

# Acute Transverse Myelitis in Post Rabies Vaccination

\*Dr.Umar Quadri, \*\*Dr. Vinit Sarode \*\*Dr.Sushant Yadav

(\*Assistant professor, \*\*Junior Resident Department of Medicine, MGM Medical College and Hospital, Aurangabad)  
sarodepvinit@gmail.com

## ABSTRACT

**Introduction:** Transverse myelitis is a clinical syndrome that involves the spinal cord, resulting in varying degrees of weakness, sensory alterations, and autonomic dysfunction. Vaccination as an etiology of ATM is frequently reported. We report a patient presenting with hyperintense cervical cord lesion in whom the involvement may be associated with rabies vaccination

**Case report:** A 60 years old female was admitted because of quadriparesis with bowel and bladder involvement. She had received a rabies vaccination 15days previous because she had been bitten by a dog.

**Findings:** Clinical and laboratory findings supported acute transverse myelitis. A hyperintense, lower cervical and upper thoracic cord lesion was detected on spinal magnetic resonance imaging.

**Conclusion:** Vaccination may have been the factor that had activated autoimmune mechanisms in this case.

**Key Words:** Rabies vaccination; Acute transverse myelitis; Methylprednisolone; MRI cervical spine, Hyperintense lesion

## INTRODUCTION

Transverse myelitis is a clinical syndrome that involves the spinal cord, resulting in varying degrees of weakness, sensory alterations, and autonomic dysfunction (1). Acute transverse myelitis (ATM) can be due to postinfectious immune-mediated inflammation of the spinal cord (2). Active or passive immunization with vaccines or sera can cause lesions

with immunomediated pathogenesis and may involve both the central and the peripheral nervous system (3). Vaccination as an etiology of ATM is frequently reported. We report a patient presenting with hyperintense cervical cord lesion in whom the involvement may be associated with rabies vaccination.

## CASE REPORT

A 60year-old female was admitted with history of neck pain and quadriparesis with bowel and bladder involvement. The complains first began with neck pain which was later followed by bilateral upper limb weakness. Her weakness progressively worsened, involving bilateral lower limb, which was later followed by bowel and bladder involvement. She had received 3 doses of rabies vaccination 15 days back after being bitten by a pet dog. Dog was healthy and still alive. Rabipur (active substance: inactivated rabies virus) was administered on the day of the bite and on the 3rd and 7th days thereafter. Patient had no associated h/o aerophobia, hydrophobia, convulsion, difficulty in deglutination, blurring of vision.

Her past medical history was non-significant.

Her General examination parameters were within normal limit. On physical examination, presence of dog bite wound near left elbow joint.

Her CNS Examination were as follows- patient was conscious, cooperative and oriented to time place and person. Muscle strength was 2/5 in bilateral upper limb and 3/5 in bilateral lower limbs. There was no sensory involvement. Deep tendon reflexes were absent in both upper and lower extremities, and plantar responses were extensor bilaterally. The remainder of the neurological examination was normal. Her rest of the systemic examination were within normal limit.

## Diagnostic Workup

Complete blood count, KFT, LFT were normal. Serum electrolytes  $\text{Na}^+$ -131 and  $\text{K}^+$ - 4.0. Marker of vasculitis anti-nuclear antibody (ANA) was negative.

The patient's cerebrospinal fluid (CSF) on physical examination was clear; microscopic examination showed 30 leucocytes/ $\text{mm}^3$  with lymphocytic predominance and no microorganisms were found. The CSF protein level was markedly elevated (1.3 gm/dL), glucose level was 35 mg/dL, and simultaneous serum glucose level was 118 mg/dL. No evidence of oligoclonal bands. Magnetic resonance imaging (MRI) whole spine was suggestive of hyperintense lesion in lower cervical and upper thoracic. Nerve Conduction Velocity (NCV) study was normal.

After hospitalization, pulse steroid therapy of methylprednisolone I.V. 1g/d for 5 days was given. After pulse methylprednisolone therapy, patient was switched to oral prednisolone at low dosage which was later tapered and stopped.

The patient underwent regular physiotherapy, and gradually her muscle strength improved completely over a period of 14 days and bilaterally in both upper and lower limb it was 5/5. Her deep tendon reflexes were present and plantar responses were flexors bilaterally. Urinary Retention was present inspite of bladder training. Patient was discharged and with plan for suprapubic catheterisation (SPC), if retention persists.



Figure 1. MRI spine suggestive of hyperintense lesion in lower cervical and upper thoracic.

## DISCUSSION

Acute transverse myelitis is an inflammatory disorder. The pathogenesis is unclear, but the probable mechanism involves an autoimmune phenomenon. Acute transverse myelitis may be an isolated entity or may occur in the context of a multifocal or even a multi-systemic disease (4). Possible causes include parainfectious and postvaccination events; ATM also can be seen in the course of multiple sclerosis (5). Das et al (6) reported the etiologies of transverse myelitis or myelopathy and found the following: 29.26% postinfectious, 19.51% demyelination, 3.65% vascular and vasculitis, 1.21% toxic, and 2.42% physical. They also reported a vaccination history in 1.21% of their patients, as in this case, in which a rabies vaccination was given 15 days prior to the onset of neurological symptoms. In this case, vaccination may be the factor that activated autoimmune mechanisms and may have caused the neurological involvement.

## CONCLUSION

Understanding the activating factors as in this case post-vaccination, may be helpful for the prevention of neurological involvement and is important for planning follow up and understanding the prognosis. Administration of rabies post-exposure prophylaxis is a medical urgency, not a medical emergency but decision must not be delayed and possible complications with it should be explained to the patient and patient relatives.

## REFERENCES

- [1] Krishnan C, Kaplin AI, Deshpande DM, Pardo CA, Kerr DA. Transverse myelitis: pathogenesis, diagnosis and treatment. *Front Biosci.* 2004;9:1483–1499.
- [2] Chan KH, Tsang KL, Fong GC, Cheung RT, Ho SL. Idiopathic severe recurrent transverse myelitis: a restricted variant of neuromyelitis optica. *Clin Neurol Neurosurg.* 2005;107:132–135.
- [3] Tezzon F, Tomelleri P, Ferrari G, Sergei A. Acute radiculomyelitis after antitetanus vaccination. *Ital J Neurol Sci.* 1994; 15:191–193.



- 
- [4] Kerr DA, Ayetey H. Immunopathogenesis of acute transverse myelitis. *Curr Opin Neurol.* 2002;15:339–347.
- [5] Iniguez C, Mauri JA, Larrode P, Lopez del Val J, Jerico I, Morales F. Acute transverse myelitis secondary to hepatitis B vaccination. *Rev Neurol.* 2000;31:430–432.
- [6] Das K, Saha SP, Das SK, et al. Profile of non-compressivemyelopathy in eastern India: a 2-year study. *Acta Neurol Scand.* 1999;99:100–105.