Cyanide poisining: An unusual case

Siyanür zehirlenmesi: Nadir bir olgu

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Abstract

Cyanide is one of the most toxic and not easy to reachable element in the nature. In the cyanide toxicity many symptoms can be seen, for example widespread red color livor mortis, widespread hyperemia, and lung edema. So that specialists should evaluate together with toxicological data, autopsy findings and examinations of the scene to define cyanide toxicity. In our case, police found corpse at home. When police examined the scene they got to white color material within a tea glass. In autopsy, specialist examined that livor mortis was formed bright in reddish color on the unpressed side of the body. At the surface of both lungs there were some subpleural hemorrhages which had a tendency to union. In cross section of lungs, %12.8 COHb, 122 mg/ml cyanide, 325 ng/ml lead, 100 ng/ml cadmium were found. As a result of these findings our case was death due to cyanide poisoning.

Key words: Cyanide, toxicology, death, forensic medicine

Özet

Siyanür bugün bilinen en etkili zehirlerden birisidir. Siyanür zehirlenmelerinde sıklıkla görülen bulgular arasında yaygın, parlak kırmızı renkte ölü lekeleri, yaygın hiperemi, akciğer ödemi sayılabilir. Siyanür zehirlenmesi tanısı koyarken; toksikolojik veriler, otopsi bulguları ve olay yeri inceleme bulguları beraber değerlendirilmelidir. Olgumuzda, evinde polisler tarafından ölü olarak bulunduğu, olay yerinde çay bardağı içerisinde erimiş beyaz sıvı madde ile katı bir maddenin olduğu görülmüştür. Otopside ölü lekelerinin açık renkte ve vücudun arka tarafında bası olmayan yerlerde oluşmuş olduğu, her iki akciğerin yüzeylerinde yer yer birleşme eğilimi gösteren yaygın subplevral kanama alanları olduğu kesitlerinde yoğun ödem ve hiperemi olduğu, özofagusta koyu kahverengi kırmızı renk değişikliği olduğu, mide mukozasında koyu kahverengi renk değişikliğiyle beraber midede 150 cc koyu renkte sıvı içerik olduğu tespit edilmiştir. Toksikolojik analizde, kanda %12,8 COHb, 122 mg/ml siyanür, 325 ng/ml kurşun, 100 ng/ml kadmiyum bulunduğu, kişinin ölümünün siyanür zehirlenmesi sonucu meydana geldiği bulunmuştur.

Anahtar kelimeler: Siyanür, zehirlenmeler, ölüm, adli tıp

Introduction

Cyanide is one of the most toxic and not so easy reachable elements in the nature (1). Cyanide can be exists in many forms such as solid, liquid and gases. People exposures cyanide toxicity accidentally or intentionally. Potential causes of acute cyanide poisoning can be inhalation of fire smoke, accidental ingestion, occupational exposure, industrial incident, homicide attempt, suicide attempt, terrorism, ingestion of cyanogenic plants and ingestion of cyanogenic drugs (2).

We can classify sources of cyanide toxicity in 5 different groups:

1. Natural and Manufactured sources:

Cyanide comes from both natural and manufactured sources. More than 2650 plant species can produce cyanide. However the largest risk of exposure comes from manufactured sources (2). Today, cyanide is used in many areas like textile, dye, fertilizing and covering

industry and gold and silver mining, production of metal shiners, disinfectants and photograph equipments (3). Manufactured products using cyanide in their population include nylon, rayon, polyvinyl chloride, polyurethane foam, polyester wadding, rubber, plastics and adhesive resins (2).

2. Cyanide in fire smoke:

A series of studies conducted by Swedish National Testing and Research Institute, illustrates the chemical process by which cyanide compounds are formed during combustion. Hydrogen cyanide is an important cause of smoke-inhalation morbidity and mortality (2). In addition to that cyanide may diffuse to air after burning of some synthetic and natural materials (1).

3. Cyanide in terrorist weapon:

William Krar was sentenced to federal prison for possessing a chemical bomb which included 800 gr of sodium cyanide on May 4, 2004. At the time of the 1995 Tokyo subway attacks, precursors of cyanide were found in the subway bathrooms. Cyanide is plentiful, readily available, requires no special skill to use, and can kill quickly – characteristics that make it an attractive terrorist weapon (2). In the past, cyanide was used to kill many people in the Halepce (1).

4. Industrial cyanide exposure:

Cyanide is used in many industries; human exposure is possible in industrial or occupational. Occupations with potential exposure to cyanide are dye makers, aircraft workers, fertilizer makers, firefighters, gas workers, gold and silver extractors, jewelers, mirror silverers, nylon makers and photographers (2). Mercury cyanide was used in the photographic industry for intensification of images before the appearance of electronic methods (4).The widespread industrial use of cyanides makes these compounds readily available for deliberate acts of self-poisoning, and sporadic cases are to be expected (5).

5. Cyanide used in some drugs (1):

In the hypertension crisis some cyanide containing drugs are used as a last resort at the intensive care units (1) – Cyanide released from sodium nitroprussid. Also cyanide toxicity can cause unexplained cardiac arrest (6).

Cyanides were used in mercuric cyanide or mercuric oxycyanide as antiseptic or hair lotion in European countries and in France before 1997. Capillia lotion in France (or Ocal in Belgium) was used for the treatment of seborrhea, alopecia and itching. They are only obtained in pharmacies with medical prescriptions (4).

Deaths from cyanide poisoning are now rare. Deaths from cyanide poisoning are now relatively rare largely owing to its restricted availability. It is a highly toxic substance and rapidly active with a historical reputation for being employed as "a suicide pill," but more recently has been used in a mass suicide (7).

Case report

He was approximately 35 to 40 years old man. In 17.03.2006 police found his corpse at entrance of the saloon across to the door and they found white color material within a tea glass. In addition to that there were solid cyanide containing bottle and a computer on the table. On the table there were 2 numbers of suicidal and farewell letter. These letters were written with computer using capital letter. At the bottom of these notes were written as “GOODBYE”, name and signature with blue pen. After analyzing of these writings scientists discovered that these handwritings were written with the same pen. In addition to that notes there were victim's daughter, brother and mother telephone numbers which were written at the bottom of the farewell letter. Police didn't find any chaotic condition in the house. When prosecutor interrogated the big brother of the victim, he said that his brother unmarried but he was living with a woman and recently they disputed with each other. Lastly they disputed 2 days ago. He said that his brother worked in a jeweler's workshop. In autopsy, specialist examined that livor mortis was formed bright in reddish color on the unpressed part of the posterior side of body. There were not examined another traumatic lesions on the body. There was hyperemia under the subcutaneous part of hairy skin (Figure 1). And except the hyperemia scientists didn't examine another lesion on the surface and cross section of the cerebrum and cerebellum.

At the surface of both lungs there were some subpleural hemorrhages which had a tendency to union. In cross section of lungs were found edema and hyperemia. Weight and cross section of the some organs can be seen at the table-1. At the surface of the esophagus again experienced dark reddish in color. And trachea was covered with foamy secretion. In the stomach was examined 150 cc dark in color gastric content and dark brown gastric mucosa. And again surface and cross sectional examinational of other part the organs were detected only hyperemia. % 12, 8 COHb, 122 µg/ml cyanide, 325 ng/ml lead and 100 ng/ml cadmium were determined in the blood sample. Specialist explained that cadmium was arisen from white color material. In toxicological examination of urine there were not any hypnotics and narcotics. Histopathological examination of the myocardium investigated huge hyperchromatic disordered cytoplasm. They were diagnosed as perivascular fibrozis. There were erythrocytes at the alveolar lumen and interstitium of the both lungs. Hyperemia was examined in the histopathological examination of the

liver, kidney, cerebrum, cerebellum, small intestine and stomach. As a result specialists evaluated together with toxicological data, autopsy findings and examination of the scene to define cyanide toxicity (1).

Discussion

Prevalence: Cyanide salts are generally colorless solids; HCN (prussic acid) is a colorless gas at room temperature. Significant blood cyanide levels can be found in many fatal and nonfatal cases of fire-related smoke inhalation. Each pack of cigarettes smoked releases 250 to 10,000 µg of cyanide, much of which the smoker may inhale. Cigarette smokers have been found to have mean whole blood cyanide levels of approximately 0.41 mg/L, more than 2.5 times the mean in nonsmokers. Natural sources of cyanide include amygdalin and similar cyanogenic substances found in a wide variety of plants (8).

In the nature cyanide exists mostly in the acrilonitril form. In addition to that cyanide can be found 3 different forms in the nature:

i. Hydrogen cyanide and salt of it

ii. Sulfocyanide, ferri and ferrocyanide

iii. Calcium cyanide

Effectiveness: After absorption, cyanide is rapidly distributed by the blood circulation throughout the body. Cyanide exerts its toxic effects by combining with the ferric iron in cytochrome oxidase, which inhibits the utilization of cellular oxygen (8). When cyanide is taken to body electron transport chains are disturbed via cytochrome oxidases. Because of inhibition of oxygen usage, pyruvate transformation stops. So that metabolic acidosis becomes more clear (1). Cyanide also inhibits other enzymes and can combine with certain metabolic intermediates (8).

Metabolism: Eighty percent of absorbed cyanide is detoxified in the liver by the mitochondrial enzyme rhodanese, which catalyzes the transfer of sulfur from a sulfate donor to cyanide, forming less toxic thiocyanate, which is rapidly excreted in urine. Other detoxification pathways include reaction with hydroxycobalamin (vitamin B12b) to form cyanocobalamin (vitamin B12). Only a small amount of cyanide is eliminated as carbon dioxide by expiration, along with small amounts of HCN (8, 9).

Symptoms: When cyanide enters to body central nervous system is effected firstly. There are 4 phases in acute period (1). The inhalation of HCN can lead to weakness and loss of muscle coordination (8).

a) If cyanide enters to the body via gastrointestinal tract or respiratory system, first symptoms can be vertigo, tachycardia, hypotension, blackout of consciousness

b) Apnea and shallow respiration

c) Convulsion phase

d) Deep comatose phase

When cyanide enters to the body chronically first symptoms can be vertigo, anorexia, mental confusion. In the body normal cyanide ratio should be 0, 01-0, 07 microgram. If cyanide is over the 2 microgram then toxicological symptoms become more pronounced. (1)

Early symptoms include lightheadedness, giddiness, tachypnea, nausea, vomiting, suffocation, confusion, restlessness, and anxiety. Stimulation of the peripheral chemoreceptors produces increased respiration. Otherwise, stimulation of the carotid body receptors slows the heart rate. These changes may be followed by hypoventilation, progressing to apnea and myocardial depression. The result is hypotension and shock, which may rapidly be fatal (8).

Peak whole blood cyanide concentrations lower than 0.2 mg/L usually do not cause symptoms, although poisoning has sometimes occurred at lower levels. Whole blood cyanide levels in smokers may reach 0.4 mg/L without causing symptoms. At cyanide concentrations between 0.5 and 1.0 mg/L, untreated patients may be conscious, flushed, and tachycardic. Stupor and agitation can appear with peak blood levels between 1.0 and 2.5 mg/L. Cyanide levels greater than 2.5 mg/L are associated with coma and are potentially fatal without treatment. According to Baselt and Cravey, the minimal lethal dose has been estimated to be 100 mg for HCN, 150 mg for sodium cyanide, and 200 mg for potassium cyanide. Nevertheless, factors such as age, body mass, state of health, and mode of ingestion may influence these values, and survival after the ingestion of larger quantities has been reported. In this respect, 37 mg of HCN has been fatal, whereas recovery has been reported after the ingestion of 300 mg (8).

Postmortem Examination: Postmortem endogenous cyanide formation in tissue is less important, because the amounts formed are not significant (8). Moreover, blood cyanide concentrations of 0.11–0.15 mg/L were measured in cases of endogenous production. However, cyanide concentration can decrease in blood and tissues significantly after death by evaporation or biotransformation to thiocyanate (4). Postmortem cyanide peak concentrations of only 0.2 mg/L are not likely to be confused with fatal cyanide poisoning (8).

In the cyanide poisoning many symptoms can be seen, for example widespread red color livor mortis, widespread hyperemia, and lung edema though the autopsy findings are, in general, nonspecific (1, 8). In the autopsy of cyanide toxicities scientists can rarely

experience shiny red color livor mortis and snuffing of bitter almond. But these findings are not used in the differential diagnosis. (10) And also snuffing of bitter almond depends on genetic variability so that reason some scientists can not smell this odor. (11) In addition, a striking hemorrhagic appearance of the gastric mucosa has been described, which may be associated with grossly edematous gastric rugae, producing a velvety appearance of the gastric lining. The underlying histologic changes constituted vacuolation of the basal mucosal cells, basal cell dissolutions, desquamation of surface epithelial cells, and discrete inflammatory infiltration of the submucosa (8).

Conclusion: Cyanide poisoning is now uncommon; its availability is largely restricted to certain occupational groups including laboratory and chemical workers. In our case, he had worked in the jewelry shop (7). Because of difficult identification of pathognomonic findings and not so easy within reach element of cyanide identification of deaths from cyanide poisoning are very difficult. So that reason death scene examination, toxicological analysis and autopsy findings should be evaluated together. We believe that sudden cardiac death and metabolic acidosis should be evaluated in the differential diagnosis.

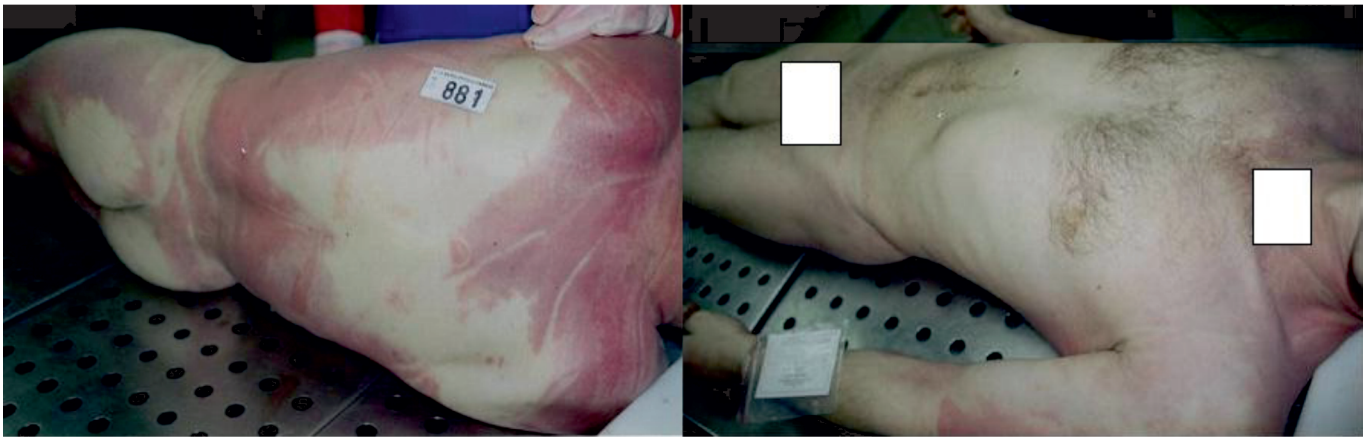

Table 1: Weight and cross section of the some organs

Organ	Weight	Cross section of the organ
Left lung	980 gr	Edema and hyperemia
Right lung	1200 gr	Edema and hyperemia
Myocardium	390 gr	Hyperemia
Cerebrum and cerebellum	1380 gr	Hyperemia
Liver	1750 gr	Hyperemia

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