Clinicopathological Aspects of Death due to Wild Mushroom Poisoning: An Autopsy Report

Abstract
Mushrooms are widely cultivated and used as a source of diet and commerce; however, unfortunately, some wild mushrooms are highly toxic to humans. Although the poisonous mushrooms have a characteristic physical appearance, sometimes it is difficult to differentiate between the poisonous and nonpoisonous variety even by the experts. We report a case of a 23-year-old married female who mistakenly harvested and consumed wild mushrooms along with her family members and subsequently died on the 5th day postingestion. Due to the medicolegal implications, a forensic autopsy was conducted in this case. Here, we discuss clinical findings along with detailed autopsy findings of this case, including histopathology examination.

Keywords: Forensic pathologists, mushroom poisoning, mycetism, wild mushroom

Introduction
A mushroom is a parasitic fungus, grows naturally in a damp environment, more during the monsoon season, and is also cultivated widely because of its nutritional and medicinal properties. In India, Punjab, Haryana, and Himachal Pradesh are the major mushroom-producing states.[1] Some poisonous mushrooms are mistakenly ingested due to their close resemblance to edible mushrooms. Although most reported cases are accidental, considering the high toxicity and easy availability, its usage for homicide cannot be neglected. Forensic pathologists have a great responsibility to find the exact cause of death and rule out any foul play, especially in the death of a married woman within 7 years of her marriage.

Case Report
Here, we present a case of a family residing in a village of Kullu district of the Indian state of Himachal Pradesh comprising a young couple with their 1-year-old child, middle-aged parents, and a brother. All the family members except the deceased’s husband and the child consumed wild mushroom dish harvested from the nearby hilly region [Figure 1]. The exact species identification could not be performed due to the nonavailability of mushroom specimen. However, the features from the photograph provided to us are suggestive of Amanita species. After 10–12 h, they developed abdominal pain, vomiting, and diarrhea. As the whole family started having the gastric symptoms postconsumption of the wild mushroom dish, they all went to primary care center from where they were referred to district-level hospital where they were treated symptomatically as a case of food poisoning, following which they showed significant improvement. Therefore, all were kept under observation, and extensive laboratory workup was not done during this period. After being stable for a day, all were discharged the next day, i.e., 3rd day postingestion. All the family members recovered except the 23-year-old wife (deceased), whose condition kept deteriorating. She was therefore taken to a higher center located in Shimla on the 5th day postingestion. A provisional diagnosis of mushroom poisoning with multiorgan failure with hemorrhagic manifestations was made, and after that, the patient was referred to our institute on the same day.

As per medical records, the deceased was brought in an unconscious state. Blood pressure was recorded as 140/90 mmHg, SpO₂ 94%, pulse rate 102/min, respiratory rate 20/min, and Glasgow coma scale 3.

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score of 3. She was pale and had bilateral subconjunctival, periorbital hemorrhages. Liver and renal function tests were found to be deranged [Table 1]. Ultrasonography abdomen report showed collapsed and edematous gallbladder with wall thickness 7–8 mm with minimal ascites. The noncontrast computed tomography head was normal. She was treated for acute liver failure with grade IV hepatic encephalopathy with acute kidney injury. She died on the same day of admission, i.e., 5th-day postingestion.

The deceased was subjected to a medicolegal autopsy as it was a case of poisoning of a female who died within 7 years of her marriage. On autopsy, the external examination revealed multiple ecchymotic patches present over the scalp, face, neck, upper limbs, and eyelids with periorbital swelling. Frank anal bleed was present. On internal examination, ecchymotic spots were found at multiple places over meninges, brain parenchyma, lungs, heart, walls of large blood vessels, liver, spleen, pancreas, kidneys, and uterus. Mesenteric venous thrombosis was noted. The stomach was congested and hemorrhagic at places [Figure 2a and b]. After postmortem examination, tissues were kept for histopathological examination; viscera and blood were sent for chemical analysis to the concerned regional forensic science laboratory (RFSL). However, the chemical analysis report came negative for toxins. On microscopic examination, the sections from the liver showed fatty changes and also extensive centrilobular necrosis. Both kidneys showed toxic acute tubular necrosis with necrotic epithelial cells, tubular dilatation, and red blood cells noted in the tubular lumen. The glomerular architecture was maintained. Splenic arterioles showed thrombus formation. Bilateral lung sections showed pulmonary edema and intra-alveolar hemorrhage [Figure 3a-c]. The brain showed mild edematous changes, and the heart showed normal morphology. The cause of death was concluded as acute liver failure, leading to disseminated intravascular coagulation (DIC) and shock due to accidental consumption of wild mushrooms.

**Discussion**

Mushroom toxicity is prevalent worldwide and seems very common in Europe, Central and South America, Australia, Asia, and Africa; however, it is uncommon in the United States. In India, around 100 poisonous species of mushrooms have been reported. Still, human poisoning is less common as the people harvesting the mushrooms are experienced enough in identifying the poisonous from nonpoisonous species. Among the mushroom toxins, amatoxin is considered to be the most toxic. Owing to its property of thermostability, amatoxins remain toxic even after cooking and long periods of cold storage. A single mushroom contains up to 15 mg of the toxin. Even a dose of 0.1 mg/kg bodyweight of amatoxin can be fatal. Due to its high human toxicity, amatoxin is responsible for more than 90% of deaths worldwide. Amatoxin undergoes first-pass metabolism in hepatocytes and undergoes enterohepatic circulation, leading to prolonged stay of amatoxins into the system and consequently the period of exposure for hepatic cells. The binding of amatoxins to eukaryotic RNA polymerase II hinders the transcription process, thus leading to the decreased hepatic formation of mRNA and protein synthesis, which eventually leads to cellular necrosis, resulting in acute liver dysfunction and failure. Faulstich has also indicated about the coagulation disorder being one of the dominant features occurring in addition to the injury
to the liver tissue and raised hepatic enzymes, resulting in thrombosis as a secondary complication.[4,5]

The clinical manifestations of mushroom poisoning follow a three-stage pattern – within first 6–24 h, there is a tremendous body fluid volume loss due to delayed gastroenteritis, there is a alleviation of symptoms in 24–36 h post-ingestion, and in usually 3–5 days post-ingestion, fulminating hepatic failure along with multiorgan dysfunction syndrome appears.[6] Most of the time, patients may be discharged with a diagnosis of food poisoning and present to the emergency room later with highlights of liver failure, adding to the generally high case fatality rate of 10%–20%.[7]

In the current report, the deceased showed typical clinical features of different stages of mushroom poisoning. As per the history given by the family members, the infant was breastfed by the deceased and subsequently developed lethargy and diarrhea. Therefore, the authors believe that the mushroom toxins could have been secreted in the breast milk. Although some textbooks state that amatoxins are secreted into breast milk, not much literature is available in this regard.[8] Within 12 h after the intake of the wild mushroom dish, the family members started having vomiting and diarrhea. It is a well-known fact that each individual reacts differently to the same toxin; hence, the deceased having a more severe response to ingested mushrooms can be attributed to this.[9] Further, as per the relatives of the deceased, she consumed more amount of the prepared dish and hence the severity.

On autopsy, the viscera and blood were preserved and sent to the RFSL. Still, the chromatographic analysis could not detect any toxin. In urine sample, amatoxins can be detected up to 4 days post-ingestion compared to the blood sample, where it is up to 30 h. Further, in the urine sample, the toxin concentration has shown to be 10–100 times higher.[10] In our case, the deceased had a survival period of 5 days, and there was no urine at the time of autopsy. Hence, as a routine, we could send only viscera and blood for chemical examination. In addition, a negative chemical analysis report does not rule out the initial toxicity due to amatoxins. As amatoxins are hepatotoxic in nature, in the current case, they led to severe hepatic dysfunction and ultimately leading to its failure, resulting in DIC and shock. As per the toxicology report issued by the RFSL, no toxins, drugs, or alcohol was detected, thus ruling out the intake of other hepatotoxic agents. In this case, the typical clinical presentation, autopsy findings, and histopathological examination confirmed the diagnosis of mushroom poisoning. Circumstantial evidence rules out the homicidal poisoning.

**Conclusion**

Amatoxin is the most common and dreadful mushroom toxin being reported. Mushroom poisoning has to be considered a differential diagnosis in patients presenting with acute gastroenteritis during the monsoon season, especially from the geographic area where occupants are known for consuming wild mushrooms.

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**Conflicts of interest**

There are no conflicts of interest.

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