Clinical Note

Painful ejaculation. Something fishy

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Painful ejaculation as an isolated form is an uncommon presentation in the emergency room, since most patients are secondary to urological conditions such as prostatic cysts, prostatitis, and seminal vesicle calculi.¹ In this clinical note, 2 patients with painful ejaculation after eating mackerel fish that contained ciguatoxin, a very potent neurotoxin are presented for its rarity and to alert practitioners regarding this entity.

Ciguatera fish poisoning (CFP) is a distinctive type of food borne illness that results from eating fish contaminated with ciguatoxin. It has long been known as a seafood linked human disease, and is a well-recognized problem in the tropics. Ciguatoxin is colorless, odorless, lipid-soluble, heat-resistant, not affected by freezing, acid-stable, cyclic polyether compound that does not alter the taste or texture of fish, and the frequency of CFP varies by region throughout the world.² Two young married men aged 25-28 years belonging to the same family presented to the emergency room for painful ejaculation at 1.00 A.M following intercourse. The pain was sharp and burning in nature and located deep in the shaft of the penis. The pain started just before ejaculation and lasted until the penis became flaccid, but urination and defecation were painless. They had no previous experience of painful ejaculation. There was no history of fever, dysuria, urethral discharge or extramarital exposure. They followed conventional heterosexual practice only. There was no history of abnormal sexual behavior or sexual perversion. Just 7 days before this episode both had an episode of watery, non-bloody diarrhea associated with tenesmus 6-8 hours after eating mackerel fish, which was diagnosed as food poisoning and treated symptomatically. At that time both had diffuse pruritus, circumoral tingling, and burning sensation in their throat and hands without any other neurosensory complaints. Systemic and general examination did not reveal any abnormalities. Examination of genitalia in both revealed mild erythema and swelling of the glans penis and per rectal evaluation was normal. Urine analysis, special serology (HIV & VDRL), expressed prostatic secretions, and transrectal ultrasonography of the prostate was within normal limits. Both avoided further sexual activity for a couple of days due to severe pain and experienced similar symptoms, when they engaged in sexual intercourse. Also, their female counterparts, although they had not consumed the fish, complained of dyspareunia following ejaculation by their husband. Their clinical examination did not reveal any abnormality. In the absence of exposure to other toxins or to sexually transmitted diseases, the diagnosis of CFP was considered in both based on clinical history and circumstantial evidences. Currently, there are no reliable bio-markers that can be used to confirm exposure to ciguatoxin.² Both of them improved after treatment with intravenous mannitol and amitriptyline. The painful ejaculation disappeared 4 days after treatment. While on follow up during a period of 3 months, they were comfortable without any sexual dysfunction.

Ciguatoxins arise from the biotransformation in the fish of precursor gambier-toxins produced by Gambierdiscus toxicus, a marine dinoflagellate that lives alongside algae on dead coral or among detritus on the sea floor of tropical and subtropical climates. The toxins are concentrated as they make their way up the food chain. Humans are the final link in a complicated food chain containing ciguatoxin. The contaminated algae are consumed by herbivorous fish, which are in turn consumed by carnivorous fish and finally by humans. The larger the fish consumed, the more likely the toxin and the toxins remain in all parts of the fish, but are concentrated in the viscera, liver, and gonads. Ciguatera toxin causes a range of gastrointestinal, cardiovascular, and neurological symptoms within 1-6 hours of ingesting fish with the toxin and it is rarely fatal.² The toxin activates the voltage-gated sodium channels in cell membranes, which increases sodium ion permeability and depolarizes the nerve cell. This depolarization of nerve cells is believed to cause the array of clinical symptoms and signs.^{3,4} The effects can last for days, months, or years. There are very few reports on painful ejaculation.⁵ Lange et al⁶ reported 2 cases of painful ejaculation followed by female partners complaining of dyspareunia due to a ciguatera toxin similar to the cases presented. They also proposed that the ciguatera toxin or a biologically-active metabolite might be excreted via genitourinary secretions and also be capable of producing dysspermia and dyspareunia upon direct contact with nerve-rich epithelial tissue. Ciguatera is solely a clinical diagnosis with no specific physical findings. Laboratory data are non confirmatory. Mannitol appears to be promising in reversal of these symptoms, and the effect of mannitol infusion is thought to be mediated by the reduction of neuronal edema.⁴ However, the pharmacological basis for the use of mannitol remains speculative. Several anecdotal reports have suggested mannitol may work well during the acute phase of the illness. Amitriptyline has been the most effective drug for chronic neurologic symptoms that often follow ciguatera poisoning. It may act by blocking fast sodium channels that have been activated by ciguatoxin.

Increased overseas travel, the globalization process, increasing consumption of fish as part of a healthy heart diet, and an increase in international exports of large exotic fishes makes CFP no longer confined to endemic areas.⁷ Warming of sea water might expand the ranges of ciguatoxin contaminated fish in many other places hitherto not recorded. As such, practitioners dealing with painful ejaculation or dyspareunia should entertain the possibility of CFP. Also, community oriented educational programmes on CFP are needed for primary prevention.

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