SHORT REPORT

Life-threatening episode after ingestion of toad eggs: a case report with literature review

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It is known that toad possesses several toxic substances in the skin and parotid glands. In the past, toad-venom poisoning had been reported from ingestion of toad soup, Kyushin and aphrodisiac pills, but the poisoning from toad eggs is observed for the first time.

The case of a healthy female who had previously eaten toad soup twice without any discomfort is reported. She developed gastrointestinal symptoms and life-threatening cardiac rhythm after ingestion of toad eggs.

A 19-year-old woman presented at the emergency department of our institution at 5:00 h with nausea, vomiting, dizziness, numbness over her mouth and general weakness. She stated that about 1 h earlier she had ingested cooked toad eggs. She was previously healthy, she reported, and had eaten toad soup twice without any discomfort. Vital signs included blood pressure 92/52 mm Hg, a pulse of 90 bpm, a respiratory rate of 24 bpm and normal body temperature. Her pupils were 4 mm reactive to light, bilaterally. No neck stiffness or focal neurological deficit was apparent. The patient's cardiac and lung examination proved to be grossly unremarkable. Mild epigastric tenderness without any peritoneal signs was apparent.

At the emergency department, an initial electrocardiogram showed first-degree atrioventricular block (fig 1A). Subsequent haematological investigation found a white cell count of 13 200 cells/ml, a haemoglobin level of 15.6 mg/dl, a haematocrit value of 45.7% and a platelet count of 342 000/mm^3. A comprehensive metabolic profile revealed the following concentrations: sodium 133 mmol/l, potassium 7.1 mmol/l, pH 7.258 and bicarbonate 19.7 mmol/l. The patient's liver and renal function test provided grossly unremarkable results. A serum–digoxin concentration of 2.42 nmol/l was noted. The patient became progressively more disoriented and lethargic as time passed. The electrocardiogram followed subsequently by complete atrioventricular block (fig 1B)?

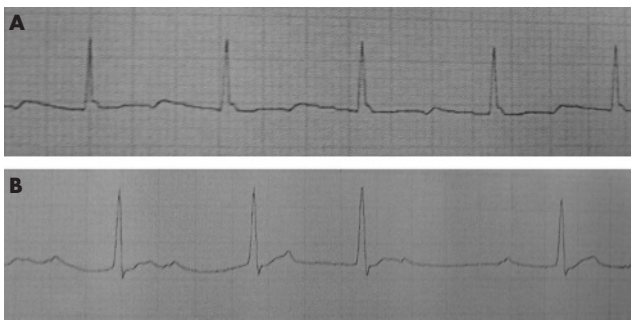

Figure 1 Initial ECG acquired in the emergency department showing (A) first degree atrioventricular block, followed by (B) complete atrioventricular block.

A transvenous temporary pacemaker was used promptly to prevent from lethal bradyarrhythmia. Prevention of further gastrointestinal absorption of toxin was undertaken with gastric lavage and activated charcoal. Hyperkalaemia was managed with insulin, glucose water and sodium bicarbonate. A digoxin-specific Fab fragment was ordered at the recommendation of the National Poison Center of Taiwan, Taipei, Taiwan

At this time, the patient was admitted to our intensive care unit for observation and life support if necessary. A total of 120 mg (an initial dose of 80 mg and a dose of 40 mg at 1 h after administration) of Fab fragment was administered to this young woman 4 h subsequent to her admission. The potassium and digoxin level declined gradually after admission. The patient's condition improved noticeably thereafter, and she was discharged in fair condition on the fifth day.

To the best of our understanding, toad-venom poisoning is rare but potentially life threatening[2–9]. Toads typically feature a variety of toxic substances in the skin and parotid glands and, even more so in the eggs of some species, such toxic substances are often resistant to break-down or denaturing by cooking.[1 3 4 7 9] Chan Su, a traditional Chinese drug, and Love stone, a topical aphrodisiac, are both made from dried toad venom. Deaths have occurred after ingestion of these products, and the clinical course resembles digoxin toxicity.[6 8 9] A number of toad species possess several toxic substances in their bodies. Poisoning by bufagins, catecholamines and indolealkylamines may occur after ingestion of toad tissue as food.[3 10] To the best of our knowledge, most patients who experienced such poisoning showed gastrointestinal symptoms consisting of nausea, vomiting and abdominal discomfort. Toad-toxin poisoning typically manifests primarily as digitalis-like toxicity-featuring cardiac effects, including bradycardia, atrioventricular block, ventricular tachycardia, ventricular fibrillation and even sudden death.[1–7] Patients having such intoxication usually feature unexplained bradycardia, possibly with a normal blood-pressure level. Laboratory studies may reveal hyperkalaemia and a detectable digoxin concentration in the afflicted individual's serum.[5–7 10]

The primary treatment modality for such cases of toad-venom intoxication is life support.[3] If such venom is ingested, treatment should be directed at the prevention of absorption of further toxin this includes—emesis, gastric lavage and the administration of activated charcoal and cathartics. Atropine, the insertion of a pacemaker and the administration of a number of different antiarrhythmic agents may be helpful in treating such cardiovascular toxicity. The majority of patients suffering from toad poisoning feature a substantially increased serum-potassium concentration, which is frequently associated with a poor prognosis and a rather high death rate.[5] An increased serum-potassium concentration features prognostic implications for individuals allegedly falling victim to toad poisoning, especially if hyperkalaemia develops, such that

subsequent treatment needs to be more aggressive than might initially be deemed necessary, in order to prevent death.[5] Immediate administration of digoxin-specific Fab antibody (an initial dose of 380 mg would appear helpful) has been reported to be life saving.[4 8] Optimal dosing of digoxin-specific Fab does not appear to be based specifically on the concentration of digoxin owing to the possibility for partial cross-reactivity. If there appears to be no clinical response to the administration of a digoxin-specific Fab fragment after around 30 min to one hour after administration, a repeat dose of 190–380 mg of this agent should be considered. Calcium should not be administered for the treatment of hyperkalaemia because it will, secondarily, increase intracellular calcium and thus elicit transient depolarisation.[5]

To the best of our knowledge, toad-venom poisoning is rare but can be life threatening. A pronounced clinician awareness of the clinical toxicity of such toxins (such intoxication often leading to digoxin-like cardiovascular and gastrointestinal effects) is clearly necessary for optimal management of such patients. Increased vigilance and prompt initiation of life-supportive measures associated with definitive treatment with digoxin-specific Fab fragments can be life saving.

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Informed consent was obtained from the patient for publication of their details in this paper.

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