Case Report

Myoglobinuric acute renal failure following cardioversion in a boxfish poisoning patient

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Introduction

Trunkfish are unique in that their bodies are enclosed in a hard box-like shell, and include boxfish, cowfish and turretfish. Members of the family Ostraciidae secrete an ichthyotoxic mucus from their skin when stressed or disturbed [1]. Fish exposed to this secretion develop irritability, gasping, quiescence, decreased rate of opercular movement, loss of equilibrium and locomotion, sporadic convulsions and death [2]. Rhabdomyolysis, a destructive skeletal muscle disease, can be caused by crush injury and other non-traumatic circumstances, e.g. alcoholism, viral infection, metabolic disorders, myopathies, drugs, etc. [3,4]. It is usually associated with elevated muscle enzymes in the serum and myoglobinuria. We report the case of a patient who suffered boxfish (Ostracion meleagris meleagris) poisoning, resulting in a cardiopulmonary catastrophe. Although cardiopulmonary resuscitation (CPR) was successful, the patient developed myoglobinuric acute renal failure as a result of the CPR electrical countershocks.

Case

A 51-year-old man ate three specimens of boxfish (Ostracion meleagris meleagris) and 30–40 min later he developed general discomfort, diaphoresis and dyspnea. He was immediately transferred to a local hospital where, due to respiratory failure, he underwent endotracheal tube insertion. He was then transferred to our hospital. On arrival at the emergency room, his blood pressure was 39/23 mmHg and an electrocardiogram showed ventricular fibrillation. The patient received CPR for 50 min, 10 electrical countershocks at a total of 3220 J, and multiple boluses of epinephrine, lidocaine and sodium bicarbonate. After successful CPR, an electrocardiogram revealed sinus rhythm without myocardial infarction. Initial laboratory data showed a white blood count of 23,500/mm³ with neutrophil 82%, haemoglobin 16.4 g/dl, haematocrit 49.4%, platelet count 236,000/mm³, serum urea nitrogen (BUN) 15 mg/dl, creatinine (Cr) 1.1 mg/dl, sodium 142 mmol/l, potassium 7.7 mmol/l, total calcium 7.9 mg/dl, phosphate 3.9 mg/dl, albumin 2.6 g/dl, and metabolic acidosis (pH 6.95, HCO₃ 8.8 mmol/l). The creatine phosphokinase (CPK) level after CPR was 375, 705 U/l (100% MM form), urinalysis showed proteinuria of 500 mg, 20–22 red blood cells/high power field, occult blood 4+, and positive myoglobin reaction. Diuretic treatment and urinary alkalinization with intravenous fluid supply was begun in the following days. During the second day of admission, the patient’s serum Cr increased to 3.2 mg/dl and his daily urine output declined to 190 cc, but renal ultrasound examination showed no abnormality. Due to remarkable fluid accumulation and exacerbated azotemia, the patient received a first haemodialysis session. The serum Cr level peaked at 8.9 mg/dl during the fifth day of admission. The CPK level decreased to 769 U/l during the ninth day of admission. The oliguric phase continued into the twentieth day of admission. In total, the patient received nine haemodialysis sessions in the hospital. He was discharged on the thirty-fifth day of admission. At that time, his serum Cr level was 7.5 mg/dl. The final serum Cr level was 2.8 mg/dl 3 months later. In the outpatient clinic, the patient complained of weakness but physical examination did not find any muscle disability.

Discussion

The Ostraciidae fish family is distributed throughout the tropical and semitropical seas of the world, where it is usually found in shallow water. The Ostraciidae
family is comprised of 13 genera containing ~30 species that secrete a toxin when agitated. Intra-peritoneal injection of ostracitoxin into mice has been reported to cause ataxia, laboured breathing, coma and death [2]. The minimal lethal dose in mice was 0.2 mg/g bodyweight. Because there have been no reports of such poisoning among humans, a review of the literature produced no toxic manifestations among humans or amounts of toxin lethal to humans. After eating Ostraciidae, our patient developed acute respiratory failure resulting in cardiac arrhythmia, resembling the toxic effects noted in the experiment with mice above.

Rhabdomyolysis with acute renal failure due to crush injury was first recognized during the Battle of Britain in 1941 [5]. Since then, there have been many non-traumatic cases reported sporadically. Electrical injury is one of the many causes of rhabdomyolysis [6,7]. Electrical injury involves tissue damage caused by heat generation and coagulation necrosis related to the intensity, duration and pathway of the electrical current and tissue resistance. The current of a lightning bolt may range from 12 000 to 20 000 A, with a voltage of ~1 billion volts, and have a duration of 1/1000 to 1/100 s. Severe muscle injury and myoglobinuria have rarely been associated in cases of lightning strikes, with myoglobinuria reported in one case only [8]. However, in two other reports, patients with acute myocardial infarction were shown to develop myoglobinuric acute renal failure after cardioversion [6,7], although the exact amount of current was not recorded. During CPR procedure, our patient received 3220 J, which probably caused rhabdomyolysis. Rhabdomyolysis may induce acute renal failure because of myoglobin’s direct toxic effect on distal tubular cells or its obstruction of the lumen of distal tubules. Dehydration, acidosis and decrease in renal perfusion may also contribute. It is crucial to manage myoglobinuria with volume expansion, alkalinization of the urine and administration of mannitol to prevent the development of acute tubular necrosis. The prognosis is good in non-traumatic myoglobinuria.

In conclusion, we report a very rare case of boxfish poisoning by Ostraciidae, resulting first in respiratory failure and ventricular fibrillation for which CPR was performed. The patient received 3220 J of counter-shocks leading to myoglobinuric acute renal failure. Prompt diagnosis and therapy are essential for adequate management of such patients.

References


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