

## Acute cyanide poisoning after eating apricot pits: a case report

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### Summary

The stones and seeds of some plants such as apples, apricots, and peaches contain significant amounts of cyanide glycosides. Apricot pits are more toxic as they contain higher amounts of cyanogens and release hydrogen cyanide more easily. A previously healthy 27-month old male patient was admitted to our emergency department as intubated. His history revealed that he was intubated in the hospital where he was taken to with the complaint of fainting after having eaten numerous apricot pits with other family members. His general status was poor and he was unconscious. Both of his pupils were reactive. His deep tendon reflexes were increased and his plantar reflex was extensor bilaterally. The case was diagnosed as cyanide intoxication and the patient was admitted to the intensive care unit. Oxygen was continuously given under observation. After administration of hydroxocobalamin and sodium bicarbonate and correction of sodium deficit the patient regained consciousness and his general health improved. On the second day of admittance, he was discharged with a stable condition. This case was presented to emphasize that parents should not feed small children with apricot pits. (*Turk Arch Ped* 2012; 47: 140-1)

**Key words:** Apricot pits, child, cyanide

### Introduction

Cyanide causes cellular hypoxia as a result of utilization of oxygen in the cytochrome oxidase system (1). The stones and seeds of some plants such as apples, apricots, and peaches contain significant amounts of cyanide glycosides. Apricot pits are more toxic as they contain higher amounts of cyanogens and release hydrogen cyanide more easily (2). When apricot pits are ingested as a whole, release of cyanide is not high, but when they are eaten by chewing the toxicity increases (3). In this article, a 2 years and 3 months-old male patient whose breathing stopped after eating apricot pits, who became unconscious and recovered with hydroxycobalamin and symptomatic treatment was presented.

### Case

A previously healthy 2 years and 3 months-old male patient was brought to our Emergency Unit as intubated. It was learned from his history that he ate plenty of apricot pits with the other family members, vomited for three times approximately one hour later, developed somnolence

afterwards, fainted and was intubated in the hospital where he was brought to because of respiratory insufficiency. Personal and familial history of the patient who was presented as intubated was normal. The patient has two healthy sisters.

On physical examination, his body weight was found to be 11 kg (25-50<sup>th</sup> percentile), his height was found to be 88 cm (10-25 percentile), his arterial blood pressure was found to be 80/50 mmHg, his respiratory rate was found to be 32/min, his apical heart beat was found to be 128/min and his body temperature (axillary) was found to be 35°C. His general status was poor, he was unconscious and he was intubated. The patient was not extubated, since his spontaneous breathing was insufficient. Ligh reflex was positive bilaterally. Deep tendon reflexes were increased. Plantar response was extensor bilaterally. Examination of the other systems was normal.

Laboratory tests revealed that complete blood count, hepatic and renal functions were normal. Sodium was found to be 126 mmol/L and the other serum electrolytes were found to be normal. Prothrombin time was found to be 15.7 s and active partial thromboplastin time was found to be 36 s. Blood gases were as follows: pH: 7.01, pCO<sub>2</sub>: 24 mmHg, pO<sub>2</sub>:204 mmHg, HCO<sub>3</sub>:5.9 mEq/L. C-reactive protein was found to be 3

g/L (Normal: 0-5 g/L). Complete urinalysis was found to be normal. Urinary culture was found to be negative.

The patient was hospitalized in the Intensive Care Unit with a diagnosis of cyanide poisoning. Gastric lavage was not performed, since he was unconscious. 70 mg/kg hydroxycobalamin was given intravenously in one hour. 2000 cc/m<sup>2</sup> 1/3 serum saline (3.33% dextrose monohydrate + 0.3% sodium chloride) was started and active charcoal at a dose of 1 g/kg/dose was given as six doses a day. The patient's spontaneous respiration became sufficient and he was extubated. He was monitored and given continuous oxygen. After administering NaHCO<sub>3</sub> blood gases were as follows: pH: 7.46, pCO<sub>2</sub>: 30 mmHg, pO<sub>2</sub>: 57 mmHg, HCO<sub>3</sub>: 21 mEq/L. Control sodium value was found to be 137 mmol/L in the patient whose serum sodium value was found to be 126 mmol/L and whose sodium deficit was corrected. Following administration of hydroxycobalamin and NaHCO<sub>3</sub> the patient improved gradually. Firstly, his consciousness was recovered. Afterwards he started to talk. He was discharged on the second day of hospitalization with cure. He is still being followed up in our outpatient clinic.

## Discussion

Cyanide inhibits cytochrome-a3. As a result of this mitochondrial oxidation metabolism is disrupted and cellular anoxia and lactic acidosis develop (4). Clinical findings start immediately after ingestion. It may lead to headache, agitation, confusion, loss of consciousness, convulsion and cardiac dysrhythmia (1). After ingestion of cyanide wheezing, deep and rapid breathing, dyspnea and acute respiratory distress may be observed (2). Excessive exposure to cyanide may lead to epileptic seizures, apnea and cardiac arrest in a few minutes (4). It may lead to death by inhibiting the respiratory regulation center (2). Metabolic acidosis with high anion gap is typical (4). However, metabolic acidosis is observed in 67% of the patients who have been acutely poisoned by oral ingestion (5). Cyanide levels can be measured in the blood, but the clinical findings are not compatible with serum cyanide level (1). The diagnosis of cyanide poisoning is made by clinical suspicion (4). The cyanide content of the apricot pit ranges between 0.122 and 4.09 mg/g with a mean value of 2.92 mg/g (6). The mean fatal dose of cyanide is 1.52 mg/kg for humans when ingested orally (7).

In our case, the level of cyanide could not be measured because of technical reasons. Our patient had eaten plenty of apricot pits before his complaints began. He fainted, developed respiratory depression and needed to be intubated following ingestion of apricot pits. He had metabolic acidosis. Many members of the family had eaten apricot pits, but only our patient was affected. The reason for this may be explained by the fact that he had a lower body weight compared to the other family members.

Especially bitter apricot pits contain more cyanide. Apricot pits still have a commercial value in many regions. Since apricot pit has been considered to be a cause of poisoning in recent years, the number of reported cases throughout the country has increased (8-11). In addition, a patient from our country was reported to have died as a result of eating bitter apricot pit (11).

The main treatment consists of using high concentration oxygen and cyanide antidote kit. Cyanide antidote kit contains amyl nitrite and sodium nitrite. These form methemoglobin by interacting with cyanide in cyanomethemoglobin. Cyanide antidote kit additionally contains sodium thiosulfate which converts cyanomethemoglobin to the less toxic thiocyanate (1). Hydroxycobalamin is another antidote. It reacts with cyanide and forms cyanocobalamin (1). Two preparations containing hydroxycobalamin are available in our country.

There are cases who were reported to be improved by giving only supportive treatment (9). Active charcoal is effective in cyanide poisoning (1). We also administered hydroxycobalamin and supportive treatment in our case. Supportive treatment consisted of intravenous fluid and active charcoal.

This case was presented to emphasize that no one should eat apricot pits and parents should not feed small children with apricot pits, since apricot pits may lead to poisoning by the amount of cyanide they contain.

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