

# Chemical poisoning: Cyanide poisoning

Shih-Yu Ko, MD; Tzong-Luen Wang, MD, PhD

## Abstract

After the terroric attack in New York on Sep 11, 2001, the medical issues about chemical, biological, radiological, and nuclear terrorism became more and more important. In order to let all emergency personnel to be familiar with the cyanide poisoning, we reviewed articles about cyanide poisoning and introduced the effects of biologic destruction, metabolic mechanism, symptoms and signs of cyanide poisoning. Besides, we also put emphasis on the decontamination, laboratory diagnosis and treatment of cyanide poisoning. We hope this review article can give the readers enough information about how to approach patients exposed to cyanide and decrease the mortality and morbidity rate if there is a cyanide poisoning event. (*Ann Disaster Med.* 2005;4 Suppl 1:S18-S22)

**Key words:** Cyanide Poisoning; Cyanide

## Introduction

Cyanide is found in a variety of substances: plastics, glue removers, wool, silks, nylons, and plants.<sup>1</sup> Numerous forms of cyanide exist, including hydrogen cyanide (HCN) and cyanogen chloride (CNCl), water-soluble potassium and sodium cyanide salts, and poorly water-soluble mercury, copper, gold, and silver cyanide salts.<sup>2</sup> Poisoning may occur iatrogenically such as nitroprusside infusion, inhalation of hydrogen cyanide gas, or during a combustion byproduct of cyanide-containing products.

Cyanide is one of the lethal chemical agent that can be spread as accidental exposure, chemical warfare and act as a terrorism. After the terroric attack in New York on Sep 11, 2001, the knowledge of lethal chemical agents

became more and more important for an emergency physician. In our country, the Republic of China, lethal chemical agents are act as threatening tools to make an illegal profit by evildoers, so all the emergency personnel have to prepare for the scenario: the next patient who appears in front of you might be the victim of chemical agents poisoning such as cyanide.

## Mechanism of cyanide intoxication:

The common sources of cyanide poisoning including: 1) Smoke inhalation of household fires during the burning of common substances such as rubber, plastic, and silk can create cyanide fumes. 2) Photography, chemical research, synthetic plastics, metal processing, and electroplating industries use cyanide. 3) Certain rare plants containing cyanide include apricot pits and

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From Department of Emergency Medicine, Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan  
 Address for reprints: Dr. Tzong-Luen Wang, Department of Emergency Medicine, Shin-Kong Wu Ho-Su Memorial Hospital, No 95, Wen Chang Road, Taipei, Taiwan  
 Received: Aug 22 2005. Revised: Aug 26 2005. Accepted: Sep 1 2005.  
 TEL: 886-2-28389425 FAX: 886-2-28353547 E-mail: M002183@ms.skh.org.tw

a type of potato called cassava. Fortunately, only chronic or massive ingestion of any of these plants can lead to serious poisoning. 4) Certain chemicals, after ingestion, can be converted by the body into cyanide. Most of these chemicals have been removed from the market, but some old artificial nail polish remover, solvents, and plastics manufacturing solutions can contain these substances.<sup>3</sup>

Cyanide is a rapidly acting poison, particularly when it is inhaled. Hydrogen cyanide has a bitter almond odor<sup>4</sup> (The odor of hydrogen cyanide is detectable at 2-10 ppm),<sup>5</sup> but some individuals cannot detect it and consequently it may not provide adequate warning of hazardous concentrations. The lethal dose for adults is 200–300 mg of potassium or sodium cyanide, or 50 mg of hydrogen cyanide.<sup>5-6</sup>

The mechanism of toxicity involves the binding of the ferric ion in mitochondrial cytochrome oxidase, preventing electron transport in the cytochrome system and bringing oxidative phosphorylation and ATP production to a halt. The inhibition of oxidative metabolism increases demands on anaerobic glycolysis, which results in lactic acid production and may produce severe acid-base imbalance.

Once ingested, cyanide is detoxified by enzymatic conversion to the less toxic, renally excreted metabolite, thiocyanate. A small amount of cyanide is also detoxified by the vitamin B12 precursor, hydroxycobalamin. This chelating agent binds cyanide to form nontoxic cyanocobalamin.<sup>8,9</sup>

### Clinical presentation

Cyanide May be absorbed through skin and eyes. If the compound was inhaled, people might have sore throat and persistent running

nose. Then the systemic symptoms appear. Early manifestations of poisoning include anxiety, dyspnea, headache, confusion, tachycardia, and hypertension. All above symptoms and signs will be followed by stupor or coma, seizures, fixed and dilated pupils, hypoventilation, hypotension, bradycardia, heart block, ventricular arrhythmias, and complete cardiopulmonary collapse.<sup>1,2,3,8,10,11</sup>

### Laboratory diagnosis

The diagnosis of cyanide poisoning is usually made on clinical grounds. Because cyanide poisoning is often in the setting of smoke inhalation, the combination with carbon monoxide and cyanide toxicity should always be kept in mind. Blood cyanide levels > 0.5 mg/L are considered toxic.<sup>10</sup> Coma, seizures, and cardiopulmonary dysfunction will happen rapidly in the presence of severe lactic acidosis or an elevated mixed venous oxyhemoglobin saturation (evidence of the blocking of aerobic oxygen utilization).<sup>13</sup> Routine laboratory studies for all exposed patients include CBC, blood glucose, and electrolyte determinations. Additional studies for patients exposed to hydrogen cyanide include ECG monitoring, determinations of serum lactate, chest radiography, and pulse oximetry (or ABG measurements). Besides, the bitter almond scent of hydrogen cyanide gas may also be present.<sup>2,4,5,6</sup>

After treatment with nitrites, serum methemoglobin levels may be monitored. However, the usual methods of monitoring methemoglobin levels are unreliable in cases of cyanide poisoning and may seriously underestimate the levels of inactive hemoglobin. Alternative methods exist, but may not be available. Whole blood cyanide tests generally require several hours and

cannot be used to guide emergency treatment. However, blood cyanide levels may be useful in documenting exposure.<sup>8,9,10,12,13</sup>

### General principles:

The general principles of handling an acute chemical emergency begin with stopping the exposure, which can be accomplished by evacuating or extricating the affected persons, and then by thorough decontamination. Ideally, the persons who might be exposed to chemicals should move upwind of the contaminant and move to an adequate shelter. Then they should remove and bag their clothing and shower thoroughly with soap and water as soon as possible.

Early decontamination of persons with hazardous exposure should be performed by trained first responders. In Taiwan, the first responder may be the EMT personnel or the triage personnel of an emergent department of hospital. And the decontamination spot should be far from the ordinary pathway. Removing contaminated clothing can eliminate 85 to 90 percent of trapped chemical substances.<sup>14,15,16</sup>

The initial supportive therapies of chemical exposure after decontamination focus on airway patency, ventilation, and circulation. Besides, patients might suffer from an explosion, we have to keep in mind that they might have concurrent injuries of burn, or trauma.<sup>1,2</sup>

In cases of ingestion, do not induce emesis. If activated charcoal has not been administered previously, and the victim is alert, asymptomatic, and has a gag reflex, administer activated charcoal (administer at 1 gm/kg, usual adult dose 60-90 g, child dose 25-50 g).<sup>11,12,16</sup> A soda can and a straw may be of assistance when offering charcoal to a child. If the patient is symptomatic, immediately institute emergency

life support measures, including the use of a cyanide antidote kit<sup>1,3,5</sup>

### Treatment

Treatment of cyanide poisoning tends to be effective if started early. Oxygen, decontamination, nitrites, and sodium thiosulfate are the main therapeutic modalities.<sup>11</sup> Oxygen therapy at 100% fraction of inspired oxygen either by face-mask or endotracheal tube should be instituted immediately. Hyperbaric oxygen is as of yet unproved in cyanide poisoning.<sup>17</sup>

Antidote: Cyanide antidotes-amyl nitrite perles and intravenous infusions of sodium nitrite and sodium thiosulfate-are packaged in the cyanide antidote kit. Amyl and sodium nitrites induce formation of methemoglobin. Cyanide has a high affinity for the ferric iron contained in methemoglobin, thereby rendering methemoglobin an effective scavenger of unbound cyanide. Amyl nitrite is administered by inhalation of crushable pearls, which are inhaled for 15 to 30 s with 30 s of rest between inhalations. One pearl lasts approximately 2 to 3 min. This therapy induces a 5% methemoglobinemia and can be used in spontaneously breathing patients or in patients receiving ventilatory support until administration of sodium nitrite.<sup>10,12</sup>

Sodium nitrite is administered IV at a dose of 300 mg ( 10 ml of 3 % solution) over 3 min to convert more hemoglobin to methemoglobin.<sup>12,15</sup> The average pediatric dose is 0.12 to 0.33 mL/kg body weight up to 10 mL infused as above. Monitor blood pressure during sodium nitrite administration, and slow the rate of infusion if hypotension develops.<sup>1,2,10,12</sup> Half this dose may be repeated after 2 h if there is persistent toxicity, and a tol-

erable degree of methemoglobinemia. Usually, methemoglobin levels remain <20% and the reduction of total oxygen-carrying capacity by the combination of carboxyhemoglobin and methemoglobin is 21%.<sup>18</sup>

Next, infuse sodium thiosulfate intravenously. The usual adult dose is 50 mL of a 25% solution (12.5 g) infused over 10 to 20 minutes; the average pediatric dose is 1.65 mL/kg of a 25% solution. Repeat one-half of the initial dose 30 minutes later if there is an inadequate clinical response. Methylene blue should be avoided as a treatment of methemoglobinemia because it will release free cyanide. Sodium thiosulfate.<sup>18,19,20</sup>

Hydroxycobalamin is a promising antidote capable of reducing RBC and plasma cyanide concentrations. In healthy adult smokers, hydroxycobalamin, 5 g IV, decreases whole-blood cyanide levels by 59%. The currently recommended dose of hydroxycobalamin in acute cyanide poisoning is 4 to 5 g IV administered as a one-time dose. However, the FDA has not approved its use in the United States.<sup>21,22,23,24</sup>

## Conclusion

In all serious cases of exposure to cyanide, a successful outcome hinges on the immediate provision of basic life support and decontamination, and follow-up with excellent supportive care. Community preparedness for possible toxic chemical releases requires well-organized emergency-medical-response systems, as well as clinicians and hospitals trained for readiness. Emergency planning should be applicable to both accidental and deliberate chemical releases. Besides, the country should intervene and stockpile antidote kit. If whole

the hinges are set down, the tragedy will be prevented.

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