Accidental ingestion of tetrahydrocannabinol by an adult who was not a regular user

Ingesta accidental de Tetrahidro-cannabinol en un paciente adulto no consumidor habitual

To the Editor,

According to data from the European Monitoring Centre for Drugs and Drug Addiction, in 2020, 19% of Spaniards aged 15 to 64 had consumed cannabis in the past 12 months, a percentage that rose to 23% among adolescents aged 15 and 16.[1]

The main psychoactive component of cannabis is Δ-9-tetrahydrocannabinol (THC). Compounds structurally similar to THC are known as cannabinoids, which can be natural or synthetic. Their effects include altered consciousness, a sense of well-being, relaxation, and increased appetite, among others. The symptoms of intoxication depend on the amount consumed and the potency of the cannabinoids used, although there is considerable interindividual variability. The clinical picture may include nausea, dizziness, tachycardia, sensory-perceptual disturbances, anxiety attacks, and even psychotic episodes.[2]

A 36-year-old woman, with no toxic habits or relevant medical history, was brought to the emergency department by ambulance after a 2-hour episode of dizziness (unsteadiness), drowsiness, and nausea. She worked as a domestic cleaner and related the episode to the ingestion, about an hour prior to symptom onset, of some gummies she had found while cleaning a private residence. She brought the product's wrapper and reported having eaten almost the entire contents (Figure 1). Upon arrival, the patient's blood pressure was 93/58 mmHg, heart rate 98 bpm, afebrile, eupneic, and with a baseline oxygen saturation of 98%. Physical examination revealed conjunctival injection, psychomotor slowing, and unsteady gait.

Since the wrapper did not list ingredients, an internet search was performed, identifying a website describing a THC content of 300 mg. Urinalysis tested positive for cannabis and negative for other widely screened toxins in hospital emergency departments. Blood tests and ECG showed no abnormalities. The patient was observed for about six hours, with full resolution of symptoms, and was subsequently discharged.

This case represents THC intoxication following involuntary oral ingestion, a situation described mainly in children but exceptional in adults[3]. In recent years, the number of emergency de-

Figure 1. Packaging of the gummies consumed, provided by the patient.

Authors:
Ruth Benito Martínez
Mariona Condom Siñol
Daniel Sánchez Ruiz

Author Affiliations:
Medicina Familiar y Comunitaria. Unidad Docente ACEBA. Hospital de la Santa Creu i Sant Pau. Universitat Autónoma de Barcelona. Barcelona, Spain.

E-mail:
RBenitom@santpau.cat

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partment visits related to cannabis intoxication has increased and is present in 44% of cases involving intoxicated patients[4]. The reasons for this increase have been linked to several factors, such as the rise in consumption and the greater potency of current cannabis strains.[5]

Various forms of synthetic cannabinoid products exist—mainly incense and liquid preparations for vaping—which are mostly sold online as alternatives to cannabis, whose use and possession are illegal in Spain. Another distinguishing feature of this case was the presentation in gummy form, whose harmless appearance and common edible nature may facilitate accidental intoxication.

In this case, despite the positive result for THC, it cannot be ruled out that the gummies also contained synthetic cannabinoids, which are undetectable with conventional toxicological assays.[6,7] Therefore, such tests should be reserved for cases with potentially severe symptoms—of unclear etiology—or when clinical history does not explain the presentation.[7]

The use of synthetic cannabinoids may thus be underestimated in emergency hospital visits, due to the inability to detect them with standard toxicological laboratory tests.[8]

Moreover, clinicians should perform a comprehensive medical history given the diverse presentations and routes of consumption of natural and synthetic cannabinoid products.

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An unidentified patient in coma

Coma en paciente nomen nescio

Authors:
Daniel N. Marco, Sandra Cuerpo, Blanca Coll-Vinent.

Author Affiliations:
Área de Urgencias, Hospital Clínic, Barcelona, Spain.
Universitat de Barcelona, Spain.

E-mail:
dnmarco@clinic.cat

Ethical Responsibilities:
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To the Editor,

Comatose patients are sometimes a diagnostic challenge in emergency departments. Approaching their evaluation through a systematic method allows not only for diagnosis of the patient but also for identification of the underlying disease causing the coma.

An unidentified patient was found on the street with a low level of consciousness and brought to our center. On arrival, he had a temperature of 39°C, a Glasgow Coma Scale score of 8, and abundant secretions, prompting orotracheal intubation to protect the airway.

Other vital signs were normal. The pupils were isocoric and reactive to light. There was no neck stiffness. Cardiopulmonary auscultation and abdominal examination were normal. The patient presented spider angiomas, decreased body hair, and numerous epidermal excoriations.

Arterial blood gas analysis showed no hypercapnia or acidosis, and the electrocardiogram was normal except for a prolonged QT interval (520 ms).

Because of an initial suspicion of acute meningitis, cultures were obtained and empirical antibiotic therapy was started. Cranial CT was unremarkable, and a lumbar puncture was performed, yielding clear cerebrospinal fluid with normal opening pressure. CSF analysis also came out normal. Blood tests revealed leukocytosis without inflammatory markers or electrolyte disturbances, and mild liver enzyme elevation (GGT, 270 U/L; AST/ALT, 62/41 U/L). A toxicology screen showed positive methadone in urine only. However, it was upon reviewing the chest X-ray that the cause of the coma was discovered: a metal structure was observed superimposed on the image of a right rib, corresponding to a TIPS (Transjugular Intrahepatic Portosystemic Shunt) (Figure 1).

This finding, along with the described clinical features, led to the diagnosis

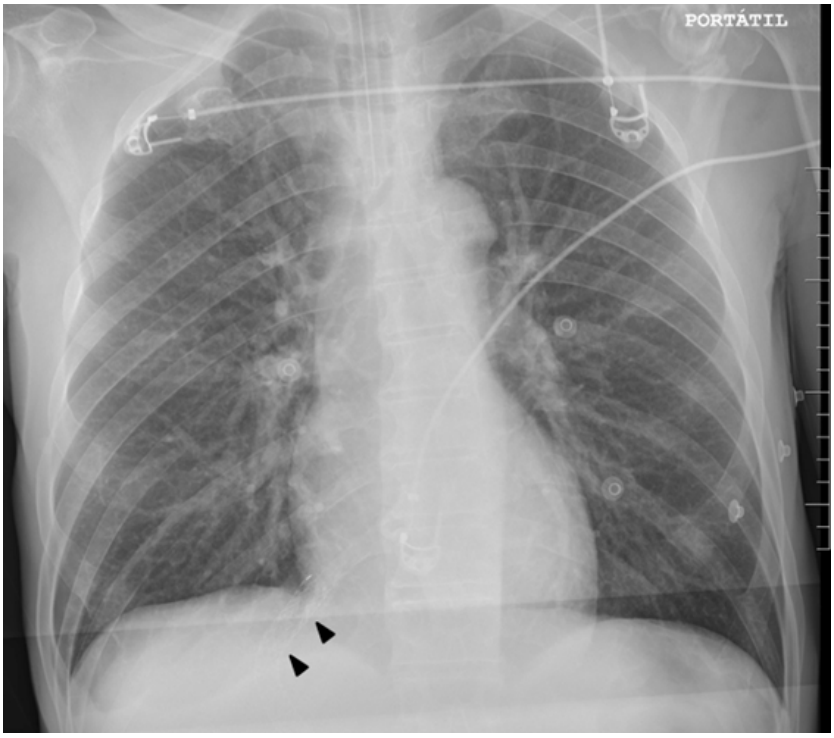

Figure 1. Portable chest X-ray on the patient's arrival to the emergency department. The shadow of the TIPS can be observed overlapping the image of a right rib (arrowheads).

of grade 4 hepatic encephalopathy, and specific treatment was initiated with antibiotics and enemas. Serum ammonia levels were elevated, and viral serologies came out positive for HCV and HIV. With these findings, along with the presence of a TIPS and an estimated age range, the patient was successfully identified. It was determined that he was on chronic methadone therapy, which explained the prolonged QT interval. Later, *S. pyogenes* was isolated in blood cultures. After 24 hours, the patient was extubated in the intensive care unit without complications.

It is common for comatose patients to arrive at emergency departments without identification (NN, *nomen nescio*). A high index of suspicion and careful physical examination are crucial in these cases when anamnesis is not possible. In this case, faced with a coma and high fever, a differential diagnosis was considered including infectious, toxicologic, metabolic, and neurological causes. After ruling out acute meningitis, reevaluation of the physical findings and liver enzyme abnormalities suggested hepatic encephalopathy as the cause of the coma, as well as a probable cutaneous infectious focus. Nevertheless, a systematic review of the chest X-ray provided the diagnostic key—a test commonly performed when fever is present, to rule out pneumonic processes, as initially suspected in this case.[3]

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Eritema migrans: Not every rash in the emergency department is hives

Eritema migrans. No todo es urticaria en urgencias

To the Editor,

Lyme disease is a zoonosis whose clinical diagnosis in the acute phase remains well below its true prevalence.[1] Cutaneous signs are common and easily accessible for clinical assessment. Delayed diagnosis carries a risk of chronicity and complications.

We present the case of a 54-year-old woman with a past medical history of systemic lupus erythematosus on methotrexate and hydroxychloroquine. She presented to the emergency department with a 24-hour history of pruritic skin lesions, which began on the left arm and progressively generalized. The previous week, she reported a self-limited episode of fever, arthromyalgias, headache, and diarrhea, treated with paracetamol. She had traveled 10 days earlier to northern Spain, denied animal contact and recalled no tick bites. Physical examination revealed erythematous-edematous plaques approximately 3–4 cm in diameter scattered over the lower limbs, lighter lesions on the arms and abdomen, and on the right flank, a large oval erythematous-edematous plaque with a slightly violaceous central area and a poorly defined pale border about 1 cm wide.

At the center of the lesion, a reddish pseudovesicular area was noted, compatible with the inoculation site (Figure 1). Blood tests, serologic studies, and a skin biopsy were performed. With a probable diagnosis of Lyme disease, given the presence of typical erythema *migrans* (EM) lesions, doxycycline treatment was initiated. Serology revealed positive IgM titers for *Borrelia burgdorferi*. Histopathological analysis showed a superficial and deep lymphoplasmacytic dermatitis with periadnexal involvement, consistent with EM.

Lyme disease is caused by 3 species of *Borrelia burgdorferi sensu lato:* sensu stricto (in America), *garinii*, and *afzelii* (in Asia). In Europe, all 3 have been identified,[1] with ticks as the usual vectors. In Spain, the most frequent species is *B. garinii*.

Endemic areas include La Rioja, Navarre, northern Castile and León, Asturias, Cantabria, and the Basque Country. Transmission occurs through the tick *Ixodes ricinus* (sheep tick),[1,2] whose life cycle includes larval, nymphal, and adult stages. Nymphal bites are the main cause of infection during late spring and summer. Due to their small size, most patients do not recall being bitten. After an incubation period of 3–32 days, the spirochete multiplies locally in the skin, giving rise to EM, the most common sign of the disease, and considered pathognomonic in endemic regions.[3,4] Other signs suggesting hematogenous dissemination include flu-like illness, lymphadenopathy, meningism, myocarditis, and the presence of multiple EM-like skin lesions (secondary EM), as occurred in this case—this form of presentation being more common in the United States.[1] Chronic signs may include arthritis, neuro-

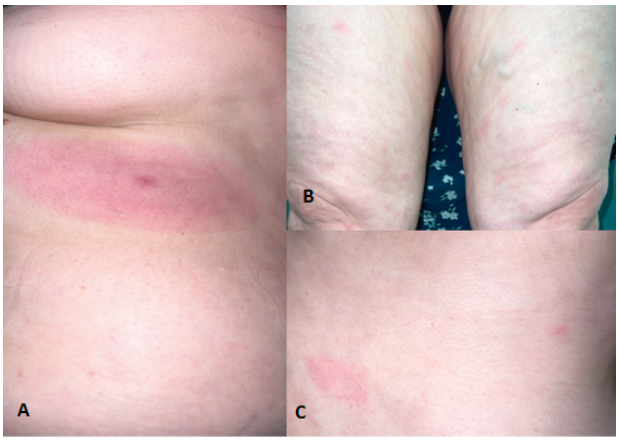

Figure 1. Erythema migrans. A. Right flank: annular erythema with a poorly defined peripheral erythematous border; a reddish area and a central pseudovesicle are observed in the center. B. Proximal half of the lower limbs: scattered, faint, poorly defined erythematous-edematous plaques. C. Left flank and abdomen: 2 erythematous-edematous plaques similar to those previously described.

Authors:
Gladis Canllavi García[1], Sonsoles Berenguer-Ruiz[2], Mar Llamas-Velasco[2], Juan Mariano Aguilar Mulet[3].

Author Affiliations:
[1]Servicio de Medicina Física y Rehabilitación, Hospital Universitario de la Princesa, Madrid, Spain.
[2]Servicio de Dermatología, Hospital Universitario de la Princesa, Madrid, Spain.
[3]Servicio de Urgencias, Hospital Universitario de la Princesa, Madrid, Spain.

E-mail:
aguilarmulet@gmail.com

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Guillermo Burillo Putze.

logical involvement (such as sensory polyneuropathy), acrodermatitis chronica atrophicans, and endocarditis. Diagnosis is primarily clinical, and the suspicion of EM alone is sufficient to initiate treatment,[5] although serologic confirmation or PCR detection of *Borrelia* from biopsy samples is recommended in atypical EM or in cases with multiple EM lesions. Histopathologically, there are no specific findings, but the most common pattern is that observed in this patient. The treatment of choice is oral doxycycline, although amoxicillin is a valid alternative.

Maintaining a high index of suspicion, especially during seasons of increased transmission, contributes to improved diagnosis and allows early treatment, preventing the development of chronic forms of the disease.

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Unexpected outcome for a professional housekeeper

Limpieza doméstica con desenlace inesperado

Authors:
Raquel Aranega,
Luana Da Costa Llácer,
Emilio Salgado.

Author Affiliations:
Hospital Clínic, Barcelona.
Universidad de Barcelona, Spain.

E-mail:
ARANEGA@clinic.cat

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To the Editor,

The ingestion of products stored in containers different from the original or without identification is a common cause of accidental poisoning in both domestic and occupational settings.[1]

A 34-year-old woman, with no relevant medical history and working as a domestic cleaner in several private homes, was performing household chores in a client's residence when she ingested an unspecified amount of a clear, odorless, and tasteless liquid from an unlabeled bottle. Approximately 30 minutes after ingestion, she developed general malaise manifested by abdominal discomfort, unsteady gait, dizziness, and drowsiness, prompting her to call a family member. About 30 minutes later, upon arriving at the home, the relative found the patient unconscious and unresponsive on the floor and alerted the Emergency Medical Service (EMS).

Upon EMS arrival, the patient was in a non-reactive coma (GCS = 3). She was administered 2 mg of IV naloxonewithout response, followed by 0.5 mg of IV flumazenil, also without improvement. Orotracheal intubation was performed, and she was transferred to the emergency department (ED).

Upon arrival at the ED, the patient was mechanically ventilated, with a RASS score of –5, without sedation. The blood test, including arterial blood gas, complete blood count, and basic biochemistry, showed no significant abnormalities. A cranial CT scan ruled out acute lesions, and toxicological testing for blood ethanol, ethylene glycol, and methylene glycol, as well as urine screening for amphetamines, benzodiazepines, cannabis, methadone, and opioids, yielded negative results.

During the first hours in the ED, the patient exhibited clonic movements of all 4 limbs, spontaneous eye opening, asynchrony with the ventilator, and no interaction with the environment, leading to the initiation of pharmacological sedation and subsequent admission to the intensive care unit (ICU) where an electroencephalogram (EEG) was performed, which showed no epileptiform activity, but rather global slowing, compatible with intoxication by an unknown central nervous system depressant.

During the first 12 hours of ICU stay, sedation was gradually reduced until withdrawal, allowing the patient to be extubated at 24 hours without immediate complications.

While in the ICU, contact was made with the owner of the residence, who reported that the ingested substance was very likely ketamine. Toxicological analysis of the urine sample obtained upon arrival at the ED, using gas chromatography and mass spectrometry

(GC/MS), confirmed the presence of ketamine and ruled out the presence of other substances.

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