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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 ($[Lactate_1 - Lactate_2]/Lactate_1$) 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% [$p = 0.0001$]). 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.

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

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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 ([Lactate$_1$ – Lactate$_2$]/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 Cochrane–Mantel–Haenzel 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

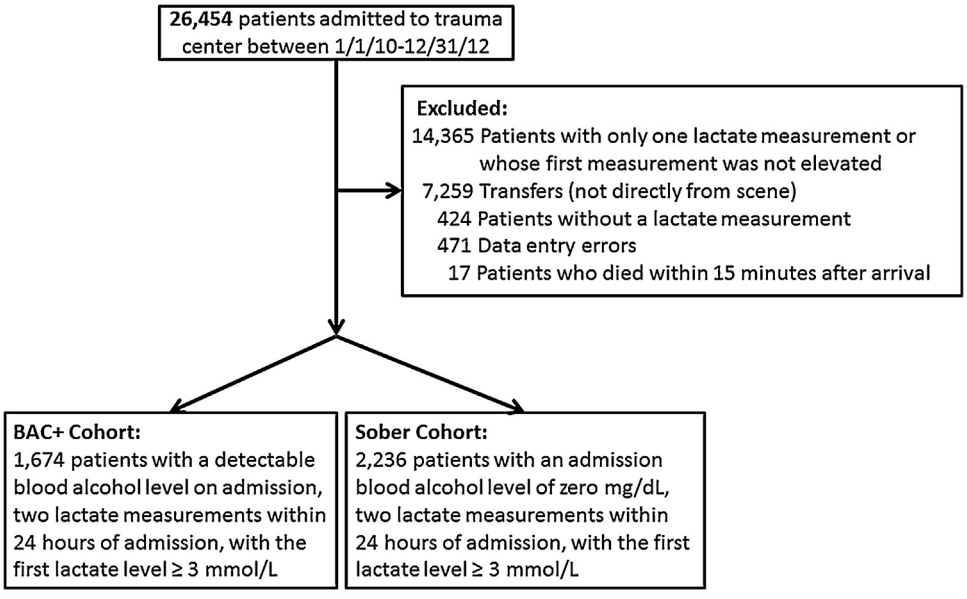

Fig. 1. Consort diagram.

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