

## CASE REPORT

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# A Fatal Case of Benzene Poisoning

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**ABSTRACT:** Chronic effects following repeated exposure to low doses of benzene have been well assessed, whereas few data are available about acute exposure to benzene. We report a case of fatal acute intoxication which occurred aboard a chemical cargo ship.

Autopsy findings included blood clots inside the heart and main vessels, multi-organ congestion, pulmonary edema and the presence of many vibices in the hypostatic areas.

Toxicological analysis of blood and urine showed a benzene concentration of 31.67 and 2.26  $\mu\text{g/mL}$ , respectively; high concentrations of benzene ( $\mu\text{g/g}$ ) were also found in the lungs (22.23), liver (378.60), brain (178.66), heart (182.57) and kidneys (75.15). The above data provide evidence for benzene distribution in various organs.

**KEYWORDS:** forensic science, acute benzene poisoning, benzene distribution, body fluid and tissue analysis

Benzene is an industrial solvent and a ubiquitous environmental pollutant. Many studies provide evidence for a positive relationship between long-term exposure to low benzene concentrations and lymphatic and haematopoietic cancer (1–3).

It is well known that acute exposure to high concentrations of volatile hydrocarbons results in central nervous system (CNS) depression and death. It is of interest to note that while several cases of death related to benzene occurred up to a few years ago (4–7), only one case report of three fatalities related to acute benzene poisoning has been recently described (8). Indeed, acute poisoning is usually associated with ingestion (9) or inhalation of toluene and xylene (7,10–12); in particular, toluene is often involved in the sudden death of “glue-sniffers” (7,13,14).

It is likely that the paucity of benzene-related deaths is related to the decrease of its industrial use due to its high toxicity. The present case report describes the postmortem findings in a fatality resulting from acute exposure to benzene.

### Case History

In a cargo ship unloading benzene, after the discharge operations, the procedures of washing and ventilating the tanks started.

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After the tanks were washed, the drainage water was expelled and the tanks were ventilated through a blower.

Afterwards, a crew member went inside a tank for inspection without protective equipment. After an unknown amount of time, another crew member heard wailing and went inside the tank to search for his colleague. The vapors forced the rescuer to go out onto the deck, where he lost consciousness, and received first aid. He was then hospitalized for three days with acute benzene intoxication.

After several attempts, the first crew member was finally recovered, unconscious, and he died before arriving in hospital, presumably due to a massive inhalation of benzene vapors.

### Autopsy Findings

This case involved a 41-year-old white male. At the autopsy, performed about 38 hours after the death, the body examination failed to reveal any signs of trauma.

The brain presented marked congestion of the meningeal vasa, edema and congestion of the parenchymal vessels.

The lungs were swollen with abundant blackish frothy liquid. Large blood clots were found inside the cardiac chamber, and multi-organ congestion was evidenced.

### Materials and Methods

The benzene concentrations in the blood and in various organs were measured by gas chromatography using the head space method: one gram of tissue was homogenized with an equal volume of cold water. The homogenate and 40  $\mu\text{L}$  of 0.5% w/v toluene in ethanol as internal standard were poured into 5 mL vials and stoppered with a rubber cap. The blood (1 mL) was treated with the same method. The vials were warmed at 60°C for 30 min, and 0.3 mL of the air phase was analyzed by gas chromatography. A DANI gas chromatograph, model 3600, with a flame ionization detector was used with a 2 m glass column (2 mm inside diameter) packed with GP 60/80 Carbowax B/5% Carbowax 20 M (Supelco). The column temperature was 120°C and nitrogen was used as the carrier gas at a flow rate of 30 mL/min. Drug-free samples of blood were treated in the same manner and used for the preparation of the assay control and calibrators, adding toluene (40  $\mu\text{L}$  of 0.5% w/v in ethanol) as internal standard and benzene (from 20 to 500  $\mu\text{g/mL}$ ).

The toxicological analysis showed the following benzene concentrations: blood 31.67  $\mu\text{g/mL}$ , brain 178.66  $\mu\text{g/g}$ , lungs 22.23

μg/g, heart 182.57 μg/g, liver 378.60 μg/g, kidneys 75.15 μg/g and urine 2.26 μg/mL.

## Discussion

The mechanism by which benzene exerts its toxic effects after acute exposure is not well documented.

It is known that inhalation of benzene results in a rapid absorption through the lungs and skin, and there is considerable evidence that acute exposure to aromatic hydrocarbons affects the CNS. In a study by Ameno et al. (15) it was reported that the brain region/blood toluene concentration ratio was higher in the brain stem region, and lower in the hippocampus and cerebral cortex. Kellerova (16) observed that workers exposed to benzene showed alterations of the electroencephalographic tracing; a rapid onset of deeper sleep stages has been considered typical for benzene exposure.

As regards benzene pharmacokinetics, it has been shown that immediately after inhalation benzene reaches high concentrations in lipid-rich tissues (brain and fat) and well perfused organs (e.g., liver and kidney) however, it is rapidly eliminated, resulting in low concentrations after 1 h (17).

In the present case, high concentrations of benzene were found in the liver, heart and brain; considerable amounts were also found in the kidneys, blood and lungs, whereas only low levels of benzene were detected in the urine. All these concentrations are higher than those reported by Avis and Hutton (8). This could be related to the different circumstances of poisoning. Indeed, in the three fatal cases described by Avis and Hutton, high concentrations were found in the blood and low concentrations in the tissues, suggesting sudden death, while in the present case death occurred within 30 to 45 min, and benzene had time to reach well-perfused and lipid-rich organs.

Probably the benzene initially caused an alteration of the central nervous system with motor incoordination which prevented the victim from escaping. A long exposure in the tank resulted in further inhalation of benzene which led to narcosis and progressive muscular relaxation. It is likely that the state of unconsciousness caused the tongue to relax, causing respiratory obstruction and asphyxia. This hypothesis is supported by the autopsy findings. The presence of vibices in the hypostatic areas could further support this hypothesis. Moreover, the presence of blood clots inside the heart and main blood vessels, multi-organ congestion, and pulmonary edema indicate a slow death.

The above findings demonstrate the peculiarities of the present

case as a typical acute intoxication due to benzene inhalation, providing evidence for its distribution in various organs.

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