Cerebrovascular Diseases

Brief Report

Cerebrovasc Dis
DOI: 10.1159/000525686

Received: March 13, 2022
Accepted: June 13, 2022
Published online: October 25, 2022

Jamaican Susumber Berry Poisoning Mimicking Acute Stroke

Jonathan Tamaiev[a] Joshua Trebach[b] Michela Rosso[c] Jeremy Moriarty[d]
Phil DiSalvo[b] Rana Biary[b] Mark Su[b] Jonathan Perk[c] Steven R. Levine[c, e, f]

[a]Department of Psychiatry, SUNY Downstate Health Sciences University, Brooklyn, NY, USA; [b]Division of Medical Toxicology, Ronald O. Perelman Department of Emergency Medicine, NYU Grossman School of Medicine, New York, NY, USA; [c]Department of Neurology, SUNY Downstate Health Sciences University, Brooklyn, NY, USA; [d]Wakefield Division, Department of Emergency Medicine, Montefiore Medical Center, Bronx, NY, USA; [e]Department of Neurology, Kings County Hospital Center, Brooklyn, NY, USA; [f]Department of Neurology, Jaffe Stroke Center, Maimonides Medical Center, Brooklyn, NY, USA

Keywords
Stroke mimic · Solanum torvum · Susumber · Susumber berries

Abstract
Background: Stroke mimics are non-vascular conditions that present with acute focal neurological deficits, simulating an acute ischemic stroke. Susumber berry (SB) toxicity is a rare cause of stroke mimic with limited case reports available in the literature. ***Objectives:*** We report four new cases of SB toxicity presenting as stroke mimic, and we performed a systematic review. ***Methods:*** MEDLINE/EMBASE/WoS were searched for "susumber berries," "susumber," or "solanum torvum." ***Results:*** 531 abstracts were screened after removal of duplicates; 5 articles and 2 conference abstracts were selected describing 13 patients. A total of 17 patients who ingested SB and became ill were identified, including our 4 patients. All but one presented with acute neurologic manifestation; 16 (94%) presented with dysarthria, 16 (94%) with unstable gait, 8 (47%) with nystagmus/gaze deviation, 10 (59%) with blurry vision, and 5 (29%) with autonomic symptoms. Six (35%) required ICU admission, and 3 (18%) were intubated. Fourteen (82%) had a rapid complete recovery, and 3 were hospitalized up to 1 month. ***Conclusions:*** SB toxicity can cause neurological symptoms that mimic an acute stroke typically with a posterior circulation symptom complex. Altered SB toxins (from post-harvest stressors or temperature changes) might stimulate muscarinic/nicotinic cholinergic receptors or inhibit acetylcholinesterase, causing gastrointestinal, neurological, and autonomic symptoms. In cases of multiple patients presenting simultaneously to the ED with stroke-like symptoms or when stroke-like symptoms fail to localize, a toxicological etiology (such as SB toxicity) should be considered.

Introduction

Stroke mimics (SM) are non-vascular conditions that present with an acute focal neurological deficit, potentially corresponding to a particular vascular distribution, simulating an acute stroke [1]. The most common SM include hypoglycemia, toxic-metabolic disturbances, seizures, functional disorders, brain tumors, infection, and migraine [2]. They represent up to a quarter of emergency department (ED) admissions for probable strokes [3].

Correspondence to:
Michela Rosso, michela.rosso@downstate.edu

The rate of treatment of SM with intravenous tissue plasminogen activator (tPA) ranges from 1.4% to 16.7% [4].

Solanum torvum berries, colloquially known as susumber berries (SB), are part of traditional Jamaican cuisine, usually served with cod and rice [5]. Susumber berry poisoning is a rare cause of food poisoning, presenting classically with gastrointestinal symptoms, and if ingested in large amounts, it can cause neurological symptoms that mimic an acute ischemic stroke [6]. Few cases of SB intoxication have been previously described. Here we describe 4 patients presenting to two different EDs in New York with stroke-like symptoms caused by SB intoxication.

Methods

New cases of SB toxicity were identified by patient self-report of SB ingestion which were called into New York City Poison Control. MEDLINE, EMBASE, and WoS databases were systematically searched from inception to November 21, 2021, using a combination of the keywords: "susumber berries," "susumber," or "solanum torvum." Retrieved abstracts were screened by MR and JT for inclusion. Full texts were screened if abstracts were unavailable. Selected full-text manuscripts and conference abstracts from abstract screening were then assessed for final inclusion in the case series. The following variables were recorded: age, sex, activation of stroke code, presenting symptoms, neurological exam, time from exposure to symptom onset, laboratory tests, radiology, and outcome. A Prisma 2020 Checklist is included as an online supplemental table (for all online suppl. material, see www.karger.com/doi/10.1159/000525686).

Results

Case 1 and Case 2 (Brooklyn)

A 72-year-old right-handed woman presented to the ED with progressively worsening slurred speech, difficulty swallowing, blurry vision, nausea, vomiting, and gait instability since waking up (more than 6 h prior to presentation). Comorbidities included hypertension and hyperlipidemia. Vitals signs were normal. Blood glucose was 92 mg/dL. Neurologic examination showed left gaze deviation, horizontal left-beating nystagmus in primary gaze and left gaze, dysarthria, and dysphagia. Sensation and strength were preserved. She had no appendicular dysmetria. Her gait was unstable with left lateropulsion. Her NIHSS score was 2, and a stroke code was activated. Head CT scan and CT angiography were normal, tPA was withheld.

Six hours later, a 67-year-old right-handed woman presented to the same ED with agitation, nausea, vomiting, incomprehensible speech, and an inability to walk, which began an hour before arrival. Comorbidities included diabetes mellitus and hypertension. Blood glucose and vitals were normal, aside from tachycardia of 118 bpm. Neurologic exam revealed agitation, inattention, severe dysarthria, and an unstable wide-based gait. Her NIHSS score was 2, and a stroke code was activated. Since the patient appeared "intoxicated," tPA was withheld. Toxicology screening, head CT, and CT angiography were unremarkable.

On day one of admission, it was discovered that these 2 patients lived together and shared a meal containing SB 12 h and 18 h prior to symptom onset, respectively. These berries were imported by a family member in Jamaica. The second patient reported eating significantly more berries (patient 1 found them to be too bitter) and, though the second patient had less severe overall symptoms, her CK was elevated far more than the first patient (online suppl. Table S1). Both patients had their serum CK and aspartate aminotransferase (AST) peak on day 2 of hospitalization and were discharged home after 5 days of observation and supportive care.

Case 3 and Case 4 (Bronx)

A 48-year-old right-handed man presented to ED with blurred vision, dysarthria, and generalized weakness. His symptoms started in the evening, with blurry vision and a sensation of things moving side-to-side. He then woke up in the middle of the night with headache, vomiting, diarrhea, slurred speech, and weakness. He had hyperlipidemia, but was not on medications. Blood glucose and vitals were normal. His neurological examination was notable for slurred speech, opsoclonus, and finger-to-nose dysmetria; strength was full in segmental examination; however, the patient was unable to walk due to subjective weakness. A stroke code was activated, NIHSS was 2. Head CT and CT angiography were normal; tPA was withheld. He ate SB 12 h before his symptoms onset and reported that the berries tasted more bitter than usual. These berries were imported by a family member from Jamaica. The patient was admitted to the medical ICU for monitoring. He was discharged home on day 4 with complete resolution of symptoms.

A 69-year-old right-handed woman presented to the ED complaining of weakness and blurry vision, shortly after patient 3 who was her son. She shared a meal containing SB with her son the previous evening. She reported that her initial symptoms started 12 h from the time of ingestion and included flickering vision, slurred speech, and leg weakness. Comorbidities included hyperparathyroidism, hypertension, and supraventricular

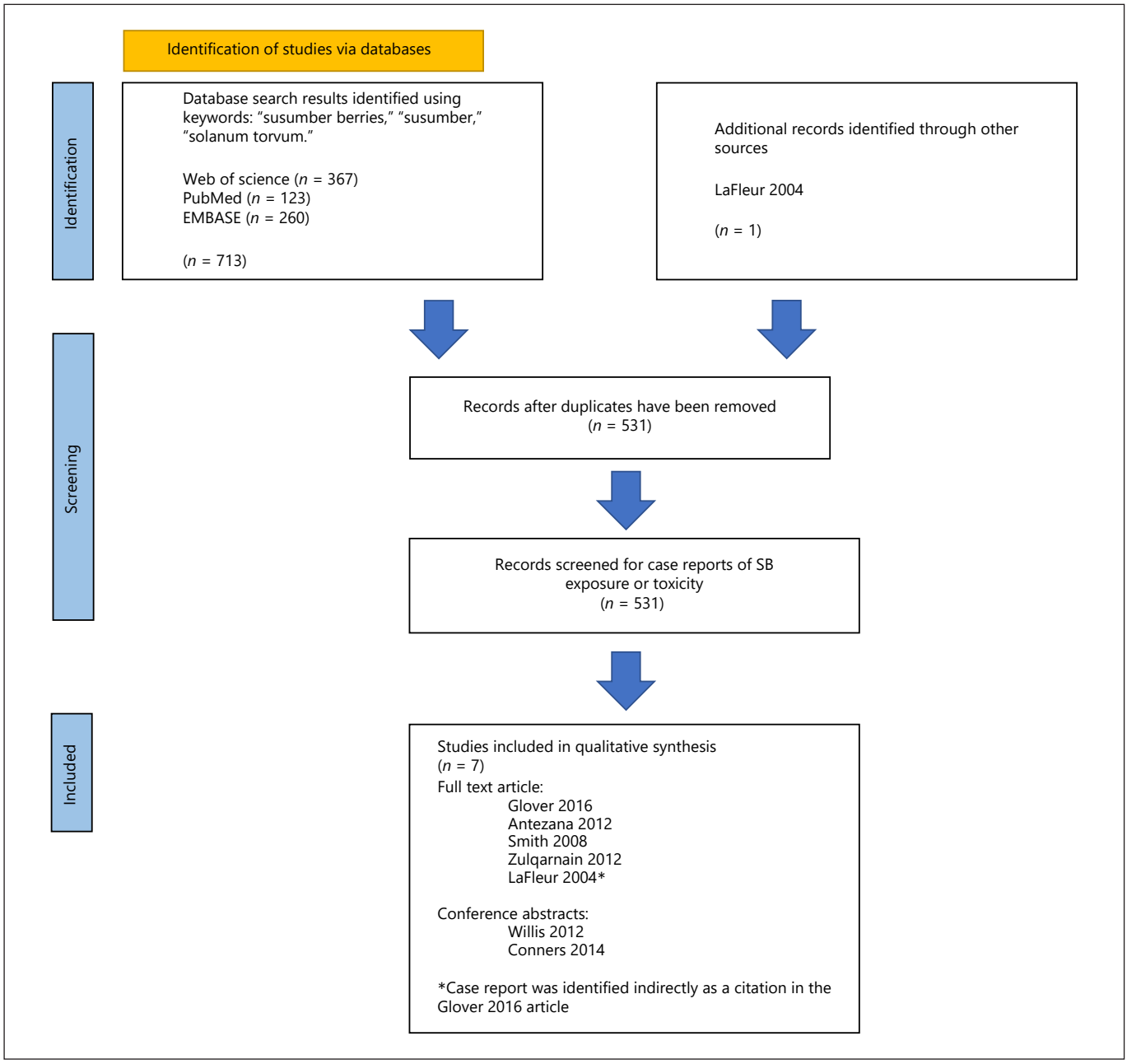

Fig. 1. PRISMA 2020 flow diagram for new systematic review [7].

tachycardia. Vitals and blood sugar were normal. Neurological examination was notable for mild generalized weakness. At the time she arrived, the history of SB ingestion was already elicited from her son; therefore, a stroke code was not activated. Her head CT was unremarkable. She was admitted to the medicine service for monitoring and was discharged 12 h later with complete resolution of symptoms.

Systematic Review

Five hundred thirty-one abstracts were screened after removal of duplicates. Four pertinent full-text articles and two conference abstracts with case reports were identified (shown in Fig. 1). An additional case report in the 2004 Florida Food and Waterborne Illness Surveillance and Investigation Annual Report was found as a citation in the paper written by Glover et al. [5] was included.

Cerebrovasc Dis
DOI: 10.1159/000525686

Table 1. Summary of cases with SB intoxication secondary to direct ingestion

Reference, city, year, age/sex, stroke code activated	Presenting symptoms	Exam	Time from exposure to symptom onset	Amount of consumption	Abnormal labs	Imaging	Outcome
Current paper Brooklyn, NY 2021 72 F Yes 67 F Yes	Dysarthria, dysphagia, unstable gait, blurry vision, dizziness, headache, N/V	T: 98.0 F, BP 125/77, HR 65, RR 17, O2 97% on RA, FS 92 mg/dL Horizontal L-beating nystagmus in primary gaze and L gaze, dysarthria, dysphagia, 4/5 shoulder add/abd, 3/5 elbow and wrist flex/ext, L lateropulsion	12 h (SB imported from Jamaica, kept frozen, boiled before serving, SB cooked with fish)	"Small amount"	Peak CK: 1,028 Peak CK-MB: 19.6 Peak AST/ALT: 70/28 QTc: 600 initially but normalized	Utox: negative CTH: unremarkable, CTA: R vertebral hypoplasia, MRI, ECHO unremarkable	Residual bilateral upper extremity weakness requiring home PT/OT. Discharged home after 5 days
	Severe dysarthria, unstable gait, dizziness, weakness, N/V	T: 99.1F, BP 129/77, HR 103, RR 17, O2 97% on RA, FS 118 mg/dL Severe dysarthria, unstable wide-based gait, 4/5 upper extremity strength	18 h (SB imported from Jamaica, kept frozen, boiled before serving, SB cooked with fish)	"Large amount"	Peak CK: 7,449 Peak CK-MB: 34.3 Peak AST/ALT: 148/30	Utox, slcohol level, salicylate, acetaminophen levels within normal limits CTH, CTA, MRI unremarkable	Symptoms fully resolved in 24 h Discharged home after 5 days
Current paper Bronx, NY 2021 48 M Yes 69 F No	Blurred vision, slurred speech, generalized weakness	VS and FS wnl. Dysarthria, opsoclonus, finger-to-nose dysmetria. Unable to ambulate due to weakness	12 h (SB imported from Jamaica, details unknown, SB cooked)	Unknown but reportedly more than patient DD (his wife) listed below	CK: 152 K+: 3.3 Lactate: 3.3	CTH, CTA, unremarkable	Admitted to critical care for monitoring at the recommendation of the regional poison control center. Discharged home after 4 days with complete resolution of symptoms
	Blurred vision, dysarthria, and leg weakness	VS and FS wnl Dysarthria, 4/5 strength throughout	12 h (imported from Jamaica, details unknown, SB cooked)	Unknown	CK: 155	CTH: no acute findings	Fully resolved Discharged home after 12 h
Antezana et al. (2012) [12] Zulqarnain et al. (2012) [8] Willis et al. (2012) [9] Brooklyn, NY 2012 (2 patients ate fish cooked with SB but did not eat the berries directly therefore are not reported) (A) 65 M No (B) 49 F No	Abdominal pain, V, blurred vision, dysarthria, bilateral hand weakness, unsteady gait	BP 210/110 Opsoclonus, dysarthria, 4/5 wrist ext, interossei, thumb opposition, dysmetria, Waltman sign, gait ataxia	11 h after ingestion (SB imported from Jamaica 3 weeks before, then frozen, consumed after boiling)	8 ounces	CK: 39,000 µ/L Low VC: 1.5–>2.8 L	MRI brain: no acute findings	Admitted to critical care Discharged home after 3 days of hospitalization with total resolution of symptoms and favorable trend in labs
	Dizziness, facial weakness, inability to open eyes, blurry vision, hoarseness, slowing of speech, N/V, abdominal cramping	BP 180/117, HR 40 opsoclonus, distal UE weakness, unsteady gait	11 h after ingestion (SB imported from Jamaica 3 weeks before, then frozen, consumed after boiling)	4 ounces	Increased CK and transaminase; AST: 1,042 Low VC: 1.8–>3.6 L	MRI brain: no acute findings	Admitted to critical care Discharged home after 3 days of hospitalization with total resolution of symptoms and favorable trend in labs
Smith SW, 2008 [6] New York City, 2006 (5 exposed to fish meal with SB, only 3 ate SB [reported here]) (A) Adult F No (B) 55 M No (C) 64 F No	Transient diarrhea	Did not seek medical attention	12 h after ingestion (SB imported from Jamaica, unknown when)	Small amount (due to bitterness)	Non performed	None	Uneventful recovery
	Dizziness, weakness, slurred speech, L facial droop, facial numbness, unsteady gait	Negative at arrival in ED	12 h after ingestion (SB imported from Jamaica, unknown when)	Moderate consumption	Negative	None	Symptoms resolved upon ED arrival
	Facial droop and numbness, dysarthria, blurry vision, dry mouth, gastric discomfort	Hypertension, facial droop and numbness, dysarthria; after 24 h worsening confusion, UE weakness, hypercapnic respiratory failure	12 h after ingestion (SB imported from Jamaica, unknown when)	"A lot"	CK: 9,471 µ/L, Bilirubin 2.8 mg/dL	MRI, CSF analysis, TTE, EMG, carotid duplex: all normal	Admitted to critical care, intubated for respiratory failure, weakness resolved within 24 h; required tracheostomy, discharged on day 27

Table 1 (continued)

Reference, city, year, age/sex, stroke code activated	Presenting symptoms	Exam	Time from exposure to symptom onset	Amount of consumption	Abnormal labs	Imaging	Outcome
Smith SW, 2008 [6] Toronto, 2003 (6 exposed to meal with SB, only 3 ate SB [reported here]) (D) 52 M No (E) 63 F Yes (F) 38 M No	2 episodes of vomiting and diarrhea, followed by blurry vision, ataxia, slurred speech, and weakness	Disconjugate gaze, ptosis, muscle fasciculations, diaphoresis and general weakness, transient dyspnea and urinary incontinence	12 h after ingestion (SB imported from Jamaica, details unknown)	Small portion	Unknown	Unknown	Recovered rapidly within 48 h
	N/V, generalized weakness, slurred speech, blurred vision	Facial droop, dysarthria, disconjugate gaze, left-sided weakness, respiratory failure	13 h after ingestion (SB imported from Jamaica, details unknown)	Moderate	CK: 1,556 μ/L	Unknown	Intubated in critical care, Persistent L hand weakness at discharge 10 days after presentation
	No symptoms at arrival in ED with the family	Dysarthria, disconjugate gaze, excess secretions, truncal weakness > peripheral weakness, respirator insufficiency	14 h after ingestion (SB imported from Jamaica, details unknown)	Large	CK: 62,000 μ/L	Unknown	Intubated in critical care, discharge after 7 days with resolved symptoms
Glover et al. (2016) [5] Conners et al. (2014) [10] New York City, 2016 54 F Yes	Vision changes, slurred speech, gait instability, diffuse myalgias, N/V	BP 166/88, HR 83, RR 14, Temp 36.8°C Myosis, opsoclonus, severe dysarthria, dysmetria, mild upper and upper lower extremity tenderness and weakness, and inability to ambulate	Approximately 12 h after ingestion (SB personally imported from Jamaica, eaten after boiling)	Large	Peak CK: 1,886 μ/L (after 12 h)	MRI brain and CSF unremarkable, EMG: spontaneous activity, early full recruitment pattern, myotonia on the needle evaluation	Discharge home day 3 with no residual symptoms. On follow-up appointment 1 month later, she reported no recurrent symptoms and had a normal neurological examination
LaFleur et al. (2004) [11] Broward County, FL 2004 (9 had meal of cod fish with boiled SB, 4 of them ate the SB with the fish and only these are reported here) 4 patients (3 female, 1 male) had hospital admission; only congregate data presented	Ataxia, slurred speech, facial paralysis, numbness and tingling of the fingers, weakness in legs and arms and altered mental state. No gastrointestinal symptoms	No additional information included	Between 10.5 and 16 h after ingestion, median incubation of 14 h (SB personally imported from Jamaica, eaten after boiling)	One patient had 3–4 tablespoons, the other 3 had 1–2 tablespoons	N/A	N/A	The patients were treated with IV fluid and electrolyte replacement, and 3 of the 4 received mannitol. Patients were hospitalized for 5 days with complete remission of all symptoms at time of discharge

Thirteen cases of toxic SB ingestion have been documented in the literature, and an additional 4 new cases are reported here [8–11]. Table 1 summarizes demographic features, presenting symptoms, physical exam, time from ingestion to symptom onset, amount of SB consumed, abnormal lab values, imaging, and outcomes for all cases.

Of the 17 patients who became ill, all but one (who only ate a "small" amount of berries) presented to the ED with acute neurologic manifestations. All the remaining 16 patients (94%) developed dysarthria as well as unstable gait (either due to weakness or ataxia). Eight patients (47%) had reported negative imaging (with no documentation of imaging studies for the remained of cases). Eight (47%) had nystagmus, opsoclonus, or gaze deviation, and 10 (59%) reported blurry vision. Seven (41%) had gastrointestinal manifestations, and 5 (29%) had autonomic symptoms. Ten (59%) had elevated CK, and 5 (29%) had elevated liver enzymes. Six (35%) required ICU admission, 3 of which (18%) were intubated. Fifteen (88%) had a complete recovery after 1 week, while 3 (12%) were hospitalized for up to a month. In none of the reported cases did patients receive tPA.

Discussion

Stroke mimics are of particular concern in neurology practice – a delay in recognition and treatment of an ischemic stroke may lead to irreversible brain damage; therefore, clinicians experience time pressure in rapidly identifying patients with suspected ischemic stroke [1, 3, 13]. However, over-treatment of patients presenting with focal neurologic deficits may lead to misdiagnosis of SM, leading to unnecessary testing, increased healthcare costs, inappropriate use of stroke facilities, unnecessary expenses, and potential patient harm from radiation (CT scans) or medication administration (tPA) [3, 4, 13–16]. Toxicologic etiologies of stroke mimics are uncommon, resulting in delayed or even inappropriate diagnoses.

SB contains steroidal glycoalkaloids (SGA): the SGA content is increased when the berries are exposed to variations in growing conditions, post-harvest stressors, and temperature changes, transforming normally innocuous berries into toxic substances [12]. In cases of toxicity, patients have subjectively reported berries tasting more bitter than usual. Chromatography studies identified two toxins, solasonine and solamargine, that might act by stimulating muscarinic/nicotinic cholinergic receptors or inhibiting acetylcholinesterase [12]. Interestingly, 5 patients in our case series showed autonomic symptoms, associated with the typical gastrointestinal and neurological presentations.

It is worthwhile to note a historic parallel of neurotoxicity caused by "Jamaica ginger extract." In the 1930s, there was a major outbreak across the United States of thousands of cases of toxic paralysis. They were discovered to be due to a ginger extract product carried in drug stores at the time, used as a remedy for a variety of common ailments. It came as a solution with a high alcohol concentration and was misused by those attempting to circumvent prohibition [17]. In many cases, the extract caused a distinct upper motor neuron syndrome via spinal cord cellular damage. The offending agent caused the neurotoxicity by means of organophosphate poisoning, with the insulting compound identified as triorthocresyl phosphate (TOCP) [18]. Like with SB toxicity, the clinical course often began with GI symptoms, however over the course of 4 days would progress to an irreversible paralysis. It did not present as a constellation of symptoms mimicking an acute stroke [19].

While a comprehensive dietary history is challenging to obtain in the harried setting of early evaluation of acute neurologic deficits, multiple patients presenting simultaneously to the ED with stroke-like symptoms or a constellation of deficits that fail to localize to a single lesion should prompt consideration of toxicologic etiology. A thorough toxicologic history may reveal such responsible ingestions including *Solanum torvum*.

Statement of Ethics

Ethics approval was not required for this study in accordance with SUNY Downstate Medical Center IRB policy on case series up to 3 individuals (Case 1 and 2): this report is granted an exemption from requiring ethics approval. Ethics approval was not required for this study in accordance with Montefiore Medical Center IRB policy on reports of a small number of cases, specifically if the second patient is a 1st-degree relative of an individual patient who manifests similar findings (Case 3 and 4), this report is granted an exemption from requiring ethics approval. Written consent was obtained from the patients for publication of the details of their medical case as part of the hospital admission process.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

All the authors contributed to the study conception and design. Jonathan Tamaiev collected the data, wrote the first draft, and performed the literature review. Joshua Trebach collected the data and wrote the first draft. Michela Rosso performed the literature review and wrote the first draft. Jeremy Moriarty, Phil DiSalvo, Rana Biary, Mark Su, Jonathan Perk, and Steven R. Levine contributed to review and critique.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

References

1 Vilela P. Acute stroke differential diagnosis: stroke mimics. Eur J Radiol. 2017;96:133–44.
2 Pohl M, Hesszenberger D, Kapus K, Meszaros J, Feher A, Varadi I, et al. Ischemic stroke mimics: a comprehensive review. J Clin Neurosci. 2021;93:174–82.
3 Moulin S, Leys D. Stroke mimics and chameleons. Curr Opin Neurol. 2019;32(1):54–9.
4 Nguyen PL, Chang JJ. Stroke mimics and acute stroke evaluation: clinical differentiation and complications after intravenous tissue plasminogen activator. J Emerg Med. 2015;49(2):244–52.
5 Glover RL, Connors NJ, Stefan C, Wong E, Hoffman RS, Nelson LS, et al. Electromyographic and laboratory findings in acute solanum torvum poisoning. Clin Toxicol. 2016;54(1):61–5.
6 Smith SW, Giesbrecht E, Thompson M, Nelson LS, Hoffman RS. Solanaceous steroidal glycoalkaloids and poisoning by solanum torvum, the normally edible susumber berry. Toxicon. 2008;52(6):667–76.
7 Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71.
8 Zulqarnain S, Fahmy S, Cheng S, Khan M, Jamaleddine G. Susumber berries, a bitter experience. Chest. 2012;142(4):971A.
9 Willis J, Garlish F, Baron B, Wiener S, Hoffman R, Alusio A, et al. Not-so-edible susumber berries. 2012 Annual Meeting of the North American Congress of Clinical Toxicology (NACCT) October 1–6, 2012 Las Vegas, NV, USA. Clin Toxicol. 2012;50(7):574–720, abstract 188.
10 Conners N, Glover R, Stefan C, Patterson D, Wong E, Milstein M, et al. Biological and botanical confirmation of solanaceous glycoalkaloid poisoning by susumber berries (*Solanum torvum*). XXXIV International Congress of the European Association of poisons Centres and clinical toxicologists (EAPCCT); 2014 May 27–30; Brussels, Belgium, Clinical Toxicology. 2012;52. p. 295–443.
11 LaFleur K, Schulze SL, Hammonds R. Foodborne outbreak attributed to solanine poisoning from susumber berries, Broward County, February, 2004 (Internet). Food and waterborne illness surveillance and investigation annual report. Florida; 2004. (cited 2021 Nov 18). Available from: http://www.floridahealth.gov/diseases-and-conditions/food-and-waterborne-disease/_documents/2004.pdf.
12 Antezana A, Policard J, Sarva H, Vas G. Susumber berries: unexpected cause of cholinergic poisoning. Neurol Clin Pract. 2012;2(4):362–3.
13 Liberman AL, Prabhakaran S. Stroke chameleons and stroke mimics in the emergency department. Curr Neurol Neurosci Rep. 2017;17(2):15.
14 Goyal N, Male S, Al Wafai A, Bellamkonda S, Zand R. Cost burden of stroke mimics and transient ischemic attack after intravenous tissue plasminogen activator treatment. J Stroke Cerebrovasc Dis. 2015;24(4):828–33.
15 Ali-Ahmed F, Federspiel JJ, Liang L, Xu H, Sevilis T, Hernandez AF, et al. Intravenous tissue plasminogen activator in stroke mimics. Circ Cardiovasc Qual Outcomes. 2019;12(8):e005609.
16 Burton TM, Luby M, Nadareishvili Z, Benson RT, Lynch JK, Latour LL, et al. Effects of increasing iv tpa-treated stroke mimic rates at ct-based centers on clinical outcomes. Neurology. 2017;89(4):343–8.
17 Friedman E. "Jake Leg," other poisonings, physicians as canaries, and the FDA. Medscape J Med. 2008;10(4):103.
18 Morgan JP, Penovich P. Jamaica ginger paralysis: forty-seven-year follow-up. Arch Neurol. 1978;35(8):530–2.
19 Merritt HH, Moore M. Peripheral neuritis associated with ginger extract ingestion. New Engl J Med. 1930;203(1):4–12.