brain injury [38]. Our current study concurs with this by showing that AP patients have a statistically significantly lower GCS, but with little clinical relevance, even when stratified by the severity of injury. It is apparent that alcohol can complicate the care of trauma patients, given the higher incidence of depressed mental status (GCS ≤ 8), since clinicians are compelled to commit more resources to these patients.

It has been shown that alcohol exposure can be immunomodulatory. In an experimental rat study, alcohol intoxication exacerbates neutrophil apoptosis and suppresses phagocytosis in hemorrhagic shock, thus, weakening host defense to infection [9]. Our current data showed an increase in pneumonia rates between the two

Table 2 Comparison of the admission characteristics of matched groups

Admission vitals	Alcohol ($n = 386$)	Non-intoxicated ($n = 386$)	p-value
SBP			
Mean ± SD	111.1 ± 53.4	122.3 ± 40.7	0.01
<90 mmHG, % (n)	18.1% (70/386)	9.6% (37/386)	<0.001
HR			
Mean ± SD	82.5 ± 39.6	90.3 ± 30.4	0.08
GCS			
Mean ± SD	11.5 ± 4.7	12.5 ± 4.0	<0.001
≤8, % (n)	24.6% (95/386)	17.1% (66/386)	0.002

SBP systolic blood pressure; *HR* heart rate; *GCS* Glasgow Coma Scale

Table 3 Comparison of the injuries of matched groups

Injuries	Alcohol ($n = 386$) % (n)	Non-intoxicated ($n = 386$) % (n)	p-value
Skull vault fracture	3.6 (14)	3.4 (13)	1.00
Basilar skull fracture	4.9 (19)	4.9 (19)	1.00
Intracranial hemorrhage	6.2 (24)	8.3 (32)	0.20
Hemopneumothorax	7.0 (27)	10.6 (41)	0.03
Lung	7.8 (30)	7.0 (27)	0.71
Cardiac	5.4 (21)	5.2 (20)	1.00
Diaphragm	4.4 (17)	2.6 (10)	0.09
Solid-organ injury	6.2 (24)	5.7 (22)	0.85
Liver	4.9 (19)	3.6 (14)	0.38
Kidney	0.8 (3)	0.8 (3)	1.00
Spleen	1.6 (6)	1.8 (7)	1.00
Hollow viscus	4.1 (16)	5.7 (22)	0.21
Stomach	1.8 (7)	1.6 (6)	1.00
Small bowel	2.6 (10)	3.4 (13)	0.58
Large bowel	1.8 (7)	2.3 (9)	0.73
Lower extremity	5.7 (22)	7.3 (28)	0.29
Femur	2.1 (8)	3.6 (14)	0.18
Tibia	3.6 (14)	4.7 (18)	0.50
Upper extremity	5.7 (22)	4.1 (16)	0.35
Humerus	3.1 (12)	1.8 (7)	0.30
Radius/ulna	3.4 (13)	2.3 (9)	0.48
Pelvic fracture	3.6 (14)	4.1 (16)	0.82

Table 4 Comparison of the surgical procedures and complications of matched groups

Variables	Alcohol ($n = 386$) % (n)	Non-intoxicated ($n = 386$) % (n)	p-value
Surgical procedures			
Craniectomy	1.6 (6)	2.6 (10)	0.42
Laparotomy	5.2 (20)	6.7 (26)	0.29
Thoracotomy	1.0 (4)	1.8 (7)	0.51
ORIF	4.1 (16)	3.4 (13)	0.63
Complications			
Had a complication	8.3 (32)	6.2 (24)	0.29
Sepsis	0.3 (1)	0.5 (2)	1.00
Pneumonia	0.8 (3)	3.1 (12)	0.02
Acute respiratory failure	0.8 (3)	0.3 (1)	0.63
Acute renal failure	0.5 (2)	0.3 (1)	1.00
Cardiac arrest	7.5 (29)	2.8 (11)	0.004

ORIF open reduction and internal fixation

groups, without effects seen on sepsis or other organ failure. Due to the small numbers of patients with complications, it is difficult to draw definitive conclusions on immunologic suppression due to alcohol ingestion in this population of trauma patients.

Data that address the interaction between alcohol use and mortality is also inconsistent but is supported by our study. Overall, the mortality was almost two-fold higher in the AP group. In an autopsy study at this institution, the authors showed that victims of penetrating trauma and

Table 5 Comparison of the admission characteristics and outcome of matched groups stratified by Injury Severity Score (ISS)

Injury Severity Score	Alcohol ($n = 386$) % (n)	Non-intoxicated ($n = 386$) % (n)	p-value
<16			
SBP < 90	6.3 (18/284)	1.8 (5/284)	0.004
GCS ≤ 8	9.9 (28/284)	5.3 (15/284)	0.05
Complication	3.2 (9/284)	0.7 (2/284)	0.07
Mortality	6.0 (17/284))	0.4 (1/284)	<0.001
≥16–25			
SBP < 90	37.7 (20/53)	18.9 (10/53)	0.03
GCS ≤ 8	49.1 (26/53)	37.7 (20/53)	0.33
Complication	20.8 (11/53)	20.8 (11/53)	1.00
Mortality	52.8 (28/53)	28.3 (15/53)	0.01
>25			
SBP < 90	65.3 (32/49)	44.9 (22/49)	0.04
GCS ≤ 8	83.75 (41/49)	63.3 (31/49)	0.02
Complication	24.5 (12/49)	22.4 (11/49)	1.00
Mortality	89.8 (44/49)	67.3 (33/49)	0.01
Total			
SBP < 90	18.1 (70/386)	9.6 (37/386)	<0.001
GCS ≤ 8	24.6 (95/386)	17.1 (66/386)	0.002
Complication	8.3 (32/386)	6.2 (24/386)	0.29
Mortality	23.1 (89/386)	12.7 (49/386)	<0.001

SBP systolic blood pressure; *HR* heart rate; *GCS* Glasgow Coma Scale

positive toxicology are more likely to have no vitals on admission in comparison to victims with negative toxicology [19]. Other studies have shown no correlation between alcohol exposure and mortality [21, 27, 30, 37, 39], while a number of recent reports have even suggested improved survival [20, 40–42]. The discordance between these investigations is a multifactorial issue. Study population selection bias is foremost amongst them. Data from autopsy-based, emergency room-based, and/or emergency medical services-based study populations will likely differ from hospital-based or trauma center-based studies. Results from multicenter studies [20, 21] will differ from single-center investigations such as the current study. Clinical data that restricts analysis to the specific mechanism of injury (penetrating only [19], pedestrians only [21] vs. motor vehicle collisions only [20]) simplifies analysis but does not facilitate the application of findings to entire populations of trauma patients. There may also be an association between injury severity and alcohol intoxication [43]. Unfortunately, like many trauma studies, particularly those examining recreational drug use, ethics preclude randomized prospective human trials. The current clinical data, however disparate, do provide valuable information that can be used to design pertinent laboratory investigations which may, in turn, lead to meaningful clinical applications.

The veracity of this report is limited by the study's retrospective design. Though most trauma patients at our institution are admitted via the resuscitation area, a number of them are not and, therefore, some patients may not receive routine drug screening. Blood alcohol levels were not consistently available; therefore, we were unable to stratify our results by the degree of intoxication. In addition, we were unable to obtain information regarding any history of chronic alcohol abuse as opposed to binge drinking. We did not include patients who died or who were discharged before 24 h following admission. Breaks in protocol due to various reasons common to a busy level I center can lead to further data loss. In addition, we did not include patients who were positive for multiple substances. In efforts to make the data manageable and results easily interpreted, this exclusion may have further introduced selection bias. Despite these shortcomings, we believe that this data has wide implication, supports further study on possible immunomodulatory effects of alcohol, and will be useful for preventative efforts in the community.

It will surely perpetuate the current controversies surrounding alcohol and its effect on trauma injuries and outcomes.

Although alcohol is a legal drug, it is associated with an increased risk of unfavorable results. In a mixed population of trauma patients, alcohol is associated with an increased

incidence of hypotension and depressed GCS on admission and, further, may be associated with an increased mortality. Despite variable findings in the literature, our current data contributes greatly to the continued controversy over this issue and mandates further study.

Conflict of interest statement None.

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