Case Report

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Binocular Diplopia After A Snakebite

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Abstract

Introduction: Ocular manifestations in snake bite injuries are quite rare. Although ophthalmoplegia following snake bites is not a severe neurotoxic complication, symptoms such as diplopia, dizziness, and ocular discomfort can be emotionally devastating for patients. We presented a case with bilateral diplopia and ptosis after snakebite and was recovered with the anticholinesterase therapy and antivenom.

Case report: Blurred vision developed at the 10th hour of the follow-up of a 48-year-old man who had complaints of nausea, vomiting, fatigue, diarrhea, as well as swelling, redness, and pain on the lateral malleolus of the right foot ankle after a snakebite. His physical examination revealed binocular diplopia and bilateral ptosis.

Conclusion: Ophthalmoplegia (diplopia and ptosis) is a rare symptom of snakebites. It may induce anxiety and ocular discomfort for an extended period. Treatment of anticholinesterase combined with antivenom may facilitate the recovery from ophthalmoplegia.

Keywords: neuromuscular paralysis, snakebite, ophthalmoplegia, anticholinesterase

Introduction

A snakebite often results in puncture wounds inflicted by the snake's fangs and sometimes resulting in envenomation. Cases related to snakebite are common in emergency departments, especially in the summer months. Venom may cause a complex condition: local damage, neuromuscular dysfunction, or systemic vascular damage leading to hemolysis.

Although the World Health Organization classified snakebite as a tropical disease, envenoming can also become a severe public health problem outside of tropical regions. In particular, neurotoxicity develops from poisoning with pit vipers such as rattlesnake. Neurotoxicity related to snakebites is the leading cause of mortality and morbidity, and it develops mainly due to the neuromuscular paralysis[1]. Neuromuscular paralysis secondary to the snakebite can be explained by the inhibition of synthesis, packaging, transport, and release of the neurotransmitter acetylcholine in the presynaptic field, blockade of postsynaptic nicotinic acetylcholine receptors, acetylcholine esterase inhibition, muscarinic effects of some snake toxins, and inhibition of voltage-gated calcium gates, changes in the neuromuscular junction. Apart from these, neurological manifestations can also result from non-neurotoxic effects of envenoming, such as cerebral hemorrhage and infarction due to coagulopathy and myotoxicity[1].

In patients with newly developed diplopia, 3rd and 6th cranial nerve pathologies, orbital trauma, especially neuromuscular junction toxins, should be considered. For differential diagnosis, botulism, tick paralysis, snake venom, organophosphate and carbamate toxicity, hypermagnesemia/hypocalcemia, and some medications such as lithium, magnesium sulfate, phenytoin, and other anticonvulsants should be evaluated[2-3]. Ocular manifestations in snake bite injuries are quite rare. Although ophthalmoplegia following snake bites is not a severe neurotoxic complication, symptoms such as diplopia, dizziness, and ocular discomfort can be emotionally devastating for patients[2].

We presented a case with bilateral diplopia and ptosis after snakebite and was recovered with the anticholinesterase therapy and antivenom. In the Black Sea region of Turkey, poisonous snake envenomation is very rare. However, Turkey's cases, resulting in neuromuscular paralysis after snakebite, are rarely seen in the literature.

Case Report

A 48-year-old man was admitted to the emergency department with complaints of nausea, vomiting, fatigue, diarrhea, swelling, redness, and pain on the right foot ankle after a snakebite. He was a farmer, and he had no comorbid disease and medication. He was not a cigarette smoker and alcohol drinker. He declared that he did not use any illicit drugs.

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The patient was awake and alert and was non-toxic-appearing. His temperature was 37.1◦C, his blood pressure was 105/82 mmHg, his heart rate was 92 bpm, and his respiratory rate was 12 /min. The initial physical examination was regular without swelling and redness on the right foot ankle (Figure 1). All systemic examinations were regular. His serum electrolytes and cardiac enzymes were normal. Prothrombin time and activated partial thromboplastin time were at normal range, but white blood cell count (WBC) was elevated (14,61 thousand/uL), creatine phosphokinase (CPK; 765 U/L) and the international normalized ratio (INR; 1.33) were minimally elevated. ECG showed a normal sinusoidal rhythm. Sultamicillin 2 gr per day and %0.9 NaCl solution were administrated intravenously. He was hospitalized to the emergency observation unit for follow-up.

At the tenth hour of the follow-up, blurred vision developed. In the second physical examination, binocular diplopia and bilateral ptosis were revealed (Figure 2). The examination of eye movements showed a presence of partial bilateral paresis of third cranial nerves. Cranial CT and diffusion MR imaging had no pathology. He was consulted with a neurologist and an ophthalmologist.

The second dose (10 ml polyvalent snake serum) of antivenom was administered intravenously. The visual field was found in the normal range by an ophthalmologist. After the neurologist's consultation, pyridostigmine 60 mg tablet (3 tabs per day) was added to his treatment. The symptoms were improved, and the eye movements were regular at the 12th hour of the treatment (Figure 3). WBC, INR, and CPK were at a normal range. The patient was discharged from our emergency department with full recovery on the third day after the admission. Informed consent was obtained from the patient for the publication of his information and images.

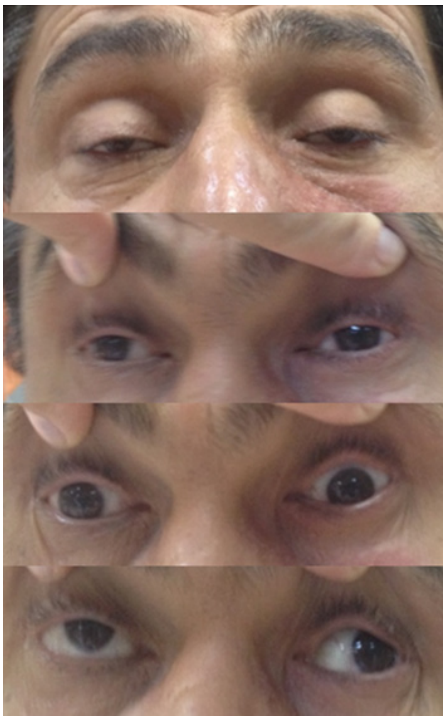

Figure 2: Diplopia and ptosis

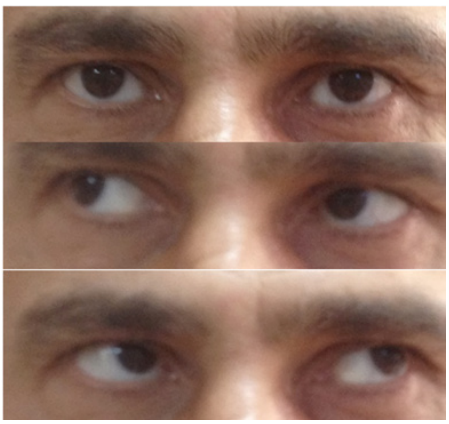

Figure 3: Improved eye findings after the treatment

Discussion

Localized and systemic symptoms may be seen after snakebites such as focal edema, pain, focal necrosis, muscle degeneration, disseminated intravascular coagulation or hemolysis, and acute renal failure[2]. Localized symptoms on his foot ankle due to snakebite were seen in our patient, but no necrosis. Neuromuscular paralysis or oculomotor nerve paralysis associated with snakebites was considered in our patient because of aberrant history and physical examination findings that exclude other causes affecting the neuromuscular junction. The normal results of our patient's cranial imaging supported the snake envenomation instead of structural brain pathologies.

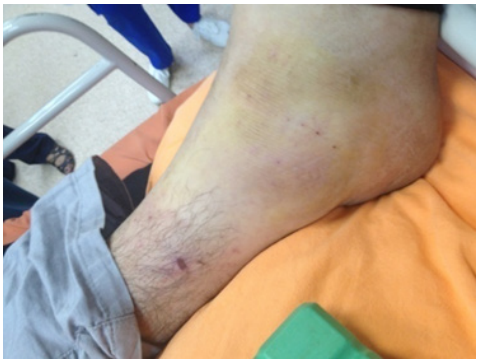

Figure 1: Swelling and redness on lateral malleolus of the right foot after a snakebite

According to the literature, a few snake species such as Europa viper (Vipera apsis), North America Viper (Agkistrodon blomhoffi), and some other vipers may lead primarily to extraocular muscle paralysis. In some reports, the most common cause of eye symptoms following a snake bite is Agkistrodon blomhoffi. The most common eye symptom is ophthalmoplegia, and the medial rectus muscle is commonly involved[4]. In our case, the patient had bilateral ptosis and paresis of the bilateral medial rectus muscle, but more

prominent in the left medial rectus muscle. The species of the snake which bit our patient was not known.

Snake neurotoxins bind to the neuromuscular junction both pre-and post-synaptically causing muscle weakness. Alpha-bungarotoxin of krait binds to acetylcholine receptors, and inhibits acetylcholine receptor sites in the postsynaptic membrane. Alpha-cobra toxin, which has a similar action, produces features of myasthenia gravis in the experimental animals. Phospholipase A2 enzyme and Beta-bungarotoxin act pre-synaptically to cause neurotoxicity[3]. Neurotoxic paralysis may also begin within the first hour of snake bites and is seen first as ptosis, then blurred vision and diplopia, followed by facial weakness and dysarthria. In severe cases, the limbs' weakness, paralysis of respiration, and fixed and dilated pupils may be observed[4]. Our patient experienced blurred vision and diplopia in the late period.

The primary treatment of neurotoxic paralysis following snakebites is an injection of antivenom. Indications for antivenom are hemostatic disturbance, cardiovascular abnormalities, neurotoxicity, elevated CPK and aminotransferases with a definite local envenomation sign, and others. Because the binding to the presynaptic portion is irreversible, clinical recovery occurs slowly and only with the formation of a new neuromuscular junction. However, the toxin binding to the postsynaptic part may act post-synaptically to produce a competitive or noncompetitive acetylcholine receptor blockade[4]. Although an antivenom may induce a certain degree of reversal of the paralysis by postsynaptic neurotoxin, the clinical recovery may be prolonged. He was brought to our emergency department from another hospital due to hypotension, unresponsive to iv fluid treatment (80/60 mmHg) after the intravenous administration of snake antivenom. In the previous hospital, the ED physician has given a polyvalent antivenom because it was challenging to identify the snake species that had bitten the patient at the treatment time[4-6].

Anticholinesterase is potentially valuable for patients with suspected myasthenia gravis[6]. Sung and Hah[7] reported a case of extraocular muscle paresis following a snakebite. It was reported that a patient, supported with a mechanical ventilator due to the neuromuscular paralysis, which was developed about 5 hours after snakebite, was resuscitated successfully using intravenous neostigmine and oral pyridostigmine[8]. In our patient, symptoms developed at the tenth hours, and after treatment, the symptoms improved fastly similar to the other cases in the literature. The patient was recovered fully on the third day of the admission. Although the absence of signs of cholinergic crisis such as muscle fasciculation and presence of improvement at the physical examination, the use of anticholinesterase may be effective for patients with neurotoxicity following snakebites.

Conclusion

Ophthalmoplegia that may induce anxiety and ocular discomfort for an extended period is a rare symptom of snakebites. Combined treatment of anticholinesterase with antivenom may facilitate the recovery from ophthalmoplegia.

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Conflict of interest: The authors have no conflict of interest.

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