A Fatality Due to Accidental PineSol\textsuperscript{TM} Ingestion*

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Abstract

The case history and toxicological findings of a fatal PineSol intoxication are presented. An 89-year-old white female with Alzheimer's disease accidentally drank PineSol and was subsequently brought to the hospital where she was pronounced dead on arrival. Significant autopsy findings included acute erosive gastritis. There appeared to be no aspiration of PineSol into the lungs. Isopropanol along with \(1\)-\(\alpha\)-terpineol are the two major toxic ingredients of PineSol. The toxicological screening and quantitation of \(1\)-\(\alpha\)-terpineol in postmortem fluids was performed by gas chromatography–mass spectrometry using a simple one-step extraction. Postmortem blood, urine, and gastric levels of \(1\)-\(\alpha\)-terpineol were 11.2 mg/L, 5.76 mg/L, and 15.3 g/L, respectively. Postmortem blood, vitreous humor, urine, and gastric acetone concentrations were 25, 31, 33, and 28 mg/dL. Postmortem concentrations of isopropanol were less than 10 mg/dL in the blood, vitreous humor, urine, and gastric contents. The cause of death was ruled acute \(1\)-\(\alpha\)-terpineol intoxication due to accidental ingestion of PineSol, presumably caused by confusion related to Alzheimer's disease.

Case History

An 89-year-old white female afflicted with Alzheimer's disease awoke from bed at approximately 5 a.m. to use the bathroom and get a drink. For reasons unknown the deceased poured herself a teacup of PineSol and drank approximately 100 mL of it. The deceased's daughter found her and put her back to bed after calling a relative for advice. The deceased appeared to sleep for the remainder of the day and evening. Just before midnight the daughter heard her mother moaning and vomiting. She observed that her mother was having difficulty breathing and her lower lip was turning blue. The daughter contacted emergency medical assistance, who arrived to find the deceased in cardiac arrest. Cardiopulmonary resuscitation was performed unsuccessfully. Death was pronounced at 1:40 a.m.

Autopsy showed the body of a well-developed white female of average nutrition with a height of 160 cm weighing approximately 50 kg. Significant findings included acute erosive gastritis, rheumatic mitral stenosis, atherosclerosis, bilateral arterionephrosclerosis, thickened pleural plaques at the right apex, and osteoporosis. Both lungs were well aerated with slightly pink-colored pulmonary parenchyma. The pulmonary vasculature was devoid of thromboemboli, and no hemorrhage

was seen in any part of the upper larynx. A chemical detergent odor emanated from the gastric contents. A general toxicological screen for drugs and ethanol was performed on cardiac blood and urine specimens collected at autopsy using enzyme multiplied immunoassay technique (EMIT), headspace gas chromatography, and gas chromatography–mass spectrometry (GC–MS).

Experimental

Materials

Pine oil and 1-α-terpineol were supplied by Clorox. C-16 hydrocarbon marker was purchased from Aldrich (Milwaukee, WI). SKF-525A (proadifen) was purchased from Sigma Chemical Co. (St. Louis, MO). Acetone, isopropanol, ethanol, n-propanol, sodium hydrosulfite, and sodium chloride were supplied by Fisher Scientific (Springfield, NJ). The chemicals used in the extraction were analytical grade.

A methanolic stock solution was made for 1-α at a concentration of 1 mg/mL. This solution was used to prepare calibration standards of 5, 10, 25, and 50 mg/L. Methanolic stock solutions of C-16 hydrocarbon marker and SKF-525A were made at a concentration of 1 mg/mL to be used as internal standard (IS).

Calibration standards for the acetone and isopropanol were prepared by diluting the purchased stock solution with deionized (DI) water. Standards were prepared at concentrations of 24, 47, and 118 mg/dL for acetone and 39, 79, and 157 mg/dL for isopropanol. Absolute ethanol was azetropically distilled and used to prepare calibration standards of 75, 150, and 300 mg/dL by diluting with DI water. Commercial whole blood controls for ethanol were obtained from Clinical Controls Inc. (Grover Beach, CA) and serum volatile controls were obtained from Utak Laboratories Inc. (Valencia, CA). The IS for the volatiles was prepared by taking 11 mL of a 5% n-propanol solution and diluting it to 4 L with DI water.

Instrumentation

A Hewlett-Packard (Palo Alto, CA) model 5890 GC with a 5971 series mass selective detector (MSD) was employed for the screening and quantitation of 1-α-terpineol. The column was an HP-Ultra-1 cross-linked methylsiloxane column (12-m length, 0.2-mm i.d., and 0.33-µm film thickness). The general toxicological screening procedure used the GC–MS in the splitless mode with a carrier gas flow rate of 0.8 mL/min. The injector temperature was set to 250°C, and the detector was set to 280°C. The initial oven temperature was 80°C held for 1 min and then ramped to 320°C at a rate of 15°C/min for a total run time of 17 min. The MSD was operated in the electron impact (EI) mode using full scan monitoring ions from 40 to 400 atomic mass units (AMUs). The EI mass spectrum and structure of 1-α-terpineol are shown in Figure 1.

The quantitative analysis of 1-α-terpineol used the GC–MS in the split mode (30:1). The oven ran an initial temperature of 40°C held for 1 min, then ramped to 220°C at 15°C/min, and finally ramped to 320 at 50°C/min and held for 1 min. The total run time was 16 min. The retention times for 1-α-terpineol and the C-16 hydrocarbon marker were 5.61 and 9.50 min, respectively (Figure 2). The MSD was operated in the EI mode using full scan monitoring ions from 40 to 160 AMUs. There was sufficient sensitivity in the full scan mode so that it was unnecessary to re-inject the samples under selected ion monitoring (SIM) conditions. The data from the full scan were used to calculate an SIM report for the quantitation of 1-α-terpineol. The quantitative ion for 1-α-terpineol was 136 with qualifiers of 59 and 121. The C16 hydrocarbon marker IS used 71 as the quantitative ion with qualifiers set at 85 and 57.

The headspace analysis for volatiles was performed on a Perkin Elmer Sigma 2000 GC equipped with a flame ionization detector (FID) and an HS-100 headspace autosampler. The column was a 6-ft x 1/8-in. 0.290 Carbowax 1500 on carbopack C support 80/100 mesh stainless steel column. The oven was set to 70°C, the transfer line was set to 90°C, and the detector was set to 250°C. The sample was heated at 60°C for 21 min prior to injection. The oven was programmed to run for 5 min at 70°C. The retention times for volatile compounds were 1.12, 1.66, 2.40, and 3.61 min for ethanol, acetone, isopropanol, and n-propanol, respectively.
Procedure

The qualitative screening procedure that was used to first identify \(\alpha\)-terpineol with GC–MS was a simple one-step liquid–liquid extraction. One-hundred micrograms of SKF-525A was added as IS to 5 mL of urine; 1 mL of pH 9.5 ammonium chloride buffer and 200 \(\mu\)L of chloroform were also added. Following vortex mixing and centrifuging, the aqueous layer was aspirated off, and the remaining chloroform was placed in a 2-mL autosampler vial. One microliter of sample was injected onto the GC–MS. Identification of \(\alpha\)-terpineol was based upon comparison of retention time and mass spectra with those of a reference standard.

Quantitative analysis of \(\alpha\)-terpineol also used a liquid–liquid extraction. To 5 mL of blood and urine and 1 mL gastric contents, 200 \(\mu\)g of C16 hydrocarbon was added as IS. The specimens were buffered with 1 mL of 0.1 M phosphate buffer (pH 6.5) and extracted with 7 mL of hexane/toluene/isoamyl alcohol (90:5:5). All samples were mixed on a rotator for 10 min and then centrifuged. The solvent was transferred to a clean, dry tube and evaporated under nitrogen. All samples were reconstituted with 100 \(\mu\)L of the extraction solvent. The injection volume was 1 \(\mu\)L.

The headspace analysis required the addition of 0.5 mL of sample and 4.5 mL of IS to a 20-mL headspace vial containing approximately 3.5 g of a 5% sodium hydrosulfite in sodium chloride mixture. The vials were capped and vortex mixed for 30 s prior to injection onto the GC.

Results

Screening panel for drugs of abuse by EMMT was found to be negative for both urine and blood. The results of headspace analysis for volatiles are listed in Table I. The limit of quantitation (LOQ) for acetone and isopropanol by this method was 10 mg/dL with a limit of detection (LOD) of 3 mg/dL. The LOD for ethanol by this method was 10 mg/dL.

| Table I. Concentrations of Acetone, Isopropanol, and Ethanol |
|-------------------|----------------|----------------|
| Sample            | Acetone (mg/dL)| Isopropanol (mg/dL)| Ethanol (mg/dL) |
| Blood             | 25             | < 10            | Not detected at 10 mg/dL |
| Urine             | 31             | < 10            | Not detected at 10 mg/dL |
| Vitreous humor    | 33             | < 10            | Not detected at 10 mg/dL |
| Gastric contents  | 28             | < 10            | Not detected at 10 mg/dL |

Postmortem blood, urine, and gastric content concentrations of \(\alpha\)-terpineol are listed in Table II. LOD, LOQ, and linearity studies were not performed for \(\alpha\)-terpineol. The calibration curve, however, was linear with a correlation coefficient of 0.998. All specimens were run at dilutions that fell within the upper and lower limits of the calibration curve.

To determine the amount of PineSol remaining in the gastric contents it was necessary to establish the amount of \(\alpha\)-terpineol in pine oil. According to its MSDS, \(\alpha\)-terpineol accounts for 50% of pine oil. To validate this, a pine oil standard was prepared at 1000 mg/L and compared against the \(\alpha\)-terpineol calibration curve. The amount of \(\alpha\)-terpineol in the 1000 mg/L pine oil standard was found to be 505 mg/L. Therefore, the pine oil used in the PineSol contained 50.5% \(\alpha\)-terpineol, which is consistent with what is reported in its MSDS. Using this information, it was calculated that there was approximately 30 mL of PineSol remaining in the 175 mL of gastric contents.

Discussion

Although there are few data showing toxic blood levels of \(\alpha\)-terpineol or pine oil, there are numerous reports related to pine oil exposure. In fact, toxic exposure to household cleaning agents containing pine oil appears to be a common event. In 1986, 5012 exposures to pine oil were reported to the American Association of Poison Control Centers National Data Collection System (8). In 1997, the number of reported cases increased to 10,232. Exposures in the home accounted for 88.1% of reported cases in 1997 with < 0.1% of exposures resulting in death (9). There appears to be an increase in mortality following pine oil ingestion with age. The basis for the increased susceptibility of the elderly includes decreased volume of distribution, decreased hepatic metabolism and renal clearance, as well as diminished skin, mucous membrane, and gut barriers that may increase absorption (2).

Most deaths attributable to pine oil occur as a result of secondary complications arising from chemical pneumonitis due to pine oil deposition in the lungs. This leads to noncardiogenic pulmonary edema that requires mechanical ventilation, which can increase the risk of infection due to bacterial invasion (7). An 88-year-old woman drank 10 oz. (296 mL) of PineSol and died 16 days later because of pneumonia and progressive multiple organ failure secondary to sepsis as a result of complications arising from secondary care (2).

Ingestion of one-half ounce (14 g) of pine oil has been reported to be fatal to a child (10). Hill et al. (10) reported a nonfatal pine oil ingestion by an 18-month-old male child that resulted in a pine odor to the child's breath and urine as well as lesions on the lips, chin, and bucal mucosal; mild respiratory depression; fever; dehydration; hematemesis; erythema of the oral pharynx; and coma (10).

The contribution of isopropanol towards respiratory and CNS depression needs to be considered because it would have certainly added to the CNS depression caused by pine oil. The isopropanol results in this case are consistent with a person ingesting isopropanol and surviving approximately 12 h. (5)
Thirty to 50% of ingested isopropanol is metabolized to acetone (6). Acetone levels in this case were slightly lower than previously reported in cases of fatalities (8). Because acetone is toxic and a more potent anesthetic than ethanol, the potency of isopropanol as a CNS depressant may be related to the generation of acetone (6).

Death due to pine oil ingestion is rare and normally results from complications arising from the secondary care of chemical pneumonitis. In this case, the deceased drank approximately 100 mL of PineSol, which is equivalent to 20 mL of pine oil. This case is unusual in that medical care was not sought until 19 h after initial exposure. Survival even with immediate medical attention could not be guaranteed, however, because of the advanced age and poor health of the individual. The Medical Examiner ruled the cause of death to be an acute intoxication of 1-α-terpineol because of accidental ingestion of PineSol, presumably as a result of confusion related to Alzheimer's disease.

References