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CASE REPORT

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Atypical leukoencephalopathy due to a snake bite in a pregnant patient: MRI findings

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Abstract

Snake envenomation significantly contributes to morbidity and mortality. Although various clinical presentations and complications are observed with different snake bites, the incidence of leukoencephalopathy is rare. Most cases of leukoencephalopathy occur following viper bites. In this article, we present a rare case of multiple infarctions and leukoencephalopathy involving the corona radiata and centrum semiovale following a neurotoxic snake bite. We emphasize the importance of considering this complication, albeit rare, in cases of snake envenomation.

Keywords: Leukoencephalopathy, snake bite, magnetic resonance imaging

Introduction

Snake bites remain a significant cause of morbidity and mortality worldwide. In Türkiye, approximately fifty species of snakes, both venomous and non-venomous, are known to exist [1]. Bites can result in local tissue necrosis, coagulopathy, rhabdomyolysis, hypotension, or acute kidney injury. Patients bitten by snakes should be moved away from the area and kept calm. All cases should be observed in a hospital setting for at least 6 to 8 hours [2]. Airway, breathing, and circulation support should be ensured, with close monitoring for respiratory depression, heart failure, and shock. Cardiac and respiratory parameters must be monitored and closely followed [3]. Tests including complete blood count, coagulation tests, urinalysis, serum creatine kinase, electrolytes, phosphate, uric acid, blood urea nitrogen, creatinine, electrocardiogram, and blood gas analyses should be conducted and repeated for abnormal values at certain intervals [4]. Timely administration of an adequate dose of antivenom can prevent adverse outcomes and complications. Patients with moderate to severe envenomation symptoms should receive supportive therapy and, if necessary, be closely monitored in an intensive care unit [5]. Neurological deficits following snake bites should also be considered, and a complete neurological examination should be performed. Common neurological deficits include intracerebral or subarachnoid hemorrhage, ischemic infarctions, brainstem infarctions, and acute disseminated encephalomyelitis [6]. Leukoencephalopathic changes following a snake bite are extremely rare [7]. In this article, we present a rare case of a pregnant woman who developed leukoencephalopathic changes involving the lentiform nuclei and juxtacortical regions, along with multiple thromboembolic infarctions, following a snake bite.

Case Presentation

A 34-year-old woman, with a history of hypothyroidism and 26 weeks pregnant, was bitten by a snake in front of her house during the daytime. The patient was immediately brought to

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the emergency department. Physical examination revealed fang marks on her left foot. At admission, the patient was conscious and in good general condition. A mild edema was observed in the left lower extremity. Vital signs included a heart rate of 86 beats/min, blood pressure of 120/74 mmHg in the right arm, and a respiratory rate of 20 breaths/min. Neurological examination and deep tendon reflexes were normal. Adequate urine output was observed.

Initial laboratory tests revealed hemoglobin levels of 11.3 g/dL, a total leukocyte count of 10,078/mm³, and a platelet count of 129,000/µL. Blood glucose was 79 mg/dL. Routine urinalysis was normal. Liver function, renal function, and coagulation test values were within the normal reference range. The patient was admitted to the intensive care unit for monitoring. Obstetric evaluation confirmed fetal health and normal fetal heart rate. On the third day of hospitalization, the patient's general condition and vital signs deteriorated. Acute kidney injury, altered consciousness, and deranged coagulation parameters were observed. Since the patient was pregnant, the obstetrics clinic was consulted for fetal evaluation. Obstetric consultation revealed intrauterine fetal demise. Due to altered consciousness, the patient underwent cranial magnetic resonance imaging (MRI), including conventional, diffusion-weighted, and susceptibility-weighted imaging (SWI). MRI findings included leukoencephalopathic edema extending from the bilateral peripheral cerebellar regions to the posterior parieto-occipital juxtacortical areas, appearing more prominent on fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted sequences. The patient also had a left cerebellar lesion. (Figure 1A-1C). Comparison with SWI sequences revealed edema and susceptibility artifacts, more pronounced on the left juxtacortical regions (Figure 2). Bilateral lentiform nuclei showed infarct-like involvement, especially in the putamen (Figure 3A,3B). Multiple parasagittal thromboembolic infarcts with diffusion restriction were noted in the bilateral frontal lobes. These findings are also seen in the apparent diffusion coefficient (ADC) map image. (Figure 4A,4B). An emergency cesarean section was performed due to intrauterine fetal demise. The patient was followed up intubated and it was decided to perform plasmapheresis. After apheresis for three days, the patient's vital parameters improved, and she was extubated. The patient was discharged on the 10th day of hospitalization without any sequelae.

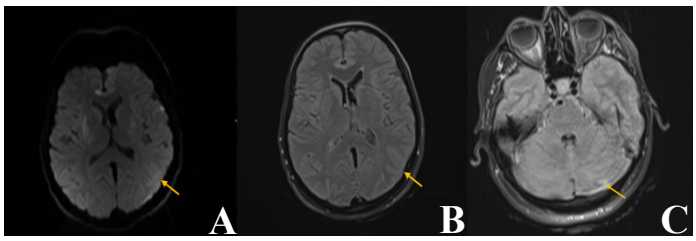

Figure 1. Leukoencephalopathic oedema seen on FLAIR (**1B**) and diffusion (**1A**) weighted sequences extending from bilateral peripheral cerebellar regions to posterior parieto-occipital juxtacortical areas. Left cerebellar lesion (**1C**)

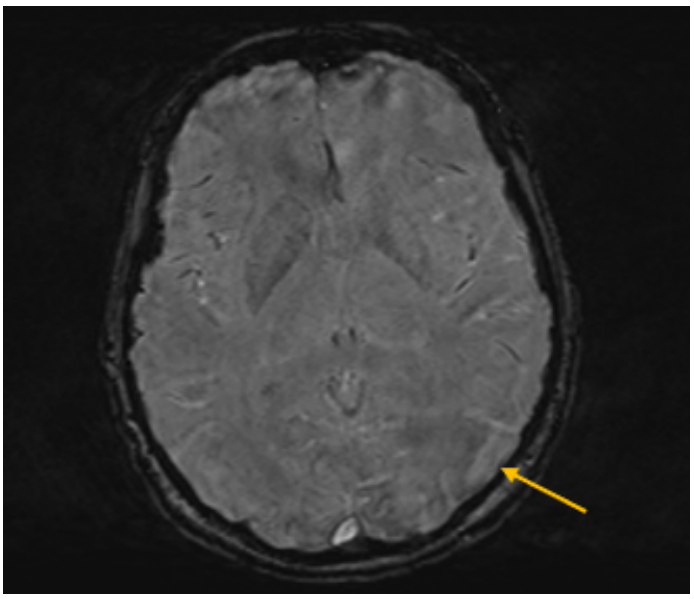

Figure 2. Edema and sensitivity artefacts, more prominent in the left juxtacortical regions

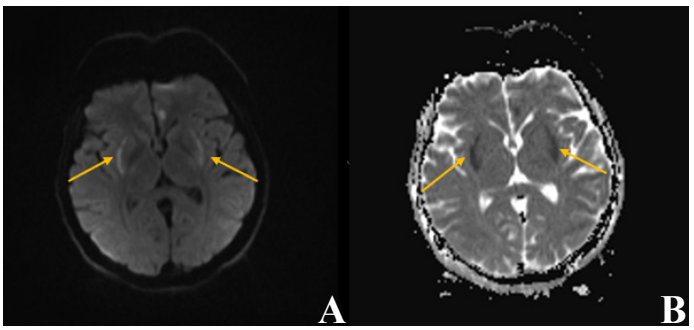

Figure 3. In diffusion weighted (**3A**) and ADC (**3B**) images, putamen involvement is present especially in bilateral lentiform nuclei

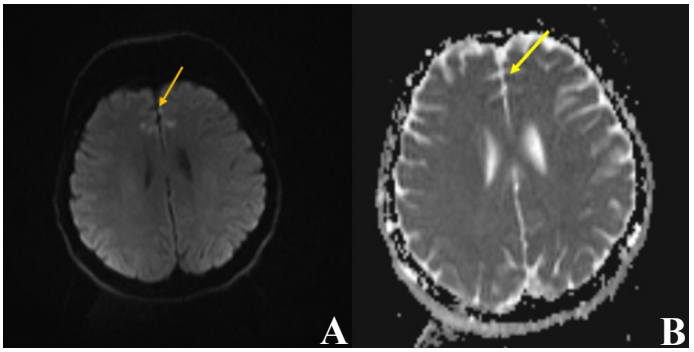

Figure 4. Multiple parasagittal thromboembolic infarcts with diffusion restriction were observed in bilateral frontal lob (**4A**) and ADC images (**4B**)

Discussion

Neurological signs and symptoms following venomous snake bites are often linked to the toxic effects of venom, including anticoagulant/procoagulant activity or neurotoxicity [8]. Factors involved in etiopathogenesis include hypercoagulability, immune-mediated vasculitis, endothelial damage, and the presence of venom toxins leading to systemic hypotension

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[1,9]. The most common central nervous system complication is intracranial hemorrhage. Some cases may develop neurological complications secondary to cerebral hypoxia. The effect of neurotoxins usually begins within minutes to several hours after the venom is introduced into the body. Findings are often in the form of muscle weakness [9,10]. Less frequently, neurological manifestations include ptosis, ophthalmoplegia, limb weakness, respiratory failure, palate weakness, and neck muscle weakness [3,11]. Altered consciousness occurs in approximately 70% of cases. Our patient had no symptoms in the first three days.

Symptoms of the condition may appear immediately following the snake bite or in the early period after antivenom treatment (10-180 minutes). These symptoms are referred to as early reactions and can range from urticaria to anaphylactic shock. Late reactions are immune-related and typically occur 5–24 days post-antivenom administration, manifesting as serum sickness syndrome [12]. Both central and peripheral nervous system symptoms can coexist in these patients. Cerebrovascular complications reported include ischemic stroke, hemorrhagic stroke involving multiple lobar hemorrhages with or without ventricular extension, subarachnoid and subdural hemorrhages, cerebellar hemorrhage, epidural hematoma, and disseminated encephalomyelitis [13,14]. Infarctions are presumed to result from venom-induced prothrombotic effects and endothelial damage. Over 60% of snake bite cases with brain infarction support this hypothesis [13]. Consistent with this, in our case, diffusion restrictions indicative of multiple thromboembolic infarctions were observed in the corona radiata and centrum semiovale.

In previous studies, the symptoms in snake bites occur within minutes to hours following the bite, whereas in our case, clear symptoms appeared after the third day. Again, when the literature is reviewed, it is observed that in radiologic imaging of such cases, either leukoencephalopathic formations or cerebrovascular findings are predominant, whereas in our case, both pathologies are reflected on MRI images simultaneously [13]. While infarct areas are observed, leukoencephalopathic changes in the basal ganglia and peripheral juxtacortical areas are also observed simultaneously.

Conclusion

Leukoencephalopathy due to snake bites is a rare neurological complication, particularly following neurotoxic snake envenomation. Therefore, this clinical condition should be promptly considered in snake bite cases presenting with features suggestive of impaired higher mental functions. High suspicion and timely diagnosis with MRI imaging help in the management of the disease and rapid recovery of the patient.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Patient Informed Consent
Informed consent was obtained from the patient.

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