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Case Report

# Cyanide Intoxication after Ingestion of Wild Cherry (Prunus Avium)

**Yilmaz BK[1], Cevik E[2]*, Cerciz H[1], Yoruk M[1], Acar YA[3], Cakmak F[4] and Ikizceli I[5]**

[1]Department of Emergency Medicine, Sisli Hamidiye Etfal Research and Training Hospital, Turkey

[2]Department of Emergency Medicine, Van Military Hospital, Turkey

[3]Department of Emergency Medicine, Etimesgut Military Hospital, Turkey

[4]Department of Emergency Medicine, Balikligol Government Hospital, Turkey

[5]Department of Emergency Medicine, Istanbul University Cerrahpasa Medical School, Turkey

***Corresponding author:** Erdem Cevik, Department of Emergency Medicine, Van Military Hospital, Van Asker Hastanesi Altintepe, Van, Turkey

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**Abstract**

Intoxications are major public health problems and can be caused from wide range of substances. Plants can be toxic to human beings and animals. Cyanogenic plants are groups of plants cause cyanide intoxication. Wild cherry is one of cyanogenic plants. In this report, we presented a 67- year old man with cyanide intoxication after eating wild cherry. He was treated successfully with hydroxocobalamin administration.

**Keywords:** Cyanides; Poisoning; Wild cherry

## Introduction

Intoxications remain a major public health problem and plant poisonings constitutes 4.7% of all poisonings [1]. However rarely encountered, early diagnosis and proper management of cyanide intoxication is crucial because of probable fatal outcome. We reported a cyanide intoxication case after wild cherry ingestion.

## Case Presentation

A 67-year old man was admitted to emergency department because of altered mental status. He had been complaining apathy, meaningless speech, and difficulty in identifying people for two days before admission. These complaints started after eating wild cherry in the forest. He denied any chronic disease and medication. His vital signs were BP: 152/73 mmHg, pulse rate: 73 bpm (regular), temperature: 36.0°C and $SaO_2$: 94%. Heart and lung auscultation and systemic examination did not show any abnormality. The patient was lethargic, and there was no lateralization finding. Venous blood gas values showed a pH of 7.36; $PCO_2$ of 45.6 mm Hg; $PO_2$ of 41.6 mm Hg, HCO3 of 25.3 mmol/L $SaO_2$ of 40.4%, lactate of 1.1 mmol/L, HHb of 58.1%, and COHb: 1.5%. Other laboratory analyses were all in normal ranges. Brain imaging (including MRI and CT) and lumber puncture were performed for differential diagnosis but any pathology could not be detected to explain the changes in consciousness.

Because of wild cherry ingestion history, cyanide intoxication was considered and the patient was hospitalized in the observation unit of emergency department. National Poison Control Center advised hydroxocobalamin therapy. The patient was 70kg weight and 5grams of hydroxocobalamin (Cyanokit®, Meridian Medical Technologies) administered intravenously in 30 minutes initially. Clinical findings were continued and a second dose (5grams) administered in 90 minutes. Symptoms were resolved quickly after second dose of hydroxocobalamin. After a 3-day follow up, patient discharged from emergency department without any complication (Figure 1).

## Discussion

Cyanide intoxication can be resulted from exposure of chemicals in industrial environment [2-6], environmental exposures [7,8], fire accidents as well as CO, [9-11] and drugs such as sodium nitroprusside [12].

Cyanogenic plants are a group of plant species and synthesize cyanogenic glycosides which can release hydrogen cyanide by enzymatic degradation [13]. Cyanogenic plant consumption can cause acute cyanide intoxication or some chronic diseases [14]. Several cyanide intoxication cases has been reported after plant ingestion such as apricot seeds [15-18], cassava [19-22], plants [23], and bitter almond [24,25]. Intoxication due to plant ingestion can

**Figure 1:** Wild cherry (Prunus avium).




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affect animals besides human beings [8].

Wild cherry caused cyanide intoxication was reported in a pony and a goat [26,27]. We could find just one human being case report in literature [28].

Cyanide causes cytotoxic hypoxia by the inhibition of cytochrome oxidase [29,30]. Most common clinical findings were reported as decreased level of consciousness, vertigo, headache, convulsion, metabolic acidosis, cardiopulmonary failure, Parkinsonism, and optic neuropathy [31-34]. Our patient's major complaint was decreased level of consciousness, also.

However there is no specific laboratory finding of cyanide intoxication, decrease of difference between arterial $PO_2$ and venous $PO_2$ and elevation of lactate levels can be a clue [31,34]. MRI findings in two patients with neurologic sequel after cyanide intoxication were reported as damage of the globus pallidus, putamen, substantia nigra, subthalamic nucleus, and cerebellum [35]. Our patient showed normal pH levels and normal brain imaging.

Treatment of cyanide intoxication needs use of an antidote besides supportive care. However there is no controlled study, hydroxocobalamin and sodium thiosulfate are two antidotes suggested for the treatment of cyanide intoxication [36,37]. Our patient was treated with hydroxocobalamin administration without any side effect or complication.

## Conclusion

Cyanide intoxication is a rare but fatal clinical condition caused by several agents. Emergency physicians must be aware of cyanogenic plants and consider especially evaluating the patients with decreased level of consciousness.

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