Wild Mushroom Poisoning in North India: Case Series with Review of Literature

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Mushroom is an important constituent of diet in many ethnic tribes in India. Ethnic Indian tribes are known to consume nearly 283 species of wild mushrooms out of 2000 species recorded world over. Although they are experts in distinguishing the poisonous from edible mushrooms, yet occasional cases of toxicity are reported due to accidental consumption of poisonous mushrooms. We report amanita like toxicity in a family after consumption of wild mushrooms resulting in fatal outcome. (J Clin Exp Hepatol 2014;4:361–365)

Mushroom poisoning (aka mycetism) in humans has been described since time immemorial, which has been witnessed by ancient writings like "Rigveda" (at least 3500 B.C.) and "Atharvaveda" (at least 1500 B. C.) The first written record about a fungus is the death from fungal poisoning, of a mother, daughter and two full grown sons, an event, which Euripides (456–450 B.C.) commemorated by an epigram.[1]

In India, Mushroom has been a source of diet and article of commerce since long time and across many cultures. Poisoning results from unintentional consumption of poisonous wild mushrooms. The cases however remain undiagnosed, underreported and unpublished. A large number of suspected cases are reported in lay press. There have been small epidemics of mushroom poisoning culminating in mortality especially during monsoon. The published literature from India is sparse and mostly in the form of case reports.[2–4] The present case report depicts one such incident of accidental death of three members of a family, owing to consumption of wild mushrooms.

CASE REPORT

A family consisting of 35 yr old man, 33 yr old lady (mother), 14 yr old daughter and 13 yr old son residents of a village Trela, district Mandi, Himachal Pradesh, India consumed wild mushrooms harvested from mountains in September 2011. Subsequently all four of them developed pain abdomen, vomiting and bloody diarrhea 4–6 h after consumption. They were taken to community health center where intravenous (IV) fluids and anti-emetics were given and diarrhea and vomiting settled after 24–36 h and they remained relatively asymptomatic for 8–10 h. This was followed by progressive altered sensorium and behavior and irritability for which referred to district hospital Mandi, where found to have transaminitis >10 times elevated and jaundice. Provisional diagnosis of toxic hepatitis secondary to poisonous wild mushrooms was made. Patients were managed with IV fluids, antibiotics, anti-emetics and antacids. The man died on day 4 of the illness and in view of worsening sensorium the remaining three patients were referred to our center, a tertiary care hospital in North India. All the patients had similar complaints of jaundice and altered sensorium at admission. On Examination all three were afebrile, tachypneic (respiratory rate 26–32/min), had tachycardia (respiratory rate 120–160/min) with normal blood pressures at presentation.

Mother was pale with deep icterus, grade 3 hepatic encephalopathy, brisk deep tendon reflexes and down going planters. Investigations revealed anemia ((hemoglobin 7.6 g/dL), leukocytosis [total leukocyte counts (TLC) 16,000/mm^3], conjugated hyperbilirubinemia (total bilirubin/conjugated bilirubin 5.7/4.6 mg/dl), coagulopathy (prothrombin time >2 min), transaminitis [aspartate aminotransferase (AST)/alanine aminotransferase (ALT) 1580/2400 IU/ml)], deranged renal function test (creatinine 2.8 mg/dL) and hyperkalemia (K+ 5.6 mEq/L) with tall 'T' waves in electrocardiogram (ECG). Ultrasound abdomen showed fatty liver, liver span 13.4 cm, gall bladder wall thickening with pericholecystic fluid, bilateral renal parenchymal disease and mild ascites (Table 1).

Son had pallor, icterus, grade 3–4 hepatic encephalopathy, generalized hypertonia, absent deep tendon reflexes and mute planters. Investigations revealed anemia (hemoglobin 10.7 g/dL), normal leukocyte count, conjugated hyperbilirubinemia (total bilirubin/conjugated bilirubin

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Abbreviations: ALT: alanine aminotransferase; AST: aspartate aminotransferase; ECG: electrocardiogram; Hb: hemoglobin; ICU: intensive care unit; Inj: injection; IU/ml: international units/milliliter; IV: intravenous; NCCT: noncontrast computerized tomography; TLC: total leukocyte counts
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Table 1 Patient Characteristics.

Parameters	Patient 1: mother	Patient 2: son	Patient 3: daughter
Residence	Rural, hills	Rural, hills	Rural, hills
Type of mushroom consumed	Wild	Wild	Wild
Onset of symptoms after consumption	4 h	4–5 h	6 h
Initial symptoms	Pain abdomen, vomitings, bloody diarrhea	Pain abdomen, vomitings, bloody diarrhea	Pain abdomen, vomitings, bloody diarrhea
Resolution of initial symptoms	24–28 h	28–30 h	30–36 h
Period of convalescence	8–10 h	8–10 h	8–10 h
Progression of symptoms	Jaundice, altered sensorium	Jaundice, altered sensorium	Jaundice, altered sensorium
Day of admission to ICU	4th	4th	5th
General examination findings	Pallor, icterus	Pallor, icterus	Icterus
CNS examination	Grade 3 hepatic encephalopathy	Grade 3–4 hepatic encephalopathy	Grade 3 hepatic encephalopathy
	Hyperreflexia	Arreflexia	Hyperreflexia
	Planters down going	Planters mute	Planters down going
	Normal tone	Hypertonia	Normal tone
Investigations: Hemoglobin	7.6 g/dl	10.7 g/dl	12.1 g/dl
Leukocyte count	16000/mm^3	5000/mm^3	9200/mm^3
AST/ALT	1580/2400 U/L	2814/3759 U/L	573/1463 U/L
Bilirubin total/conjugated	5.7/4.6 mg/dl	5.2/3.1 mg/dl	5.2/3.6 mg/dl
Prothrombin time	>2 min	>2 min	>2 min
Serum sodium	125 meq/L	135 meq/L	137 meq/L
Serum creatinine	2.8 mg/dl	0.5 mg/dl	3.0 mg/dl
Arterial gases	Metabolic acidosis	Metabolic acidosis	Metabolic acidosis
Urine routine	Normal	Normal	Normal
USG abdomen	Raised liver echogenicity, 13.4 cm GB wall thickening, pericholecystic fluid, bilateral renal parenchymal disease, ascites	Raised liver echogenicity, 12.4 cm No ascites	Raised liver echogenicity, 11.5 cm GB wall thickening, pericholecystic fluid, bilateral renal parenchymal disease, ascites, bilateral mild pleural effusion
Management	ICU based including penicillin and silibinin	ICU based including penicillin and silibinin	ICU based including penicillin and silibinin
Course			
DIC	Present	Present	Present
Onset of hypotension	10 h	20 h	24 h
Demise	12 h	24 h	60 h
Terminal events	Refractory shock, MODS, sinus bradycardia	Refractory shock, MODS	Refractory shock, MODS, ventricular fibrillation, seizures

5.2/3.1 mg/dL), coagulopathy (prothrombin time > 2 min), transaminitis (AST/ALT 2814/3759 IU/ml), normal renal function test, electrolytes and ECG. Ultrasound abdomen showed fatty liver; span 12.4 cm and no free fluid. Noncontrast computed tomography (NCCT) head showed cerebral edema (Table 1).

Daughter had icterus, no pallor, grade 3 hepatic encephalopathy, brisk deep tendon reflexes and down going planters. Investigation revealed hemoglobin 12.1 g/dL, TLC 9200/mm^3, conjugated hyperbilirubinemia (total bilirubin/conjugated bilirubin 5.2/3.6 mg/dl), coagulopathy (prothrombin time >2 min), transaminitis (AST/ALT: 573/1463 IU/ml), normal Renal function test, electrolytes and ECG. Ultrasound abdomen showed fatty liver, liver span 11.5 cm, Gall bladder wall thickening with pericholecystic fluid, bilateral renal parenchymal disease, mild