

Cyanide Poisoning in a Children Caused by Apricot Seeds

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Introduction

Childhood exposure to cyanide is rare despite multiple potential sources including inhalation, dermal absorption or ingestion of cyanogenic foods [1]. Cyanogenetic glycosides, naturally occurring compounds, are found in foods such as apricot, almond, and cassava. The most significant of these compounds is amygdalin. Unlike western world, cyanide poisoning in children in developing countries is mainly related to ingestion of foods containing amygdalin. In Turkey, apricot seeds are the most common food causing to acute cyanide poisoning in children [2]. Because of its infrequent occurrence, pediatricians might have difficulty recognizing cyanide poisoning, confirming its presence, and treating it. Here, we report a fatal cyanide poisoning case caused by apricot seed ingestion.

Case Report

A 28 month old girl presented to emergency department with sudden onset unconsciousness and seizure. Her parents reported that she had eaten apricot approximately ten apricot seeds. Fifteen minutes later she had headache, dizziness and suddenly became unconsciousness. At the presentation she was unconsciousness. Her blood pressure was 92/42 mm Hg. She could not able to breathe spontaneously. Her pupils were miotic with negative light reflex. Glasgow coma score was four. She was transported to intensive care unit and mechanical ventilation started. Gastric lavage performed and pieces of apricot seeds were observed. On laboratory investigation blood gas analysis revealed that pH: 6.8, paO_2 80 mmHg, PaCO_2 15 mmHg, HCO_3 5.5 mmol/L, Base excess -29.6 mmol/L. Plasma lactate level measured as 10 mmol/L. Plasma glucose level was 290 mg/dl The rest of laboratory test including complete blood count, urine analysis and blood biochemistry were all within normal limits. With help of apricot seed eating and laboratory findings, the patient was diagnosed as acute cyanide poisoning. After collecting whole blood sample for measurement of cyanide level, Cyanide antidote dicobalt edetate (Kelocyanor) was given ten hours after presentation to hospital. Repeated arterial blood gas analysis showed that the difference between arterial and venous PO_2 levels was 8 mmHg. The result of whole blood cyanide level was more than 3mg/L at the 20th hour of presentation. Dicobalt edentate was repeated. She died on the 22th day of hospitalization following supportive care in intensive care unit.

Discussion

Most of cases cyanogenic intoxications in Turkey are related to ingestion of apricot seeds by children [2]. Cyanogenetic glycosides, naturally occurring compounds, are found in peache, almond and cassava also. The most significant of these compounds is amygdalin which is a cyanogenic compound found in apricot core. An endogenous enzyme in the seeds converts amygdalin into glucose, benzaldehyde, and hydrogen cyanide. If seeds swallowed completely without chewing, less cyanide is released than if they are chewed. After apricot seeds are eaten cyanide is released in alkaline environment of the small intestine and, with emulsification, is absorbed quickly and circulates in the body [1,2]. Onset of symptoms might varies depend on amount of apricot seeds eaten. This time was 15 minutes for our patient after eating ten

apricot seeds. Akyildiz et al. [2] reported that the mean time to onset of symptoms was 60 minutes and median number of seeds ingested by patients was eight. Although the quantity of cyanide in apricot seeds is not certain, it is reported that intoxication may occur from eating 5 and 25 apricot seed, depending on the age and weight of the child [3]. Also it has been reported that children are more susceptible than adult to poisoning by ingestion of cyanogenic foods. The apparently greater vulnerability of children to poisoning by cyanogenic foods has been attributed to children's lower body mass and children's high gastric acidity than that of adults [1].

The endogenous metabolic pathways for cyanide are rapidly and easily overcome in the event of cyanide poisoning. Cyanide might have multiple toxic mechanisms, some of which have not been established with certainty. The best established and most important toxic action of cyanide is incapacitation of the cell's mechanism for using oxygen resulting in chemical asphyxiation. Cyanide, which has a chemical structure similar to that of oxygen, binds to the ferric iron portion of cytochrome oxidase. The binding of cyanide inhibits the ability of cytochrome oxidase to use oxygen and thereby reduces production of ATP. Because their oxygen-utilization mechanism is disabled, cells are forced to rely on anaerobic metabolism instead of aerobic metabolism. The reduced availability of ATP results in cellular dysfunction and death. In addition, cyanide affects multiple neurotransmitter systems, including dopaminergic, GABAergic, and glutamatergic pathways, either directly or indirectly through changes in iron regulation [4].

Early signs and symptoms of acute cyanide poisoning reflect reflexive attempts of the respiratory, neurologic, and cardiovascular systems to overcome tissue hypoxia. Headache, vomiting, dizziness, and abdominal pain were common presentation symptoms of patients in the study of Akyildiz et al. [2]. Exposure to large doses, particularly by ingestion or inhalation, can produce marked symptoms nearly immediately, like our case, and result in death in minutes.

Diagnosis of cyanide poisoning is difficult. Hallmark laboratory findings in acute cyanide poisoning are metabolic acidosis with marked elevated plasma lactate and excessive venous oxygenation. Plasma lactate concentration normally ranges from 0.5 to 2.2 mmol/L. Like our patient, a concentration > 8 mmol/L suggests the possibility of cyanide poisoning. However, elevated plasma lactate is not specific to cyanide poisoning and therefore does not definitively signify cyanide toxicity [5].

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Received January 20, 2011; Accepted June 30, 2011; Published July 15, 2011

Citation: Sahin S (2011) Cyanide Poisoning in a Children Caused by Apricot Seeds. J Health Med Informat 2:106. doi:10.4172/2157-7420.1000106

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Excessive venous oxygenation, the second main laboratory finding in acute cyanide poisoning, is reflected in a low (< 10 mmHg) arteriovenous oxygen saturation difference (SaO_2-SvO_2) on arterial and venous blood gas analysis. We also found that blood gas analysis collected from arterial and venous lines showed that the difference was 8 mmHg. Excessive venous oxygenation is attributed to the inability of cells to extract and use oxygen from arterial blood [5].

The result of whole blood cyanide level of our patient was greater more than 3mg/L. Blood cyanide concentrations currently have only a confirmatory role in the initial management of acute cyanide poisoning because the results of standard assays are generally not available within the time required to initiate intervention. Although blood cyanide concentrations are not practical in initial management, they are used to confirm toxicity. For cyanide in whole blood, the toxicity threshold for cyanide alone ranges from 0.5 to 1.0 mg/L, and the lethal threshold ranges from 2.5 to 3.0 mg/L [5].

Acute cyanide poisoning is treated by terminating exposure and administering supportive care and antidotal therapy. In the event of poisoning by ingestion gastric lavage and administration of activated charcoal should be performed. Initial supportive care involves the basic life support airway, breathing and circulation (ABC) triad. Administration of 100% oxygen is viewed as an important component of supportive care despite the fact cyanide poisoning involves deficient oxygen utilization rather than deficient oxygen availability. It is hypothesized that increased oxygen delivery could increase respiratory excretion of cyanide [6].

Several cyanide antidotes are available in one or more countries around the world. Dicobalt edentate is the only one present in our country. The hypothesized mechanism of action of dicobalt edentate is chelation of cyanide to form cobaltcyanide ($CoCN_6$), which is much less than toxic cyanide. Dicobalt edentate differs from other commercially available antidotes in that it seems to improve survival when administered very late during course of cyanide poisoning

[7]. Dicobalt edentate treatment was performed at the 10th hour of hospitalization of our case. In some or previously reported cases treated with dicobalt edentate, administration of dicobalt edentate was associated with serious side effects, including cardiac arrhythmia and anaphylactic reaction [7]. No of these was present in our patient. Dicobalt edentate may be particularly harmful when administered to patients without cyanide poisoning. Because of the way dicobalt edentate manufactured, toxic free cobalt ions are present in solutions containing antidote. In the absence of cyanide to bind the cobalt, serious cobalt toxicity can develop [1]. We used dicobalt edentate before obtaining the result of blood cyanide level measurement. Because the clinical features of the case were helpful to us to diagnose cyanide poisoning.

In conclusion; pediatricians might have difficulty recognizing cyanide poisoning, confirming its presence, and treating it. Cyanide poisoning should be considered in patients who presented with metabolic acidosis characterized by high levels of lactate level and venous pO_2 .

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