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CASE REPORT

CLINICAL, RADIOGRAPHICAND ANALYTICAL FINDINGS OF ATYPICAL RABIES ENCEPHALITIS IN A FIVE-YEAR-OLD BOY: A CASE REPORT

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ABSTRACT

Introduction: *Rabies is an almost always fatal disease that doctors and patients fear because of its bleak prognosis and limited treatment choices. In the absence of special laboratory investigations at the point-of-care facility, the diagnosis is usually clinical. We present a case of rabies encephalitis with a dog bite history and standard MRI findings that can be used for early identification in the absence of usual clinical signs and specialized diagnostic testing.*

Keywords: *Dogs. MRI, Rabies*

INTRODUCTION

Rabies is a zoonotic illness caused by an RNA virus of the Rhabdoviridae family, genus Lyssavirus. The rabies virus can infect any creature, although dogs are the most common source of human rabies. Despite the fact that the required evidence and methods are in place to control and eliminate rabies, the virus continues to spread globally, posing a substantial health and economic burden mostly on poor countries in Africa and Asia.[1]

Typically, two types of clinical symptoms of rabies are described: paralytic and furious rabies. Furious rabies is distinguished by vegetative symptoms, behavioral abnormalities, hydrophobia, aerophobia, and paresthesia, which frequently manifest on the affected limb. Paralytic rabies, on the other hand, is characterized by ascending weakness and peripherally sensitive symptoms that can lead to coma.[2]The usual time from clinical disease onset to death in furious rabies is 5-7 days and 11 days in paralytic rabies.[3]

Rabies is a vaccine-preventable disease. Modern cell culture-based and embryonated egg-based anti-rabies vaccines (ARV) have proven to be safe and effective in preventing human rabies. Pre-exposure prophylaxis (PrEP) is recommended for individuals who will be at continual, frequent or increased risk of exposure to the rabies virus.[4]

This article reports the unusual case of a child who contracted an uncommon form of rabies despite undergoing rabies post-exposure prophylaxis; the youngster had survived for a month with neurological sequalae.

CASE SUMMARY

A 5-year-old child from a hamlet in Nepal's central terai was bitten by a stray dog, resulting in a category III wound on his left shoulder. His father quickly rushed him to the local primary health care center, where the

child's wound was cleansed with running tap water. After 8 hours, the youngster received the first dose of rabies vaccination, 0.1ml, administered intradermal in the bilateral deltoid region. He also received two additional deltoid dosages on the third and seventh days. On the seventh day after the dog bite, the youngster received rabies immunoglobulin (kind and dose unknown), which was locally infiltrated around the shoulder bite site.

After the bite, the child appeared to be fine for 21 days until developing mild to moderate fever (Tmax 102F) and the occasional bout of vomiting. The child had weakness in bilateral lower limbs on day 6 of febrile illness and was steadily advancing with decreased degree of consciousness by day 7. With these complaints, the patient was admitted to a neighboring clinic for 20 hours before being sent to our facility (National Medical College and Teaching Hospital, Birgunj, Nepal) on the eighth day of the febrile illness/ 29th day after the dog bite.

The child's vitals were within normal ranges in our hospital during presentation. E4V3M4 was the Glasgow Coma Scale score. Motor assessment revealed bilateral lower limb hypertonia with a power score of 3/5 and bilateral plantar extension. Tone was normal in the upper limb, power was 4/5, and reflexes were normal. His pupils were equal and reactive on both sides. During a 5-day hospital stay, the child's weakness progressed, involving upper limbs, and his GCS plummeted to E2V2M3 at 40 hours. Child developed abnormal body movement, which was generalized tonic clonic type with up rolling of eyes at 48 hours of hospital stay. There was no hydrophobia or aerophobia. Differential diagnosis of viral encephalitis, paralytic form of rabies and GBS were considered and kept under Meropenem, Vancomycin, Acyclovir & phenytoin. The convulsions were controlled by phenytoin but the child continued to remain in semi-comatose state with GCS of 7. GCS when assessed at 3rd day of hospital stay dropped to E2V2M2 6, and the child was intubated & kept under ventilator support.

CSF obtained by lumbar puncture revealed 350 cells/L, 95% lymphocyte & 5% polymorphs, protein 137 and sugar 72% of blood sugar. A contrast enhanced Computed tomography scan of brain was done and reported as normal.

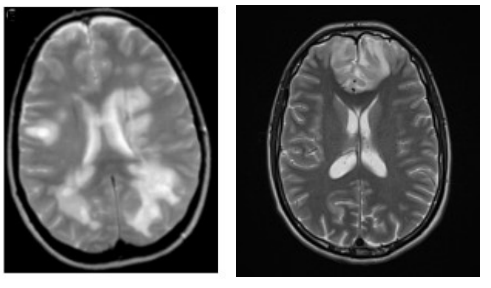

Figure 1. Images of the patient showing worsening of hyperintensities involving the bilateral basal ganglia and periventricular and deep white matter. (a) On admission, showing T2 hyperintensity in the bilateral basal ganglia, thalami, and deep and periventricular white matter. (b) At 15 days after admission

Patient was extubated on 5th day of intubation. A magnetic resonance imaging (MRI) scan of the brain done on 8th day of hospital admission was suggestive of acute disseminated encephalomyelitis (ADEM). A provisional diagnosis of post-PCEC vaccine ADEM was considered.

A standard course of intravenous methylprednisolone 30mg/kg/day was given once daily for 3 days and there was no clinical improvement so intravenous immunoglobulin therapy was given 0.4g/kg/day once daily for 5 days. MRI scan of the brain was done on day 15 after admission (Figure 1). Both MRI scans of the brain, was revised and radiological diagnosis of 'probable' rabies encephalitis was made. For the hypertonia, baclofen and diazepam were prescribed, as well as phenytoin as an anticonvulsant. After educating the parents on caring for a semi-comatose patient and giving nasogastric tube feeding, the patient was discharged after a 20-day stay in our center.

A follow-up two weeks after discharge, on day 34 of the sickness, revealed that the infant had died two days prior.

DISCUSSION

The current case had none of the classic symptoms of furious rabies, but rather those of the paralytic form of rabies, such as progressive ascending paralysis, stupor, and coma. Based on the initial MRI scan of the brain, a diagnosis of post-PCEC vaccine ADEM, a highly rare illness, was proposed.[5]

There were departures from WHO recommendations; the immunoglobulin amount was unknown, and it is best when supplied concurrently with the first or second dose of vaccine, but in this case, it was administered on the seventh day after the dog attack.[6]

The current case was diagnosed as paralytic rabies based on a history of exposure, a compatible clinical state, and imaging features, however RVNA detection in serum and CSF was not possible. According to a recent paper, just six victims survived rabies encephalitis caused by a dog attack. All six patients had received insufficient rabies post-exposure prophylaxis, and none of them had displayed the characteristic indications of furious rabies. Three of these individuals survived with serious neurological effects, while the others died, as in the current case.[7]

CONCLUSION

This paper emphasizes the necessity for quick, effective, and appropriate post-exposure management of human cases exposed to rabies, particularly in rural Nepal. Health authorities must devote greater resources to educate rural medical practitioners in prophylactic measures for this nearly always fatal disease, which is virtually entirely preventable with appropriate post-exposure prophylaxis.

CONSENT

An informed and written consent was obtained from the mother for publication.

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