K21 Arizona Tea, It's Not For Everyone: An Anabasine Accidental Lethal Ingestion

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After attending this presentation, attendees will be afforded a review of poisoning and fatalities due to anabasine, including the symptoms of anabasine toxicity and the procedure for analysis of the compound by GC/NPD and GC/mS.

This presentation will impact the forensic community and/or humanity by providing the forensic community with data from a recent postmortem case in which anabasine toxicity was determined to be the cause of death. There is scant toxicological literature regarding the minimal lethal concentration of this drug, and presentation of this case may help in compiling such data.

The authors will present forensic science information regarding a death attributed to acute anabasine toxicity. Anabasine (3-(2-piperidiyl) pyridine) or neonicotine is the major alkaloid of the plant *Nicotiana glauca*, commonly known as tree tobacco. The shrub can grow up to 6 meters in height, has large fleshy gray-green leaves, and tubular yellow flowers. The plant has worldwide distribution including Israel, Australia, and South and North America. In Arizona and the desert southwest, it is commonly found along riverbeds. Anabasine ($C_{10}H_{14}N_2$) is similar in chemical structure and pharmacological effects to nicotine. A 50-year-old male transient was living near the river basin of the Salt River outside Phoenix, Arizona. He was witnessed to drink a heated tea-like solution consisting primarily of desert shrubbery and then complained of feeling numb from the level of his mid chest down to his toes. Other transients summoned emergency personnel but resuscitation efforts were unsuccessful, and he was pronounced dead at the scene. The decedent's prior medical history is unknown. A full autopsy was performed approximately 19 hours after death with significant findings being a slightly enlarged heart; moderately congested lungs, and mild diffuse cerebral edema. Routine specimens consisting of femoral blood, urine, vitreous fluid, bile, liver, kidney, brain, and stomach contents were collected for toxicological analysis as well as the tea solution recovered from the scene. What appeared to be leaves were observed in the gastric contents. Blood and urine specimens were subjected to a qualitative analysis using a basic pH drug screen performed by liquidliquid extraction and analyzed by GC-NPD and GC-MS, with volatiles being assayed by GC-FID. The blood was also screened by ELISA for methamphetamine, benzodiazepines, barbiturates, opiates, and benzoylecgonine, with negative results. A trace amount of methamphetamine was found in the urine by GC-NPD and GC-MS. Quantitative analysis of anabasine was performed on all specimens as follows: briefly, to each tube was added 2 mL of specimen, a 100 uL aliquot of internal standards (0.20 mg/L alpha-phenethylamine, mepivicaine, and dibucaine) and a 100 uL aliquot of concentrated ammonium hydroxide. This was then extracted into 10 mL of n-butyl chloride. A back extraction was performed into 3 mL of 0.2N sulfuric acid. A wash was done with 3 mL of n-butyl chloride and then a 100 uL aliquot of 10N NaOH was added and a re-extraction was done into 10 mL of n-butyl chloride. The solvent was decanted to a conical evaporation tube containing 25 uL of isoamyl acetate and evaporated to 10uL. 1 uL of extract was injected into an Agilent model 6890 gas chromatograph equipped with an Agilent nitrogen-phosphorous detector (NPD) and an Agilent 25 meter HP-5 capillary column (0.33 um film thickness). Split injection (10:1) was done at 260ºC. The temperature program was 60ºC for 1 minute then increased to 315ºC for 5.5 minutes at 9º/minute. Under these conditions the retention time of anabasine was 0.65, relative to mepivicaine. The concentration was determined by comparing the peak area ratios of anabasine to the internal standard against a standard curve with linearity demonstrated up to 1.0 mg/L. Fractional volumes were used for samples exceeding linearity. The concentration of anabasine in the decedent's femoral blood was found to be 0.81 mg/L while tissue levels were: brain 1.11 mg/kg, liver 1.78 mg/kg, kidney 1.58 mg/kg, and gastric 34.4 mg/L. The concentration of the tea like solution was determined to be 151.7 mg/L. Anabasine, like other nicotine alkaloids is rapidly absorbed through the gastrointestinal mucosa as well as by the respiratory mucosa and skin. The symptoms of anabasine poisoning are similar to those of nicotine and include hypersalivation, vomiting, diarrhea, hypertension, tachycardia, diaphoresis, headache, dizziness, twitching, auditory and visual hallucinations, and paralysis. The initial mechanism is stimulation of the nicotine receptors but this may be followed with a blockade at the neuromuscular junction, leading to skeletal and respiratory muscle paralysis. Death is always due to respiratory failure and the few previously reported deaths have occurred within one hour of the onset of symptoms.

Anabasine, Nicotiana Glauca, Gas Chromatography/Mass Spectrometry

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