Original Article

# Mushroom Poisoning

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## ABSTRACT

**Objective.** We aimed to review characteristics of mushrooms and mushroom poisoning and compare clinical picture, laboratory data, treatment modalities and prognostic factors in children with amanita intoxication and non-amanita mushroom poisoning.

**Methods.** We analyzed 39 pediatric patients through 1994-2004, retrospectively from the patient files and evaluated the patients in two groups as patients with amanita intoxication and patients with non-amanita mushroom poisoning. All of the cases were admitted to the hospital in autumn. Twenty three (59%) of the patients were female and 16 (41%) were male. Mean age of the patients was 8.05 ± 2.10 years.

**Results.** Amanita phalloides toxin was detected in the serum in 8 patients. Eleven (28%) of the cases were strongly suggestive of amanita poisoning but alpha amanitin level could not be studied. The average time of appearance of symptoms after mushroom consumption, duration of symptoms, hospital stay, serum AST, ALT, PT and creatinin levels were significantly higher in patients with amanita poisoning ($p<0.01$). Conventional therapy, antidote therapy together with hemoperfusion were carried out in 16 (41%) of the patients. Four of the patients in whose blood amatoxin was detected (50%) and 3 of the patients highly suggestive of amanita poisoning (30%), totally 7 patients died of hepatic coma. The average time of admission to hospital, mean AST, ALT, creatinin and PT values at 3rd day were significantly higher in patients who died of hepatic coma. Prognosis was better in case of early admittance to hospital in patients with amanita poisoning.

**Conclusion.** Early diagnosis and treatment in mushroom poisoning can be life saving. Public awareness is very important in prevention of intoxication as well as encouraging early admission to hospitals. **[Indian J Pediatr 2007; 74 (9) : 847-852]** *E-mail: oznuryilmaz@yahoo.com*

***Key words :*** *Mushroom; Poisoning; Amanita phalloides; Hemoperfusion*

The habit of eating naturally grown mushrooms is quite common in our country especially among the people living in rural areas. Hundreds of people are admitted to hospitals with mushroom poisoning every year and many lose their lives because of the complications.

Mushroom are the visible fruit of a fungus with more than 5000 known species. But of these only about 100 are poisonous and responsible for most of the cases of mushroom poisoning. The clinical picture caused by poisonous compounds in mushrooms is called mushroom poisoning or ''mycetismus''.[1] Mushroom poisoning is most commonly seen in spring and autumn seasons with cool, damp evenings which promote mushroom growth. Most of the times, nontoxic and poisonous mushrooms grow nearby and unfortunately, many mushrooms are difficult to identify even by a trained mycologist (Figs. 1,2).

**Fig. 1.** Amanita phalloides'', also known as ''the death cap'' that is responsible for most of the fatalities.

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**Fig. 2.** ''Agaricus bisporus'', also known as ''chest nut mushroom'' frequently consumed in daily nutrition.

Our country is very rich in growth of mushroom flora because of its convenient ecological environment. Mushroom poisoning is very common especially among people from low socioeconomic level who consume mushroom in daily nutrition.[2]

The time interval between the ingestion of the mushrooms and the appearance of first symptoms is very important in terms of prognosis. Symptoms appear earlier in mushrooms with low toxicity while symptoms appear later in cases of poisoning with toxic mushrooms. Mushrooms with short incubation period (usually less than 6 hs) contain muscarine, coprin, ibotenic acid and psilocybin toxins.[2] In these cases, the clinical picture is mild and symptoms resolve in shorter period of time.

Symptoms start to appear after 6-24 hr in mushrooms with longer incubation period. Gyromytra and Amanita (that contain amatoxin) are in this group. Nearly 95% of all deaths caused by mushroom poisoning are due to the group called amatoxins. Within the amatoxin group, the species responsible for most fatalities are *amanita phalloides* (''the death cap'') and *amanita virosa* (''the destroying angel''). Amatoxin is a termostabile, dialyzable octapeptide and inhibit mRNA and DNA synthesis by inhibiting RNA polymerase II.[3] This toxin is very strong and is lethal at a dose of 0.1-0.3 mg/Kg.[4] It can stay stabile 1- 5 yr in frozen state. There is a case report about severe mushroom poisoning by eating the mushroom that is kept in deep freezer.[5] No known enzymes are capable of degrading and heating them to 100ºC for several minutes does not destroy them.

Symptoms of amanita poisoning occur in different stages or phases. In the 1st phase, abdominal cramping, nausea, vomiting, severe watery diarrhea occur 6-24 hr after eating the mushroom and last for about 24 hr. In the 2nd phase, there is a period of remission of symptoms that last 1-2 days. During this time, the patient feels better, but blood tests begin to show evidence of liver and kidney damage. In the 3rd phase, liver and kidney failure develops and either leads to death within about a week or recovery occurs within 2-3 weeks.

Variations in amount ingested amatoxin and individual susceptibility probably account for mortality rates ranging from 10-90%. Toxin is absorbed from the intestines and carried in blood bound to albumin. Amatoxins lead to death of many cells, especially those that reproduce frequently such as in the liver, intestines and kidney. Early treatment can be life-saving. The aim is to remove the toxin from the body as soon as possible.[6]

In the present study, we aimed to review characteristics of mushrooms and mushroom poisoning and compare clinical pictures, laboratory data, treatment modalities and prognostic factors in children with amanita intoxication and non-amanita mushroom poisoning.

## MATERIALS AND METHODS

In the present study, we analyzed 39 children admitted to our hospital between the yr 1994-2004, retrospectively from the patient files; in terms of age, gender, seasonal factors, family history, time of occurrence of symptoms, time of admittance to hospital, symptoms on admission, laboratory values, duration of hospital stay, response to treatment and prognosis.

We evaluated the patients in two groups (i) patients with amanita intoxication and (ii) patient with non-amanita mushroom poisoning. While making this distinction, we considered the physical features of the mushrooms, time of appearance of symptoms, the clinical condition of the patient and the other family members that are poisoned, laboratory data and serum alpha amanitin levels. Serum alpha amanitin level was studied by high pressure liquid chromatography method in Istanbul Hifzisihha Institute only in 8 patients.

In cases with short latent period, firstly gastric lavage was carried out in case the patients were admitted to hospital in the first 4 hr. Then, activated charcoal (1g/Kg every 4 hr) was given and forced diuresis (intravenous fluid administration 3000cc/$m^2$/day together with diuretics) was performed for 48 hr, as enterohepatic circulation continues 48 hr. If there are no gastrointestinal symptoms 48 hours after mushroom ingestion and the laboratory parameters being normal, the therapy was discontinued.

In cases with long latent period and if there were gastrointestinal symptoms, amatoxin poisoning is strongly suspected, and antidote (penicillin 300.000-1million units/Kg/day, silibinin 25-50 mg/Kg/day) was given without waiting for the results of toxicological tests- if performed. Steroids and vitamin C were also used. Liver, kidney functions, electrolytes, blood glucose and protrombin time was checked at certain intervals. Hemoperfusion was carried out in patients in whom

amatoxin was detected in blood, or amatoxin poisoning was strongly suspected. Carbon filter (Alucard) was used in hemoperfusion. As intravenous form (IV) of silibinin is not available in our country, oral form of silibinin was used for our patients.

In patients with fulminant hepatic failure, maintenance therapy was given. Even though orthotopic liver transplantation is the only treatment modality in these patients, it could not be performed in any of them.

For statistical analysis, SPSS (Statistical Package for Social Sciences) for Windows 10.0 was used. In addition to definitive tests like standard deviation analysis, Mann Whitney U test was used in comparing quantitative data. Chi-square and Fisher's Exact chi- square test was used in comparing qualitative data. The results were evaluated in 95% confidence interval and $p<0.05$ values were accepted as statistically significant.

## RESULTS

The present study involved 39 children, treated for mushroom poisoning in department of Internal Medicine of our clinic between the years 1994-2004. All the children were admitted to the hospital in autumn. Twenty-three (59%) of them were female and 16 (41%) were male. Mean age of the patients was 8.05 ± 2.10 yr (4.5-12 yr).

Symptoms on admission were: vomiting and diarrhea in 79.5%, only vomiting in 10.3%, abdominal pain 5.1%, abdominal pain and vomiting in 5.1 % of the patients. There was family history of intoxication in 20 (51.28%) of the patients. Average time of appearance of symptoms was 12.31±12.39 hours (2-72 hours). Average time of admittance to hospital was 16.26±14.26 hours. Symptoms lasted 22.10±15.69 hr in average. The average duration of hospitalization was 8.08 ± 5.57 days.

Amanita phalloides toxin was detected by high pressure liquid chromatography method in blood in 8 cases (in Istanbul Hifzisihha Institute). Eleven (28%) cases were strongly suggestive of amanita poisoning (according to time of appearance of symptoms, anamnesis, clinical and laboratory data) but alpha amanitin level could not be investigated (Table 1). Among the 19 patients accepted as amanita poisoning, the average time of appearance of symptoms was 20.31±13.83 hr. False well-being lasted 24.10±3.87 hr in amanita poisoning. Aspartate aminotransferase (AST) levels were 2243.7±613.6 U/l (26-3156), and alanine aminotransferase (ALT) levels were 2820.2±546.8 U/l (16-3434) on 3rd day in average. Protrombin time (PT) was prolonged in all of them and was infinite in 2 of the patients. Renal functions were also impaired. The average duration of symptoms was 33.68±15.39 hr and average duration of hospitalization was 15.25±4.27 days.

**TABLE 1. Distribution of Types of Mushroom Poisoning**

| Mushroom poisoning | n:39 | % |
|---|---|---|
| Amanita phalloides | 19 | 48 % |
| Non-amanita | 20 | 52% |

The average time of occurrence of symptoms in non-amanita poisoning was 4.70±0.98 hr. Vomiting was predominant among these patients. Average AST levels were 39.10±5.69 U/l and ALT levels 30.10±6.08 U/l. Renal function was impaired in only one patient and since it accompanied symptoms of dehydration and returned to normal after hydration, it was thought to be of prerenal origin. The average duration of symptoms was 11.10±2.47 hr and average duration of hospitalization was 4.00±0.86 days. Anticholinergic symptoms accompanied vomiting in one patient but improved without antidote therapy.

The average time of appearance of symptoms after mushroom consumption, average duration of symptoms and hospital stay were significantly higher in patients with amanita poisoning ($p<0.01$) (Table 2). There was no statistically significant difference in terms of symptoms. Serum AST and ALT values were also significantly higher in patients with amanita poisoning ($p<0.01$) (Table 2). Presence of family anamnesis was significantly higher in amanita group (89.5%) ($p<0.01$) (Table 2).

Conventional therapy (maintenance of fluid-electrolyte balance, decontamination, forced diuresis), antidote therapy together with hemoperfusion was carried out in 16 (41%) of the patients (among the patients with definite amanita poisoning and in patients strongly suggestive of amanita poisoning). Three of them who were admitted very late did not receive hemoperfusion. The rest were treated with only conventional therapy.

Four of the patients in whose blood amatoxin was detected (50%), 3 of the patients highly suggestive of amanita poisoning (30%), totally 7 patients died of hepatic coma (Table 3). Death from hepatic coma was observed 5-10 (average 7) days after the mushroom ingestion.

There was no statistically significant difference in terms of age and gender between survivors and dead in amanita group ($p>0.05$) (Table 4). The average time of admission to hospital after ingestion was significantly higher in patients who died of hepatic coma ($p<0.01$) (Table 4). The mean AST, ALT, creatinin and PT values on 3rd day were also significantly higher in patients who died (Table 4). Thirty-two (82%) of the patients were discharged from the hospital with clinical and laboratory improvement.

## DISCUSSION

Turkey is very suitable for mushroom growth because of

**Table 2. Clinical and Laboratory Features of Amanita and Nonamanita Poisoning**

| | | Amanita poisoning Mean ± SD | | Non-amanita poisoning Mean ± SD | | Test stat.; p |
|---|---|---|---|---|---|---|
| Time of appearance of symptoms (hours) | | 20.31 ± 13.83 | | 4.70 ± 0.98 | | Z:-5.406; p:.0.001* |
| Duration of hospital-stay (hours) | | 15.25 ± 4.27 | | 4.00 ± 0.86 | | Z:-4.389; p:0.001* |
| Duration of symptoms (hours) | | 33.68 ± 15.39 | | 11.10 ± 2.47 | | Z:-5.291; p:0.001* |
| AST (U/l) | | 2244.7 ± 613.6 | | 39.10 ± 5.69 | | Z:-5.343; p:0.001* |
| ALT (U/l) | | 2820.2 ± 546.8 | | 30.10 ± 6.08 | | Z:-5.342; p:0.001* |
| | n | % | n | % | | |
| vomiting | Yes | 19 | 100 | 18 | 90.0 | $F\chi^2$ |
| | No | | | 2 | 10.0 | p:0.487 |
| diarrhea | Yes | 17 | 89.5 | 14 | 70.0 | $F\chi^2$ |
| | No | 2 | 10.5 | 6 | 30.0 | p:0.235 |
| Abdominal pain | Yes | 1 | 5.3 | 3 | 15.0 | $F\chi^2$ |
| | No | 18 | 94.7 | 17 | 85.0 | p:0.605 |
| Family anamnesis | Yes | 17 | 89.5 | 2 | 10.0 | $\chi^2$: 24.633; |
| | No | 2 | 10.5 | 18 | 90.0 | p: 0.001* |

Z: Mann Whitney U test $\chi^2$: X-square test $F\chi^2$: Fisher's Exact X-square test
*p<0.01 statistically high significance

**Table 3. Mortality Rates Among Cases of Mushroom Poisoning Between the Years 1994-2004.**

| Amanita poisoning | Patient (n) | Mortality | % |
|---|---|---|---|
| Definite amanita | 8 | 4 | 50 |
| Highly suggestive of amanita | 11 | 3 | 27.3 |
| Total | 19 | 7 | 36.8 |

**Table 4. Comparison of Survivors and Dead in Amanita Poisoning**

| **Amanita poisoning** | | **Dead Mean±SD** | | **Survivors Mean±SD** | | **Test stat.; p** |
|---|---|---|---|---|---|---|
| Age (years) | | 7.07±1.79 | | 8.54±2.37 | | Z:-1.494; p:0.135 |
| Time of admission to hospital (hours) | | 37.14±20.67 | | 17.92±7.50 | | Z:-2.840; p:0.005** |
| AST (U/l) | | 2849.4±1891.9 | | 1891.9±469.6 | | Z:-3.550; p:0.001** |
| ALT (U/l) | | 3162.7±302.9 | | 2620.3±566.6 | | Z:-2.113; p:0.035* |
| PT (sec.) | | 28.20±1.30 | | 25.42±2.46 | | Z:-2.293; p:0.022* |
| Creatinin (mg/dl) | | 1.71±0.21 | | 1.03±0.29 | | Z:-3.347; p:0.001** |
| | n | % | n | % | | |
| Gender | female 5 | 71.4 | 6 | 50.0 | | $F\chi^2$ |
| | male 2 | 28.6 | 6 | 50.0 | | p:0.633 |

Z: Mann Whitney U test $F\chi^2$: Fisher's Exact X-Square test
*p<0.05 statistically significance ** p<0.01high significanc

its convenient ecological environment. In a study carried out in East-Anatolian part of our country, it is reported that 143 people between the yr 1996-2000 have been diagnosed and treated as mushroom poisoning and 12 patients died of fulminant hepatic failure.[7] The first symptoms seen were loss of consciousness, fatigue, dizziness, severe headache, abdominal discomfort and vomiting. In the present study, most of the cases admitted were with vomiting and diarrhea. In another study from Central Anatolia, 64 children with mushroom poisoning were enrolled, most of them had presented in May and June and gastrointestinal symptoms were most commonly seen symptoms of presentation.[8] Out of 64 cases, 5 patients (7.8%) had died of hepatic failure and all of them had delayed presentation. In a study in Iran between the yr 1992-2002, 37 cases of mushroom toxicity were recorded.[9] Most of them (80%) were admitted in autumn and the rate of hepatic encephalopathy was 50%. The rate of mortality changes in different parts of the world from 10-90%.[10,11] This difference depends on variations in amount of ingested amatoxin, individual susceptibility and probably due to difference in amatoxin content of different species of mushrooms all over the world.

Poisoning with mushroom having long incubation period is very dangerous and has significantly higher mortality rate.[12] As both types of poisoning mostly present with gastrointestinal symptoms, the time of appearance of symptoms gain importance in planning the treatment. In our study, the average time of appearance of symptoms after mushroom consumption, average duration of symptoms and hospital stay were significantly higher in patients with amanita poisoning (p<0.01) (Table 2). Besides this, the average time of admission to hospital after ingestion was significantly higher in patients who died of hepatic coma in amanita group (p<0.01) (Table 4). The mean AST, ALT, creatinin and PT values on 3rd day were also significantly higher in patients who died (Table 4). Therefore, all the cases that ended with mortality had late onset of symptoms.

Jager *et al* examined amatoxin in plasma, urine,

gastroduedonal fluid, feces and tissue in 45 patients by high-performance liquid chromatography method and found out that amatoxins can be found upto 5 days in urine and as the renal and hepatic failure results from the direct toxic effect of amatoxin, early elimination is very important in treatment.[13] Patients with history of mushroom ingestion should be hospitalized promptly no matter when the gastrointestinal tract symptoms start and alpha amanitin level should be checked, if possible. The aim is to eliminate the toxin from the body as soon as possible after the stabilization of the patient.

Many treatment modalities aiming detoxification are suggested by many different studies from all over the world.[14,15] However, since it is very difficult to carry out a controlled clinical study, no certain treatment modality could be settled. There are many studies with different results and treatment is more likely to be based on individual experiences. Extracorporeal detoxification techniques like hemodialysis and hemoperfusion and more recently MARS (Molecular Albumin Reabsorbent System) seem to be the most effective methods in elimination of the toxin.

We administered nonspecific antidotes like penicillin G, silibinin, corticosteroids and vitamin C in the patients that we suspected strongly of amatoxin poisoning, Penicillin G prevents binding of toxin to hepatocytes and together with steroids, also binds to serum albumin and enhances the elimination of the toxin from the kidneys.[16] Steroids are also effective in cases with shock and hypotension. Penicillin G in combination with silibinin is shown to be effective in studies conducted on mushroom poisoning.[17] Silibinin blocks the lysosomal proteases and by stabilizing the cell membrane, prevents the absorption of toxin by hepatocytes. In a study, 2108 patients diagnosed as mushroom poisoning in the last 20 yr in North America and Europe were investigated, silibinin was found to be effective, whereas steroids did not have any efficacy.[18] There are many more studies about efficacy of silibinin.[19] vit C is used for its cytoprotective effect. Effect of corticosteroids and vit C are still under debate.

Hemoperfusion is the most commonly used and life saving method of elimination of poison. It was first described by Chang in 1973. Hemoperfusion cleans alpha amanitin and neurotoxic amino acids like metionin, tryptophan and phenylalanine from plasma and helps in recovery from hepatic encephalopathy. The importance of early hemoperfusion in amanita poisoning has been emphasized in many studies.[15,20,21] In one study it is shown that early hemoperfusion by itself or together with hemodialysis and plasmapheresis decrease mortality due to renal and hepatic failure to a great extent.[22]

More recently, MARS seems to be the most promising dialysis method as in many studies its efficacy is referred to in treatment of mushroom poisoning even in cases with severe hepatic dysfunction and encephalopathy.[17,23,24,25] Extracorporeal Albumin Dialysis (ECAD) is also a choice of treatment in which the treatment has to start within 72 hr and it is shown to help hepatic regeneration.[26] Orthotopic liver transplantation is the only treatment modality in case of fulminant hepatic failure.[27,28] We did not have MARS and transplantation unit in our hospital.

In our patient group, hemoperfusion therapy was performed in 16 of the patients in the first 48 hr if possible. Eight of them were the patients in whom amatoxin was detected in plasma and the other 8 were found to be strongly suspected of amatoxin poisoning. In other words, hemoperfusion was performed regarding alpha amanitin levels, the incubation period (in case of delayed onset of symptoms), features of the ingested mushroom, family history of intoxication, clinical and laboratory data. It could not be performed in 3 patients because of late admission and these patients died of fulminant hepatic failure. Prognosis and life expectancy were better in the patients admitted to hospital earlier. 50% of the patients with definite amatoxin poisoning and 66.3% of the patients with possible amanita poisoning recovered.

## CONCLUSION

Early diagnosis and treatment in mushroom poisoning can be life saving. Every patient with the history of mushroom ingestion should be hospitalized, conventional therapy should be given and the patients should be followed up. Alpha amanitin levels should be checked as soon as possible, if amanita poisoning is suspected. If laboratory tests to detect the toxin cannot be performed the time of occurrence of symptoms, the features of the ingested mushroom, the clinical picture, and the family anamnesis can give defined important clues about the type of intoxication. Early hemoperfusion can be lifesaving in amanita poisoning. Moreover, providing public education on mushroom poisoning is extremely important in prevention of intoxication as well as encouraging early admission to hospitals.

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