Environmental Health

CIGUATERA IN THE PACIFIC: A LINK WITH MILITARY ACTIVITIES

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Summary Ciguatera fish poisoning is widespread in the Pacific. Outbreaks and the rise in incidence of the disease are related largely to military activities that disturb coral reef ecology. Nuclear test explosions and the setting up of the infrastructure for these uses are major components of such military activity.

INTRODUCTION

Ciguatera, the commonest type of marine fish poisoning world wide, is caused by the ingestion of tropical fish containing toxins produced by coral reef dinoflagellate plankton species. The toxins have no observable effect on fish, and the highest concentrations are found in large predatory fish that feed on herbivorous fish, favoured by man for food. Ciguatera is especially common in the Caribbean and in the Pacific. Ciguatera most commonly presents as an acute self-limiting syndrome of vomiting, diarrhoea, and abdominal pain, accompanied by sensory and/or motor disturbances. Weakness may necessitate ventilatory support. Bradycardia, myocardial depression, and vasomotor collapse may occur. Ciguatera during pregnancy may result in abortion, premature labour, and neurological disease in the newborn, and the disease may be transmitted by breast-feeding. Ciguatera is occasionally fatal, but more commonly it runs for weeks, months, or years, with constitutional symptoms, diffuse tingling pain, and ataxia often being the most persistent symptoms. The diagnosis is clinical and therapy is predominantly supportive and symptomatic. Despite reports of striking improvement with mannitol, no treatment is of proven efficacy. Subsequent attacks tend to be more severe than first attacks, and symptoms may recur with ingestion of non-toxic fish, alcohol, and other foods, or with factors such as intercurrent illness. Such “sensitisation” may persist for years after an attack of ciguatera.

The dinoflagellate species usually implicated in ciguatera is Gambierdiscus toxicus, the predominant benthic dinoflagellate in Pacific coral reefs. Ciguatera endemicity correlates with the environmental density of G toxicus in French Polynesia, but not in other areas (e.g., Queensland). The most striking factors precipitating ciguatera outbreaks are disturbances in the ecology of coral reefs, by natural events such as storms, earthquakes, tidal waves, and heavy rains, or by human activities such as construction works, explosions, and dredging. In this paper the relation with military activity is examined.

MILITARY-RELATED OUTBREAKS

The Pacific Islands are divided ethnogeographically into three broad areas: Micronesia, Melanesia, and Polynesia (fig 1). The Pacific has been used extensively for military activities. Naval, air, and communication facilities are maintained in the region and three countries fire missiles to a number of splashdown points. Nuclear test explosions have been conducted by the British in Australia (12 in 1952–57) and at Christmas and Malden Islands (9 in 1957–58). US Pacific tests have been carried out at Eniwetok and Bikini in

E. A. S. NELSON AND OTHERS: REFERENCES—continued

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10. Ciguerita most commonly presents as an acute self-limiting syndrome of vomiting, diarrhoea, and abdominal pain, accompanied by sensory and/or motor disturbances. Weakness may necessitate ventilatory support. Bradycardia, myocardial depression, and vasomotor collapse may occur.

**Marshall Islands, Micronesia**

These islands were the site of several battles during the 1939–45 war. As a result of military activity then, Kwajalein and Majuro replaced Jaluit as the administrative and commercial centre of the Marshalls. As the populations here increased there were reports of ciguatera in these two areas, especially at Majuro, the rise in the 1950s being attributed by the Marshallese to the nuclear test programmes carried out there between 1946 and 1958.

Despite the reported absence of ciguatoxic fish from Eniwetok in the years following nuclear test explosions there, severe outbreaks of ciguatera occurred in other atolls in the Marshalls. Outpatient department records of the Marshall Islands Memorial Hospital on Uliga Island of Majuro Atoll for 1955–57 suggested an average annual fish poisoning attack rate for the local population of 9.5%. Whereas clinic visits overall increased by 60% over this period, gastrointestinal illness (much of which may have been ciguatera) and fish poisoning increased two to three fold. Household surveys on Rita, Uliga, and Majuro Islands suggested an incidence rate for the previous year of around 15%. In health statistics for the whole US Micronesian Strategic Trust Territory of the Pacific Islands, fish poisoning used to be included under “accidents”—and in the late 1950s the accident rate in the Marshalls was at least 50% higher than that for the whole Territory.

In a 1982 survey requested by the WHO Regional Office, 56% of families in the Marshalls reported that at least one member had been poisoned within the last year. Only since 1982 have health statistics for the former US Strategic Trust Territory of the Pacific Islands (TTPI) been recorded separately for its components—the Northern Mariana Islands, the Federated States of Micronesia, the Marshall Islands, and Belau. Between 1982 and 1987, the reported annual ciguatera incidence rate for the Marshall Islands averaged over 300 per 100 000 per year, more than three times the rate of any other Micronesian territory (table 1). There is little doubt that ciguatera continues to be a greater problem in the Marshalls than elsewhere in Micronesia. The most plausible explanation is the extensive military infrastructure and activities related to the 66 nuclear test explosions at Eniwetak and Bikini between 1946 and 1958, and to the Kwajalein Missile Range. Alternative explanations have not been offered.

**Gilbert Islands, Kiribati (Central Pacific)**

On several of the Gilbert Islands, outbreaks of ciguatera were closely related temporally to naval shipping activities and battles in the 1939–45 war, the link being clearest for those at Butaritari and Tarawa (site of the 1943 Battle of Tarawa).

**Line Islands, Kiribati**

The first recorded outbreak at Fanning Island, previously free of ciguatera, followed the dumping of war material (tank mines, ammunition, batteries, and so on) by the US Army in July, 1943, before evacuation of the island. 95 cases of ciguatera were recorded in a population of 224 between February, 1946, and April, 1947. Except for 1 case illness followed the eating of fish caught in areas where war material had been dumped. Toxicity decreased during the 1950s. This outbreak at Fanning was part of the more widespread appearance of ciguatera through the northern Line Islands in the 1940s that started during the 1939–45 war. Local residents reported that there had been no ciguatoxicity before then. The only island unaffected was Washington Island—the sole island in the group where military forces were not stationed and which no military vessels visited. Palmrya Island, Kingman Reef, and Christmas and Fanning Islands, along with Johnston and Midway Islands further to the north, were affected.

**Hao Atoll, Tuamotu Archipelago, French Polynesia**

The outbreak at Hao is the best known example of a military-related outbreak of ciguatera. Ciguatera had not occurred in Hao before January, 1965, when it is generally said that the French Commissariat à l’Energie Atomique (CEA) began to convert the atoll for use as a staging base for nuclear testing at Moruroa and Fangataufa Atolls, several hundred kilometres to the south. Newspaper and Territorial Assembly records in fact show that members of the French Foreign Legion were illegally dispatched to both Moruroa and Hao, to begin construction work in July, 1963. The building of an army camp for 2000 soldiers, a permanent base for nuclear bomb assembly, and a large military airfield with a 3500 m runway required the construction of piers and dredging of large quantities of coral.

The first case of ciguatera reported from Hao involved fish caught at the original French landing site in August, 1966. The disease then spread contiguously to other areas of the atoll over the next 2 years. The first fish to become toxic were herbivores; by April, 1968, carnivores were causing poisoning. By mid-1968, 43% of the population of 650 had been affected by ciguatera. Not till 1975 did toxicity in herbivorous fish begin to fall.

**Gambier Islands, French Polynesia**

A dinoflagellate organism was first clearly implicated as a cause of ciguatera during the 1968 outbreak in the Gambier Islands, after which it was named. Construction of the military base at the Gambiers began in 1967. Before the detonation of the first hydrogen bomb on Aug 24, 1968, a fallout shelter was built for the local population of
was a 100-140 kilotonne explosion beneath the reef at Moruroa, Tuamotu Archipelago, French Polynesia. The most striking example of direct damage to the military infrastructure and by the explosions has been extensive coral damage caused by the building of a military base, and reported to have been heavily contaminated by the first French thermonuclear explosion (1968). 23-32

Other Islands of Tuamotu Archipelago, French Polynesia

Military activity may also be implicated in the following outbreaks of ciguatera that have occurred since the 1960s in several atolls of the Tuamotus, 29-31 especially since no other explanation has been offered: (1) at Fakarava (western Tuamotus), which, like Mangareva, was visited by contaminated warships; and (2) at Pukaru and Reao (eastern Tuamotus), which lay within the “danger zone” for aircraft that applied during atmospheric tests.

No data on ciguatera are available for Fangataufa, site of a military base, and reported to have been heavily contaminated by the first French thermonuclear explosion (1968). 23,32

RELATION BETWEEN RADIOACTIVITY AND CIGUATERA

The outbreaks cited above raise the question of whether radioactivity is related to ciguatera. The only reported study addressing this issue was conducted in the Central Pacific 30 years ago and was supported by the US Atomic Energy Commission. 31 No correlation between radioactivity and ciguatoxicty was found in fish from a variety of locations, including Eniwetak and Majuro; the possibility that nuclear explosions may have caused ciguatera by other means was not considered. Whether radioactive fallout is related to ciguatera deserves further study, especially in view of the likelihood of sizeable leakage of radionuclides from Moruroa within 10-100 years. 33

INCIDENCE

Ciguatera is substantially underreported—the South Pacific Commission estimates that official statistics reflect only 10–20% of actual cases. 14 The South Pacific Epidemiological and Health Information Service has collected ciguatera morbidity and mortality data since 1973. 15 Although incomplete, their records form the best available database on overall ciguatera epidemiology in the South Pacific. For the region as a whole (excluding Papua New Guinea), the reported annual incidence for 1987 was 219 cases per 100 000 population per year. 15

For the period 1973–87 rates more than three times the regional average were recorded in French (eastern) Polynesia, some of the isolated island groups in the north central Pacific (Kiritati, Tokelau, and Tuvalu), the Marshall Islands in north-east Micronesia (data for 1982-87 only), and Vanuatu (1985-87). 15 Compared with the experience in French Polynesia, ciguatera occurs less commonly in the Melanesian nations to the west (Papua New Guinea and the Solomon Islands), where the population is less dependent on marine resources, and where morbidity reporting is less complete. 14

TABLE II—CIGUATERA INCIDENCE BY ARCHIPELAGO, FRENCH POLYNESIA, 1960-84

<table>
<thead>
<tr>
<th>Archipelago</th>
<th>Average annual incidence per 100 000 population, 1960-84</th>
</tr>
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<tbody>
<tr>
<td>Gambier</td>
<td>22 700</td>
</tr>
<tr>
<td>Marquesas</td>
<td>4300</td>
</tr>
<tr>
<td>Tuamotu</td>
<td>1700</td>
</tr>
<tr>
<td>Society</td>
<td>500</td>
</tr>
<tr>
<td>Austral</td>
<td>100</td>
</tr>
</tbody>
</table>

Based on: Danielsson and Danielsson 15
Between 1960 and 1984 there was clearly a general flare-up of ciguatera in French Polynesia, with more than 24,000 cases being recorded among a population that grew from 84,500 in 1962 to 174,000 by mid-1985. The incidence rose sharply through the 1960s, peaking in 1972-75 at 1200 per 100,000, a ten-fold increase over the 1960 figure (fig 2). Improved case reporting has never been presented as a major reason for the increase.

In the areas most affected—the eastern Tuamotu, Gambier, and Marquesas Archipelagos—the decline in annual regional incidence varies widely (table 1), with sustained and the incidence in the 1980s remains high. The poisonings recorded in Tahiti (Society Islands) were from fish caught in the Tuamotus. Pooling of data by archipelago tends to dilute high incidence figures from particular islands.

The basic biogeographical phenomenon of decreasing diversity of marine and terrestrial fauna from west to east in the Pacific may help explain the susceptibility of eastern Polynesia and some small island chains further north to ciguatera. Reduced biological diversity and interspecies competition may be advantageous for opportunistic organisms such as ciguatera plankton. Simple ecosystems may be less resilient to a variety of insults than their more diverse counterparts.

CIGUATERA AND NUCLEAR TEST EXPLOSIONS

The examples of ciguatera outbreaks and the changes in incidence of ciguatera cited above indicate that in the Pacific nuclear test explosions may be related to ciguatera both directly and indirectly. Directly, nuclear explosions damage plants and animals: a guide for Australia. Brisbane, Queensland Museums, 1986: 15-36.

REFERENCES

33. References continued at foot of next page
Communicable Disease

HEPATITIS B INFECTIONS AFTER GYNECOLOGICAL SURGERY

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Summary When acute hepatitis B developed in 3 patients who had had gynaecological surgery, the surgeon was found to be a carrier of hepatitis B e antigen. Of 268 patients operated on by this surgeon in one hospital, 247 were screened for markers of recent or current hepatitis B. 22 (9%) had such markers, associated with symptoms in 5. The operations carrying greatest risk of infection were hysterectomy (10/42) and caesarean section (10/51). These findings strengthen the case for vaccination of all surgeons and medical students against hepatitis B.

INTRODUCTION

The occupational risk to health-care workers of acquiring hepatitis B, although small, is well-recognised. Transmission from staff to patients has also been described in association with gynaecological surgery,¹² cardiac surgery,¹³ dentistry,¹⁴ and general practice,¹⁵ although it appears to be an uncommon cause of acute hepatitis B virus (HBV) infection. We describe a seroprevalence study of contacts after an outbreak of hepatitis B which was traced to a gynaecologist.

THE OUTBREAK

In April, 1987, 2 women were found to be positive for hepatitis B surface antigen (HBsAg), neither of whom had any obvious risk factors for infection. The investigations were done because one had acute hepatitis, and the other—a symptom-free diabetic—had abnormal liver function when tested to see if she was suitable to be entered into a drug trial. A computer search for previous laboratory investigations on these patients revealed that both women had been patients on the same gynaecological ward several months before. The lists of patients found to be HBsAg-positive during the preceding months were examined, and another woman with no obvious risk factors was identified; she had had a caesarean section several months before. The notes of these 3 patients were obtained; no patient had any apparent risk factors for hepatitis B, but all had been operated on by the same surgeon. A blood sample from this individual was obtained and found to be positive for HBsAg and hepatitis B e antigen (HBeAg). Electron microscopy of serum showed an abundance of HBsAg and numerous Dane particles.

The surgeon stopped operating immediately. A committee to manage the outbreak was set up, and included representatives from the hospital’s unit management and infection control teams, and obstetrics and gynaecology department; the Department of Health and Social Security; and the Communicable Disease Surveillance Centre. It was decided that all patients who had been operated on by this surgeon during the 5 months in post and during research appointments in the previous year should be identified and offered serological screening, as should all staff who had assisted at these operations.

PATIENTS AND METHODS

Patients who had been operated on by the surgeon between October, 1985, and February, 1987, were identified from theatre records and patients’ notes. Patients were classified as at high, medium, or low risk of exposure to hepatitis according to the type of operation. (Eg, major gynaecological surgery or caesarean section was considered high-risk; cone biopsy or forceps delivery with episiotomy as medium-risk; and dilatation and curettage or termination of pregnancy as low-risk.) Letters were sent to all patients, who were urged to attend for screening; their general practitioners were also informed. A telephone number was set aside for urgent inquiries by worried patients and general practitioners. Information notices were prepared for hospital staff and the Information Liaison Officer to anticipate any media inquiries and to reduce alarm generated by misinformation.

Arrangements were made for special sessions in which groups of patients were counselled and blood samples taken. Screening of patients was continued until 6 months after the date of surgery; high-risk patients were screened monthly; medium-risk women at the first session and then 3-monthly; and those at low risk initially and at 6 months after surgery. The obstetric patients had been screened for rubella antibodies in early pregnancy; these samples had been stored and could be tested for preoperative evidence of hepatitis B infection.

Patients infected with HBV were interviewed and counselled by a virologist and the infection control nurse, and their sexual partners and babies were also screened. Prophylaxis was offered to partners who had had sexual contact in the post-exposure period, and for infants who were breast-fed or who had had other potential exposure. (High titre anti-HBs immunoglobulin and hepatitis B vaccine were given to 3 partners of HBsAg-positive women, and vaccine alone to 3 babies of women who had seroconverted after caesarean section.)

Members of the scrubbed teams who had operated with the carrier surgeon were identified, counselled by the occupational health department, and screened for HBV markers.

All blood samples were first tested for HBsAg by radioimmunoassay (RIA); if positive they were tested for HBeAg and its antibody (anti-HBe) by RIA. HBsAg-negative samples were tested for the presence of antibodies to the hepatitis B surface antigen (anti-HBs) and antibody to hepatitis B core antigen (anti-HBc) by enzyme immunoassay; if positive they were tested for hepatitis B surface antibody (anti-HBs) by RIA. Stored antenatal serum samples were also tested, when available, for all patients found to have hepatitis markers. (Abbott reagents were used for these tests.) Samples with evidence of infection by hepatitis B were tested for IgM antibody to core antigen (anti-HBc IgM) by RIA, and HBsAg-positive samples were subtyped, at the Middlesex Hospital.

Since patients were thought likely to ask about their risk of infection by human immunodeficiency virus (HIV), we tested the surgeon’s blood for antibodies to HIV, after consent was obtained; the test was negative.

RESULTS

266 patients were identified to be at risk, of whom 114 were classified as high-risk, 117 as medium-risk, and 37 as low-risk. 22 (9%) of 247 patients tested had evidence of recent or current infection, 21 of them in the high-risk group