SMU Sikkim Manipal University

SMU Medical Journal

(Volume – 1, No. – 1, January 2014)

The Fatal Recipe of Mushroom

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Manuscript received : 11.12.2013
Manuscript accepted : 08.01.2014

Introduction

Mushroom is commonly ingested in various parts of the world including the Himalayan state of Sikkim in spite of being aware of the fact that ingestion of wild mushroom if poisonous may be life threatening. Mushroom poisoning is usually the result of ingestion of wild mushrooms after misidentification of a toxic mushroom as an edible species. The most common reason for this misidentification is close resemblance in terms of colour and general morphology of the toxic mushrooms species with edible species. The presentation after eating poisonous mushroom may be varied depending on the species of the mushroom and the toxins present in it. Patients commonly present with features of acute gastroenteritis, fulminant hepatic failure and renal failure. Other manifestations like rhabdomyolysis, methemoglobinemia, hemolysis, seizures or hallucinations and severe muscarinic symptoms may also occur depending on the species of mushroom. Identification of the species is a difficult process and needs the help of an expert mycologist. Here we report one family who presented with mushroom poisoning after consuming mushroom obtained from their kitchen garden, from where they used to consume earlier also without any ill effects.

Case report

From the year 2008 onwards, 29 cases of mushroom poisoning with 5 deaths were admitted at Central Referral Hospital, a tertiary care hospital in East Sikkim associated hospital of Sikkim Manipal Institute of Medical Sciences, Gangtok, Sikkim. The clinical presentation of four members of one family is being described below.

A 32 years old male (case A) along with 28 years old wife (case B) , 10 years son (case C) and 6 years old daughter (case D) presented with profuse watery loose motions and vomiting 16 hours after consumption of cooked mushroom collected from their kitchen garden. All of them were alright before this presentation. They presented to the hospital within 2hrs of consumption. After 36 hours of ingestion, the frequency of loose motion and vomiting diminished to 2-3 times with conservative treatment which mainly included volume resuscitation. 48 hours later, all of them were passing black tarry stools and after 60 hours were complaining of cough streaked with blood. There was no complain of fever,convulsions,hallucinations or other muscarinic symptoms.There was no history of bleeding disorders in the family.

Examination on admission revealed mild dehydration in all and mild icterus in Case A. The general condition was fair, all were afebrile and vital parameters were normal.

On systemic examination, there was diffuse tenderness over abdomen. There was no rigidity, guarding or organomegaly. Examination of respiratory, cardiovascular and central nervous system did not reveal any abnormality.

The investigations done revealed a normal blood count, Kidney Function tests and electrolytes. The Liver function tests were deranged.

Case A

Day 1	Serum Bilirubin	: 4.5mg/dl,
	Direct Bilirubin	: 2.6mg/dl
	Prothrombin Time	: (Patient) 24.55seconds.
		(Control) 13.90seconds.
Day 3	Serum Bilirubin	: 2.2mg/dl,
	Direct Bilirubin	: 1.4mg/dl
	Prothrombin Time	: (Patient) 16.44seconds.
		(Control) 13.45seconds.

Case B

Day 1	Serum Bilirubin	: 2.9mg/dl,
	Direct Bilirubin	: 1.3mg/dl
	Prothrombin Time	: (Patient) 18.45seconds.
		(Control) 13.60seconds.
Day 3	Serum Bilirubin	: 8.8mg/dl,
	Direct Bilirubin	: 0.3mg/dl
	Prothrombin Time	: (Patient) 19.55seconds.
		(Control) 13.75seconds.

Case C

Day 1	Serum Bilirubin	: 1.0mg/dl,
	Direct Bilirubin	: 0.3mg/dl
	Prothrombin Time	: (Patient) 15.15seconds.
		(Control) 14.50seconds.
Day 3	Serum Bilirubin	: 11.2mg/dl,
	Direct Bilirubin	: 2.0mg/dl
	Prothrombin Time	: (Patient) 55.40seconds.
		(Control) 14.50seconds.

Case D

Day 1	Serum Bilirubin	: 0.8mg/dl,
	Direct Bilirubin	: 0.3mg/dl
	Prothrombin Time	: (Patient) 13.55seconds.
		(Control) 13.20seconds.
Day 3	Serum Bilirubin	: 1.0mg/dl,
	Direct Bilirubin	: 0.3mg/dl
	Prothrombin Time	: (Patient) 13.45seconds.
		(Control) 13.25seconds.

Stool for occult blood was positive in all cases.

Treatment and outcome:

The patients were treated conservatively. Gastric lavage with normal saline was done and activated charcoal was used to adsorb the remaining toxins. Dehydration was corrected with administration of intravenous fluids.

Hemodynamic monitoring of the patients was done in the Intensive Care Unit. Husband (Case A), wife (Case B) and their 6 years old daughter (Case D) were stable after 4-5 days of conservative treatment but their son (Case C) started deteriorating as he developed profuse hemoptysis and was unable to maintain arterial oxygen saturation. His prothrombin time was markedly prolonged along with the marked increase in serum bilirubin levels.Inspite of all conservative measures and ventilatory support, he succumbed after 6 days of consuming poisonous mushroom. Rest all patients were stable in their follow up.

Autopsy of Case C showed thick gelatinous type sub mucosal hemorrhage. Hemorrhagic patches were present over whole intestine, omentum, liver, pericardium and lungs. There were bleeding points over lower esophagus. Other organs were congested. Brain was pale and there was no evidence of intracranial hemorrhage. Liver was yellowish and uniformly congested.

Discussion

In our case the patients were unable to provide us the sample of mushroom consumed as they consumed all that were grown in their kitchen garden but going retrospectively from their presentations and investigations showing fulminant hepatic failure it appears that it was due to *Amanita sp*[1]. Ingestion of *A. phalloides* may account for approximately 90% of deaths attributable to mushroom ingestion worldwide. *Amanita sp.* has no specific antidote. The main treatment is vigorous intravenous fluid replacement and correction of electrolyte disturbances. The only definitive treatment may be liver transplantation once fulminant liver failure occurs[2,3,4]. Fatal outcomes are associated with age less than 10 years, a short latency between ingestion and onset of symptoms and severe coagulopathy[2]. Other syndromes seen with mushroom poisoning, such as acute renal failure, encephalopathy (convulsive or non convulsive) and erythromelalgia were not observed in these cases[5].

Mushrooms are source of protein and essential amino acids and are low in calories and cholesterol and hence consumed widely. There are many folk traditions concerning the defining features of poisonous mushrooms. Unfortunately there are no general identifiers for poisonous mushrooms and so such traditions are unreliable guides and are a frequent cause of mushroom poisoning. Some common myths are

- Poisonous mushrooms are brightly coloured but in reality most poisonous mushrooms are not unique in appearance and can be mistaken for nonpoisonous species.
- Poisonous mushrooms are bad to taste and are destroyed by cooking and drying but actually it has no distinct taste or smell, and though some toxins maybe inactivated by cooking or drying others are not.
- Myths like mushroom growing on wood are safe does not hold true.

Incidents of mushroom poisoning dates back to 479BCE when there are some unconfirmed reports of Gautama Buddha being a victim of the same. Holy Roman Emperor Charles VI and Tsaritsa Natalia Naryshkina are believed to have died from eating the death cap mushroom

The parents of the physicist Daniel Gabriel Fahrenheit, who created the Fahrenheit temperature scale, died in Danzig on 14 August 1701 from accidentally eating poisonous mushrooms. According to a popular legend, the composer Johann Schobert died in Paris, along with his wife, one of his children, maidservant and four acquaintances after insisting that certain poisonous mushrooms were edible.

Education regarding the poisonous nature of wild mushrooms may act as a deterrent to careless mushroom foraging and ingestion.

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Authors Column

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SMU Medical Journal, Volume – 1, No. – 1, January, 2014, PP. 175 – 178