CASE REPORT

Scombroid fish poisoning: successful treatment with cimetidine

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Guss DA. Scombroid fish poisoning: successful treatment with cimetidine. Undersea Hyper Med 1998; 25(2):123–125.—Reported is a patient with a clinical syndrome characteristic of scombroid fish poisoning after ingesting yellowfin tuna that may have been allowed to sit at room temperature for some time before preparation. The patient was treated with an intravenous infusion of cimetidine with prompt resolution of a diffuse, well demarcated, erythematous, pruritic rash. The treatment was without sequelae and permitted early discharge from the emergency department. A brief review of scombroid fish poisoning and its treatment is provided.

scombroid fish poisoning, H2 receptor blocker, H1 receptor antagonists, histamine, cimetidine

Scombroid fish poisoning is one of the most common causes of toxic fish poisoning. It is caused by the ingestion of fish in the suborder Scombroidiae; typically tuna, mackerel, skip jack, and bonito, but has also been associated with non-scombroidal varieties such as mahi mahi and Australian salmon (1–3). Scombroid poisoning is associated with a distinctive clinical syndrome characterized by abdominal cramps, nausea, vomiting, diarrhea, oral burning or metallic taste, flushing, headache, pruritus, and a well circumscribed, raised, erythematous rash characteristic of urticaria. The illness can be of varying severity, may include some or all of the above features, and may occasionally be complicated by hypotension and bronchospasm. Most cases are self-limited and will resolve spontaneously within 4–10 h (4). The illness is believed to be secondary to the ingestion of improperly stored fish which permits overgrowth of bacteria. Microorganisms in turn metabolize histidine present in the flesh of the fish producing histamine. Treatment has traditionally been supportive, comprised of hydration, antiemetics, and H1 receptor antagonists such as diphenhydramine. A review of the medical literature revealed only a single report of treatment with an H2 receptor blocker (5). Reported here is a typical case of scombroid fish poisoning managed effectively with only a single injection of cimetidine.

CASE REPORT

The patient, a 32-yr-old male, chiefly complained of a rash. He indicated that approximately 30 min before arrival at our institution he developed an erythematous, raised, pruritic rash on his arms, legs, and torso. The rash was initially described as splotchy but progressed rapidly to become confluent over large areas of his upper body. The patient denied fever, chills, nausea, vomiting, abdominal cramps, dyspnea, wheezing, or light headedness. Before the development of the rash, the patient noted a period of flushing and a metallic taste in his mouth. These symptoms had resolved before arrival. The patient reported having ingested a cooked meal of tuna about 30 min before symptom onset. The tuna had been caught locally 3 days previously and, although it had been refrigerated in the interim, it may have been allowed to sit at room temperature for some time before cold storage. The patient was accompanied by a friend who had eaten portions of the same fish and indicated a brief period of dysgeusia and flushing but had not developed pruritus or rash. The patient was not taking any medications and could not recall any drug, chemical, or toxic exposures. Past medical history and review of systems were negative. He had a questionable history of penicillin allergy.

Physical examination revealed a pulse of 98, temperature 36.8°C (98.3°F), respiratory rate 18, and blood pressure of 123/77. Head, neck, and cardiac exams were normal. Lungs were clear without wheezes or crackles. The abdomen was soft without tenderness. Examination of the skin revealed sharply demarcated areas of confluent erythema over both arms, the neck, and the upper torso. The rash was slightly raised, blanched with compression, and was nontender.

A presumptive diagnosis of scombroid fish poisoning was made and it was decided to treat the patient with a 300-mg i.v. infusion of cimetidine.

Ten minutes after cimetidine administration, the rash was noted to be somewhat lighter and all pruritus had subsided. Thirty minutes after the infusion, the rash was completely resolved. The patient was observed for an additional 30 min without recurrent symptoms and then discharged home.

DISCUSSION

The precise mechanism of scombroid fish poisoning is not known. It is believed to be secondary to the overgrowth of bacteria present on or under the surface of specific varieties of fish that contain large amounts of histidine in their flesh. A variety of bacteria have been implicated, including Klebsiella pneumoniae and oxytoca, Morganella morganii, Serratia marcescens, Enterobacter intermedium, and Plesimonas shigelloides (6,7). All of these organisms contain one or more enzymes such as histidine decarboxylase which are capable of converting histidine to histamine (8). Although enteral absorption of histamine is limited, and the compound is readily metabolized by the liver, there is good evidence supporting the role of orally absorbed histamine in many, if not all, of the clinical manifestations of illness (9). Histamine is heat stable and once formed by bacterial metabolism will not be inactivated by cooking. Prevention of illness can only be achieved by assuring proper cold storage of fish at all times before preparation and ingestion.

H1 receptor antagonists are the favored treatment for most histamine-related clinical syndromes and have been widely cited in the management of scombroid fish poisoning (1,4,10). H2 receptor antagonists have been used in the management of histamine-related illnesses such as urticaria, but with mixed results. While several studies have indicated efficacy for the H2 receptor antagonist cimetidine to be equivalent to or exceed that achieved with H1 receptor antagonists, others have failed to demonstrate consistent clinical utility or benefit beyond that achieved with H1 blockers alone (11–14). In consideration of the histamine basis for the clinical symptoms associated with scombroid poisoning, it is reasonable to anticipate some benefit from an H2 blocker such as cimetidine. The use of cimetidine in the treatment of scombroid fish poisoning has been noted only once in the medical literature (5). In that report, four patients with presumed scombroid poisoning were treated with intravenous cimetidine with prompt relief of symptoms and no adverse effects. The patient reported here is only the second citation in the medical literature on the successful use of cimetidine in the management of scombroid poisoning.

Cimetidine is easy to administer and, when taken for a limited time, has a low incidence of significant side effects. The principal advantage of this agent and other H2 receptor antagonists when compared to most H1 receptor antagonists is the lack of sedating side effects. Over-sedation from H1 blockers can delay emergency department discharge and limit transportation options.

Case reports concerning the therapy of scombroid fish poisoning will always be limited because the illness remits in time without treatment and, in most circumstances, the diagnosis is based on clinical criteria alone. It is possible that the clinical improvement noted would have occurred without therapeutic intervention, as it is possible that the illness attributed to scombroidiae fish ingestion is secondary to some unrelated etiology. In this case, however, the highly characteristic clinical syndrome and history of recent ingestion of yellowfin tuna that may have been allowed to stand at room temperature for some time before preparation strongly supports the diagnosis of scombrotoxism. Furthermore, the close temporal relationship between cimetidine administration and the rapid and complete clinical response strongly supports the drug's efficacy.

CONCLUSION

H2 receptor antagonists such as cimetidine may prove to be valuable adjuncts in the management of scombroid fish poisoning. If further study or experience with these agents substantiates the clinical utility suggested in this case report, they may be favored over currently utilized medications such as diphenhydramine, chlorpheniramine, and hydroxyzine.

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