

Ingestion of Black Locust Tree Bark: Case Report

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Abstract

Background: Case reports of black locust tree (*Robinia pseudoacacia* L.) bark ingestions are scarce in modern medical literature. The black locust tree is native to the Appalachian Mountains as well as parts of the Midwestern United States. The seeds and bark of the tree are known to be toxic when ingested. Since oral absorption of the toxins is limited; symptoms are primarily gastrointestinal in nature.

Case Report: A twelve-year-old male presented with weakness, nausea, vomiting and diarrhea after ingestion of black locust tree bark. Pertinent vital signs and laboratory results include a temperature of 101.5°F, a heart rate of 133 beats per minute, pH of 7.31, a White Blood Cell (WBC) count of 25.6×10^3 cells/L, a hematocrit of 50.1%, and an alkaline phosphatase of 216 U/L. Intravenous ondansetron and fluids had been administered at a referring hospital. He was transferred to our facility where he was admitted for observation and did not experience any additional episodes of emesis or diarrhea. Due to the early recognition of the etiology of his symptoms, no further workup was required and the patient was discharged home in stable condition after tolerating a liquid diet the next day.

Conclusion: Ingestion of black locust tree bark can result in clinical toxicity with primarily gastrointestinal symptoms. Supportive care with appropriate medical observation is the mainstay of treatment and generally results in positive outcomes. The effectiveness of gastric decontamination by activated charcoal is unknown but a single dose may be considered if the patient presents within one hour of ingestion without contraindications.

Keywords: Black locust; *Robinia pseudoacacia*; Tree bark; Ingestion

Abbreviations: CNS: Central Nervous System; WBC: White Blood Cell

Introduction

The black locust (*Robinia pseudoacacia* L.) tree is native to the Appalachian Mountains as well as parts of the Midwestern United States and is often used for fencing or landscaping [1]. Lectins are a diverse group of proteins often found in plants as well as microorganisms that bind carbohydrates, precipitate glycoproteins, and agglutinate cells [2,3]. Some toxic lectins are found in the seeds, leaves, and bark of the black locust tree (Figure 1) [4-8].

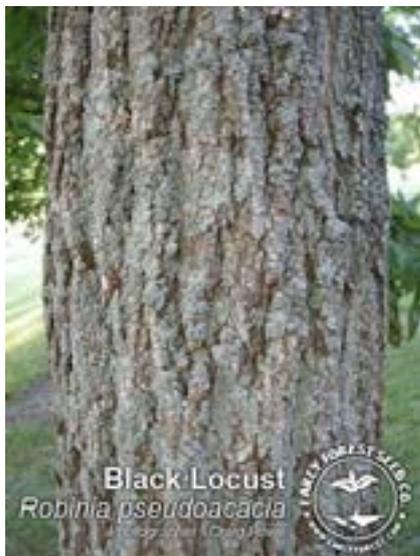


Figure 1: The black locust tree (*Robinia pseudoacacia* L.).

As early as 1890, the toxic constituents of the black locust tree, robin and robinin, were described as large proteins called toxalbumins similar to ricin [4,9,10]. Ricin, which is well known as a potential bioterrorism agent [11], binds to cell surfaces with its A chain and inactivates ribosomes with its B chain. This leads to tissue edema and cell death causing a vascular leak syndrome [7,12,13]. While this mechanism of toxicity has not been confirmed for the substances contained in *R. pseudoacacia*, they are known to bind similar cell surfaces and are comprised of A and B subunits [14].

While there are many cases of poisoning with the castor bean (*Ricinus communis* L.), there are very few incidences of black locust bark ingestion. The most recent report occurred in New York during 2004. The patient developed nausea and vomiting with an elevated alkaline phosphatase and WBC count that was treated with activated charcoal and supportive care for five days before being discharged in stable condition [7]. Additional cases involving several children have been reported from Spain, all of which were treated with gastrointestinal decontamination and recovered quickly [4,5,15,16]. Prior to these few cases, only one other ingestion of black locust bark has been reported since 1887 when 32 orphans ate the bark off of a fence. Although some were stated to be in severe condition, they were all treated with supportive care appropriate for their time and discharged after two days [17].

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Case Presentation

A previously healthy 12-year-old Caucasian male presented to the pediatric emergency department with nausea, vomiting, non-bloody diarrhea and fatigue after being transferred from an outside hospital. The symptoms began shortly after eating lunch with his father who did not become ill. The patient's mother attributed these symptoms to the meal and tried a home remedy of yellow mustard for possible food poisoning. The vomiting was soon accompanied by a watery, profuse diarrhea with severe generalized weakness to the point where he was stated to be unable to rise from a prone position. The patient also reported episodes of "blacking out". Upon further questioning, the boy's older brother revealed that he had seen the patient eat approximately two handfuls of the soft bark from a tree between earlier that day, citing a sweet taste. The tree was soon identified as a black locust tree.

The patient's parents report no family history of gastrointestinal disease, no allergies, no medications, and no recent illness or any sick contacts. The patient was up to date on his immunizations and denied use of tobacco, alcohol, or recreational drugs. Upon presentation, the patient was alert and oriented for his age with the physical examination being largely unremarkable other than a mild sinus tachycardia and abdominal pain. A complete metabolic panel with a complete blood count were drawn and revealed a pH of 7.31, a White Blood Cell (WBC) count of 25.6×10^3 cells/L, a hematocrit of 50.1%, and an alkaline phosphatase of 216 U/L. All other results were within normal limits.

One liter of normal saline and 4 mg of ondansetron had been administered at the outside hospital with the last episode of emesis occurring a few hours prior to his arrival at 10:00 pm. Upon consultation with a toxicologist, the decision was made by the pediatrics team to admit the patient for observation and supportive care. The use of gastric decontamination was deemed unnecessary due to the recent episodes of vomiting and the prolonged time since ingestion. Antibiotics were also avoided as the elevated WBC count was thought to be inflammatory in nature.

Once admitted, telemetry was initiated to monitor for cardiac involvement but revealed no changes. No further medications were given during his stay with the exception of intravenous fluids. The patient did not experience any additional episodes of diarrhea or emesis, nor did he complain of worsening muscle weakness. By the next day, he had regained his appetite and was tolerating a liquid diet. All vital signs were stable at that time with an improved WBC count of 13.3×10^3 /L and an alkaline phosphatase of 138 IU/L. The patient was then discharged home with instructions to return if symptoms worsened.

Discussion

The lethal oral dose of black locust bark is not well defined and mortality data is lacking, however, there are a few reported cases of death in animals following large ingestions [15]. Systemic absorption of the toxins is thought to be poor, however, exposure is increased when the *R. pseudoacacia* bark is masticated [3,14,18]. Of note, hepatic Kupffer cells are equipped with lectin receptors that enable rapid uptake and subsequently impair the patient's ability to detoxify the substance [12,13]. Laboratory detection of the black locust toxins is not yet clinically validated and minimum toxic serum levels are unknown. Laboratory levels may be beneficial for environmental surveillance since both plasma and urine samples have been tested in human and animal studies with ricin. As another example, the less toxic alkaloid ricinine has successfully been used as a surrogate marker for ricin since it can be detected in the urine for up to 48 hours [12,19].

Symptoms generally appear within several hours of *R. pseudoacacia* ingestion. The clinical course progresses over a few days so patients should be admitted for observation [4,7,15]. The typical presentation includes nausea, vomiting, diarrhea (with or without gastrointestinal hemorrhage), abdominal pain, tachycardia, fever, leukocytosis, and elevated alkaline phosphatase [4,7,15,19]. Severe symptoms may develop if left untreated and include hypovolemic shock, QT interval prolongation secondary to electrolyte disturbances, and metabolic acidosis [20]. Disturbances of the central nervous system (CNS) such as mydriasis, headache, dizziness, seizures, and cerebral edema have been observed in black locust bark ingestions and may be caused by other harmful substances contained in the plant [19]. For example, ricinine is thought to be responsible for the CNS effects seen in some *R. communis* ingestions [12].

There is no antidote for *R. pseudoacacia* ingestion, however, supportive care is often enough to manage the toxic state [7,9]. The extent of toxin adsorption by activated charcoal is unknown but a single dose of 0.5–1 g/kg in children or 25–50 g in adults may be considered if the patient presents within one hour of ingestion without contraindications [7,15]. Large proteins, like the toxins from the bark, are generally not dialyzable and cardiopulmonary, renal, and hepatic function should be monitored carefully [18]. Patients who remain asymptomatic for several hours are unlikely to develop toxicity and may be discharged [4].

Summary

From the few reported cases, toxicity of the black locust tree appears to be caused by the lectin robin. Gastrointestinal effects are the most common manifestation with supportive care and medical observation being the standard therapy. Particular caution should be taken in regards to pediatric cases as toxic doses are considerably less in this population; however even those patients are expected to fully recover.

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