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Case Report

# Acute Cyanide Poisoning from Jewelry Cleaning Solutions

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## Abstract

Cyanide is one of the most lethal and devastating poisons. It causes acute toxicity through smoke inhalation simultaneously with carbon monoxide, or by ingestion of cyanide salts that are commonly used in metallurgy and in jewelry or textile industries. Cyanide intoxication is an extremely rare event; in the present study, we report a case of cyanide poisoning involving a 25-year-old jeweler, who ingested a jewelry cleaning solution containing potassium cyanide in a suicide attempt.

**Key words:** Cyanide, Poisoning, Jewelry Cleaning Solution, Suicide.

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**التسمم الحاد بالسيانيد الناتج عن محاليل تنظيف المجوهرات: تقرير حالة**

يُعد السيانيد أحد أكثر السموم القاتلة والأشد فتكاً. حيث أنه يسبب السمية الحادة عن طريق استنشاق الأدخنة الناتجة عن الحرائق مع أول أكسيد الكربون في آن واحد، أو بسبب تناول أملاح السيانيد التي يشيع استخدامها في الصناعات المعدنية وصناعة المجوهرات أو الصناعات النسيجية. ويعتبر التسمم بالسيانيد حدث نادر للغاية. في هذه الدراسة، تم توثيق حالة تسمم بالسيانيد، والتي تمثلت في صائغ يبلغ من العمر 25 عاماً، تناول عن طريق الفم محلول لتنظيف المجوهرات يحتوي على سيانيد البوتاسيوم في محاولة للانتحار.

## Introduction

Cyanide is one of the most deadly rapid-acting poisons. Cyanide toxicity is generally considered to be a rare form of poisoning [1]; however, cyanide has many natural, industrial and even household sources. Exposure occurs frequently in patients with smoke inhalation from residential or industrial fires. Cyanide poisoning may also occur in industry, particularly in the metal trades, mining, electroplating, jewelry manufacturing, and processes involving silver recovery from radiographic films and other silver-containing medical waste. In addition, cyanide salts such as mercury cyanide, copper cyanide, gold cyanide and silver cyanide produce hydrogen cyanide gas when

combined with acids, thus creating the opportunity for industrial accidents or purposeful harmful exposures [2, 3].

Although not a common cause of poisoning, natural sources can produce cyanide poisoning when taken in large quantities or when they are packaged as alternative medicines, such as Laetrile [4]. Cyanide occurs naturally in the form of cyanogenic glycoside (amygdalin) in apricot kernels, bitter almonds and apple and cherry seeds [5]. Cyanogenic glycoside releases hydrogen cyanide after enzymatic hydrolysis when seeds are crushed and moistened.

Cyanide causes intracellular hypoxia by reversible binding to mitochondrial cytochrome oxidase a3 [6]. Signs and symptoms of cyanide poisoning usually occur less than 1 minute after inhalation and within a few minutes after ingestion [7]. Cyanide toxicity is largely attributed to the cession of aerobic cell metabolism, resulting in accumulation of lactate; lactic acidosis is a recognized hallmark of acute cyanide poisoning in humans [8, 9].

## Case report

A 25-year-old jeweler was found dead in his jewelry store a few days after his disappearance. Two flasks containing white and brown fluids were found near the victim and then sent to the toxicology laboratory for analysis.

The body was transported to the morgue for autopsy to determine the cause and circumstances of death. Samples of blood, urine and gastric contents were taken and sent to the toxicology laboratory for further analysis.

The external examination of the body revealed an intense cyanosis on the face and the conjunctival hyperemia without any trauma or signs of violence. The internal examination revealed a congestion of the viscera, pulmonary edema and congestion of submucosal vessels of the upper respiratory tract.

## Methods

A toxicological screening was conducted in order to find out the possible cause of death; the first phase began with a liquid-liquid and solid-liquid extraction of various fluid samples. Acid and basic extracts were separated by thin layer chromatography (TLC) and analyzed by gas chromatography/mass spectrometry (GC/MS).

In addition, barbiturates, opiates, amphetamine, cannabinoids, and cocaine were also evaluated semi-quantitatively by fluorescence polarization immunoassay (FPIA). Cyanide in biological fluids (urine and gastric contents) as well as in the white and brown solutions found at the crime scene was determined potentiometrically using a cyanide ion-selective electrode. This method is based upon the measurement of electrode potential as a function of cyanide concentration in the sample.

## Results

There were no detectable opiates, barbiturates, salicylates, or any other drug in the samples analyzed. The toxicological tests for ethanol and carboxyhemoglobin were entirely negative. However, analysis of urine, gastric contents and two unknown solutions (glass bottles) for cyanide concentration turned out to be highly positive and the results are shown in the table 1.

**Table 1-** ***Cyanide concentration in urine, gastric contents, and white and brown solutions.***

| Samples | Cyanide concentration |
|---|---|
| Urine | 15.5 mg/L |
| Gastric contents | 146.2 mg/L |
| White solution | 16.5 g/L |
| Brown solution | 4.4 g/L |

## Discussion

Cyanide ingestion is frequently lethal because of the early onset of severe symptoms and the difficulty in making an immediate diagnosis. While cyanide poisoning is rarely encountered by physicians, it continues to be used in suicides and homicides [10]. As an intracellular poison, cyanide is potentially lethal because it diffuses into tissues and binds to target sites within seconds [3].

Symptoms depend on the dose taken and the time since ingestion. Oral or transdermal ingestion may result in gradual increases in cyanide concentration levels in the bloodstream, which may cause a delay in signs and symptoms [11]. The three most frequently reported signs of cyanide intoxication are unconsciousness, dyspnea and cyanosis [12]. These clinical manifestations are largely a reflection of intracellular hypoxia [13].

In our case, significantly high cyanide concentrations were found in urine (15.5 mg/L) and gastric contents (146.2 mg/L). Serum concentration of cyanide greater than 0.5 mg/L is typically associated with acute cyanide poisoning [14].

In serious cases of poisoning, cytotoxic anoxia causes an anion gap metabolic acidosis with elevated lactate [6]. The presence of a severe lactic acidosis with a high anion gap may be the most valuable and reliable clue to acute cyanide poisoning [15]. Unfortunately, the blood sample of our victim was not suitable for blood gas analysis. However, lactic acidosis is not specific to cyanide poisoning, and future research is still needed to find a rapid test to aid in diagnosing it.

Concerning the white (16.5 g/L, Table 1) and brown solutions (4.4 g/L) found at the crime scene, the contents of both the bottles were probably metal-shining solutions containing cyanide salts, which are routinely used by jewelers to polish precocious metals such as silver and gold.

The lethal oral dose of the absolute acid (Hydrogen cyanide - HCN) has been reported as 50mg and that of its potassium salt is 200-300mg [13].

These data should confirm the cyanide poisoning of the jeweler by ingesting metal cleaning solution containing cyanide salts in a suicide attempt.

## Conclusion

Ingestion of cyanide salts is a common method of suicide. Its lethality is related to the rapid onset of toxicity, nonspecific nature of symptoms and the failure to consider the diagnosis. Because of the rapidly lethal effects of this toxin, any patient suspected of being poisoned by cyanide should be removed from the source of the exposure, treated with oxygen therapy and an antidote as soon as it is available.

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