Case Reports in Neurology

Case Rep Neurol 2022;14:167–172
DOI: 10.1159/000523770
Received: December 27, 2021
Accepted: February 16, 2022
Published online: March 24, 2022

Single Case – General Neurology

Delayed Visual Loss in a Patient with Snake Bite: Case Report of an Unusual Neuro-Ophthalmic Presentation

Sandip Kuikel[a] Suman Rimal[a] Rajeev Ojha[b] Sanjeeta Sitaula[c] Ragesh Karn[b] Bikram Gajurel[b] Reema Rajbhandari[b] Niraj Gautam[b] Sunanda Paudel[b] Aashish Shrestha[b]

[a]Department of Internal Medicine, Tribhuvan University Institute of Medicine, Kathmandu, Nepal; [b]Department of Neurology, Tribhuvan University Institute of Medicine, Kathmandu, Nepal; [c]Department of Ophthalmology, Tribhuvan University Institute of Medicine, Kathmandu, Nepal

Keywords
Snake bite · Optic neuritis · Anti-snake venom · Visual loss

Abstract
Neurotoxin-related optic neuritis (ON) after snake bite is uncommon. Here, we present a case of a 70-year-old female who developed bilateral painless loss of vision after she received treatment with anti-snake venom (ASV). She had only perception of light on assessment of visual acuity on admission which then improved drastically after administration of intravenous methylprednisolone (MP) after making the provisional diagnosis of ON on the basis of history and clinical findings of the patient. Imaging and visual-evoked potential could not be done initially, and they were done after the administration of intravenous MP which had normal findings. ASV, though being a lifesaving treatment, has been sometimes associated with ON.

Introduction

Snake bite is a common public health problem in many tropical countries. According to the WHO, annually 4.5–5.4 million people get bitten by snakes, and the reported death after snake bite can range from 81,000 to 138,000 [1]. In Nepal, the reported annual incidence of

Correspondence to:
Rajeev Ojha, rajeevnet@hotmail.com

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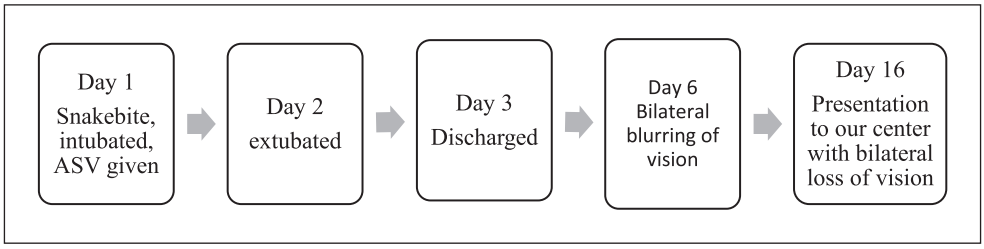

Fig. 1. Timeline of the onset of the symptoms.

snake bite is 1,162/100,000, and the annual incidence of envenomation is 604/100,000 [2]. After a snake bite, the clinical manifestation can range from mild local inflammation to severe systemic manifestation which can be hematotoxic, cytotoxic, or neurotoxic [3]. Neurotoxins related to optic neuritis (ON) after snake bite has been a rarely reported condition [4]. Here, we present a case of a 70-year-old female patient who developed bilateral painless sudden loss of vision following treatment for snake bite.

Case Presentation

A 70-year-old female developed a sudden onset of slurring of speech and dryness of mouth after snake bite. During presentation in local hospital after 2 h of the event, the patient was unconscious and needed intubation. Anti-snake venom (ASV) was given immediately according to the local protocol. She regained her consciousness the next day and was extubated. There was no limb weakness, blurring of vision, diplopia, or other neurological symptoms. The patient did not recall any specific character of the snake; however, the geographic region suggested the snake was most likely to be a cobra snake. She was discharged after 3 days of admission; slurring of speech completely improved during discharge. After 16 days of the episode, the patient presented to our center with a complaint of blurring of vision for 10 days followed by bilateral loss of vision. The detailed timeline of events is shown in Figure 1. Loss of vision was painless and progressive. The vision loss was diffuse, and no postural variation was present. There was no redness of the eye, photophobia, increased lacrimation, or pain in eye movement. There was no headache, vomiting, limb weakness, slurring of speech, nasal intonation of voice, or nasal regurgitation. She did not complain of any tingling sensation, numbness, paresthesia, abnormal body movement, and weakness of the limbs. She was a diabetic under regular oral antidiabetic drugs for 16 years. She was also diagnosed with hypertension 10 years back for which she has been under regular antihypertensive medication. There is no history of cigarette smoking or consumption of alcohol.

On examination, her vitals were stable with the pulse rate of 72 beats/min, 120/80 mm of Hg blood pressure, respiratory rate of 20 breaths/min, and temperature of 98°F. Visual acuity was assessed using the Snellen Chart, but as the patient could not read the largest letter, could not count finger or see hand movement, and only see the light shined in bilateral eyes, visual acuity of «perception of light» in both eyes was recorded. The cup to disc ratio was 0.1:1 in the right eye and 0.2:1 in the left eye, and the disc was hyperemic. On neurological examination, higher mental function was intact with normal cranial nerves except CN II where visual acuity was limited to perception of light. Pupils were regular and round while both direct and consensual light reflex were sluggish. Examination of the anterior and posterior segments, ocular motility, and intraocular pressures were normal. Motor and sensory system examinations were normal. There were no signs of cerebellar dysfunction and meningeal irritation.

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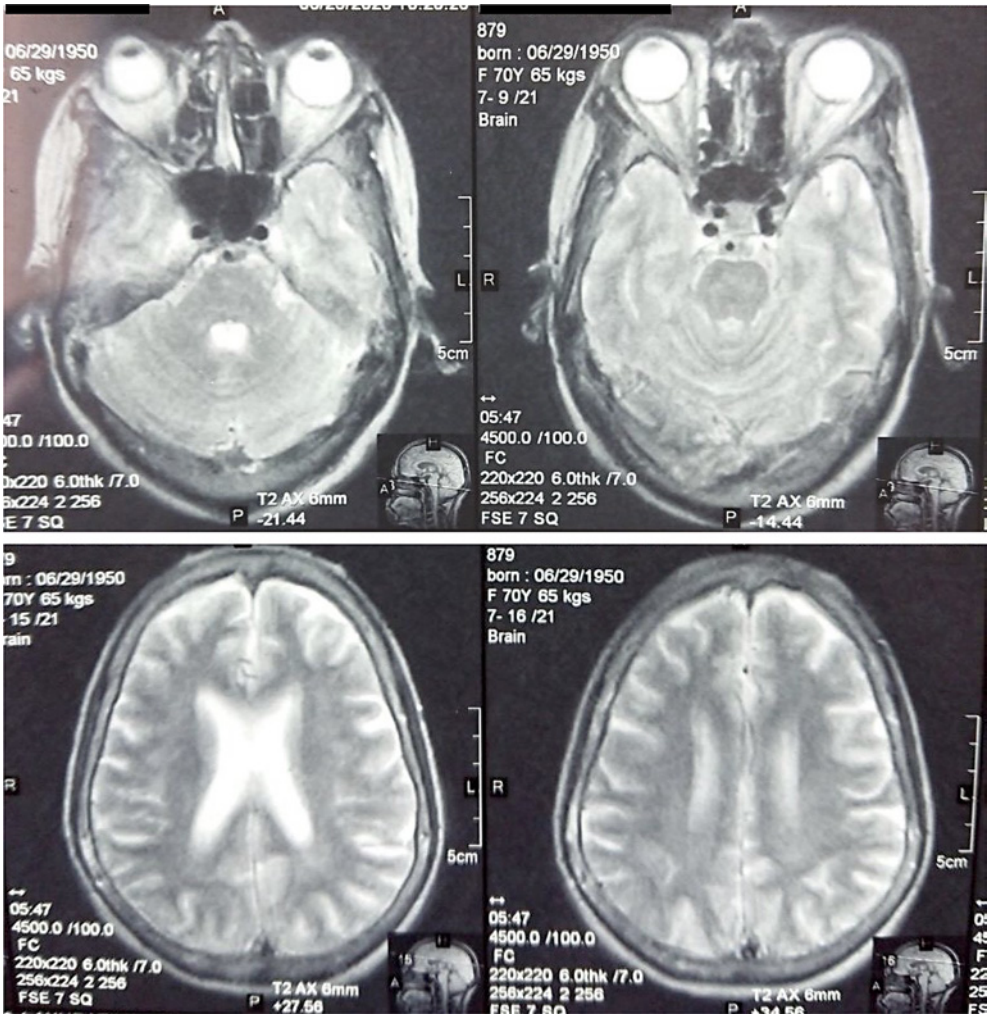

Fig. 2. MRI of the brain in transverse section with normal findings on the fifth day of intravenous MP administration.

Routine blood investigations such as complete blood count and renal and hepatic function tests were normal. CSF examination for total protein, albumin, IgG, IgA, IgM, glucose, lactate, cell count, microbiological/virological analysis, and the oligoclonal band was normal. With this history and examination findings, a provisional diagnosis of ON was made. and visual-evoked potential (VEP) and MRI of the brain were planned but were not done due to patient uncooperativeness. On the basis of our provisional diagnosis, 1 g intravenous methylprednisolone (MP) was given for 5 days along with oral thiamine and methyl cyanocobalamin supplements. Chest X-ray was performed before administrating the steroid to rule out pulmonary tuberculosis, and it was normal. On the 2nd day of treatment, the patient was able to perceive hand movements. The patient's visual symptoms gradually improved to bilateral visual acuity of 6/60 on the fifth day of admission. Pinhole correction further did not show any visual acuity correction. There was abnormal color vision assessed by the Ishihara chart. MRI of the brain was also done which showed no abnormal findings (shown in Fig. 2). Intraocular pressure, VEP, and visual perimetry test were normal. Examination of the fundus showed a bilateral normal optic disc (shown in Fig. 3). The patient was given oral steroids at an initial dose of 1 mg/kg and tapering over 4 weeks after completion of intravenous MP. On

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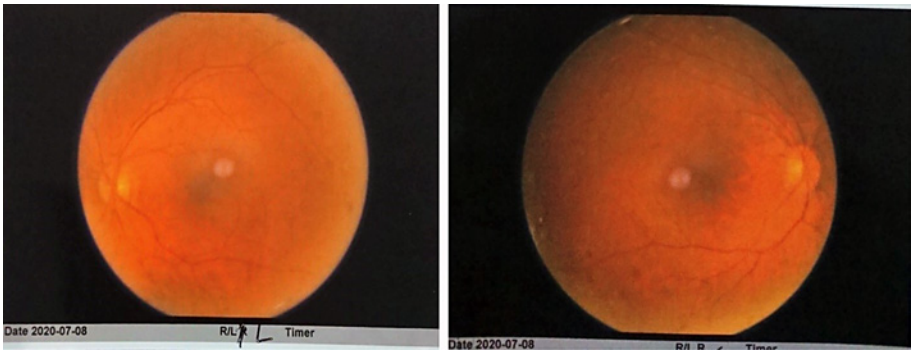

Fig. 3. Bilateral normal optic disc appearances on the fifth day of intravenous MP administration.

the ninth day, the patient was discharged with improved visual acuity to 6/36 on left and 6/12 on right. On 2 months' follow-up, the patient's visual acuity improved to 6/9 on the left side and 6/6 on the right. Blood sugar monitoring was done throughout the course of treatment, and it did not rise to the level that warranted possible discontinuation of steroids or other treatments.

Discussion

Many ocular complications caused after injection of the snake venom are described in the literature, but there is little to no evidence that a direct toxic effect of snake venom causes ON [5]. Based on the reported cases of ON after snake bite, ASV is one of the causes of ON in patients after snake bite [4, 6]. These cases presented after 5–6 days following ASV administration. Snake venom allergy, retinal hemorrhages, or capillary damage are some of the possible causes of ON after administration of ASV [7].

The clinical features of ON are periocular pain associated with eye movement, unilateral or bilateral loss of visual acuity, photophobia, ipsilateral relative afferent pupillary defect (RAPD), visual field defect, decrease in color vision, pallor of the optic disc, the presence of phosphenes, and reduction in vision in bright light. For diagnosis and establishing the cause of ON, a detailed ophthalmological and neurological examination is necessary. Ophthalmological examinations include slit-lamp examination, RAPD test, aided visual acuity, color vision testing, contrast sensitivity, visual field determination, examination along with orbital, and brain MRI should be performed. Other tests include CSF analysis for the determination of total protein, albumin, IgG, IgG index, IgA, IgM, glucose, lactate, cell count, microbiological/virological analysis, and oligoclonal bands. To rule out infective and inflammatory causes, blood culture and serological tests should be done. In patients suspected to have tuberculosis, chest X-ray and Mantoux test should be done before starting steroid treatment [8].

Here, in our case, the patient developed a sudden onset painless visual loss 6 days after administration of ASV, which matches the cases of ON reported beforehand. We suspected ON in this case based on the normal neurological examination except for sluggish light reflex, normal CSF, and MRI brain study. The absence of RAPD is due to the involvement of the bilateral optic nerve. VEPs test could not be done due to the uncooperativeness of the patient. Based on the clinical picture, ON was suspected, and the patient was treated on the line of it. Other possible differential diagnoses of these cases are posterior reversible encephalopathy syndrome, vascular causes, and vasculitis due to the snake toxin. Normal fundoscopic findings rules out the possibilities of vascular events like central retinal artery occlusion and central

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retinal vein obstruction. Also, the rapid response to intravenous steroids supports our diagnosis. The clinical presentation and rapid improvement of visual acuity after administration of intravenous steroids support the diagnosis of ON than posterior reversible encephalopathy syndrome. This case of ON improved in visual acuity drastically from perception of light to hand movement after the first dose of iv MP 1 g and visual acuity to 3/36 in bilateral eyes after 5 days of treatment. This further supported the diagnosis of our patient to be ON. We suppose the retrobulbar ON in our patients could be either due to of ASV or could be a delayed inflammatory response after snake bite.

In a similar case report from India, a 24-year-old female developed ON 6 days after ASV administration and improved on administration of steroids [4]. Similarly, another Indian case report described the development of bilateral retrobulbar neuritis on a 50-year-old male 2 weeks following a black-cobra bite. He was treated with intravenous dexamethasone for 3 days which gradually improved his vision to visual acuity of 6/18 in both eyes at 8 weeks [6].

Further studies and evaluations are needed to identify the exact factor responsible for the neuro-ophthalmologic presentation in patients with snake bite who were treated by ASV. Limitations of our study include the inability to do VEP and a brain MRI of the patient at the time of presentation and treating the patient on the basis of clinical grounds.

Conclusion

ASV has been a lifesaving treatment in the management of poisonous snake bites, but there are many complications associated with this. In our presented patient, none of the reported or well-known imaging and ophthalmological findings of ON were evident but with her history and response to intravenous MP consistent with the ON. ON has been reported a few times after administration of ASV and opens a potential site for research for the cause.

Statement of Ethics

Ethical approval was not required for this study in accordance with local/national guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

No funding was received for the preparation and publication of this manuscript.

Author Contributions

Rajeev Ojha conceptualized the study, reviewed, and edited the manuscript and was in charge of the case; Suman Rimal and Sandip Kuikel wrote the original draft and were in charge of the case; Sanjeeta Sitaula, Bikram Gajurel, Ragesh Karn, Reema Rajbhandari,

Sunanda Paudel, Niraj Gautam, and Aashish Shrestha were in charge of the case and reviewed the manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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