Effect of Alcohol on Death Rate in Organophosphate Poisoned Patients

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Purpose: Many patients who are acutely poisoned with organophosphorus pesticides have co-ingested alcohol. The purpose of this study was to identify the factors that influence mortality in organophosphate intoxication and the differences between alcohol coingested patients and non-coingested patients, looking at vital signs, length of admission, cholinesterase activity, complications, and mortality.

Methods: All patients visiting one Emergency Department (ED) with organophosphate intoxication between January 2000 and December 2012 were reviewed retrospectively. The patients were divided into two groups, alcohol coingested group and non-coingested group.

Results: During the study period, 136 patients (alcohol coingested group, 95 patients; non-coingested group, 41 patients) presented to the ED with organophosphate intoxication. Seventy-one alcohol coingested patients (74.1%) vs. 16 non-coingested patients (39.0%) received endotracheal intubation, with results of the analysis showing a clear distinction between the two groups ($p=0.001$). Twenty-three alcohol coingested patients (24.2%) vs. 1 non-coingested patient (2.4%) required inotropics, indicating a significant gap ($p=0.002$). Twenty-eight alcohol coingested patients (29.5%) vs. 2 non-coingested patients (4.9%) died, with results of the analysis showing a clear distinction between the two groups ($p=0.002$).

Conclusion: In cases of organophosphate intoxication, alcohol coingested patients tended to receive endotracheal intubation, went into shock, developed central nervous system complications, and more died.

Key Words: Organophosphate poisoning, Alcohols, Mortality

Introduction

Two hundred thousand people die globally every year from organophosphate poisoning, and it represents an important medical problem, with a death rate of 15%~30%\cite{1}. Poisoning by organophosphate agents inhibits acetylcholinesterase, inducing cholinergic...
hyperstimulation in the central nervous system, the neuromuscular junction, and the autonomic nervous system, resulting in symptoms such as anxiety, miosis, increased secretion and bradycardia. In serious cases, organophosphate poisoning may cause death through respiratory failure and shock, requiring intensive ICU care. Respiratory failure reportedly occurs in up to 70% of organophosphate poisoned patients, and Grmec et al. reported that respiratory failure often occurs in patients with conscious degradation. Eddleston et al. reported a high death rate in response to the combination of organophosphate and alcohol due to the aggravation of conscious degradation and respiratory failure of the central nervous system. However, studies on the effect of alcohol consumption in organophosphate poisoned patients are rare, and the few existing investigations only reported the relationship between organophosphate blood concentration and blood alcohol concentration, but did not consider clinical characteristics.

Therefore, we analysed clinical characteristics in organophosphate poisoned patients who consumed alcohol, and determined its relationship with the death rate.

Methods

1. Study design

This was a retrospective cohort study conducted, for 12 years from January 2000 through December 2012, on organophosphate poisoned patients visiting 5 emergency centres. Organophosphate poisoning was determined through the poisoning registry, and only registered patients poisoned with a single organophosphate agent were included in this study. Patients under the age of 18 with a medical history of cirrhosis, cancer, or deteriorated nutritional status were excluded from the study. A total of 136 patients were selected as subjects.

2. Subject and Data collection

This study targeted patients that visited the ER with organophosphate poisoning, and the data was collected through handwritten charts and electronic medical records. Data regarding general patient characteristics (age, gender, vital signs, poison amount, reason of poisoning, concomitant alcohol consumption and cholinesterase concentration), clinical characteristics (major symptoms, Acute Physiology and Chronic Health Evaluation [APACHE] II score, Namba classification, and aspiration pneumonia), treatment (ICU hospitalization, intubation and inotropics usage, and atropine dosage) and results (length of stay in ICU and hospital in-hospital death rate) were collected. Patient and guardian statements were used to determine the poison amount, and Gas Chromatography (7694 Series II, HewlettPackard, Avondale, PA, USA) was used to determine the patient’s blood alcohol concentration. Pseudocholinesterase concentration was measured using a colorimetric assay (Cobas c701, Roche, Indianapolis, IN, USA). The first concentration measured at the ER visit was used as a standard.

3. Data Analysis

First, we divided organophosphate poisoned patients into two groups according to concomitant alcohol consumption. Differences in general and clinical characteristics, treatment, and results were compared between the two groups. The difference in the death rate between the two groups was compared as the primary result, and complication and treatment differences were compared as secondary results. The normality of the data was verified through the Kolmogorov-Smirnov test. Chi-square or the Fisher’s exact tests were used for categorical variable analysis, and presented as percentage (%) and frequency. The Mann-Whitney U test was used to analyse continuous variables, which are presented as median and quartile. SPSS 18.0.0 (SPSS, Inc., Chicago, IL, USA) was used for statistical analysis, and its statistical significance was set to 0.05.