Impact of blood alcohol concentration on hematologic and serum chemistry parameters in trauma patients: Analysis of data from a high-volume level 1 trauma center

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ABSTRACT

Background: Alcohol (EtOH) intoxication is common among trauma patients. While providers are familiar with the clinical aspects of acute EtOH intoxication, few studies have investigated the effects that EtOH levels may have on common laboratory markers. The aim of this study was to identify hematologic and serum chemistry parameters that may be affected by the blood alcohol concentration (BAC), hypothesizing that BAC influences both comprehensive blood count (CBC) and comprehensive serum chemistry (CSC) components.

Methods: We performed an IRB-exempt institutional registry review of all trauma patients who had serum EtOH levels measured between January 2009 and June 2015. Data for each patient included: patient demographics, BAC determinations (g/dL), injury mechanism/severity information (ISS), hematologic parameters included in a CBC (hemoglobin, hematocrit, white blood cell [WBC] count, and platelet count), and CSC panel components (sodium, potassium, chloride, bicarbonate, blood urea nitrogen [BUN], creatinine, glucose, and hepatic function tests). Laboratory markers were contrasted across predefined categories of BAC: <0.10%, 10%–15%, 15%–20%, and >20%. Statistical comparisons were performed using SPSS 18 Software, employing analysis-of-covariance with adjustments performed for the patient demographics and injury characteristics. Statistical significance was set at $\alpha = 0.005$.

Results: A total of 2167 patient records were analyzed. After adjusting for patient age, gender, and ISS, increasing BAC correlated with 4.8% increase in hemoglobin and 32.5% higher hematocrit (both $P < 0.001$), as well as a 27.8% decrease in WBC count. There were also statistically significant differences between low (<0.10%) and high (>0.20%) BAC groups across multiple CSC parameters, with largest impact on BUN (32.2% decrease); creatinine (31.5% decrease); and glucose (13.6% decrease) values. Elevated BAC (>0.20 g/dL) was also associated with 81.8% increase in total bilirubin, and hepatic transaminases were elevated among patients with BAC >0.10.

Conclusion: Due to the paucity of literature relating to the effects of BAC on serum hematologic and biochemical markers in acute trauma, this study provides a foundation for further exploration of these relationships and their clinical impact. More specifically, we found that BAC levels significantly...
influenced key laboratory markers, suggesting that acute EtOH intoxication may lead to hematologic and CSC changes that are potentially important in acute trauma management by frontline clinical staff.

Key Words: Alcohol intoxication, hematology, injury, laboratory values, serum chemistry, trauma

INTRODUCTION

Excessive alcohol (EtOH) consumption is a leading cause of mortality in the United States, directly and indirectly contributing to approximately 1 in 10 deaths among working-age adults. Each year, approximately 100,000 people die secondary to excessive EtOH use in the United States alone. Moreover, an estimated $185 billion in annual costs can be attributed to excess disability from secondary events related to EtOH abuse (e.g., motor vehicle collisions, drowning, falls, burns, and assaults). On a global scale, EtOH-related injuries account for approximately one-third of years of healthy life lost, quantified in disability-adjusted life years.

The prevalence of unhealthy EtOH use ranges from 7 to 20% among outpatients, 30%–40% among patients in emergency departments (EDs), and as much as 50% among patients who present with acute traumatic injuries. As such, EtOH abuse may be considered to be one of the most common chronic illnesses in injured patients, and is associated with increased risk of readmission for new/subsequent trauma. Some even view trauma as a marker for EtOH abuse. Prior studies examined the effects of acute EtOH intoxication on morbidity and mortality of injured patients, showing an overall harmful effect.

Acute EtOH intoxication commonly complicates the initial assessment and management of the injured patient. Unique issues encountered in assessing trauma victims with either acute intoxication or chronic EtOH abuse include, but are not limited to: mimicking of brain injury, masking of intra-abdominal injuries, reduction of immune response, and alteration of hepatic metabolism. Not infrequently, trauma patients with EtOH use disorder present without known (or declared) alcohol abuse history, with early-stage issues, or with complaints that are not seemingly EtOH related. Therefore, any serum markers that could potentially help identify EtOH as a confounding factor in the presenting trauma patient may be of benefit.

The measured impact of blood alcohol concentration (BAC) on various subcomponents of the comprehensive blood count (CBC) and comprehensive serum chemistry (CSC) during the initial evaluation of the injured patient remains largely unexplored. Rather than assuming that such impact is “irrelevant or already known,” the authors believe that potentially useful information could be gleaned from dedicated research in this area. Consequently, this study seeks to investigate correlations between BAC and serum hematologic and chemistry markers using our institutional trauma registry, with a goal of providing traumatologists with potentially useful diagnostic information in the setting of the co-presence of traumatic injury and acute EtOH intoxication. We hypothesized that increasing BAC would correlate with nontrivial alterations in the majority of CBC and CSC subcomponents, without predetermined directionality of the effect.

METHODS

This is an Institutional Review Board exempt study, with data abstracted from our Level 1 Trauma Center institutional registry. We retrospectively reviewed admission records (total, n = 14,057) beginning January 1, 2009, and ending June 1, 2015. Included were all injured patients who underwent formal serum BAC (g/dL) testing on their presentation during the study period and concurrently underwent CBC and/or CSC assessments as part of the initial trauma evaluation. Collected data included patient demographics, BAC, injury mechanism/severity information (ISS), hematologic parameters (hemoglobin, hematocrit, white blood cell (WBC) count, and platelet count), as well as components of the CSC panel (sodium, potassium, chloride, bicarbonate, blood urea nitrogen [BUN], and glucose. When available, hepatic function testing was abstracted from the medical record, including total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and serum albumin.

Hematologic and serum chemistry data were contrasted across predefined BAC strata (0.10, 0.10–0.15, 0.15–0.20, >0.20). The legal definition of acute alcohol intoxication in the most U.S. states is a BAC of 0.08–0.10 g/dL (80–100 mg/dL) or greater, and served as a suitable reference for this study’s analysis. This allowed our data to be potentially applicable across a broad number of geographic settings than the more restrictive BAC of 0.08% g/dL. Trauma physician discretion was used to determine whether a specific trauma patient underwent EtOH screening during the study period. The overall patient inclusion/exclusion diagram is shown in Figure 1.

In addition to standard descriptive statistics and measures of central tendencies, we utilized Chi-squared
methodology to analyze categorical data. For continuous data with normal distribution, statistical comparisons were performed using analysis-of-covariance (with adjustments made for patient demographics and injury characteristics) on SPSS 18 Software (PASW Statistics for Windows, Chicago, Illinois, USA). Due to relatively large data sample, statistical significance was set at \( \alpha = 0.005 \). Because the directionality of the effects of BAC on CBC/CSC components was not known \textit{a priori}, no assumptions were made in this respect. Moreover, due to of lack of reliable preliminary data (and thus very limited information regarding expected intergroup differences), we did not perform a formal power analysis.

**RESULTS**

A total of 2167 patient records were reviewed in the current analysis. Mean patient age was 41.1 ± 17.4 years, with median ISS of 9 (interquartile range [IQR]: 5–17) and 70.4% male predominance. Blunt injuries were most common (2068/2167, or 95.4%). The most common mechanisms of injury included motor-vehicle crash (46.9%), fall (25.1%), and motorcycle crash (7.8%). The remaining 20.2% of mechanisms comprised a combination of pedestrian struck by vehicle, stabbing, gunshot wounding, and various other occurrences. The average hospital length of stay was 5.4 days. Overall mortality of the study sample was 4.1%.

For the overall study sample, the median BAC was 0.10% (IQR: 0–0.13). Examining the four BAC strata, 76% (1648/2167) of patients had BAC < 0.10 g/dL, 7.9% (172/2167) had BAC 0.10–0.15 g/dL, 6.4% (139/2167) had BAC 0.15–0.20 g/dL, and 9.6% (208/2167) had BAC > 0.20 g/dL. Tables 1 and 2 provide a detailed summary of the behavior of various CBC and CSC components, organized according to predetermined BAC strata defined above. Corresponding results will be discussed in the next two paragraphs.

After adjusting for patient age, gender, and ISS, increasing BAC correlated with significant increases in both hemoglobin and hematocrit [Table 1]. When comparing the lowest bracket (BAC < 0.10 g/dL) to the highest bracket (BAC > 0.20 g/dL), the overall increase in hemoglobin concentration of 4.8% was noted. For hematocrit, the same comparison shows a 32.5% increase. When examining WBC count, a decline of 27.8% was noted. Platelet counts demonstrated a more binary behavior, with BAC levels of <0.15 associated with approximately 10% lower platelet counts than BAC levels > 0.15. All of the above relationships were statistically significant (\( P < 0.001 \)).

Similar procedure was followed during the examination of the relationship between BAC and CSC across the four predefined patient groups. There were statistically significant differences across BAC groups for multiple CSC component parameters [Table 2]. On comparison of the lowest (BAC<0.10 g/dL) with the highest (BAC > 0.20 g/dL) patient strata, we found significant differences for the majority of serum chemistry parameters studied. Serum potassium was 8.7% lower in the BAC > 0.20 group. Serum albumin increased by 14.2% in the same group comparison. BUN was 32.2% lower in the BAC > 0.20 group. Similarly, serum creatinine was 31.5% lower in the same comparison. Of interest, serum glucose tended to decrease with increasing BAC (13.6% decrease when comparing the lowest and the highest BAC groups). Although statistically different, the clinical significance observed in serum bicarbonate is likely negligible, with only a 4% difference between the highest and lowest BAC strata. No significant differences were found for sodium and chloride across the four BAC groups.

When examining hepatic function tests, total bilirubin was 81.8% higher in the BAC > 0.20 stratum when

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**Table 1: Key hematologic parameters stratified by blood alcohol concentration**

| Parameter (n) | BAC \textless{} 0.1 | BAC 0.1 < x \textless{} 0.15 | BAC 0.15 < x \textless{} 0.2 | BAC > 0.2 | Significance (\( P \)) |
|---------------|-----------------|-----------------|------------------|-----------|-----------------|
| Hemoglobin (2167) | 13.62 ± 0.07 (n = 1648) | 13.59 ± 0.26 (n = 172) | 14.12 ± 0.39 (n = 139) | 14.28 ± 0.40 (n = 208) | <0.001 |
| Hematocrit (2167) | 39.86 ± 0.46 (n = 1648) | 39.73 ± 1.68 (n = 172) | 40.64 ± 2.48 (n = 139) | 52.81 ± 2.60 (n = 208) | <0.001 |
| White blood cell | 13.45 ± 0.36 (n = 1545) | 11.92 ± 1.38 (n = 161) | 11.10 ± 2.33 (n = 116) | 9.71 ± 2.21 (n = 151) | <0.001 |
| Indent slightly | 229.67 ± 3.04 (n = 1493) | 225.11 ± 11.50 (n = 147) | 244.77 ± 19.04 (n = 87) | 252.37 ± 18.40 (n = 152) | <0.001 |

Data shown as mean ± SEM. BAC shown as g/dL. SEM: Standard error of mean, BAC: Blood alcohol concentration.

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**Figure 1:** Patient inclusion/exclusion diagram. Out of 2352 patients with EtOH/blood alcohol concentration testing, 185 did not have a documented comprehensive blood count or comprehensive serum chemistry and thus were excluded. *Of note, 45 patients had documented comprehensive blood count but no available comprehensive serum chemistry assessment.
compared to the BAC < 0.10 group. Finally, we noted a parallel pattern of behavior for ALT and AST. More specifically, there was a marked increase in both enzymes when proceeding from BAC < 0.10 to BAC 0.10–0.15 strata, with 54.4% increase in AST and 49.8% increase in ALT. However, this trend did not continue in the higher strata (BAC 0.15–0.20 and BAC > 0.20). Rather, a gradual decline was noted, with ALT and AST levels being 44.0% and 26.0% lower for the BAC > 0.20 stratum when compared to the BAC < 0.10 grouping. Details of the above comparison are shown in Table 2.

## DISCUSSION

Alcohol abuse and intoxication play an important role as contributors to a significant proportion of traumatic injuries.\[14,15\] Previous studies have established the link between acute effects of EtOH and specific causes of injury, such as motor vehicle crashes, bicycling, boating, and fires.\[15,16\] In fact, BAC may be the best indicator of hazardous EtOH drinking in young adults and working-age patients who present with trauma.\[17\] Yet despite these well-known epidemiological associations, little is known about the measured hematologic and serum chemistry changes associated with acute EtOH intoxication in trauma. Consequently, the current manuscript’s primary aim is to demonstrate that increasing BAC has the potential to significantly impact key hematologic and serum chemistry parameters. Due to paucity of previously published data in this area, no directionality of the effect was implied by the investigators.

Although data regarding routine CSC variables in the setting of trauma are scant, there is some evidence that acute EtOH intoxication can modulate serum gamma glutamyl transferase (GGT); aspartate aminotransferase (AST); high-density lipoprotein-cholesterol activity; carbohydrate-deficient transferrin (CDT); and erythrocyte mean corpuscular volume (MCV).\[17,18\] In one study of patients admitted to the ED in Innsbruck, Austria, a number of serum electrolyte, hematologic, and hepatic function abnormalities were identified in 121 subjects with acute EtOH intoxication.\[19\] From hematologic perspective, the most frequently observed changes, in descending order, included elevated mean corpuscular hemoglobin (MCH, 40.8%); reduced erythrocyte count (33.3%); elevated MCV (30.6%); decreased hematoctrit (23.3%); leukocytosis (23.1%); thrombocytopenia (14.1%); and reduced hemoglobin (11.7%).\[19\] In decreasing order of frequency, serum chemistry alterations in acute EtOH intoxication included hypernatremia (41%); hyperchloremia (21%); hypermagnesemia (17%); hypocalcemia (15%); hypokalemia (5%); and hypophosphatemia (3.4%).\[19\] Hyperglycemia occurred in 42.2% of patients, with no episodes of hypoglycemia reported.\[19\] Elevated serum AST (38.9%) and ALT (17.8%), low serum creatinine levels (21%), and reduced serum BUN (8%) were observed as well.\[19\] Results of the above investigations will now be discussed in the context of the current study’s findings.

### Impact of acute alcohol intoxication on the complete blood count components

Our findings support previous data on elevations of hemoglobin and hematocrit levels in concert with increasing BAC levels. Prior research has demonstrated EtOH’s ability to increase blood viscosity, considered mainly to be due to hemoconcentration and erythrocyte-related abnormalities.\[20-22\] Our data show a nearly 5% increase in hemoglobin and approximately 33% greater hematocrit for the most intoxicated patients (BAC > 0.20 g/dL) when compared to patients with BAC > 0.10 [Table 1]. When using the strict definition of leukocytosis (WBC ≥11,000/mm³), we noted that patients with BAC levels of ≤0.20 g/dL had findings consistent with previously published data.\[19\] However, the group with BAC > 0.20 g/dL in the current study had normal WBC counts (average of 9710/mm³). This finding, although somewhat surprising, may demonstrate the appearance of cytotoxicity at higher BAC levels — something that previous data did not examine vis-à-vis EtOH blood
concentrations in excess of 0.20 or greater, likely due to insufficient data granularity. Finally, our study demonstrated significantly elevated platelet levels for patients with BAC > 0.15 g/dL. Although previous literature shows the presence of thrombocytopenia in approximately 14% of acutely intoxicated patients, there are no stratified data regarding mean or median serum platelet counts.

Impact of acute alcohol intoxication on comprehensive serum chemistry markers

Sodium, chloride, and potassium
After adjusting for injury severity, patient age and sex, the current study shows no clinically meaningful differences across the BAC strata in terms of serum sodium and chloride levels [Table 2]. We did, however, demonstrate a significant drop of approximately 9% in serum potassium as the BAC level increased. Previously published data suggest that acutely-intoxicated patients may experience some degree of hyponatremia in the phase of increased BAC, pointing to possible suppression of pituitary antidiuretic-hormone release and associated polyuria, but this has not been demonstrated by others. Of note, there tends to be an increased serum osmolality in EtOH intoxicated trauma patients, and changes in serum chloride closely correspond to those in sodium. Hypokalemia may be associated with EtOH intoxication, consistent with our findings of progressively lower serum potassium levels as BAC increased. In other studies, the estimated incidence of hypokalemia in EtOH-intoxicated patients is between 8% and 22%. The aminotransferases, ALT and AST, have been traditionally used as markers of excessive EtOH intake. They have also been noted to be elevated in acute EtOH intoxication. While ALT and AST are excellent markers of hepatocellular injury, aminotransferase levels tend to be only modestly elevated (<300 U/L) in acute EtOH-induced hepatotoxicity, unless other insults such as hemorrhagic shock-induced liver injury are superimposed on alcoholic liver disease. Our study corresponds with these previously published reports, with average ALT/AST consistently below 300 U/L for all BAC categories. Of interest, our data show a pronounced increase in both ALT and AST for patients presenting with BAC between 0.10 and 0.15 g/dL, with notably lower levels for groups presenting with BAC > 0.15 g/dL [Table 2]. Elevations in hepatic function tests have been traditionally seen in acutely intoxicated subjects older than age 30 and in the setting of chronic EtOH abuse.

Elevated bilirubin levels have prognostic significance in alcoholic hepatitis, primary biliary cirrhosis, and in acute liver failure. Not surprisingly, significantly increased serum bilirubin levels, both conjugated and unconjugated, have been observed in patients with acute EtOH intoxication. Our study shows that bilirubin elevations were most prominent with BAC > 0.20, although this was not statistically significant (likely due to a relatively smaller sample of available bilirubin measurements) and the increase was not noted for patients with BAC levels <0.20 [Table 2]. This is generally consistent with previous data showing significant increase in bilirubin in acute EtOH intoxication, with a maximum observed effect at approximately 2 h postexposure. Hypoalbuminemia has been consistently seen in alcoholic patients, often credited to either chronic alcoholic liver disease or the decreased nutritional status of this patient population. In the current study, albumin levels were consistently below 4.0 g/dL across all BAC groups. As has been reported in other clinical scenarios, elevations in albumin can also be seen, attributed to several proposed etiologic mechanisms including acute hemoconcentration. Again, this is consistent with our data, wherein albumin was approximately 14% higher for patients with BAC > 0.15 [Table 2].

Miscellaneous aspects of alcohol abuse in trauma population
Hypertension, stroke, cardiomyopathy, cirrhosis, chronic pancreatitis, brain atrophy, hypogonadism with osteoporosis and sexual dysfunction, gastroesophageal reflux, esophagitis, peptic ulcers, pancreatitis, seizures, and arrhythmias are among the diseases associated with excess alcohol use. Unhealthy alcohol use leads to a myriad of medical, psychiatric, and behavior-related complications. Elevated risk is typically associated with higher levels of EtOH consumption. Of importance, heavy episodic drinking may lead to acute poisoning, a medical emergency where high blood EtOH levels suppress the central nervous system and may cause
loss of consciousness, hypotension and hypothermia, coma, respiratory depression, and potentially death.\[42\] Moreover, previous study published by our group shows a significant correlation between EtOH and polysubstance abuse.\[43-45\]

It is important for health-care providers to remember that many acutely intoxicated patients may suffer from chronic EtOH abuse, along with its associated long-term complications.\[44,45\] These patients may require longer periods of clinical observation and frequent monitoring due to symptoms of withdrawal – a high morbidity and mortality occurrence.\[46\] The aforementioned alcohol-related comorbidities, such as pneumonia, liver cirrhosis, and esophageal varices, often necessitate additional dedicated management and resource utilization.\[45\] The presence of the above associations further underscores the need for accurate diagnosis and identification of alcohol use, particularly in the complex trauma patient. CAGE or AUDIT questionnaires should be utilized to screen for potential alcohol abuse or dependence.\[47-49\]

**Study limitations**

There are several important limitations of this study. The current report represents a single institution analysis, with an arguably preselected (and thus biased) subset of trauma patients. It must be noted that our routine trauma patient evaluation does not usually entail the assessment of CSC, and therefore our study suggests that patients who received BAC screening also were more likely to undergo CSC assessment. This, in itself, is likely a manifestation of selection bias. In addition, possible discrepancies in timing of alcohol consumption, as well as in timing of the BAC laboratory draw could also have significantly impacted our results. The final limitation to consider is that trauma patients included in the current analysis may have had other comorbid health conditions that could have influenced their hematologic and serum chemistry markers.

Research findings on alcohol use in the setting of traumatic injury remain limited by the methodology employed and the populations studied.\[5\] Other useful biomarkers for detecting acute alcohol intoxication that were not evaluated in this manuscript include serum GGT, CDT, and MCV of erythrocytes.\[18,50\] Nonetheless, given the overall paucity of data in this largely unexplored area, we strongly feel that the current contribution will add an important descriptive dimension to the existing body of scientific literature on this important topic, thus encouraging further research. Fleming et al.\[18\] make a strong case for the routine performance of BAL determinations in all patients admitted with traumatic injury.

**CONCLUSION**

The current study carries both descriptive and clinical significance because the knowledge of alcohol-induced hematologic and biochemical changes has the potential to inform both future research in this area and provider perception of hematologic and electrolyte homeostasis in trauma patients. Given the presence of alcohol-mediated variability in key laboratory components, a non-trivial proportion of alcohol intoxicated patients may be at risk of electrolyte and hematologic result misinterpretation. For this reason, the reliability and behavior of biochemical markers in the setting of alcohol abuse should be further explored.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

**Research quality and ethics statement**

This study was approved by the Institutional Review Board. The authors followed applicable EQUATOR Network (http://www.equator-network.org/) guidelines during the conduct of this research project.

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