PUFFER FISH POISONING: FOUR CASE REPORTS

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SUMMARY

Four cases of puffer fish poisoning with one death in a family are described. Acute respiratory cessation probably resulted from both depression of the medullary centres and muscular paralysis by the neurotoxin. Artificial ventilation is the single most effective therapeutic measure and should be considered in all severe cases. Delay in treatment may result in death. Anticholinesterases are not known to confer any benefit. Medical staff in coastal hospitals should familiarise themselves with the management of puffer fish poisoning.

INTRODUCTION

Puffer fish (Tetraodontidae), also known as globe fish or blow fish and locally called ikan buntal, is commonly found in the shallow waters around Malaysia (Fig. 1). It is easily caught and when irritated or landed, it takes in air or water into a diverticular sac in its gullet and blows itself up into its typical spherical shape. There are several species of puffer fishes in the Malaysian waters. All are poisonous and only one, T. lunaris — or ikan buntal pisang — is occasionally eaten by

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village fishermen along the Malaysian, Indonesian and southern Philippine coast.

A family of four who became extremely ill shortly after eating puffer fish is reported here. One of them died, the remaining three were hospitalised for a period between two to seven days.

CASE REPORTS

A Filipino family of four consisting of father, daughter, son and daughter-in-law had a meal of fried puffer fish which the men had caught earlier in the coastal waters off Sandakan. The species of the puffer fish is not known. Although it is common knowledge that the fish is poisonous, the father had apparently eaten it before in the southern Philippines without any ill effect and had persuaded the rest of the family that it was safe for consumption. Within half an hour, both father and son who had consumed the bulk of the puffer fish meal developed severe symptoms of poisoning. The women developed symptoms two hours later.

CASE 1

Initially, the father, aged 60, complained of flushing in the face. This was rapidly followed by numbness and paraesthesiae of the lips and tongue, spreading to the fingers. He vomited several times. Speech became slurred and he complained

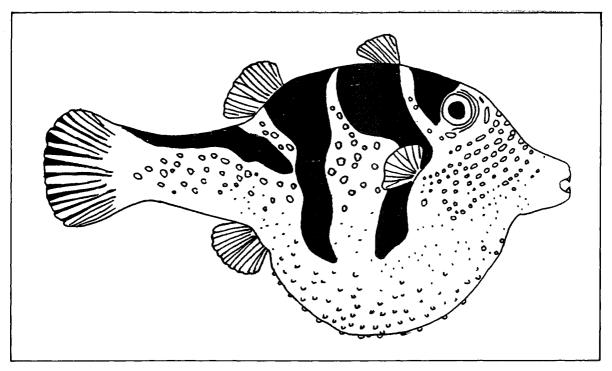


Fig. 1 Puffer fish (Tetraodontidae).

of blurred vision. There was extreme weakness of his arms and legs and he had to be carried into the casualty department.

The casualty doctor reported a low blood pressure (100/60) with a pulse rate of 50. The patient had bulbar palsy and was unable to open his eyes or move his limbs. His breathing was shallow. While being moved from the casualty department to the ward, he suddenly stopped breathing, and had a cardiorespiratory arrest. Attempt at cardiopulmonary resusitation failed. The time from ingestion of the fish to death was approximately one-and-a-half hours. No ECG was done in casualty.

Case 2

The son, aged 29, was admitted at the same time as his father. The initial symptoms were identical and he also had to be carried into the casualty department. He had bulbar palsy; his speech was slurred and he had marked bilateral facial weakness. There was bilateral ptosis with opthalmoplegia.

Pupillary reflexes were sluggish. All tendon reflexes were reduced, and there was marked weakness in all limbs (MRC grade 1-2). His breathing was shallow but not laboured and he was not cyanosed. His distress arose mainly from difficulty in swallowing his saliva. The blood pressure was normal and no rhythm disturbances were present on the ECG. Serum electrolytes, calcium, liver function tests were all normal.

He was given oxygen, intravenous chlorpheniramine (10mg) and hydrocortisone (200mg) and put on an ECG monitor. A tensilon test (Ephodronium hydrochloride 10mg) showed little objective improvement in limb weakness or respiration but he was started on pyridostigmine 30mg four hourly on an empirical basis for the first three days. There was no further deterioration in respiration and no ventilatory support was needed. He made a slow recovery and was eventually able to sit up on the third day, speak without slurring on the fourth and walk with minimal aid on the fifth day. Full recovery occured within a week.

Cases 3 and 4

The daughter was aged 30, with the daughter-inlaw being 31. The two women had less marked symptoms and presented some four hours after the meal with nausea, vomiting, dizziness and paraesthesiae. There was minimal weakness in their limbs but blood pressure, pulse rate and reflexes were all normal. All basic investigations including ECG were within normal limits. They recovered fully and were discharged on the second day.

DISCUSSION

The poison or tetrodotoxin of the puffer fish is a neurotoxin concentrated mainly in the liver and intestines. The flesh is relatively safe to eat and is considered a delicacy (called 'fugu') in Japan where it is usually eaten raw after careful preparation by trained and licensed chefs. Normal cooking processes however do not destroy the toxins which can stand prolonged boiling for up to four hours.

Pharmacological studies have shown that the primary action of the tetrodotoxin is on the nervous system and produces both central and peripheral effects. Intravenous injection of the poison in cats results in severe vomiting with rapid cessation of respiration and a precipitous fall in blood pressure. There may also be a marked reduction in myocardial contractibility.3 It is believed that the tetrodotocin has a direct action on the respiratory and vasomotor centres with severe inhibition of the sympathetic nerve fibres supplying the myocardium;^{1,3} there does not seem to be a direct cardiotoxic effect. The toxin also causes marked muscular paralysis by rapid and severe inhibition of conductivity of the motor fibres.^{1,3} The site of action does not seem to be at the neuromuscular junction and there is at present no known antidote to the toxin:

Treatment of puffer fish poisoning includes initial him. gastric lavage to suffer any remaining gastric toxins.

Because our patients had all vomited profusely by The time they were seen, gastric lavage was not done.

Furthermore, gastric lavage in the presence of bulbar palsy is not without risk of aspiration pneumonitis. Intravenous fluids, chloripheniramine, and steroids are of unproven benefit but are often recommended and were given in our three patients who survived. Oxygen should also be given to reduce cerebral hypoxia.

Artificial ventilation is the single most effective therapeutic measure in preventing death from respiratory arrest, and should be considered urgently in all severe cases.3 Although anticholinesterase drugs have not been found useful in puffer fish poining,3 pyridostigmine was used following the tensilon test is one of our severe cases (Case 2) in the knowledge that it probably did no harm and in the hope that it could benefit. This was before a search of the literature showed it to be of no proven benefit. Subsequent events suggested that the pyridostigmine probably had little effect on the recovery time of Case 2. All patients with puffer fish poisoning should have daily ECGs and cardiac activity should be monitored in the first 24 hours, ECG abnormalities in such cases are however unusual and animal experiments have indicated that only occasionally sinus bradycardia, A/V nodal block and atrial inactivity may be seen.³ All our three patients who survived had normal ECGs throughout their hospital stay.

Puffer fish poisoning is one of the three most common types of icthyosarcotoxic fish poisoning in the world. It is also the most lethal. The mortality from puffer fish poisoning remains disturbingly high at around 60%, 1 and death usually occurs within the first 24 hours after ingestion of the toxin.

Case 1 probably died of sudden respiratory arrest followed by a cardiac arrest. (Post-mortem was refused by the relatives.) Early intubation and artificial ventilation is casualty might have saved him.

Because our patients had all vomited profusely by These case reports underline two important the time they were seen, gastric lavage was not done. points. Firstly, early onset of severe symptoms

probably indicate ingestion of a large and potentially lethal amount of the toxin. The toxin is extremely potent. Animal experiments have shown that the intraperitoneal L.D. 50 for the cat is less than 10 μ g/kg, and the lethal ingested dose for humans is less than 1 mg.^{1,2} Secondly, urgent artificial ventilation should be considered in all cases of severe puffer fish poisoning. Blood pressure, respiration rate and electrocardiographic activity should be closely monitoried, and oxygen given. Delay in treatment may result in death. Doctors in coastal hospitals should therefore be aware of the potentially lethal complications of puffer fish poisoning and the need for early treatment.

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