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An Extremely Rare Cause of Acute Symptomatic Seizure in Toddlers: Apricot Seed Ingestion

Çocuklarda Akut Semptomatik Nöbetin Oldukça Nadir Bir Nedeni: Kayısı Çekirdeği Yutulması

İpek DOKUREL ÇETİN [1*], Cansen ÇENELİ [2]

[1] Balıkesir University Medical Faculty Hospital, Department of Pediatrics, Division of Pediatric Neurology, Balıkesir, Türkiye

[2] Balıkesir University Medical Faculty Hospital, Department of Pediatrics, Balıkesir, Türkiye

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Abstract

One of the most frequent causes of seizures in children is an acute symptomatic seizure. To forecast risk of recurrence of seizure, it's crucial to identify the trigger element at the root of the problem. Cyanide inhibits cell oxygen utilization and cellular respiration through inactivation of mitochondrial cytochrome oxidase. Cyanide is one of the most lethal toxins ever discovered for the human body. Early treatment enables the avoidance of harmful outcomes, including death. This case report represents case of a 2-year-old girl presented with an acute symptomatic seizure after eating a few apricot kernels and her quick recovery with antidote after cyanide poisoning was suspected. The patient was discharged without experiencing any long-term effects of cyanide poisoning.

Keywords: Cyanide, Children, Acute Symptomatic Seizure

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Öz

Çocuklukta nöbetlerin en sık nedenlerinden biri akut semptomatik nöbettir. Nöbetin tekrarlama riskini tahmin etmek için sorunun kökündeki tetikleyici unsuru belirlemek çok önemlidir. Siyanür, mitokondriyal sitokrom oksidazın inaktivasyonu yoluyla hücre oksijen kullanımını ve hücresel solunumu inhibe eder. Siyanür, insan vücudu için şimdiye kadar keşfedilen en öldürücü toksinlerden biridir. Erken tedavi, ölüm dahil zararlı sonuçların önlenmesini sağlar. Bu vaka sunumu, birkaç kayısı çekirdeği yedikten sonra akut semptomatik nöbet ile başvuran ve siyanür zehirlenmesinden şüphelenildikten sonra panzehirle hızla iyileşen 2 yaşındaki bir kız çocuğunu sunmaktadır. Hasta siyanür zehirlenmesinin uzun süreli etkisi görülmeden taburcu edilmiştir.

Anahtar Kelimeler: Siyanür, Çocuk, Akut Semptomatik Nöbet

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[*] **Sorumlu Yazar (Corresponding Author):** İpek Dokurel Çetin, **e-mail**: dripekdokurel@gmail.com

Introduction

The immature brain of children is more prone to epileptogenesis and propagation of seizures. Acute symptomatic seizures were provoked by any acute condition such as a toxic or metabolic disturbance, fever, head trauma, or acute stroke, are differed from epileptic seizures. The American Academy of Neurology has advised having laboratory screening tests based on certain clinical conditions in evaluating a child's first seizure (1). However, due to its rarity, cyanide poisoning in pediatric seizures is challenging for clinicians in terms of diagnosing, verifying, and treating it early.

Cyanide is particularly hazardous to the central nervous, respiratory, and cardiovascular systems. Cyanide poisoning is uncommon in children, but it is potentially fatal. In general, inhaling smoke from a fire, consuming pesticides, polishing metal, and consuming cyanogenic foods are all potential sources of cyanide exposure (2-4). Several plants, including apricot, peach, plum, and cherry contain cyanide glycosides (5,6). Amygdalin was the first cyanogenic glycoside isolated from plants. Amygdalin was originally found in bitter almonds [Prunus dulcis Mill. var. amara (DC.) H. Moore] (7) Amygdalin is found in many Rosaceae seeds and is mostly derived from bitter apricots, semen prunus, almonds, and peach kernel (8).

The amygdalin is hydrolyzed and converted to hydrogen cyanide in the stomach (9). Cyanide binds to ferric iron in the cytochrome oxidase a-a3 complex and inhibits aerobic metabolism. The rate of lactate is increased, due to Krebs cycle disruption, resulting in metabolic acidosis. As a result, cyanide significantly reduces the level of ATP and increases the level of lactate in the brain (10).Patients with delayed diagnosis and treatment have higher mortality and morbidity rates (11). Antidotes such as sodium thiosulfate, sodium nitrite, amyl nitrite, and hydroxocobalamin can be utilized. Though, hydroxocobalamin stands out in its efficacy and safety profile (12,13).

In this article, we aimed to present a rare case of acute symptomatic seizure due to cyanide intoxication with apricot kernel ingestion, is successfully treated with hydroxoycobalamin.

Case Report

A previously healthy 2-year-old girl was admitted to the pediatric emergency department (PED) for loss of consciousness. In the PED, she had generalized tonic clonic contraction of the extremities, staring and pallor lasting for 5 minutes. After the seizure, the initial evaluation revealed post-ictal confusion, tachycardia (174 beats/min) (Figure 1), prolonged capillary refill time (>3s) tachypnea(36/min), and hypotension(66/47mmHg) with low GCS (9/15). Intravenous fluid and dopamine(5mcg/kg/min) infusion were initiated. Her complete blood count, biochemical and coagulation analyzes were within normal limits. Tympanic temperature was 36.7°C, acute phase reactants were negative, and empirical intravenous acyclovir (40mg/kg/day) and ceftriaxone (100mg/kg/day) were administered with high suspicion of encephalitis.

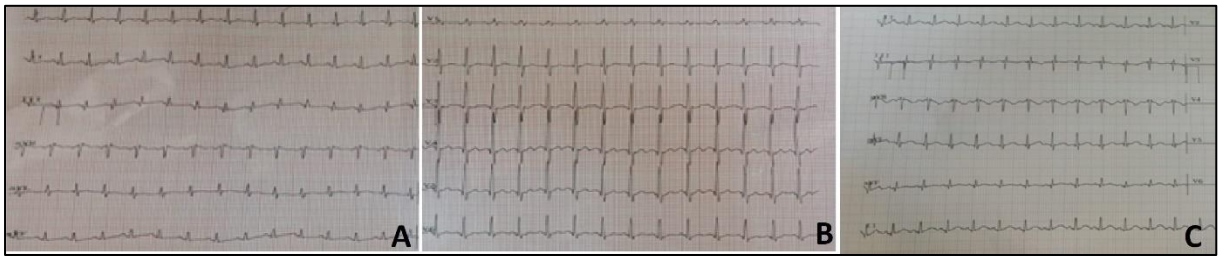

Figure 1. The electrocardiogram of 2-year-old girl with sinus tachycardia performed after acute symptomatic seizure due to cyanide ingestion. A, B are before the treatment with hydroxycobalamin. C is after the treatment with hydroxycobalamin.

As metabolic disorders such as hypoglycemia may produce loss of consciousness accompanied by seizures, the blood glucose (124mg/dL) and ammonia (54µmol/L) levels were normal. High anion-gap metabolic acidosis and high lactate levels were detected in blood gas analysis and sodium bicarbonate replacement therapy was initiated (Table 1). The patient's mother said she ate 10–15 apricot kernels for 45 min before she was collapsed. In the PED, gastric lavage was performed with activated charcoal. Hydroxycobalamin (70 mg/kg/dose) was given by intravenous infusion over one hour as antidote. She was consulted by pediatric cardiologist for sinus tachycardia and mildly elonged QTc(440ms) (Figure 1). Informed consent form is taken from the family.

Discussion

An acute symptomatic seizure is defined as a clinical seizure occurring at the time of a systemic insult or in close temporal association with a documented brain insult by the International League Against Epilepsy. Approximately 25,000 to 40,000 children per year in the United States have a first unprovoked seizure. However, most children do not go on to develop epilepsy later in life. In our case, cyanide poisoning due to apricot kernel consumption provoked an acute symptomatic seizure, and this was successfully managed with hydroxocobalamin.

The main action of cyanide is for the production of essential cellular energy sources of oxygen in the form of ATP. It inhibits oxidative phosphorylation, a process in which it is used. Ingestion of cyanide by inhalation symptoms begin within minutes, and in a few minutes with oral ingestion. Early symptoms are headache, dizziness, confusion, and mydriasis. These symptoms result from brain tissue hypoxia and coma may develop with convulsions afterwards. Early respiratory and cardiovascular findings are tachypnea and tachycardia, followed by apnea, hypotension, and cardiac arrhythmias (14).

Since this clinical picture, which can be fatal, is rare, it can be difficult to recognize by clinicians. Therefore, to consider the oral poisoning by cyanide is important for early diagnosis and treatment. Ünal et al. reported that the symptoms of a 3.5-year-old boy started 45 minutes after he ate the apricot kernels. This period after poisoning is a long period of time that leads to the development of serious symptoms if left untreated. After the patient came to the hospital, 2 cardiac arrests developed and he was resuscitated. After 24 hours, a cyanide antidote kit was found and treatment with an antidote was started. In the brain MRI of the patient, hemorrhagic damage was detected in the bilateral lentiform nuclei. After 12 days of hospitalization, the patient was discharged with neurological sequelae (tetraparesis and rigidity), and tetraparesis continues after 8 months of follow-up (6). In the article of Peddy et al., a 17-year-old male patient with potassium cyanide toxicity, who presented with seizures, apnea and cardiovascular collapse, was reported. In this case, there was a similar clinical picture due to potassium cyanide intake, cardiac arrhythmias, cerebral infarction were observed and resulted in brain death (2). In these two cases, a cyanide antidote kit containing sodium nitrite and sodium thiosulfate was applied as antidote treatment. In the study of Bıcılıoğlu et al., 4 cases aged 2, 2.5, 3 and 3.5 years due to apricot kernel ingestion were presented. Three of these cases were in severe poisoning state. In one, the symptoms developed 30 minutes after the apricot kernel ingestion, and in the other two, 2 hours later. These three cases of severe poisoning were administered hydroxycobalamin at a dose of 70mg/kg after 6 hours,8 hours and 12 hours. All three cases were discharged without any sequelae (15). Our patient was also a case of severe cyanide poisoning and recovered without sequelae for early diagnosis and early hydroxoycobalamin treatment.

Most of the studies on cyanide poisoning in the literature consist of animal experiments. These experiments show that the amount of cyanide administered to observe signs of poisoning in rats or mice represents a relatively large volume compared to that given for the same effect to occur in a human. Rice et al. observed rats for overt signs of toxicity immediately following poisoning across a broad range of doses (4– 128 mg/kg) (16). Sodium nitroprusside (SNP) which is an inorganic cyanogen, is a potent vasodilator commonly used as an anti-hypertensive agent in postoperative cardiac surgical patients. Despite, cyanide toxicity can occur with SNP therapy. Any concentration of sodium nitroprusside exceeding 3.5 mg/kg/min,

based on the Federal Drug Administration, may be fatal to humans. There are certain risk factors that have been associated with the development of harmful levels of cyanide and negative effects in children. Nevertheless, there is a lack of proof regarding the indications and symptoms of toxicity associated with SNP infusions in pediatric patients. (17). Based on the current literature, it appears that there is limited evidence supporting a connection between elevated cyanide levels and symptoms of cyanide poisoning. Although randomized controlled studies with multiple cases of cyanide poisoning due to ingestion of apricot kernels are not available in the literature due to the rarity of their observation, considering the small number of case reports, early diagnosis and early hydroxycobalamin treatment seem to be effective for good results.

In conclusion, clinicians should consider cyanide poisoning in patients who have presented an acute symptomatic seizure, a change in consciousness. Apricot seeds consumption should be interrogated. It should behold that hydroxocobalamin antidote medication administered quickly can save lives.

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