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Mushroom Poisoning: A retrospective Study of Prognostic Indices to Predict Outcome

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[1#]Soutrik Dutta, [2]Anupam Dutta

[1]Post-graduate resident doctor (2nd year), Department of Medicine, Assam Medical College and Hospital, Dibrugarh, Assam
[2]Assistant Professor, Department of Medicine, Assam Medical College and Hospital, Dibrugarh, Assam

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#Corresponding author: post graduate student, Department of Medicine,Assam Medical College and Hospital, Dibrugarh, Assam. Email: soutrikdutta.kpc@gmail.com.

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ABSTRACT

Aim: To determine the environmental, clinical and laboratory parameters in cases of mushroom poisoning. To correlate those parameters with the outcome of the patient, calculate the Kings College Criteria(non-paracetamol) in cases with acute liver failure, and to correlate the King's college Criteria (non-paracetamol) with the outcome of the patient.

Materials and Methods: A hospital based retrospective study was done at a tertiary care centre of upper assam on the presenting patients showing symptoms after recent mushroom ingestion over the span of six months. Retrospective analysis of history charts, examination findings, laboratory investigations was done to determine environmental, clinical and laboratory parameters which were then correlated with the outcome of patients using statistical analysis. In the subset of patients showing acute liver failure, Kings college Criteria (non -paracetamol) was applied and correlated with the outcome.

Results: Out of 45 total patients ,41 patients could take part in study. Significant difference in mortality (p=0.003) was noted with delayed onset of symptoms (>6 hrs) when compared to early symptoms. 14 patients expired in our study. We found that AST,ALT ,PT,INR values from day 2 in expired group (AST,ALT>2500 and PT/INR>30/ 3.0)are significant with mortality.(p<.05).In our study it was found that there is a significant difference in mortality in those who present to hospital early and those who present late(p=0.01) .In our study ,out of nine patients who had documented acute liver failure , only four of them fulfilled kings college criteria and all of them expired ,thus having a 100 percent positive predictive value for mortality.

Conclusion: we have coupled all the biochemical and clinical factors which have a relationship with severity. We have shown that elevated liver enzymes (>2000), elevated coagulation profile, delayed time of onset of symptoms, delayed hospitalization, development of hepatic encephalopathy to be directly predictive of severe poisoning and fatality

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INTRODUCTION

Mushroom poisoning although rare is still prevalent in parts of the globe and has an increased number of mortality. In India also some regions suffer from this catastrophe every year leading to significant mortality There are around 5000 mushroom species all around the globe of which 100 species are said to be poisonous[1] Mushroom poisoning accounts for 50-100 deaths per year in western Europe.[1] Among the mushroom toxins, amatoxin causes 95 percent of the fatal cases.[2] *amanita phalloids,* a species of genus *amanita* produces amana toxin, a cyclopeptide which accounts for 90-95 percent of fatal poisoning in Europe.[3] A genus *galerina* also produces cyclopeptide, although reported poisoning by the latter is rare. Another fatal toxin to be found is gyometrin with a 50 percent mortality rate in the United States. Both these groups have a late onset of symptoms and symptoms are cholera-like. Both have effects on the liver and kidney although the latter group has effects on the nervous system and haematopoetic system Misidentification between edible and non-edible mushrooms plays an important role in mushroom poisoning. In India, mushrooms forms an important part of the diet in ethnic populations. Most of the cases of mushroom poisoning occur due to consumption of wild mushrooms. Though traditional wisdom helps in differentiating edible from the poisonous variety, poisoning mainly occurs due to misidentification. In india the reported cases are mainly from tribal areas of South India, the Eastern ghats, Northern India and north eastern part of the country though many cases go unreported. Mushroom poisoning usually happens in certain months of the year, especially the first monsoons. There are many factors which contribute to this fatality and such factors can be used as prognostic markers. Many studies conducted in India and world wide have demonstrated the relationship with various biochemical and clinical and environmental parameters with the severity of poisoning. A study by A Joshi suggests that the effect of amanita is mainly on the liver and kidneys and fatality is also due to the involvement of the two .[4] In a study by Mohammad Ali Jan it was noted that in 83 percent cases there is involvement of liver with 50 percent having hepatic encephalopathy.[5] In the same study it was also noted that the earliest sign of renal damage was found to be oliguria. As mushroom poisoning causes an acute liver and renal failure, an understanding of the relationship with various biochemical, clinical and environmental parameters with severity of the poisoning will help us to triage. In our study, we aim to determine the prognostic factors which could predict mortality by retrospective analysis of hospital records. The fatalities suffered after mushroom ingestion is mostly due to the amanatoxins present in certain species. This toxin mainly affects the liver and kidney resulting in multi organ failure. As the mushroom toxins cause acute liver failure, we also aim to use the kings college criteria (non paracetamol) (Figure.1) for assessing the outcome of mushroom induced acute liver failure.[6]

MATERIALS AND METHODS

Duration of the study - April 2020 to September 2020 Study type-Hospital based Retrospective observational study. Study Place - Department of medicine and Paediatrics of a tertiary health care centre in Dibrugarh, Assam. Study population-Different parts of upper Assam including certain parts of Arunachal Pradesh presenting to our institution with symptoms after mushroom ingestion. Sample – All the patients presenting to the hospital with symptoms after intake of mushroom during the study period. Insclusion Criteria – All the patients presenting to the hospital with symptoms after intake of mushroom during the study period.

Exclusion Criteria

1. Patients who did not gave informed consent
2. Patients who absconded from the hospital before the study was complete Laboratory parameters AST and ALT was done by UV and P5P method.

Total bilirubin and fraction was calculated by Diphylline diazonium salt method and PT/INR by Coagulometer CA-500 series Important definitions. Acute liver failure ALF is defined as the development of severe acute liver injury with encephalopathy and impaired synthetic function (INR of 1.5 or higher) in a patient without cirrhosis or preexisting liver disease and with an illness of fewer than 26 weeks duration.[7] Hepatic Encephalopathy is an altered level of consciousness as a result of liver failure. Its onset may be gradual or sudden. Other symptoms may include movement problems, changes in mood, or changes in personality.[8]

West haven criteria

The severity of hepatic encephalopathy is graded with the West Haven Criteria; this is based on the level of

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impairment of autonomy, changes in consciousness, intellectual function, behaviour, and the dependence on therapy.

- Grade 0 - No obvious changes other than potentially mild decrease in intellectual ability and coordination
- Grade 1 - Trivial lack of awareness; euphoria or anxiety; shortened attention span; impaired performance of addition or subtraction
- Grade 2- Lethargy or apathy; minimal disorientation for time or place; subtle personality change; inappropriate behaviour
- Grade 3- Somnolence to semistupor, but responsive to verbal stimuli; confusion; gross disorientation
- Grade 4- Coma

Table 1. King's college hospital criteria for liver transplantation in acute liver failure.[6]

Patients with paracetamol toxicity
pH < 7.3 or
Prothrombin time > 100 seconds and
serum creatinine level > 3.4 mg/dL (> 300 µmol/l) if in grade III or IV encephalopathy

Other patients

Prothrombin time > 100 seconds or
Three of the following variables:

- Age < 10 yr or > 40 years
- Cause:
  - Hepatitis C or E
  - halothane hepatitis
  - idiosyncratic drug reaction
- Duration of jaundice before encephalopathy > 7 days
- Prothrombi®n time > 50 seconds
- Serum bilirubin level > 17.6 mg/dL (> 300 µmol/l)

Acute Kidney Injury

Based on KDIGO

AKI can be diagnosed if any one of the following is present: [9]

- Increase in SCr by ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours; or
- Increase in SCr to ≥1.5 times baseline, which has occurred within the prior 7 days; or
- Urine volume < 0.5 ml/kg/h for 6 hours.

Methodology

After informed consent, proper records of history including diet history and past history are accessed and noted. Monitoring charts of initial symptoms along with charts of evolution of symptoms of organ failure were accessed. Detailed clinical examination done at admission and also during hospital stay at intervals were accessed. Routine laboratory investigations including Liver function tests done during hospital stay were also recorded. Patients were given treatment according to our our institiutional protocol (Ryles tube indwelling, 200 ml/kg body weight of fluids: injection N-acetyl cysteine@150 ml/kg body weight over 15-30 minutes, followed by 12.5 mg/kg/hour over 4 hours followed by 6.25 mg/kg/hour for 16 hours; Injection vitamin C 3 gm iv till clinical improvement.)This protocol was followed only in those patients who presented to hospital within 24 hours of ingestion. Gastric lavage has been done in all those who presented within 6 hrs. Syrup Silymarin, injection vitamin K, mannitol infusion were given according to requirement in cases of acute liver failure. Clinical and biochemical improvement / deterioration which was recorded was accessed. All these environmental, clinical and laboratory parameters were then determined and correlated with outcome (survival or mortality). Kings College Criteria (non paracetamol) was applied to each patient. The prognosis predicted by the Kings College Criteria was then correlated with the outcome in each patient. Statistical analysis was done using Chi square tests and Student T test. Ethical clearance was taken from the instititutional Ethics Committee before starting the study.

RESULTS

Forty five cases were admitted in both department of Medicine and Paediatrics in the study span Out of them four did not consent for the study. Thus, forty one cases were included in our study after informed consent.These patients were divided into thirteen cohorts.

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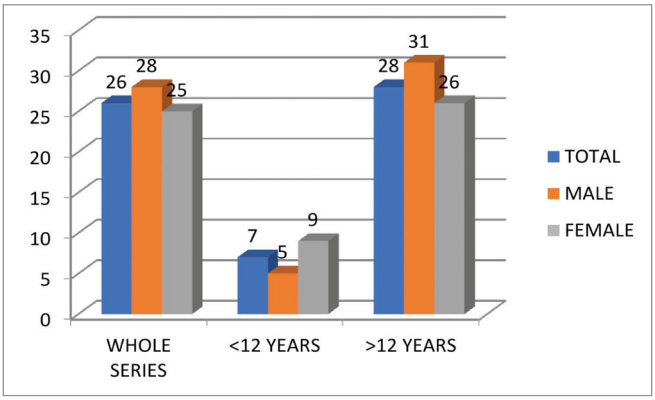
Tables 1: Table showing gender distribution of patients

	Total	<12	>12
Male	19	2	17
Female	22	2	20

Chart 1: graph showing age distribution of patients



Tables 2: Table showing mortality pattern vs gender distribution

	Total	<12	>12
Male	6	1	5
Female	8	2	6
Total	14	3	11

Chart 2: graph showing gender-wise mortality

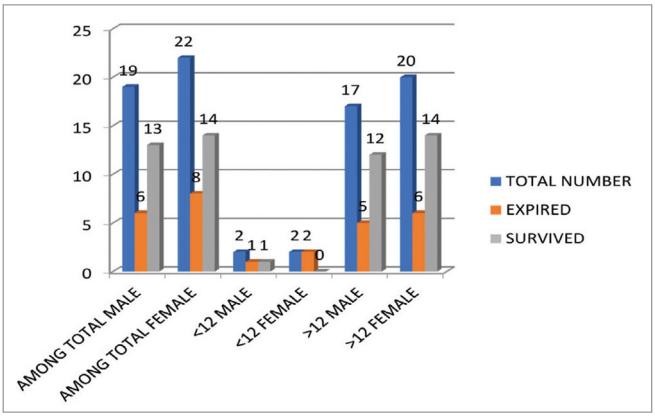
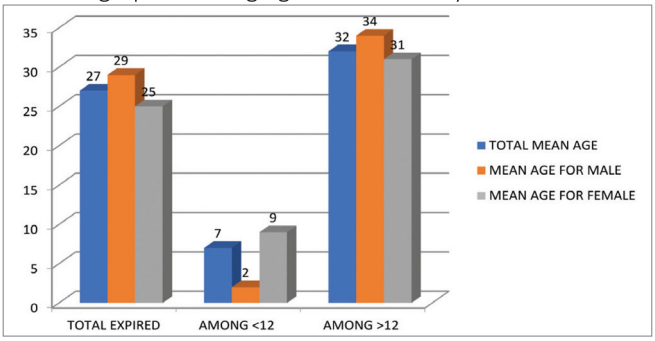


Chart 3: graph showing age wise mortality



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