ORIGINAL

Fish Poisoning in American Samoa

JOHN M. DAWSON, M.D., *Honolulu*

• *The first recorded outbreak of fish poisoning in the tropical Pacific occurred in 1606 as recorded by the Spanish explorer, Pedro de Quiros. In American Samoa, Jordan[1] made the first survey of poisonous fish in 1902, and several more recent articles on fish poisoning have referred to American Samoa as an endemic area for fish poisoning.[2,3,4] In this present study, an average of 40 persons were hospitalized annually in American Samoa during 1970 through 1974 due to illnesses resulting from the ingestion of marine animals. Ciguatera accounted for more than one-third of these hospital admissions. Fish poisoning or "ichthyosarcotoxism" is an important economic and medical problem in American Samoa.*

Illnesses due to eating fish and other marine life can be related to poisoning by putrefied products, viral, bacterial, or protozoal pathogens, allergies, fish made toxic by chemical pollution of the marine environment, naturally toxic fish, or by toxins produced by microorganisms ingested by fish.[5]

Ciguatera is an example of the latter group, and ciguatera is the most important type of fish poisoning in the tropical Pacific. Ciguatera results from eating any of a wide variety of fish associated with coral reefs.[4,6,7-10] A fish probably becomes ciguatoxic from a toxic microorganism in its food chain,[11] although the exact identity of the microorganism is not known.

Ciguatera is a symptom complex produced by eating fish containing ciguatoxin.[12,13] Secondary toxins,[14] the concentration of ciguatoxin in the fish, the amount eaten, and the individual's sensitivity account for the variability of the clinical syndrome.

Symptoms commonly begin several hours after ingestion, but may be almost immediate. Gastrointestinal disturbances of nausea, vomiting and diarrhea; neurosensory disturbances of paresthesias and dysesthesias—sensation of burning or electrical discharge on contact with cold; arthralgias and myalgias; neuromuscular disturbances of paresis or fasciculations; and cardiovascular changes of hypotension and bradycardia occur singularly or as complexes.[3,8,15] The toxicity of a specific species may differ according to the area or time; generally the larger specimens are more toxic than the smaller, and the viscera (liver, intestines, testes, ovaries) are more toxic than the flesh. Ciguatoxin is thermostable and a peculiar phenomenon of reduced tolerance occurs with repeated intoxications.[10]

Research Method

Data were reviewed retrospectively from inpatient records (1970-1974, inclusive) at the Lyndon Baines Johnson Tropical Medical Center, Pago Pago, American Samoa, of 198 admission cases related to the ingestion of marine animals, as determined by the admitting physician, discharging physician or medical librarian. We reviewed data prospectively, using standardized questionnaires,[8] of 136 outpatients who presented themselves to the LBJ Tropical Medical Center, and the dispensaries on the islands of Ta'u, Fitiuta, Ofu (Manu'a group), and Swain's Island for illnesses related to the ingestion of marine animals during the period September, 1974 to September, 1975. Questionnaires were in both English and Samoan. Thirdly, we distributed questionnaires to Samoan medical officers and local fishermen, and conducted personal interviews with the Director of Marine Resources.

Results

Of the 198 inpatient charts reviewed, 33% were admissions for ciguatera; of the 136 outpatients surveyed, 43% had ciguatera poisoning (Table 1). Illnesses resulting from the consumption of fish were recorded as ciguatera only if the

Accepted for publication December, 1976.

TABLE 1.—*Number of patients ill due to the ingestion of marine animals.*

	INPATIENTS 1970 - 1974	OUTPATIENTS 09-71 - 08-75	TOTAL
Marine animals other than fish	64	33	97
Fish—other than ciguatera	69	44	113
Fish—ciguatera	65	59	124
Total	198	136	334

neurosensory symptoms of paresthesias or dysesthesias or the cardiovascular signs of hypotension and bradycardia (50/minute or less) were present. The category, "other than ciguatera," included food poisonings, scombroid poisonings and allergic reactions. Patients who presented with the symptoms of only vomiting, diarrhea, arthralgia, myalgia and/or weakness were included in the "other than ciguatera" group. However, some of these may have been ciguatera that were not clinically distinguishable from other poisonings.[8]

The 10 varieties of animals implicated in the illnesses resulting from eating marine animals other than fish are presented in Table 2. Reported in the ciguatera poisonings were 13 types of fish (Table 3). A higher incidence of ciguatera occurred during the period from October to December (Table 4).

TABLE 2.—*Number of patients ill due to eating marine animals other than fish.*

SAMOAN	COMMON	SCIENTIFIC		NO. OF CASES
se'a	sea cucumber	*Holothuria atra* (Cuverian organs)		25
palolo	marine sea worm	*Eunice viridis*		18
fe'e	octopus	several species		15
'ulatai	lobster	*Panulirus penicillatus*		11
pa'a	crab	several species		10
matamalu	sea anemone	*Rhodactis howesi*		5
tugane	clams	several species		2
'ula	shrimp, prawns	several species		2
vana	sea urchin	*Diadema paucispinum*		1
'ali'ao	top shell	*Trochus niloticus*		1
			Total	90*

*Figure excludes 7 patients who had been included both as inpatients and outpatients.

TABLE 3.—*Types of fish reported in the ciguatera cases.*

SAMOAN	COMMON	SCIENTIFIC	CASES	%	PREVIOUSLY REPORTED BY:*
mu	red snapper	*Lutjanus bohar*	32	28	J; B; H
malie	shark	Carcharhinidae	22	19	
sapatu)		*Sphyraena obtusata*			
saosao**)	barracuda	*Sphyraena barracuda*	10	9	
pusi	eel	*Gymnothorax* spp.	5	4	J; B; H
ata ata	grouper	*Epinephelus merra*	4	3	J; B
gatala	grouper	Serrandidae	3	3	
filoa	emperor	*Lethrinus* spp.	3	3	J
savani	snapper	*Lutjanus kasmira*	2	2	
lupo***)					
ulua)	jacks	*Caranx* spp.	2	2	
malai	snapper	*Lutjanus gibbus*	1	1	B; H
malau	squirrel fish	Holocentridae	1	1	B; H
not specified			29	25	
		Total	114	100****	
Marine Resources[16] reports additional fish producing ciguatera:					
taiva	snapper	*Lutjanus monostigma*	"sometimes toxic"		J
pa'pa	grouper	*Variola louti*	1 case		

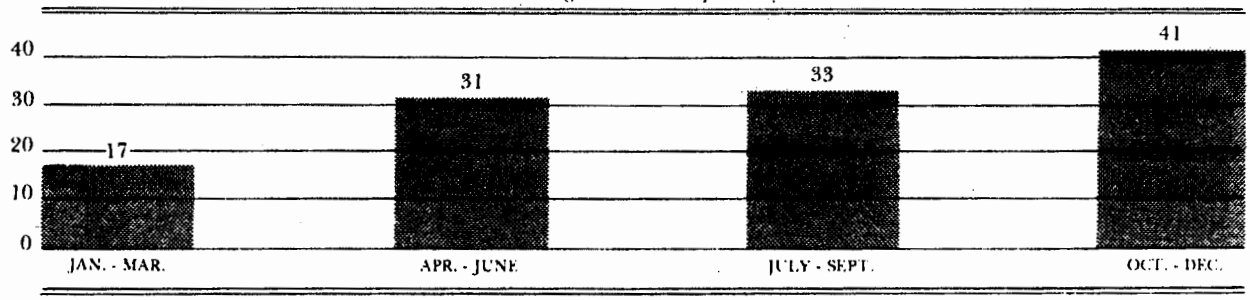
*Previously reported by: J-Jordan;[1] B-Banner and Helfrich;[2] H-Halstead.[3]

**Differentiation between these two species is not always made; sapatu was more commonly reported by patients, but saosao is generally considered more often poisonous.

***Lupo is the Samoan name for a jack under approximately one pound, and it is generally considered to be nontoxic.

****Total excludes 10 patients who had been included both as inpatients and as outpatients during the September-December 1974 period.

TABLE 4.—*Ciguatera cases reported by month.*



Chi square analysis comparing four groups:
$x^2 = 9.80$ df3 $p<.05$

Discussion

These data represent only those persons who, becoming ill after ingesting marine animals, attended a medical facility, either as outpatients or as inpatients. The director of Marine Resources estimates that no more than 10% of ciguatera poisonings reach the hospital or dispensaries.[16] A house-to-house survey, as performed on other tropical islands,[17,18] might yield more accurate data regarding the extent of fish poisoning in American Samoa.

Our inpatient figures necessarily rely on the accuracy and completeness of the admission records; the outpatient figures were collected with the assistance of the outpatient department staff. Therefore, the resulting numbers cannot be considered completely accurate. However, the outpatient department physicians and nurses reliably recorded the outpatient cases during the survey year.* Ciguatera resulted in 15 hospital admissions each year (1970-1974 average) and four times this number of ciguatera cases were seen as outpatients during the survey year. If the 10% approximation given by Marine Resources for the fraction of ciguateric illnesses seen at the hospital and dispensaries is accurate, then there is a ciguatera incidence of more than 20 per 1,000 (100/10 × 59/29, 191 × 1,000) population in American Samoa.

Several serious cases with marked hypotension and bradycardia and one case of severe paresis were observed during 1974 and 1975. The usual course for the ciguateric syndrome in the territory is gastroenteritis for 1-2 days, weakness for 2-7 days, and paresthesias for 2 days to 3 weeks.** No fatalities were recorded due to fish poisoning during the study period (1970-1975). However, according to a previous report, 3 people died in 1958 with symptoms similar to ciguatera after consuming a green sea turtle (*Chelonia mydas*).[2]

The indicated seasonal variation of ciguatera is consistent with reports from Fiji where a peak is seen in October.[19,20] Our study sample was biased in that only cases seen at the medical facilities were reported. Therefore, the hypothesis that there is a seasonal variation in ciguatera or in ciguatoxicity in the reef fish of American Samoa requires further testing.

Of the 13 types of fish listed as potentially toxic, 3 kinds accounted for more than 50% of the ciguateric cases: mu (red snapper)—*L. bohar;* malie (shark)—*Carcharhinida;* and sapatu and saosao (barracuda)—*Sphyraena* spp. The shark (elasmobranch) poisonings resulted only after the ingestion of the liver or gills, and these poisonings were commonly associated with the cardiovascular signs of bradycardia and hypotension. Gymnothorax (eel) and elasmobranch (shark) poisonings were included as ciguatera in this report in accordance with more recent publications.[13,21]

The prevention of food poisonings and scombroid poisonings requires hygienic production and adequate storage and distribution. Other illnesses could be prevented if sensitive individuals avoided allergenic species and if marine gourmets adequately cooked sea anemones.[22] However, the prevention of ciguatera is a difficult problem, since many of the common table fish (Table 3) of American Samoa are occasionally toxic.

The Department of Public Health has restricted the sale of mu (*L. bohar*); and the major fish market, on its own initiative, does not sell taiva (*L. monostigma*) or very large filoa (*Lethrinus* spp.). These 3 fish account for approximately 25% of the total bottom fish caught by the Samoan fishing fleet.[16]

Research efforts to find an inexpensive, efficient screening technique for ciguatoxic fish is promising for the future,[23] but the only available screening techniques now are bioassay procedures, eg, with the mongoose[10] which are too complex, inaccurate, and expensive for an endemic area such as American Samoa. Efforts to reduce the incidence of fish poisoning must rely on health education. Specifically, the following should be stressed for the prevention of ciguatera in American Samoa:

1. 13 fish have been implicated in ciguatera cases (see text for complete list), but 3 have produced more than 50% of cases: mu - red snapper; malie - shark (liver and gills) and sapatu and saosao - barracuda (Table 3).

*Personal observation and cross check with day books.

**Personal observation as outpatient physician, December 1973-August 1975.

2. Although a seasonal variation in ciguatera is indicated, no season is reliably safe (Table 4).
3. Most reef areas may yield toxic fish.[16]
4. The viscera of fish is more toxic than the flesh.[10,21]
5. Larger fish are more toxic than smaller fish.[10,25]
6. Cooking techniques have little effect on toxicity.[25]
7. Common home tests in Samoa including the coin test (a silver coin cooked with the flesh of a toxic fish turns dark) and the fly test (a fly will not alight on a toxic fish) are experimentally invalid,[25] but prefeeding the viscera to a cat would be of benefit if the animal was observed closely for symptoms (lack of coordination, inability to stand) or regurgitation.[25]
8. The early administration of an emetic is useful if vomiting has not already occurred.

Treatment Regimes

Although the ciguatera phenomena is not strictly comparable to anticholinesterase poisoning *in vivo,*[26] a confirmation test to aid in the diagnosis as suggested by Li[27] may still prove useful: an intramuscular adult dose of 1 mg. of atropine (a safe procedure[28]) will fail to produce the expected signs of atropinization: dry mouth, lips and throat, dry skin, pupil dilatation, and tachycardia, if ciguatera poisoning is present.

The following pharmacologic agents have been experimentally or clinically tested in ciguatera poisoning:

1. Native Plants[3,24,30]
2. Neostigmine and physostigmine[25,31]
3. tubocurarine chloride[32]
4. vitamins[8,15,20,34]
5. EDTA (calcium disodium edetate)[34]
6. cortisone[8,25,34]
7. edrophonium[25]
8. magnesium sulfate[32]
9. methyl phenidate hydrochloride[32]
10. procaine amide[33]
11. oximes[8,15,26,27,31,32,34]
12. atropine[8,15,20,25-27,31,32,34]
13. calcium salts[8,20,25,32,34,35]

The oximes, eg, protopam chloride[R] (pyridine-2-aldoxime methchloride), have been extensively used by Bagnis;[15] however, his results and the results of others[32,34] would indicate that the oximes are effective and safe only during the early phases of the syndrome. Atropine has been used in conjunction with other pharmacologic agents in many treatment regimes and is safe if administered after cyanosis has been corrected and if the dosage is adequately monitored (doses up to 33.6 mg. of atropine have been used during the acute stages.[32]) Atropine has reliably given symptomatic relief from abdominal pains and, together with electrolyte infusions, is effective for the correction of hypotension and bradycardia.[34]

Ciguatera toxin has been shown to have a widespread direct action on excitable membranes.[36,37] Calcium has been shown to be a competitive inhibitor* of ciguatoxin on frog membrane.[38] Reports of the ineffectiveness of calcium infusions[20,35] may be related to the small dosages of calcium utilized (eg, 10 ml. of calcium gluconate[35]) considering the competitive type of interference suggested for ciguatoxin.

Respiratory therapy including ventilation support should be utilized in combating respiratory failure if it occurs. This is the common mechanism for ciguatera fatalities.[26,31]

The general initial regime of intravenous electrolyte solution, vitamins and atropine at therapeutic dosages (0.4-1.0 mg. q 3-4 hours subcutaneously) together with additional symptomatic treatment was used in American Samoa during the period of this study (1970-1975) and no ciguatera related deaths occurred during this period. Until more data accumulate, the possible benefits of any therapeutic modality should be weighed carefully against the potential risks. The early administration of an oxime (Bagnis[15]) and high doses of atropine (Okihiro[32]) have received empirical validation; however, if the mechanism of ciguatoxism in man is consistent with the laboratory results of Rayner,[38] large infusions of calcium salt might prove to be the treatment of choice for life-threatening ciguatera if adequate clinical and biochemical monitoring is available.

Acknowledgements

I am grateful to the personnel at the LBJ Tropical Medical Center, especially the OPD physicians and nurses and the medical records department for their assistance in compiling the data, and to Dr. Richard Wass, Fisheries Biologist, Office of Marine Resources, Pago Pago, for his help with the scientific names of local fish.

*Following Michaelis-Menten kinetics, except at low calcium and high ciguatoxin concentrations.

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