

OP – 06

Toad poisoning: clinical characteristics and outcomes.

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Objective: Toad or bufotoxin poisoning has been reported in cases reports. However, the clinical data of this poisoning is not well described and still very limited. This study was performed to describe and analyze the clinical characteristics and outcomes of the cases of toad poisoning in Thailand.

Methods: We carried out a retrospective cohort study of the cases of toad poisoning from Ramathibodi Poison Center Toxic Exposure Surveillance System, during a 5-year period (2012-2016).

Results: There were 24 consultations with totally 36 poisoning cases. The median age was 31 years-old (8-month-old to 93-year-old). Most cases were male (66.7%) and from the north-east region (33.3%). Most (43.6%) ingested eggs of toads. The most common presenting symptom was gastrointestinal (GI) symptoms (91.7%) with the median onset of 2 hours after ingestion. Fourteen cases presented with bradycardia, 9 cases with shock and 1 case with cardiac arrest. Initial EKG showed sinus bradycardia (37%) and one case had complete heart block. No patients complained of visual abnormalities. Two patients developed cardiac arrested during management in emergency room within only 30 minutes after ER visit. The EKG of these 2 patients with cardiac arrest was recorded as pulseless electrical activity. Four cases developed bradycardia including second-degree AV block Mobitz type 2 during hospitalization. No tachyarrhythmia such as ventricular tachycardia (VT) was detected in all patients. Some (11.5%) had hyperkalemia at presentation. Seven cases were measured for serum digoxin levels by immunoassay method, 5 cases were detected serum digoxin levels (0.43 - >8) at 2-11 hours after ingestion. One with cardiac arrest had serum level more than 8 ng/mL at 2 hours after ingestion. Most cases (69.4 %) were admitted in the hospital including intensive care unit (8.3%) and the median of hospital stay was about 24 hours (17-120 hours). The mortality rate was 8.3% from all who ate eggs and developed cardiac arrest. Most cases received supportive care including gastric lavage (33.3%), activated charcoal (33.3%), inotropic drugs (13.8%) and management of hyperkalemia (13.9%). One received intravenous calcium for hyperkalemia, but did not develop arrhythmia after calcium administration. One case received digoxin immune FAB, after that he clinically improved and had resolution of arrhythmia about 24 hours after treatment.

Conclusion: Toad poisoning commonly caused GI symptoms, bradycardia and bradyarrhythmia. However, in severe cases, death could occur. Tachyarrhythmia including VT was not found. Supportive and symptomatic care might be the main therapies in this type of poisoning.