CASE REPORT

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Phenol:Tissue Distribution in a Fatality

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ABSTRACT: A case is reported where phenol, a disinfectant, was ingested and resulted in the death of a 40-year-old white female. Concentrations of phenol were determined in blood (130 mg/L), urine (47 mg/L), bile (187 mg/L), brain (486 mg/kg), kidney (331 mg/kg), muscle (204 mg/kg), liver (228 mg/kg), and stomach content (668 mg) and compared to other cases reported in the literature.

KEYWORDS: toxicology, phenol, tissues (biology), tissue distribution

Phenol, or carbolic acid, is used commercially as a disinfectant. Exposure may occur by inhalation of the vapor, by cutaneous absorption, or by oral ingestion [*1*]. Phenol produces its toxic effect by denaturing and precipitating cellular proteins. The minimum lethal dose by mouth is about 1 g [*2*]. Oral ingestion can result in acute mucocutaneous and gastrointestinal corrosive burns resulting in severe pain and vomiting. Both oral ingestion and extensive application to skin can cause systemic toxicity, which is manifested by transient central nervous system (CNS) stimulation followed by CNS and cardiovascular depression [*3*]. Phenol is metabolized by conjugation to yield phenyl glucuronide and phenyl sulphate; phenol is a metabolite of benzene [*2*]. Deichmann [*4*] has reviewed the local and systemic effects following skin contact.

Case History

A 40-year-old woman with a history of mental problems including alcoholism spoke to her boyfriend the night before her death complaining about her drinking problem. The next morning he stopped to see her and found her dead. Underneath the sofa, the police found a half-full bottle of an unknown liquid, later identified as phenol. Investigators were unable to determine from where or how the decedent obtained the phenol. The body was sent to the

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Office of the Chief Medical Examiner, State of Maryland, for further investigation and examination.

External examination of the body revealed a 5-ft 7-1/2-in. (171.5-cm), 137-lbs (62-kg), normally developed white female with no evidence of recent medical intervention or injury. White foam was present in the nostrils. The back of both hands and nail beds were deeply cyanotic. On opening the body cavities, the pungent odor of phenol was detected. A semi-fluid white froth was observed in the upper airway and this extended into the trachea and both main stem bronchi. All lobes of both lungs were moderately congested with a pale tan edema fluid easily expressed from the cut surface.

There was a tan, crusted corrugated appearance to the folds of the stomach most pronounced in the region of the cardia, beginning just distal to the gastroesophageal junction. This corrugated thickening did not involve the esophagus. Approximately one half of the surface of the gastric mucosa was affected with the prepyloric zone, the pylorus itself being spared. Approximately 400 mL of dark brown liquid were present in the stomach exuding a strong pungent odor. The liver weighed 2130 g and had a smooth surface with blunt anterior margins. The remainder of the examination was normal. Specimens were sent to the laboratory for toxicological analysis.

Experimental Procedures

Materials

Phenol was Baker reagent grade and a 1.7-mg/mL solution in methanol was prepared.

Valproic acid was obtained from Abbott Laboratories (Chicago, Illinois), and a 0.9-mg/mL methanolic solution served as the internal standard (IS) solution.

Hydrochloric acid was Baker reagent grade.

Chloroform was Fisher pesticide grade.

Instrumentation

A Perkin Elmer gas chromatograph with a flame ionization detector and a Sigma 1 Data Station was used for the phenol analysis. The column was GP 5% DEGS-PS on 100-120 Supelcoport (2-m by 2-mm inside diameter [ID]). Helium was the carrier gas at a flow rate of 25 mL/min. The injector temperature was 250°C and the detector temperature was 300°C. The oven was operated isothermally at a temperature of 140°C.

A Hewlett-Packard 5890 gas chromatograph interfaced with a model 5970 mass selective detector was used to confirm the presence of phenol in the urine. A HP-1 cross-linked methyl silicone gum capillary column (12-m by 0.33-μm ID) was used for the analytical separation. Helium (3 mL/min) was the carrier gas. The oven temperature was set and held at 50°C for 1 min, then increased by 10°C/min to a final temperature of 200°C. The injector temperature was 275°C and the source temperature was 200°C.

Phenol Analysis

One millilitre of blood, urine, or tissue homogenate (1 g of tissue to 1 mL of deionized water) was added to 1 mL of 1*N* HCl and 2 mL of chloroform. Internal standard (100 uL) was added. After vortexing for 30 s, the tubes were centrifuged. Two microlitres of the chloroform were injected into the gas chromatograph. The retention time for phenol was 1.47 min; the retention time of the IS was 0.85 min. Quantitation was based on the peak height ratio of phenol to IS in comparison to fortified blood standards. The assay measures free phenol and no endogenous phenolic substances interfered.

TABLE 1—*Phenol concentrations reported in the literature.*

Specimen	Case 1	Case 2	Case 3	Case 4
Blood, mg/L	130	56	27	47
Urine, mg/L	47	NR[a]	NR	NR
Bile, mg/L	187	NR	NR	NR
Brain, mg/kg	486	NR	NR	NR
Kidney, mg/kg	331	NR	NR	NR
Muscle, mg/kg	204	NR	NR	NR
Liver, mg/kg	228	76	NR	33
Stomach content, mg	668	NR	NR	neg
Lung, mg/kg	NR	NR	NR	neg
Blood ethanol, %	0.19	0.30	0.30	NR
Reference	present	*2*	*2*	*5*

[a]NR = not reported.

Results and Discussion

The blood submitted in this case was screened for: (1) ethanol, methanol, isopropanol, and other volatiles by gas chromatography; (2) ethchlorvynol, salicylates, and acetaminophen by color tests; and (3) barbiturates and basic drugs by gas chromatography. An ethanol concentration of 0.19% was detected; color tests and the barbiturate and basic drug screen did not detect the presence of any drugs. Drug concentrations of phenol in biofluids and tissues from the present case are shown in Table 1, Case 1, and suggest that phenol was well distributed throughout the central body compartments.

No reliable estimates of the mean lethal dose in man have been documented. Animal studies have estimated that 2% of the ingested amount of phenol is sequestered by both the blood and the liver [*6*]. Assuming this, it can be estimated from the blood and liver that the amount ingested was between 24 and 26 g. This is substantially greater than the minimum lethal dose of 1 g. Blood and liver phenol concentrations in the present case are the highest reported. Relatively, the brain showed the highest concentration, which may be due to the lipophilic property of phenol. It was concluded that phenol intoxication was the cause of death. Because of the limited history surrounding the death, the manner of death was undetermined.

References

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