CASE REPORT

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Homicide by Intravenous Injection of Naphtha

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ABSTRACT: A case of homicide by the intravenous injection of Energine®, a petroleum distillate spot remover, is presented. This case is the only known homicide committed with naphtha. This elderly man had severe natural disease in addition to chest trauma sustained in the assault leading to death; however, the rapid injection of approximately 25 mL of Energine was the overwhelming cause of death.

KEYWORDS: toxicology, homicide, naphtha, Energine®, petroleum distillate, homicide by injection

Naphtha is a petroleum distillate consisting of a mixture of aliphatic hydrocarbons and mono- and di-cyclohydrocarbons with a molecular skeleton of five to eleven carbon atoms, as well as trace amounts of benzene and alkylbenzenes. The literature contains only two case reports of intravenous injection of naphtha in humans, both were attempted suicides [*1,2*]. We present a unique case of intravenous injection of naphtha as a method of homicide.

Case Report

An 80-year-old man was found on the floor of his apartment. His eyeglasses and dentures were lying nearby. His apartment was not in disarray but certain papers, several valuable art objects, and his car were missing. He had last been seen alive the previous afternoon.

At autopsy 4 h later, there was well developed livor mortis over the anterior body surface. There were fresh contusions over the right forearm, left wrist, and both hands. Two fresh needle puncture sites were present in the left antecubital fossa. On incision, these were freshly hemorrhagic and intravenous. There were fresh fractures of the left fourth, fifth, sixth, and seventh ribs anteriorly. The heart weighed 880 g. The epicardial surface was covered with a fibrinous exudate and posteriorly appeared hemorrhagic. There was severe coronary atherosclerosis

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with a recent thrombus occluding the left anterior descending artery. Extensive old fibrous scarring was present in the anterior interventricular system. There was subepicardial hemorrhage in the posterior left ventricle corresponding to the area of external hemorrhage.

Microscopically, there was fresh hemorrhage in the epicardial fat and superficial myocardium of the posterior left ventricle that appeared to be contusion rather than ischemic necrosis.

The police were notified of the autopsy findings and informed that there were intravenous puncture sites that could not be accounted for.

A search was begun for a young male companion of the deceased who had been living in the apartment as a male nurse. Five days later, this man was apprehended and related that he and an accomplice had jumped on the deceased's chest and with a 50-Ml syringe injected him intravenously with Energine®, a naphtha-based spot remover.

Toxicology

Qualitative identificaton of naphtha was based on the comparison of gas chromatograms obtained from blood samples of the decedent with those from drug-free blood to which naphtha (Energine®) or pure hydrocarbon standards (pentane, hexane, heptane, octane) had been added.

Gas chromatographic analysis was by the headspace method as described by Dubowski [*3*]. Analyses were performed using a Perkin Elmer Sigma 2 gas chromatograph equipped with a flame ionization detector. The detector response was recorded with a Perkin Elmer Sigma 10B data station. Analyses were performed using two columns. The first column was a 1.8-m by 2-mm inner diameter glass column packed with 3% SE-30 on Chromosorb WHP, 80–100 mesh. The temperature of the injector was 100°C; the column, 50°C; and the detector, 125°C. The gas flow rate for the nitrogen carrier gas was 28 mL/min; air, 300 mL/min; and hydrogen, 30 mL/min. The retention times (t_R) of the hydrocarbon standards were pentane, 0.56 min; hexane, 0.87 min; heptane, 1.61 min; and octane, 3.3 min. Typical chromatographs obtained from drug-free postmortem blood (blank), naphtha-spiked blood, and the decedent's blood are presented in Fig. 1.

The second column was a 1.87-m by 2-mm inner diameter glass column packed with 10% Carbowax 1500 on Chromosorb, 80–100 mesh. The temperatures of the injector was 125°C; the column, 100°C; and the detector, 150°C. The gas flow rate for the nitrogen carrier gas was 30 mL/min; air, 300 mL/min; and hydrogen 30 mL/min. The t_R of heptane was 0.8 min. Additional samples were analyzed by gas chromatography-mass spectroscopy (GC-MS) using a Hewlett Packard Model 5942 GC-MS equipped with a 1.5-m by 1 2-mm inner diameter glass column packed with 3% OV-101 on 20-mesh Chromosorb. The temperature of the injector was 150°C; the column, 70°C; and the ionization chamber, 180°C. The helium carrier gas flow rate was 30 mL/min. The t_R of heptane was 2.9 min. The resultant electron impact spectra of eluted chromatographic peaks were indicative of hydrocarbons, with consistent cleavage of mass/charge fragments of 14 or 15 mass units. For example, the apparent heptane (molecular weight 100) peak produced major mass/charge fragments of 86, 85, 72 (base peak), 70, 58, 53, and 44 units.

Additional blood and urine samples were analyzed by the Gerarde and Skiba colorimetric method for the detection of kerosene constituents in blood [*4*]. This procedure is based on the reaction of aromatic hydrocarbons in petroleum distillates with formaldehyde–sulfuric acid reagent, forming a chromophore measured spectrophotometrically at 490 nm. Naphtha-free blood and urine specimens from other medical examiner cases produced no color changes. The blood and urine from the decedent in this case were grossly positive.

With the exception of trace amounts of acetaminophen in the urine, general toxicologic screening for acidic, neutral, basic, and amphoteric drugs and volatile compounds failed to disclose the presence of other toxicants.

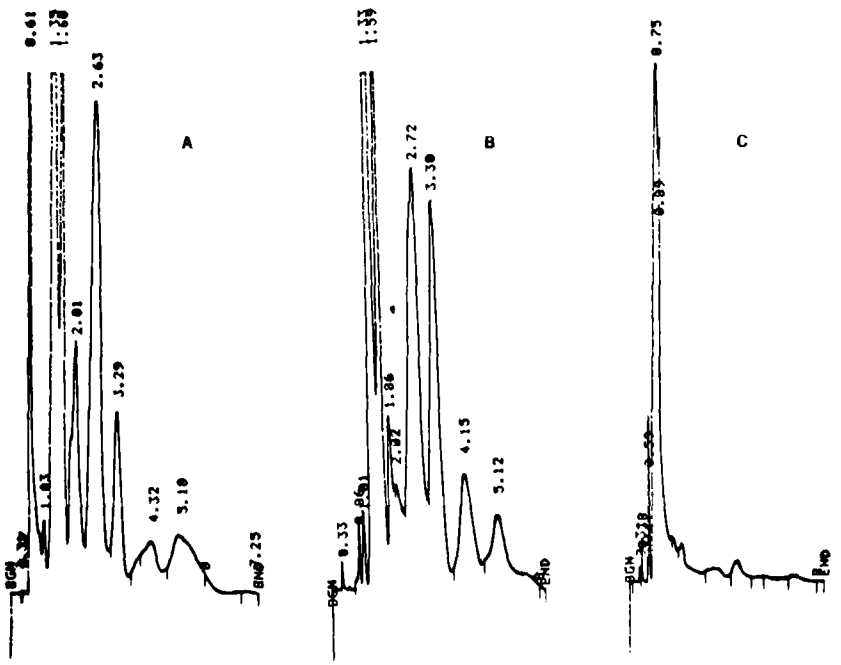

FIG. 1—*Chromatograms on SE-30% liquid phase: (a) blood from decedent, (b) toxicant-free blood to which naphtha (Energine) had been added, and (c) toxicant-free blood. Peaks at 1.60, 3.30, and 5.12 min were consistent with standard heptane, octane, and nonane, respectively.*

Discussion

An unsuccessful suicide attempt by a 21-year-old man who injected about 2 mL of Energine intravenously into each arm was reported by Green [*1*]. The patient had also consumed four to six beers. On admission to the emergency room a short time later, he was lethargic and drowsy but his vital signs and physical examination were normal. Laboratory studies were normal except for a white blood count of 14 300 with left shift and a creatinine phosphokinase (CPK) of 220 (normal 30 to 140). Chest X-ray was normal. He was placed in intensive care on a cardiac monitor for 24 h without the appearance of any cardiac arrhythmias. At no time did pulmonary disease develop. He became febrile on the second hospital day and developed a sterile abscess of the left antecubital fossa that gradually healed. This abscess indicates that possibly the naphtha intended to be injected intravenously was actually injected subcutaneously.

Another intravenous injection of naphtha by a 40-year-old man who injected 3 mL of charcoal lighter fluid in a suicide attempt was reported by Vaziri et al [*2*]. In 2 h, he developed burning chest pain and dyspnea. On admission to the hospital, he had a normal physical examination and chest X-ray. Within 8 h, he developed severe chest pain, shortness of breath, and epigastric pain. He was febrile and tachypneic and had bibasilar rales. During the first two hospital days he produced a bloody sputum and was treated with steroids. A chest X-ray after 12 h showed the development of ill-defined densities in both lungs. Laboratory studies were remarkable for a marked leukocytosis with left shift. On the third day he improved and was discharged two days later without residual pulmonary complication. In contrast to Green's [*1*] case, this case demonstrated a diffuse hemorrhagic pneumonitis following the intravenous injection of only 3 mL of naphtha. This further suggests that Green's patient may have actually injected little naphtha into either vein, as the patient in Vaziri et al showed quite severe toxicity by the intravenous route.

Because of the major problem that petroleum distillates present as a cause of accidental poisoning in children, a large number of studies are available on the effect of the oral ingestion of those products. Accidental ingestions of petroleum distillates in children under 5 years old in the United States number 28 000 annually [5]. These products are the second most common cause of hospital admission for poisoning in children. Of these, kerosene has been the leading agent and remains so in the South. Elsewhere, however, charcoal lighter fluid has taken the lead over kerosene. Charcoal lighter fluid is a naphtha derivatve but somewhat more volatile than kerosene [6]. An evaluation of the toxicity of both products is relevant to the present case.

The ingestion of kerosene results in a hemorrhagic pneumonitis with fever, dyspnea, and rhonchi. The lungs show pulmonary edema, extravasation of blood into the alveolar spaces, and marked congestion of the septal capillaries. Both polymorphonuclear and mononuclear cells appear [7,8]. There are also symptoms of central nervous system depression, as evidenced by lethargy, stupor, coma, and convulsions [8]. The pulmonary effects of kerosene are more the effect of aspiration of some of the hydrocarbon than direct absorption from the gastrointestinal system into the circulation [9].

Results of animal studies substantiate that the pathogenesis of the pneumonitis following kerosene ingestion is due to direct aspiration rather than its absorption into the circulation. In studies of rats, rabbits, and chickens it was found that hemorrhagic pneumonitis results when kerosene is directly instilled into the lungs by the intratracheal route or when it is given by mouth to an anesthetized animal. When kerosene is instilled directly into the stomach, no pneumonitis results. Rats receiving subcutaneous doses develop sterile abscesses at the injection site. Rabbits given rapid intravenous kerosene die suddenly from cardiac arrest, while slower injection causes death in several minutes from pulmonary edema and hemorrhage [10].

Animal studies on charcoal lighter fluid reveal similar findings [6]. If rats have charcoal lighter fluid directly instilled into the stomach, no pulmonary changes occur. When the naphtha product is injected into the trachea, however an acute pneumonitis develops. Injection of the naphtha into the tail vein also results in development of respiratory symptoms and frequent death; at autopsy the lungs show edema and hemorrhagic pneumonitis. The livers of these animals are normal. Injection of the naphtha into the portal vein produces no symptoms; at autopsy the lungs are normal while the livers are large and pale and have extensive necrosis. The authors postulate that naphtha appears to injure the organ bearing the first capillary or sinusoid system through which it passes.

During ingestion of charcoal lighter fluid, kerosene, or similar petroleum distillates, some of the fluid is inadvertently aspirated because of its low viscosity and low surface tension. Clinically, this is evidenced by coughing, gagging, and respiratory distress. Vomiting frequently occurs, with its potential for further aspiration. The damage then is to the lung by aspiration rather than absorption from the gastrointestinal tract. The lethal amount of kerosene and other petroleum distillates is reported to be 90 to 120 mL (3 to 4 oz) when ingested orally [9].

Lethal amounts for the intravenous route of kerosene and naphtha are also uncertain. The animal studies cited above indicate that these substances may certainly be lethal by this route of administration in animals. Rabbits die from cardiac arrest following rapid intravenous injection of kerosene and rats die from pneumonitis following intravenous injection of naphtha.

In the present case, the elderly victim had severe coronary artery atherosclerosis, with a recent thrombus in the left anterior descending artery. The assailants admitted to jumping on his chest, which broke four ribs over the anterior left chest and caused cardiac contusion with considerable traumatic hemorrhage.

The exact amount of naphtha injected into the victim by his assailants is not known. By testimony of one of the attackers, it was established that at least 25 mL (half a 50-mL syringe) and as much as 40 mL may have been administered. Naphtha constituents may cause death by a number of toxic mechanisms: central nervous system depression, anoxia, or cardiac sensitization [11,12]. Given the victim's advanced age and chronic cardiovascular disease, and the severe trauma and stress suffered during physical assault, the rapid injection of 25 mL of

naphtha would be acutely life-threatening or lethal. While stress and cardiac injury may have proved lethal in time, the detection of naphtha in heart blood and urine indicates the victim was alive when administered the spot remover. The absence of severe hemorrhagic pneumonitis indicates that death occurred rapidly following the lethal injection of naphtha.

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