G42 Determination of β — Phenylethylamine Blood Levels in Carbon Monoxide Intoxicated-Related Fatalities

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The goal of this presentation is to study the β-Phenylethylamine (PEA) blood levels and its metabolic enzyme, an oxygen-dependent monoamine oxidase B (MAOB), during the hypoxic status induced by carbon monoxide intoxication cases with or without oxygen resuscitation.

PEA, a specific substrate of MAOB, is a biogenic amine that acts as a sympathomimetic amine through its release of dopamine. The rate-limiting step of the MAOB activity of monoamine deamination is a highly oxygen-dependent phenomenon. Carbon monoxide (CO) has a high affinity for hemoglobin that is about 200 times greater than oxygen. CO causes a decrease of the oxygen-carrying capacity of the blood and induces a hypoxic, irreversible status even after re-inhalation of oxygen during resuscitation. The hypothesis is that reduction of the activity of MAOB during the hypoxic status could cause an accumulation of PEA and may be associated with the duration of hypoxic and agonal status. Elevation of PEA blood levels in asphyxia-related fatalities may be related and can be reversed after additional oxygen resuscitation.

A retrospective study consisting of 67 cases of carbon monoxide poison-related fatalities and 121 control cases of CO-unrelated asphyxia and cardiogenic fatalities were collected from the Institute of Forensic Medicine, Ministry of Justice during a medicolegal investigation in Taiwan. Gas Chromatography/Mass Spectrometry was performed to determine the PEA concentrations of each victim's blood. Carboxyhemoglobin (COHb) satu- ration was determined by Oximeter. Data are reported as mean ± standard error mean (SEM). The statistical analyses were carried out with ANOVA by SPSS and *p* values of less than 0.05 were considered to be statistically significant in this study.

Base on COHb saturation levels, PEA blood levels of groups of COHb 20-50%, 50-70%, and higher than 70% were 140.72±41.81 (n= 16), 107.34±25.63 (n= 26) and 66.36±18.03 (n= 25) ìg/ml, respectively. The PEA blood levels of asphyxia cases (including strangulation and suffocation) recognized as non-CO intoxicated-related fatalities with and without resuscitation were 1.6 ± 0.4 ìg/ml (n= 11) and 31. 7 ± 6.3 ìg/ml (n= 48), respectively. The PEA blood levels of CO poison related fatalities with and without resuscitation were 64.75 ± 32.42 ìg/ml (n= 9) and 105.49 ± 17.47 ìg/ml (n= 58), respectively. The mean PEA concentrations in the blood of strangulation and suffocation cases were 83 and 98 fold higher than those of control values, respectively.

In comparison with medical rescue group with decreases in the PEA levels of non-CO intoxicated fatalities during oxygen resuscitation, the CO intoxicated cases with and without resuscitation both have significant elevations in the PEA level. These results reveal that the reversible MAOB activity during oxygen resuscitation can be blocked by the CO saturation of hemoglobin. The high affinity between CO and hemoglobin molecules and the sequential blocking of the oxygenation of hemoglobin can elevat the blood PEA of CO-intoxicated cases without reactivation of the MAOB activity after sequential oxygen re-inhalation. In conclusion, the PEA can play a crucial role of vital reaction in asphyxia-related fatalities. This study strongly supports that the pathological elevation of PEA in the blood during a hypoxic-agonal status can be reversed by oxygen resuscitation but not in CO-intoxicated fatalities.

β-Phenylethylamine, Asphyxia, Biomarker

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