

Food poisoning due to money tree seeds: a case report of toxic encephalopathy

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Abstract

Background: The money tree is a favored indoor potted plant in China. Its seeds resemble chestnuts in both shape and flavor when cooked, which are generally considered non-toxic and safe for consumption.

Case presentation: This report presents the first published case of food poisoning due to the ingestion of large amounts of money tree seeds. A 67-year-old man exhibited toxic encephalopathy symptoms and dyspnea after consuming the seeds for approximately 6 months. After empirical mechanical ventilation, therapeutic plasma exchange, and other medical interventions, the patient's clinical status improved significantly. He was subsequently discharged from the hospital.

Conclusion: Consuming a substantial quantity of money tree seeds within a limited timeframe can lead to poisoning, resulting in adverse outcomes, particularly toxic encephalopathy. Moreover, plasma exchange may have some therapeutic effect.

Keywords: Case report, Money tree, *Pachira macrocarpa*, Respiratory failure, Toxic encephalopathy

Introduction

Pachira macrocarpa, commonly known as the money tree, French peanut, Guinea peanut, lucky tree, and *Pachira glabra*, is a favored indoor potted plant in China. Its seeds (Fig. 1A) resemble chestnuts both in shape and flavor when cooked, which were generally considered non-toxic and safe for consumption. To our knowledge, no documented cases of toxicity resulting from the consumption of money tree seeds have been reported.

Harchelroad et al.^[1] reported that 13% of plants could be identified as poisonous. Between 1983 and 2009 in the United States, 45 cases of fatal plant poisonings were recorded.^[2] In China, 597 poisoning events caused by poisonous plants were reported between 2004 and 2013. The top 3 plants that caused poisoning were kidney beans (124 events, 59.68%), aconite (49 events, 15.91%), and tung tree (16 events, 5.19%).^[3] Acute plant poisoning is more common; however, the acute onset of chronic poisoning has been rarely reported.^[4–7]

Here, we report the case of a 67-year-old man who exhibited toxic encephalopathy symptoms and dyspnea after consuming money tree seeds for approximately 6 months (Fig. 1B). His clinical condition significantly improved following empirical mechanical ventilation,

therapeutic plasma exchange, and other medical interventions. The study was conducted in accordance with the Declaration of Helsinki.

Case presentation

A 67-year-old man from Sanya, Hainan Province of China was admitted to our hospital with chest tightness and dyspnea for 2 days, which had worsened over the preceding day, accompanied by impaired consciousness in October 2022. He underwent partial gastrectomy in 1990 due to a gastric ulcer and reported no other significant medical history.

Over the preceding 6 months, the patient had been collecting money tree seeds from his local area to incorporate into his rice or porridge meals, adding 3–10 seeds each time. Recently, as the seeds matured, he collected more and added 30–40 to each meal.

Two days prior to admission, the patient had experienced chest tightness and dyspnea, accompanied by facial numbness and involuntary angulus oris twisting. Breathing difficulties were further exacerbated in the afternoon of admission, prompting a visit to our emergency department at 21:00 that night. His condition deteriorated with symptoms of confusion, slurred speech, agitation, and difficulty in opening his eyes. Physical examination revealed a respiratory rate of 30–40 breaths per minute, an SpO₂ of 90%, poor bilateral pupillary light reflexes, and no Babinski's reflex. The arterial blood gas findings indicated respiratory alkalosis and metabolic acidosis, with a pH of 7.49, PaCO₂ of 16 mmHg, PaO₂ of 120 mmHg, lactic acid of 1.4 mmol/L, HCO₃⁻ level of 18.5 mEq/L, and base excess of 11.1 mmol/L. No apparent abnormalities in hepatic, renal, and cardiac functions or in electrolyte levels were observed. However, brain computed tomography revealed low-intensity lesions in both cerebellar hemispheres and the brainstem (Fig. 2). Upon admission to our emergency department, tracheal intubation was performed, followed by assisted ventilation and analgesic sedation.

Differential diagnosis

The main differential diagnoses for toxic encephalopathy included encephalitis and cerebral infarction.

Encephalitis

The leading causes of encephalitis are infectious and autoimmune diseases. Herpes simplex virus (HSV) infection is the most common

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figure 1. Money tree seeds. (A) Money tree seeds provided by the relatives of the patient in our case report. (B) The patients incorporated money tree seeds into their meals of rice or porridge, adding 3–10 seeds each time.

and treatable cause of encephalitis. The involvement of the temporal lobe in HSV encephalitis is typically visualized on magnetic resonance imaging (MRI), characterized by abnormal T2 signal abnormalities and contrast enhancement, often exhibiting asymmetric involvement. Infectious encephalitis typically manifests with initial prodromal flu-like illness. The presentation of autoimmune encephalitis typically includes an infectious prodrome and can be differentiated from infectious causes through MRI, cerebrospinal fluid analysis, and neuronal autoantibody testing.

Cerebral infarction

The onset of cerebral infarction is typically abrupt, primarily presenting with symptoms such as paralysis, headache, speech impairment, visual disturbances, and gait abnormalities. The high-signal

lesions observed on diffusion-weighted imaging (DWI) are commonly asymmetric.

Treatment

He was transferred to the intensive care unit on day 2, and MRI revealed symmetric DWI hyperintensity surrounding the bilateral cerebellar dentate nucleus, dorsal medulla oblongata, and midbrain aqueduct (Fig. 3A and B). We administered plasma exchange (totaling 112.5 U) on days 3, 6, and 9 in conjunction with anti-infective therapy, trophic neurotherapy, and rehabilitative exercise. The patient's level of consciousness gradually improved while the difficulty of eye opening diminished. On day 11, tracheal intubation was removed. However, residual dysarthria, dysphagia, bucking, right lower-limb numbness, and hypoesthesia persisted along with grade 4

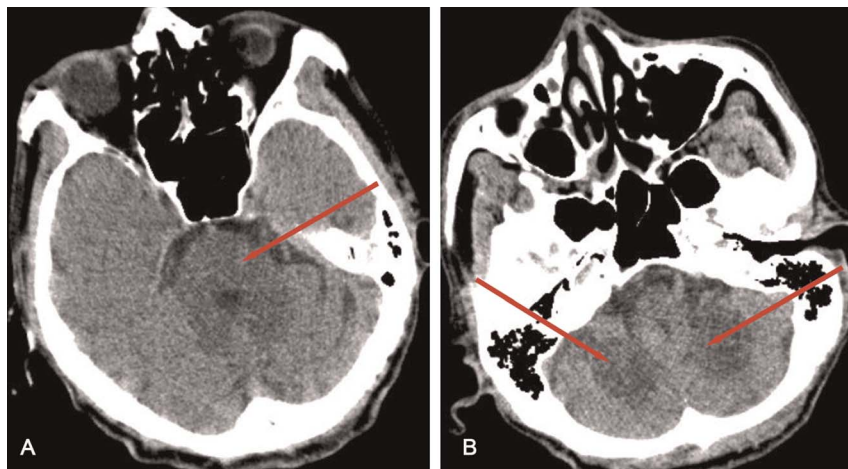


Figure 2. CT scan of a 67-year-old man with toxic encephalopathy due to food poisoning from money tree seeds. (A) Low-density lesions in the brainstem and the patient's dyspnea might have been attributed to the brainstem lesions. (B) Low-density lesions located in the bilateral cerebellar dentate nucleus. CT, computed tomography.

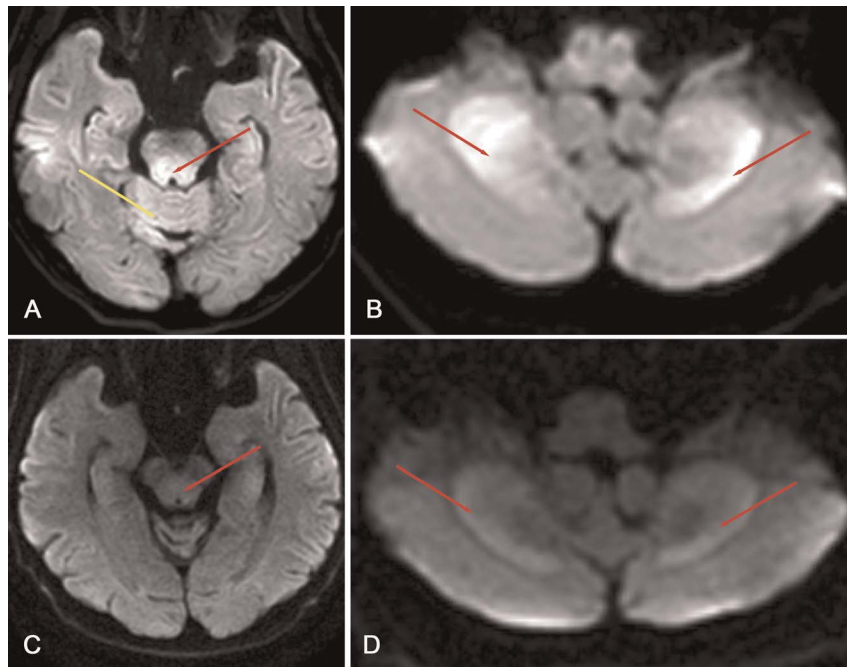


Figure 3. Diffusion-weighted imaging (DWI; $b = 1000$) of a 67-year-old man with toxic encephalopathy due to food poisoning from money tree seeds. (A–B) High-signal DWI at the initial presentation demonstrated symmetric signal hyperintensity surrounding the bilateral cerebellar dentate nucleus (red arrow in Fig. 3B), dorsal medulla oblongata, midbrain aqueduct (red arrow in Fig. 3A), and cerebellar vermis (yellow arrow in Fig. 3A). (C–D) Follow-up DWI after 15 days revealed a reduction in lesion scope (red arrow in Fig. 3C–D) and mostly resolution of the diffusion-restricted areas.

weakness of the right limb. On day 13, the patient's brain MRI revealed a reduction in lesion scope and DWI signal hyperintensity (Fig. 3C and D).

Patient outcome

By day 40, the patient had regained enough function to live independently with improved swallowing dysfunction, speech impairment, and right limb movement disorder. Additionally, the patient's oral diet intake returned to normal before high-pressure oxygen treatment was initiated.

Case of cohabitated wife

The patient's cohabitated wife developed the same symptoms. The woman first exhibited limb and facial numbness 3 days prior to the onset of her husband's symptoms. Within 24 hours, her condition rapidly deteriorated with symptoms of chest tightness, facial spasms, and blepharoptosis, leading to hospitalization at another medical facility. Arterial blood gas analysis revealed respiratory alkalosis and metabolic acidosis (pH: 7.459, PaO₂: 121 mmHg, lactic acid: 6.21 mmol/L). Despite hospitalization, her dyspnea worsened rapidly and culminated in cardiac arrest on the fourth day. The patient died on the sixth day following unsuccessful cardiopulmonary resuscitation.

Discussion

Money tree, also known as *Pachira macrocarpa* of the genus *Platyptoniaceae* or *P. glabra*, is a popular large indoor potted plant from tropical rainforests. In Sanya, Hainan, where the couple resided, it can grow into a tall tree and bear fruit with seeds resembling chestnuts in shape and taste. The seeds are composed of lipids

(43.34%, including palmitic, linoleic, and oleic acid), protein (15.21%), moisture (6.23%), and ash (3.90%).^[8]

In this case, poisoning was primarily diagnosed by comprehensively analyzing the patient's dietary history, clinical manifestations, and characteristic imaging findings indicative of encephalopathy toxica.^[9,10] The patient had been consuming the seeds since 6 months prior, with a recent increase in dosage. The onset was acute and rapidly progressive, with clinical manifestations predominantly affecting the respiratory, extraocular, and facial muscles. Consequently, dyspnea, dysarthria, and weakness of the right distal limb accompanied by confusion were observed. His spouse, who shared meals with him, experienced similar but more severe symptoms 3 days prior to him, which had rapidly progressed to progressive dyspnea. Imaging examination revealed symmetric DWI signal hyperintensity around the bilateral cerebellar dentate nucleus, dorsal medulla oblongata, and aqueduct in the midbrain, consistent with the findings of toxic encephalopathy. Following improvement in the patient's condition, the intracranial lesions decreased in size, and the DWI signal hyperintensity diminished, further confirming our diagnosis as the imaging and clinical manifestations were congruent. Post-hypoxic encephalopathy, characterized by initial improvement followed by neurological decline, is a possible etiology of the leukoencephalopathy observed on MRI.

Few cases of money tree seed poisoning have been reported, and the seeds are generally considered non-toxic and safe for consumption. However, the reason behind the poisoning of this couple remains unclear. We hypothesized that prolonged consumption and recent high-volume consumption of money tree seeds, particularly of undercooked seeds, might have contributed to the accumulation of toxins in their system. Unfortunately, the toxicological test was not completed in this patient. However, given the high risk associated with the disease and the absence of a specific detoxifying agent,

prompt plasma exchange effectively ameliorates the symptoms by rapidly eliminating toxic substances from circulation.^[11–17] Additionally, trace amounts of toxic cyclopropenoid fatty acids have been detected in some peanuts and cottonseed oil^[18–20]; therefore, future research can focus on whether money tree seeds contain cyclopropenoid fatty acids.

In view of the severe worsening symptoms and a predicted high mortality, we conducted empirical therapeutic plasma exchange for the present patients, to remove toxic substances and inflammatory mediators from the blood compartment rapidly.^[21] No clinical indications have been established for the use of therapeutic plasma exchange in the treatment of such patients.^[22–24]

Conclusion

This case presents the first reported case of money tree seed poisoning. The attempted treatments included plasma exchange, anti-infective therapy, trophic neurotherapy, and rehabilitative exercise.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Zhao Y participated in the writing of the paper, Quan X participated in reviewing of the draft, and Wang T participated in project administration and supervision.

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Ethical approval of studies and informed consent

The study followed the principles of the Declaration of Helsinki as revised in 2013. The guidelines of the Ethics Committee of Hainan Hospital of Chinese PLA General Hospital state that the publication of case reports is exempt from ethical approval. Written informed consent was obtained from the 2 patients' legal representative because they were intubated and sedated. This article has the consent of the patients for the use of their data and for the publication of the data that appear in the article.

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