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Chelonitoxism: New case reports in French Polynesia and review of the literature

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Abstract

Eating the flesh of some marine turtles can cause a type of seafood poisoning called chelonitoxism. The purpose of this article is to report a new case of mass poisoning caused by consumption of sea turtle flesh in French Polynesia. The episode involved 19 members of the same family. Three persons required hospitalization after consuming two consecutive meals including turtle flesh. One 26-year-old woman who was pregnant at 14 weeks of amenorrhea lapsed into a coma and died due to multiorgan failure on the third day after the meal. This case confirms the potential severity of chelonitoxism as reported in several series in the literature showing high mortality rates. The causative toxins are currently unidentified. Further study is needed to better understand chelonitoxism.

Keywords: Chelonitoxism; Marine turtles; Eretmochelys imbricata; French Polynesia; Seafood poisoning

1. Introduction

Chelonitoxism is an uncommon type of food poisoning caused by eating the flesh of marine turtles (Brodin, 1992). In the literature two species have been clearly implicated in sea turtle poisoning, i.e. the Hawksbill Turtle (*Eretmochelys imbricata*) and the Green Sea Turtle (*Chelonia mydas*). However, other species may be toxic including the Leatherback Turtle (*Dermochelys coriacea*) and the Loggerhead Turtle (*Caretta caretta*). Sea turtle poisoning is a severe intoxication with a high mortality rate. It is mainly observed in the Indo-Pacific region but has also been reported in the intertropical zone where the offending species live. The purpose of this report is to describe a new case of mass turtle poisoning episode observed in French Polynesia.

2. Case report

In October 2002, 19 members of a Polynesian family ate turtle flesh during a traditional meal in Rangiroa in the Tuamotu Islands (located 350 km northeast of Tahiti in French Polynesia). The turtle that had been caught the preceding day was identified by several villagers as a young specimen of a species considered as edible by the local

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population, i.e. the Green Sea Turtle (*C. mydas*). In accordance with local customs, the turtle flesh was thoroughly cooked. Within 3 h after the meal, all people that ate the turtle flesh presented digestive symptoms with nausea and vomiting. The next day despite signs, three persons decided to eat the leftover turtle flesh because it was considered as a great delicacy. After consuming even larger quantities than the day before, all three developed severe symptoms and required medical evacuation to the Papeete Hospital in Tahiti.

One patient was a 40-year-old man with a history of gout. He presented vomiting, abdominal pain and polyarthralgia that was attributed to gout. He was admitted to the Rheumatology department and treated with analgesics. His condition improved quickly with disappearance of all clinical signs in less than 12 h.

The second patient was a 64-year-old man with a highly significant medical history including arterial hypertension, hypertrophic heart disease, previous stroke, and non-insulin-dependent diabetes. He was admitted to the critical care unit of the Renal department after exhibiting moderate functional renal failure associated with neurological signs (flaccid tetraparesis and coma) and abnormal laboratory findings (moderate liver cytoloysis, hypoglycemia and pancreatic disturbances). Renal insufficiency and neurological signs resolved spontaneously within a few hours. Following resolution CT-scan was performed revealing the presence of lacunar lesions suggesting previous ischemic infarcts but no evidence of recent lesions was found. Upon awakening the patient reported upper digestive tract pain. Oeso-gastro-duodenal fibroscopy was undertaken on D1 demonstrating involvement of the whole zone with grade III oesophagitis, erosive antral gastritis and ulcerative bulbo-duodenitis that was treated using proton pump inhibitors. Gastrointestinal signs disappeared within 2 days and the patient returned home to the Tuamotu Islands.

The third patient was a 26-year-old woman who was pregnant at 14 weeks amenorrhea but had no medical history. After consuming the turtle flesh for the second time, she quickly developed severe gastrointestinal signs requiring hospitalization in Papeete. Upon admission abdominal ultrasound was carried out to rule out the possibility of extrauterine pregnancy. Upon clinical examination the patient already presented progressing neurological manifestations, i.e. alternating periods of drowsiness interrupted by periods of agitation. The patient soon lapsed into a coma with bilateral constricted pupils reacting to lights and reduced tendon reflexes with no evidence of meningeal involvement. Mild increase in respiratory frequency was also observed. Other clinical findings were normal, i.e. normal cardiopulmonary sounds, soft abdomen, stable hemodynamic status without vasoactive drugs and normal diuresis. Laboratory testing demonstrated extensive abnormalities including severe metabolic acidosis, rhadomyolysis, hyponatremia, low prothrombin rate (68%), hyperammonemia, hypoglycemia that was unaffected by intravenous glucose/electrolyte solution and elevated reactive protein C level. Findings of lumbar puncture, electrocardiography, toxic and renal assessment and thoracic radiography were normal. After the patient went into a coma on D1, her respiratory status deteriorated rapidly requiring intubation for ventilatory assistance and multiorgan failure appeared with liver cytolysis and agranulocytosis. A second ultrasound examination on D2 revealed a dead fetus in the process of expulsion. The patient's renal function deteriorated suddenly on D3 (creatinine clearance, 10 ml/min) and cardiovascular failure occurred requiring infusion of macromolecule solution and treatment with vasoactive drugs. Despite intensive care the patient died of shock associated with uncontrollable lactic acidosis, hypocalcemia and hypoglycemia.

The death of this young patient triggered an investigation by the French police who seized the shell of the sea turtle as evidence. The shell was given to expert zoologists for identification. Findings determined that the villagers had been mistaken and that the specimen was a Hawksbill Turtle (*E. imbricata*).

3. Discussion

Chelonitoxism is rare in comparison with other types of seafood poisonings such as ciguatera or scombrotoxism that have been more extensively described (Brodin, 1992; Isbister and Kiernan, 2005). The low frequency of chelonitoxism is probably related to the fact that sea turtles are an endangered species protected by international regulations and subject to a number of religious restrictions (taboo species for many ethnic groups) (Champetier de Ribes et al., 1998). Nevertheless episodes of turtle poisoning have been reported among low-income coastal populations for whom a captured sea turtle can represent a non-negligible

source of sustenance. Under these circumstances it is not unusual to observe mass poisonings since the flesh of the captured reptile is often shared by a whole family or even village (festive occasion).

Chelonitoxism has long been recognized with case reported by Europeans dating back to the 17th century (Table 1) (Chevallier and Duchesne, 1851). However, detailed clinical descriptions have been rare and most information is anecdotal, i.e., testimonials (sometimes contradictory) based on the stories told by local fishermen (Brodin, 1992). Another reason for the paucity of reliable data on sea turtle poisoning is that victims often live in remote geographical locations with no access to health care facilities (Pillai et al., 1962; Ranaivoson et al., 1994; Champetier de Ribes et al., 1997; Robinson et al., 1999). It should also be noted that consumption of sea turtles is illegal and the fear of sanctions may keep some victims from seeking medical attention.

The clinical signs of chelonitoxism are clearly distinguishable from other types of seafood poisonings (Brodin, 1992; Isbister and Kiernan, 2005) and are relatively stereotyped (see table of gradation in

Table 1
Chelonitoxism in the literature

Year	Localisation	Turtle species	Patients	Death	References
1697	West Indies	*Eretmochelys imbricata*	2	0	Chevallier and Duchesne (1851)
1840	Panadura (Sri Lanka)	*Chelonia mydas*	28	18	Tennent (1861)
1888	Karuppankudiyirupu (Sri Lanka)	*E. imbricata*	12	12	Deraniyagala (1939)
1912	Queesland (Australia)	*E. imbricata*	1	0	Banfield (1913)
1917	Cebu (Philippines)	*C. mydas* or *Dermochelys coriacea*	33	14	Taylor (1921)
1921	Mandaitivu (Sri Lanka)	*E. imbricata*	24	7	Loveridge (1945)
1927	Vaddukodai (Sri Lanka)	*E. imbricata*	?	4	Deraniyagala (1939)
1935	Batavia, Java (Indonesia)	*C. mydas* or *Caretta caretta*	4	1	Siegenbeek van Heukelom (1936)
1935	Wooi (Papua New Guinea)	*E. imbricata*	52	9	Bierdrager (1936)
1949	Ararae (Kiribati)	*E. imbricata*	?	5	Cooper (1964)
1950	Befandefa (Madagascar)	*D. coriacea*	6	0	Robinson et al. (1999)
1950	Yaeyama, Ryukyu (Japan)	*E. imbricata*	80	6	Hashimoto et al. (1969)
1954	Kaipuri (Papua New Guinea)	*E. imbricata*	6	2	Romeyn and Haneveld (1956), Campbell (1960)
1954	Mindanao (Philippines)	*E. imbricata*	14	11	Ronquillo and Caces Borja (1968)
1961	Quilon, Kerala (India)	*E. imbricata*	130	18	Pillai et al. (1962)
1964	Ambatomilo (Madagascar)	*E. imbricata*	25	0	Robinson et al. (1999)
1965	Namatanai (Papua New Guinea)	*E. imbricata*	17	5	Dewdney (1967)
1967	Raiatea (French Polynesia)	*E. imbricata*	12	1	Bagnis and Bourligueux (1972)
1974	Panapai (Papua New Guinea)	*E. imbricata*	6	3	Likeman (1975)
1978	Andrevo (Madagascar)	*E. imbricata*	?	4	Robinson et al. (1999)
1982	Ambohimailaka (Madagascar)	*E. imbricata*	?	5	Robinson et al. (1999)
1982	Rangiroa (French Polynesia)	*E. imbricata*	6	0	Brodin (1992)
1985	Talpe (Sri Lanka)	*E. imbricata*	15	2	Ariyananda and Fernando (1987) Chandrasiri et al. (1988)
1985	Anakao Bas (Madagascar)	*C. mydas*	15	0	Robinson et al. (1999)
1987	Bora Bora (French Polynesia)	*E. imbricata*	1	1	Brodin (1992)
1989	Tahaa (French Polynesia)	*E. imbricata*	1	0	Brodin (1992)
1990	Huahine (French Polynesia)	*E. imbricata*	9	0	Brodin (1992)
1993	Tulear (Madagascar)	*E. imbricata*	200	15	Champetier de Ribes et al. (1997)

Table 1 (*continued*)

Year	Localisation	Turtle species	Patients	Death	References
1993–1998	Toliara and Antalaha (Madagascar)	*E.* and *Chelonia mydas*	422	29	Champetier de Ribes et al. (1998)
1994	Antalaha (Madagascar)	*E. imbricata*	32	5	Ranaivoson et al. (1994)
1995	Ankiembe (Madagascar)	*C. mydas*	40	0	Robinson et al. (1999)
1995	Antalaha (Madagascar)	*C. mydas*	95	9	Champetier de Ribes (1997)
1995	Tsimenatse (Madagascar)	*C. mydas*	21	0	Robinson et al. (1999)
1996 and 1997	Four provinces in Madagascar (20 collective poisonings)	*E. imbricata* and *C.helonia mydas*	571	81	Champetier de Ribes et al. (1999)

Table 2
Gradation of chelonitoxism

Grade	Onset delay	Clinical signs	Biological disturbances	Evolution
Grade 1	Few hours to 4 days after the meal	Gastrointestinal signs ± dizziness, sweating, throat and mouth pain	No	If no aggravation, recovery over a week
Grade 2	1–2 days after the onset period	Glossitis, dysphagia, ulcerative oeso-gastro-duodenal lesions, drowsiness, mild renal failure	Hyponatremia, hypocalcemia, hypoglycemia, hyperuricemia	If no aggravation, recovery over 3 weeks
Grade 3	1–3 days after the onset period	Coma, multiorgan failure (tubular nephropathy, liver cytolysis, respiratory distress)	Neutropenia, thrombopenia, pancytopenia, uncontrolled acidosis, hypocalcemia and hypoglycemia	High percentage of death (65–100% of the grade 3 in recent series in Madagascar—see references in Table 1). Survivors with frequent neurological sequels

Table 2). Onset occurs from few hours to 4 days after consumption. The *onset period* is characterized by the appearance of gastrointestinal signs (nausea, vomiting, epigastric pain and, occasionally, diarrhea). Other symptoms possibly present at onset include dizziness, malaise, sweating, sore throat and chest pain. Most patients do not develop any further effects and recover over a week (Bagnis and Bourligueux, 1972). The real percentage of patients who will have more severe grade is not precisely known as in almost all published case series the grade 1 patients are not included in the statistical analysis. The *typical moderate poisoning* is characterized by pathognomonic oral and pharyngeal involvement with a burning mouth and throat sensation and dysphagia with hypersialorrhea followed by glossitis (25–75% of patients depending on series). Ulcerative oeso-gastro-duodenal lesions have also been reported. At this stage neurological manifestations, best indicators of severity, may appear with alternating periods of drowsiness and full consciousness (or psychomotor agitation). Most patients with moderate forms (grade 2) recover fully within about 3 weeks. However some patients may lapse into coma as an initial phase of a more *severe grade* characterized by multiorgan involvement (tubular nephropathy, liver cytolysis, hypotonic coma, respiratory distress). The most frequent abnormal laboratory test findings are metabolic acidosis, hyperuricemia, hyponatremia, hypocalcemia, hypoglycemia, neutropenia and thrombopenia (Brodin, 1992). Mortality is high among patients with severe forms especially in children. Upon emerging from coma, survivors often present complex central and/or peripheral neurological sequels (e.g., hemiplegia, tetraplegia, dementia, sensory-motor deficit, cerebellar syndrome). There is no antidote for sea turtle poisoning and treatment is strictly supportive and symptomatic with intensive care if necessary.

The sea turtle species most commonly cited in the literature and apparently responsible of the highest mortality is *E. imbricata* (Table 1). Most authors stress that all organs of this chelonian are potentially toxic regardless of preparation (thermoresistant toxin ?) (Pillai et al., 1962; Ariyananda and Fernando, 1987; Champetier de Ribes et al., 1999). It has also been noted that toxic effects are dose dependent since symptoms are most severe in victims that eat the most flesh. Another feature of chelonitoxism described in the literature is passage of toxins into breast milk with several reported cases of poisoning in breast-fed children that did not consume the turtle flesh itself (Dewdney, 1967; Ariyananda and Fernando, 1987; Ranaivoson et al., 1994). Transplacental contamination may have occurred in our case since the fetus died at the beginning of the severe phase (night between D1 and D2) when the metabolic status of the mother was still under control.

The causative toxins for sea turtle poisoning have not been identified. However several investigators have suggested that contamination of turtle flesh is due to the accumulation of toxic compounds from the food chain with no effect on the health of the sea turtle itself. Investigators in Japan identified lyngbyatoxins (toxins in *Lyngbya* sp. seaweed) in the flesh of the herbivorous species *C. mydas*, the second taxon implicated in chelonitoxism (Yasumoto, 1998; Ito et al., 2002). However, it seems unlikely that phycotoxins are involved in poisoning by *E. imbricata* since they are strictly carnivorous species mainly eating sponges. There are no external signs that the turtle is poisonous and almost all sayings and customs are unreliable. Prophylaxis depends on enforcement of regulations to protect endangered species and controlling the sale of sea turtle flesh.

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