Mushroom Poisoning and Treatments

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Abstract. The poisoning mushroom can affect the human body and is even lethal. This review summarizes gastroenteritis, hemolytic and liver damage caused by poisoning mushroom, as well as therapy methods for a variety of toxins. The main symptoms of gastroenteritis include acute nausea, abdominal pain, and diarrhea. For that, the common treatments include vomiting, gastric lovage and catharsis. The manifestation of hemolytic contains interic, acute anemia, liver adds spleen enlargement. The main approaches used to alleviate the effects are cortisone and atrium biacrbonate. Moreover, serious anemic people can use adrenal cortical hormones or Transfusion Therapy. The symptoms of liver damage are quite similar to gastroenteritis and hemolytic, but the manifestation is instruction regarding form doctors and over-the counter medicines. With prompt treatment, most minor symptoms can be cured. However, if the toxin has damaged the endoplasmic reticulum from liver nucleus, it would be difficult for patients to recover. Future research may focuse on precise treatment methods of mushroom positioning. More efforts should be made to facilitate the awareness of people about poisoning mushrooms to prevent mushroom poisoning.

Keywords: Mushroom poisoning, treatments, gastrointestinal, liver damage, hemolytic.

1. Introduction

Mushrooms contain many nutrients of human requirement, but some mushrooms which are found in the wild are toxic. It is hard to discern whether this mushroom is toxic by observing the appearance of mushrooms cause of they are similar by eyes. Thus, there are many people who poison in order to eat mushrooms in the world. The number of known mushrooms is approximately 14,000 around the world and 10 to 50 species have fatal toxin in them [1]. On average, 10-20g of raw mushrooms can cause people poisoning and 500g can kill people [2]. Thus, mushrooms poisoning is common in most of region of the world, such as China, North America, Russia, Italy, Japan [3]. Poisoning mushrooms may cause gastrointestinal toxicity, such as hallotoxing and anatoxins. There are some treatments of intaking these poisoning mushrooms. People can use Gastric lovage and oral dehydration to deal with gastrointestinal poisoning. In terms of hemolytic toxicity, corticosteroids and biological agents are the most suitable treatment i.e., azathioprine biological. Injecting Mercaptan detoxifying agents, N-acetyl cysteinc, large amouts of vitamin C, etc. are common treatments of liver poisoning. Thus, it is important to judge the toxicities of mushrooms and their treatments rapidly to the healthcare workers.

This review discusses the ordinary clinic symptoms and their treatments after mushroom poisoning, including, gastrointestinal, hemolytic and hepatic toxicities.

2. Gastrointestinal toxicity

Gastrointestinal toxicity is one of main toxicities of mushroom and it is usually appearing with others together as prodromal symptoms. Gastrointestinal symptoms happened in 90% of all symptomatic patients who eat toxic mushrooms [4]. The main features of it contains short incubation periods (10min-6h), high morbidity and low mortality rate.

2.1. Clinic symptoms

The common symptoms are caused by gastrointestinal toxicity including different gastrointestinal (GIT) effects, for example, vomiting, nausea, diarrhea, abdominal pain. Dehydration occurs due to the gastrointestinal fluid decrease (Fig. 1) [5]. Table 1 showed symptoms of associated dehydration. There are two cases about patients who ate the mushrooms and carrying gastrointestinal toxicity.

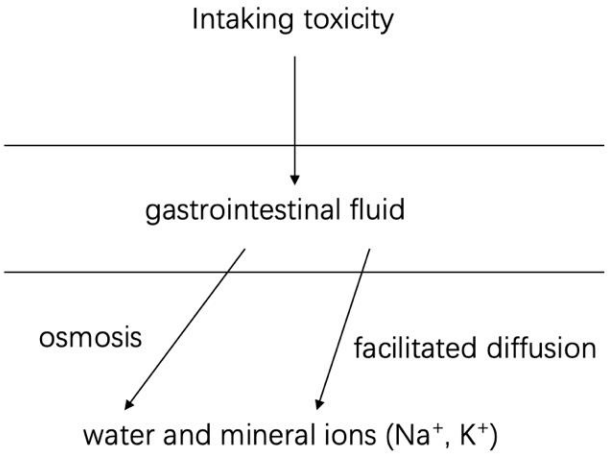

Figure 1. The mechanism of gastrointestinal dehydration

A case happened in 2008, Japan showed patients with hemorrhagic enteritis after ingestion of *Omphalotus guepiniformis* (Fig. 2), which was initially recognized as shitake (Fig. 2) [6]. In terms of appearance, they are extremely similar. They both have the brown fruit bodies and some white grains on them randomly. Their sizes are small, and they both have coarse and short stems. Thus, people usually eat them carelessly in the wild. The same thing happened in Fujian province, China. On November 24th and 26th, 2018, there were eight workers who work in Jiangyang District, Nanping City, Fujian Province poisoned due to intake poisonous mushrooms [7]. They appeared nausea, vomiting and abdominal pain after eating 10min-90min. There were two workers also poisoned due to intake the same poisonous mushrooms in two days. The sample of mushrooms were determined *Omphalotus guepiniformis* by morphological and molecular biological identification [7]. Then, researcher's further analysis revealed that the serving size of poisoning mushrooms was larger, the incubation period was shorter, and the duration of nausea and vomiting is longer than the patients who intake few poisoning mushrooms [7]. Both two poisoning incidents only cause GIT symptoms and they do not cause the damages for other organs.

Figure 2. Omphalotus guepiniformis [8] and Lenutinus edodes [9]

2.2. Treatments

Patients who poisoned gastrointestinal toxicity take medicines, such as lithium, digoxin, anti-arythmic drugs within 1-3 hours after onset of symptoms and dehydration improves [10]. The other treatments include gastric lavage, inducing vomiting and oral rehydration therapy. The treatments of the second example above were gastric lavage, inducing diarrhea, acid suppression and stomach protection and oral rehydration. Patients who had been poisoned were healed within 1-3 days of receiving treatments [7]. The function of gastric lavage, inducing diarrhea and vomiting both to eliminate the toxic residual in stomach and intestines. These methods also prevent the absorption of toxicity. The aim of oral rehydration is the replacement of fluids and electrolyte to prevent dehydration [5].

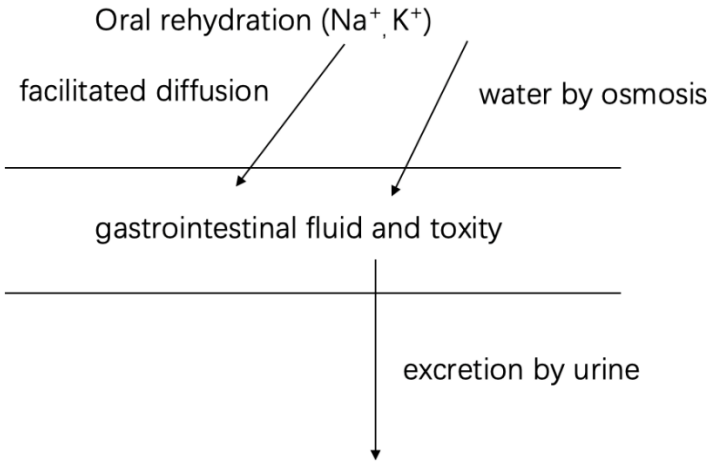

Figure 3. The mechanism of oral rehydration

If there were no professional detoxification drugs and devices, surround people must administer first aid to a poisoned person immediately. On the one hand, if the poisoned people have conscious, ask them to drink a large amount of water. This way can dilute the toxicity in human body and delay the onset of poisoning. Poisoned people should consume small amounts of water with salt and sugar in order to replenish lost electrolytes. On the other hand, the poisoned patients have been in coma and cannot swallow when they are unconscious. This method can cause the poisoned people asphyxiation [13]. There are five levels of disorders of consciousness and people depend on the right level of them to take the right first aid approach (Table 2).

Table 1. Symptoms of associated dehydration [11, 12]

Symptoms	Minimal or no dehydration (<3% loss of body weight)	Moderate dehydration (3%-9% loss of body weight)	Severe dehydration (>9% loss of body weight)
Mental status	Well; alert	Normal, fatigued or restless, irritable	Apathetic, lethargic, unconscious
Thirst	Drinks normally, might reject liquids	Thinsty; eager to drink	Drinks poorly; unable to drink
Heart rate	Normal	Noramal to boosted	Tachycardia
Breathing	Normal	Normal; fast	Deep
Eyes	Normal	Slightly sunken	Deeply sunken
Skin fold	Instant recoil	Recoil in <2 seconds	Recoil in >2 seconds
Extremities	Warm	Cool	Cold; mottled; cyanotic
Urine output	Noraml to declined	Delined	Minimal

Table 2. Five levels of disorders of consciousness

Level of disorders of consciousness	Features
Somnolence	The level of consciousness decreases, mental depression, reduction in movement. Patients maintain continuously asleep, but they can be wakened and answer the questions rightly. When the stimulus stops, the patients goes back to sleep.
Stupor	The level of consciousness is reduced compared to the former. The patients need loud shouting or stronger pain to awaken and their expression in blank. They cannot answer questions clearly.
Shallow coma	The patients there is a response to severe pain, a few spontaneous movements are present, corneal reflex, pupil-to-light reflex, gag reflex present in their body and inhibition reaches the cortex.
Moderate coma	The patients no response to pain, corneal reflex, pupil-to-light reflex, swallowing reflex present but all declined. Inhibition reaching subcutaneous.
Severe coma	The pupils of patients are dilated, their eyes are fixed and all reactions have disappeared. There is impaired respiratory and thermoregulation. Inhibition reaches the brainstem.

3. Hemolytic toxicity

Myotoxicity is one of hemolytic toxicity and there are two types of it: rapid onset and delay on set.

The rapid onset of myotonic is usually caused by a carboxylic acid, cydroprop-2-ene carboxylic acid [14]. For example, 7 patients of one family poisoned by eating *Russell subigricans Hongo.* One of them dies due to rhabdomyolysis. Many causing toxicities of delay onset is unknown. The new research shows that *saponaceolide B and M* are the main myotoxicity, but people do not this myotoxicity whether or not cause people poisoning [15].

3.1. Symptoms

In terms of rapid onset of myotoxicity, GIT is one of the prodromal symptoms, including serious vomiting, nausea and diarrhea within 2 hours after intaking cycloprop-2-ene carboxylic acid [16]. When the peak CK is more than 200,000 IU/l, rhabdomyolysis appears [17]. It is a series of striated muscle damages that include the cell surface membrane changing completely, myoglobin, creatine kinase, and other substances being lost, and so on. Rhabdomyolysis is characterised by muscle pain, swelling, and weakness, as well as fever and general weakness [18]. For the delay onset of myotoxicity, the case showing the yellow Tricholomoa glavoriens can cause delay onset of human rhabdomyolysis. People eat the mushrooms which has a few myotoxicities continuously 1-3 days and onset of symptoms contains delayed fatigue and myalgia [19].

3.2. Treatments

There are three methods to treat haemolytic toxins. The accustomed administration is bed rest and patients also can drink liquid salts as appropriate or intravenous fluids to correct water and electrolyte imbalance [20]. The second is antimicrobial therapy. The patients who have moderate disease normally do not require antimicrobial drugs, but the patients with severe symptoms can use gentamicin, amikacin and norfloxacin etc [20]. The third process is symptomatic treatment [20]. The analgesics are possibly given as appropriate when patients feel abdominal pain. For patients with shock, they can use vasoactive drugs as appropriate, except replenishing blood volume and correcting acidosis.

4. Liver toxicity

Toxicity is one of the major causes of muschroom poisoning induced death.

The main toxic is amatoxins with α-amanitin, β-amanitin, γ-amanitin, ε-amanitin, amanine and amanulin [21]. In addition, the phallotoxins also include phallisin, phalloidin, phallisin, phallicidin and phallinB. The toxins can directly destroy the liver nucleus so that cells rapidly damage, and the toxic mainly act on the endoplasmic reticulum of cell [21]. In addition, the toxic peptides act rapidly compared with amatoxins, but the toxicity of amatoxins is higher than the toxic peptides. And the devastating dose of amatoxins is less than 0.1mg/kg body weight, but the toxic peptides have 2mg/kg body weight [21]. Thus, the amount of amatoxins approximately 50g results from a healthy adult dying. Moreover, amatoxins are soluble in water, which may cause opportunities for poisoning to be raised.

4.1. Symptoms

The incubation period, gastroenteritis period, visceral stage, psychotic phase, and convalescence period are the six stages of liver injury [21]. The incubation period usually is found six hours even if one to two days. Result in the symptoms can't be timely found when people incautionsly eat posioning mushrooms. The gastroenteritis period may appear nausea, emesis, celialgia and diarrhea. These symptoms usually disappear after one to two days but may also be lethal. The latent period, when the symptoms of gastroenteritis disappear, patients have good spirits and don't have obvious symptoms. This moment the toxin enters into the liver or other organs through blood, then the organs are damaged. After latent period, the patient may turn into visceral damage stage. There is a significant amount of damage to the crucial organs such as heart and brain injury. Liver symptoms are among the most severe conditions, for example Acute Hepatopancreatic Necrosis and Hepatic Coma. Patients who severe poisoning rapidly access psychotic symptoms such as mania and convulsions, and going into coma, or even dying quickly from shock due to respiratory failure during the gasteroenteritis period. The final period is the recovery period, the symptoms of poisoning gradually diminish and the liver damage gradually improves. It generally takes 10 to 15 days of active treatment for liver function to totally return to normal [21].

4.2. Treatments

The treatment that a combination of therapeutic measures can be taken, with timely treatment focusing on liver protection [21]. Medication can also be used, such as (1) Mercaptan detoxifying agents such as sodium dimercaptopropionate, sodium dimercaptosuccinate, etc. The sodium dimercaptopionate at a does 0.25g, intramuscular injection1to2 times one day. A course of treatment needs three to fiive days and also need an interval of three to four days during treatment [22]. The sodium dimercaptosuccinate at a dose 1g, intravenous injection 1 to 2 times one day and course of treatment and interval are simliar as sodium dimercaptopionate [22]. (2) Adreno cortico hormones are hormones that are produced by the adrenal glands. Adrenocorticotropic hormones have the following side effects: they promote protein decomposition and inhibit protein synthesis, resulting in a negative nitrogen balance; they can increase calcium and phosphorus metabolism and have an anti-vitamin D effect, affecting calcium absorption; long-term use can also inhibit the vitality of bone cells, resulting in impaired bone formation, which can lead to osteoporosis and even fractures; and they can increase calcium and phosphorus metabolism and have an anti-vitamin Furthermore, adrenocorticotropic hormone inhibits bone development and protein synthesis by acting as an anti-growth hormone. Cushing's syndrome is caused by long-term use of high doses of adrenocorticotropic hormones, which can cause growth and development problems in children as well as dwarfism. (3) N-acetyl-L-cysteine. There are two ways to use N-acetyl-L-cysteine. The first method is spray. This can only be used in non-emergency situations. spray inhalation with 10 percentage solutions, one to three ml each day and two to three times one day. The second method is tracheal drip. This is suitable for use in emergency situations. se a 5-percentage solution via tracheal intubation or directly into the

trachea, one to two ml each day and two to six times one day. But this medicine has side effects such as nausea, vomiting, constipation or diarrhoea, if drugs are used excessively. (4) thiozincic acid (5) decoction of Ganoderma lucidum; (6) silymarin. This medicine is oral. In three divided doses, provide 2.5-20 grammes each day. Each therapy course lasts four weeks [23]. (7) large amounts of vitamin C and B vitamins [21]. At the same time, the patient also uses medicine that protects the liver such as Hepatite, Heparin, Inosine & Sodium Inosinate, Energy Combination, etc. [21].

4.3. Cases

There's a well-known case of a person being poisoned by lethal Amanita phalloides poisoning from preserved mushrooms. He was awake and completely alert at admission, and his vital signs were all normal. Except for dehydration, his physical examination was ordinary [24]. Using a polymerase chain reaction, the DNA of the hepatitis B virus was discovered to be Inverse. A patient was admitted to a private intensive care unit for three days. 50 grammes of activated charcoal were administered every 6 hours after a nasogastric tube was placed to lavage the stomach. The unwell person was rehydrated intravenously (iv) with 0.9 percent sodium chloride and 5% dextrose to avoid hypoglycemia. A bolus infusion of 5mg/kg silibinin was started at the same time, followed by a 20 mg/kg/day continuous infusion. Acetylcysteine (150mg/kg over 1 hour, 50mg/kg over 4 hours, and 150mg/kg over 16 hours) was given intravenously continuously for 21 hours at a budget dose of 300 mg/kg. A daily infusion of penicillin G (1,000,000 U/kg) and alpha lipoic acid, as well as a multivitamin, were given to the patients [24].

The other case of pediatric acute liver failure in northern China was due to mushroom poisoning [25]. He was able to recover as a result of a liver transplant. A previously healthy 9-year-old child developed nausea, vomiting, jaundice, and coma after consuming mushrooms for 5 days. Mushroom poisoning and abrupt liver failure were the causes of his condition. His condition worsened despite cautious treatment. He had LT on the seventh day after positioning due to grade IV hepatic encephalopathy [28]. He was discharged after twenty days of recovery. This story shows that poisonous mushrooms are harmful to human health.

The poisoning caused by Amanite phalloides-type mushrooms has been well-documented as being fatal [26]. Amanita phalloides and Amanita verna intoxicated four family members on August 21, 1991. The person who died had liver and kidney failure. Among the survivors was a pregnant woman in her 23rd week of pregnancy. Clinical signs and symptoms (lowest prothrombin activity 23%), as well as blood chemical results, suggested middle austerity intoxication. The patient was cured with intravenous hydration, forced diuresis, and the administration of high-potency penicillin, thioctic acid, hydrocortisone, vitamin K, and fresh frozen plasma, in addition to the administration of high-potency penicillin, thioctic acid, hydrocortisone, vitamin K, and fresh frozen plasma [26].

Three individuals took a high dose of Amanita in March 2019, and one of them had a liver transplant at Sun Yat-sen University's Zhongshan hospital. [27]. All patients all appear vomiting and diarrhea after consuming wild mushrooms within 8 to 12 hours. These patients were identified with amanita poisoning at the beginning. There was one case that was so complicated that he was diagnosed with mushroom poisoning. (amanita fatalis), acute liver failure, toxic encephalopathy, hemorrhagic colitis, toxic myocarditis, disseminated intravascular coagulation, and pregnancy are all possible side effects [27]. One instance was very complex, and he was eventually diagnosed with mushroom poisoning. Acute liver failure, toxic encephalopathy, hemorrhagic colitis, toxic myocarditis, disseminated intravascular coagulation, and pregnancy are all possible side effects of the mushroom (amanita fatalis) [27]. One case improved significantly in the indexes of liver, kidney, coagulation function, and infection after liver transplantation as a result of the therapy. In two other cases of intracerebral haemorrhage, it was also fatal. According to this case, liver transplantation may improve the chances of survival for patients suffering from noxious liver failure caused by acute liver failure caused by mushroom poisoning.

5. Conclusion

The poisoning mushroom includes lots of toxins and mushroom poisoning has been showing a high mortality. This review introduces various types of poisoning when consuming poisonous mushrooms and the treatment for different toxins. On the other hand, medicines are major methods for toxic phenomena. The gastrointestinal toxicity frequently appears in prodromal symptoms. The major character only has short incubation periods with high morbidity and low mortality rate. The treatment generally reveals medicines which are lithium etc. Another therapy is gastric lavage. The hemolytic toxicity is accelerated onslaught and prolonged outbreak, and the symptoms are vomiting which is similar to gastrointestinal. Myotoxicity is rapid onset, the treatment takes immunosuppressive agents. The last one liver toxicity chiefly caused by amatoxins and symptoms has six different periods. The medicines which mercaptan detoxifying agents and adrenocorticosteroids are used. Future research may focus on the methods to recognize that mushroom poisoning, as well as exploring efficient treatment.

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