Case Report

Ocular Hypotonia and Transient Decrease of Vision as a Consequence of Exposure to a Common Toad Poison

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Received 8 October 2019; Accepted 7 January 2020; Published 17 January 2020

The common toad produces venom (bufotoxin) that is produced in the parotid gland of the toad as well as in the skin. This toxic compound is a potent inhibitor of Na+/K+-ATPase activity. Physiological effects of bufotoxin are similar to those of digitalis and cause increased heart rate and muscle contractions. Ocular toxicity was described. A 67-year-old female patient was admitted to the emergency service because of sudden vision loss and a burning sensation in both eyes after she had been exposed to the poison of a toad. Slit lamp examination showed conjunctival hyperaemia and signs of ocular hypotonia. Topical antibiotic treatment was administered, and after 24 hours, corneal oedema and ocular hypotonia were in remission. Inhibition of Na+/K+-ATPase is a well-known effect of the toad venom. Na+/K+-ATPase is a part of corneal endothelial cells, ciliary body, and iris, and its inhibition caused by exposure to bufadienolides induces corneal dysfunction, decreased vision, and ocular hypotonia. Effects of bufadienolides on the decrease of ocular pressure appear to be very strong, with quick action. This rarely described effect of the bufotoxin can be used as a basis for further research of toad venom and its pharmacological potential.

1. Introduction

The common toad, or European toad (Bufo bufo), is an amphibian found in almost all of Europe (with the exception of Iceland, Ireland, and some Mediterranean islands) and in parts of North Asia and North-west Africa. It can live up to 50 years in captivity, and its age is counted by growth rings on their phalanges [1]. It has green-gray-brown skin covered with lumps that produce bufadienolides. Bufadienolides are produced in parotid glands and the skin of a toad [2]. These toxic compounds are potent inhibitors of Na+/K+-ATPase activity, and toads use these as a natural repellent against predators and also as an immune defence against pathogens [3, 4]. Bufadienolides are divided into bufagenins, the smaller, hydrolysed molecules which have stronger cardiotoxic effects, and bufotoxins, the larger bufadienolide molecules with an amino-acid side chain [5]. Some research suggests that the ratio of bufadienolides depends of environmental factors [6]. Bufotoxin was first isolated by Hienrich Wieland in 1922, and its structure was described by the same team 20 years later (C_{40}H_{60}N_{4}O_{10}) [7]. The physiological effects of bufotoxin are similar to those of digitalis and causes increased heart rate and muscle contractions. Therefore, it is used worldwide in traditional medicine as an aphrodisiac [8] or as an anti-inflammatory agent [9]. Exposure to large amounts of toxin may cause cardiovascular and respiratory symptoms, such as paralysis and seizures, increased salivation, vomiting, hyperkalemia, cyanosis, and hallucinations [10]. Cases of poisoning of children were described after kissing a frog [11]. Ocular toxicity was also described [12, 13].

2. Case Report

A 67-year-old female patient was admitted to the emergency service because of sudden vision loss and a burning sensation in both eyes after she had been exposed to the poison of a common toad. The patient stated that she had been working in a garden when she noticed something moving under a
The ingestion of the common toad’s venom causes corneal oedema and ocular hypotension. Bufadienolides, which are responsible for the venom’s toxicity, inhibit Na+/K+-ATPase, leading to increased sodium permeability across the basolateral surface of the ciliary epithelium. This results in a decrease of ciliary body epithelial sodium concentration, which is critical for aqueous humor production. The inhibition of Na+/K+-ATPase is reversible, and the venom’s effects are temporary.

One hour after exposure, typical symptoms included corneal oedema, ocular hypotension, and ciliary body dysfunction. The venom’s effect on Na+/K+-ATPase is reversible, and the venom’s effects are temporary. In the present case, the venom’s effects were reversed by the administration of topical antibiotic and anti-inflammatory medications.

3. Discussion

The toxic effect of bufadienolides on the cornea is consequent to the inhibition of Na+/K+-ATPase, which is a critical enzyme for maintaining corneal clarity. The inhibition of Na+/K+-ATPase leads to an increase in sodium concentration, which disrupts the balance of sodium and potassium in the aqueous humor, resulting in corneal oedema and ocular hypotension.

In conclusion, the ingestion of the common toad’s venom caused ocular hypotonia and corneal oedema. The administration of topical antibiotic and anti-inflammatory medications reversed the venom’s effects, which were temporary and reversible. Future research is needed to understand the long-term effects of venom ingestion and to develop strategies for preventing and treating such cases.


