

Case report

Brugmansia suaveolens (angel's trumpet) tea poisoning in a previously healthy young patient: a case report***Intoxicação por chá de Brugmansia suaveolens (trombeta de anjo) em paciente jovem previamente hígido: relato de um caso***

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ABSTRACT: INTRODUCTION: *Brugmansia suaveolens*, popularly known as “angel's trumpet,” is a plant with anticholinergic alkaloids that inhibit the activity of muscarinic receptors in the central and peripheral nervous system. Its toxicity results in agitation, hallucinations, hyperthermia, tachycardia, rhabdomyolysis, renal failure, and death. OBJECTIVE: Report a case of acute intoxication due to ingestion of “angel's trumpet” tea, with associated fulminant hepatitis. METHODS: We present a case report with clinical and laboratory data collected from medical records. The study was approved by the Research Ethics Committee, and Written Informed Consent was obtained from a legally responsive relative. CASE REPORT: A healthy 19-year-old male was hospitalized with a fever of 41°C and generalized tonic-clonic seizures, progressing to orotracheal intubation. He exhibited fixed mydriasis and neck stiffness. We were informed by the family that the patient had made use of the tea from the “angel's trumpet” plant. At admission, he performed a computerized cranial tomography, which showed no midline shift, cerebrospinal fluid without alterations, arterial blood gas analysis with severe metabolic acidosis, acute renal failure requiring dialysis, and fulminant acute liver failure. CONCLUSIONS: “Trumpet tea” intoxication is a neurological emergency that must be quickly recognized for its potential fatal effects. With meningitis as a differential diagnosis, it is important to collect a complete history taking to investigate possible previous contact with the plant. To our knowledge, this is the first case report of fulminant hepatitis caused by “angel's trumpet,” in addition to the neurological symptoms already described in the literature, which further corroborates the importance of the topic in question.

KEYWORDS: *Brugmansia suaveolens*; Anticholinergic alkaloid; Mydriasis; Acute renal and hepatic failure.

RESUMO: Introdução: A *Brugmansia suaveolens*, conhecida como “trombeta de anjo,” é uma planta com alcaloides anticolinérgicos que provocam inibição da atividade de receptores muscarínicos no sistema nervoso central e periférico. Sua toxicidade resulta em agitação, alucinações, hipertermia, taquicardia, rabdomiolise, insuficiência renal e morte. Objetivo: Descrever um caso de intoxicação aguda por chá de trombeta, cursando com hepatite fulminante. Materiais e Métodos: Trata-se de um relato de caso, com dados clínicos e laboratoriais coletados a partir dos registros em prontuário. Termo de Consentimento Livre e Esclarecido foi obtido com familiar e o projeto foi aprovado pelo Comitê de Ética em Pesquisa. Relato de Caso: Masculino de 19 anos, saudável, internado por quadro de febre de 41°C e crises convulsivas tônicas generalizadas, evoluindo para intubação orotraqueal. Apresentava midríase fixa e rigidez de nuca. Família relata que o paciente tinha feito uso de chá de “trombeta de anjo”. À admissão, realizou tomografia computadorizada de crânio, que não evidenciou desvio de linha média, líquido sem alterações, gasometria arterial com acidose metabólica grave, insuficiência renal aguda com necessidade dialítica e insuficiência hepática aguda fulminante. Conclusões: A intoxicação pelo “chá de trombeta” é uma emergência neurológica que deve ser rapidamente reconhecida por ser potencialmente fatal. Tendo meningite como diagnóstico diferencial, é importante obter história clínica completa para investigar possível contato prévio com a planta. Para nosso conhecimento, esse é o primeiro caso a relatar quadro de hepatite fulminante pela “trombeta de anjo,” além dos sintomas neurológicos já descritos em literatura, o que corrobora ainda mais para a importância do tema em questão.

PALAVRAS-CHAVE: *Brugmansia suaveolens*; Alcaloide anticolinérgico; Midríase; Insuficiência renal e hepática aguda.

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INTRODUCTION

Brugmansia suaveolens, commonly known as “angel’s trumpet” or “trumpet tea,” is a plant typical of tropical regions and famous for its hallucinogenic, euphoric, and sedative effects¹, being consumed for medicinal or recreational use. Its mechanism of action is due to substances present in its leaves or seeds, such as hyoscyamine, scopolamine, and atropine (anticholinergic alkaloids), which cause inhibition of the activity of muscarinic receptors in the central and peripheral nervous system². Anticholinergic symptoms usually begin within an hour after ingestion and can continue for days. Severe toxicity results in agitation, hallucinations, hyperthermia, tachycardia, rhabdomyolysis, and renal failure³.

In addition to its psychotropic effects, signs and symptoms of anticholinergic toxicity are common, such as elevated temperature, xerostomia, visual changes, urinary retention, hypertension, cardiorespiratory collapse, coma, and death⁴. Without aggressive supportive care, death can result from renal failure induced by rhabdomyolysis, disseminated intravascular coagulation, arrhythmias, or uncontrolled seizures⁴.

We present a clinical case of angel’s trumpet tea poisoning in a young patient, which led to renal failure and liver dysfunction.

METHODS

This is a descriptive observational case report. Clinical and laboratory data were obtained from medical records. All laboratory analyses were performed in the hospital unit’s laboratory where the patient was hospitalized. A Free and Informed Consent Form for access to data and publication of the case was obtained from family members. The present study was approved by the Research Ethics Committee (CAAE: 64756422.0.0000.5262).

CASE REPORT

A 19-year-old male patient with no previous comorbidities was admitted due to a fever of 41°C associated with generalized tonic-clonic seizures, which led to orotracheal intubation on the same day. Upon physical examination, he was sedated with midazolam and fentanyl, had fixed dilated pupils and neck stiffness, and an ultrasound examination showed an optic nerve sheath measuring 0.5 cm. The diagnostic hypothesis of meningitis was raised, and a computed tomography (CT) scan of the skull was performed, which showed effacement of sulci without midline shift. A lumbar puncture was performed, and cerebrospinal fluid showed no alterations (12 cells, 20% polymorphonuclear, 80% mononuclear, glucose 54 mg/dL, protein 57 mg/dL, rapid molecular test for *Mycobacterium tuberculosis* not detected, Ziehl-Neelsen staining did not

demonstrate microorganisms). On admission, arterial blood gas analysis showed significant metabolic acidosis (pH 7.09 / HCO₃ 13.7 mEq/L / BE -15.2 / lactate 4.8 mg/dL), the patient presented with dark gastric residual and required high doses of vasopressors. Empiric treatment was initiated with ceftriaxone, vancomycin, and dexamethasone. Laboratory tests showed negative serologies for hepatitis viruses (hepatitis A, B, and C), human immunodeficiency virus (HIV), *herpes simplex virus* (HSV), toxoplasmosis, *Epstein-Barr virus* (EBV), cytomegalovirus (CMV), and syphilis. Due to the development of anuria, increased nitrogenous waste, and refractory metabolic acidosis, the patient underwent renal replacement therapy by hemodialysis. A family member informed the medical team that the patient had ingested high doses of “angel’s trumpet tea” recreationally with his cousin, who was in critical condition in another hospital with similar clinical conditions. On the third day of hospitalization, the patient presented a significant increase in transaminases and markers of liver dysfunction (Table 1), characterizing a case of fulminant hepatitis. In light of this progression, fresh frozen plasma transfusion was performed, in addition to other blood products, and immediate transfer to a liver transplant referral hospital was arranged. However, due to hemodynamic instability, the patient died the day after transfer.

DISCUSSION

The *Brugmansia* plant family is native to tropical regions such as Brazil and is widely cultivated in gardens for its stunning beauty. One way of consuming it is through “trombone tea,” which can be made from its leaves, flowers, or seeds⁵.

The tea contains a high concentration of atropine and scopolamine, which can cause anticholinergic poisoning and have a high potential for chemical dependence. The degree of toxicity depends on the part of the plant used, the season, and the stage of maturity. All parts of the plant can be toxic, especially the seeds⁶.

The typical clinical presentation includes increased heart and respiratory rate, hypertension, elevated temperature, mydriasis, xerostomia, seizures, and in severe cases, coma, and death⁷. Symptoms develop rapidly, about 5-10 minutes after ingestion, and are due to the anticholinergic effects of atropine and scopolamine. Each flower of the plant contains approximately 0.3 mg of atropine and 0.65 mg of scopolamine. The ingestion of just 10 flowers for tea preparation has been reported as being associated with fatal cases⁵.

After recreational use of the tea, which was later reported by a family member, our patient presented with generalized tonic-clonic seizures and fever, in addition to being admitted with mydriatic pupils. Mydriasis is described as one of the main findings of intoxication.⁸ In

a study by Isbister *et al.* (2003)⁹, between July 1990 and June 2000, 33 patients intoxicated by different plants were included, of whom 31 ingested “angel’s trumpet,” and 100% of these had mydriasis with an average duration of 29 hours, in addition to 88% presenting *delirium* with an average duration of 18 hours.

Doan *et al.* (2019)², in a retrospective study of case series reported to the Taiwan Poison Control Center

between 1986 and 2015 (203 cases of *Datura* exposure and 114 cases of *Brugmansia* exposure), found that the most frequently observed clinical effect was mydriasis (53.2%), followed by confusion (40%), tachycardia (35.5%), xerostomia (35.5%), vertigo (34%), xeroderma (32.5%), and delirium (31%). In addition to visual disturbances, neurological manifestations such as seizures, stroke¹⁰, and Guillain-Barré syndrome^{11,12} are very common.

Table 1 - Laboratory evolution during hospitalization period

	04/05/2022	05/05/2022	06/05/2022
Hemoglobin (g/dL)	14,6	13,3	10,8
Hematocrit (%)	44,4	41,3	32,4
Leukocyte count (Cell/mm ³)	10000	14700	13800
Bands (%)	-	25	10
Platelets (Células/mm ³)	89000	37000	28000
CRP (mg/dL)	-	1,2	1,3
Urea (mg/dL)	48	119	26
Creatinine (mg/dL)	3,0	5,9	2,2
TGO/AST (mg/dL)	496	1675	4437
TGP/ALT (mg/dL)	75	887	3253
ALP (mg/dL)	-	60	-
GGT (mg/dL)	-	61	59
Total proteins (g/L)	6,3	-	4,2
Albumin (g/L)	4,0	3,2	2,7
Total bilirubin (mg/dL)	1,87	3,1	6,15
Direct bilirubin (mg/dL)	-	1,09	1,9
Indirect bilirubin (mg/dL)	-	2,01	4,25
INR	2,05	6,6	5,01
TAP (s)	21,8	72	54,4
PTT (s)	45,3	81,7	57,8
LDH (U/L)	1039	2389	4446
CPK (U/L)	2818	-	12793
Chlorine (mEq/dL)	92	-	-
Sodium (mEq/dL)	143	152	137
Potassium (mEq/dL)	3,2	6,2	4,8
Phosphorus (mEq/dL)	-	13,8	5,6
Magnesium (mEq/dL)	-	2,0	1,7
Calcium (mEq/dL)	-	5,6	-

Legend: CRP: C-reactive protein; TGO/AST: aspartate transaminase/oxaloacetic transaminase; TGP/ALT: alanine transaminase/pyruvate transaminase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase; INR: international normalized ratio; TAP: prothrombin time; PTT: activated partial thromboplastin time; LDH: lactate dehydrogenase; CPK: creatine phosphokinase.

Treatment for severe cases involves intravenous administration of physostigmine (2 mg), an acetylcholinesterase inhibitor. Within less than 48 hours of consumption, gastric lavage with activated charcoal may also be performed⁶, with a dose of 1 to 2 g/kg diluted in 100 to 250 ml of saline or water. However, the effectiveness of this method is extremely reduced after 6 hours of ingestion. For cases of seizures, the use of benzodiazepines is indicated¹³.

The patient in our case report presented with seizures, mydriasis, and neck stiffness. The initial

diagnostic hypothesis was meningitis and a lumbar puncture was performed, and empiric antibiotic therapy was initiated. However, he did not improve and rapidly progressed to renal and hepatic failure. We believe that he must have ingested high doses of “angel’s trumpet” to present with acute and multiple organ dysfunction.

Acute hepatic failure is a rare but highly fatal condition characterized by sudden onset of coagulopathy, jaundice, and hepatic encephalopathy, associated with laboratory evidence of hepatocellular injury. The latter is characterized by an increase of 2-3 times the reference

value of transaminases (AST and ALT). The classification into acute or chronic is defined according to the time elapsed between the onset of symptoms, usually jaundice, and the development of encephalopathy. Cases where this progression occurs in up to 8 weeks, are classified as fulminant, while those with more than 8 weeks are called chronic. The *International Association for the Study of the Liver* (IASL) uses the subdivision into hyperacute (progression in up to 10 days), fulminant (between 10 and 30 days), and subacute/subfulminant (between 5 and 24 weeks)¹⁴.

The patient in our case presented marked elevation of transaminases, demonstrating liver injury. Changes in coagulation parameters - reflected in the observed increase in INR, TAP, and PTT - and increased total bilirubin levels are associated with confirming organ dysfunction. The presence of significant neurological alteration and the time until its development characterizes the condition as a fulminant hepatic failure.

Classically, according to the IASL classification, hyperacute hepatitis presents with severe coagulopathy, a marked increase in transaminases, and a moderate increase in bilirubin. Subfulminant hepatitis often has a less intense increase in transaminases, deep jaundice, and mild to moderate coagulopathy.¹⁴ Based on the progression of symptoms and laboratory tests, our patient had a picture of hyperacute hepatitis, with the rapid development of neurological alteration, a significant increase in transaminases, and alteration of coagulation parameters, but with a moderate increase in total bilirubin.

The most common causes of fulminant hepatic failure include viral and drug-induced hepatitis. Poisonings resulting from the consumption of plants and derived

compounds are also described as a cause of fulminant hepatitis. Early renal replacement therapy and therapeutic plasmapheresis play an important role in its treatment^{14,15,16}. Liver transplantation is the definitive therapy necessary in some cases where clinical support treatment is not enough¹⁴. The patient, in our case, was referred to a specialized liver transplant unit but died before the procedure was performed.

In the presented clinical case, liver dysfunction may have occurred due to multiple factors, such as an ischemic injury due to hemodynamic shock and the administration of drugs with hepatotoxic potential. To date, there are no other reports in the literature of fulminant hepatic failure associated with poisoning by the trumpet tea plant. However, the rapid progression, in a few days of hospitalization in a previously healthy individual, and the history of ingestion of high doses of "angel's trumpet" strongly suggest that it may have been a manifestation resulting from the consumption of the plant.

CONCLUSION

The poisoning by "trumpet tea" is a neurological emergency that must be quickly recognized due to its potentially fatal effects. As meningitis is a differential diagnosis, it is important to gather a complete clinical history to investigate a possible previous contact with the plant. To our knowledge, this is the first case reporting fulminant hepatitis secondary to the ingestion of "angel's trumpet," in addition to the neurological symptoms already described in the literature, which further reinforces the importance of the topic at hand.

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