

Extraocular muscle palsy after snakebite

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Summary

An 11-year-old male was admitted with the diagnosis of snakebite. The bite mark, edema and ecchymosis were recorded on the left hand back. The patient reported binocular diplopia on the fourth day. Restriction was detected in upper and upper-right gazes in the left eye. Inferior oblique muscle palsy was diagnosed and the patient was started to be followed up. Diplopia improved in time and completely disappeared in the fourth month. Eye movements returned to normal. This case was decided to be reported in order to draw attention to extraocular muscle paralysis which is rarely seen in snake envenomation and usually improves spontaneously. (*Turk Arch Ped 2012; 47: 134-6*)

Key words: Diplopia, paralytic strabismus, snakebite

Introduction

Snake bites may be fatal. The determinant factors include the type of the toxin and the immune response of the individual. The toxins which lead to vital complications usually include hemolytic toxins and neurotoxins. The toxins which cause extraocular muscle paralysis are usually weak neurotoxins (1-4). The paralysis of these muscles following snake bite is a very rare complication and the internal rectus muscle has been reported to be the most commonly affected muscle (5,6). Studies have reported involvement of multiple muscles (3). In this study, a case of snake bite with involvement of the lower oblique muscle was reported.

Case

An 11-year-old male patient was presented to our Pediatric Emergency Outpatient Clinic because of snake bite which occurred 2 hours ago. On physical examination of the patient, edema in the left hand, a bite mark and ecchymosis on the left hand back were found. Systemic examination findings were normal. The patient was administered dexamethasone (8mg), phenyramine hydrogen maleate (45.5 mg) and ranitidine hydrochloride (50 mg) in the healthcare center where he was primarily presented. The patient was hospitalized in our

Pediatric Intensive Care Unit with a diagnosis of snake bite. The patient was given 40 mg polyvalent snake venom antiserum in 100 ml normal saline. Dextran 75 and dextran 40 (500 cc/day) pentoxifylline (8 mg/day), ampicillin-sulbactam (100 mg/kg/day), paracetamol (500 mg tablet 2x1) and fluid treatment with 2500 cc/m²/day were started. "Elevation" was applied in the arm with the bite and cold application was performed on the wound site. On the fourth day of hospitalization, a complaint of diplopia occurred. The Ophthalmology Clinic was consulted. On ophthalmologic examination, vision was found to be 10/10 (uncorrected) in both eyes. Diplopia increased especially with upper and upper-right gazes. Light reactions were normal bilaterally. The patient was orthophoric on the first sight. In the left eye, complete restriction was found on the upper-internal gaze, mild restriction was found on the upper direct gaze and increase in movement was found on the lower-external gaze. The orbital movements were found to be normal in the right eye (Picture 1). No pathology was found in the anterior and posterior segments. Magnetic resonance imaging of the brain and orbita was found to be normal. With these examination findings a diagnosis of lower oblique muscle paralysis in the left eye was made. The patient who was followed up and treated in the Pediatrics Clinic for 11 days was discharged, since no other complication developed. The patient was followed up with regular intervals by the

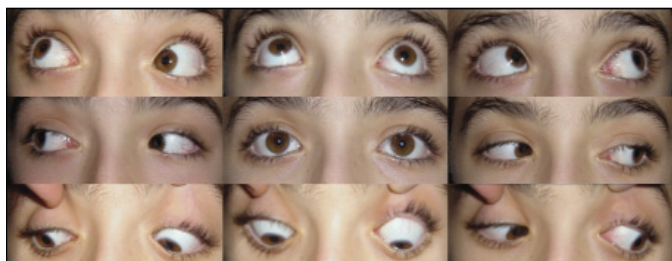
Pediatrics Clinic and Ophthalmology Clinic. On the follow-up visit performed one month later, it was found that diplopia was decreased, the restriction in the upper internal gaze persisted and the restriction in the upper direct gaze was decreased to a great extent. On the follow-up visit performed four months later, diplopia was found to be disappeared completely and eye movements were found to be improved (Picture 2).

Discussion

When the sources are screened, a few snake species which lead to extraocular muscle paralysis are in the front line. The primary ones include Europa viper (*Vipera aspis*), North America Viper (*Agkistrodon blomhoffi*) and some other vipers (1-4). Although the species of the snake which bit our patient was not known, this snake was described as a gray snake with a length of 1.5 m by the patient.

Europa viper is frequently seen in the western part of the North Europa. With its venom it causes vomiting, diarrhea, hypotension, shock, coagulation disorder, thrombocytopenia, weakness, paresthesia and bleeding. Neurotoxicity is observed very rarely and generally involves the muscles in the head (3). Re et al. (3) reported that all muscles innervated by the third, fourth and sixth cranial nerves were involved in a case of ocular muscle paralysis which occurred with bite of an Europa viper. In this article, it was emphasized that the sensitivity of extraocular muscles against snake envenomation might be related to some main features of the neuromuscular conjunction as in diseases involving specifically ocular muscles including myasthenia gravis.

Local edema, pain, local necrosis, muscle degeneration, disseminated intravascular coagulation or hemolysis and acute



Picture 1. Left lower oblique muscle paralysis which appeared on the fourth day in the patient who presented with snake envenomation. There is restriction on the upper internal and upper direct gaze in the left eye



Picture 2. The fourth month of the follow-up. Eye movements are found to be improved

renal failure have been reported in cases of envenomation with the toxin of North America Viper (7-9). The toxin of North America Viper is a hemorrhagic or thrombotic toxin, but it also has a weak effect of neurotoxicity and it may lead to ptosis or diplopia with this effect (4,8,9). Extraocular muscle paralysis occurs more commonly in cases of bite of a cobra snake whose toxin is mainly a neurotoxin (10). Tekeshita et al. (4) reported that strabismus developed in both eyes after three days and paresis was found in the left internal rectus and lower oblique muscles in a case who was bit by a North America Viper.

Mainly, strabismus related to paralysis is observed following snake bites. However, Kim et al. (1) reported a case who developed comitant extropia in both eyes following a viper bite.

We could not find any case of extraocular muscle paralysis related to snake bite among our national publications related to snake bite. It has been reported that the viper family causes snake envenomations which occur in rural areas in Turkey (11). In cases of this type of snake envenomations, hospitalization and supportive treatment are recommended, if envenomation findings are mild (11). Hospitalization and supportive treatment were performed in our case who presented with mild symptoms. No systemic pathology was found in our case, but fatal cases of snake bite have been reported in our country, though with a low rate (12). Therefore, snake bites should absolutely be taken seriously and the patients should be hospitalized and monitored.

When the few studies in the sources related to eye involvement in snake bites are examined, it is realized that different extraocular muscles are found to be involved and no muscle paralysis is observed in other parts of the body. These data suggest that the extraocular muscles have a special sensitivity against snake toxins as Re et al (3) also stated. Again, when these publications are examined, it is observed that paralysis of the extraocular muscles is transient and improve in a few months without leaving sequelae. In our case, diplopia completely improved approximately four months after the incident and orbital movements returned to normal.

Conclusively, we think it should be kept in mind that extraocular muscle paralysis may be observed following snake bite and improves spontaneously without any treatment. These patients should be followed up for a while with regular intervals and surgical intervention does not need to be performed immediately.

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