Case Report:
Sudden Death Due to Acetone Toxicity

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ABSTRACT

Background: Acetone or propanone is a volatile liquid that can be absorbed by ingestion, inhalation, and dermal routes, and is distributed throughout the body.

Case Presentation: This study presented a case of an employee of the Emergency Medical Center of North Khorasan Province, Iran, who died due to acetone poisoning. He was last seen alive approximately 4 hours before his body was found. Thus, the deceased was found 4 hours after death. He was a 25-year-old unmarried male nurse. He had a history of smoking for the last 4 years and reported diarrhea and nausea on the day before death. There was no history of diabetes or alcoholism as well as no other definite causes. According to interviews with his colleagues and family, he had no history of depression. A postmortem examination was performed to define the cause of death. Postmortem quantitative toxicological analysis with Gas Chromatography with Flame-Ionization Detection (GC-FID) presented acetone in the vitreous (35 mg/dL), blood (28 mg/dL), and urine (77 mg/dL) samples. Furthermore, in postmortem pathological analysis, highly microvascular changes and the diffuse necrosis of hepatocytes were reported. Other pathological changes in the brain and lungs were observed. No other definite medical cause of death was found. The cause of death was determined to be acute acetone intoxication.

Conclusion: Contrary to public opinion, acetone is a toxic and dangerous substance; thus, it is necessary to improve and implement public safeguards concerning acetone usage, as well as its handling, and disposal. Organ toxicity due to acetone can be a major cause of death. Other alcohol-related poisoning deaths, such as acetone and other metabolites, should be considered.

Keywords:
Propanone, Acetone, Intoxication, Forensic, Solvent poisoning, Overdose

Introduction

Acetone or propanone is a volatile liquid, i.e., absorbed by ingestion, inhalation, and dermal, and distributed throughout the body [1, 2]. Acetone is a solvent abused in “sniffing glue” [3]. Acetone is used as a solvent for paints, adhesives, and plastics; it is endogenously produced during fasting or diabetic ketoacidosis [4]. Forensic toxicologists usually encounter high levels of acetone in body

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fluids whenever individuals ingest isopropyl alcohol for intoxication purposes, as ketones are metabolites of secondary alcohols [5].

**Case Presentation**

This study reported the case of a 25-year-old unmarried male nurse with a history of smoking over the last 4 years as well as diarrhea and nausea on the day before death. The deceased worked in the Emergency Medical Center of North Khorasan Province, Iran, and his body was found there 4 hours after death. According to interviews with his colleagues and family, he had no history of depression. A postmortem examination was performed to define the cause of death. In the autopsy, severe pulmonary congestion was reported. Besides, the heart was unremarkable.

Postmortem quantitative toxicological analysis signified acetone in the vitreous (35 mg/dL), blood (28 mg/dL), and urine (77 mg/dL) samples. No isopropanol, drugs, opioids, carbon monoxide, or phosphides were detected in the bile, kidney, liver, or stomach contents. Postmortem pathology examination highlighted diffuse congestion, mild atherosclerotic changes in the coronary arteries, congestion in the lung, pulmonary hemorrhage, highly microvascular changes (Figures 1), the diffuse necrosis of hepatocytes, and the infiltration of inflammatory cells in the liver and brain vascular hyperemia. He was poisoned with acetone and had no other definite medical problems. There was no evidence that how s the amount of liquid was consumed by him. It also remained undiscovered whether it was solvent abuse, suicide, or accidental occupational exposure at work. The cause of death was determined to be acute acetone intoxication.

**Discussion**

The toxicity of acetone is the same as ethanol; although the anesthetic potency of acetone is much greater [6, 7]. Acetone-associated Central Nerves System (CNS) depression can range from mild lethargy with ataxia and slurred speech to deep coma [7]. Rarely, the massive ingestion of acetone results in hypotension, respiratory arrest, and negative cardiac inotropic effects [7]. The 1991 Annual Report of the American Association of Poison Control Centers (AAPCC) National Data Collection System documented 1137 incidents of human exposure to acetone without fatalities [8]. The clearance of acetone is slow and recovery from acetone narcosis does not occur.

![Figure 1. The hematoxylin and eosin staining of the hepatic sample, 400 X](image-url)
for at least 9 hours post-exposure [9]. The excretion of acetone occurs via the urine and breath, especially the breath [1, 6, 10]. Acetone is metabolized to methanolic and ethanoic acids, which induce mild acidosis [9]. Severe toxic effects of Acetone are associated with blood levels of 20-30 mg/dL, and a blood level of 55 mg/dL was detected in fatal cases [3]. The high concentration of acetone in the human samples was observed after the direct ingestion of acetone and isopropanol metabolized to acetone in patients with diabetes [5, 10]. In diabetic ketoacidosis, the range of acetone concentrations almost equals 10-70 mg/dL [10]. Therefore, contrary to public opinion, acetone is a toxic and dangerous substance; it is necessary to improve and implement public safeguards concerning acetone usage, as well as its handling and disposal. Organ toxicity due to acetone can be a major cause of death. Other alcohol-related poisoning deaths, like acetone, should be considered.

**Conclusion**

This report highlighted rare sudden death due to solvents poisoning. Contrary to public opinion, acetone is a toxic and dangerous substance; thus, it is necessary to improve and implement public safeguards concerning acetone usage, as well as its handling, and disposal.

**Ethical Considerations**

**Compliance with ethical guidelines**

All ethical principles were considered in this article. The study protocol was in conformity with the ethical guidelines of the 1975 Declaration of Helsinki, revised in 1983. The authors confirm that an informed consent was obtained from the legal relatives of the decedent prior to drafting this report. Private information, including name and surname was removed from the data sheet to comply with ethical concerns.

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**Authors' contributions**

All authors equally contributed to preparing this article.

**Conflict of interest**

The authors declared no conflict of interest.

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**References**


