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Lupin Intoxication Presenting as a Stroke Mimic in an Elderly Patient: A Case Report

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Abstract

Bitter lupin beans are traditionally consumed in the Eastern Mediterranean region and contain quinolizidine alkaloids (QAs), including lupanine and sparteine. Inadequate processing may result in clinically significant exposure to these alkaloids, which interfere with cholinergic neurotransmission and can produce neurological and autonomic manifestations. Although lupin intoxication is classically associated with features such as mydriasis, xerostomia, tachycardia, visual disturbances, and urinary retention, the clinical presentation may be variable. We report the case of a 78-year-old man with multiple vascular risk factors and a history of bioprosthetic aortic valve replacement who presented to the emergency department (ED) with acute dysarthria, severe dizziness, gait instability, and urinary retention shortly after consuming approximately one cup of home-prepared bitter lupin beans. Because of the abrupt neurological presentation, an acute stroke protocol was initiated. Brain magnetic resonance imaging (MRI), including diffusion-weighted sequences, showed no definite evidence of acute ischemic injury. Following neurological assessment and consideration of the exposure history and clinical course, lupin-induced neurotoxicity was considered the most likely clinical diagnosis. The patient received supportive care, with complete resolution of urinary and neurological symptoms within approximately 24 hours. This case highlights an uncommon presentation of lupin intoxication in which neurological manifestations closely mimicked an acute cerebrovascular event. Recognition of dietary exposures may be particularly important in emergency settings when evaluating patients with acute neurological symptoms while maintaining appropriate consideration of cerebrovascular disease.

Categories: Neurology, Nutrition, Emergency Medicine
Keywords: anticholinergic toxidrome, emergency medicine, lupin intoxication, quinolizidine alkaloids, stroke mimic, toxicology

Introduction

Lupin beans (*Lupinus* species), locally known as tourmos or termos, are widely consumed as a traditional snack across Lebanon and the Mediterranean region [1,2]. Bitter lupin varieties contain quinolizidine alkaloids (QAs), including lupanine, sparteine, and related alkaloids, which are responsible for their characteristic bitter taste and toxic potential [3]. Acute anticholinergic toxicity following consumption of inadequately processed bitter lupin has been described in case reports and case series [1,2,4,5].

QAs interfere with cholinergic neurotransmission, including effects on nicotinic acetylcholine receptors, and may consequently affect both the central and peripheral nervous systems [3]. Reported manifestations include mydriasis, xerostomia, blurred vision, tachycardia, dizziness, weakness, urinary retention, gastrointestinal symptoms, altered mental status, and impaired coordination [1,2,4,5].

The onset of toxicity is generally rapid, occurring within minutes to hours after ingestion [2,4,5]. However, the clinical presentation may vary according to the type and concentration of QAs, the quantity consumed, the degree of processing, and individual susceptibility [2,3,6]. Importantly, not all patients develop the complete classic anticholinergic toxidrome.

Neurological manifestations of lupin intoxication have previously been reported and are therefore not unique to this case [2,5,7,8]. However, the predominance of acute dysarthria and truncal ataxia in an elderly patient with substantial vascular risk factors created a particularly challenging clinical resemblance to posterior circulation ischemia. This report highlights the importance of considering dietary toxicological exposures within the differential diagnosis of acute neurological presentations while maintaining an appropriate stroke evaluation. Importantly, the absence of definite acute ischemic changes on initial neuroimaging should not by itself lead to diagnostic closure, and other differential diagnoses, including infectious and metabolic causes, as well as cerebrovascular events, should also be considered according to the clinical context.

Case Presentation

A 78-year-old man presented to the ED with sudden-onset dysarthria, severe dizziness, and gait instability. His medical history was significant for type 2 diabetes mellitus, essential hypertension, benign prostatic hyperplasia, and bioprosthetic aortic valve replacement in 2013. The initial valve replacement was complicated by infective endocarditis, requiring redo aortic valve replacement in 2018.

His regular medications included lercanidipine 20 mg daily, bisoprolol 2.5 mg daily, perindopril 10 mg daily, clopidogrel 75 mg daily, alfuzosin 10 mg daily, and rosuvastatin 20 mg daily. He was a lifelong nonsmoker and denied alcohol or recreational drug use.

Collateral history obtained from his wife revealed that he had consumed approximately one cup of home-prepared bitter lupin beans at 11:30 PM. He had been asymptomatic before ingestion. Approximately 30 minutes later, he developed dizziness, lightheadedness, gait instability, and urinary retention, followed by diaphoresis and slurred speech. He denied fever, dyspnea, chest pain, palpitations, headache, visual disturbances, or focal limb weakness.

The exact weight of the consumed lupin beans was not available, and their QA concentration was not measured; therefore, the ingested alkaloid dose could not be quantified.

On arrival, his blood pressure was 159/72 mmHg, heart rate was 76 beats/minute with a regular rhythm, respiratory rate was 16 breaths/minute, temperature was 36.7°C, and oxygen saturation was 99% on room air. Bedside capillary blood glucose was 139 mg/dL.

The patient was alert and fully oriented, with a Glasgow Coma Scale score of 15/15 [9]. Neurological examination demonstrated prominent dysarthria and truncal ataxia. Cranial nerve examination was otherwise unremarkable, with preserved extraocular movements and no facial asymmetry. Motor strength was 5/5 in all four extremities, and sensation was intact. His pupils were equal, round, and briskly reactive to light, measuring 3 mm bilaterally, without mydriasis or anisocoria. His National Institutes of Health Stroke Scale score was 1 [10]. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

Given the abrupt onset of neurological symptoms, advanced age, vascular risk factors, and history of aortic valve replacement, an acute stroke code was activated because of concern for a posterior circulation or cardioembolic ischemic event.

Laboratory investigations were obtained as part of the evaluation for acute neurological symptoms and are summarized in Tables *1*-*3*. Most findings did not identify an acute metabolic, inflammatory, or cardiac explanation for the presentation. Relevant abnormalities included mild anemia, mild hyponatremia, elevated blood urea nitrogen and serum creatinine with reduced estimated glomerular filtration rate, and mild elevation of aspartate aminotransferase. Cardiac biomarkers and C-reactive protein were within their respective reference ranges.

Test Parameter	Patient Result	Reference Range	Units
White blood cell count (WBC)	9.28	4.0-10.0	$\times 10^3/\mu L$
Red blood cell count (RBC)	4.04	4.5-6.5	$\times 10^{12}/L$
Hemoglobin (HGB)	12.2	13.0-18.0	g/dL
Hematocrit (HCT)	34.9	40.0-54.0	%
Mean corpuscular volume (MCV)	86.4	80.0-94.0	fL
Mean corpuscular hemoglobin (MCH)	30.2	27.0-31.0	pg
Mean corpuscular hemoglobin concentration (MCHC)	35.0	30.0-35.0	g/dL
Platelet count (PLT)	159	150-400	$\times 10^9/L$
Mean platelet volume (MPV)	10.6	7.2-11.1	fL
Neutrophils	69.7	40.0-65.0	%
Lymphocytes	19.2	25.0-40.0	%
Monocytes	8.3	2.0-8.0	%

TABLE 1: Complete blood count

CBC, complete blood count; HCT, hematocrit; HGB, hemoglobin; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPV, mean platelet volume; PLT, platelet count; RBC, red blood cell count; WBC, white blood cell count

Test Parameter	Admission Value	Reference Range	Units
Sodium	135	136-145	mEq/L
Potassium	3.6	3.5-5.1	mEq/L
Chloride	98	98-107	mEq/L
Carbon dioxide (CO_2)	28	22-29	mmol/L
Blood urea nitrogen (BUN)	37.0	8.4-25.7	mg/dL
Serum creatinine	1.46	0.7-1.3	mg/dL
Estimated glomerular filtration rate (eGFR)	45	>60	mL/min/1.73 m^2
Aspartate aminotransferase (AST)	60	11-34	U/L
Alanine aminotransferase (ALT)	23	<45	U/L
Gamma-glutamyl transferase (GGT)	18	<55	U/L
Lipase	57	≤60	U/L

TABLE 2: Biochemistry, renal function, and liver enzyme panel

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CO_2, carbon dioxide; eGFR, estimated glomerular filtration rate; GGT, gamma-glutamyl transferase

Test Parameter	Admission Value	Reference Range	Units
C-reactive protein (CRP)	2.1	<5.0	mg/L
High-sensitivity troponin I	16.7	<34.2	ng/L
Creatine kinase-MB (CK-MB)	1.1	<5.2	ng/mL
Creatine phosphokinase (CPK)	63	30-200	U/L

TABLE 3: Cardiac biomarkers and inflammatory profile

CK-MB, creatine kinase-MB; CPK, creatine phosphokinase; CRP, C-reactive protein

Brain MRI, including diffusion-weighted sequences, was performed during the acute evaluation and showed no definite evidence of acute ischemic injury. Posterior fossa imaging demonstrated no definite acute abnormality involving the cerebellum or brainstem (Figure *1*), while higher axial images demonstrated mild age-related cerebral atrophy with no signs of acute ischemic stroke (Figure *2*).

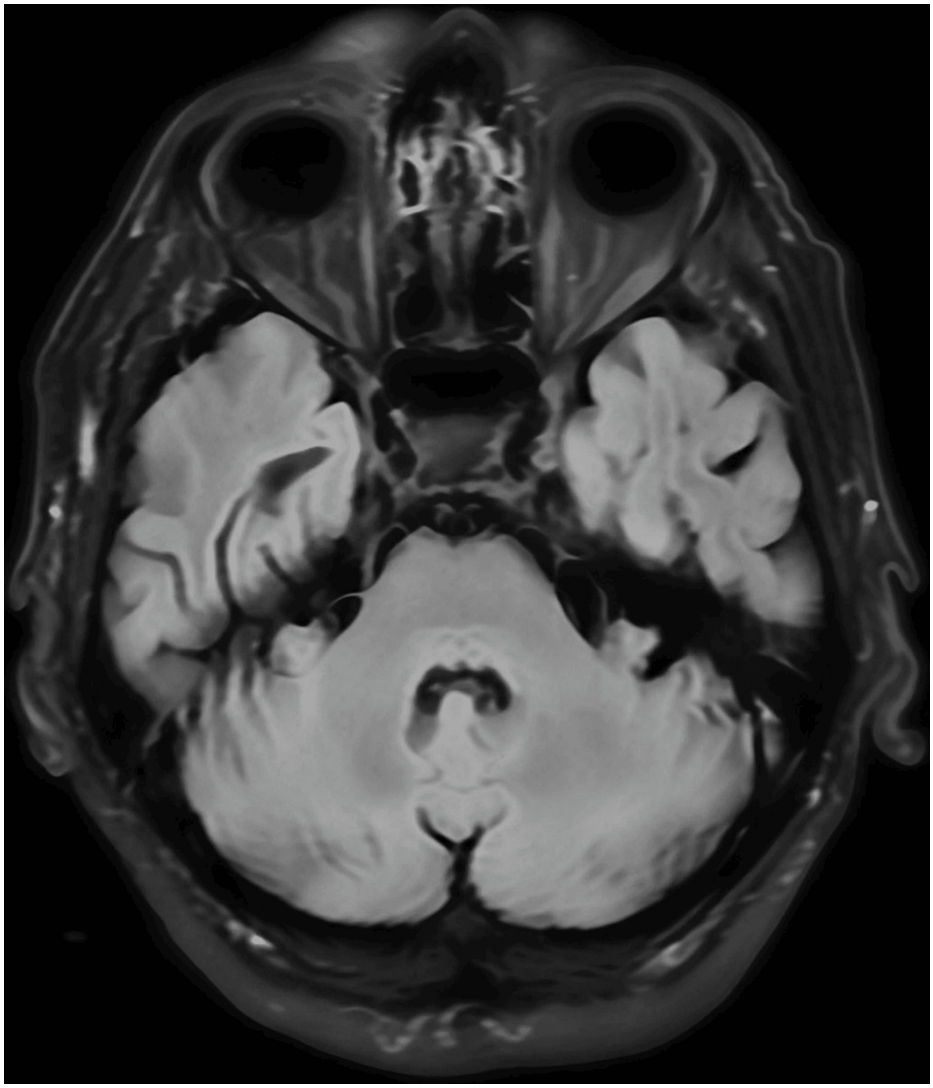


FIGURE 1: Axial brain magnetic resonance imaging at the level of the posterior fossa

Axial fluid-attenuated inversion recovery (FLAIR) magnetic resonance image demonstrating unremarkable cerebellar hemispheres and brainstem without evidence of acute ischemic injury or mass effect

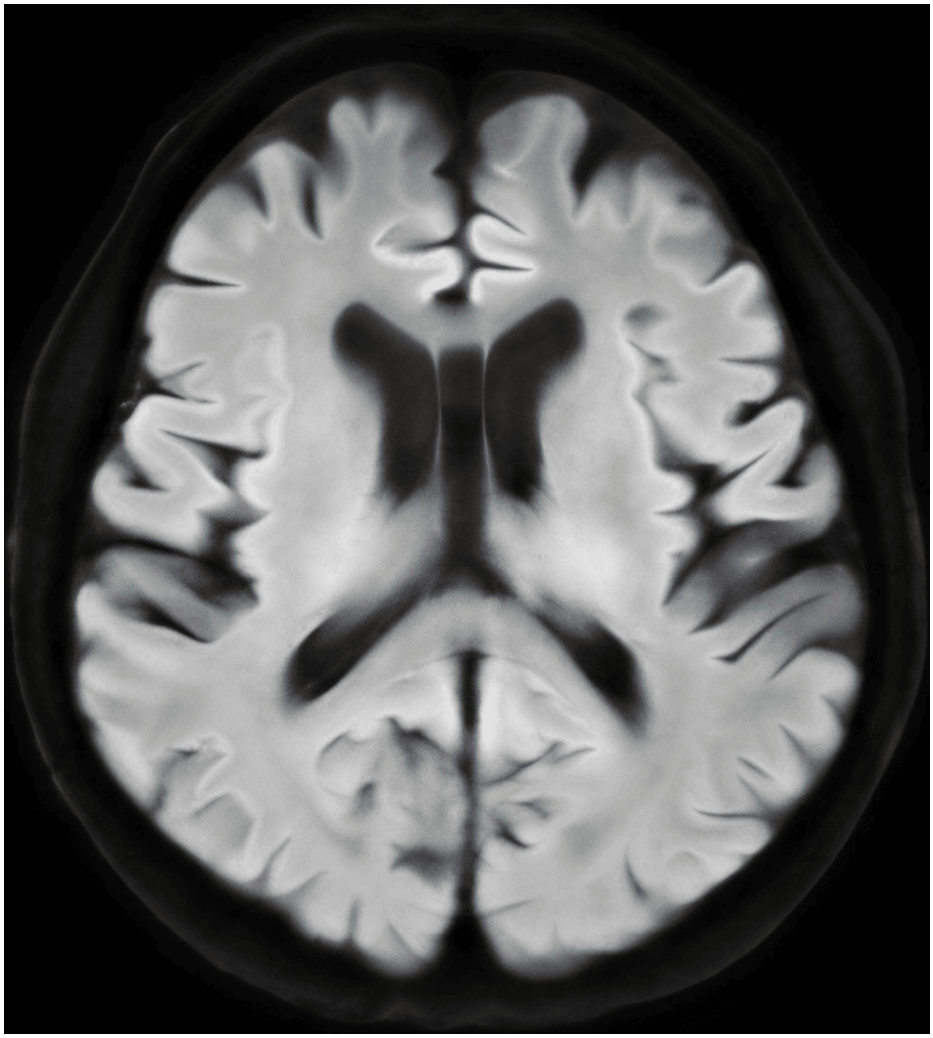


FIGURE 2: Axial brain magnetic resonance imaging at the level of the cerebral hemispheres

Axial brain magnetic resonance image without evidence of acute ischemic injury or mass effect

Following the initial stroke evaluation, the differential diagnosis was reconsidered. The neurology service evaluated the patient, and lupin-induced neurotoxicity was considered the most likely clinical diagnosis based on the documented exposure history, the combination of neurological and autonomic manifestations, and the subsequent clinical course.

The patient was admitted for supportive management and neurological observation. He did not require cholinesterase inhibitor therapy. His urinary retention resolved over the following 12 hours, and his dysarthria and gait instability subsequently resolved, with return to his neurological baseline by approximately 24 hours after ingestion. He was discharged home in stable condition with counseling regarding the risks associated with inadequately processed bitter lupin beans. No formal outpatient follow-up was performed after discharge.

Discussion

This case highlights lupin intoxication as an uncommon toxicological condition that can produce acute neurological manifestations resembling cerebrovascular disease. In this patient, the predominance of dysarthria, severe dizziness, and truncal ataxia, combined with advanced age and vascular comorbidities, appropriately prompted an urgent stroke evaluation. The accompanying urinary retention and history of recent bitter lupin ingestion subsequently raised consideration of a toxicological etiology.

Lupin toxicity results primarily from QAs such as lupanine and sparteine, which interfere with cholinergic neurotransmission and may affect both central and peripheral neurological function [3]. Clinical manifestations vary considerably. Classic findings include mydriasis, xerostomia, blurred vision,

tachycardia, and urinary retention, while neurological manifestations such as dizziness, weakness, impaired coordination, confusion, and other alterations in neurological function have also been described [1,2,4,5,7,8].

Neurological involvement itself is therefore not novel. What distinguishes the present case is the predominance of acute stroke-like neurological manifestations, particularly dysarthria and truncal ataxia, despite the absence of prominent mydriasis or tachycardia.

The variability of lupin intoxication may reflect differences in lupin species, QA composition and concentration, preparation methods, quantity consumed, and individual susceptibility [2,3,6]. Previous Lebanese cases described anticholinergic toxicity after ingestion of approximately 200-500 g of inadequately soaked bitter lupin beans [1]. In the present case, the patient reported consuming approximately one cup; however, the beans were not weighed, and their QA concentration was not analyzed. Consequently, the patient's exposure cannot be directly compared quantitatively with previously reported toxic doses. This represents an important limitation when interpreting the exposure-response relationship in this case.

This case illustrates the importance of maintaining a broad differential diagnosis while simultaneously prioritizing time-sensitive cerebrovascular disease. Acute dysarthria and gait ataxia in an elderly patient with vascular risk factors warrant urgent stroke assessment irrespective of a possible toxicological exposure.

Brain MRI in this patient showed no definite acute ischemic injury; however, an early negative diffusion-weighted MRI does not completely exclude acute ischemic stroke, particularly small or posterior circulation lesions [11]. The subsequent complete clinical resolution and compatible exposure history supported lupin intoxication as the most likely clinical diagnosis but cannot establish the diagnosis with absolute certainty.

A detailed dietary and exposure history can therefore provide important diagnostic information in patients with unexplained neurological presentations. When acute neurological manifestations occur shortly after ingestion of a potentially toxic food, food-related intoxication should be incorporated into the differential diagnosis without prematurely abandoning evaluation for neurological emergencies. Recognition of the exposure may help guide observation and supportive treatment when the overall clinical course is compatible with toxicity.

Several limitations should be acknowledged. First, the diagnosis of lupin intoxication was clinical and was not confirmed by measurement of QAs in the consumed beans or in biological samples. Second, the exact weight of lupin consumed was unavailable, preventing estimation of the ingested QA dose. Third, although the initial brain MRI showed no definite acute ischemic injury, early diffusion-weighted MRI cannot completely exclude acute ischemic stroke, particularly in the posterior circulation [11]. Finally, no formal outpatient follow-up was performed after discharge; therefore, longer-term clinical outcome data are unavailable.

Conclusions

Lupin intoxication may produce acute neurological manifestations that closely resemble a cerebrovascular event. In elderly patients with vascular risk factors, symptoms such as sudden dysarthria and gait instability should continue to prompt appropriate emergency stroke evaluation, even when a potentially relevant dietary exposure is identified. A careful dietary history may nevertheless reveal an important toxicological alternative, particularly when neurological symptoms are accompanied by autonomic manifestations such as urinary retention. In the present case, the exposure history, clinical pattern, absence of definite acute ischemic injury on initial MRI, and complete spontaneous resolution supported lupin-induced neurotoxicity as the most likely clinical diagnosis.

Importantly, the absence of acute abnormalities on initial neuroimaging should not lead to premature diagnostic closure. Other potential causes of acute neurological symptoms, including urinary tract infection, metabolic disturbances, and ischemic stroke, should remain in the differential diagnosis and be considered in light of the clinical presentation, particularly when symptoms persist or evolve. Awareness of this presentation may assist emergency physicians in maintaining an appropriately broad differential diagnosis while avoiding premature diagnostic anchoring.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Joelle Kalaji, Zahia Eid, Charbel Tannous, Nancy Matar

Acquisition, analysis, or interpretation of data: Joelle Kalaji, Zahia Eid, Charbel Tannous

Drafting of the manuscript: Joelle Kalaji, Zahia Eid, Charbel Tannous, Nancy Matar

Critical review of the manuscript for important intellectual content: Joelle Kalaji, Zahia Eid, Charbel Tannous, Nancy Matar

Supervision: Nancy Matar

Disclosures

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References

1. Awada A, Atallah D, Zoghbi A: Anticholinergic syndrome after intoxication by lupine seeds (Tourmos) . J Med Liban. 2011, 59:233-4.
2. Barmo N, Sarieddine A, Al Lawati D, et al.: Anticholinergic toxicity from lupin bean consumption: a case series. Toxicol Commun. 2025, 9:2569241. 10.1080/24734306.2025.2569241
3. Schrenk D, Bodin L, Chipman JK, et al.: Scientific opinion on the risks for animal and human health related to the presence of quinolizidine alkaloids in feed and food, in particular in lupins and lupin-derived products. EFSA J. 2019, 17:e05860. 10.2903/j.efsa.2019.5860
4. Pingault NM, Gibbs RA, Barclay AM, Monaghan M: Two cases of anticholinergic syndrome associated with consumption of bitter lupin flour. Med J Aust. 2009, 191:173-4. 10.5694/j.1326-5377.2009.tb02732.x
5. Ozkaya PY, Ari HF, Turanli EE, et al.: Severe lupin bean intoxication: an anticholinergic toxidrome . Pediatr Emerg Med J. 2021, 8:108-11. 10.22470/pemj.2021.00262
6. Schryvers S, Arinzechukwu C, Miserez B, Eeckhout M, Jacxsens L: The fate of quinolizidine alkaloids during the processing of lupins (Lupinus spp.) for human consumption. Food Chem. 2023, 429:136847. 10.1016/j.foodchem.2023.136847
7. Flores-Pamo AE, Pisano E, Carreazo NY: Anticholinergic toxicity in a one-year-old male following ingestion of Lupinus mutabilis seeds: case report. Sao Paulo Med J. 2018, 136:591-3. 10.1590/1516-3180.2016.0157220517
8. Alessandro L, Wibecan L, Cammarota A, et al.: Pupillary disorders in the emergency room: lupinus mutabilis intoxication. J Clin Toxicol. 2017, 7:360. 10.4172/2161-0495.1000360
9. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale . Lancet. 1974, 2:81-4. 10.1016/S0140-6736(74)91639-0
10. Brott T, Adams HP Jr, Olinger CP, et al.: Measurements of acute cerebral infarction: a clinical examination scale. Stroke. 1989, 20:864-70. 10.1161/01.str.20.7.864
11. Oppenheim C, Stanescu R, Dormont D, et al.: False-negative diffusion-weighted MR findings in acute ischemic stroke. AJNR Am J Neuroradiol. 2000, 21:1434-40.