A Case Report of *Chlorophyllum palaeotropicum* Mushroom Poisoning

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Abstract

Mushroom poisoning is an important yet underreported public health issue. We present a case report of acute *Chlorophyllum palaeotropicum* mushroom poisoning in a family of five, involving both adult and paediatric subjects. They exhibited mainly gastrointestinal symptoms with varying severity. Two of the patients sustained acute renal injury from profuse vomiting and diarrhoea whilst the rest had mild dehydration and required outpatient treatment alone. There was no fatality with all of the patients recovered without any complications after receiving timely supportive treatment. This report highlights the challenges in managing mushroom poisoning in a low-resource setting where mycological and toxin identification facilities are not readily available.

Keywords: chlorophyllum; palaeotropicum; mushroom; poisoning; Malaysia

Introduction

Mushroom poisoning is a recognised global public health problem.¹ It is estimated that more than a million species of fungi exist with at least 16,000 species of mushroom identified.² Most cases of mushroom poisoning occur because of inadvertent misidentification of edible species. This is not surprising considering that wild mushroom foraging is a common practice throughout...
the world. Most commonly reported source of poisoning include species such as *Amanita* spp., *Microporous* spp. *Lepiota* spp., and *Coprinus* spp. In Malaysia, mushroom poisoning is uncommon with majority of the reported cases linked to *Chlorophyllum molybdites* (CM) ingestion. Another closely related species known as *Chlorophyllum palaeotropicum* (CP) was classified as a distinct new species in recent years. To date, there are no CP poisoning cases reported in the scientific literature.

**Case Report**

A 41 year-old Malay lady with unremarkable medical history presented to the emergency department in September 2022 complaining of more than 10 episodes of watery stool and vomiting, mild central abdominal discomfort and giddiness after allegedly consuming a home-cooked “tom yum” soup dish about 2 hours prior to onset of symptoms. The dish contained some wild mushrooms foraged by the husband a day earlier from the school garden where he works as a school gardener. She claimed that they were thoroughly cooked. Four of her children aged 13, 11 and a pair of twins aged 10, all with unremarkable past medical history, also presented alongside her with similar gastrointestinal (GI) symptoms. They have consumed the mushroom at almost identical time during lunch and experienced similar symptom onset. None of them had any bloody stool, fever, chest or neurological symptoms.

The raw leftover mushrooms were obtained from the mother and promptly dispatched to the Mushroom Research Centre at Universiti Malaya for analysis [Figure 1]. The culprit mushroom was suspected to be CM considering the gross morphology, local epidemiological context and the rapid onset of GI symptoms within 3 hours of ingestion.

When examined, the mother appeared lethargic. Her blood pressure (BP) was slightly low at 99/62 mmHg and tachycardic (122 bpm). She was commenced on intravenous (IV) normal saline 5 pints/24 hours for rehydration and oral activated charcoal. Initial blood tests showed raised total white cell (TWC) of 23.1 x 10⁹/L (reference range: 4-11) and creatinine at 139 umol/l (reference range: 50-98). Her venous blood gas (VBG) showed mild metabolic acidosis (pH: 7.29) with normal plasma lactate. Liver function test (LFT) however was normal. She was admitted to the acute medical ward with strict fluid status monitoring. After 24 hours, her
symptoms resolved with normal BP (110/70 mmHg) and she was able to tolerate fluid well. Her blood tests repeated were normal.

Similar to the mother, the eldest child also appeared lethargic on arrival. His BP was normal but had slight tachycardia (126 bpm). His mucosa appeared dry with normal skin turgor. IV normal saline/dextrose 5% (NSD5) was commenced at 4 pints/24 hours to cover for the fluid deficit and daily maintenance. Initial blood tests showed elevated TWC (13.8 x 10^9/L) and creatinine at 105 umol/L (reference range: 46-72) with a normal LFT. His VBG showed slight metabolic acidosis (pH: 7.27). After 24 hours of rehydration, his symptoms had diminished and blood tests repeated were normal. Both the mother and the child were discharged within 48 hours of presentation after satisfactory oral intake was ensured.

The other three children displayed milder symptoms. At presentation, their vital signs were stable. Although the renal profile/LFT were normal, all had leukocytosis (range: 13.6–23.6 x 10^9/L). IV NSD5 was administered to replace ongoing fluid loss. Within 6 hours, their symptoms had reduced substantially. By 12 hours of presentation, adequate oral intake was established and they were able to be discharged. Table 1 provides a summary of the salient clinical findings and treatment for all the patients involved.

A follow-up visit arranged at a local health clinic found that the patients have recovered well without any complications. The mycological report obtained about 3 weeks later identified the culprit mushroom as the young stage of CP.

**Discussion**

Mushroom poisoning or mycetism is increasingly being reported worldwide each year.\(^6\) *Chlorophyllum* mushroom is widely distributed throughout the world with a predilection for tropical and subtropical regions.\(^5\) The mushroom is often mistaken for certain edible look-alike species such as *Termitomyces* spp. (termite mushrooms, also known as cendawan busut in the local Malay language) and *Macrolepiota procera* (parasol mushroom).\(^3,6,7\) Misidentification of edible mushroom is the most common cause of mushroom poisoning,\(^1\) as exemplified in this report. There is no fail-safe way to differentiate between edible and poisonous species.\(^1,4\) In
addition, the same species may exhibit morphological variations depending on the season, geographical location and the maturity of the mushroom.\(^1\)

In this report, all of the subjects started having GI symptoms within 2-3 hours of ingestion. This resembles symptom profile of CM poisoning, a notorious GI irritant.\(^4,7\) The exact mechanism of CM toxicity is unknown, but a protein isolated from CM known as molybdophyllysin is thought to be responsible for its GI toxicity.\(^7,8\) Molybdophyllysin can maintain thermostability after heating for 10 minutes at various temperatures between 30-70 °C.\(^7\) The thermostability of the CP toxins in this case is uncertain, as the reliability of the mother’s claim that the meal was thoroughly cooked is questionable given that the cooking duration is unknown and the mushrooms could have been added at any point of the cooking process. Despite comparable GI toxicity, it is uncertain if CP contains similar toxin as in CM because different species of mushroom within the same genus may have varying toxin content.\(^9\)

Distinguishing between CM and CP morphologically is extremely challenging. It is not until recent years that CP was classified as a separate species, made possible through molecular phylogenetic analyses.\(^5\) Hence, the possibility that some of the earlier literature on CM poisoning may have actually involved CP cannot be ruled out. Apart from GI symptoms, autonomic nervous dysfunction such as altered perception, dilated/pinpoint pupils, dizziness, blurry vision, lacrimation, salivation and tachycardia have been reported in CM poisoning in addition to electrocardiographic abnormalities such as T-wave inversion. Haematological abnormalities such as leukocytosis, bleeding and disseminated intravascular coagulation may also occur. Severe dehydration from GI symptoms may lead to acute renal insufficiency and electrolyte imbalance or even hypovolaemic shock.\(^7\) In comparison, all of the subjects reported here had leukocytosis whilst two sustained transient renal impairment from protracted GI symptoms that resolved with adequate rehydration.

Diagnosis of mushroom poisoning ideally should entail the identification of the species ingested, determination of the time interval between ingestion and symptom onset (latency period) and laboratory toxin analysis (if available).\(^10\) Formal macroscopic and microscopic examination of the mushroom or digestive samples by trained expert may allow quick and reliable identification
of the species.\textsuperscript{7,10} This facility however is not readily accessible in resource-constrained settings,\textsuperscript{11} resulting in limited clinical use apart from providing retrospective confirmation as in this case. Toxin analysis has limited value as well because most mushroom toxins are not well-characterized except for detection of amatoxin (the main toxin contained in \textit{Amanita phalloides} which causes 90\% of all lethal cases) in serum or urine.\textsuperscript{10} As a result, provisional diagnosis often is established on a clinical basis to avoid unnecessary delay in treatment.\textsuperscript{10,11}

Diaz previously proposed a syndromic approach to mushroom poisoning based on latency period and the predominant target organ toxicity to facilitate earlier diagnosis and treatment.\textsuperscript{12} More recently, Wennig classified ten of the most important poisoning syndromes encountered clinically into short latency (< 6 hours) and long latency (> 6 hours).\textsuperscript{10} Such syndromic classification may serve as a helpful guide when prompt mycological identification is not available [Table 2]. Short latency is classically associated with limited toxicity and milder clinical course compared to longer latency (> 6 hours) which is suggestive of a more lethal poisoning.\textsuperscript{10,11} This handy classification may serve as a general guide rather than an infallible rule as a short latency does not always preclude lethal amatoxin poisoning such as in the instance of a mixed mushroom ingestion.\textsuperscript{10} In this case, the diagnosis of a probable \textit{Chlorophyllum} poisoning was established based on the gross morphology of the mushroom, the prominent GI irritant syndrome and the short latency period.

The GI syndrome caused by \textit{Chlorophyllum} poisoning typically resolves within 48 hours with good supportive care which mainly include aggressive rehydration and GI decontamination.\textsuperscript{4,7} Our experience here suggest that CP poisoning may be approached and managed clinically in a manner similar to CM poisoning. As illustrated by this case, fluid and electrolyte replacement should not be delayed in \textit{Chlorophyllum} poisoning particularly in children and fragile patients.\textsuperscript{7} This is because prolonged dehydration from protracted GI symptoms may risk complications such as acute renal insufficiency, electrolyte imbalance and hypovolaemic shock.\textsuperscript{4,7}

In this report, there was no fatality and none of the patients developed serious or long-term complications. This is comparable to CM poisoning which is rarely fatal.\textsuperscript{4} However, mushroom poisoning may occasionally lead to organ failure and even death.\textsuperscript{13} Patients may rarely end up
with acute liver failure and require liver transplantation as a lifesaving procedure. Overall mortality rate for mushroom poisoning is estimated at 8 to 12% and significantly higher (up to 30%) if Amanita spp. is involved. The single most important strategy to prevent mushroom poisoning is to educate the public especially the at-risk population to avoid consuming wild mushrooms or at least to keep a sample or take a photograph of the raw mushrooms prior to consuming them. Further research to characterize the toxins in CP is necessary to provide insight on the actual mechanism of toxicity. The major limitations of this study include the small number of subjects and the fact that the possibility of a mixed mushroom poisoning cannot be ruled out.

Conclusion

Chlorophyllum palaeotropicum poisoning causes severe gastrointestinal toxicity and has a short latency period. With timely supportive care, speedy and complete recovery is achievable. Clinicians can make use of the gross mushroom morphology, the prominent gastrointestinal symptoms and presence of short latency period to help establish a provisional diagnosis of Chlorophyllum poisoning in a resource-constrained setting where rapid mycological and toxin analysis may not be readily accessible. This will avoid unnecessary delay in treatment initiation and improve outcome.

Consent

Written informed consent was obtained from the patient/guardian for their anonymized information to be published in this manuscript.

Authors’ Contribution

MRZ, HHS and HH conceived the idea. All authors were involved in the design, data interpretation and drafting of the manuscript. All authors approved the final version of the manuscript.
References


Figure 1: Morphology of the mushroom sample depicted here as having a hemispherical cap (black arrow) and light brown scales/squamulose on the surface. The stipe (white arrow) appears smooth with an enlarged base.
Table 1: Key clinical features and management for all the patients.

<table>
<thead>
<tr>
<th>Subject (Age in years)</th>
<th>Symptoms</th>
<th>Onset from time of ingestion</th>
<th>Clinical findings</th>
<th>Treatment</th>
<th>Laboratory findings</th>
<th>Time from admission to discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother (41)</td>
<td>Watery stool Vomiting Abdominal pain Dizziness</td>
<td>2 hours</td>
<td>Lethargy Tachycardia</td>
<td>Admitted to ward - oral activated charcoal - IV NS</td>
<td>Cr: 139 TWC: 23.1 VBG: pH 7.29 Lactate: N LFT: N</td>
<td>42 hours</td>
</tr>
<tr>
<td>Eldest Child (13)</td>
<td>Watery stool Vomiting Abdominal pain</td>
<td>2 hours</td>
<td>Lethargy Tachycardia</td>
<td>Admitted to ward - oral activated charcoal - IV NSD5</td>
<td>Cr: 105 TWC: 13.8 VBG: pH 7.27 Lactate: N LFT: N</td>
<td>42 hours</td>
</tr>
<tr>
<td>Second Child (11)</td>
<td>Watery stool Vomiting Abdominal pain</td>
<td>2 hours</td>
<td>Tachycardia</td>
<td>Not admitted - IV NSD5</td>
<td>TWC: 23.6 RP/LFT: N</td>
<td>12 hours</td>
</tr>
<tr>
<td>Twin A (10)</td>
<td>Watery stool Vomiting Abdominal pain</td>
<td>2 hours</td>
<td>Tachycardia</td>
<td>Not admitted - IV NSD5</td>
<td>TWC: 23.6 RP/LFT: N</td>
<td>12 Hours</td>
</tr>
<tr>
<td>Twin B (10)</td>
<td>Watery stool Vomiting Abdominal pain</td>
<td>2 hours</td>
<td>Tachycardia</td>
<td>Not admitted - IV NSD5</td>
<td>TWC: 13.6 RP/LFT: N</td>
<td>12 hours</td>
</tr>
</tbody>
</table>

✝ Cr: creatinine; TWC: total white cell count; VBG: venous blood gas; RP: renal profile; LFT: liver function test; N: normal; NS: normal saline; NSD5: normal saline/dextrose 5%.
### Table 2: Clinically important mushroom poisoning syndromes

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Latency</th>
<th>Clinical findings</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal irritant</td>
<td>Short, 30 min – 6 hours</td>
<td>Vomiting, diarrhoea, hypotension</td>
<td>Fluid/electrolyte replacement</td>
</tr>
<tr>
<td>Muscarinic poisoning</td>
<td>Short, 30 min – 2 hours</td>
<td>Vomiting, diarrhoea, miosis, diaphoresis, bradycardia, hypotension</td>
<td>Fluid/electrolyte replacement, activated charcoal, atropine</td>
</tr>
<tr>
<td>Panthercap / Fly agaric poisoning</td>
<td>Short, 30 min – 3 hours</td>
<td>Hallucinations, euphoria, myoclonus, seizures, mydriasis/miosis, tachy- or bradycardia, somnolence</td>
<td>Cardiovascular monitoring, benzodiazepines in agitated patients</td>
</tr>
<tr>
<td>Psilocybin poisoning</td>
<td>Short, 15-60 min</td>
<td>Hallucinations, euphoria, nausea, tachycardia, hypotension, mydriasis, headache, psychosis</td>
<td>Observation and benzodiazepines/neuroleptics if required</td>
</tr>
<tr>
<td>Coprine poisoning</td>
<td>Short, 15 min – 2 hours</td>
<td>Flushing, diaphoresis, tachycardia, nausea &amp; vomiting, hypotension</td>
<td>Cardiovascular monitoring, beta blockers if needed</td>
</tr>
<tr>
<td>Paxillus poisoning</td>
<td>Short, 60 min – 2 hours</td>
<td>Vomiting, diarrhoea, flank pain, hypotension, kidney damage and multiorgan failure in chronic ingestion</td>
<td>Activated charcoal, blood transfusion, may require haemodialysis</td>
</tr>
<tr>
<td>Amatoxin poisoning</td>
<td>Long, 6-24 hours</td>
<td>Vomiting, diarrhoea, hypotension, coagulopathy, encephalopathy, acute renal and liver failure</td>
<td>Fluid/electrolyte replacement, activated charcoal, blood products transfusion, antidotes (silibinin and N-acetylcysteine), may require haemodialysis and liver transplant</td>
</tr>
<tr>
<td>Gyromitrin poisoning</td>
<td>Long, 6-12 hours</td>
<td>Nausea, vomiting, impaired consciousness, seizures, liver and kidney damage</td>
<td>Activated charcoal, intravenous pyridoxine</td>
</tr>
<tr>
<td>Orellanine poisoning</td>
<td>Long, 36 hours – 17 days</td>
<td>Thirst, flank pain, weakness, oliguria/acute renal failure, tubulointerstitial nephritis</td>
<td>Steroids, haemodialysis</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
<td>Long, 1-3 days</td>
<td>Muscle pain, elevated creatine phosphokinase, arrhythmias, myocarditis, renal failure</td>
<td>Urine alkalinization, haemodialysis</td>
</tr>
</tbody>
</table>