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Alcohol consumption decreases lactate clearance in acutely injured patients[☆]

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Abstract

Introduction—Alcohol, a common risk factor for injury, has direct toxic effects on the liver. The use of lactate clearance has been well described as an indicator of the adequacy of resuscitation in injured patients. We investigated whether acutely injured patients with positive blood alcohol content (+BAC) had less lactate clearance than sober patients.

Methods—We conducted a retrospective cohort study of acutely injured patients treated at an urban Level 1 trauma centre between January 2010 and December 2012. Blood alcohol and venous lactate levels were measured on all patients at the time of arrival. Study subjects were patients transported directly from the scene of injury, who had an elevated lactate concentration on arrival (≥ 3.0 mmol/L) and at least one subsequent lactate measurement within 24 h after admission. Lactate clearance ($[\text{Lactate}_1 - \text{Lactate}_2]/\text{Lactate}_1$) was calculated for all patients. Chi-squared tests were used to compare values from sober and intoxicated subjects. Lactate clearance was plotted against alcohol levels and stratified by age and Injury Severity Score (ISS).

Results—Serial lactate concentration measurements were obtained in 3910 patients; 1674 of them had +BAC. Patients with +BAC were younger (mean age: 36.6 [SD 14.7] vs 41.0 [SD 19.9] years [$p = 0.0001$]), were more often male (83.4% vs 75.9% [$p = 0.0001$]), had more minor injuries (ISS < 9) (33.8% vs 27.1% [$p = 0.0001$]), had a lower in-hospital mortality rate (1.4% vs 3.9% [$p = 0.0001$]), but also had lower average lactate clearance (37.8% vs 47.6% [$p = 0.0001$]).

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Author contribution statement

All of the authors were members of the research team and (1) participated in the conception, design, and interpretation of the data, (2) contributed to the drafting and revision of the manuscript, and (3) gave approval for the final manuscript being submitted.

Conflict of interest statement

My co-author and I have no commercial associations or sources of support that suggest or constitute a conflict of interest.

The lactate clearance of the sober patients (47.6 [SD 33.5]) was twice that of those with +BAC >400 (23.5 [SD 6.5]). Lactate clearance decreased with increasing BAC irrespective of age and ISS.

Conclusions—In a large group of acutely injured patients, a dose-dependent decrease in lactate clearance was seen in those with elevated BAC. This relationship will cause a falsely elevated lactate reading or prolong lactate clearance and should be taken into account when evaluating patients with +BAC.

Keywords

Trauma; Resuscitation; Alcohol; Lactate; Mortality

Introduction

Traumatic injury remains the most common cause of death in persons between 1 and 45 years of age [1,2]. Lactate, a product of anaerobic metabolism, is produced by all cells in low-oxygen settings [3], particularly traumatic shock [4]. High peripheral venous lactate levels on admission have been associated with a higher mortality rate in patients with sepsis and those who are acutely injured [5–7] and can identify high-risk populations of injured patients who are in shock yet have normal vital signs [4,8]. Lactate clearance, the change in the lactate level in response to resuscitative efforts, has become one of the cornerstones of monitoring trauma care; poor lactate clearance is a marker of both short-term and in-hospital mortality in patients with traumatic shock [8–11]. The value of lactate measurements in trauma care is enhanced by the fact that samples are technically easy to obtain, with arterial and venous sources being essentially equivalent [12–14]. Improvements in portable technology [3,15], have increased interest in the measurement of lactate levels in the prehospital arena to help with triage and identification of high-risk patients.

Alcohol is a common risk factor for traumatic injury. Multiple studies have documented that up to half of all people who die as a result of trauma have a positive alcohol screen [16–18]. Lactic acidosis can be a complication of chronic alcoholism [19], as alcohol has direct toxic effects on the liver [20–22], where the majority of lactate is metabolized [23]. Furthermore, chronic alcoholism is a known cause of cirrhosis and is associated with hepatitis [24], both of which can reduce the functional ability of the liver to metabolize lactate [23]. Previous work has shown that patients with positive alcohol screens have higher initial lactate levels and higher rates of intensive care unit admission than sober patients, but these differences did not result in differences in mortality rate or hospital length of stay (LOS) [9]. The effect of alcohol on lactate clearance in injured patients remains unknown. We hypothesize that injured patients who have a +BAC have slower or lower lactate clearance than those who are sober, in a dose-response relationship.

Materials and methods

Population and setting

This report is based on a single-centre, retrospective, cohort study of patients admitted to a Level I trauma centre between 2010 and 2013. Our data source was the trauma centre's

patient registry, which contains prospectively collected data, including demographics (age, sex, race), clinical information (Injury Severity Score [ISS] [25], vital signs), laboratory values (serial lactate levels), and morbidity and mortality information. The registry is designed to capture data from patients with acute injuries; those with complications of old injuries (e.g., cellulitis and sepsis in a patient with paraplegia) are excluded. Patients with non-traumatic medical (e.g., stroke) and surgical emergencies (appendicitis, ruptured abdominal aortic aneurysm) are seen in an affiliated emergency department, so their data are not recorded in our trauma registry.

From all trauma admissions, we selected those patients who were admitted directly to the trauma centre from the incident scene and who had at least two lactate measurements within the first 24 h after arrival to the trauma centre, the first being elevated (≥ 3 mmol/L) (Fig. 1). Per protocol, the first lactate is drawn immediately on arrival and the second is drawn 6 h later. At our institution, initial lactate concentrations >3 mmol/L are considered abnormal. This threshold has been shown to be more sensitive than the traditional 4 mmol/L without a significant decrease in sensitivity [10]. Patients transferred from other facilities, patients without traumatic injuries, those admitted with complications of previous injuries, and patients who died within 15 min after arrival were excluded. Incomplete records and those with obvious data errors were also excluded. This study was approved by the Institutional Review Board at the institution with which the authors are affiliated.

Data analysis

Lactate clearance, defined as the difference between the first and second lactate level, divided by the first lactate ($(\text{Lactate}_1 - \text{Lactate}_2)/\text{Lactate}_1$), was calculated for every subject and reported as a percentage [3–11,15]. Patients were separated based on whether their initial serum alcohol level (a standard admission test) was positive (i.e., a non-zero value, termed “+BAC”) or zero (“sober”). Patient demographics (age, sex, race), injury characteristics (ISS, mechanism of injury, cause), vital signs, past medical history (alcoholism, cirrhosis, hepatitis, or other liver disease), laboratory values, LOS, and documentation of death at 24 h were obtained for every subject.

Demographics, injury characteristics, vital signs on arrival, initial laboratory values, mortality at 24 h, and past medical history were compared between the sober and +BAC populations using chi-squared tests. The distribution of those subjects with abdominal injuries was compared using a Cochran–Mantel–Haenszel test for ordinal variables. Lactate clearance for the +BAC and sober patients was calculated, and lactate clearance was plotted as function of blood alcohol content for all patients in the +BAC cohort. This group was then stratified by age (14–30, 31–65, >65) and ISS (mild [<9], moderate [9–16], severe [16–25], and critical [>25] injuries), similar to previous studies [18,26]. All analyses were completed using SAS 7.3.

Results

Inclusion criteria were met by 3910 of the 26,454 patients admitted to the trauma centre them (Fig. 1); 2236 (57.2%) of them were sober and 1674 (42.8%) had +BAC when admitted (Table 1). Eight and 424 patients were excluded because of missing blood alcohol

and lactate levels, respectively. There were no significant differences in age, injury severity, vital signs, prevalence of liver disease, or mortality rate across the range of blood alcohol levels, so all patients with a +BAC were grouped together. The intoxicated patients were younger (36.6 ± 14.7 vs 41.0 ± 19.9 years [$p = 0.0001$]), were more often male (83.4% vs 75.9% [$p = 0.0001$]), and had milder injuries (ISS < 9) (33.8% vs 27.1% [$p = 0.0001$]). The proportions of blunt and penetrating mechanisms were similar between the two groups, but the +BAC cohort had a greater proportion of stabbings (14.1%) and assaults (12.7%) and the sober cohort had more motor vehicle crashes (44.0%) and gunshot wounds (14.1%). There were no differences in the distribution of abdominal injuries between the sober and +BAC cohorts ($p = 0.9$). The +BAC cohort had a lower proportion of critical injuries (ISS > 25) (24.2% vs. 29.9%). The +BAC cohort had lower in-hospital mortality (1.4% vs 3.9% [$p = 0.0001$]) and lower lactate clearance (37.8% vs 47.6% [$p = 0.0001$]). The sober patients had a higher average initial lactate level than the +BAC patients (5.8 [SD 3.3] vs 5.4 [SD 2.7] mmol/dL [$p = 0.0001$]), yet they were better able to clear lactate (sober, 47.6% [SD 33.5]; +BAC, 37.8% [SD 31.1] [$p = 0.0001$]). This difference increased with increasing BAC: Sober patients had twice the lactate clearance of those with BAC >400 (23.5% [SD 26.5]).

The sober group had a significantly higher proportion of patients with abnormal INR (international normalized ratio) measurements, but the difference was small (11.3% vs 7.2, Table 2). There was no difference in heart rate or diastolic blood pressure on arrival, and the sober group had a slightly higher systolic blood pressure (140.3 [SD 34.1] vs. 138.2 [SD 31.1] mmHg [$p = 0.03$]). A large proportion of the +BAC cohort had a discharge diagnosis of alcohol dependence (25.8% vs 3.4%, $p = 0.0001$) and cirrhosis (0.5% vs 0.1%, $p = 0.012$), but the two groups were similar in the incidence of hepatitis and other liver disease. To examine patients who could have subclinical liver disease, we compared those with and without a diagnosis of alcohol dependence within the +BAC group. Those with a diagnosis of alcohol dependence had significantly lower lactate clearance (39.8 ± 30.1 vs 32.4 ± 33.7 [$p < 0.0001$]).

Table 3 shows how lactate clearance consistently decreased as admission blood alcohol increased, to the point where patients with a BAC >400 had approximately half the clearance of the sober patients. When made into a scatter plot, lactate clearance rate decreased slightly overall (Fig. 2A). This relationship persists even when stratified by age (14–30 [Fig. 2B], 30–65 [Fig. 2C], 65 [Fig. 2D]) or ISS (25 [Fig. 2H]).

Discussion

In this large single-centre population of injured patients with serial measurements of lactate concentration, we demonstrated that the presence of alcohol decreases lactate clearance in a dose-dependent manner. Lactate concentration remains an important marker of resuscitation; it is useful for identifying injured patients who have normal vital signs but are still at high risk of early death. Relative lactate clearance, a calculation used to determine how a patient is responding to resuscitative efforts, has become an indispensable guide for trauma surgeons and emergency physicians as a predictor of death [4,27]. It can identify those in need of transfusion or surgical intervention, those at risk of organ failure, and those suffering from sepsis. Similar to previous studies, 42.8% of our injured patients had +BAC [8,14–17],

demonstrating the continued need for clinicians to be aware of how to interpret biomarkers such as lactate in the context of common confounding comorbidities such as alcohol intoxication. A number of studies [8,28] sought to determine whether the predictive value of lactate levels measured on arrival changed in the context of acute alcohol ingestion. In this large study of injured patients with serial lactate measurements, those with +BAC exhibited a dose-dependent decrease in lactate clearance across a wide range of ages and injury severity.

Substance abuse is common in trauma patients and is often a contributing factor to their injury. Alcohol, with its ability to lengthen reaction time and lower inhibitions, continues to be a common risk factor for traumatic injury and impedes our ability to evaluate injured patients [9,16]. Dunne and colleagues examined a large undifferentiated cohort of injured patients and found that those with positive alcohol screens had higher initial lactate levels, ISS, and rates of ICU admission compared with sober controls [9]. Gustafson demonstrated that patients with +BAC had elevated lactate concentrations compared with controls on admission to the trauma centre (despite lower injury severity), fewer ICU days, and a shorter overall LOS compared with sober controls. In our cohort, the mortality rate among the +BAC patients was lower than that of the sober patients [28]. The +BAC patients were younger and had less severe injuries (33.9% had an ISS <9; 24.3% had an ISS >25). In our study, the +BAC population also had shorter LOS and lower ISS, in addition to a lower initial lactate concentration compared with controls. Admittedly, we examined an idealized cohort with serial lactate measurements derived from the general population of admitted patients. While this approach allowed us to create a natural experiment from which we could calculate lactate clearance and surmise the effects of alcohol on the clearance rate, the selection process likely introduced biases, explaining why our results diverged from those of Dunne and Gustafson. The difference in mortality rate between our groups is likely explained by the younger age of our +BAC group and their lower ISS.

Hyperlactemia is caused by a combination of increased lactate generation and decreased lactate elimination. All of the patients in this study were injured, and it is reasonable to assume their elevated lactate concentration on arrival was due in part to an increase in lactate in response to the injury. In healthy individuals, lactate generated under stress is quickly normalized, with a half-life of 15–30 min [29]. Patients with cirrhosis have normal lactate levels at rest but, when stressed, can have prolonged elevations of lactate because of their diminished liver function. These same patients regain the ability to clear elevated lactate levels quickly after liver transplantation [30]. Liver disease could be a confounder in this study, but very few subjects had hepatitis, cirrhosis, or liver dysfunction documented in their medical history, and the AST, ALT, alkaline phosphatase levels on admission were similar between sober and +BAC cohorts.

The elimination of alcohol relies on the availability of oxidized cofactor nicotinamide adenine dinucleotide (NAD⁺) [31]. This cofactor is first consumed when ingested alcohol is converted into acetaldehyde via alcohol dehydrogenase (ADH) and again when acetaldehyde is oxidized to acetic acid by aldehyde dehydrogenase (ALDH). This first oxidation reaction step is reversible and takes place primarily in the cytosol of hepatocytes. The second oxidation step by ALDH is irreversible and helps to quickly remove acetaldehyde, which is

toxic, from the body. Both ADH and ALDH have high affinity for their substrates and quickly become saturated. This reaction pathway produces the reduced form of nicotinamide adenine dinucleotide (NADH) and decreases the availability of NAD⁺ when alcohol is consumed.

Lactate metabolism also relies on the availability of NAD⁺, which is also used when lactate is converted reversibly into pyruvate, NADH, and H⁺ by lactate dehydrogenase in the liver. These metabolic pathways therefore compete for the same substrates. In acute injury, lactic acidosis caused by the increased production of lactate in under-perfused tissues may be exacerbated by decreased elimination secondary to NAD⁺ substrate being made scarce by alcohol consumption. This explanation is supported by two observations: (1) a dose-dependent effect, that is, increasing blood concentrations of alcohol were associated with decreased lactate metabolism, and (2) +BAC patients with diagnosed alcohol dependence, who likely have chronically low reserves of NAD⁺, had lower lactate clearance compared with +BAC patients who were not alcohol dependent.

Clinicians often use the lactate level in conjunction with other laboratory test results to screen patients for occult injuries, especially intoxicated individuals. Our study suggests a flaw in this strategy: a +BAC patient with an elevated lactate concentration could have impairment in the ability to clear lactate in response to resuscitative interventions. Laboratory results might suggest an occult injury and cause the clinician to pursue a workup more extensive than is actually needed. In this era of efforts to contain medical costs and avoid the risks of unnecessary diagnostic procedures, the effect of alcohol on lactate clearance should be taken into account.

Limitations

This work is retrospective and therefore vulnerable to unknown biases. By selecting the high-lactate cohort, we might have introduced confounding. Our data do not prove causality, i.e., that a patient who had consumed alcohol before being injured would experience a slowing of lactate clearance. We do not know if there was a larger proportion of liver-specific injuries in one cohort or the other, but the distribution of abdomen-specific abbreviated injury scores was nearly identical between the two groups. Lactate clearance is time dependent, and the difference in the time between the first and second lactate measurements in the sober and +BAC groups was statistically significant. However, the mean time lapse for the +BAC group was longer, which would have given them more time to process lactate. This time difference likely decreases the magnitude of the observed effect of alcohol on lactate metabolism.

Conclusions

The presence of alcohol decreases lactate clearance in a dose-dependent manner. This effect might be due to the diversion of metabolites needed for the conversion of lactate into pyruvate in the alcohol metabolic pathway.

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Fig. 1.
Consort diagram.

Fig. 2.

Lactate clearance as a function of increasing blood alcohol content [Fig. 2A], stratified by age (14–30 years [Fig. 2B], 30–65 [Fig. 2C], >65 [Fig. 2D]) and ISS (ISS < 9 [Fig. 2E], 9 < ISS < 16 [Fig. 2F], 16 < ISS < 25 [Fig. 2G], ISS > 25 [Fig. 2H]).

Table 1

Demographics of sober and +BAC cohorts.

Population	2 lactates and initial lactate ≥ 3		
	Sober	+BAC	<i>p</i> -value
N	2236	1674	
Age, mean (SD)	41.0 (19.9)	36.6 (14.7)	0.0001
Male sex, count (percent)	1698 (75.9)	1396 (83.4)	0.0001
White	1227 (55.1)	817 (48.8)	0.0006
Black	854 (38.4)	747 (44.6)	
Hispanic	46 (2.1)	47 (2.8)	
Other	100 (4.5)	58 (3.5)	
Injury severity, count (percent)			
<9	607 (27.1)	566 (33.8)	0.0001
9 <ISS <16	530 (23.7)	394 (23.5)	
16 <ISS <25	403 (18.0)	291 (17.4)	
ISS >25	668 (29.9)	405 (24.2)	
Missing data	28 (1.3)	18 (1.1)	
Injury mechanism, count (percent)			
MVC	984 (44.0)	675 (40.3)	0.0001
Falls	435 (19.5)	291 (17.4)	
Stabbing	180 (8.1)	236 (14.1)	
GSW	315 (14.1)	157 (9.4)	
Assault	158 (7.1)	212 (12.7)	
Other	164 (7.3)	103 (6.2)	
Blunt	1658 (74.2)	1238 (74.0)	0.4
Penetrating	506 (22.6)	402 (24.0)	
Missing data	72 (3.2)	34 (2.0)	
Abdominal abbreviated injury score, count (percent)			
0	1661 (74.3)	1152 (68.8)	0.9
1	226 (10.1)	189 (11.3)	
2	103 (4.6)	109 (6.5)	
3	108 (4.8)	112 (6.7)	
4	104 (4.7)	79 (4.7)	
5	34 (1.5)	33 (2.0)	
Initial lactate (mmol/L), mean (SD)	5.8 (3.3)	5.4 (2.7)	0.0001
Relative lactate clearance, percent (SD)	47.6 (33.5)	37.8 (31.1)	0.0001
Minutes between first and second lactate, mean (SD)	304.5 (241.6)	321.0 (219.1)	0.04
Mortality at 24 h, count (percent)	87 (3.9)	24 (1.4)	0.0001

Table 2

Admission clinical data for the sober and +BAC cohorts.

Population	Sober	+BAC	<i>p</i> -value
Admission vitals			
Heart rate (beats per minute), mean (SD)	99.0 (26.0)	99.1 (23.2)	0.8
Systolic blood pressure (mmHg), mean (SD)	140.3 (34.1)	138.2 (31.1)	0.03
Diastolic blood pressure (mmHg), mean (SD)	82.1 (20.9)	82.1 (21.1)	0.9
Past medical history, count (percent)			
Alcohol dependence	77 (3.4)	432 (25.8)	0.0001
Cirrhosis	2 (0.1)	9 (0.5)	0.012
Hepatitis (B, C, or unspecified)	38 (1.7)	29 (1.7)	1.0
Other liver disease	0	1 (0.0)	1.0
Admission lab values			
AST >40 IU/L	802 (35.9)	562 (33.6)	0.1
ALT >40 IU/L	102 (4.6)	81 (4.8)	0.6
ALP >130 IU/L	26 (1.2)	17 (1.0)	0.6
INR >1.2	253 (11.3)	120 (7.2)	<0.0001

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Table 3

Lactate clearance as a function of admission blood alcohol content.

BAC (mg/dL)	Sober	1–99	100–199	200–299	300–399	>400
Percent lactate clearance, mean (SD)	47.6 (33.5)	49.9 (30.4)	36.8 (30.5)	28.6 (25.5)	24.1 (37.6)	23.5 (26.5)