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Acute cyanide poisoning due to apricot kernel ingestion

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ABSTRACT

Cyanide is a toxin and one of the most rapidly acting fatal poisons that human being is aware. If it is not treated promptly, encountering to cyanide poison will lead to die in minutes. Cyanide avoids cellular oxygen usage by inactivating mitochondrial cytochrome oxidase thus inhibits cellular respiration. In this case, we represent a case report describing uncommon cyanide intoxication owing to consumption of a few portion of apricot kernels and its rapid treatment with dicobalt edetate after suspection of cyanide poisoning.

1. Introduction

Cyanide poisoning is often rapidly lethal if it is not treated aggressively. Cyanogens can be found in many plants including peaches, almonds and apricots. Multiple cases of paediatric poisoning as a result of consumption of apricot kernel were reported. Apricot kernel has amygdalin that is hydrolyzed to hydrogen cyanide by the catalyzing effect of β-glucosidase emulsin. Chewing apricot liberates emulsin and augments the toxicity of cyanide[1].

2. Case report

A 60-year-old woman was brought to the emergency service with excessive vomiting, headache and diminished level of consciousness. Her blood pressure was 120/60 mm/Hg. Pulses were 119. Respiratory rate was 24. Tympanic temperature was 35.2 ˚C. Blood-glucose level was 105 mg/dL. Oxygen saturation was 95. She was anxious. Her cardiac, pulmonary, abdominal and neurologic examinations were normal. She had excessive vomiting with the headache complaints. An emergent cranial CT was taken. There was no sign of cranial bleeding in CT. Intravenous hydration and antiemetics were given to the patient. The patient said she ate 10–15 apricot kernels for 3 h before she came to the hospital. A blood gas was studied, and slight metabolic acidosis was seen (pH 7.33, PO_2 80 mmHg, pCO_2 34 mmHg and HCO_3 was 20.7). She was mildly hypothermic. No significant electrocardiograph changes were noted. Acute cyanide poisoning due to apricot kernel was considered. The antidote, Kelocyanur (dicobalt edate) of 300 mg IV bolus was given. Patient had a prompt response to antidotal therapy. After the following 24 h, patient was discharged. Patient's cyanide level was 1639 μg/L and > 50 μg/L was considered high. After the therapy, patient's cyanide level was < 50 μg/L considered normal. This is the first case which cyanide toxicity level is observed due to apricot kernel ingestion.

3. Discussion

There are a number of common sources of cyanide. It can be found in the end products of fire, cigarette smoke, artificial nail remover, metal, wood, plastic refineries, laetrile plants, cassava,

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lima beans, grass (*Sorghum*), and the pits of peaches and apricots. Cyanide toxicity has also been reported as an iatrogenic complication from medications, such as sodium nitroprusside[2].

The patient of our case was poisoning as a result of the intake of apricot kernel. Amygdalin is the cyanogen compound found in apricot kernels. Apricot kernels are represented as little cyanide pellets as a result that they additionally contain a β-glucosidase emulsin that works as a catalyzer of amygdalin hydrolysis. Apricot kernels might not liberate a lot of cyanide once swallowed completely, but rather granulating or biting can increase toxicity by discharging emulsin from lysosomes[3].

In our case we suspected very early in diagnosing cyanide poisoning before most of lethal conditions came up.

Cyanide avoids cellular oxygen usage by inhibiting mitochondrial cytochrome oxidase and therefore, causes a shift of cells from aerobic to anaerobiotic cellular respiration. Anaerobiotic respiration produces lactic acid. Therefore, clinical appearances of cyanide poisoning are mostly vague and chiefly indicate oxygen deficiency of neurologic and cardiovascular system. After forceful exposure, quick loss of life can follow. After less serious contact, early appearance comprises malaise, weakness, confusion, dizziness, headache and shortness of breath. Later findings include nausea and vomiting, hypotension, seizures, coma, apnea, cardiac arrhythmias and death due to cardiorespiratory arrest. Supplementary findings occasionally comprise cherry-red discoloration of the skin developing from the lack of ability of cells to get oxygen from the blood. Venous oxygen levels increase and cyanosis is typically not involved in naturally breathing patients[4].

Minor amounts of cyanide that are not metabolized can be sent out through breath, urine and sweat. It has a bitter almond-like aroma which is occasionally noticed on the breath or in gastric contents of patients. Nevertheless, the capability to smell this flavor is genetically established, and up to 50% of the population lacks the gene[5].

There are antidotes in cyanide poisoning that vary from regional availability. The cyanide antidote kit contains three products, amyl nitrite, sodium nitrite and sodium thiosulfate. The nitrates induce methemoglobinemia and thus allow cyanide to dissociate from cytochrome oxidase and preferentially bind to methemoglobin. This process sequesters cyanide in the serum and allows cellular metabolism to resume. The sodium thiosulfate component enhances the innate pathway of cyanide metabolism. The amyl nitrite component of the cyanide antidote kit is contained in pearls that are crushed to produce a vapor that is inhaled. Methemoglobinemia and hypotension are adverse effects which can be seen[6].

Hydroxocobalamin is an successful antidote. It conjoins with cyanide to develop an even less poisonous outcome, cyanocobalamin. Cyanocobalamin is easily excreted in urine. It has light toxicity in itself and does not establish methemoglobinemia or hypotension as sodium nitrite. Hydroxocobalamin has a blood pressure raising effect and improves hemodynamic status[7].

Combination of hydroxocobalamin and sodium thiosulphate demonstrated complimentary outcomes in several cases of cyanide poisoning. Urticaria and impermanent reddish discoloration of urine are the merely reactions reported. Dicobalt-ethylene diamine tetraacetic acid chelates cyanide to make the less poisonous cobalticyanide. It's an extremely effective agent however its toxicity is great once it's not chelating cyanide. Severe high blood pressure or hypotension, cardiac insufficiency and severe hypersensitivity reactions could occur. As a result, it's not used in cases of diagnostic confusion and in moderate toxication[8]. 4-Dimethylaminophenol is practiced in several European countries as an antidote. It's a methemoglobinemia causing antidote and establishes methemoglobin earlier than the nitrites. Attention should be taken in its practice as a result of 4-dimethylaminophenol could cause excessive methemoglobinemia and hemolysis[7].

4. Conclusion

In our case, we used dicobalt edetate for antidotal therapy. We have achieved our treatment by dicobalt edetate. The up-to-date therapy should be hydroxycobalamin because of its little adverse effects. We did not experience any side effect with dicobalt edetate. If your institution has hydroxycobalamin, you should firstly use it. If not, you can use the other antidotal therapies. In literature, there is also a recovery with non-specific supportive therapy.

Conflict of interest statement

The authors report no conflict of interest.

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