Anticholinergic Syndrome in Response to Lupin Seed Toxicity

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Abstract

Lupin (also known as lupine or *turmus*), a member of the legume family, is an important source of protein in areas of the Middle East and the Andean highlands. It has recently come into worldwide use as a soybean substitute. An elaborate cooking process is necessary to remove toxic alkaloids in the seeds.

We describe a case of anticholinergic toxicity in a patient who ingested improperly prepared lupin seeds. Presenting symptoms included blurred vision, dry mouth, nervousness, and malaise**.** The diagnosis was based on past history and clinical findings. The condition resolved spontaneously within one day. This report is intended to alert emergency medicine physicians to the potential toxicity of edible lupin and the importance of taking patient herbal and food consumption into account in the differential diagnosis of toxic syndromes.

MeSH Words: Lupin/lupine toxicity, Anticholinergic toxicity; Quinolizidine alkaloids

Introduction

Plant poisoning is often encountered in emergency medicine. It usually occurs when people mistakenly ingest a product they thought was edible or deliberately ingest a toxic produce as an herbal remedy of for purposes of self-harm.

Lupin, a member of the legume family, is commonly consumed as a snack in the Middle East and is coming into more widespread use as a high-protein soy substitute. An elaborate cooking process is needed to remove toxic alkaloids in the lupin seeds before consumption. We describe a woman who presented with anticholinergic toxicity after eating improperly prepared lupin seeds.

Case Report

A 35-year-old woman presented to the emergency department in the evening with complaints of blurred vision, dry mouth, nervousness, and malaise. Anamnesis revealed that the symptoms had started suddenly, about one half-hour after she consumed a handful of lupine seeds.

Previous medical history was noncontributory. The patient denied known allergies, current intake of medications, or use of alcohol or recreational drugs.

On physical examination, she appeared alert but uncomfortable and agitated. Her general health condition was good. Pulse rate was 120 bpm,

blood pressure 123/90 mmHg, temperature 36.4^0 C p.o. with 98% oxygen saturation in room air. The oral mucosa was dry, but the oropharynx was otherwise unremarkable. The skin appeared normal. The pupils were dilated to 6 mm bilaterally, with minimal response to light. Cardiovascular examination revealed tachycardia but no murmurs. Findings on chest and abdominal examination were normal.

Neurological examination confirmed bilateral mydriasis with normal optical fundi. No abnormalities were noted in the other cranial nerves, or in limb strength, deep tendon reflexes, sensitivity, or coordination. The electrocardiogram showed sinus tachycardia of 120 bpm and a normal QRS and QTc interval.

On laboratory work-up, blood count, blood sugar level, electrolyte level, and kidney and liver function were within normal range. Chest X-ray and head computed tomography revealed no abnormalities.
The clinical diagnosis was partial anticholinergic toxidrome due to ingestion of lupin seeds. The patient was admitted for observation. The following morning, she was completely asymptomatic. No additional tests were done, and the patient was discharged home.

The patient was questioned about the process of lupin preparation. She reported that after she had bought the seeds at the market, she boiled them for half an hour and then soaked them in water for two days, changing the water once in between. The seeds had been eaten when they were still bitter.

Discussion

Lupin is the common name for members of the genus *Lupinus* of the legume family. It is known as lupines in the United States and as *turmus* in the Middle East. The plant is characterized by long flowering spikes in a range of colors. The genus includes more than 500 species; some are used for ornamental purposes and others have formed part of the traditional diet of the Mediterranean area and Andean highlands for thousands of years [1]. The most common species produced today worldwide are *L. angustifolius, L. albus* and *L. luteus.*

Although lupin seeds are currently gaining global popularity as a good protein source, their high alkaloid content (2-3%) is a significant limiting factor to their more widespread consumption. The alkaloids confer a bitter taste to the seed and are toxic when ingested. To eliminate them, an elaborate cooking process is necessary. The seeds must first be soaked in water overnight, then boiled for several hours, then again soaked for several days with 2 to 3 changes of water each day. When the "debittering" process is complete, the seeds may be salted and eaten [2], with or without the skin. In the Mediterranean area, they are also preserved in a salty solution (like olives) and served as an appetizer or in salads. To take advantage of their high protein content, researchers have cultivated a low-alkaloid (about 0.001% of the natural plant), nontoxic variant of the lupin seed, called "sweet lupin", for use as an alternative to soybean, as flour or bran or a dairy substitute, and for livestock feed [1]. As its name indicates, sweet lupin is not bitter and does not need to be soaked in water before ingestion.

Allergic reactions to lupin are very rare. They may occur either because of cross-reactivity in people with peanut allergy or de novo in people with no previous food allergy [3,4]. The severity of the reaction ranges from mild symptoms to anaphylaxis.

There are also reports of reactions to the many toxic alkaloids in lupin species, especially the more than 70 types of quinolizidine alkaloids. The frequency and distribution of the alkaloids varies by species. The most relevant in terms of toxicity are sparteine and lupanine, which are metabolized by the liver. Studies in rodents have shown that sparteine, the more toxic of the two, has cardiac, oxytocic, and ganglioplegic effects, whereas lupanine has anticholinergic effects [2]. The severity of the toxicity depends on the amount of alkaloid ingested. It may be more severe in slow metabolizers, who account for 6-10% of the general white population [5].

Most of the published reports so far on acute human toxicity have been anecdotal. Case studies from Europe described a 51-year-old woman who presented with an anticholinergic toxidrome after ingesting bitter lupine seeds [6] and a man with lupin-induced xerostomy [7]. Others described anticholinergic toxicity induced by ingestion of lupin seeds as a home remedy for diabetes mellitus [5] or by drinking the water in which the seeds had been soaked [8]. All patients

recovered uneventfully. Findings of rapid excretion of the alkaloids in animal studies support the assumption that chronic toxicity is unlikely. [2]

Lupines are the lest common of the many plants with anticholinergic properties (Table 1) [9] and the most clinical relevant are those from the Atropa Belladona and Datura species.

Table 1.

Anticholinergic Plants
Atropa Belladonna (Deadly Nightshade)
Mandrafora officinarum (Mandrake)
Datura species (Datura)
Hyocyamus niger (Henbane)
Lupinus species

The diagnosis of lupin toxicity is based on the history and the clinical findings. In some cases, manifestations are subtle, and a high index of suspicion is necessary. There are no specific tests. Treatment, like for other anticholinergic toxicities, is mainly supportive, with monitoring of vital signs. In severe or persistent cases, physostigmine may be considered [2]. Benzodiazepines may be prescribed to alleviate anxiety.

Conclusion

We describe a patient who presented with partial anticholinergic toxicity after eating lupin beans that had undergone an incomplete debittering process. The patient was carefully followed for 24 hours. No intervention was required. All symptoms resolved spontaneously.

This report is intended to alert physicians to the potential toxicity of edible lupins that are improperly prepared and to the importance of taking herbal and food consumption into account in the differential diagnosis in patients who present with toxic syndromes.

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