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Ciguatera Fish Poisoning in San Francisco, California, Caused by Imported Barracuda

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CIGUATERA FISH POISONING is endemic to tropical waters, where reef-dwelling fish ingest toxins produced by the dinoflagellate *Gambierdiscus toxicus*. The disease, which is caused by the ingestion of fish contaminated with ciguatera toxins, produces gastrointestinal and neurologic symptoms. These consist of an early gastrointestinal phase (abdominal pain, vomiting, diarrhea) followed by a neurologic or constitutional phase (pruritus, muscle pain, weakness, "burning tongue," blurred vision, dizziness, headache, and hot-cold reversal). We report an outbreak of ciguatera poisoning that occurred in 1989 in San Francisco, California, caused by barracuda (*Sphyraena* species) imported from Florida. This disease is rare in California, having previously been reported in Monterey, California (caused by mullet imported from Florida), and in the San Francisco Bay Area (jack fish from Midway Island).[1]

Reports of Cases

Four members of a Vietnamese-American family became ill after sharing a meal. The family included a 54-year-old man, a 53-year-old woman, their 21-year-old daughter, and 24-year-old son. All patients were in good health before the meal, which consisted of fresh corn on the cob, commercially canned mushrooms, and steamed fish. Only the fish was eaten by all four people. No shellfish had been ingested.

The fish was said to be a "nhong" fish, described as 1.2 m (4 ft) long with a pointed snout. It was purchased from an open-air farmers' market in San Francisco. The family purchased and ate only the head of the fish, which they said was large and contained abundant flesh. Investigation by the county health department determined that the fish eaten was from a shipment of frozen barracuda flown in from Fort Pierce, Florida, and apparently sold to several Asian-American families.

(Geller RJ, Olson KR, Senécal PE: Ciguatera fish poisoning in San Francisco, California, caused by imported barracuda. West J Med 1991 Dec; 155:639-642)

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Case 1

At five hours, the father complained of weakness, burning of the tongue, and blurred vision. He became profoundly weak and by eight hours could not get up off the floor. He had no nausea, vomiting, or diarrhea. Paramedics found the patient prostrate with a pulse of 40 beats per minute and a systolic pressure of 80 mm of mercury. He did not have chest pain, and there were no signs of heart failure. Atropine sulfate, 0.5 mg, was administered intravenously (IV). Twenty minutes later, his blood pressure was 132/52 mm of mercury, and his pulse was 88 beats per minute.

In the Emergency Department at San Francisco General Hospital, he had numbness to the face and extremities, weakness, and periumbilical abdominal pain. His blood pressure was 144/90 mm of mercury, pulse rate 84 beats per minute, and he was afebrile. Initial laboratory values included a leukocyte count of 9.0×10^9 per liter (9,000 per μl), hemoglobin 120 grams per liter (12.0 grams per dl), and hematocrit 0.37 (37%). The serum glucose level was 12.7 mmol per liter (229 mg per dl), blood urea nitrogen 7.9 mmol per liter (22 mg per dl), and creatinine 106.1 μmol per liter (1.2 mg per dl). Serum sodium was 139, potassium 3.3, chloride 103, and bicarbonate 27 mmol per liter. Arterial blood gas determinations with the patient receiving supplemental oxygen showed a Po_2 of 344 mm of mercury, a Pco_2 of 44.8 mm of mercury, and pH 7.35.

Because of abdominal pain, a surgical evaluation was done. Bradycardia and hypotension recurred, but additional atropine was not given. Instead, fluid administration was begun. After six hours, he had received 4,000 ml of fluid IV and his systolic blood pressure had still fallen to 70 mm of mercury, with a pulse of 54 beats per minute. An abdominal x-ray series was initially read as showing free air under the diaphragm. A subsequent exploratory laparotomy revealed no intra-abdominal disease.

His hospital course was remarkable for persistent weakness, recurrent bradycardia, and hypotension. His pulse rate stayed between 45 and 55 beats per minute with systolic blood pressures in the range of 80 to 90 mm of mercury. He tolerated these values generally without symptoms and was discharged after nine days.

Two days later, he was readmitted with severe pruritus unresponsive to the administration of diphenhydramine hydrochloride, which had led to a significant cellulitis around his buttocks and presacral area due to excoriation. Mild orthostatic changes in pulse and blood pressure were noted but did not cause symptoms.

Case 2

At 12 hours, the mother became ill in the emergency department while waiting for her husband. She complained of fatigue, nausea, and chest pain. Initially her pulse rate was 68 beats per minute and her blood pressure was 132/90 mm of mercury. At 18 hours, she had nausea and vomiting and complained that her face and throat felt swollen. Her blood pressure was then noted to be 80/38 mm of mercury, and her pulse was 60 beats per minute. Thereafter, her pulse and blood pressure remained low.

Initial laboratory tests revealed a leukocyte count of 11.6×10^9 per liter (11,600 per μl), with 0.69 granulocytes and 0.27 lymphocytes; hemoglobin 122 grams per liter; and hematocrit 0.375. Serum sodium was 141, potassium 3.8,

chloride 104, and bicarbonate 22 mmol per liter. A glucose level was 7.8 mmol per liter (140 mg per dl), blood urea nitrogen 7.1 mmol per liter (20 mg per dl), and creatinine 61.9 μmol per liter (0.7 mg per dl). Arterial blood gases measured with the patient receiving 100% oxygen showed a Po_2 of 499 mm of mercury, a Pco_2 of 53.8 mm of mercury, and a pH of 7.27.

Her gastrointestinal symptoms resolved within 24 hours of admission. She had prolonged and severe bradycardia and orthostatic hypotension, treated with IV fluids with transient benefit. Her resting pulse was about 50 beats per minute, with resting systolic blood pressures averaging 80 to 88 mm of mercury. Standing blood pressures were about 60 mm of mercury, with the pulse increasing to only 80 beats per minute.

On day 9, the patient's supine blood pressure was 106/68 mm of mercury and the pulse was 48 beats per minute. After standing for a minute, her pulse was 83 beats per minute and her blood pressure was 58/39 mm of mercury. After eight minutes of standing, the patient reported dizziness and a sensation of blindness. Her blood pressure was 47/30 mm of mercury, and the pulse was 87 beats per minute. She was returned to the supine position with a prompt resolution of symptoms. Atropine sulfate was then administered intravenously. A total dose of 2 mg given in 0.5-mg increments over 30 minutes completely abolished the orthostatic hypotension. Lasting recovery from symptomatic orthostatic hypotension did not occur until four weeks after the onset of the illness. Severe pruritus developed on the 12th day.

TABLE 1.—*Onset of Symptoms in Patients With Ciguatera Fish Poisoning*

Symptom	Patients, No. (%)	Range of Onset, days*	Average Day of Onset	Median Day of Onset
Pruritus	16 (100)	1-16	1.8	3
Weakness	15 (94)	0-3	0.7	0
Muscle pain	15 (94)	0-3	0.8	1
Abdominal pain	12 (75)	0-1	0	0
Vomiting	11 (69)	0	0	0
"Burning" tongue	11 (69)	0-3	0.9	0
Blurred vision	9 (56)	0-15	2.2	0
Dizziness	9 (56)	0-7	0.9	0
Diarrhea	9 (56)	0	0	0
Metallic taste	8 (50)	1-7	3.0	3
Headache	8 (50)	0	0	0
Hot-cold reversal	8 (50)	1-7	2.1	1.5
Lip numbness	6 (38)	0-2	0.8	1
Finger or toe numbness	6 (38)	0-4	1.8	1.5
Skin rash	5 (31)	2-14	5.2	7
Dyspareunia (♀)	5 (31)	4-6	4.8	4.5
Joint pain	5 (31)	1-3	1.3	1
Dysuria	5 (31)	0-3	2.0	3
"Burning" feet	5 (31)	0-2	0.5	0
Dry mouth	4 (25)	0-14	5.0	1
Dentalgia	3 (19)	0-14	5.7	3
Nasal burning	3 (19)	0-4	2.0	2
Vertigo	2 (12)	0	0	0
Anorexia	2 (12)	1-8	4.5	4.5
Hiccups	1 (6)	13	13.0	13
Painful intercourse (♂)†‡	1			

*Day 0=day of exposure.
†This symptom occurred in a man who had not eaten the fish but whose wife had, when he later had intercourse with her.
‡Data are not available for the onset of symptoms.

Case 3

At five hours, the daughter had nausea, vomiting, and an unusual "burning" sensation of the tongue, plus mild weakness. She did not seek medical attention. At 22 hours, she presented to the emergency department, where she was evaluated for vomiting, weakness, and burning in the tongue and was released. The daughter returned at 74 hours complaining of a freezing sensation while stepping into a tub of hot water (hot-cold reversal) and generalized weakness. She was again released.

Case 4

At 22 hours, the son also presented to the emergency department, complaining of perioral paresthesias and burning of the tongue. He reported having vomited at 14 hours. He was released. Eight days later, he returned with severe pruritus and excoriation, unresponsive to parenteral diphenhydramine. A regimen of amitriptyline hydrochloride, 25 mg twice a day, hydroxyzine hydrochloride, 25 mg four times a day, and a steroid cream was prescribed. Two days later, he was admitted with a presacral and perigluteal cellulitis due to excoriation. His pruritus had abated slightly.

No family members had rash or immediate pruritus, cranial nerve palsies, or pulmonary symptoms.

Other Cases

On day 8, a family of four ethnic East Indians from the Fiji Islands presented with gastroenteritis to another hospital. Several hours after they had eaten a barracuda purchased on day 1 from the same farmers' market and frozen for a week, they complained of burning of the tongue and perioral paresthesias. One patient had severe bradycardia (pulse rate, 30 beats per minute) and hypotension (systolic blood pressure, 60 mm of mercury); his hospital course was prolonged.

On day 11, two members of a Filipino family who had also eaten barracuda on day 1 presented with severe pruritus. Six other family members who had shared the meal also had symptoms.

All patients identified as having ingested the contaminated fish were contacted and interviewed similarly by staff of the San Francisco Bay Area Regional Poison Control Center. Information collected included the types of symptoms suffered by each patient and the time of the onset of symptoms relative to ingesting the fish. This is presented in Table 1.

Of special interest is a patient who did not eat the barracuda but in whom pain developed at the tip of his penis after intercourse with his wife, who had eaten the fish. Lange and co-workers reported dyspareunia in an unaffected woman following intercourse with her affected male partner.[2] We think that this is the first case suggesting the transmission of symptoms from an affected woman to an unaffected man. A woman who was pregnant at the time of her illness with ciguatera poisoning was delivered of a healthy full-term child five months later.

Discussion

Ciguatera fish poisoning is common in tropical regions between 35° north and 35° south latitude. In the United States, 90% of cases occur in Hawaii and Florida.[3] Sporadic

cases have been reported in Chicago (fish from Florida and the Bahamas),[4,5] Iowa (fish from the Virgin Islands),[6] Texas,[7] Vermont (barracuda from Florida),[8] Maryland (grouper from Florida),[9] and Louisiana, Massachusetts, and Washington, DC.[10] One previous epidemic in the San Francisco Bay Area occurred in 1977 and was caused by jack fish caught off Midway Island and brought by merchant ship into the port of Oakland.[1] A recent report documented an outbreak of ciguatera fish poisoning in ten people who ate fish caught off the middle third of the North Carolina coast on the western edge of the Gulf Stream.[11] The 35°-north latitude line passes through the mid-North Carolina coast.

Fish commonly implicated include barracuda, red snapper, amberjack, and grouper,[3] but more than 400 species have been causative. In a given area, a particular fish is usually responsible. Red snapper is most often the vector in the South Pacific, while in Hawaii amberjack is the most common cause. In Dade County, Florida, where more than a third of barracuda tested have contained ciguatoxin, it is illegal to sell barracuda for human consumption.[12] There are no regulations to prohibit exporting barracuda from Florida or importing it into California.

Pathophysiology

At least three toxins are thought to be responsible for the clinical effects seen in ciguatera fish poisoning: ciguatoxin, maitotoxin, and scaritoxin.[13] The toxins are created by a one-celled organism, the dinoflagellate *Gambierdiscus toxicus*.[14] The organism is similar to the dinoflagellates that cause shellfish poisoning in colder waters,[5] but its growth is restricted to reefs in tropical climates. The dinoflagellate is consumed by small fish, and the toxins are passed on up the food chain as large fish eat smaller fish. The largest fish appear to have the highest concentration of toxin.[15] In some areas, fish are empirically judged as safe or unsafe to eat based on their relative size.

The toxins are lipid-soluble, acid-stable, and are not destroyed by freezing or cooking. They are without taste or smell, making detection difficult.[16] The toxins are concentrated in some organs more than others. Natives of some South Pacific islands test their fish by feeding the liver, ovary, or testis of a fish to cats to observe for signs of illness.

The exact structure, site of action, and mechanism of the toxins are not completely understood. In studies on neuroblastoma cells, ciguatoxin has been shown to act on sodium channels to increase sodium permeability.[17] One site of action appears to be the stimulation of central or ganglionic cholinergic receptors.[13]

Clinical Presentation

Symptoms are largely gastrointestinal, neurologic, and cardiovascular. Neurologic and cardiovascular symptoms, especially pruritus, dizziness, paresthesias, and postural hypotension with inappropriate bradycardia, may be prolonged (months) and severe. Depression has been reported.[5] Symptom patterns vary among ethnic groups and even between the sexes. The sequence of symptom onset may also have a geographic variability. Neurologic symptoms often precede gastrointestinal symptoms in the South Pacific, while the reverse is true in the West Indies.[12] All of our patients had transient, early gastroenteritis followed by prolonged or delayed neurologic symptoms.

The largest series of cases examined in the literature was from the South Pacific.[18] In 3,009 cases, only 3 deaths occurred, each of whom had a neurologic catastrophe (coma, seizures). The distribution by sex was 59.3% in males and 40.7% in females, with 49.7% of cases occurring in the third or fourth decades of life. Common symptoms or signs included circumoral paresthesias (89% of patients), paresthesias of the extremities (89%), burning or pain to skin on contact with cold water (87.6%), arthralgia (85.7%), myalgia (81.5%), diarrhea (70.6%), asthenia (60%), headache (59.2%), and chills (59%). Abdominal pain occurred in 46.5% and hypotension in only 12.2%.

In a series of cases from Hawaii, more than 175 clinical manifestations of ciguatera fish poisoning were reported.[19] Of these, nausea, vomiting, diarrhea, paresthesias (oropharyngeal or acral), and temperature sensation reversal were noted to be common. The last symptom, where hot objects feel cold and cold objects seem hot, occurred in several of our patients on the third day. This phenomenon is strongly suggestive of ciguatera poisoning but is not pathognomonic. It also occurs in neurotoxic shellfish poisoning with exposure to cyclic ethers such as brevitoxins A, B, and C.[20]

Early and persistent hypotension with bradycardia and late, severe pruritus were the most striking features of our cases. Ho and colleagues reported three cases in Toronto, Ontario, that resulted from ingesting grouper from Florida. Profound bradycardia and hypotension occurred in two patients. Late neurologic symptoms—paresthesias, pruritus, and postural dizziness—lasted as long as three months. As in our cases, the outbreak in Toronto occurred in Vietnamese immigrants, suggesting that further outbreaks among Southeast Asians may occur.

Diagnosis is at this time based on the history and clinical presentation. An ingestion of a large fish (especially barracuda or grouper) from tropical reef waters followed by gastroenteritis accompanied by unexpected neurologic and cardiovascular symptoms should suggest the diagnosis. There are no serum or urine tests to detect human poisoning. A radioimmunoassay has been developed that will confirm the presence of ciguatera toxin in suspected fish, but it is not widely available.[21] The remains of one fish eaten by four of our patients were sent to the University of Hawaii for analysis. A stick enzyme immunoassay carried out there was positive in the "high toxic range" for ciguatoxin and related polyethers. Local and state health officials may be able to assist in sending portions of the fish for toxin assay.

Treatment

Treatment is largely supportive. Gastrointestinal symptoms are usually brief and self-limited, requiring little more than the administration of fluids. Mild pruritus will usually respond to antihistamine therapy, but severe pruritus may be difficult to treat. Administering antihistamines and amitriptyline failed to relieve severe pruritus in our patients, but the use of morphine was effective. The relief of neurologic symptoms has been reported with amitriptyline therapy, with variable success.[4-6,22] The use of nifedipine and tocainide has been suggested.[4,23] Intravenous mannitol was given to two patients with suspected cerebral edema and one patient in shock.[24] Recovery within minutes was described. The same study reported further the empiric and uncontrolled use of intravenous mannitol in 21 other patients and claimed a notable lessening of neurologic and muscular symptoms within minutes of administration. In a pilot study from Australia,

infusing 0.5 to 1.0 grams per kg of mannitol (10% or 20%) resulted in "significant" improvement in 5 (42%) and "dramatic" improvement in 3 (25%) of 12 patients with ciguatera fish poisoning.[25] There have been no controlled studies of any of these agents in ciguatera poisoning, and appropriate caution should be used in employing these therapies.

Public health officials should be notified as soon as a case of ciguatera poisoning is suspected, as an epidemic may rapidly unfold. Ciguatera poisoning is not specifically a reportable disease, but it is a food-borne illness. In California, physicians are required to report immediately by telephone when two or more cases or suspected cases of food-borne illness from separate households are suspected to have the same source of illness (title 17, California Code of Regulations, sections 2500, 2504, 2574).

Acknowledgements

Tim McCarthy, PharmD, San Francisco Bay Area Regional Poison Control Center, assisted with the prompt diagnosis of the outbreak; Fran Taylor, MD, and Mr Ben Gale, San Francisco Department of Public Health, assisted with notifying the county and providing clinical consultation; Tim Berger, MD, Department of Dermatology, San Francisco General Hospital Medical Center, provided clinical assistance and manuscript review; and Ben Werner, MD, and Mr Warren Crawford, Infectious Diseases Branch, California State Department of Health Services, assisted with notifying the state and providing clinical consultation.

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Sarcoidosis of the Breast, Central Nervous System, and Exocrine Glands in a Patient With Sicca Symptoms

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SARCOIDOSIS IS A SYSTEMIC inflammatory disease of unknown etiology characterized by noncaseating epithelioid cell granulomata.[1] The lungs and reticuloendothelial system are typically involved, and virtually any organ system, including the salivary glands, may be affected. Clinical involvement of the breast, however, is exceedingly rare.[2]

Sjögren's syndrome is an autoimmune disorder characterized by keratoconjunctivitis sicca, xerostomia, and clinical or laboratory evidence of systemic autoimmunity.[3] The disorder is pathogenetically defined by mononuclear cell infiltration into exocrine glands, and the diagnosis can be confirmed by minor salivary gland biopsy. Sarcoidosis can also affect exocrine glands and cause sicca symptoms.[4] In such patients, minor salivary gland biopsy will show typical noncaseating granulomata that will distinguish sarcoidosis from Sjögren's syndrome.

Herein we report the case of a woman who presented with sicca symptoms and lymphadenopathy consistent with Sjögren's syndrome and with a hard left breast mass suggestive of breast cancer. Minor salivary gland and breast biopsies revealed only noncaseating granulomata diagnostic of sarcoidosis.

Report of a Case

The patient, a 70-year-old white woman, was seen in November 1989 because of a firm left axillary lymph node enlarged to about 4 cm in diameter. A thorough breast examination revealed no abnormalities. A complete blood count, liver function test values, a chest x-ray film, mammography, and computed tomography (CT) of the chest and mediastinum were all normal. Fine-needle aspirate of the node revealed lymphocytes, histiocytes, and a rare "immunoblastic" cell. The patient refused open biopsy. By April of 1990, she had begun to have neck and jaw pain, xerostomia, and xerophthalmia with significant bilateral submandibular gland enlargement. A needle biopsy of one of the submandibular glands revealed lymphocytic infiltration of benign salivary gland tissue. Treatment with fast-acting nonsteroidal anti-inflammatory agents resulted in symptomatic improvement.

In July, acute diplopia and vertigo developed. On physical examination, the patient had a left lateral rectus muscle palsy, sublingual and labial salivary gland hypertrophy, persistent submandibular gland enlargement, left axillary lymphadenopathy, and a hard mass 3 cm in diameter in the lower inner

(Fye KH, Hunter RH: Sarcoidosis of the breast, central nervous system, and exocrine glands in a patient with sicca symptoms. West J Med 1991 Dec; 155:642-644)

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