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Psilocybe Poisoning: *Pathophysiology*, Classification and Treatment. A Clinical Case Review

Intoxicación por *Psilocybe*: Fisiopatología, Clasificación y Tratamiento. Revisión de caso clínico

Omar Azuara-Antonio[a], Erika Rubí De La Cruz-Elizalde[b], José Eduardo Carmona-Rodríguez[c] Lesly Idaliht Hernández-Martínez[d]

Abstract:

The *Psilocybe cubensis* mushroom is recognized as the primary source of psilocybin in the Americas, occurring naturally across various regions. This fungus has a long history of use in Mesoamerican rituals due to its capacity to induce altered states of consciousness. The defining characteristic of *Psilocybe* mushrooms is their psilocybin content. Following ingestion, psilocybin is metabolized into psilocin, which acts as a potent serotonergic agonist by interacting with serotonin receptors. The resulting physiological and psychoactive effects are linked to the activity at 5-HT receptors within the central nervous system, along with the release of glutamate. Throughout history, diverse Mesoamerican cultures incorporated hallucinogenic mushroom consumption into their ritual ceremonies. The Aztecs, for example, revered them as *Teonanácatl*, or " flesh of the gods," valuing their ability to shift the perception of reality. Interest in psilocybin has seen a resurgence in the scientific community, spanning from the ethnobotanical studies of R. Gordon Wasson in the 1950s to contemporary research into its therapeutic applications for depression. Studies have indicated that psilocybin can sustainably alleviate depressive symptoms, often with fewer side effects compared to conventional pharmacological treatments. The combination of the ancient ceremonial and religious use of *Psilocybe* mushrooms with their demonstrable therapeutic potential is prompting a reevaluation of their legal status as a Schedule I drug. Ongoing research is actively exploring the impact of psilocybin on various psychiatric disorders, yielding promising results, particularly in the treatment of major depressive disorder. As the evidence supporting its therapeutic benefits continues to accumulate, it suggests a future where these psychedelic compounds could play a vital role in global mental health.

Keywords:

Psilocybe, mushrooms, poisoning, pathophysiology, treatment

Resumen:

El hongo *Psilocybe cubensis* es la principal fuente de psilocina en América, presente naturalmente en varias regiones. Se ha utilizado tradicionalmente en rituales mesoamericanos por su capacidad para inducir estados alterados de consciencia. Los hongos *Psilocybe* se caracterizan por contener psilocibina, que se convierte en psilocina una vez ingerida, actuando como un agonista serotoninérgico y afectando los receptores de serotonina. Los efectos fisiológicos y psicóticos se deben a la actividad de los receptores 5-HT en el sistema nervioso central, así como a la liberación de glutamato. El consumo de hongos alucinógenos en ceremonias rituales se ha practicado en diversas culturas mesoamericanas a lo largo de la historia. Los aztecas los llamaban Teonanácatl y los consideraban sagrados por su capacidad de alterar la percepción de la realidad. Desde los estudios de etnobotánicos como Robert Wasson en la década de los 50 hasta las investigaciones más recientes sobre sus propiedades terapéuticas para tratar la depresión, la psilocibina ha despertado un renovado interés en la comunidad científica. Se ha demostrado que la psilocibina puede reducir los síntomas de la depresión de manera sostenida y con menos efectos secundarios que los tratamientos farmacológicos convencionales. El uso milenario de los hongos *Psilocybe* en contextos ceremoniales y religiosos, así como su potencial terapéutico actual, ha contribuido a reevaluar su estatus legal como droga tipo 1. Las investigaciones en curso están explorando el impacto de la psilocibina en el tratamiento de trastornos psiquiátricos y se han obtenido resultados prometedores en el tratamiento de la depresión mayor. A medida que se acumula evidencia sobre los beneficios terapéuticos de la psilocibina, se vislumbra un futuro en el que estos compuestos psicodélicos puedan jugar un papel crucial en la salud mental de la población mundial.

***Palabras Clave**:*

[a] Universidad Autónoma del Estado de Hidalgo, https://orcid.org/0000-0002-8648-4573, Email: azuara3000@icloud.com

[b] Instituto de Ciencias y Estudios Superiores de Tamaulipas, https://orcid.org/0009-0009-5562-9213, Email: eriicka_rubi@hotmail.com

[c] Universidad Autónoma del Estado de Hidalgo, https://orcid.org/0009-0002-9952-3356, Email: eduardo.crmed@outlook.es

[d] Universidad Autónoma del Estado de Hidalgo, https://orcid.org/0009-0002-2027-9574, Email: Idalihtmartinez@gmail.com

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Psilocybe, hongos, intoxicación, fisiopatología, tratamiento.

INTRODUCTION

Fungi are eukaryotic organisms within the kingdom Fungi, characterized by organized nuclei and well-defined nuclear membranes. The hallucinogenic fungi containing the psychotropic alkaloid psilocybin belong to the order Agaricales, which includes the genus *Psilocybe*, as well as the genera *Panaeolus, Conocybe, Inocybe, Gymnopilus, Lycoperdon* and *Pluteus*. These mushrooms contain psilocybin and psilocin. Species within the genus *Psilocybe* are saprophytes found in wood, dung, moss, soil, and leaf litter. The mushroom *Psilocybe cubensis* is the most common source of psilocin in the warmer regions of the United States; it also occurs naturally in Mexico, Central America, and South America. Furthermore, it is the most frequently cultivated species for recreational use.[1-3]

Hallucinogens are substances that, when ingested in non-toxic doses, can cause altered states of consciousness and induce non-existent perceptions or environmental distortions. Throughout history, numerous societies have isolated substances with hallucinogenic properties from fungi, plants, and animals. From an ethnobotanical and anthropological perspective, the American continent is a privileged region due to the vast diversity of naturally occurring hallucinogens.[4]

The various civilizations that settled in Mesoamerica possessed extensive knowledge and precise handling of numerous hallucinogens. Archeological, ethnohistorical, and ethnographic evidence confirms that, throughout history, pre-Columbian Mesoamerican cultures utilized hallucinogenic substances for magical, therapeutic, and religious purposes.[5,6] They are known by the term "entheogens," as they were used to stimulate mysticism and communication with the divine. The objective was to achieve a state of trance, heightened enlightenment, and openness of mind. This desired altered state of consciousness was characterized by spatio-temporal disorientation, a sense of ecstasy and inner peace, vivid hallucinations, a tendency toward introspection, and a feeling of union with nature and divinities.[7]

The primary entheogenic mushrooms belong to the genera *Psilocybe*, *Panaeolus,* and *Stropharia*. There are approximately 230 species within the genus *Psilocybe,* of which at least 54 are found in Mexico and were utilized as hallucinogens by pre-Columbian Mesoamerican cultures. Notable species include *P. semilanceata*, *P. mexicana*, *P. aztecorum*, *P. cubensis* and *P. caerulescens*. These are small mushrooms, ranging from 2.5 to 10 cm in height, with a slender, long, fibrous stem and a cap measuring 1 to 3 cm. The active ingredient present in *Psilocybe* spp. is an indole-alkylamine, 0-phosphoryl-4 hydroxy-N,N dimethyltryptamine or psilocybin. Following ingestion, it undergoes a dephosphorylation process and is converted into psilocin (4-hydroxy-N,Ndimethyltryptamine), which is a more potent hallucinogen. Psilocybin is absorbed from both the fresh, unboiled mushroom and the dried, powdered form. [8]

Psilocybin is metabolized via dephosphorylation into psilocin, transforming it into its active metabolite. Psilocin possesses a structure similar to serotonin, enabling it to bind and activate serotonin receptors as a serotonergic agonist. Due to its relatively lipophilic nature, psilocin effectively crosses the blood-brain barrier, with its effects typically lasting between 4 and 6 hours.[1,2] Both physiological and psychoactive effects result from agonist activity at serotonin (5-HT) receptors within the central nervous system, primarily through the activation of the 5-HT_{2A} receptor. Furthermore, evidence suggests that psilocin additionally increases glutamate release, which in turn activates the excitatory N-methyl-D-aspartate (NMDA) and α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) receptors. It has been determined that these effects may also be partially mediated by dopamine through indirect pathways. Based on pharmacochemical structure-activity relationship (SAR) studies, it has been established that psilocin binds with high affinity to the 5-HT_{2A} (K_i=6 *nM*) and 5-HT_{2C} (K_i=10 nM) receptors, and with lower affinity to the 5-HT_{1A} receptor (K_i=190 nM).[1,2,9]

Table 1. *Dose-dependent effects of psilocybin[10]*

Dose	General Effects	Perceptual Changes	Psychic Effects
Low (0.1 mg)	Slight	Visual distortions	Mood changes
Medium (0.2 mg)	More pronounced	Bodily distortions accompanied by agitation and hallucinations	Combination of illogical events and actions (primary process thinking)
High (0.3 mg)	Maximum	Disrupted motor activity. Complex hallucinations	Distortion of the "Self" (the Ego) and the environment

Dosage ranges are referential. Individual sensitivity, the specific mushroom species, and the context of use (set and setting) can significantly modify clinical effects.

Psilocybin concentrations vary across mushroom species; however, doses exceeding 5 mg can induce hallucinogenic effects. Approximately thirty minutes after ingesting, a clinical presentation emerges, characterized by euphoria, depersonalization, visual field distortions, and deep introspection, often accompanied by a tendency toward isolation from the surrounding environment. The duration of the oneiric symptoms typically ranges from 4 to 6 hours. Subjects often present with cutaneous and facial flushing, diaphoresis, tachycardia, and elevated blood pressure. Furthermore, high doses (20-30 mg) may induce cholinergic-like symptoms,

including xerostomia, urinary retention, and intensified hallucinations. Approximately 8 hours post-consumption, the subject typically returns to a baseline state. Occasionally, effects such as headache, fatigue, and a lingering sense of well-being may persist for a few days (Table 1).[8,10] Psilocybin is classified under Schedule I of the 1971 Convention on Psychotropic Substances and is, therefore, considered a controlled and banned substance.

The consumption of hallucinogenic mushrooms in ritual ceremonies was widespread in Mesoamerican cultures. The cult of sacred mushrooms spread from the Valley of Mexico throughout Central America and is estimated to be at least 3,500 years old. The Maya consumed "*kaizalaj okox*" (*Psilocybe cubensis*), while the Aztecs called them "*teonanácatl*". They were also consumed by the Huastec, Totonac, Mazatec, and Mixtec peoples. In Teotenango, the practice of grinding mushrooms with water −dating back to prehistoric times− was performed on stone models of temples on rocks featuring petroglyphs. Furthermore, archaeological evidence of their use exists across Mexico, Guatemala, Honduras, and El Salvador, where "mushroom stones" (lithic artifacts representing hallucinogenic fungi) have been discovered. In Kaminaljuyú, Guatemala, nine mushroom stones were discovered; their stems featured anthropomorphic figures depicting the pre-Hispanic practice of grinding sacred mushrooms into powder. Furthermore, the Mixtec deity *Siete Flores* (Seven Flowers) was traditionally represented holding a pair of mushrooms in his hands. The sculpture of *Xochipilli*, the Aztec god of flowers, discovered in the 16th century on the slopes of the Popocatepetl volcano, depicts various medicinal and hallucinogenic plants, including *Psilocybe aztecorum*, whose habitat is endemic to that region. Other psychoactive plants carved into the sculpture include *Nicotiana tabacum*, *Heimia salicifolia* (*sinicuichi*), *Turbina corymbosa* (*ololiuhqui*), and *Psilocybe* spp. The Tepantitla mural in Teotihuacan, dating back to 500 AD, is also highly illustrative; it depicts the rain deity *Tlaloc* and, beneath his falling water droplets, priestly figures emerging alongside hallucinogenic mushrooms.

In the Mayan Dresden and Madrid codices, mushrooms appear in scenes depicting human sacrifices. Psychoactive mushrooms were also utilized in the coronation ceremonies of several Aztec emperors, including Tizoc, Ahuizotl and Moctezuma II. Furthermore, several 16th-century chroniclers−such as Durán, Sahagún, and Motolinía−recorded the use of sacred mushrooms by the Aztecs in their religious ceremonies. In his *Historia de las Cosas de Nueva España*, Fray Bernardino de Sahagún, described the uses and properties of these hallucinogenic mushrooms as follows: "There are mushrooms in this land that are called *teonanácatl*. They grow under the hay in the fields or moors. They are round and have a thin, round stem, a foot high. Eaten they have a bad taste; they hurt the throat and make you drunk. They are medicinal against fever and gout. They should be eaten two or three at most. Those who eat them see visions and feel heart vessels, and see visions at times frightening and at times of laughter. Those who eat many of them provoke lust, even if they are few. And to the crazy and mischievous, tell them that they have eaten *nanácatl*".[8]

The historical use of *Psilocybe* mushrooms has been so significant that it has even been suggested that they may have acted as evolutionary catalysts for certain human cognitive functions.[11,12] Likewise, it has been posited that the hypothetical consumption of these mushrooms by hominid ancestors, dating back to the Pliocene, could have enhanced social interaction, group cohesion, and the emergence of collective rituals.[13] What has been widely documented is the millennial use of *Psilocybe* to induce altered states of consciousness within religious and ceremonial contexts by pre-Columbian civilizations, such as the Aztec, Olmec, Zapotec and Maya.[8,14]

For the Aztecs, for example, these mushrooms represented *teonanácatl* ('the flesh of the gods') and were considered sacred beings capable of altering the perception of reality.[8] Such a "magical" conception of mushrooms is not relegated solely to those historical periods of history; on the contrary, it has become even more pronounced over time. In the 1950s, R. Gordon Wasson, one of the pioneers of ethnobotany, popularized the psychotropic potential of mushrooms in the West by sharing a series of testimonies regarding the healing rituals performed by the Mazatec curandera María Sabina with *los niños santos* (the holy children)−the name she gave to *Psilocybe*.[15] Subsequently, in the early 1960s, the chemist Albert Hofmann isolated psilocybin and its psychedelic derivative, psilocin; consequently, their use spread to clinical and scientific fields, giving rise to numerous publications on its therapeutic potential.[16,17] However, the subsequent excessive recreational consumption of these mushrooms and the resulting legal restrictions on their use hindered the continuity of research regarding their benefits.[18] Consequently, since the 1970s, psilocybin and psilocin have remained categorized as Schedule I drugs according to the Convention on Psychotropic Substances.[19] However, the evidence gathered decades earlier served as the foundation for a renewed path of scientific inquiry that resurfaced in the late 1990s. At that time, isolated research began to receive endorsement in countries such as the United States and, more recently, across several academic centers worldwide. [17] The resurgent interest in the therapeutic properties of psilocybin and other classic psychedelics has led to a new wave of publications regarding the potential benefits in the treatment of various psychiatric disorders.[20]

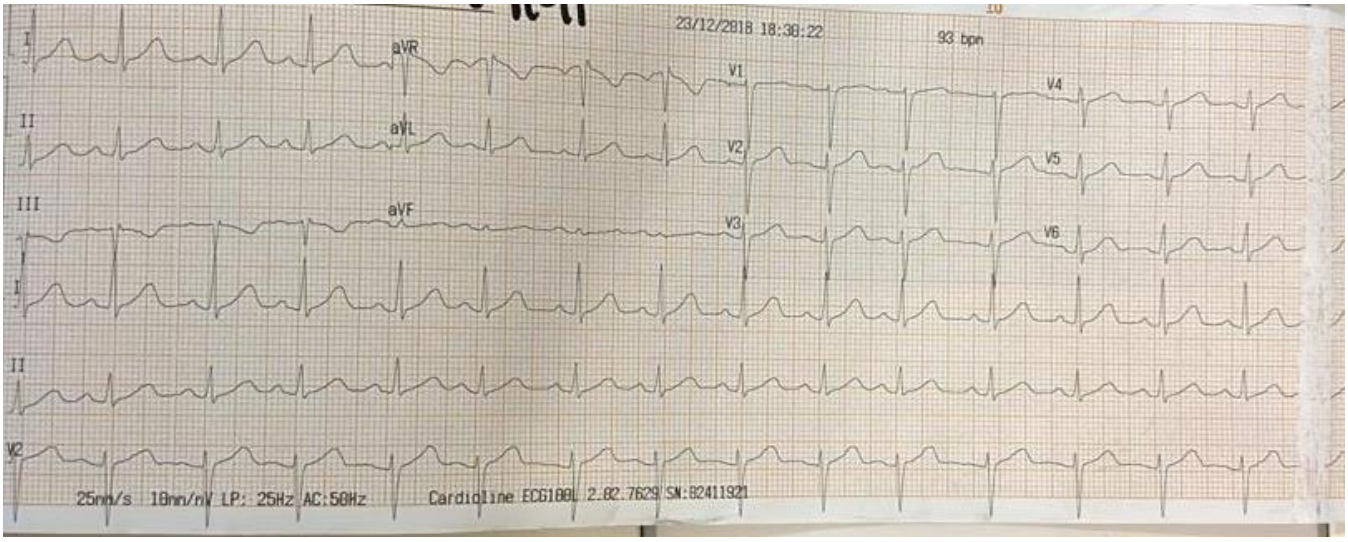

Figure 1. Electrocardiogram on admission

In this context, research has focused on the role of this alkaloid in treating major depressive disorder (MDD), a highly disabling condition that affects approximately 5% of the world's population.[21] Thus far, promising results indicate that psilocybin may not only sustainably reduce depressive symptoms but also do so within a shorter timeframe and with fewer side effects compared to conventional pharmacological therapies.[22-24]

CLINICAL CASE

We present the case of a 52-year-old female patient with a significant medical history of hypotension diagnosed at approximately 30 years of age and without established treatment. Symptoms began on June 15, 2023, after the ingestion of eight capsules of a *Psilocybe*-based dietary supplement. The patient reported dizziness, anxiety, visual hallucinations, abdominal pain, headache, and generalized pruritus. Additionally, she experienced tonic-clonic seizures for the first time, which lasted approximately four minutes, notably without loss of consciousness. Prior to hospital admission, the patient was administered 0.9% saline solution, 20 mg of furosemide, 500 mg of hydrocortisone, and three ampoules of atropine at 30-minute intervals; however, her symptoms persisted, leading her to seek evaluation at the hospital.

Upon admission, the patient's vital signs were as follows: blood pressure 117/64 mmHg, heart rate 65 beats per minute, respiratory rate 18 breaths per minute, temperature 35.6°C, oxygen saturation 97%, and a weight of 54 kg. Physical examination revealed the patient to be neurologically oriented with a Glasgow Coma Scale (GCS) score of 15 (O4, V5, M6). Further assessment showed symmetrical eyes with isochoric and normoreactive pupils, patent nares, and hydrated oral mucosa. The neck was short and cylindrical. The thorax was symmetrical with adequate expansion and respiratory excursion; heart sounds were rhythmic. Extremities were symmetrical with preserved motor strength and sensation, and capillary refill was immediate.

The initial management plan included fasting, intravenous crystalloid solutions for eight hours, potassium replacement, antiemetics, magnesium sulfate, and benzodiazepines. Additionally, activated charcoal was prescribed at a dose of 1 g/kg in 350 cc, to be administered orally over a one-hour period. In addition to pharmacological management, paraclinical and imaging studies were obtained (Figures 1 and 2; Table 2). Monitoring included cardiac monitoring, pulse oximetry, capillary glucose checks per shift, abdominal girth measurements, strict fluid intake and output quantification, and continuous neurological surveillance.

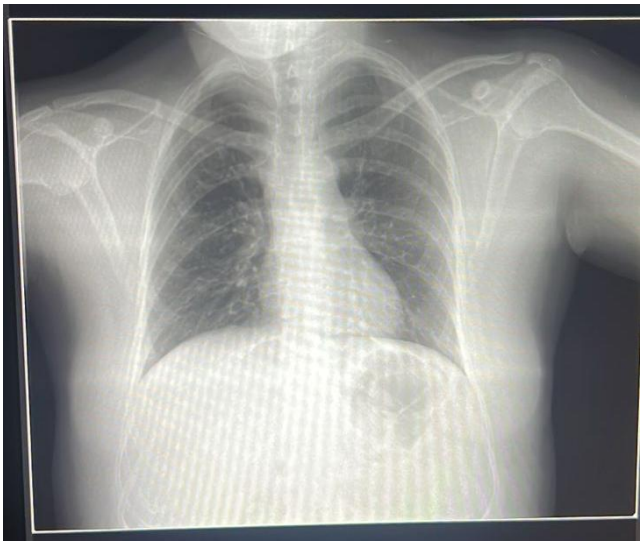

Figure 2. Anteroposterior (AP) chest X-ray upon admission. No acute cardiopulmonary findings are evident.

Table 2. Arterial blood gas (ABG) analysis upon admission

pH	PCO	PO2	Gluc	Lacta	HCO

	2 (mm Hg)	(mm Hg)	ose(mg/d L)	te (mm ol/L)	3 (mEq /L)
7.47	30	67	97	1.1	24.1

Sample obtained upon hospital admission. The findings are consistent with mild respiratory alkalosis (elevated pH, low pCO2) with adequate oxygenation. This is a frequent clinical finding in patients presenting with anxiety or psychomotor agitation.

During the initial days of clinical management, paraclinical tests returned within normal limits; consequently, a diet as tolerated was initiated, and treatment was maintained solely with magnesium sulfate. Given the patient's continuous clinical improvement, follow-up laboratory tests were obtained (Table 3), including a Complete Blood Count (CBC), a Comprehensive Metabolic Panel (CMP) with serum electrolytes (SE), and Liver Function Tests (LFTs). Following the confirmation of normal results, the patient progressed to a normal diet with oral liquids as tolerated. Since she remained clinically stable with vital signs within normal ranges, hospital discharge was authorized with a follow-up appointment scheduled in five days.

Table 3. *Laboratory results of the patient upon clinical improvement*

CBC	CMP	SE	LF	Other
Hb: 15.1	Glucose: 144.4	Na: 138	TB: 0.80	
HCT: 44.2	Urea: 24.5	K: 3.5	DB: 0.03	
WBC (Leu): 5.40	BUN: 11.4	Cl: 108	IB: 0.77	
Neu: 75.7	Uric Acid: 4.8	Ca: 10.1	AST: 30.1	Serum ethyl alcohol <10 mg/dL
Lymp:19.4	Trig: 55.2	P: 4.0	ALT: 22.3	
PLT: 265	Cholesterol: 214	Mg: 1.9	ALP: 91.6	
			LDH: 187	

Key: CBC: Complete Blood Count; CMP: Comprehensive Metabolic Panel; SE: Serum Electrolytes; LFTs: Liver Function Tests; Hb: Hemoglobin; HCT: Hematocrit; WBC (Leu): White Blood Cell count (Leukocytes); Neu: Neutrophils; Lymp: Lymphocytes; PLT: Platelets; BUN: Blood Urea Nitrogen; TB: Total Bilirubin; DB: Direct Bilirubin; IB: Indirect Bilirubin; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; ALP: Alkaline Phosphatase; LDH: Lactate Dehydrogenase.

DISCUSSION

POISONING SYNDROMES

Severe mushroom poisoning typically presents with clinical manifestations after an incubation period of more than six hours. Since the clinical presentation can mimic other illnesses, an accurate medical history −specifically inquiring about mushroom consumption in recent days− is vital for differential diagnosis.[25] The following classification is used for clinical assessment (Table 4).

Table 4. *Classification of Mushroom Poisoning Syndromes* .[25]

Group I: Primary Hepatotoxicity
Group II: Delayed Primary Nephrotoxicity
Group III: GABA Blockers
Group IV: Neurotoxic Hallucinogens.
Group V: Autonomic Nervous System Neurotoxins
Group VI: Central Nervous System (CNS) Neurotoxins
Group VII: Neurotoxins
Group VIII: Disulfiram-like Effect
Group IX: Hyperprocalcitonemia

CNS: Central Nervous System; GABA: Gamma-Aminobutyric Acid

Group I: Primary Hepatotoxicity

This group presents with initial gastrointestinal symptoms, which may be accompanied by tachycardia, hematuria or fever. This phase is followed by a period of apparent improvement, known as the latent period. The third stage involves hepatorenal failure[26], often associated with Central Nervous System (CNS) damage, leading to disorientation, confusion, somnolence, vertigo, convulsions, or coma.[27] Impaired liver function is evidenced by alterations in liver enzymes, such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), and bilirubin levels.[28] If jaundice develops following an acute gastrointestinal episode, amatoxin consumption should be suspected.[29,30] In rare cases, cardiogenic shock may occur. This phalloid or cyclopeptide syndrome is caused by *Amanita phalloides*, *Lepiota* spp., *Galerina* spp., *Amanita verna,* and the recently described *A. virosa*.[31]

This approach integrates clinical strategies aimed at palliating symptoms, considering absorption and transport routes to minimize gastrointestinal distress. For all species, a common symptomatic and supportive treatment is initiated, complemented by specific pharmacological agents or specialized techniques as needed. Initially, aggressive rehydration is required to restore fluid and electrolyte balance. Furthermore, hemodialysis may be considered during the latency period in specific cases of severe poisoning. Another option proposed for a time window of less than one hour post-ingestion is the administration of activated charcoal at a dose of 0.5 to 1g/kg body weight. Other evidence-based alternatives include nasogastric aspiration, induced diuresis, or gastric lavage. However, these therapeutic strategies are contraindicated in patients with an absent gag reflex, impaired consciousness (coma), or a high risk of gastrointestinal hemorrhage. Thirdly, pharmacological therapy with penicillin

G, silibinin, and N-acetylcysteine has shown positive results, even in combination. Silibinin stabilizes the hepatic membrane by blocking lysosomal proteases and preventing the entry of α-amanitin into hepatocytes[32]. Its administration within the first 48 h of ingestion reduces the likelihood of severe hepatic damage. Furthermore, silymarin has been observed to exert protective effects on cell membrane damage resulting from osmotic and oxidative stress, while also blocking the metabolism of toxins such as α-amanitin.[33] It can be combined with penicillin G;[32] Intravenous Vitamin K and fresh frozen plasma (FFP) are necessary, because hepatocellular damage causes impaired coagulation. These drugs can be used empirically without the need to confirm amatoxin poisoning; however, it is important to seek new alternatives. The use of thioctic acid remains controversial. Lactulose inhibits the uptake of neurotoxic metabolites such as ammonium.[34]

The management of liver failure requires extracorporeal support, such as the Molecular Adsorbent Recirculating System (MARS) or other albumin-based dialysis techniques. If there is no clinical response to these treatments, liver transplantation remains the last resort. [35,36]

Regarding toxicodynamics, three types of protoplasmic poisons have been identified: amanitins, virotoxins and phalloidins[37]:

Amanitins are the most toxic compounds of this group; they are chemically characterized as thermostable and cold-resistant bicyclic octapeptides. These toxins interfere with the bridge helix, thereby triggering the inhibition of the elongation process. It has been suggested that they can generate Reactive Oxygen Species (ROS), leading to oxidative stress, or even that there is synergistic activity between amatoxins and Tumor Necrosis Factor (TNF) that induces apoptosis. The primary target organ is the liver, where the toxins can cause an elevation of serum transaminases, which can progress to liver failure. These compounds undergo enterohepatic circulation, resulting in intestinal reabsorption that forces them to pass through the liver multiple times. Ultimately, they are eliminated almost entirely via the kidneys.[35] They act as inhibitors of protein synthesis and penetrate both hepatocytes and enterocytes, causing extensive cell necrosis. Some studies indicate that the concentration of α-amanitin is higher in the kidneys than in the liver; furthermore, these toxins are shown to cause Central Nervous System (CNS) damage.[37] However, several experimental studies in dogs and mice, along with published human case reports, appear to indicate the opposite: the amount of amatoxins eliminated in the bile is considerably higher than that eliminated in the urine.[38,39] Survival depends on the extent of hepatic destruction, the regenerative capacity of the liver cells, and the effectiveness in counteracting complications. The lethal dose of amatoxin in humans is approximately 0.1 mg of toxin per kg of body weight.[36]

Phalloidins exert their toxicity by binding to the F-actin filaments within cells, causing an interruption in the depolymerization process, which subsequently alters membrane functions. The actin polymer that binds to the *Amanita phalloides* peptide delays the release of inorganic phosphate produced by ATP hydrolysis.

Finally, virotoxins are monocyclic peptides with a structure similar to phallotoxins; however, they are chemically distinct due to their monocyclic nature and the presence of a sulfur atom in the form of a sulfoxide group. New data regarding these species are added annually; notably, while Amanita phalloides is responsible for 90% of fatal mushroom poisonings, virotoxins themselves appear to be the least toxic compounds within this group.[26]

There is ongoing controversy regarding the classification of these syndromes, as different authors propose grouping species into different toxic categories. It has been suggested that *Amanita phalloides*, all toxic *Lepiota* species, *Galerina* spp., and *Amanita verna* should be grouped within the same category. However, authors such as Yilmaz et al.[11], argue that *Amanita verna* is even more toxic than *A. phalloides* and is associated with a higher mortality rate. Consequently, within each classification, there may be varying degrees of symptomatic severity based on factors such as: (a) the species, (b) the time of collection, and (c) the portion of the mushroom that has been ingested. Regarding the latter, the highest concentration of amatoxins is found in the ring, gills, and cap, whereas the volva is richer in phallotoxins.[40] In their study, they argue that the concentrations of α-amanitin and β-amanitin are three times higher in this species, while γ-amanitin is found in lower proportions. Additionally, phalloidin −a specific type of phallotoxin− is also present in higher concentrations in *Amanita verna*. Furthermore, a rapid, simple, and sensitive method exists for detecting amatoxins in both mushroom fragments and urine samples.[41]

Group II: Delayed primary nephrotoxicity

This group presents with general symptoms such as fatigue, intense thirst, dry mouth and lips, a burning sensation on the tongue, loss of appetite, and headaches. These are accompanied by renal impairment that can be complicated by tubulointerstitial nephritis, which may progress to chronic renal failure and death.[35] *Cortinarius orellanus* causes orellanic syndrome −also known as orellanine or nephrotoxic syndrome− which can take several days to manifest. Between 2016 and 2020, seven cases were reported, likely due to confusion with edible species such as *Cantharellus cibarius* or, more frequently, *Chroogomphus* spp.[42] The recommended treatment involves monitoring urea and creatinine levels to correct electrolyte imbalances. In patients whose symptoms appear in less than a week, hemodialysis and plasmapheresis are often useful for removing toxins from the bloodstream. Furthermore, some studies indicate that hemofiltration is a much more effective treatment than hemoperfusion, and it is recommended in combination with conventional therapy.[32] In general, forcing diuresis seems advisable, although further studies are needed to reinforce this approach. On one hand, studies such as that by Palmier-Peláez[28] recommend intense hydration to prevent acute

renal failure; however, others advise against it, arguing that forced diuresis accentuate the accumulation of toxins. Meanwhile, studies in rats have shown that this nephrotoxic compound can be rapidly eliminated through urine or dialysis.[43] Regarding the toxin and its biological activity, it is known that the primary compound is orellanine, accompanied by cyclic decapeptides such as cortinarines A, B, and C. Orellanine is a bipyridyl protoplasmic poison with a marked tropism for the kidneys.[26] Some studies suggest that the cytotoxic effects may be attributed to the production of reactive oxygen species (ROS) and subsequent oxidative stress. When cyclopeptide orellanine oxidizes, it generates oxygen radicals; furthermore, it inhibits the synthesis of ribonucleic acid (RNA) and deoxyribonucleic acid (DNA), and promotes non-competitive inhibition of alkaline phosphatase activity. Its metabolites may also inhibit protein synthesis.[45]

Group III: GABA Blocker

This group presents with gastrointestinal and neurological effects, acute hemolysis, hypoglycemia, ataxia, liver damage, and cardiocirculatory collapse. It may even manifest as methemoglobinemia, convulsions, or coma, potentially accompanied by fever, stupor, or agitation. This is the gyromitrinic or gyromitrian syndrome, caused by *Gyromitra esculenta*.[46] *Gyromitra* species are frequently confused with species of the Helvellaceae family. Until now, treatment has consisted of monitoring symptoms and administering intravenous pyridoxine (vitamin B6), which antagonizes gyromitrin activity. Additionally, fluid resuscitation, electrolytes, and glucose replacement are administered. Gastric decontamination with activated charcoal is not recommended due to the long latency period. If seizures occur, diazepam or a complementary treatment including pyridoxine is recommended.[47,48] When methemoglobinemia occurs, the administration of methylene blue is recommended at a dose of 1 to 2 mg/kg administered intravenously over 5 minutes.[35] Gyromitrin is known to be a cytotoxic poison that is less dangerous than amatoxin. It is water-soluble and volatile; therefore, poisoning may occur if the mushrooms are not thoroughly cooked and subsequently dried. Furthermore, intoxication can occur through the inhalation of cooking vapors. In the stomach, gyromitrin is hydrolyzed, forming cytotoxic hydrazines that irritate the mucous membranes. The higher homologues of gyromitrin are MFH and MH (monomethylhydrazine), whose toxicity may also be carcinogenic.[2,26] In cases of mild poisoning, the incubation period is less than 4 hours, presenting with minor gastrointestinal or neurological symptoms. However, certain species can be fatal. It is crucial to determine whether the patient has previously self-medicated, as the intake of antiemetics or antidiarrheals can mask symptoms, thereby complicating the diagnosis. There may also be cases of mixed syndromes, therefore it is recommended that the patient remains under clinical observation for 18 to 20 hours.

Group IV: Neurotoxic hallucinogen.

This group presents with visual, auditory, and tactile illusions, altered perception of space and time, synesthesia, and euphoria. These manifestations may lead to anxiety, dilated pupils, vomiting, paresthesia, tachycardia, hypertension, headaches, psychotic episodes. In pediatric patients, seizures, hyperthermia, or coma may occur.[49] Nef et al.[48] described abnormalities in cardiac muscle mobility within the apical region, resembling Takotsubo syndrome. These cases primarily involve various species of the hallucinogenic genus *Psilocybe*.
In cases presenting with mild symptoms, gastric lavage, supportive care, or the induction of emesis is performed. For patients exhibiting anxious or aggressive behavior, benzodiazepines alongside pulmonary ventilation are recommended.[2] Over a four-year period, approximately 43 cases associated with voluntary ingestion for recreational-hallucinogenic purposes were documented.
The toxins are psilocybin and psilocin. The former acts as a prodrug, as its metabolite, psilocin, is the active agent. Due to its lipophilic nature, it readily crosses the blood-brain barrier. The hallucinogenic effect of psilocybin is attributed to the activation of serotonergic receptors (5-HT_{2A}). Psilocybin induces neural excitatory effects when 5-HT_{2A} receptors increase in the postsynaptic membrane. Psilocin is a mixed serotonergic receptor agonist that has a high affinity for the 5-HT_{2A} receptor in the brain, where it mimics the effect of serotonin. Hallucinogenic effects homologous to psilocybin have been described for baeocystin, which can also bind to specific subtypes of the 5-HT receptor.[2]

Group V: Neurotoxic of the autonomic nervous system

This group presents with individual intolerances that are common in immunosuppressed patients, children, pregnant women, and the elderly following the ingestion of edible mushrooms such as *Clitocybe nebularis*.[50-51] Symptoms typically initiate with headaches, nausea, and vomiting, subsequently progressing to sialorrhea, lacrimation, miosis, and blurred vision. Other symptoms include abdominal cramps, diarrhea, bronchoconstriction, and severe dyspnea. Bradycardia, hypotension, and vasodilation may occur, potentially leading to circulatory shock. Ingestion of a mushroom containing 0.33% muscarine by dry weight can be lethal, although such fatalities occur only in very rare instances.[2,52] The species are *Inocybe* spp., *Mycena* spp., and *Clitocybe* spp. *Clitocybe* spp. can induce acro-erythromelalgia, although they typically cause gastrointestinal distress. Given the mild nature of these cases, they do not usually result in significant discomfort; consequently, management typically involves gastric lavage or supportive treatment. In the case of *Inocybe* spp., treatment with atropine combined with induction of emesis, activated charcoal, and rehydration is indicated. If convulsions occur, benzodiazepines are indicated.[53,54] For acro-erythromelalgia caused by *Clitocybe* spp., management includes conventional analgesics combined with nicotinic acid,

followed by cold-water immersion of the extremities or elevation. Furthermore, the administration of opiates, acetylsalicylic acid, antidepressants (clomipramine), or clonazepam is indicated for pain management.[55] Some species of *Inocybe* exhibit psychotropic effects due to the presence of several toxic alkaloids, including psilocin, psilocybin, muscarine, aeruginascin, and baeocystin.[2,56] This presentation is similar to the poisoning caused by certain *Clitocybe* species, whose toxin, acromelic acid, can induce a disorder of tryptophan metabolism. In this process, the ACRO-A isomer causes damage to the unmyelinated fibers of the autonomic nervous system (ANS).[26,57] *Clitocybe* poisoning is classified into groups 2B and 6B in the White study[46], as it can induce autonomic nervous system symptoms through different mechanisms of action and, consequently, diverse clinical presentations depending on the species. Generally, an early onset of symptoms requires mild supportive treatment; however, a delayed onset −occurring over several days− indicates the development of acromelalgia syndrome

Group VI: CNS neurotoxins

This group presents with symptoms of intoxication characterized by visual hallucinations, ataxia, muscle spasms, mydriasis, and cutaneous erythema. These may be accompanied by hyperthermia, dry mucous membranes, or, conversely, profuse sweating. Cardiovascular manifestations such as bradycardia or tachycardia, and hypotension may occur, along with neurological signs including miosis, headache, dizziness, and paresthesias. Furthermore, gastrointestinal disturbances may be present. These symptoms are caused by the delusional mycoatropinic syndrome produced by *Amanita muscaria* and *A. pantherine.* The recommended treatment includes gastric lavage, administration of activated charcoal, hydroelectrolytic support, as well as diazepam for cases of psychomotor agitation. Should anticholinergic effects manifest, physostigmine should be administered intravenously in repeated doses every 15 to 30 minutes.[26] The primary psychoactive toxins are ibotenic acid (IBO) and muscimol. While IBO is structurally similar to glutamic acid in animals and is rapidly converted to muscimol, the latter exhibits a high affinity for the GABA receptor, mimicking its inhibitory action by modulating the recruitment and propagation of nerve impulses. Conversely, IBO is a potent agonist of the *N*-methyl-D-aspartic acid (NMDA) receptor. Other considerations include the phenological differences between the species in this group. Specifically, *Amanita muscaria* contains higher concentrations of IBO and lower levels of muscimol compared to *A. pantherine,* the latter of which is associated with the induction of coma. A retrospective analysis of patients poisoned by these two species between 1980 and 2013 led to a proposed clinical division into two distinct syndrome subtypes. Mechanistically, ibotenic acid acts as an excitatory amino acid on glutamate receptors, while muscimol exerts a depressant effect on the central nervous system. The White J[45] study discusses the separation of group 2B muscarines from group 2C ibotenic acid/muscimol. As previously mentioned, it is not uncommon to find cases of poisoning from spoiled edible mushrooms. These incidents may result from incorrect cooking or preparation, transport in plastic containers or at high temperatures, poor storage, or even specimens in good condition that trigger the so-called intolerance syndrome. This occurs when mushrooms are eaten in excessive quantities, are decayed, or have been parasitized.[35] Furthermore, it is suggested that the rise in such cases may be linked to the introduction of exotic species into new regions.[58] Consequently, it is recommended that comprehensive toxicological profiling be conducted over a specified period before introducing new species for human consumption. [59]

Group VII: Neurotoxins

This group presents with symptoms that are mainly gastrointestinal, occurring alongside neurological and other effects such as sweating, fainting, hyperthermia or hypothermia, salivation, headache, paresthesia, confusion, or dysarthria. In rare cases, symptoms such as instability and tremors may persist for several days, although they eventually disappear completely.[42] This identifies the cerebellar syndrome (which may be hemolytic or related to intolerance) caused by *Morchella* spp. (morels) and *Helvella* spp. Since these mushrooms are considered edible, poisoning typically occurs when they are consumed raw or have not been sufficiently dried or cooked. Insufficient cooking appears to be linked to cases of gastroenteritis which, when combined with individual intolerance, can become severe. However, there does not seem to be a similar correlation regarding cerebellar syndrome, as it can occur even after the consumption of fresh, well-cooked morels. Furthermore, while rare, there are reports of digestive symptoms classified under toxic mushroom poisoning patterns. [60] In conclusion, it is necessary to dry specimens for an extended period and consume them in limited quantities. Moreover, it may be advisable to avoid those collected under ash trees, as studies are currently investigating the possibility of a specific symbiosis between the genus *Fraxinus* and neurotoxic morels. Additionally, freezing should not be utilized as a preservation method. According to Lagrange & Vernoux[58] a portion of the toxicity in raw morels may be attributed to the concentration of toxins within the spores.

Group VIII: Disulfiram Effect

This group presents with symptoms that appear 15 to 30 minutes after consuming any alcoholic beverage, or after ingestion of the mushroom if alcohol has been previously consumed. This effect may reappear for up to four days following the initial ingestion of the toxin. It causes an erythematous rash on the trunk and extremities, accompanied by facial flushing, headache, dyspnea, sweating, nausea, vomiting, tachycardia, ventricular contractions, atrial fibrillation, vertigo, confusion, and a metallic taste. This syndrome is recognized as coprine or antabuse effect, which is induced by species of the genus *Coprinus*−particularly *Coprinus atramentarius*−*as well as*

Clitocybe clavipes. Regarding treatment, gastric lavage is typically unnecessary as the poison is generally mild; supportive therapy and fluid and electrolyte replacement are usually sufficient. Furthermore, high doses of intravenous vitamin C have shown a favorable effect, acting as a "redox" factor. An effective antidote is 4-methylpyrazole, which prevents the formation of acetaldehyde by blocking the initial oxidative step of ethanol. The toxin is a cycloaminopropanone derivative with biochemical properties similar to disulfiram. Coprine inhibits the conversion of acetaldehyde to acetate, leading to the accumulation of aldehyde in the liver and blood. There is one reported[61-62] case of death following the consumption of "inky caps"; however, the fatality was not directly attributed to mushroom toxicity. Instead, it resulted from an esophageal rupture, likely due to the intensity of vomiting.

Group IX: Hyperprocalcitonemia

This group presents with symptoms such as vomiting, diarrhea, pain, cramps, sweating, salivation, or fever, which may indicate resinoid syndrome. This condition is caused by several species, including *Boletus* spp., *Boletus Satanas* (currently classified as *Rubroboletus satanas*), *Omphalotus olearius*, and *Lactarius* spp. The recommended treatment is consistent with that of other non-severe gastrointestinal poisonings. Additionally, research has found that galactose may prevent the toxin from binding to the cellular membrane.[63] Vitamin K may be useful in cases of coagulopathy.[29] Antiemetics, such as metoclopramide, may be administered to manage nausea and vomiting resulting from raw ingestion; similarly, butylscopolamine may be prescribed for colic, alongside esomeprazole to reduce gastric acid secretion.[64] It is essential to determine whether the medications administered are the most appropriate, as there have been reports of exacerbated poisoning following the use of a drug such as ramipril.[29] Currently, the specific toxins for most species in this group remain unidentified, with the exception of *Boletus Satanas* (currently classified as *Rubroboletus Satanas*), which contains bolesatin. Certain *Boletus* species contain sufficient levels of muscarine to produce the muscarinic toxicity characteristic of cholinergic syndromes.[48] Specifically, *Boletus satanas* (currently *Rubroboletus satanas*) exhibits procoagulant or prothrombotic activity.[60] Bolesatin possesses a D-galactose binding site, which characterizes it as a potent agglutinator of erythrocytes and platelets.[60] The incidence of poisoning is high, which, as Schenk-Jaeger et al.[63] point out, may be attributed to the fact that numerous cases are linked to the incorrect preparation of these mushrooms. This suggests that many species are not inherently toxic, but rather require adherence to very specific protocols regarding their cooking, storage, and transport. An increasing number of poisoning cases are being reported for species previously considered safe for consumption, highlighting the urgent need for further research in this field.[62] Consequently, other types of poisoning have been excluded from the current study due to a lack of documented information regarding their clinical presentation and symptoms.

CONCLUSION

The clinical presentation of mushroom poisoning can often mimic other pathologies; therefore, it is vital to inquire whether mushrooms have been consumed in the preceding days. Impaired hepatic function is evidenced by alterations in liver enzymes, such as AST, ALT, and bilirubin levels. Treatment remains common across all species, consisting of symptomatic and supportive care, supplemented by specific pharmacological agents or specialized techniques. Hemodialysis may be performed if the patient is within the latent period. Additionally, the administration of activated charcoal at 0.5 to 1 g/kg body weight is an option, though it is strictly recommended only if administered within one hour of ingestion. To the same end, and if performed within one hour of ingestion, the placement of an aspiration tube, induction of diuresis, or gastric lavage has proven successful −though these interventions are contraindicated in patients presenting with a diminished cough reflex, coma, or a high risk of hemorrhage. Additionally, pharmacotherapy with agents such as penicillin G, silibinin, and N-acetylcysteine has proven successful, even when administered in combination. Silibinin's efficacy stems from its ability to stabilize the hepatic membrane by blocking lysosomal proteases and preventing α-amanitin from entering hepatocytes. Evidence suggests that administering silibinin within the first 48 hours of ingestion significantly reduces the likelihood of severe liver damage. It has also been observed that silymarin exerts protective effects against cell membrane damage caused by osmotic stress and free radicals, effectively blocking the entry of toxins such as α amanitin. In cases of liver failure, MARS or other albumin-based dialysis techniques may be employed as a last resort until transplantation can be performed. Regarding toxicodynamics, three types of protoplasmic poisons have been identified: amanitins, virotoxins, and phalloidins.

Amanitins represent the most toxic compounds within this group. The primary target organ is the liver, where they trigger a significant elevation in transaminases that can progress to fulminant liver failure. It has been suggested that these toxins promote the formation of reactive oxygen species (ROS), inducing oxidative stress; furthermore, amatoxins may act synergistically with TNF, thereby inducing apoptosis. Beyond the hepatic damage, they affect the central nervous system (CNS), and certain studies indicate a higher concentration of α-amanitin in the kidneys than in the liver. However, various experimental studies in dogs and mice, along with published human case reports, suggest the contrary: the quantity of amatoxins excreted via bile is considerably greater than that eliminated through urine. Regarding the molecular mechanism, the actin polymer that binds to the *Amanita phalloides* peptide delays the release of phosphate generated by ATP hydrolysis. Finally, virotoxins are monocyclic peptides with structures similar to those of phallotoxins, distinguished by their

monocyclic nature and the presence of a sulfoxide group. They appear to be less toxic. Significant controversy persists regarding the classification of these syndromes, as authors frequently propose disparate groupings of species within toxicological frameworks. Furthermore, some researchers argue that *Amanita verna* is even more toxic than A. *phalloides,* presenting a higher mortality rate; this suggests that within any given classification, there should be a gradient to account for varying degrees of toxicity.

REFERENCES

[1] Stebelska K. Fungal hallucinogens psilocin, ibotenic acid, and muscimol: analytical methods and biologic activities. Ther. Drug. Monit. 2013; 35(4): 420-42.

[2] Patocka J, Wu R, Nepovimova E, Valis M, Wu W, Kuca K. Chemistry and Toxicology of Major Bioactive Substances in Inocybe Mushrooms. Int. J. Mol. Sci. 2021; 22(4): 2218.

[3] Kosentka P, Sprague SL, Ryberg M, Gartz J, May AL, Campagna SR, et al. Evolution of the toxins muscarine and psilocybin in a family of mushroom-forming fungi. PLoS ONE. 2013; 8(5): e64646.

[4] Carod-Artal FJ. Neurological syndromes associated with the ingestion of plants and fungi with a toxic component (II). Hallucinogenic fungi and plants, mycotoxins and medicinal herbs. Rev. Neurol. 2003; 36(10): 951–60.

[5] Manzanilla, L., Lujan. L. L. Historia antigua de México: Aspectos fundamentales de la tradición cultural mesoamericana. 1a ed México: UNAM; 2000

[6] Carod-Artal FJ, Vázquez-Cabrera CB. An anthropological study about epilepsy in native tribes from Central and South America. Epilepsia. 2007; 48(5): 886-93.

[7] Schultes, R. E. Who uses hallucinogenic plants?; Plants of the Gods: their sacred, healing, and hallucinogenic powers. E.U: CreateSpace; 2001: 62-5.

[8] Carod-Artal FJ. Hallucinogenic drugs in pre-Columbian Mesoamerican cultures. Neurologia. 2015; 30(1): 42-9.

[9] Vollenweider FX, Kometer M. The neurobiology of psychedelic drugs: implications for the treatment of mood disorders. Nat. Rev. Neurosci. 2010; 11(9): 642-51.

[10] Duchi Ñauta P. La psilocibina, sustancia psicodélica del hongo orientada a ser el nuevo enfoque de la medicina. Rev. Juv. Cie. Sol. 2022; 12: 57-9.

[11] Yilmaz I, Kaya E, Sinirlioglu ZA, Bayram R, Surmen MG, Colakoglu S. Clinical importance of toxin concentration in Amanita verna mushroom. Toxicon. 2014; 87: 68-75.

[12] Mckenna, terence k. Food of the gods. Bantam, 1992.

[13] Rodríguez Arce JM, Winkelman MJ. Psychedelics, Sociality, and Human Evolution. Front. Psychol. 2021; 12: 729425.

[14] Nichols DE. Psilocybin: from ancient magic to modern medicine. J. Antibiot (Tokyo). 2020; 73(10): 679-86.

[15] R. Gordon Wasson. A velada in Huautla. The Wondrous Mushroom. Mycolatry in Mesoamerica. United States of America: Sungazer Press 2021. Pág 3-31

[16] Hofmann A, Heim R, Brack A, Kobel H, Frey A, Ott H. Psilocybin und psilocin, zwei psychotrope wirkstoffe aus mexikanischen rauschpilzen. Helvetica Chimica Acta. 1959; 42(5): 1557-72.

[17] Zeiss R, Gahr M, Graf H. Rediscovering Psilocybin as an Antidepressive Treatment Strategy. Pharmaceuticals (Basel). 2021; 14(10): 985.

[18] Lieberman JA, Shalev D. Back to the future: Research renewed on the clinical utility of psychedelic drugs. J. Psychopharmacol. 2016; 30(12): 1198-1200.

[19] Marks M, Cohen IG. Psychedelic therapy: a roadmap for wider acceptance and utilization. Nat. Med. 2021; 27(10): 1669-1671.

[20] Mithoefer MC, Grob CS, Brewerton TD. Novel psychopharmacological therapies for psychiatric disorders: psilocybin and MDMA. Lancet Psychiatry. 2016; 3(5): 481-8.

[21] World Health Organization. Depression [Internet]. World health organization, world health organization, 2019 [cited 2025 Jan 25]. Available from: https://www.who.int/health-topics/depression#tab=tab_1

[22] Bogenschutz MP, Ross S. Therapeutic Applications of Classic Hallucinogens. Curr. Top. Behav. Neurosci. 2018; 36: 361-91.

[23] Johnson MW, Griffiths RR. Potential Therapeutic Effects of Psilocybin. Neurotherapeutics. 2017; 14(3): 734-40.

[24] Vargas MV, Meyer R, Avanes AA, Rus M, Olson DE. Psychedelics and Other Psychoplastogens for Treating Mental Illness. Front. Psychiatry. 2021; 12: 727117.

[25] Schmutz M, Carron PN, Yersin B, Trueb L. Mushroom poisoning: a retrospective study concerning 11-years of admissions in a swiss emergency department. Int. Emerg. Med. 2016; 13(1): 59–67.

[26] Moreno G, Manjón JLG. Guía de hongos de la Península Ibérica: con la descripción de 584 especies más de 1000 fotografiás en color y numerosos dibujos. Turín, Italia: Omega; 2010.

[27] Bonnet MS, Basson PW. The toxicology of Amanita phalloides. Homeopathy. 2002; 91(4): 249-54.

[28] Palmier Peláez, Esmeralda. Estudio epidemiológico de la intoxicación aguda por setas [Trabajo de grado]. Valladolid, España: Universidad de Valladolid. Facultad de Medicina; 2016

[29] Erden A, Esmeray K, Karagöz H, Karahan S, Gümüşçü HH, Başak M. Acute liver failure caused by mushroom poisoning: a case report and review of the literature. Int. Med. Case Rep. J. 2013; 6: 85-90.

[30] Diaz JH. Amatoxin-Containing Mushroom Poisonings: Species, Toxidromes, Treatments, and Outcomes. Wilderness Environ. Med. 2018; 29(1): 111-8.

[31] Alvarado P, Gasch-Illescas A, Morel S, Dagher-Kharrat MB, Moreno G, Manjón JL, et al. Amanita Section Phalloideae Species in the Mediterranean Basin: Destroying Angels Reviewed. Biology (Basel). 2022; 11(5): 770.

[32] Keller SA, Klukowska-Rötzler J, Schenk-Jaeger KM, Kupferschmidt H, Exadaktylos AK, Lehmann B, et al. Mushroom Poisoning-A 17 Year

Retrospective Study at a Level I University Emergency Department in Switzerland. Int. J. Environ. Res. Public Health. 2018; 15(12): 2855.

[33] Colak S, Kandis H, Afacan MA, Erdogan MO, Gunes H, Kaya E, et al. Assessment of patients who presented to the emergency department with mushroom poisoning. Hum. Exp. Toxicol. 2015; 34(7): 725-31.

[34] Ahishali E, Boynuegri B, Ozpolat E, Surmeli H, Dolapcioglu C, Dabak R, et al. Approach to mushroom intoxication and treatment: can we decrease mortality? Clin. Res. Hepatol. Gastroenterol. 2012; 36(2): 139-45.

[35] Grabhorn E, Nielsen D, Hillebrand G, Brinkert F, Herden U, Fischer L, et al. Successful outcome of severe Amanita phalloides poisoning in children. Pediatr. Transplant. 2013; 17(6): 550-5.

[36] Ramírez-Terrazo A, Garibay-Orijel R, Reyes-Chilpa R, Casas A, Méndez-Espinoza C. Alternativas para la atención oportuna de las intoxicaciones por consumo de hongos en México y Centroamérica. Gac. Med. Mex. 2023; 159(4).

[37] Garcia J, Costa VM, Carvalho A, Baptista P, de Pinho PG, de Lourdes Bastos M, et al. Amanita phalloides poisoning: Mechanisms of toxicity and treatment. Food Chem. Toxicol. 2015; 86: 41-55.

[38] Sun J, Niu YM, Zhang YT, Li HJ, Yin Y, Zhang YZ, et al. Toxicity and toxicokinetics of Amanita exitialis in beagle dogs. Toxicon. 2018; 143: 59-67.

[39] Piqueras J. Hepatotoxic mushroom poisoning: diagnosis and management. Mycopathologia. 1989; 105(2): 99-110.

[40] Jaeger A, Jehl F, Flesch F, Sauder P, Kopferschmitt J. Kinetics of amatoxins in human poisoning: therapeutic implications. J. Toxicol. Clin. Toxicol. 1993; 31(1): 63-80.

[41] Bever CS, Swanson KD, Hamelin EI, Filigenzi M, Poppenga RH, Kaae J, et al. Rapid, Sensitive, and Accurate Point-of-Care Detection of Lethal Amatoxins in Urine. Toxins (Basel). 2020; 12(2): 123.

[42] Piqueras J. More Mushroom Toxicity: An Update. Fungi. 2021;14(2):45-52

[43] Najar D, Haraldsson B, Thorsell A, Sihlbom C, Nyström J, Ebefors K. Pharmacokinetic Properties of the Nephrotoxin Orellanine in Rats. Toxins (Basel). 2018; 10(8): 333.

[44] Dinis-Oliveira RJ, Soares M, Rocha-Pereira C, Carvalho F. Human and experimental toxicology of orellanine. Hum. Exp. Toxicol. 2016; 35(9): 1016-29.

[45] White J, Weinstein SA, De Haro L, Bédry R, Schaper A, Rumack BH, et al. Mushroom poisoning: A proposed new clinical classification. Toxicon. 2019; 157: 53-65.

[46] Lheureux P, Penaloza A, Gris M. Pyridoxine in clinical toxicology: a review. Eur J Emerg Med. 2005; 12(2): 78-85.

[47] Graeme KA. Mycetism: a review of the recent literature. J. Med. Toxicol. 2014; 10(2): 173-89.

[48] Nef HM, Möllmann H, Hilpert P, Krause N, Troidl C, Weber M, et al. Apical regional wall motion abnormalities reminiscent to Tako-Tsubo cardiomyopathy following consumption of psychoactive fungi. Int. J. Cardiol. 2009; 134(1): e39-41.

[49] Wennig R, Eyer F, Schaper A, Zilker T, Andresen-Streichert H. Mushroom Poisoning. Dtsch .Arztebl. Int. 2020; 117(42): 701-8.

[50] Pauli JL, Foot CL. Fatal muscarinic syndrome after eating wild mushrooms. Med J Aust. 2005; 182(6): 294-5.

[51] Diaz JH. Evolving global epidemiology, syndromic classification, general management, and prevention of unknown mushroom poisonings. Crit. Care Med. 2005; 33(2): 419-26.

[52] Diaz JH. Syndromic diagnosis and management of confirmed mushroom poisonings. Crit Care Med. 2005; 33(2): 427-36.

[53] Nogué S, Simón J, Blanché C, Piqueras J, Menarini L. Intoxicaciones por plantas y setas. Wordpress [cited 2025 Oct 31]. Available from: https://joseppiqueras.wordpress.com/wp-content/uploads/2016/06/2009-menarini.pdf

[54] Stebelska K. Fungal hallucinogens psilocin, ibotenic acid, and muscimol: analytical methods and biologic activities. Ther. Drug Monit. 2013; 35(4): 420-42.

[55] Flammer R, Schenk-Jäger KM. Pilzvergiftungen-die Schattenseiten des Myzetismus [Mushroom poisoning-the dark side of mycetism]. Ther. Umsch. 2009; 66(5): 357-64.

[56] Lima ADL, Costa Fortes R, Garbi Novaes MRC, Percário S. Poisonous mushrooms; a review of the most common intoxications. Nutr. Hosp. 2012; 27(2): 402-8.

[57] Piqueras-Carrasco J. Intoxicaciones por setas, una actualización. Rev. Esp. Med. Legal. 2014; 40(1): 19–29.

[58] Lagrange E, Vernoux JP, Reis J, Palmer V, Camu W, Spencer PS. An amyotrophic lateral sclerosis hot spot in the French Alps associated with genotoxic fungi. J. Neurol. Sci. 2021; 427: 117558.

[59] Mayer JH, Herlocher JE, Parisian J. Esophageal rupture after mushroom-alcohol ingestion. 1971; 285(23): 1323

[60] Kretz O, Creppy EE, Dirheimer G. Characterization of bolesatine, a toxic protein from the mushroom Boletus satanas Lenz and it's effects on kidney cells. Toxicology. 1991; 66(2): 213-24.

[61] Keller SA, Klukowska-Rötzler J, Schenk-Jaeger KM, Kupferschmidt H, Exadaktylos AK, Lehmann B, et al. Mushroom Poisoning-A 17 Year Retrospective Study at a Level I University Emergency Department in Switzerland. Int. J. Environ. Res. Public Health. 2018; 15(12): 2855.

[62] Gachet C, Ennamany R, Kretz O, Ohlmann P, Krause C, Creppy EE, et al. Bolesatine induces agglutination of rat platelets and human erythrocytes and platelets in vitro. Hum. Exp. Toxicol. 1996; 15(1): 26-9.

[63] Schenk-Jaeger KM, Rauber-Lüthy C, Bodmer M, Kupferschmidt H, Kullak-Ublick GA, Ceschi A. Mushroom poisoning: a study on circumstances of exposure and patterns of toxicity. Eur. J. Intern. Med. 2012; 23(4): e85-91.

[64] Bernardino Sahagun RP. Historia general de las cosas de nueva España: Tomo 1. Outlook Verlag; 2022.