

## CASE REPORT

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### A Case of Suicide Involving the Concomitant Intravenous Injection of Barbitol and Oral Ingestion of Arsenic

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**ABSTRACT:** A case of suicide involving the intravenous injection of barbitol and the oral ingestion of arsenic trioxide is reported. The pathologic and toxicologic findings are discussed.

**KEYWORDS:** toxicology, barbiturates, arsenic, poisoning, toxicologic analysis, thin-layer chromatography

Drugs and other potentially toxic compounds are commonly used in suicide attempts. The use of multiple agents during a single episode is common [1]; however, the administration of more than one agent via multiple routes is unusual. We herein report a case of suicide in which barbitol was injected intravenously and arsenic ingested orally.

#### Case History

A 51-year-old black man was brought to the emergency room by ambulance at 12:45 pm, after his wife found him lying unconscious on the front room sofa. The ambulance crew noted shallow respiration. No therapeutic measures were instituted before examination at the hospital by the emergency room physician, who noted fresh needle marks in the left antecubital fossa. The patient was subsequently intubated and admitted to the medical intensive care unit. The patient was lethargic but responded to pain by moving all extremities. A nasogastric tube drained 1000 mL of light brown fluid. Blood pressure was maintained at 85/60 using dopamine. Jugular venous pressure was less than 5 cm water. No acute electrocardiographic changes were detected. A chest radiograph demonstrated pulmonary edema. A lumbar puncture demonstrated no abnormalities. Laboratory studies revealed the following: ar-

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terial blood (intubated, 100% oxygen) had a pH of 7.09, an oxygen pressure of 329 mm Hg, a carbon dioxide pressure of 60 mm Hg, and a 7-mmol/L bicarbonate content; serum—sodium 140 mEq/L, chloride 105 mEq/L, potassium 3.2 mEq/L, blood urea nitrogen 3.7 mg/dL, glucose 249 mg/dL, lactic acid 12.1 mEq/L, amylase 90 units/dL, calcium 10 mg/dL, serum glutamate oxaloacetate transaminase 140 units/L, and serum glutamate pyruvate transaminase 140 units/L. Ketones were not detected in the blood. The acidosis was refractory to the administration of bicarbonate. Over the subsequent 18 h, he received 14-L of fluid, eight ampules of sodium bicarbonate, Solu-Medrol<sup>®</sup>, thiamine, and antibiotics. Output during the same period was 250 mL of urine, 250 mL of gastric drainage, and five copious mucoid yellow stools. The patient deteriorated and died at 6:45 am the next day.

He had been emotionally depressed because of marital difficulties and the loss of his job as a result of alcoholism. He had been employed as a hospital laboratory technician. Two days before admission he had been arrested for flourishing a weapon. His wife stated he sought psychiatric aid on that day at a public mental institution but was allegedly refused.

On the day of the fatal episode his wife had gone to the neighbor's house to use the telephone, at which time the victim was reportedly asleep on the sofa. Upon her return home she noted that her husband was incoherent and slurred his speech. She went to get dressed for work and 1 h later found him unconscious, at which time she called the ambulance. Adjacent to the sofa was an open bottle of arsenic trioxide. In the same room were multiple laboratory supplies, among them several syringes (including a recently used one), sodium fluoride, and barbitol. The victim also had access to Darvon<sup>®</sup>, Tylenol<sup>®</sup> #3, Percodan<sup>®</sup>, and lidocaine. The presence of any of these substances in the house was not reported to either the treating physicians or to the medical examiner until several hours after the death of the victim. The treating physicians interpreted the needle marks to be evidence of drug abuse.

### Autopsy Results

The body was 173 cm (68 in.) long and weighed 116 kg (256 lb). The anterior abdominal wall fat was 4 cm thick. Several recent antecubital fossae needle punctures were present. The fresh left antecubital fossa puncture marks noted upon admission to the hospital could not be distinguished from the punctures that resulted from later therapeutic endeavors. No old injection sites were identified. Each pleural space and the peritoneal cavity contained approximately 200-mL serous fluid. The oral cavity and esophagus were unremarkable. A small amount of bloody fluid was the only material present in the stomach. The gastric and duodenal mucosae were diffusely hemorrhagic. Microscopic sections of the stomach and duodenum disclosed mucosal and submucosal polymorphonuclear leukocytic infiltration and edema. The muscularis was unremarkable. The submucosal infiltrate tended to congregate around blood vessels. The remainder of the gastrointestinal tract showed no significant alteration. The 490-g heart contained multifocal subepicardial and subendocardial hemorrhages. The latter were confined to the left ventricular outflow tract. The left ventricle was hypertrophied. The coronary arteries had no significant lesions. Microscopic sections demonstrated several foci of interstitial acute inflammation throughout the myocardium. Scattered myocardial fiber necrosis was present. The liver, which weighed 1570 g, showed evidence of marked fatty metamorphosis along with fibrosis and early cirrhosis. Mitotic figures were not observed. The lungs weighed 1550 g. Pulmonary congestion and edema were present, and many pulmonary hyaline membranes were observed. The brain was unremarkable except for gliosis in the right temporal lobe, probably the result of previous surgery related to an old gunshot wound.

### Toxicologic Findings

Barbital was extracted from urine by XAD-2<sup>®</sup> resin and the resultant residue analyzed by thin-layer chromatography (TLC) [2]. Barbital was indicated by a mercuric sulfate-positive

spot (purple) at  $R_f$  0.56. Barbitol was extracted from blood and tissue homogenates (diluted 1:1 with distilled water) at physiologic pH with chloroform, which was then washed with distilled water and pH 7.4 phosphate buffer and evaporated to dryness. The residues were analyzed by TLC. Spots presumed to be barbitol were eluted from the TLC plates by the method of Freimuth [3] and analyzed by differential ultraviolet spectrophotometry (UV) [4] and gas-liquid chromatography (GLC) [5]. The retention index of barbitol on SE-30 liquid phase was 1490.

Arsenic was identified in urine and tissue homogenates by the Reinsh test as described by Kaye [6]. Quantitative determination of arsenic in blood and tissues was made by the silver diethyldithiocarbamate colorimetric method as described by Sunshine [7]. Routine drug screening procedures utilized in our laboratory [8] and specific analysis for fluoride failed to disclose the presence of other toxicants.

The results of toxicologic analysis of the victim are shown in Table 1. The fluid drained at the hospital from the gastrointestinal tract had been discarded and was not available for toxicologic evaluation.

## Discussion

Neither barbitol, as opposed to other more common barbiturate preparations, nor arsenic is a common suicide agent. In the past ten years in St. Louis City and County, one other death due to barbitol (also in a laboratory technician) has been recorded. Including this case, five arsenic deaths have been encountered. These have occurred in an area currently encompassing 1 500 000 persons of which 18 000 die annually. Of the 6000 deaths reported to the medical examiner, 1500 undergo autopsy.

Although there are several reasons for the infrequent use of either of these drugs, one of the major factors is access. Barbitol is neither as widely available nor as prescribed for therapeutic use as are many of the other barbiturates. In our area, barbitol is not listed in the therapeutic formularies of pharmacies. Barbitol is, however, a common constituent of laboratory buffer solutions [9]. If barbitol is identified in an individual, a laboratory source should be strongly suspected. Both barbitol and arsenic were readily available to the deceased at his former place of employment. Limited toxicologic knowledge probably played a role in the selection of the two agents from among the many lethal compounds available. Whether he intended to take his own life using barbitol alone and then added arsenic when barbitol failed to produce the desired effect (or vice versa) or intended to use both substances from the outset is not known. The onset of symptoms and disability associated with use of either agent may be delayed, for example, an intravenous injection of barbitol usually takes effect approximately 20 min following injection [10].

Lethargy and slurring could be expected to occur at a serum barbitol concentration of 72 mg/L. Patients were found to be awake or rousable at concentrations of 43 to 155 mg/L in a

TABLE 1—Results of toxicologic analysis.

Specimen	Concentration, mg/L or mg/kg	
	Arsenic	Barbitol
Antemortem blood (serum)	0.3	72
Postmortem blood	1.3	53
Injection site	trace	26
Kidney	35	50
Liver	14	57
Spleen	11	69
Urine	present	present
Syringe	absent	present

series of studies by Bailey and Jatlow [9]. The administration of large amounts of fluid may be responsible for the shortened half-life (40.7 h). Barbitol concentrations in the case reported here were below lethal concentrations described in the literature [11].

The gastroduodenal and cardiac findings are typical of acute arsenic poisoning. Arsenic concentrations in this case were within the reported lethal range [12] and within the range seen in our previous experience with arsenic deaths. The discrepancy between antemortem and postmortem blood arsenic concentrations reflects intercurrent continuing absorption.

### Conclusions

We herein report a case of suicide involving the separate intravenous injection and oral ingestion of two uncommonly used toxic agents. The findings support the value of a complete toxicologic evaluation in all cases, including those in which a single obvious lethal substance and route of administration is involved.

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