
CLINICAL NOTE

Paralytic shellfish poisoning

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We report the case of a man with paralytic poisoning caused by eating mollusks. The patient presented with severe respiratory failure and required mechanical ventilation for 48 hours. This syndrome, which is very rare in Spain, can be fatal if the respiratory muscles are affected and proper treatment is not provided. [Emergencias 2009;21:306-308]

Key words: Paralytic shellfish poisoning. Dinoflagellates. Saxitoxin. Tetrodotoxin. Red tide.

Introduction

Paralytic shellfish poisoning (PSP) is paralyzing biological poisoning due to the ingestion of marine shellfish, mainly bivalve mollusks. In Spain the most common are mussels. Shellfish may accumulate, on a seasonal basis, potent neurotoxins produced by a type of unicellular organism of oceanic phytoplankton called dinoflagellates^{1,2}. Massive proliferation is called the red tide³.

Neurotoxins have a selective action on the peripheral nervous system and skeletal muscle. Saxitoxin is one of the most common neurotoxins isolated and studied in cases diagnosed in humans. It binds to the voltage-dependent sodium pump of cell membranes, blocking it and interfering with the transmission and conductivity of nerve impulses, and produces neuromuscular paralysis without loss of consciousness. The incubation period of symptoms is short, from seconds to minutes⁴⁻⁶. Overall mortality is estimated at 10% and prognosis is related to how quickly adequate ventilation is established and maintained⁷.

Due to the lack of an antidote, immediate treatment is symptomatic and supportive; recommendations include induced vomiting, gastric lavage, enemas, use of saline solution and mechanical ventilation^{5,6,8}. There are different degrees of human susceptibility to the toxin. There are people who have a natural tolerance to large amounts of the toxin and others who show acquired tolerance⁹, which raises the question of the role of the immune system in the development of the disease.

Case report

This patient was a 49-year-old male who habitually consumed alcohol without visceral impact. While at home preparing food, after breaking the shell of a sea snail to remove the animal (Figure 1), he ate a piece of bread soaked in the visceral juice. After ten minutes he noticed a tingling sensation of the mouth, general malaise, dysarthria and generalized loss of strength.



Figure 1. Triton Sea Snail.

He alerted the emergency medical system, which transferred him to a hospital. On arrival he presented agitation and progressive dyspnea which was followed by decreased level of consciousness and coma; he received endotracheal intubation for airway maintenance. He did not require sedation but presented an episode of tachycardia rhythm with self-limiting narrow QRS which became a sinus rhythm, blood pressure of 130/65 and excellent O_2 saturation, at which point he was transferred to our centre. Here his blood pressure was 90/50 mmHg (which responded to crystalloid infusion), with hemodynamic and respiratory stability. Neurological examination showed: a Glasgow Coma score of 3, symmetric bilateral unresponsive mydriasis, complete absence of corneal, brainstem and tendon reflexes, and lack of rigidity. Emergency department abdominal ultrasound was normal. Haemogram, coagulation, biochemistry, blood gases, venous D-dimer and cranial CT scan were normal. Urine and blood tests for illicit drugs were negative, and alcohol level was 41 mg/dL (0.41 g/L). The patient was admitted to the intensive care unit.

He received 3 vials of slowly infused antbotulin, without improvement. Electroencephalogram showed normal brain bioelectrical activity; electromyogram and electroneurogram showed complete block of peripheral electrical transmission, compatible with severe mixed polyneuropathy. He presented nosocomial *Haemophilus influenzae* (blood and bronchoaspirate cultures), for which amoxicillin-clavulanate antibiotic was empirically prescribed, later followed by 3rd generation cephalosporin. The patient recovered gradually from flaccid tetraparesis and presented trunk reflexes; he was extubated 48 hours later with complete functional recovery and without evidence of residual neurological damage. Ten days later he was discharged without sequelae.

We identified the mollusk responsible (Figure 1) as the gastropod *Charonia lampas lampas* (triton sea snail), a carnivorous predator widely distributed in Spain and the North Atlantic, which had been part of a catch by Portuguese fishermen sold at markets in Malaga. Toxicological analysis of the sample involved in this poisoning (consisting of the remains of the shell partially consumed by the patient), was performed at the Provincial Laboratory of the Malaga Public Health service. Analytical mouse bioassay was applied, following the AOAC procedure, published in our legislation as the official method⁷. First, part of the foot or flesh was analyzed for paralyzing biotoxins, after the process of extraction and sample preparation, which yielded a result equivalent to 151 mg STX/100 g (toxicity refers to saxitoxin⁷). Although this exceeded the legal limit established at 80 mg/100 g, it was not so high as to produce intoxication. Second, the liver and pancreas remains of the mollusk were analyzed, and in this case paralyzing biotoxins in amounts equivalent to 25,500 mg STX/100 g. were found. A peculiarity of the analysis by mouse bioassay is that it values the overall effect of all toxic compounds present in the sample, after the process of extraction and preparation.

The samples and/or extracts were subsequently analyzed by high performance liquid chromatography (HPLC) with mass spectrometry, and the group of tetrodotoxin (TTX) is identified: in particular 5,6,11-trideoxyTTX at a concentration of 2.75 mg/100 g. The presence of saxitoxin was not detected.

Discussion

PSP presents in the form of violent sporadic outbreaks, associated or not with the presence red tide and massive proliferation (or not) of toxic dinoflagellates. The intake of contaminated seafood results in a variety of symptoms depending on the type of toxin present, its concentration in the shellfish and the amount consumed¹¹.

There are a considerable number of marine biotoxins of the PSP type, consisting of 21 chemical compounds of which the most toxic and representative is saxitoxin⁵. Tetrodotoxins are another paralyzing neurotoxin found in puffer fish and some marine snails, with some effects and mechanism of action being similar to PSP.

In addition to paralyzing poisoning, there are other common types of poisoning causing diarrhea, nervous disorder and amnesia. Diarrhea may

be underdiagnosed because the symptoms are non-specific and self-limiting. Amnesic poisoning caused by the biotoxin domoic acid produces gastrointestinal symptoms and neurological disorders such as confusion, memory loss, disorientation and even coma^{11,12}. Neurotoxic intoxication is due to exposure to brevetoxin-type polyethers^{11,12}.

The diagnosis of PSP is based on symptoms observed and recent food intake; it is confirmed by identification of the biotoxin in the shellfish epidemiologically implicated. Mouse bioassays are used to detect the presence of toxins, and chemical tests with liquid chromatography coupled to a mass spectrometer mass are used for specific identification^{7,10}. To prevent outbreaks of PSP, shellfish samples are regularly collected in breeding areas and analyzed. When toxin levels exceed the level permitted, these areas fall under quarantine and sales are prohibited⁴. Epidemiological surveillance is based on the detection of toxin levels in shellfish. Each program or system must be adapted to the area, region or country in which it is applied. Currently, prevention programs focus on non-consumption of shellfish^{4,11,12}, especially mussels with high doses of toxins, and are not directed specifically towards the elimination the toxin-producing dinoflagellate.

Since PSP may present as an isolated case, difficult to prevent in light of globalization of the seafood market, it is necessary to alert Emergency Department professionals to this possibility, without the presence of red tide and outside endemic areas. Once life-support measures are initiated, electro/neurophysiological tests are most useful

for the diagnosis. It is essential to maintain a fluid relationship between the hospital and reference laboratories in the approach to PSP.

Finally, the public at risk must be alerted, and we would emphasize health education to promote awareness and vigilance for any indication of the disease.

References

- 1 Odum E. Ecología. 3.ª ed. Interamericana p. 368. 1972.
- 2 Paez de León L. Aspectos epidemiológicos sobre la intoxicación parálitica por mariscos. *Veterinaria Tropical*. 1985;10:59-87.
- 3 Pacheco Troconiz G, Toro Alayon G. La marea roja y el problema de la toxina paralizante de los moluscos. *Boletín de Salud Pública (Caracas, Venezuela)*. 1978;31:13-6.
- 4 Fernández R. Descripción de las intoxicaciones producidas por consumo de mejillones (Pernaperna) (Rio Caribe, estado Sucre). 1977;15 p.mimeo.
- 5 López Capont F. La toxina del mejillón y otros moluscos. Su problemática e importancia para España. *Rev de Agro-Química y Tecnología de Alimentos*. 1978;18:47-63.
- 6 Mira Gutiérrez J. Ictiosarcotismo: un riesgo médico y sanitario de la fauna marítima. *Rev San Hig Pub (España)*. 1970;4:460-515.
- 7 U.S.A Food and Drug Administration. Proceeding of the second national conference for food protection. Washington D.C. 1984:141-3.
- 8 Fernández R. Intoxicación por moluscos en el oriente del país. Dola vigilante ante cualquier indicio de enfermedad. *Boletín de Salud Pública (Caracas, Venezuela)*. 1978;31:5-12.
- 9 Medcorf JC, Leim AH, Needler AB, Needler AWH. Paralytic shellfish poisoning on the Canadian Atlantic Coast. *Bull Pish Res Bd Can*. 1947(LXXV):32.
- 10 Reichardt PB, Neve RA, Gershey RM, Hall S, Musgrave DL, Seaton PI, et al. Chemical investigations of paralytic shellfish poisoning in Alaska. *Primeras Jornadas Técnicas Moluscos Gallegos: Mejillón. Asociación de Científicos y Tecnólogos de Alimentos de Galicia; Santiago de Compostela*; 1982. p. 49.
- 11 De Schrijver K, Maes I, Den Man L, Michelet J. Brote de intoxicación alimentaria por consumo de marisco en Amberes, Bélgica. *Euro Surveill*. 2002;7:138-41.
- 12 CFSAN. Various shellfish Associated Toxins. In: *Foodborne Pathogenic Microorganisms and Natural Toxins Handbook*. Maryland. United States. Center for Food Safety and Applied Nutrition; 1993. (Consultado 15 de diciembre de 2007). Disponible en: (<http://www.cfsan.fda.gov/mov/chap37.html>).

Intoxicación parálitica por consumo de marisco

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Se presenta el caso de un paciente con intoxicación parálitica por ingesta de moluscos que cursó con insuficiencia respiratoria grave y requirió ventilación mecánica durante 48 horas. Se trata de una intoxicación muy rara en nuestro entorno y puede ocasionar la muerte por parálisis de los músculos respiratorios si no se atiende adecuadamente. [*Emergencias* 2009;21:306-308]

Palabras clave: Intoxicación parálitica por moluscos. Dinoflagelados. Saxitoxina. Tetrodotoxina. Marea roja.