G70 β—Phenylethylamine as a Biomarker in Mechanical Asphyxia-Related Fatalities

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The goal of this presentation is to establish β—Phenylethylamine (PEA) as a biomarker of asphyxia during medicolegal investigation by characterizing the rate-limited step of oxygen-dependent monoamine oxidase B (MAOB).

This presentation will impact the forensic community and/or humanity by showing how the elevation of PEA concentration in blood may play a crucial role in asphyxia-related fatalities. A PEA blood level higher than 2.7 £gg/ml can play a diagnostic role in the determination of asphyxia during the medicolegal investigation. An endogenous substance, PEA allows forensic scientists to develop a state-of-the-art biomarker using the rate-limiting step of MAOB to specify the cause of death in asphyxia.

Identification of asphyxia depends on various non-specific parameters in forensic medicine, such as signs of petechial hemorrhages, cyanosis, engorgement of right heart chambers, lung congestion, and a variety of signs in mechanical asphyxia. The cause of death in asphyxia depends on the history and the exclusion of other causes. It is imperative to develop a quantitative and specific biomarker to interpret the scientific evidence and to ensure a precise diagnosis of asphyxia during the medicolegal investigation. PEA, a specific substrate of MAOB, is a biogenic amine and acts as a sympathomimetic amine through its release of dopamine. The ratelimiting step of the MAOB activity of monoamine deamination is a highly oxygen-dependent phenomenon. The hypothesis is that reduction of the activity of MAOB during the hypoxic status could cause an accumulation of PEA in human body fluids.

A retrospective study consisted of forty-one cases of mechanical asphyxia and thirty-seven cases unrelated to asphyxia that were collected from the Institute of Forensic Medicine, Ministry of Justice, during medicolegal investigation in Taiwan. There were sixteen strangulation fatalities where the causes of death were by manual strangulation (hand or ligature) or hanging. In twenty-five cases of suffocation with mostly choking on food, fixing a pad or gag over the face, and drowning were concluded to be the causes of death. The control group of fatalities unrelated to asphyxia included sudden death by cardiac failure, gunshot injury, violence, and traffic and falling accidents. *In vitro* study in human platelets and *in vivo* animal models in rats were used to monitor the PEA alternation during the hypoxic status. Gas chromatography/mass spectrometry was performed to determine the PEA concentrations of each forensic fatality's body fluids and of each animal specimen.

The PEA blood concentrations of strangulation, suffocation and control cases were 34.2 ± 7.7 µg/ml (mean ± SEM, n=16), 33.0 ± 6.7 µg/ml (n=25) and 0.16 ± 0.03 µg/ml (n=37), respectively. The PEA blood levels of asphyxia-related fatalities were significantly higher than those of control cases (p<0.005). There was no difference in PEA blood levels between suffocation and strangulation cases. The PEA urine concentrations of strangulation, suffocation and control cases were 1.5 ± 1.1 µg/ml (n=8), 0.3 ± 0.2 µg/ml (n=9) and 0.2 ± 0.1 µg/ml (n=12), respectively. The PEA gastric content concentrations in strangulation, suffocation and control cases ranged from 0.03 to 124.1µg/ml with no statistical difference between asphyxia and control group. The postmortem interval for the asphyxia and control groups was 4.3 ± 0.8 days and 5.0 ± 1.0 days, respectively.The PEA profile was not affected by postmortem alteration up to 13 days. Decreasing oxidative activity of MAOB and accumulation of PEA are observed during a hypoxic status in human platelets. An animal model to induce a hypoxic status in rats resulted in elevation of PEA blood levels up to two to four times the control value.

In conclusion, elevation of PEA concentration in blood may play a crucial role in asphyxia-related fatalities. The PEA blood level higher than 2.7 µg/ml can play a diagnostic role in determining asphyxia during the medicolegal investigation. An endogenous substance, PEA allows forensic scientists to develop a state-of-the-art biomarker using the rate-limiting step of MAOB to specify the cause of death in asphyxia.

β—Phenylethylamine, Asphyxia, Strangulation

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