AMANITA MUSHROOM POISONING: A STUDY OF CLINICAL CHARACTERISTICS AND OUTCOME OF TOXICITY

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Objective: To analyze the clinical characteristics and outcome of amatoxin poisoning cases

Methods: We performed a retrospective study from Ramathibodi Poison Center Toxic Exposure Surveillance System, during a three-year period. The diagnosis was made from the history of mushroom exposure and delayed onset (> 5 hours) of gastrointestinal symptoms after eating mushrooms

Results: There were 30 consultations with totally 55 poisoning cases. Most cases were male (51%) and from the north-east region (71%). The mean onset of gastrointestinal symptoms after taking mushrooms was about 10 hours. Two cases presented to the hospital with hypotension. Hepatitis, acute kidney injury (AKI), jaundice and coagulopathy accounted for 74%, 46.3%, 44.7% and 40% of cases, respectively. Interestingly, 27% of all had increased indirect bilirubin levels without definite evidence of hemolysis in some cases who were investigated. Almost all of cases (92.7%) were admitted to the hospital and the median of hospital stay was 4 days (1-28 days). The mortality rate was 29%. Seventy-three percentages of cases received the treatment including multiple-dose activated charcoal (67.5%), intravenous N-acetylcysteine (87.5%) and benzylpenicillin (45%). In 60% of cases, the treatment was initiated within 24 hours after eating mushrooms. Exchange transfusion and liver transplantation were performed in one severe case, however, this case was dead eventually. Because intravenous silybinin is not available in Thailand, we used oral silymarin instead. Eight cases who all had hepatitis, were treated with oral high dose silymarin (5 cases with 4.48 g/day, 2 cases with 1.68 g/day and 1 case with 1.4 g/day) for a couple days. One of these cases was dead, nevertheless, this case was treated quite late (>72 hours after taking mushrooms) and received the 1.68 g/day dose. Thus, the fatality in oral silymarin treatment group was 12.5%. We performed the subgroup analysis into 2 groups; the dead and survived group. We found that hepatitis, initial and peak serum aspartate transaminase, initial and peak serum alanine transaminase, AKI were statistically significantly different ($p < 0.01$) between 2 groups. While the age, sex, onset of gastrointestinal symptoms, increased indirect bilirubin, the interval from eating mushrooms to starting treatment (day) and different types of therapies showed no statistically significant difference.

Conclusions: Amanita mushroom poisoning still caused high fatalities. Serum transaminase and creatinine might be prognostic factors. Increased indirect bilirubin levels need further studies to elucidate the pathophysiology. Treatment with oral high dose silymarin should be investigated more as the novel therapy in amatoxin poisoning.