

Chlorophyllum Molybdites Poisoning in Singapore

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Abstract

Chlorophyllum molybdites is one of the most common causes of mushroom poisoning worldwide. The majority of cases are from consumption of misidentified mushrooms while foraging. To date, there have been no reported cases of mushroom poisoning in Singapore.

We describe the first reported case of Chlorophyllum molybdites poisoning in Singapore who presented to the emergency department with acute gastroenteritis, complicated by hypovolemia and several laboratory abnormalities, including lactic acidosis, deranged liver enzymes and elevated creatine kinase. The patient received supportive treatment with intravenous fluids, analgesia and anti-emetics. The patient made a complete recovery with the resolution of all symptoms and laboratory abnormalities after 48 hours.

Chlorophyllum molybdites is known to be a gastrointestinal irritant, and ingestion is often associated with a rapid onset of profound gastrointestinal symptoms, including abdominal cramps, nausea, vomiting and diarrhea. Treatment is mainly supportive, and the majority of patients improve rapidly within 24 hours although children and the elderly can be more susceptible to complications such as dehydration and electrolyte abnormalities.

However, Chlorophyllum molybdites can be confused for other species of more toxic mushrooms, such as amatoxin-containing Lepiota, which can present similarly with acute gastrointestinal symptoms but subsequently develop severe delayed toxicity such as hepatic failure and multisystem organ failure. Hence clinicians managing patients with mushroom poisoning should be aware of the potential for delayed toxicity. Close monitoring for at least 24 hours is recommended for all cases of mushroom poisoning, especially if there is doubt regarding the identity of the mushrooms ingested or if there was ingestion of multiple species of mushrooms.

Keywords: Chlorophyllum molybdites; mushroom poisoning; Singapore

Introduction

Chlorophyllum molybdites is a well-known cause of mushroom poisoning worldwide. It is known to be a gastrointestinal irritant, producing significant nausea, vomiting, abdominal cramping and diarrhea within hours of ingestion [1].

The majority of cases are from consumption of misidentified mushrooms by foraging adults [2].

Chlorophyllum molybdites poisoning has been reported across an extensive geographic distribution, including the USA, South America, Europe, Australia and Asia [5].

Chlorophyllum molybdites can be mistaken for more dangerous amatoxin-containing mushroom species, and additionally, the initial signs and symptoms from Chlorophyllum molybdites poisoning can also resemble poisoning from amatoxin-containing mushroom species.

Singapore has a tropical climate and is home to a wide variety of mushrooms, several of which are considered toxic. To date, there has been no previous reported cases of mushroom poisoning reported from Singapore.

We describe the first reported case of Chlorophyllum molybdites poisoning in Singapore.

Case Report

A 55 year-old woman presented to the Emergency Department of Khoo Teck Puat Hospital, Singapore at 3 am with chief complaints of abdominal pain associated with profuse vomiting and diarrhoea.

She complained of severe, generalised crampy abdominal pain, with multiple episodes of non-bilious, non-bloody vomiting and watery non-bloody diarrhoea.

The patient ingested the cooked mushrooms around 12 midnight and her symptoms started acutely around 1 hour later.

The patient reported that she had foraged and picked the wild mushrooms growing around her neighbourhood. The patient worked as a gardener and had shown pictures of the mushroom to another gardener friend beforehand, who claimed that the mushrooms were edible as he had eaten the same mushrooms previously.

She had cooked around 20 grams of the foraged mushroom by stir-frying with butter and garlic, and reported that the mushrooms tasted pleasant.

She had shared a meal with her family members, however she was the only one who ate the mushrooms.

She did not report chest pain, breathlessness or any other signs of cholinergic toxicity.

Her past medical history was unremarkable and the patient was not consuming any chronic regular medications.

The patient was able to provide photos of the mushrooms she had eaten which are shown below.



Figure 1: Photos of the ingested mushrooms provided by the patient

Physical Examination

At presentation, her vital signs were: blood pressure of 128/100 mmHg, pulse rate of 80 beats per min (bpm), respiratory rate of 18 breaths per minute and a peripheral oxygenation saturation (SpO₂) of 100% on room air. Patient was afebrile with a temperature of 36.3 °C.

On physical examination, the patient was alert and oriented with a Glasgow Coma Scale score of 15. However, the patient was diaphoretic and uncomfortable from her ongoing abdominal cramps, nausea, vomiting and diarrhoea.

The patient was clinically dehydrated with dry mucus membranes on examination.

Pupils were of normal size, equal and reactive. Breath sounds were normal and no cardiac murmur was heard on auscultation.

Her abdomen was soft but with generalised tenderness on palpation. Bowel sounds were active. There was no abdominal distension, guarding or rigidity.

Her neurological examination was normal with no lateralising signs.

The prominent and acute onset of gastrointestinal symptoms such as abdominal tenderness combined with the reported history of prior mushroom ingestion led the medical team to suspect mushroom poisoning.

Investigations

An electrocardiogram (ECG) was done on presentation which showed sinus tachycardia with no other abnormalities.

Laboratory investigations were done and are shown in Table 1.

The patient had several mild laboratory abnormalities, including mildly raised creatinine of 82 $\mu\text{mol/L}$, urea of 7.2 mmol/L , corrected calcium of 2.52 mmol/L , amylase of 110 U/L , creatine kinase of 222 U/L , and AST of 49 U/L , ALT of 66 U/L .

A venous blood gas was performed which showed a mixed picture of metabolic acidosis and metabolic alkalosis, with a component of respiratory alkalosis as well.

A venous lactate was also performed which was elevated at 3.2 mmol/L .

Intravenous access was established and IV fluid resuscitation was performed with 500mls of normal saline over 30 minutes. Subsequent fluid resuscitation was continued with 1 liter of IV Lactated Ringer's solution for a total of 1.5 liters of IV crystalloids given over 3 hours.

IV metoclopramide 10mg was given as an anti-emetic and IV tramadol 50mg was given for analgesia as well.

Table 1: Laboratory Investigations of the Patient on Presentation

Test	Result	Reference Values
Hemoglobin (g/dL)	14.2	11.2 – 14.9
White Blood Cells	10.01	$3.82 - 9.91 \times 10^9$
Platelets	314	$173 - 414 \times 10^9$
INR	0.96	
PT (seconds)	10.5	9.7 – 11.6
APTT (seconds)	22.8	25.5 – 35.8
Urea, Serum (mmol/L)	7.2	2.4 – 6.6
Creatinine, Serum ($\mu\text{mol/L}$)	82	44 – 79
Bicarbonate, Serum (mmol/L)	22	23 - 29
Sodium, Serum (mmol/L)	144	135 – 145
Potassium, Serum (mmol/L)	4.2	3.5 – 5.1
Magnesium, Serum (mmol/L)	1.0	0.6 – 1.1
Calcium, Serum (mmol/L)	2.89	2.15 – 2.50
Calcium, Corrected (mmol/L)	2.52	2.15 – 2.50
Phosphate, Serum (mmol/L)	0.86	0.85 – 1.45
Chloride, Serum (mmol/L)	103	96 - 108
Liver Function Test		

Total Protein, Serum (g/L)	94	63 – 83
Albumin, Serum (g/L)	59	35 – 50
Bilirubin, Total (µmol/L)	8	3 – 21
AST (U/L)	61	10 - 30
ALT (U/L)	74	10 - 36
ALP (U/L)	99	22 - 104
Creatine Kinase (U/L)	222	24 – 200
Lactate, venous (mmol/L)	3.2	0.6 – 1.4
Venous Blood Gas		
pH	7.548	7.310 – 7.410
PCO ₂ (mmHg)	27.3	41.0 – 51.0
PO ₂ (mmHg)	24.0	30.0 – 55.0
Base Excess (mmol/L)	1.0	-2.0 – 3.0
Bicarbonate (mmol/L)	23.8	23.0 – 28.0
O ₂ Saturation (%)	54	40 – 85

AST, Aspartate Transaminase; ALT, Alanine Transaminase; ALP, Alkaline Phosphatase; INR, international normalized ratio; PT, prothrombin time; APTT, activated partial thromboplastin time;

The patient improved dramatically within a few hours, with near resolution of her abdominal cramps and nausea. The patient was admitted to the medical general ward for further monitoring and continued to improve over the next 48 hours with symptomatic medical management. The patient made a complete and uneventful recovery and was discharged from the hospital on her 2nd day of admission with complete resolution of her gastrointestinal symptoms and initial biochemical abnormalities.

Discussion

Mushroom poisoning is an important cause of intoxication worldwide, with approximately 6000 to 8000 mushroom exposures annually in the United States³ and an estimated 100 deaths/year in Europe alone [4].

The majority of cases are from consumption of misidentified mushrooms while foraging.

The warm and humid tropical climate of Singapore supports a wide variety of flora and fauna, such as more than 150 different species of mushrooms, including poisonous species such as *Lepiota* and *Chlorophyllum molybdites*. However, there has been no prior previously reported cases of any mushroom poisoning in Singapore.

Mushroom poisoning has been classified into 14 described clinical syndromes determined by their predominant symptoms and toxins.

Chlorophyllum molybdites, also known as *Lepiota morgani*, *Chlorophyllum esculentum*, and *Lepiota molybdites* is one of the most common cause of mushroom poisoning in North America [5]. It is also known by other names, some of which include false parasol, green-spored parasol, green-spored *lepiota* and the vomiter.

When mature, *Chlorophyllum molybdites* can be recognized from its characteristic green spores and gills, often with a cap that

ranges from 7 – 30 cm. The appearance of the cap can also vary from being broad and convex, to being knobbed or flat. The stalk is often 10-25 cm long, and 10-25 mm thick.

However younger specimens are associated with white spores, making them difficult to differentiate from other edible species. In addition, the cap is often dotted with brown scales, which resemble the scales of the edible *Macrolepiota procera*.

Cases of *Chlorophyllum molybdites* poisoning are often due to misidentification of *Chlorophyllum molybdites* for other edible species such as *Macrolepiota procera* (parasol mushroom) [15].



(A)



(B)

Figure 2: Comparison between *Chlorophyllum molybdites* (A) and *Macrolepiota procera* (B) Photos by Michael Kuo.

Chlorophyllum molybdites belongs to the group of toxic mushrooms associated with prominent gastroenteritis symptoms. Ingestion of *Chlorophyllum molybdites* is associated with rapid onset of prominent gastrointestinal symptoms within 1-3 hours of ingestion, including abdominal cramps, nausea, vomiting and diarrhea. In severe cases, diarrhea may be bloody [7].

The time from ingestion to onset of symptoms is important, as delayed onset of gastrointestinal symptoms (6 – 24h post-ingestion) is suggestive of the ingestion of amatoxin-containing mushrooms, which are significantly more toxic. However, it is worth noting that an early onset of gastrointestinal symptoms does not rule out ingestion of amatoxin-containing mushrooms, especially if there was ingestion of multiple species of mushrooms.

Chlorophyllum molybdites remains toxic even after being cooked, with numerous reports of toxicity after eating cooked mushrooms [8].

The toxins and associated mechanisms of action responsible for the gastrointestinal effects of *Chlorophyllum molybdites* are not clearly understood.

An in-vitro study [17] by Sai Latha et al, 2018 showed significant intestinal cell toxicity such as cell shrinkage, vacuolation, and distortion in cell shape upon exposure to extracts of *Chlorophyllum molybdites*. This correlates to the classical clinical presentation of severe gastrointestinal symptoms associated with *Chlorophyllum molybdites* poisoning.

Another study [9] by Yamada et al, 2012 isolated a toxic protein, Molybdophyllysin, suspected to play a role in the gastrointestinal effects of *Chlorophyllum molybdites* toxicity. The protein was able to maintain its thermostability despite being exposed to temperatures up to 70 °C for 10 mins, explaining the observation that *Chlorophyllum molybdites* maintains its toxicity even after being cooked.

The diagnosis of *Chlorophyllum molybdites* poisoning is made on the basis of a history of *Chlorophyllum molybdites* ingestion and the usual clinical features of rapid and prominent gastrointestinal symptoms.

There is no specific antidote for *Chlorophyllum molybdites* poisoning.

Gastrointestinal decontamination with activated charcoal can be considered for patients who present within 1 hour of ingestion of potentially toxic mushrooms. Enhanced elimination with multidose activated charcoal (MDAC) has not been shown to be effective in majority of mushroom poisoning, including *Chlorophyllum molybdites*, with the exception of amatoxin-containing mushrooms [10]. Extracorporeal removal of mushroom-related toxins such as hemodialysis has not been shown to be effective as well.

The mainstay of treatment is supportive, including symptomatic treatment and rehydration. Possible complications include hypovolemic shock from dehydration, renal insufficiency and electrolyte imbalances [11].

Although *Chlorophyllum molybdites* poisoning can cause severe symptoms in children [14] or the elderly, there has been no reported deaths from *Chlorophyllum molybdites* poisoning.

The majority of cases of *Chlorophyllum molybdites* poisoning respond well to supportive management, with rapid resolution of symptoms within 24h.

It is recommended that patients be admitted and monitored for at least 24h, especially if the identity of the ingested mushroom is not known and/or the patient had ingested multiple types of mushrooms.

Our patient was able to provide pictures of the mushrooms she had ingested, which aided in identifying the culprit mushroom species, however the species of mushroom is not known in up to 80% of mushroom-poisoning cases [2].

Singapore is also home to the *Lepiota* species, majority of which are known to contain amatoxin [13]. Ingestion of amatoxin-containing mushrooms can be associated with early onset of acute gastroenteritis syndrome mimicking the initial phase of less

toxic mushrooms (e.g. *Chlorophyllum molybdites*). Patients then show an apparent recovery of gastrointestinal symptoms, however patients subsequently develop fulminant hepatic and multiorgan failure [12] 48-96h after ingestion. Several toxic species of *Lepiota* are also known to morphologically resemble *Chlorophyllum molybdites* as well.

Given that the vast majority of mushroom-poisoning cases are due to misidentification, even by experienced pickers, it is strongly advised that the general public refrain from consuming wild mushrooms unless identified by an expert [16].

Conclusion

This is the first reported case of *Chlorophyllum molybdites* poisoning in Singapore. *Chlorophyllum molybdites* is one of the most common causes of mushroom poisoning worldwide, and is known to cause acute gastroenteritis soon after ingestion. Treatment is often supportive and majority of patients recover rapidly within 24-48 hours.

However *Chlorophyllum molybdites* can be confused for other mushroom species, such as *Lepiota* species, which are capable of producing acute gastrointestinal symptoms, followed by severe delayed toxicity. Hence the potential for delayed toxicity from the more toxic amatoxin-containing mushrooms must be considered when managing patients with suspected *Chlorophyllum molybdites* poisoning, especially if there is doubt regarding the identity of the mushroom and/or there was ingestion of multiple species of mushrooms.

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