SPC/ICHT/WP.2
16th April, 1968.

SOUTH PACIFIC COMMISSION

SEMINAR ON ICHTHYOSARCOTOXISM
(Rangiroa, French Polynesia, 16th - 22nd August, 1968)

CLINICAL FORMS OF FISH POISONING OF THE "CIGUATERA" TYPE IN FRENCH POLYNESIA

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Cases of poisoning due to the ingestion of venenous fish have for a very long time been known to occur in French Polynesia.

The reflexions set out below are to a large extent the results of personal observations gathered more particularly in the Tuamotu Group and on the Island of Tahiti. Also, a certain amount of information was provided by various Medical Officers of the Territorial Health Department. (2)

In all, 350 observations have been analysed.

In French Polynesia, ciguatera presents a polymorphous clinical picture with, however, a few characteristic features which enable a ready diagnosis to be made.

We shall adopt as typical for the purpose of our description the most complete form with its various syndromes: digestive, neurological, cardiovascular and algic with repercussions on the general condition (69 personal observations).

Incubation

Very variable according to the individual, the species, the quantity of toxic flesh ingested, but generally short: from two to twenty hours; this latent period may be reduced to thirty minutes or so in severe cases.

Onset

The first symptom to appear is paresthesia: prickling about the lips, tongue and nose, a tingling sensation in the extremities of the limbs.

At the same time: a state of indisposition accompanied by facial congestion, cold perspiration and nausea which herald the digestive phase.

This initial period lasts approximately two hours.

Established Case

The full clinical picture is forming. It is that of toxic polyneuritis with four fairly clearly individualized syndromes:

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a) Digestive Syndrome

This sets in early. The feeling of nausea is rapidly followed by slight vomiting, then by repeated attacks of abundant, watery diarrhoea accompanied by abdominal cramp and colic.

These symptoms generally subside within twenty-four hours, leaving the patient asthenic and dehydrated.

b) Cardiovascular Syndrome

- Slow pulse, between 35 and 50 beats per minute, often irregular.
- Muffled heart-beats.
- Reduced blood pressure.
- An electrocardiogram will show:-
    1) disturbances in the rhythm of regular sinus bradycardial type and of the supraventricular or ventricular extra-systole type, in bursts.
    2) Interference of conduction of first degree atrioventricular heart block type.

However, the number of recordings made (22 in all) is still insufficient to enable any conclusions to be drawn and we intend to pursue our investigations in this field.

c) Neurological Syndrome

- Dysthesia affecting mainly sensitivity to cold objects, with painful tingling of the extremities on contact with cold water.
- Superficial hyperesthesia with sensation of burning or electric discharge.
- Frequent mydriasis.
- More rarely, paresis localised in the lower limbs with hypo-reflexia of the knee and ankle.

d) Algic Syndrome

- Arthralgia localised mainly in the large joints: knee, ankle, shoulder, elbow.
- Myalgia affecting in particular the thigh and leg muscles.
- Cephalgia, dorso-lumbar rachialgia.

e) Accompanying Syndromes

- Severe asthenia making walking difficult and sometimes confining the patient to bed for several days.
- Feelings of malaise and dizziness.
- Subsequent appearance - two to three days after ingestion - of intermittent pruritus persistent at times.
- Constant and marked chills.
- Frequent oliguria during the first forty-eight hours.
- No heat regulation disorders.

LABORATORY TESTS

In medical wards at the Papeete Hospital we systematically carry out a certain number of supplementary tests: white cell count, ionogram, alkaline reserve, protein electrophoresis, colorimetric dosage of serum cholinesterases.

Up to the present, results of these tests have not revealed any characteristic or consistent anomalies; in particular, using the "Acholest" colorimetric dosage techniques, we have never been able to find a concrete example of a cholinesterases inhibition, even in cases of severe poisoning.

DEVELOPMENT

In French Polynesia ciguatera is rarely fatal. In the course of the last three years, only three deaths have been ascribed to it, and even then it was not possible to determine the precise cause of death with any degree of certainty.

The clinical cardiovascular symptoms begin to subside within a few days; the electrocardiographic disorders are rectified within forty-eight to seventy-two hours.

The neurological symptoms regress more slowly. They persist for at least a week, but it is not uncommon for dysesthesia of contact to continue for one month.

The asthenia, chill and pruritus also persist for a fortnight or so.

CLINICAL FORMS

The symptomatology is not always as clearly defined, since each individual's ways and means of reaction can produce an infinite variety of clinical forms.

However, we felt it would be useful to differentiate between a few characteristic clinical forms according to the grouping or the degree of severity of certain symptoms, the frequency with which they manifest themselves and the causal species.

A) According to the symptomatology:

- Forms showing increased neurosensitivity (112 cases)

  Paresthesia, dysesthesia of contact, chills, myalgia and arthralgia dominate the picture, causing the mild digestive symptoms to take second place. These are often of the diarrhoea type, unaccompanied by vomiting.

- Forms where digestive disorders predominate (58 cases)

  Vomiting and diarrhoea go on for three to four days and cause a state of dehydration accompanied by intense thirst, dryness of the tongue and skin, severe oliguria.

- Pruriginous forms (29 cases)

  Intense pruritus, beginning towards the third day of development, at first localised in the extremities, then generalised, continuous, particularly irritable at night, causing insomnia and lesions through scratching.

- Erythematous form (14 cases)

. A genuine rash appearing approximately twelve hours after ingestion, with flushes and puffiness of the face, patches of the urticaria type, extremely pruriginous in the folds of the limbs, diffuse infiltration of the integuments; resolved in four to five days after a desquamation phase.

These forms frequently appear in children.

- Forms where cardiovascular disorders predominate (7 cases)

Bradycardia at less than 40/minute, hypotension with reduction of the differential, tendency towards collapse and constant electrocardiographic disorders.

- Forms where neuro-motor disorders predominate (7 cases)

Paresis of the lower limbs is in the forefront, making walking impossible. Dysphonia is often associated with it.

- Forms where sensory disorders predominate (4 cases)

In addition to the symptoms of motor incoordination, severe attacks of dizziness, visual or auditive hallucinations during the day and nightmares with zoopsia at night may be noted.

- Asymptomatic forms (12 cases)

When the same fish has been ingested by several people, some appear unscathed, whereas others are affected. In the case of the former, the threshold of toxicity has not been reached whereas it has been crossed in the case of the latter.

B) Forms caused by the effect of summation (8 cases)

These occur when two toxic fish, whether or not they belong to the same species, are eaten at one or two days' interval. The quantity of toxin ingested separately during each of the meals is not large enough to reach the stage at which clinical symptoms appear, whereas the two together give rise to a severe intoxication.

C) Reiterative forms (28 cases)

These are pure anaphylactic forms, neurological symptoms predominating, with dysesthesia, paresthesia and pruritus, mild most of the time, but may be particularly severe in subjects who have become allergic to one or several species of fish.

D) Forms depending on the causal species

It has not, to date, been possible to establish a clinical scale of toxicity in relation to the causal species. However, in the light of our personal observations during the past three years, it is significant to note that:-

- surgeon fish which are herbivorous appear as the origin of neuro-digestive or neurological forms, with sensory disorders predominating and with very few cardiovascular repercussions.

- on the other hand, groupers, sea bass and other piscivorous fish give rise to more complete forms where cardiovascular attacks are very frequent and where, nevertheless, the scale of neurodigestive symptoms is not in any way reduced on that account.

The establishment of a clinical fact of this kind should be confirmed by a toxicological investigation of the various species.

POSITIVE DIAGNOSIS

This will be made first of all on the basis of interrogation, which will reveal the fact that a fish belonging to a known or suspect toxic species was ingested in the hours preceding the onset of the clinical symptoms; then, on the basis of three characteristic clinical symptoms: disturbance of sensitivity, bradycardia and mydriasis.

DIFFERENTIAL DIAGNOSIS

- Intoxication by tetraodons or moon-fish (2 observations)

The time of incubation, initial and accompanying symptoms, digestive symptoms, hypotension and mydriasis appertaining to a typical case of ciguatera are to be found here.

On the other hand, tachycardia, rapid pulse, flaccid paralysis which is neither fixed nor systematized (except in cases of respiratory paralysis) are specific symptoms.

- Histaminic intoxications (16 observations)

The rash accompanied by congestion of the face, edema of the eyelids, injection of the conjunctival vessels, scattered pruriginous urticaria patches - these symptoms may recall the erythematous form of ciguatera.

However, a knowledge of the causal species (tuna and skipjack most of the time), the long interval between the time of fishing and the time of consumption, the symptoms which appeared simultaneously amongst all those who partook of the fish, and finally a constant feverish condition - 100.4°F or over - all these factors enable a ready diagnosis to be made.

- Allergic reactions (2 observations)

The clinical picture of pruriginous urticaria is to be compared both with the erythematous forms of ciguatera and the cases of histaminic intoxication.

The difference lies in the sudden and early nature of its appearance (a few minutes after ingestion), in the anamnesis which leads to the discovery of similar accidents whenever any kind of sea-food, however infinitesimal the quantity, is being eaten, and the frequent association of other allergic symptoms (asthma attacks in the two cases observed).

PROBLEM OF NATURAL IMMUNITY

A certain number of people declare they have never been poisoned, although they eat species which come from suspect areas. Others say that they have on several occasions eaten the flesh of fish which gave rise to ciguatera symptoms in the case of other people who partook of the same fish, and yet have themselves experienced no disorders. It seems difficult to speak of natural immunity as long as we have not made further advances in our biological knowledge of the human phase of ciguatera, the more so as we very often receive people who are convinced that they are immune, do not hesitate to eat suspect fish of whatever kind, and end up one day by presenting a typical case of ciguatera.

TREATMENT

- Symptomatic

Up to the present the treatment of ciguatera has been purely symptomatic.

- Antispasmodic drugs, lactic-acid bacilli, vegetable charcoal, for example for the digestive dis.rders;

- Combination: vitamin B complex - colchicine - salicylic acid, for the neurological disorders;

- Synthetic antihistaminic drugs, magnesium hyposulfite, for the pruriginous symptoms;

- Cardiovascular analeptic treatment, steroïds in cases of shock or conditions of pre-collapse.

- Etiologic

Taking into account the results of pharmacological studies of the poison (Kwang Ming Li 1965), we tested in the Hospital at Tahiti the oxime type cholinesterases regenerators.

In this way we have used successively "2 Formyl 1 methyl pyridium chloride" (Protopam) and "Methyl sulphate of pyridine 1 aldoxime" (Contrathion), the dosages ranging from 200 mg to 1 g in intravenous perfusions of 250 cc of glucose serum to be given fairly rapidly. To this we add $\frac{1}{2}$ to 1 mg. of atropine and a vitamin B complex with 250 g of vitamin B 6.

Within a few hours a spectacular improvement may be observed, and the only symptoms which persist for three or four days are dysesthesia, myalgia and a slight pruritus.

The results are all the more rapid and patent that these are serious forms in which cardiovascular symptoms predominate and that therapy was brought into play at an early stage.

It must be pointed out, however, that we subsequently obtained almost as good results through the independent use of vitamin B complex with 250 mg of B 6 in intravenous perfusion. Furthermore, recent pharmacological studies (Rayner, Kosaki and Fellmeth) have not confirmed the initial observations made by Kwang Ming Li.

In conclusion, the symptomatology, whilst extremely varied, presents a few permanent features which enable a ready diagnosis to be made.

Unfortunately, most of the disorders are purely subjective and the absence of consistent biological symptoms, or failure to recognize them, prevents our understanding the precise mechanics of the action of various toxins in the body.

However, with the help of precise interrogation, more complete clinical observations, systematic laboratory examinations, we hope to be able to identify a certain number of "combinations of clinical and biological symptoms" corresponding to specific fish families.

In this way perhaps, in addition to administering symptomatic therapy in a more rational way, we shall be able to develop a rapid specific treatment in relation to the cause.

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