MUSHROOM POISONING IN CHILDREN: CLINICAL PRESENTATION AND OUTCOME

Mohammad Ali Jan, Tahir Saeed Siddiqui*, Nasir Ahmed, Israr Ul Haq, Ziaullah Khan**

Department of Child Health, Saidu Teaching, Hospital Swat, *Department of Paediatrics, **Department of Medicine, Ayub Medical College, Abbottabad, Pakistan.

Background: A variable clinical picture characterizes mushroom poisoning. The Amatoxin, the main toxic component of these fungi, are responsible for gastrointestinal symptoms as well as hepatic and renal failure. As acute gastroenteritis is extremely common in our set up, so every patient presenting with these symptoms is treated as gastroenteritis of viral aetiology. The authors present the clinical picture of the phalloid syndrome, its treatment and immediate outcome. **Methods:** All children age less than 16 years admitted in Saidu Hospital Swat from January to December 2006 with mushroom poisoning were included in the study. Patients with doubtful history or with associated illness were not included. The diagnosis was based on the clinical picture of the patient, history and the laboratory data. In addition to maintenance of fluid and electrolyte balance and treating sepsis, oral Silymarin and intravenous penicillin was started. Liver function tests, renal functions tests, serum electrolytes and coagulation profile was done in all the patients. The severity of poisoning was graded according to hepatic transaminase elevations and prolongation of prothrombin time. **Results:** Of the 18 patients, fifteen were above five years of age. Female were twice in number. Fifteen patients developed hepatic failure and three patients developed renal failure. Thirteen patients expired. **Conclusion:** To start timely management, Mushroom poisoning should be considered in the differential diagnosis in patients presenting with food poisoning particularly coming in groups. Delay in diagnosis is associated with high mortality.

Keywords: Mushroom poisoning, Food poisoning, Amanita poisoning

INTRODUCTION

Mushroom poisoning refers to the severe and often deadly effects of various toxins that are found in certain types of mushrooms. Mushrooms are fungi, Saprophytic in nature, use organic material from dead plants and animals. Severity of mushroom poisoning depends on type of mushroom eaten, early diagnosis and correct treatment.[1] Fatalities due to mushroom poisoning are increasing world wide, more than 90% of deaths resulting from ingestion of amatoxin containing species.[2] If necessary expert knowledge is present, consuming wild mushrooms is relatively safe. However tragic deaths or illnesses can occur if toxic mushrooms are ingested unintentionally.[3,4] Ingestion of high doses of Amanita phalloides for suicidal purpose has been reported in literature.[5]

Clinical manifestations of Amanita phalloides are the result of the cyclopeptide toxins, Phalodin and alpha Amantinin. Phalodin is cyclic heptapeptide that interrupts the actin polymerization-depolymerization cycle and impair cell membrane function. It has limited GI absorption and cause gastroenteritis like effects.[6,7] Alpha Amantinin produces deleterious effects on liver and kidney when circulating in the blood .[7] Clinical features occur in four phases.[8] Latent phase lasts for 0.5-12 hours.[1] Gastroenteritis phase includes diarrhoea, vomiting, abdominal pain during which patient becomes severely dehydrated. During the phase of temporary improvement, patient feels well for 6–8 hours. During Hepatic and Renal phase (4^{th} and 5^{th} day) patient lapses into hepatic coma and renal failure ending in death.[9]

Patient's history and initial symptoms are important in diagnosis. It is important to obtain specimen of ingested mushroom if possible, as treatment is species specific.[10] Liver function tests can be deranged severely in seriously poisoned patients.[11]

The therapy includes 1: stabilization of the patient with the correction of hypoglycaemia and electrolyte imbalance, substitution with coagulation factors Fresh Frozen Plasma (FFP) and red cells and the treatment of septic complications, 2: decontamination, which consists of gastric levage, the administration of activated charcoal and laxatives as well as the forced diuresis, and 3: therapy with high doses of penicillin or ceftazidime and of silibinin.[8] Silibinin is the most promising new treatment for Amanita mushroom poisoning.[10]

Impending hepatic failure needs supportive measures including lactulose, low protein diet, vitamin K and fresh frozen plasma. Haemodialysis/Plasma Pharesis may be helpful in some cases. If response is not satisfactory liver transplantation[7] can be considered as final option depending on availability and affordability.

Purpose of present study is to present clinical features, treatment and immediate outcome in children with mushroom poisoning in our setup.

MATERIAL AND METHODS

This study was conducted in the Department of Child Health Saidu Teaching Hospital Swat between February and November 2006. A total of 18 cases of Mushroom poisoning were included in the study. All patients were below 15 years. Patients with

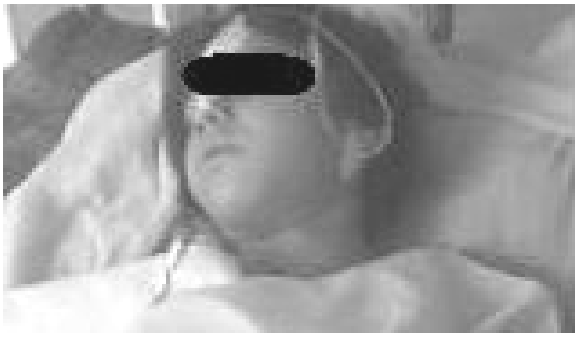
associated illnesses were not included in this study. Diagnosis was history based, when patients were specially enquired about mushroom ingestion when element of hepatic dysfunction was noted in patients presenting with acute gastroenteritis. All information was recorded on specially prepared proforma. After admission, supportive (intravenous fluids, oxygen, electrolytes replacement) treatment was started in all the patients. Specific treatment in the form of stomach decontamination, Silymarin and intravenous penicillin was started when history of mushroom ingestion was obtained. Silymarin was given orally due to non-availability of intravenous preparation. In patients who developed hepatic encephalopathy lactulose and vitamin K was also given.

Fresh frozen plasma was administered in cases with bleeding due to disturbed coagulation profile. All these patients were admitted as cases of acute gastro-enteritis/food poisoning. Thorough history was taken; only three of them clearly gave the history of Mushroom ingestion. Rests of the fifteen patients were treated as gastro-enteritis/food poisoning till the appearance of liver involvement, when they were specifically asked about mushroom ingestion. In all these cases along with non-specific tests (serum electrolytes, blood smear) liver and renal function tests were performed. Prothrombin time and hepatic transaminases were serially determined to grade severity of illness and to monitor progress of hepatic dysfunction.

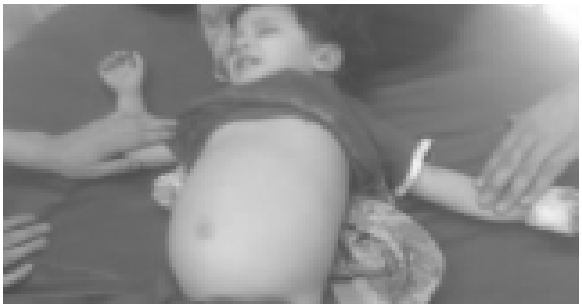
RESULTS

Out of 18 patients 6 (33.3%) were male and 12 (66.7%) were female. Three patients (16.7%) were between 1 and 5 years, six (33.3%) between 5–10 years and nine (50%) were between 10 and 15 years. The most frequently reported symptoms were vomiting (100%), diarrhoea (100%) with signs of moderate to severe dehydration and abdominal pain (83.3%). In 15 (83.3%) cases hepatic involvement was noted, 50% of them developed hepatic encephalopathy. In most of these cases signs of liver involvement were noted after forty-eight hours. Renal failure was noted in 4 cases (22.3%).

Non-specific laboratory investigations were inconclusive. Specific Investigations include total bilirubin level, SGPT, Prothrombin time, Blood urea and Serum Creatinine. Bilirubin was noted between 7 and 30 mg/dl and SGPT between 50 and 3000 IU. Prothrombin time was between 50 and 120 seconds. Thirteen (72.3%) patients expired and five (27.8%) patients survived and discharged home. The maximum stay in the hospital was one week and the minimum stay 24 hours. In survivors the liver involvement was minimal and there was no renal involvement. All of them presented with profuse vomiting and diarrhoea. In three of them the diagnosis was clear from the history with in two hours and the specific management, including gastric levage was started early. In families with high mortality, the diagnosis was not clear until they showed signs of liver involvement.



14-years old girl with Hepatic Coma



3-year old baby with renal failure and ascites

Table-1: Age-wise distribution

Age	No. of cases	Percentage
1–5 years	3	16.7%
5–10 years	6	33.6%
11–15 years	9	50%

Table-2: Sex-wise distribution of cases

Gender	No. of Cases	Percentage
Male	6	33.3%
Female	12	66.7%

Table-3: Area-wise Distribution

Area	No. of Cases	Percentage
Swat district	8	44.4%
Shangla district	7	38.9%
Kalam	3	16.7%

Table-4: Clinical presentation of Mushroom poisoning

Symptoms	No. of cases	Percentage
Diarrhoea	18	100%
Vomiting	18	100%
Dehydration	18	100%
Abdominal pain	15	83.3%
Jaundice	15	83.3%
Renal failure	4	22.2%

Table-6: Specific Laboratory investigations

Blood Index	No of Cases	Percentage
Serum Bilirubin (7–30 mg)	15	83.3%
SGPT 50–3000 IU	15	83.3%
Prothrombin time 50–120 S	13	72.2%
Blood Urea 80–200 mg/dl	4	22.2%
Serum creatinin 3–12 mg/dl	4	22.2%

DISCUSSION

In this study the male to female ratio is 1:2. In an Indian study female (59%) were more than males (41%),[11]

other people reported equal incidence. The double number of female could be due to the more herbivorous nature of females in our area and as most of the time the female brought the mushroom home. Maximum numbers of children affected in our study were between 5 and 15 years, similar to reports by other workers.[6,12] In some localities mushroom poisoning has been reported to be more common in children less than 6 years.[3]

Less severe cases can present with history of mushroom ingestion without any effects.[3] Most children have clinical signs and symptoms of acute gastroenteritis.[3,9,12,13]

In localities with wild mushrooms possibility of mushroom poisoning should be considered in differential diagnosis in children presenting with unexplained gastrointestinal symptoms.[14] Early diagnosis and treatment decreases morbidity and mortality.[11] In present study 100% patients presented with signs of acute gastroenteritis.

Acute liver failure as a result of acute mushroom poisoning is associated with high mortality despite optimum medical management.[15] Hepatic involvement is common and serious complication of mushroom poisoning. Jaundice is the early sign of liver involvement. In present study 15 (83.3%) patients had liver involvement and 50% of these developed hepatic encephalopathy. Pajoumand A *et al* reported similar results.[1] Signs of liver involvement were noted after forty-eight hours of admissions in most cases. Signs of hepatic encephalopathy were noted in most cases ending in death. Renal failure was noted in 4 cases and the earliest sign was oliguria. Alpha-Amanitin causes kidney damage in addition to liver damage.[7] Of all the laboratory parameters evaluated prolongation of prothrombin time and elevation of hepatic transaminases were associated with increased mortality rate in our study; similar correlation has been seen in other studies.[2] Duration of hospitalization was 1–7 days. Hospital stay depends upon severity of poisoning and timely aggressive management. Durkan P *et al* have reported 1–4 days duration of hospitalization without any mortality.[1]

The mortality rate in our study is high (72%) as compared to the studies done in developed areas.[2,12] The reasons of high mortality rate in our set up was delayed diagnosis and treatment as well as lack of specific treatment, for example, intravenous Silymarin, Haemodialysis and hemoperfusion[15], MARS (molecular absorbent regenerating system)[15], Acetylcysteine[7], liver transplantation.[7]

CONCLUSIONS & RECOMMENDATIONS

Search of literature shows that this study is the first of its kind in Pakistan and needs further research to decrease the mortality rate due to this highly lethal poisoning. Mushroom poisoning should be considered in all patients presenting as acute food poisoning, particularly patients coming in group from a family.

Even slight suspicion should lead to gastric levage at the earliest. For better outcome, Intravenous Silymarin should be made available. Intensive combined treatment applied is highly effective in improving patients with both moderate and severe amanitin poisoning, that's why it is recommended that haemodialysis should be performed in these severe cases of mushroom poisoning.

Central poison control centre should be established to provide instant identification of the poisonous mushroom. Public should be made aware by mass media to avoid eating unidentified mushroom.

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Address for Correspondence:

Dr. Ali Jan, Assistant Professor of Paediatrics, Saidu Teaching Hospital, Swat, Pakistan.
E-mail: alijan56@hotmail.com