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**Case Report**

# Acute Cyanide Intoxication Related to Apricot Seed: The Findings of Cranial Magnetic Resonance Imaging

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## Abstract

Cyanide is a strong poison which acts rapidly and which may lead to death in minutes even at low doses. The substance of amigdaline which is found in seeds of fruit including plum, cherry, apricot, peach and bitter almond can be converted into hydrogen cyanide in the stomach. Cyanide intoxications are rarely observed following the ingestion of food products that contain cyanide glycosides. Brain magnetic resonance imaging was performed 9 days after intoxication in a 3.5-year old male patient in whom cyanide poisoning developed as a result of intake of apricot seed. On brain magnetic resonance imaging, hemorrhagic damage was found in bilateral lentiform nuclei and damage was found in the caudate nuclei. Our aim was to present magnetic resonance imaging findings related to cyanide poisoning which occurred after the ingestion of apricot seed.

**Keywords:** Apricot, cyanide, magnetic resonance imaging

## Kayısı Çekirdeğine Bağlı Akut Siyanür İntoksikasyonu: Beyin Manyetik Rezonans Görüntüleme Bulguları

## Özet

Siyanür düşük dozlarda bile hızlıca dakikalar içerisinde ölüme yol açabilen güçlü bir zehirdir. Erik, kiraz, kayısı, şeftali, acı badem gibi meyvelerin çekirdeklerinde bulunan amigdalin maddesi midede hidrojen siyanüre dönüşebilmektedir. Siyanür zehirlenmeleri nadiren siyanür glikozitleri içeren gıda ürünlerinin alımını takiben gözlenir. Kayısı çekirdeği yenmesine bağlı siyanür intoksikasyonu gelişen 3,5yaşındaki erkek hastaya intoksikasyonun 9. gününde beyin manyetik rezonans görüntüleme yapıldı. Beyin Manyetik rezonans görüntülemede, bilateral lentiform nükleusta hemorajik hasar ve kaudat nükleuslarda hasar gözlemlendi. Amacımız kayısı çekirdeği yenmesinden sonra meydana gelen siyanür zehirlenmesi olgusunun beyin manyetik rezonans görüntüleme bulgularını sunmaktır.

**Anahtar Kelimeler:** Kayısı, Siyanür, Manyetik Rezonans Görüntüleme

## INTRODUCTION

Cyanide is one of the most powerful and deadly poisons. The people can be exposed to cyanide from various sources. Cyanide poisoning can occur by inhalation, ingestion, or skin contact of the substance. Existing in gaseous, solid, and liquid forms, cyanide is used in many industries, found in certain household substances, and produced by the combustion of common materials such as fabrics containing nylon,

silk, or wool and many plastics such as melamine, polyurethane, and polyacrylonitrile[4]. Amygdalin found in the seeds of various fruits such as plum, cherry, apricot, peach, and bitter almond is converted to hydrogen cyanide in the stomach. Cyanide poisoning can be observed after ingestion of food containing cyanide glycosides[3,9].

In the developing countries, cyanide poisoning in children primarily occurs as a result of ingesting amygdalin-containing food. The ingestion of apricot seeds is the most common cause of cyanide poisoning in children in Turkey[1]. In the current case report, we present magnetic resonance imaging (MRI) findings after the ingestion of apricot seed.

## CASE PRESENTATION

A 3.5-year-old boy was admitted to the hospital due to fainting, muscle contraction, and difficulty in breathing. The children lived in the village in Turkey. According to the medical history obtained from the parents, the patient have had abdominal pain with a sudden onset followed by fainting that lasted 2-3 minutes and then regained consciousness after which he cried and had difficulty in breathing with the eyes deviated and had muscle contractions and shaking. The patient then lost consciousness for the second time and did not awake until arrival to the hospital. According to the family, the patient did not have a known medical problem and he ate apricot seeds 45 minute before the complaints have started. The parents had no information on how many apricot seeds the child might have eaten.

On physical examination, the patient's general condition was poor, and he was unconscious. The light reflex was bilaterally weak. There was an increase in nasopharyngeal and oropharyngeal secretions. The respiration was superficial and secretory rales were heard on auscultation. The pulse rate was 60 bpm and blood pressure could not be obtained.

The pathological laboratory findings were as follows: lactate dehydrogenase 1725 U/L, alanine aminotransferase: 91 U/L, aspartate aminotransferase: 250 U/L, creatine kinase: 2312 U/L, creatine kinase-MB: 86 U/L, amylase: 374 U/L (all higher than normal ranges), and blood glucose was 44 mg/dl, (lower than normal). Blood anion gap 19 higher than normal. The patients had lactic acidosis (arterial blood gases; ph:7.06, lactat 4.41 mmol/L).

The patient sustained cardiac arrest twice in the same day. In the first resuscitation attempt, the patient responded at 7 minute, and in the second attempt the patient responded immediately. The patient was thought to have acute cyanide poisoning and activated charcoal was administered. A blood sample obtained from the patient was sent to an external center for the analysis of cyanide levels. After the receipt of cyanide antidote kit (after 24 hours), the treatment was continued with the cyanide antidote kit.

The patient underwent MRI of the brain in the 9$^{th}$ day of the treatment period. MRI showed heterogeneous hypointense area with peripheral hyperintense signal changes in bilateral lentiform nuclei on T2-weighted, GRE and FLAIR sequences, and hypointense area with peripheral hyperintense signal changes on T1-weighted sequences suggestive of hemorrhagic damage. Both caudate nuclei showed increased signal intensity on T2-weighted GRE and FLAIR sequences and decreased signal intensity on T1-weighted sequences suggestive of damage. The diffusion-weighted images showed bilateral and symmetric peripheral restriction in lentiform nuclei and heterogeneous appearance at the center and diffusion restriction in both caudate nuclei (Figure 1A-D and Figure 2A, B). The blood cyanide level measured in an external center was reported to be >2.0 mg/l. This value confirmed the diagnosis of cyanide poisoning together with clinical

appearance, history, and other emergency laboratory tests.

After 12 days of hospitalization period, the patient was discharged with slightly increased deep tendon reflexes, slightly impaired consciousness, rigidity, and tetraparesis. During the patient's follow-up at 8 months, the patient had tetraparesis, cogwheel rigidity in the upper extremity and a decrease in intellectual activity.

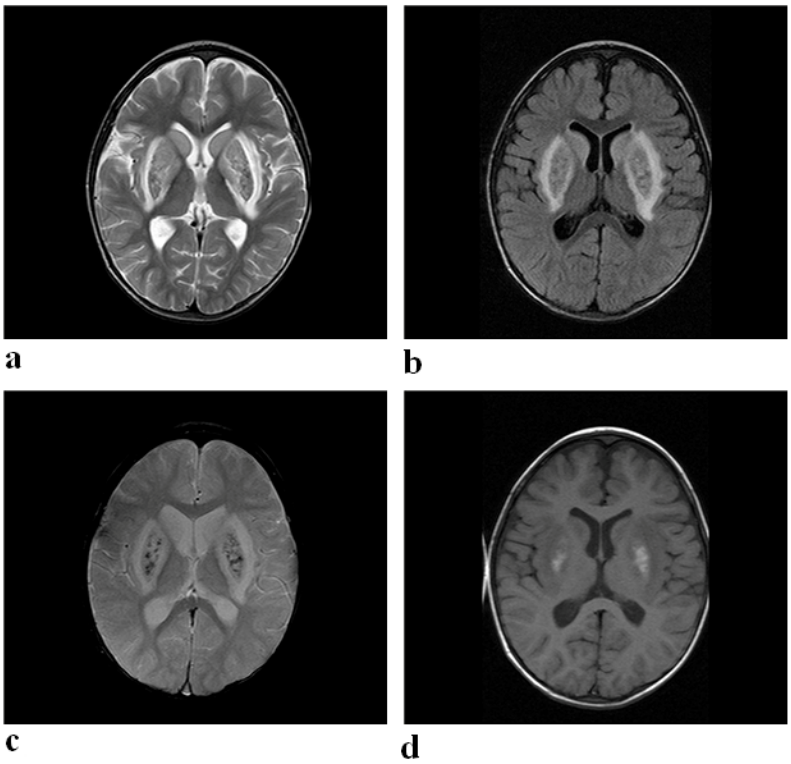


***Figure 1 A-D:*** *An area of hemorrhagic damage is observed in bilateral lentiform nuclei with central heterogeneous hypointense and peripheral hyperintense appearance on T2-weighted (Fig. 1A), Flair (Fig. 1B), and GRE (Fig 1C) sequences, and central hyperintense and peripheral hypointense appearance on T1-weighted (Fig. 1D) axial images. Both caudate nuclei showed increased signal intensity on T2-weighted GRE and FLAIR sequences and decreased signal intensity on T1-weighted sequences suggestive of damage (Figs. 1A-D).*

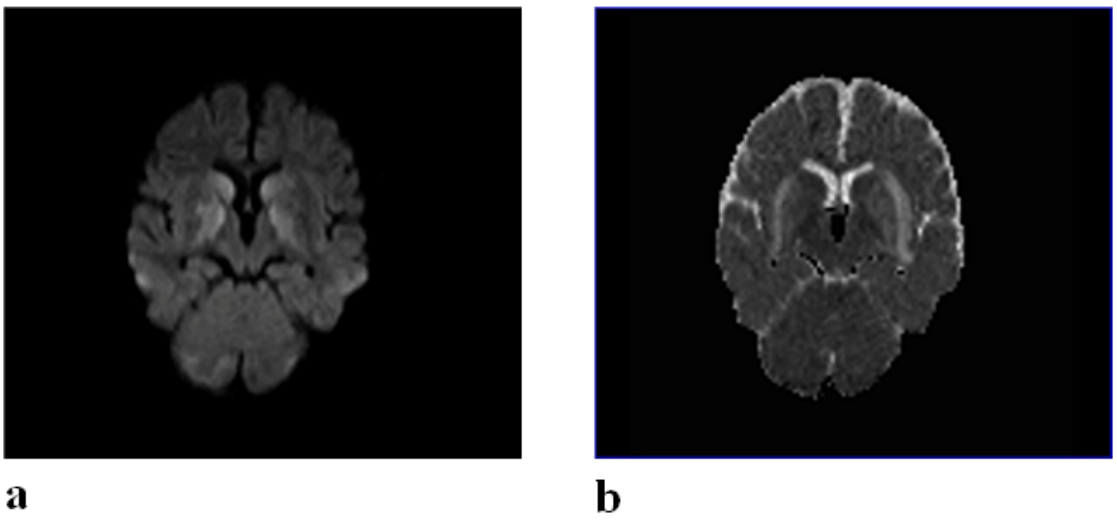


***Figure 2 A, B:*** *Diffusion-weighted (b 1000) images (Fig. 2A) and ADC map (Fig. 2B) show hemorrhagic damage in both lentiform nuclei and damage site in both caudate nuclei.*

## DISCUSSION

Acute cyanide poisoning can occur in children with the ingestion of cyanide-containing herbs or other food contaminated by cyanide-containing products. The seeds of peach and apricot, large- and flat-seeded beans (Lima bean), and cassava plant are known to contain cyanide. Intentional or inadvertent ingestion of these plants can cause to cyanide poisoning. Due to the fact that cyanide-containing food products are part of the diet in tropical countries, cyanide poisoning has been mostly reported from these countries[4,1]. The apricot seeds contain amygdalin, which is hydrolyzed to cyanide and benzaldehyde by glycosidase and emulsion enzymes after ingestion[9]. The apricot seeds would not cause excessive cyanide release if ingested as a whole; however, if the seeds are chewed and fragmented, cyanide is released in greater amounts with the action of a lysosomal enzyme emulsion. The lethal dose of cyanide for humans ranges from 0.56 to 3 mg/kg cyanide content of the apricot seeds changes from 4.09 to 112 mg/g, which is accepted to be 2.92 mg/g in the latest publications[3,7].

The current case was 3.5 years old and weighted 13.5 kilograms, and the parents did not know how many seeds the patient might have eaten. The symptoms have become manifest 45 minutes after the ingestion of apricot seeds. The possibility that the patient might have exposed to another substance that could cause this clinical picture was ruled out by the finding of >2.0 mg/dl of blood cyanide level.

The essentials of cyanide treatment include oxygen supplement, cardiopulmonary support, and administration of specific antidote. The treatment with specific antidote must be administered as early as possible[1]. The fact that cyanide antidote was administered to the patient approximately 24 hours after the initial presentation may have caused residual symptoms.

Cyanide exerts its effect at the cellular level by arresting oxygen utilization. Cyanide inhibits the reduction of molecular oxygen to water by cytochrome oxidase a3 enzyme which is the last step of oxidative phosphorylation. Cyanide exhibits a particular affinity for metal-containing enzymes, and it binds to ferric (Fe+3) iron which is found in the hem part of the cytochrome enzyme. As a result of the disturbed oxidative phosphorylation, cellular hypoxia occurs despite normal oxygen supply to the tissues[2].

The differential diagnosis of acute conditions bilaterally involving basal ganglia in the childhood include hypoxia, hypoglycemia, carbon monoxide poisoning, hemolytic uremic syndrome, osmotic myelinosis, and encephalitis. The chronic conditions in the childhood include Leigh's disease, glutaric academia type 2, MELAS, wilson, maple syrup urine, juvenile huntington's, canavan disease, neurofibromatosis type 1, and metachromatic leukodystrophy[8].

Cyanide causes a damage exhibiting high demand for oxygen. The basal ganglia exhibit high demand for oxygen, and cyanide poisoning leads to hemorrhagic necrosis in these areas. The cerebellar and sensorimotor cortex showing high demand for oxygen can also be affected. In cases where cerebellar and sensorimotor cortex is affected, it may not be clear if the poisoning was caused by cyanide itself or to hypoxia as the component of poisoning. The preservation of hippocampus that exhibits very high demand for oxygen may be helpful to differentiate cyanide poisoning for hypoxic damage as being the component of poisoning. In cyanide poisoning occurring as a result of suicide attempt, hemorrhagic necrosis can be observed in corpus striatum and pseudolaminar necrosis can occur in

cerebral cortex due to cyanide intoxication[7].

In the present case, cerebellar cortex, sensorimotor cortex, and hippocampus were intact, and therefore hypoxia associated with poisoning and hypoxic damage due to cardiac arrest were ruled out. Furthermore, although the lesion in caudate nuclei were not clearly visible, the observation of lesions in basal ganglia on computerized tomography before the patient has sustained cardiac arrest indicates cyanide poisoning as the possible cause.

Our literature search returned one case report of brain involvement on MRI after ingestion of apricot seed[3]. In that case report, a 2.5-year patient underwent cranial MRI at day 5 after the ingestion of apricot seed, and MRI showed increased signal intensity in both lentiform nuclei on T2-weighted and FLAIR sequences, diffusion restriction in diffusion-weighted images, and increased signal intensity in both parietal cortex adjacent to interhemispheric fissure on FLAIR sequences. We found few reports of cyanide poisoning in the literature other than the ingestion of apricot seed suggesting brain involvement on MRI images[7,6,8]. In all these publications, lentiform nuclei were the common injury site but the involvement of substantia nigra, subthalamic nucleus, cerebellar damage, and cerebral pseudolaminar necrosis have also been reported.

In conclusion, food intoxication associated with cyanide poisoning should be kept in mind and food history should be obtained in cases presenting with the involvement of basal ganglia and particularly in cases with acute hemorrhagic necrosis. In addition to conventional MRI sequences, GRE sequences which is superior in showing hemorrhages and DAG for early diagnosis of the condition can be helpful in diagnosis. It must be kept in mind that therapy must be instituted as early as possible in cyanide poisoning and the antidotes are required to be dispatched from limited centers.

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