



Fatal Mushroom Poisoning: A Case-series

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ARTICLE INFO

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How to Cite this article: Sarma B, Dash SK, Chavali K. Fatal Mushroom Poisoning: A Case-series. Journal of Indian Society of Toxicology 2019;15(1):48-53. DOI: [jist.org.in/10.31736/2019v15i1/p48](https://doi.org/10.31736/2019v15i1/p48)

Keywords:

Amanita phalloides, hepato-renal toxicity, autopsy, fulminant hepatic failure.

Conflicts of Interest and Fundings: Nil

Received – 11th Mar 2019
Accepted – 22nd May 2019
Published – 30th Jun 2019

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ABSTRACT

Mushroom poisoning is frequently reported from different geographical locations. Due to high case fatality rate, it is considered as a medical emergency and is seen among communities with the habit of hunting and cooking its wild variety. Amongst all identified species, poisoning caused by Amanita Phalloides is considered to be the fatal one because of onset of late symptoms with hepato-renal toxicity and persistence of toxins even after cooking or freezing. Symptoms appear usually after 6-8 hours of ingestion and by the time patient is brought to the emergency department, early hepatic complication already develops.

In Assam, there has been increasing incidences of toxicity following poisonous mushroom ingestion since last few years. In the year 2008, a significant number of cases of mushroom poisoning were reported from few districts of upper Assam. In two separate incidences, eight victims of wild mushroom poisoning were admitted at tertiary care institute of Dibrugarh district of Assam during the month of April, 2014. All of them died within a period of 7 days. Autopsy was conducted in department of Forensic Medicine of the same institute. Out of eight deceased, five were male adult and two children of age below 12 years. Autopsy findings were suggestive of fulminant hepatic and renal failure and their complications in all cases. Present case series focuses on fatalities due to poisonous mushroom in respect to their clinical presentation, laboratory investigation and autopsy findings.

Conclusion: Pesticides, especially cholinergic pesticides were the commonest group of agents used for deliberate self-poisoning in the study. There was a reduction in overall mortality each year due to self-poisoning over the study period with a reduction in pesticide related admissions each year. Regulation of pesticide use should be considered while planning prevention strategies of suicide.

INTRODUCTION

Poisoning following consumption of poisonous variety of mushroom is considered as a medical emergency. It has been reported throughout the year from different parts of the world. Such incidences are because of consumption of wild poisonous variety of mushrooms amongst those communities, where hunting and eating is a common habit. Although, these ethnic groups are expert in differentiating edible from non edible one, accidental poisonings are seen very often among themselves. The earliest report of documented fatal mushroom poisoning was attributed to the Greek poet Euripides, who described the death of his wife and three children after consuming poisonous mushroom.^[1] Published literature does not highlight exact incidence and fatalities due to poisonous mushroom throughout different part of our country as most of the cases remain undiagnosed, underreported and published articles are mostly in the form of case reports.^[2,3] It is very difficult to differentiate edible one from wild variety. Till now, approximately 5000 species of mushroom are identified globally; out of which 100 are poisonous.^[4] *Amanita phalloide* is the deadliest variety containing amatoxin as active principle. It is not even destroyed by cooking or freezing and have a longer incubation period (usually >6 hours), targeting mainly hepato-renal system.^[5,6] Even a single piece of amatoxin containing mushroom can be fatal causing death of an individual.^[5] Following its absorption, it causes depletion of protein synthesis by inhibiting RNA polymerase II; followed by DNA transcription.^[7] Incidences of fatal mushroom poisoning are increasing in Assam since last few years with 3-4 reported outbreaks per year. A significant number of cases were reported in 2008 from a few districts of upper Assam mostly among tea tribe communities.^[8] Herewith; we have described eight cases occurring in and around Dibrugarh district of Assam with the focuses on fatalities due to poisonous mushroom on its clinical presentation, laboratory investigation and autopsy findings.

CASE REPORT

All the eight victims from two separate incidences of mushroom poisoning, undergoing treatment in our hospital were categorized into two groups depending on the day and location of unfortunate incident.

Group 1: The incident occurred in the night of 01/04/2014 inside a tea estate of Dibrugarh district. The five victims,

two children and three adults were from two different families. Adults were daily worker at the tea garden and the two families were residing in the same campus. The head of one family (termed as 'Family A') collected some mushroom from a nearby field on 01/04/2014 in the evening. His wife prepared the mushroom curry for dinner and all of them (parents and their 11 years old daughter) consumed it at around 9:30 pm. They also offered the same mushroom curry to the family in their neighborhood (termed as 'Family B') where a widow was staying with her 9 years old boy. In the next day morning, all of them became ill with complains of vomiting, abdominal pain and mild diarrhea. They were immediately taken to the nearby primary health centre. After stabilizing, doctors planned to shift them to the tertiary care hospital, Dibrugarh for higher treatment on the same day. But due to ignorance and relative feeling of well-being, all of them took discharge against medical advice, went home and did not receive any further treatment. On 03/04/2014 early in the morning all family members including kids in family A and B felt ill. They were hospitalized in our hospital as their condition worsened. All the patients were in semiconscious state with signs of severe dehydration. Laboratory investigation reports showed abnormally raised liver enzymes (AST/ALT) and electrolyte imbalance (Na^+/K^+). Even after continuous monitoring and treatment, the daughter (Case 1) and mother (Case 2), belonging to 'Family A' died on 06/04/2014 at 7 am and 4 pm respectively i.e. on 4th day of hospital stay. Thereafter, the mother (Case 3) and child (Case 4) of 'Family B' and father (Case 5) of 'Family A' died on 07th and 8th April respectively, in the afternoon.

Group 2: This group comprised three adult security guards from another tea estate of Dibrugarh district. They all were deputed for night duty on 02/04/2014 in the garden premises. On the fateful night, one of the guards prepared the dinner with fish and mushroom which were collected from a nearby field. After completing a routine check-round they had their dinner at around 12 am. They all became ill by the next day morning with complaints of vomiting, abdominal pain and developed dehydration. After initial treatment at PHC, all of them were admitted to our hospital with suspicion of mushroom poisoning. Treatment was started immediately in ICU and necessary investigations sent. Gastric aspirate at the time of admission from all three patients were sent to forensic science laboratory for identification of species. They all became stable by evening. On 07/04/2014 (5th day of

hospital stay), condition of all three worsened suddenly with extremely high liver enzymes (AST/ALT) and electrolyte imbalance (Na^+/K^+); even after continuous treatment and monitoring. One of them (Case 1) died next day morning i.e. on 08/04/2014 and other two (Case 2 & 3) on 09/04/2014 in the afternoon.

Autopsy Findings: All eight cases as described above (in group 1 and 2) were brought for medico-legal autopsy on different dates in the month of April, 2014 to the mortuary of tertiary care institute; Dibrugarh. Autopsy findings of all cases are shown in table 1. Some photographs, showing important findings are also given (Fig.1,2,3,4). In the department laboratory, stomach contents of all cases from group 2 were examined under microscope. A drop of material placed on a slide and covered with a cover slip was viewed under high power magnification which revealed spores mixed with debris. They were light coloured spores with thin wall and almost uniform in size. Exact interpretation could not be drawn due to lack of experienced mycologist in the institute. Later on, chemical analysis of stomach contents from the same group (i.e. group 2) were done by high performance liquid chromatography and mass spectrometry (HPLC-MS) at Forensic Science Laboratory (FSL). Biological samples were deproteinized by mixing equal volume of organic solvent and centrifuged. Then the supernatant was injected and identified by comparing against a reference standard. It showed positive result of mushroom from amanita species in all three cases. Due to limited supply of control (high cost) agents and lack of stomach contents in other cases, both qualitative and quantitative analysis could not be performed in all by the FSL. Histopathology reports showed hepatic and renal toxicity in all cases. There was necrosis of hepatocytes either focal (case numbers 1,2,3,8) or diffuse (case numbers 4,5,6,7) with few surviving cells showing features of cholestasis along with vacuolization of renal tubular epithelium.

DISCUSSION

Mushroom poisoning is considered as medical emergency globally in affected population. It is responsible for 50 to 100 deaths per year in Western Europe.^[9] Most of the incidents are reported usually during rainy season because of its highest growth. Incidences are mostly seen among ethnic Indian tribes where hunting and eating wild mushroom is a common habit.^[10] Such fatalities has been reported very often in Assam, mostly from upper Assam districts since last few years because eating wild variety

is still a common habit among tea tribe communities. Mushroom is an important component of their diet and they usually pick it from open fields, gardens or roadsides during rainy seasons, cook them and eat. Due to lack of awareness amongst them and habit of hunting, cases have complex and challenging clinical presentation causing high mortality and morbidity. Initial clinical presentation following poisonous mushroom ingestion is mainly gastrointestinal.^[9,10] The incubation period is considered to be an independent prognostic criterion which is different for different species. If it is more than 6 hours, prognosis is usually not considered as good and amanita group (the deadliest one) is suspected to be the offending species.^[12] This species mainly contains phallotoxin and amatoxin. Former is toxic to cell membrane of enterocytes leading to initial diarrhea like illness whereas later is responsible for the toxic effect leading to acute liver and renal failure with associated potential toxicities to pancreas, adrenal glands, and testes.^[13] Clinical presentation usually comprises 3 stages- initially for at least 6 hours patient is symptomless, followed by gastrointestinal symptoms (First stage), then there will be apparent recovery even without any treatment; although there is an elevation of liver enzymes during 12-48 hours (Second stage), ultimately patient will develop fulminant hepatic failure and its complications (Third stage).^[7] Victims usually seek medical attention late due to longer incubation period. Moreover, initial symptoms often simulate acute gastroenteritis leading to delay in diagnosis. In some situations, patient is discharged to home in the second stage of mushroom poisoning i.e. after apparent recovery. Hence, by the time patients are referred to tertiary care centre, they must be in a stage of fulminant hepatic and renal failure. Involvement of liver usually is seen after 48 hours and the victim dies because of hepato-renal complications.^[14] Hence, when a patient is brought to hospital with alleged history of consumption of wild mushroom it is very difficult to diagnose because of clinical presentation similar to acute gastroenteritis. Although, treatment in that line is started based on history and baseline laboratory investigations; species confirmation is possible only with examination of gastric aspirate either under microscopy by mycologist or by HPLC-MS, Gas chromatography- Mass spectrometry. But the main problem is non availability of either in most of the government institutes. Moreover, there are no specific autopsy findings in fatal mushroom poisoning; some authors have described jaundice, pleural effusion and ascites along with findings suggestive of coagulopathy.^[14,15,16] Hence, in the absence of specimen; offending

species is predicted clinically based on case fatality rate and incubation period along with the geographical location.^[17,18] *Amanita phalloides* has longer incubation period and high case fatality rate amongst all, causing 90% cases of fatal mushroom poisoning.^[19] In these two reported incidences, mushrooms were mainly collected from open fields grown naturally. They consumed it after proper cooking. In each case, initial symptoms were mainly gastrointestinal and appeared after a gap of more than 6-8 hours. Five cases were reported to our center very late, after 36 hours of exposure and by the time they reached; they all started developing signs of acute liver dysfunction with abnormally raised enzyme levels. Rapidly it progressed into fulminant hepatic failure. Although, other 3 cases of second incidence reached hospital immediately after diagnosis; they too started developing hepatic and renal dysfunction even with continuous treatment and monitoring. This highlights that the mushroom species involved was a deadly one. Although, we could not locate the exact field of alleged mushroom origin; it was identified to be of amanita group in three cases by HPLC-MS. In rest of the cases we were unable to recover traces from stomach contents. Moreover, there was inadequate supply of control for high performance liquid chromatography due to its high cost. However,

involvement of amanita species was strongly suspected in other five cases based on similar clinical presentations; longer incubation period and persistence of toxicity even following cooking. Poor prognosis of amanitin induced acute liver failure has also been illustrated by various authors.^[20,21] Due to compromised liver function, synthesis of clotting factors was depleted, prothrombin time increased (more than 2 minutes); leading to multiple hemorrhagic foci in different organs with different extent. Ganzert et al found prothrombin index along with serum creatinine level as prognostic indicator in a retrospectively series following amatoxin intoxication. Overall, the prognosis of amanitin induced acute liver failure remains quite poor.^[20,21,22] Externally appearance of jaundice was noted over skin and mucous membrane in all cases. Ascitis seen in few cases was also due to compromised liver. In our case series, cause of death was confirmed following autopsy and histopathology findings in all cases as because of complications of hepatic and renal failure. The earliest death occurred within 4 days of exposure and the affected victims were a mother and her 12 years old daughter. All adult deceased male in this case series survived for at least 6 days. Similar kind of early fatalities among children and female population has been documented in various literatures.^[8,23]

Table 2: Case-wise autopsy features.

Groups	Victims	Age/Sex	External findings	Internal findings
1	Case 1	11 years/Female	Body pale, features of jaundice, froth around mouth and nostrils.	Sub-capsular hemorrhage of liver, cortical hemorrhages of kidney, petechial hemorrhages over lungs and stomach mucosa.
	Case 2	33 years/Female	Body pale and features of jaundice.	Petechial hemorrhage over stomach mucosa, Fatty infiltration of liver with sub-capsular hemorrhage, Cortical hemorrhage over kidney.
	Case 3	35 years/Female		Sub-capsular hemorrhage of liver and hemorrhage in peri-nephric fatty tissue.
	Case 4	9 years/Male		Enlarged liver, cortical hemorrhage over kidney, petechial hemorrhage over stomach mucosa.
	Case 5	36 years/Male		Ascitis, sub-capsular hemorrhage of liver with nodularity, hemorrhagic foci over lungs and mesentery of small intestine.
2	Case 6	27 years/Male	Body pale and features of jaundice.	Ascitis, contracted nodular liver with sub-capsular hemorrhage, hemorrhagic foci over omentum, mesentery, lungs.
	Case 7	32 years/Male		Ascitis, contracted nodular liver with sub-capsular hemorrhage, hemorrhagic foci over lungs, heart, omentum and kidney.
	Case 8	25 years/Male		Nodular liver with sub-capsular hemorrhage, cortical hemorrhage of kidney.

CONCLUSION

Preventive measures and early treatment are the two important steps to decrease fatalities in mushroom poisoning. Most of the cases are reported in a late stage to the tertiary care center with already abnormal liver functions. Hence, all the cases brought to the primary care system must be dealt promptly and if required referred as early as possible. Patients with signs and symptoms suggestive of acute gastro enteritis especially reported during rainy season must be evaluated thoroughly to rule out mushroom poisoning. Preventive measure comprises of legislation for selling mushroom only in designated

center. Awareness program must be organized periodically especially among communities where hunting and eating mushroom collected from open field is a regular habit. They should be taught the severity of wild variety and scientific cultivation should be promoted.

There must be facility for prompt identification of mushroom species by laboratory tools, which is lacking in our region due to non-availability of chemicals (because of its high cost) and expert mycologist. This can be considered a limitation of this study.

Fig 1: Photograph showing yellowish discoloration of sclera.



Fig 2: Photograph showing sub-capsular hemorrhage of liver.

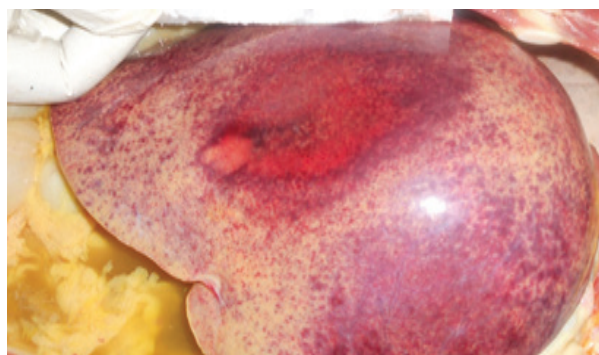


Fig 3: Photograph showing multiple supleural hemorrhagic foci in lungs.

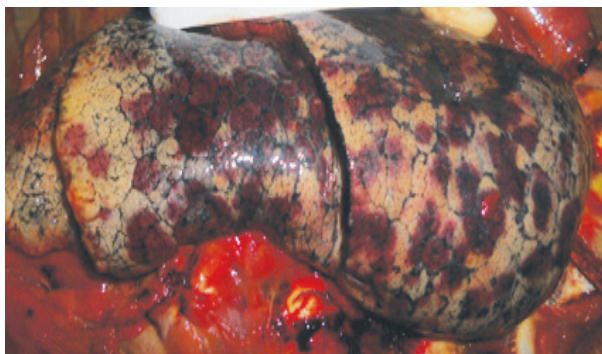
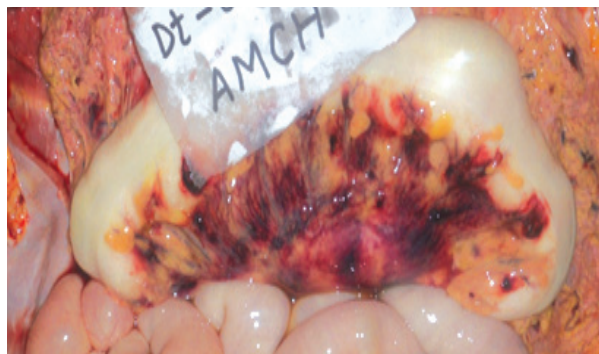


Fig 4: Photograph showing difuse confluent areas of mesenteric hemorrhage.



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