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ACUTE DEMYELINATING ENCEPHALOMYELITIS FOLLOWING VIPER BITE

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ABSTRACT The most serious complications of the central nervous system that occur after venomous snake bite are intracranial hemorrhage and ischemic stroke. We present a rarely seen central nervous system complication, acute demyelinating encephalomyelitis, after a treated viper bite.

On 10th July 2017, a 45-year-old Female farmer was bitten on his right leg by a viper. The bite rendered the victim unconscious for 6 days, during which he was treated with tetanus toxoid and polyvalent antisnake venom. Acute demyelinating encephalomyelitis (ADEM) was suspected after magnetic resonance imaging of the brain. After a high dose of methylprednisolone was used as diagnostic treatment, the patient started recovering fast. ADEM is a rare complication after snake bite, and is triggered by venom or antivenin. Magnetic resonance imaging helps in the early diagnosis of ADEM, and high-dose corticosteroid therapy appears to be effective in ADEM after viper bite or antivenin management.

Abbreviations: ADEM = acute disseminated encephalomyelitis, CNS = central nervous system, CSF = cerebral spinal fluid, MRI = magnetic resonance imaging.

KEYWORDS : antivenin, demyelinating encephalopathy, neuropathy, viper bite

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is an autoimmune-mediated nervous system disease, which occurs mainly following animal or insect bites. However, ADEM after a snake bite has rarely been reported. [1,2] Here, we present a rarely seen central nervous system (CNS) complication, acute demyelinating encephalomyelitis, after a treated viper bite. The patient recovered after a high dose of methylprednisolone therapy. We discuss the role of magnetic resonance imaging (MRI) and methylprednisolone in the diagnosis and treatment of this rare complication.

Case report

A previously healthy 45 -year-old female presented to our emergency department after being bitten by a viper on 10th July, 2017. He described severe pain and significant swelling on his right leg. One hour later, he was transported to a local hospital and given broad-spectrum antibiotics (amoxycillin sodium and clavulanate potassium 1.2 g intravenously), administered 3 times daily. She was also administered tetanus toxoid, and polyvalent antivenom therapy immediately. She received polyvalent antisnake venom with a dose of ten vials (100 units) stat. Five hours after the treatment,

the patient was unconsciousness and showed no response to external stimulation. Emergency endotracheal intubation was performed to maintain an open airway and mechanical ventilation. Meanwhile, the patient was treated with anti-infective and other supportive treatment.

The patient's medical history was unremarkable. On examination, she was febrile with a temperature of 38.5°C, with normal blood pressure and pulse. There was no skin rash and no lymphadenopathy, but she had mild residual swelling at the bite site. The Glasgow Coma Scale score was 6.In Laboratory findings: blood culture was negative. The patient's blood glucose was normal. Complete hemogram was normal .Renal function: serum creatinine was 211 umol/L, blood urea nitrogen 33.7mmol/L. Liver function: alanine aminotra nsferase 105 U/L, aspartate aminotransferase 47 U/L. His erythrocyte sedimentation rate was 98mm/h and his C-reactive protein was 45mg/L. Cytology and tuberculosis examination of the cerebral spinal fluid (CSF) were normal. Coagulation profile was normal. MRI of the brain was used to confirm whether CNS complications had resulted in long-term unconsciousness. The examination demonstrated T2/Flair hyperintensity involving B/L cerebeller hemisphere and cortical,sub cortical and juxta –cortical in B/L frontal,parietal ,occipital lobe and caudate nucleaus.On SWI micro haemorrhages are seen in B/L Cerebellers hemispheres .On post contrast study, diffuse gyriform enhancement is noted in B/L Cerebral hemisphere ,B/L basal ganglia ,B/L Cerebeller hemisphere suggestive of acute demyelinating encephalomyelitis.Initially, methylprednisolone (1000mg/d) was used as a diagnostic treatment. The patient's body temperature returned to normal within 3 days. Pulse therapy was given for 5 days.Then, prednisone (60mg/d) was administered for 10 days. A physical examination showed that the patient's Glasgow Coma Scale score was 10. Meanwhile, the patient was given antibiotics, a blood transfusion, protection of liver and kidney function, supportive nutrition, and right leg debridement and dressing to ameliorate his health.

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The final diagnosis was demyelinating encephalopathy. The patient recovered fairly well and was discharged from our hospital. He was advised to take prednisolone (50mg/d) orally and reduce the doses gradually (minus 2 times a week, each 5mg). Follow-up 1 month later showed that the lesions revealed by the MRI had almost disappeared.

DISCUSSION

Snake bite is a neglected public health issue that affects millions of people worldwide each year. Depending on the species of the offending snake, the clinical manifestations may be different, but include swelling and necrosis in the bitten limb or neurological symptoms. [1] The most common serious CNS complications following venomous snake bite are intracranial hemorrhage and ischemic stroke. Some patients also experience CNS complications such as delayed cerebellar ataxia or ADEM after a snake bite. These manifestations may relate to the immune-mediated damage triggered by venom or antivenom. ADEM is an autoimmune demyelinating disease of the nervous system, which often follows viral or bacterial infections, or less often, vaccination for measles, mumps, or rubella. [3] It has rarely been reported in the literature following animal or insect bites, and there is only 1 recorded case that was caused by snake bite. ADEM can be suspected based on clinical features, laboratory features, and magnetic resonance imaging, which is the

imaging modality of choice to demonstrate white matter lesion. Among the treatment methods used for ADEM are supportive care, high- dose methylprednisolone, immunomodulation, plasmapheresis, and physical and rehabilitation therapy. More than half of ADEM patients can achieve full recovery after therapy. In the case of our patient, MRI revealed T2/Flair hyperintensity involving B/L cerebeller hemisphere and cortical,sub cortical and juxta –cortical in B/L frontal,parietal ,occipital lobe and caudate nucleaus.On SWI micro haemorrhages are seen in B/L Cerebellers hemispheres .On post contrast study, diffuse gyriform enhancement is noted in B/L Cerebral hemisphere ,B/L basal ganglia ,B/L Cerebeller hemisphere suggestive of acute demyelinating encephalomyelitis

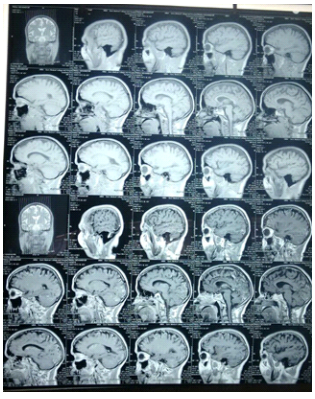

Figure 1a

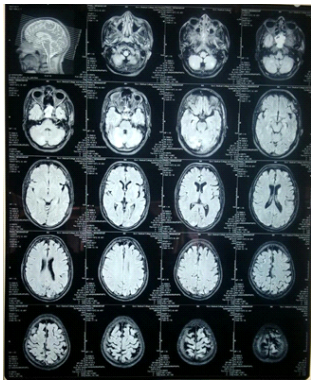

Figure 1b

Figure 1 a & b. Axial T1-weighted MR image showing decreased signal intensity (A, B). T2-weighted MR image showing increased signal intensity in the lesions. MR = magnetic resonance

Since the patient did not have any history of CNS disorders, we considered the findings to be more likely due to inflammatory and autoimmune origins than tumor. However, cytology and a tuberculosis examination of the CSF did not show evidence of infectious diseases. After the exclusion of other possible causes of coma, we considered demyelinating encephalopathy. Moreover, the patient's sensitivity to glucocorticoids also supported our diagnosis. The relationship between cerebral damage and viper bite is unclear based on current information.[4–7] Some researchers have hypothesized that injury to the nerve endings or demyelination may be related to an immune reaction to the venom or antivenom. Since the patient in our case had no history of diseases involving the central nervous system, such as multiple sclerosis, the occurrence and turnover of the nervous system were concomitant with the occurrence of a viper bite. Following snake bite, some patients develop delayed neurological symptoms. The vast variety of symptoms often makes the diagnosis of ADEM difficult. It is easy to be misdiagnosed as an intracranial infection or tumor. We should pay more attention to the history and take account of the possibility of central nervous system damage caused by systemic diseases. The brain MRI and sensitivity to glucocorticoids helped us to make a provisional diagnosis of ADEM, but eventually biopsy may be needed for definitive diagnosis which could not be done in our case. (4).

CONCLUSION

ADEM is a rare complication after snake bite, and is triggered by venom or antivenom. MRI helps in the early diagnosis of ADEM, and high-dose corticosteroid therapy appears to be effective in ADEM after viper bite or antivenom management.

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