Puffer Fish Poisoning : Clinical Features and Management Experience in 25 Cases

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Abstract

Between 1989 and 1999, 25 cases of puffur fish poisoning (PFP) were admitted to the medical service of Chon Buri Hospital. The severity of the poisoning was classified into four stages based on clinical signs and symptoms of PFP. Of the 25 patients, 23 were males and 2 were females. Three patients were in stage 1, four were in stage 2 and eighteen were in stage 4. Paresthesia was the early presenting complaint of all patients. Paresthesia consisting of either numbness or tingling of lips, tongue, around the mouth, hands, and feet. Muscle weakness, dizziness, vertigo, nausea and vomiting were common complaints. Eighteen patients developed acute flaccid paralysis and respiratory failure requiring ventilatory support. All patients received symptomatic and supportive treatment and general supportive care, including gastric lavage and intravenous fluid. Intubation and mechanical ventilation was considered especially when paralysis was progressing rapidly. Most were taken off the respirator 12-48 hours later. All patients completely recovered without any sequelae. Clinical features of PFP, toxicity of puffer fish and management were discussed.

Key word : Puffer Fish Poisoning

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Illness resulting from seafood consumption is not uncommon. It usually takes the form of gastroenteritis, probably bacterial or viral in origin, or of an acute allergic reaction confined to a few hypersensitive individuals and some marine illnesses which result from toxic fish and shellfish consumption[1-4]. Between 1994 and 1996, an outbreak of horseshoe crab poisoning by eating toxic eggs of the horseshoe crab Carcinoscorpius rotundicauda affected over 100 persons in Chon Buri which

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is located on the eastern coast of Thailand. This was the first large outbreak of horseshoe crab poisoning recognized in Thailand. The horseshoe crab toxin was identified as tetrodotoxin (TTX)[5].

Puffer fish poisoning (PFP) is not uncommon in Thailand[6]. In this paper, the author reviews the experience with 25 cases of PFP seen at Chon Buri Hospital from 1989 through 1999.

MATERIAL AND METHOD

The medical records of 25 patients with PFP, admitted to Chon Buri Hospital between 1989 and 1999, were reviewed. Patients who were initially treated at other hospitals but were transferred to this hospital for further therapy were also included. The severity of the poisoning was classified into four stages based on clinical signs and symptoms of PFP[7] : (1) Numbness of the lips and tongue and often of fingers occurs. (2) Numbness progresses markedly. Muscular paralysis of extremities occurs without loss of deep tendon reflexes. (3) Motor incoordination progresses and paralysis develops, but consciousness is maintained. Voice production is difficult because of bulbar muscle paralysis. (4) Consciousness may progressively deteriorate, and respiratory paralysis can cause death.

RESULTS

There were 25 patients, 23 males and 2 females. The severity of the poisoning was classified into four stages according to the stages of progression as described above. Three patients were in stage 1, four were in stage 2 and eighteen were in stage 4.

Clinical features

Most patients had the onset of their symptoms within six hours after puffer fish consumption. Onset time of the symptoms ranged from 30 minutes, in severe cases, to a few hours, in most cases, but seldom exceeded six hours. Paresthesia was the early presenting complaint of all patients. Paresthesia consisting of either numbness or tingling of lips, tongue, around the mouth, hands and feet. Muscle weakness, dizziness, vertigo, nausea and vomiting were common complaints. Eighteen patients developed acute flaccid paralysis and respiratory distress requiring ventilatory support. Nine patients developed transient hypertension.

Table 1. Frequency of certain symptoms and signs.

Symptom and sign	No. of cases	%
Circumoral paresthesia	25	100
Paresthesia of the extremities	25	100
Muscle weakness	22	88
Dizziness and vertigo	18	72
Respiratory failure	18	72
Nausea and vomiting	16	64
Transient hypertension	9	36
Fixed dilated pupils	8	32
Ophthalmoplegia	8	32

Eight totally paralyzed patients with or without altered consciousness had fixed dilated pupils with ophthalmoplegia. Frequency of certain symptoms and signs is shown in Table 1.

Management

All patients received symptomatic treatment and general supportive care, including gastric lavage and intravenous fluid. The patients were closely observed for respiratory failure. Intubation and mechanical ventilation was considered especially when paralysis was progressing rapidly. Most were taken off the respirator 12-48 hours later. All recovered uneventfully without any sequelae.

DISCUSSION

PFP is notorious in Japan, where it most commonly occurs from eating puffer fish, or fugu. Fugu is considered a gourmet delicacy despite its toxicity and requires a special training and license to prepare. In the case of PFP in Thailand, there have been some occasional reports of such incidences. Most victims ingested toxic puffer fish without proper knowledge of the fish and its toxicity. Some people were not familiar with puffer fish characteristics. Most puffer fish have a smooth skin without having long spines but only short spines partly covering the dorsal and ventral part. This may make some people misunderstand that they are not puffer fish and consume them without careful attention. The simple way to recognize whether they are puffer fish of the family Tetraodontidae or not is their set of four teeth which are large and fused to a beak - like plate[8]. Some people, especially those from North - East Thailand, used to consume non-toxic local freshwater puffers and thought that marine puffers were also non-toxic. Some people thought that cooking can destroy the puffer toxin.

Although puffer fish inhabits freshwater as well as seawater, most cases of PFP have been caused by marine species. The toxin of marine puffers was named tetrodotoxin (TTX). In Thailand, cases of PFP have been caused by both marine puffers and freshwater puffers(6). Freshwater PFP was occasionally occurred in North - East Thailand(6,9,10). Some reports have suggested that TTX was the causative agent of freshwater PFP (9-11). Recently, screening on the toxicity of fresh water puffers was conducted in Udonthani province which is located in North - East Thailand. The toxins found in Thai freshwater puffers were not TTX, as usually found in marine puffers, but saxitoxin (STX) and its analogues(12).

TTX is a heterocyclic guanidine. It is heat - stable so it cannot be destroyed by cooking. TTX is highly concentrated in the skin and viscera (liver, gonads, gut) of puffer fish. TTX is one of the best known marine toxins because of its frequent involvement in fatal food poisoning, its unique chemical structure, and its specific action of blocking sodium channel of excitable membranes. The toxin derives its name from the puffer fish family, Tetraodontidae, but past studies have revealed its wide distribution in both terrestrial and marine animals(13). In many of the TTX - containing animals, toxin content fluctuates according to individuals, region, and season. Several studies indicated that TTX was first produced by bacteria and transmitted to other animals through the food chain(14-18). TTX - producing bacteria also occur in a freshwater environment(19).

STX, like TTX, is a heterocyclic guanidine. It is produced by dinoflagellates of the genus Gonyaulax and is found in large concentration in clams, mussels, and other shellfish that feed on these organisms. The toxin has been named STX because it has been extracted from the Alaska butter clam, Saxidomus giganteus. Both TTX and STX block nerve conduction by lowering the conductance of sodium currents through membranes. Because of this uniquely selective action, these toxins were the first chemical markers employed to locate the sodium channel, and have remained important tools in sodium - channel research(2,4, 20).

Paralytic shellfish poisoning (PSP) is an acute paralytic illness which follows ingestion of shellfish contaminated with STX. PSP occurs when toxic dinoflagellates increase in number and are ingested by bivalve mollusks. The mollusks concentrate the neurotoxin in their tissues but are not affected.

STX was also found in some marine puffers. The puffers were assumed to accumulate STX by feeding on bivalve mollusks that have ingested toxic dinoflagellates. As the mode of action of TTX and STX is the same, it is conceivable that puffers can tolerate STX(21).

Paresthesia is considered the earliest clinical hallmark of PFP. Paresthesia is frequently localized to the lips, tongue, around the mouth, hands and feet. Muscle weakness may start in the upper or lower extremities or it may involve all four limbs simultaneously. Weakness may progress to involve bulbar and respiratory muscles. The muscle stretch reflexes tend to be lost in muscles that are very weak. Fixed dilated pupils with ophthalmoplegia usually developed in totally paralyzed patients with or without alteration of consciousness. Totally paralyzed patients with fixed dilated pupils, ophthalmoplegia, and negative oculocephalic movement may mimic the clinical picture of irreversible brain damage. Some patients thought to have anoxic brain damage, but later recovered and described vividly their helpless state of paralysis, which the physicians regarded as deep coma but in which they could hear their conversation. According to old Japanese accounts, some victims of PFP were pronounced dead and subsequently recovered in the morgue or on the way to the crematorium(22).

Hypertension has been found in every stage of PFP. Hypertension may be mild, moderate or severe. It was usually transient and required only a modest dose of antihypertensive drugs or no treatment. The degree of hypertension did not correlate with clinical severity. There were two reports of hypertension in human tetrodotoxication following consumption of unidentified species of fish and gastropod mollusks(23,24). Hypertension may come either from an exaggerated response to sympathetic stimuli, or due to various responses of the vasomotor centre to a small dose of TTX. Further studies are required to elucidate the mechanism of hypertension in PFP. Hypotension usually developed in severe cases with total paralysis and respiratory failure.

Treatment is symptomatic and supportive. There are no specific antidotes to TTX and STX. Gastric lavage and administration of activated charcoal are recommended, but vomiting produced by

the toxin itself may make the procedure unnecessary. Mortality from PFP is related to respiratory failure and cardiovascular collapse, therefore, ventilatory and circulatory support are the mainstay in the management of PFP. Artificial respiration must be initiated as quickly as possible by any available artificial respiration apparatus, and the treatment must be powerfully and continuously practiced to prevent cardiorespiratory arrest. If respiration and circulation is supported for 12-24 hours after ingestion, many patients, seemingly dead or near death, can be revived. Therefore, artificial respiration must be employed even in advanced cases. Severe cases with hypotension should be treated with intravenous dopamine or adrenaline.

Recovery from muscular paralysis often begins in ocular, facial, bulbar, respiratory and limb muscles respectively. Pupils may remain fixed and dilated for a short time after clinical recovery. This suggest that TTX has direct local effects on sphincter muscles.

Cholinesterase inhibitors are of no practical use in PFP[7]. However, there have been case reports of their effectiveness[25,26]. The drugs were used to treat many cases of human tetrodotoxication from puffer fish and horseshoe crab poisoning without clinical improvement. Recently, TTX - specific monoclonal antibodies were reported to protect mice from lethal TTX challenge. In the future, such reagents may have therapeutic potential for treating human TTX poisoning[27].

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พิษปลาปักเป้า : อาการทางคลินิกและประสบการณ์การรักษาในผู้ป่วย 25 ราย

*จีรศักดิ์ กาญจนาพงศ์กุล, พ.บ.**

ระหว่างปี พ.ศ. 2532 – 2542 มีผู้ป่วยได้รับพิษจากการกินปลาปักเป้า 25 ราย เข้ารับการรักษาในโรงพยาบาลชลบุรี อาการเป็นพิษของผู้ป่วยแบ่งออกเป็น 4 ขั้น ตามความรุนแรงมากน้อย มีผู้ป่วยชาย 23 คน ผู้ป่วยหญิง 2 คน ผู้ป่วย 3 ราย อยู่ในขั้นที่หนึ่ง 4 รายอยู่ในขั้นที่สอง และ 18 รายอยู่ในขั้นที่สี่ อาการนำที่พบในผู้ป่วยทุกราย คือ อาการชาริมฝีปาก, ชาลิ้น ชารอบปาก และชาปลายมือปลายเท้า อาการอื่นที่พบบ่อยได้แก่ กล้ามเนื้ออ่อนแรง เวียนและงงศีรษะ คลื่นไส้อาเจียน ผู้ป่วย 18 ราย มีอาการกล้ามเนื้ออ่อนแรงมากจนถึงกับเป็นอัมพาตทั้งตัว หายใจเองไม่ได้ ต้องใช้เครื่องช่วยหายใจ ผู้ป่วยทุกรายได้รับการรักษาแบบประคับประคอง และรักษาตามอาการ ได้รับการล้างท้อง ให้สารน้ำทางหลอดเลือดดำ ผู้ป่วยที่มีอาการกล้ามเนื้ออ่อนแรงมากจนเป็นอัมพาต หายใจลำบาก จะได้รับการใส่ท่อหลอดคอและใช้เครื่องช่วยหายใจ ส่วนใหญ่ใช้เวลาประมาณ 12–48 ชั่วโมง จึงหยุดเครื่องช่วยหายใจได้ ผู้ป่วยทุกรายหายเป็นปกติดี ได้อภิปรายถึงอาการทางคลินิกของผู้ป่วยที่ได้รับพิษปลาปักเป้า พิษวิทยาของปลาปักเป้า และวิธีการดูแลรักษาผู้ป่วย

คำสำคัญ : พิษปลาปักเป้า

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