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Original Contribution

Quantitative analysis of high plasma lactate concentration in ED patients after alcohol intake☆

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ABSTRACT

Introduction: Plasma lactate concentration is known to increase after alcohol intake. However, this increase has rarely been analyzed quantitatively in emergency department (ED) settings. Evaluating plasma lactate elevation in ED patients after alcohol intake is important because it can affect patients' evaluation based on the plasma lactate level.

Methods: This study analyzed venous lactate concentrations of 196 continuous patients presented to our ED after alcohol intake. The control group comprised 219 successive ED patients without alcohol intake. Patients who had conditions that might induce lactate elevation were excluded from both groups.

Results: Venous lactate concentration was significantly higher in the alcohol intake group (2.83 mmol/L; 95% confidence interval, 2.69-2.96 mmol/L) than in the control group (1.65 mmol/L; 95% confidence interval, 1.53-1.77 mmol/L; $P < .05$). Lactate concentrations exceeding 3 mmol/L and exceeding 4 mmol/L were found, respectively, in 41.8% and 12.2% of the alcohol intake group compared with in 8.7% and 2.3% of the control group ($P < .05$). Lactate concentrations do not correlate with patients' level of consciousness. Therefore, a higher plasma ethanol level is apparently unrelated to elevated lactate.

Discussion and conclusion: Analyses show that plasma lactate concentration is significantly higher in ED patients after alcohol intake and to a greater degree than previously reported, even in patients without previously known alcohol-related diseases. Emergency department physicians must be careful when interpreting the lactate level of the patients with alcohol intake.

1. Introduction

1.1. Plasma lactate concentration as a triage tool or prognosis predicting tool

Plasma lactate concentration is increasingly regarded as important as a prognostic indicator for emergency department (ED) patients. In patients with infection who present to the ED, venous lactate concentration is known to correlate well with the prognosis [1]. Even slightly higher venous lactate concentration (2.0-3.9 mmol/L) can indicate worse prognosis in this population [2]. Venous lactate concentrations tend to be higher than arterial lactate concentrations, but they are equally reliable quantitative data to predict prognosis. Moreover, venous blood tests have the benefit that they are less invasive [3]. In addition to being useful for patients with infection, plasma lactate concentration testing is expected to be useful at a point of care to help determine a triage level at the entrance to an ED [4,5]. For drug overdose patients, plasma lactate concentration is reported at the ED as an independent prognosis predictor [6].

1.2. Alcohol induced elevation of plasma lactate level

Plasma lactate concentration is frequently elevated in ED patients after alcohol intake. Alcohol intake, especially chronic intake, is known to be able to increase plasma lactate concentration slightly by several physiological mechanisms such as elevated NADH/NAD ratio, followed by ethanol metabolism by alcohol dehydrogenase mainly in the liver, or decreased liver function for conversion of lactate into glucose (gluconeogenesis) because of chronic alcoholic liver injury [7]. Only 2 reports have described inebriated ED patients' plasma lactate concentration, but they were conducted more than 20 years ago [8,9]. One report describes that alcoholic liver injury induced by chronic alcoholic intake might contribute to lactate elevation [8]. The other report explains that plasma lactate elevation in many cases is attributable to conditions other than alcohol intake [9]. However, young and healthy patients who present to the ED with simple alcohol intoxication without signs of baseline liver injury or other clinical conditions also frequently present with high plasma lactate concentration. This higher concentration might

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Table 1
Detailed exclusion criteria for the patients

Exclusion criteria	Details
Age	<20 y
Severe conditions	Any conditions that required or caused intensive care unit admission
Specific diseases	Suspected seizure
	Cardiac arrest (anytime before or during hospital arrival)
	Organ ischemia (acute coronary syndrome, arterial or venous thrombosis, aortic dissection, cerebral infarction)
Hypoxia	Oxygen saturation <85% anytime before or during hospital arrival
Low blood pressure	Systolic blood pressure <80 mm Hg anytime before or during hospital arrival
Hypoglycemia	Blood sugar <70 mg/dL
Intoxication	Any kind of medication (including metformin), carbon monoxide intoxication other than ethanol
Anemia	Hemoglobin <7.0 g/dL
Liver dysfunction	Any of the following: total bilirubin >2.0 mg/dL, AST >70 IU/dL, ALT >90 IU/dL, γ-GTP >100 IU/dL, ALP > 480 IU/dL
Renal dysfunction	Acute renal failure with creatinine >2.0 mg/dL

Abbreviations: AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP alkaline phosphatase; γ-GTP, γ-glutamyl transpeptidase.

affect patients' triage, evaluation, or clinical decision at the ED. Analyses of plasma lactate concentration after alcohol intake in healthy volunteers [10] or analysis of blood chemistry changes that do not include lactate after alcohol intake [11] has been reported. However, no report describes a quantitative analysis of plasma lactate concentrations in ED patients after alcohol intake without another condition that might affect the plasma lactate concentration. To settle this clinical question, we conducted a quantitative analysis of plasma lactate concentrations by comparing data of an alcohol-consuming ED patient group to those of a non–alcohol-consuming ED patient group.

2. Methods

Data of people who had recently consumed alcohol and who were seen by emergency medical staff at our ED were collected from the patient database at our hospital from April 1, 2015, through October 20, 2015. These alcohol-consuming patients were selected by searching the text in the "history of present illness" data field in patient notes and the database using specific words such as ethanol, alcohol, drunk, beer, sho-chu, and wine. Then each case was judged. Recent alcohol intake was defined as alcohol intake during the prior 6 hours at the time of presentation to the ED. This cohort was designated as the "alcohol group." A "control group" included people who presented to our ED without recent alcohol intake. Data were collected from August 5, 2015, through October 6, 2015. At our ED, venous blood gas data, including lactate concentrations, were checked for all patients having any abnormality in vital signs (judged according to the criteria shown in Table 1) or any change in consciousness level compared with their baseline (judged using Japan Coma Scale). First venous gas data of both groups obtained after arrival were analyzed. In addition, objective information was collected from each patient. This information included the patient's age, sex, final diagnosis, disposition, vital signs (on arrival), blood test results (including venous blood gas; on arrival), and medical history. Patients were also asked about the history of the present illness, especially the presence or absence of alcohol intake, injury, seizure, syncope, and drug overdose.

Although the patient data were collected retrospectively for this study, the data are objective except for reports of "history of present illness." Regarding "history of present illness," all patients' detailed data were stocked in a comprehensive database. Electronic health records were searched for specific words. The results were subsequently reviewed for individual cases. Consequently, there might be only slight biases related to collection of the patients' data and division of patients into groups. In addition, patients who presented with potential conditions that can affect plasma lactate concentrations were excluded. The conditions included severe diseases that caused intensive care unit admission, suspected seizure, conditions that caused tissue hypoperfusion, drug intoxication other than ethanol, and chronic live disease. Furthermore, people younger than 20 years, the legal drinking age in Japan, were excluded from this study. The patient inclusion and exclusion

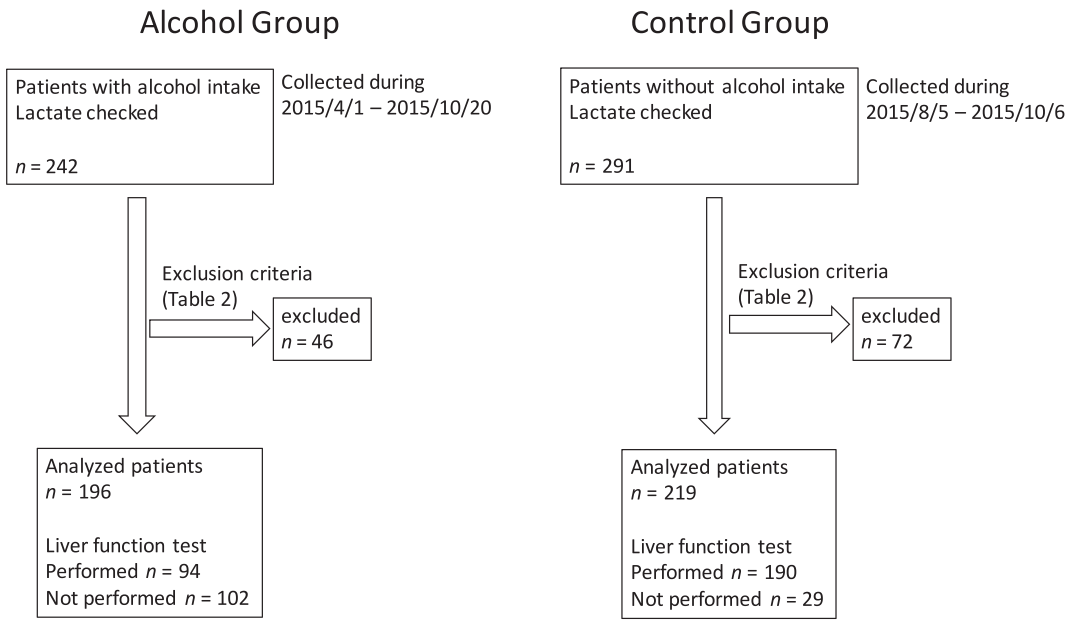

Fig. 1. Patient collection algorithm.