Six groups of poisonous mushrooms: classified according to clinical symptoms

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Abstract. Since they have been consumed for a very long time, mushrooms have been linked to numerous ailments that are primarily toxin-induced. The precision and speed of diagnosis will be hampered by a lack of knowledge of the signs of mushroom poisoning, which poses a serious risk to public health and safety. This paper mainly discus cytotoxic, neurotoxic, myotoxic, metabolic, endocrine and related toxicity, gastrointestinal irritant, and miscellaneous adverse reactions of mushrooms, indicating that mushrooms can be categorized based on the kind of harmful reaction, which is helpful for the evaluation and analysis of clinical problems. By having a better understanding of various symptoms, one can make wiser decisions and develop effective coping mechanisms in the event of poisoning, thereby minimizing the damage caused by mushrooms and refocusing attention on the investigation of whether mushrooms may have any potential benefits.

Keywords: poisonous mushrooms, clinical symptoms.

1. Introduction

Macrofungi, sometimes known as mushrooms, are fungal taxa that develop above or below ground and have massive, readily visible spore-bearing structures. Worldwide, 56,679 different species of macrofungi (mushrooms) have been recognised [1]. Gastrointestinal irritation, other unpleasant reactions, neurotoxicity, myotoxicity (rhabdomyolysis), metabolic (including endocrine and related toxicity), and cytotoxicity are the six categories of symptoms caused by poisonous mushrooms. [2]. Toxic mushrooms that are cytotoxic and myotoxic are responsible for the majority of fatal poisonings [1]. Consuming poisonous mushrooms is still a health risk in many nations, increasing incidence and mortality. One can only recognize and treat symptoms more accurately if a deeper grasp of the underlying causes is within reach.

2. Cytotoxic mushroom

For cytotoxic mushrooms category which includes poisonings that cause main hepatotoxicity or primary nephrotoxicity due to specific significant internal organ disease [2]. The liver and kidneys are the primary targets of cytotoxic mushrooms. Amanitin, aminohexadienoic acid, and orellanine are the toxins in charge of harming the liver and kidneys. The majority of the species in Amanita Pers. and Cortinarius (Pers.) Gray that produce these toxins are [1,3]. Both primary hepatotoxicity and primary nephrotoxicity are subgroups of cytotoxic mushrooms. After then, the main nephrotoxicity can be further split into two categories: delayed primary nephrotoxicity and early primary nephrotoxicity [2].

Toxins like amatoxins that produce potentially primary hepatotoxicity and have a particular constellation of clinical characteristics are included in the main hepatotoxicity subgroup [2]. Medically, this kind of poisoning manifests as a protracted development of pre-hepatotoxic symptoms that start with typically severe gastrointestinal (GI) effects following consumption (nausea, vomiting, diarrhoea, abdominal pain, secondary dehydration). In severe cases, hepatotoxicity will progress to full liver failure (coagulopathy, bleeding, and hepatic encephalopathy), frequently in conjunction with renal failure, and result in death from hepatic failure on or around day 7 if corrective measures are unsuccessful. Hepatotoxicity will first manifest on or around day 3 in the form of abnormal laboratory findings (liver function, coagulation), but this won't happen until that day or the next. A liver

transplant, which is occasionally either unavailable or medically impractical, may be the sole chance for survival in severe circumstances [2].

Those mushrooms are under the early primary nephrotoxicity category and cause acute direct renal injury. Amanita smithiana and Amanita pseudoporphyria, two mushrooms high in aminohexadienoic acid (AHDA), are linked to this [4]. The symptoms of A. smithiana poisoning, which have been reported, include a variety of constitutional (or "pre-renal") symptoms that start anywhere between 30 minutes and 12 hours after ingestion. These signs and symptoms include diarrhoea, vomiting, stomach cramps, headaches, weakness, and exhaustion. Acute renal failure may start 2–5 days after ingestion and last for a few days before renal recovery is complete. This may last for a few days or more. Dialysis is required for a period of 9 to 180 days in renal failure. Liver function enzymes (LFTs) may rise early and inconsistently before quickly returning to normal [4]. This preliminary indication of potential liver toxicity can lead to diagnostic misunderstanding with amatoxin poisoning.

The subgroup of mushrooms that cause delayed primary nephrotoxicity includes those that result in renal failure. It is linked to some Cortinarius species, particularly those that contain orellanine [5]. The clinical manifestation of this kind of poisoning is delayed onset renal failure, which develops 4–15 days after intake [5,6,7,8,9,10]. Pre-renal symptoms may manifest 12 hours to 14 days after swallowing the chemical and include nausea, vomiting, diarrhoea, dehydration, thirst, anorexia, headaches, chills, parasthesia, sleepiness, sweating, rash, dyspnea, and lumbar pain (mean 3 days)[5,7]. The more quickly renal failure develops, the worse the prognosis; acute kidney injury (AKI) with an onset time of 2-3 days is severe; AKI with an onset time of >10 days is often mild [7]. The earliest indication of renal failure is frequently polyuria, which is then followed by oliguria or anuria. Chronic renal failure is a possibility, and the restoration of renal function can take some time.

3. Neurotoxic mushroom

Macrofungi that have neuroexcitatory properties include mushrooms that are neurotoxic. Psilocybin, muscarines, and isoxazoles are the recognised poisons. Agaricales mushrooms like Amanita, Clitocybe, Inocybe, Psilocybe, and Gymnopilus are the main producers of these poisons. Several Pezizales mushrooms, including Morchella spp., have also been linked to neurotoxic syndrome, however the exact toxins responsible for this are still unknown [1]. Hallucinogenic mushrooms, autonomic toxicity mushrooms, central nervous system neuroexcitatory mushrooms, and morel neurologic illness are the four main classifications of neurotoxic mushrooms [2].

Any mushroom that has hallucinations or other psychoactive effects as its primary presenting symptom is considered hallucinogenic. clinically insignificant, this kind of trying to poison manifests as symptoms or signs between 10 and 30 minutes after ingestion, effects on the central nervous system (CNS), illusions (visual, auditory, or tactile), true hallucinations (in 50% of cases), altered perception of time and space, synaesthesia, and feelings of euphoria, which are frequently described as a mystical experience that may last for a while [11,12,13]. Some people have the potential to act aggressively, especially those who have consumed more alcohol. It's possible to hurt oneself or other people. There could be cardiac arrhythmias, myocardial ischaemia, ataxia, tachycardia, hyperreflexia, parasthesia, anxiety, nausea, vomiting, and discomfort in the abdomen [2].

The subset of mushrooms known as autonomic toxicity includes those that directly affect the autonomic nervous system. It has to do with the fact that mushrooms contain muscarines and other toxins that are comparable to them. Clinically, this type of poisoning shows up as a sudden (15 min–2 h) onset of normal parasympathetic activation, which comprises the trinity of enhanced sweating, salivation, and lachrymation. Other signs include constricted pupils, blurred vision, painful or urgent urination, nasal discharge, bronchoconstriction, asthma, skin flushing, hypotension, bradycardia, vomiting, and stomach/colic pain [2].

Mushrooms that produce neuroexcitatory effects, often involving "hallucinations," are referred to as neuroexcitatory mushrooms. Ibotenic acid and muscimol are the poisons. Amanita muscaria, Amanita patherina, and Amanita ibotengutak are just a few of the mushrooms that are involved [14].

In terms of clinical manifestation, this kind of poisoning occurs very quickly (within minutes, up to 3 hours) after ingestion, with initial drowsiness (especially in children), followed by a period of manic excitement, and other symptoms that may include nausea, vomiting, diarrhoea, abdominal pain, rash, sweating, ataxia, incoordination, dizziness, and other symptoms (especially in children). The final stage of these symptoms/signs, which is drowsiness, can last for up to 48 hours (often deep sleep). There could be forgetfulness for the time spent under the influence. Seizures and coma (GCS 4) may develop quickly[15].

The illness known as "morel neurologic syndrome," which occurs after eating some morel mushrooms, causes neurological and gastrointestinal (GI) symptoms, is now poorly understood [16]. Since it was first described in 1889, this type of "poisoning" has occasionally been detected, but no toxin has been found to be the cause, and its status is unknown. [2]. Clinically, eating a lot of morel mushrooms has been linked to this kind of poisoning. It's unclear when the symptoms started. In the majority of instances, there were neurological side effects as well as GIT side symptoms (vomiting, diarrhoea, nausea, and abdominal discomfort). Dizziness and unsteadiness/ataxia were some of the consequences that were most noticeable. Asthenia, sweating, dizziness, hyperthermia, hypothermia, salivation, headache, parasthesia, trismus, muscle spasms, drowsiness, disorientation, and dysarthria are other less frequent side effects. Nystagmus, miosis, mydriasis, dysphagia, hallucinations, agitation, and convulsions are a few of the seldom documented side effects. There hasn't yet been any indication of renal or hepatic involvement. All of the known cases to date have come to a conclusion on their own [2].

4. Myotoxic mushroom

This group of poisonings includes those in which rhabdomyolysis is the main symptom, therefore the name myotoxic mushroom poisoning [2]. Myotoxic mushrooms, which mostly come from Russula and Tricholoma, are strongly linked to rhabdomyolysis symptoms. Russula subnigricans Hongo, Tricholoma equestre (L.) P. Kumm, and T. terreum (Schaeff.) P. Kumm are the common species mentioned [1]. The two main subgroups of myotoxicity are rapid onset and delayed onset [2].

The mushrooms that cause quick onset myotoxicity fall into the rapid onset myotoxicity subgroup [17]. Cycloprop-2-ene carboxylic acid, a highly strained carboxylic acid, has reportedly been identified as the toxic substance [17,18,19]. It has to do with consuming particular Russula species. Cyclopropylacetyl-(R)-carnitine has been recognised as the chemical that best identifies this species[20]. Clinically, this kind of poisoning manifests as the beginning of GIT symptoms (perhaps severe nausea, vomiting, and diarrhoea), which typically begin within 2 hours of consumption but may occasionally be delayed even longer [19]. The majority of incidents end within 24 hours, leaving no trace of rhabdomyolysis. After more severe GIT effects, rhabdomyolysis (peak CK > 200,000 IU/l), myalgias, hypertension, renal failure, hyperkalemia, and cardiovascular collapse may occasionally happen.

The mushrooms that cause delayed onset myotoxicity fall into the delayed onset myotoxicity subgroup. Even while the clinical manifestation of rhabdomyolysis may be relatively similar, the mechanism may vary based on the type of mushroom and toxin. It is linked to the consumption of specific Tricholoma species, including T. equestre, T. terreum, and probably T. auratum. Clinically, this kind of poisoning manifests as myalgia and delayed-onset lethargy one to three days after eating mushrooms for numerous consecutive meals. It might matter how many mushrooms you eat over the course of several meals. Following this are progressive weakness, leg stiffness, myoglobinuria (black urine), face erythema occasionally, mild nausea (but not vomiting), and heavy sweating. The kidneys, serum potassium, coagulation, and liver regularly function despite the potential for a large rise in plasma CK. However, a tiny percentage of them result in dyspnea, fever, acute myocarditis, renal failure, cardiac failure, hyperkalemia, and catastrophic cardiac collapse. The majority of cases go away within 15 days [2].

5. Metabolic, endocrine and related toxicity mushroom

The inclusion of many poisonous mushrooms in this large category was done so for convenience even though they rarely exhibit clinical commonalities [1]. There is a link between museum poisonings and the following metabolic, endocrine, and related toxicological subgroups: Pancytopenia, trichothecene poisoning, GABA-blocking poisoning, disulfiram-like poisoning, hypoglycemia poisoning, hyperprocalcitoninemia poisoning, and polyporic mushroom poisoning These are all instances of mushroom poisoning [2].

GABA-blocking mushrooms are those that inhibit the production of GABA and hence cause metabolic-based disease with multi-organ consequences. It is connected to some Gyromitra species, which contain gyromitrins [21]. Theoretically, this kind of poisoning manifests as GIT symptoms five to twelve hours or more after intake. More severe poisonings may also result in the following signs and symptoms: vertigo, hypoglycemia, methaemoglobinemia, sweating, diplopia, headache, dysarthria, incoordination, ataxia, hemolysis, and liver damage (usually develops in first 48 h). Gyromitrin poisoning does, however, appear to be influenced by the patient's acetylator status; "fast acetylators" (high levels of acetalhydrazin) typically develop hepatotoxicity (mild transaminase elevation, but high bilirubin levels), whereas patients with normal acetylator status typically present with neurotoxicity dominating the symptoms [21]. A severe poisoning could be fatal.

When alcohol is consumed subsequently, the mushrooms in the disulfiram-like mushroom poisoning subgroup elicit a reaction similar to disulfiram (after eating the mushrooms). It is connected to certain Coprinus species, which contain coprines (notably C. atramentarius) [2]. Clinically, this kind of poisoning manifests after eating the mushrooms and in response to consuming alcohol earlier or later. There has been a reported delay between the mushroom meal and alcohol use of anything between two hours and many days. Shortly after taking the alcohol, the patient develops a blotchy, erythematous rash across their body and limbs. Some potential symptoms and warning signs include headache, dyspnea, sweating, nausea, vomiting, tachycardia, premature ventricular contractions, atrial fibrillation, vertigo, confusion, and a metallic taste. Hypotension is less common when compared to disulfiram, and coma is exceedingly unusual to rare. Usually only lasting for about 30 minutes, symptoms might occasionally extend up to 24 hours [2].

The grouping of polyporic mushrooms that cause neurologic and multi-organ symptoms includes these fungi. It is connected to polyporic acid-containing mushrooms, particularly Hapalopilus rutilans [22]. Clinical signs of this type of poisoning include a delayed (12 hours or more) onset of GIT symptoms (abdominal pain, nausea, vomiting, and diarrhoea), neurologic symptoms (diplopia, blurred vision, disordered balance, nystagmus, and occasionally visual hallucinations), mild AKI, mild hepatotoxicity, proteinuria, leukocyturia, and purple urine (a diagnostic feature). Within two to seven days, the syndrome disappears [2].

The subgroup of trichothecene mushrooms that leads to bone marrow failure and lamellar desquamation of the face, palms, and soles of the feet, among other multi-organ failures. Trichothecenes-containing mushrooms, particularly Podostroma cornu-damae, are linked to it. Theoretically, this kind of poisoning manifests as a multiorgan illness that frequently ends in death and can happen either right away after intake or only after taking mushrooms on a regular basis for a few days or weeks. The first GIT side effects of nausea, vomiting, diarrhoea, and dehydration are probable. Hypotension, oliguric renal failure, impaired mental status, pancytopenia, lamellar desquamation of the face, palms, and soles, as well as generalised hair loss, may then follow these consequences. Multi-organ failure death is frequently the end result in many cases[2].

The subgroup of mushrooms that quickly cause hypoglycemia is known as hypoglycemic mushrooms. According to clinical standards, the symptoms of poisoning, which include palpitations, fainting, "chest uneasiness," shortness of breath, stomach pain, and syncope, come suddenly. In severe circumstances, death could occur approximately two hours after consumption [2].

One small case series from France makes up the hyperprocalcitoninemia mushroom poisoning subgroup. Low grade temperature (38.5 °C) and early GIT symptoms (vomiting, diarrhoea) start to appear about 2 hours after ingesting the poison. Procalcitonin (ProCT) and C-reactive protein (CRP)

plasma levels are elevated 12 hours after ingestion, although hepatic and renal function are unchanged. After 24 hours, ProCT and CRP levels steadily decline and all symptoms quickly go away [2].
An uncommon poisoning is included in the pancytopenia mushroom poisoning subgroup. Clinically, this type of poisoning manifests as an inexplicable fever, which upon analysis reveals pancytopenia (anaemia, leukopenia, and thrombocytopenia), with no involvement of any other organ systems [2].

6. gastrointestinal irritant mushroom

A vast number of mushrooms that just produce gastrointestinal irritation without having any other discernible consequences is referred to as "gastrointestinal irritant mushroom poisoning." Medically, this type of poisoning has a wide range of effects on the gastrointestinal tract (GIT), and in severe cases, dehydration-related problems due to fluid loss from the GIT are a potential. It's crucial to remember that now the GIT effects could result from a range of factors and aren't always brought on by pollutants. Certain mushrooms could be hard to digest, especially if you eat a lot of them. GIT distress and symptoms/signs resembling those seen in the early stages of poisoning may develop from this.

7. Miscellaneous adverse mushrooms

These sorts of mushroom "poisoning" that don't fall under one of the previous five categories are included in this section. Not all are brought on by certain "poisoning" responses [2]. The four subgroups of group 6 include shiitake mushroom dermatitis, erythromelalgia-like mushroom poisoning, Paxillus disease, and encephalopathy syndrome [2].
The subgroup of shiitake mushrooms that cause acute post-ingestive dermatitis includes these mushrooms. It has a connection to shiitake mushrooms (Lentinola edodes) [2].

The mushrooms that cause an erythromelalgic-like syndrome are included in the erythromelalgic-like mushroom poisoning subgroup [23]. It is related to Paralepistopsis (Clitocybe) acromelalga/amoenolens, also called the toxic dwarf bamboo mushroom or the paralysis funnel [2].

The subset of paxillus syndrome includes mushrooms that, following repeated exposure, cause an autoimmune hemolytic anaemia. It is associated with Paxillus species, often known as brown roll-rim mushrooms (P. involutus). Theoretically, this sort of poisoning is highly uncommon and then only appears after consuming the mushroom repeatedly. Before proceeding to intravascular hemolysis, anaemia, and maybe renal failure, the mushroom's immunological reaction first appears as acute respiratory failure, shock, DIC, and GIT symptoms (nausea, vomiting, stomach pain, and diarrhoea) [2].

It is believed that HydroCyanic Acid (HCN) poisoning caused by eating mushrooms with high HCN contents is the cause of the encephalopathy syndrome subgroup poisoning syndrome, which has been reported from Japan. Some of the mushrooms connected to this type of poisoning are Pleurotus eringii, Grifola frondosa (hen-of-the-woods, ram's head, sheep's head, maitake, and signorina), and Pleurocybella porrigens (angel wing) (king trumpet, French horn, oyster, king brown, boletus of the steppes, and trumpet royale) [24]. In terms of medicine, this kind of poisoning results in "cramps" and a gradual onset of unconsciousness [2].

8. Conclusions

People are particularly excited about eating mushrooms in China, which occasionally results in mushroom poisoning. The scenarios that arise in real life are a lot more complex than the cases that are tested in experiments. For instance, whether combining various mushrooms would result in new toxins or symptoms that won't occur when the two are separated, or whether consuming food with mushrooms will have different impacts on the effects of toxins, causing the important but less noticeable symptoms. The patient's personal health must occasionally be taken into consideration (some diseases or abnormal hormone levels). The likelihood that the patient would receive prompt

and effective care is higher in terms of the current medical circumstances, the more cases there are, the more specific the classification, and the more referential the clinical diagnosis. Additionally, if awareness of mushroom categorization could be spread, the risk of unintentional consumption due to the following knowledge gaps could be reduced. Dialectical thought should be applied to everything. Some types of mushrooms offer a lot of potential for medication development. If the presence of psilocybin in psilocybin mushrooms can be further identified [25].

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