

Rabies – An Unusual Presentation

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Abstract: Every year more than 20,000 peoples die in India due to Rabies. It is an acute infection involving the CNS in human and other mammals caused by an RNA virus of the rhabdovirus.

1. Case Report

64 year old female patient referred from privet Hospital and RH Murgud on 4th of September with history of sudden weakness in both lower limbs since 2 days she was brought with support of two people there was history of Class III dog bite to right leg on 11th August 2012 for which she had received both active and passive immunization in RH Murgud, there was no history of hydrophobia/aerophobia/violent behavior, not associated with loss of sensation, loss of bladder/bowel control, trauma, No history suggestive of root pain there was history of cellulitis of Right Lower limb . 10 days (25th August) prior to admission proximal muscle weakness of both lower limbs for which she was and was referred to our hospital for further management. Patient is a non diabetic, non hypertensive with no history of major medical/surgical illness in the past.

On admission (Day1)-the patient was conscious oriented to time, place and person Afebrile, P=80bpm, BP=120/80 mm of Hg in the right arm supine position. The patient was paraparetic (Power Grade I/V), with flaccid type of paralysis without sensory and bladder and cranial nerve involvement with bilateral extensor plantar responses with are flexia in lower limbs. EMG/NCV was done.

Day 2-Patient developed disorientation with irrelevant talk and mild weakness of both upper limbs with loss of all deep tendon reflexes. There was no evidence of respiratory muscle weakness/cranial nerve or sensory nerve involvement. Saturation by Arterial blood gas sampling, blood glucose and serum electrolytes were within normal ranges.

Day 3-Patient developed frank quadriparesis with respiratory muscle involved with bulbar palsy.

Day 4, 5, 6 – Patient remained comatose for 3 days and patient succumbed to death on 6th day and patient's relatives were not willing for post mortem examination.

2. Investigations

Reports

Hb-12.8
TLC-12,700/cmm
Plt-3.12 Lacs

BSL-93mg/dl
BUL-42 mg/dl
S.Creat -0.8
Bili(T)-0.6
SGOT-18
SGPT-14

CSF Analysis Revealed

Sugar-55mg/dl
Protins-24.8 mgs/dl
Erythrocyte count -4-5/HPF
Total Nucleated count -87/cumm
Differential count –Polymorphs -00%, Lymphocytes-100%
Gram stain / ZN Stain / India Ink preparation and culture revealed no organisms CSF for rabies antibodies was sent to NIMHANS, Bangalore (report awaited)

EMG / NCV

Upper limb MNC is normal. the lower limb MNC shows normal CMAF amplitude with normal distal latency and conduction velocity in the left tibial nerve. The CAMPS from the right tibial nerves.

MRI Brain with wlike Spine Screening

Bilateral caudate and lentiform nuclei are swollen and shows abnormal T2FLAIR hyperintense signal.

T2FLAIR hyperintense signal abnormality is also seen involving the midbrain (predominantly tectum and periaqueductal region) and B/L superior cerebellar peduncle.

Tiny/FLAIR hyperintensity is also seen involving subcortical white matter in right high parietal region.

The lower dorsal spinal cord and cauda equine are swollen and show abnormal T2 hyperintense signal abnormally.

The above mentioned imaging features are suggestive of acute encephalomyelitis.

3. Discussion

The term rabies is derived from the old Indian root word rabh, meaning to make violent. Rabies encephalitis is an

acute infection caused by a type of RNA virus of the rhabdovirus family. Transmission to humans is mainly through bites of infected rabid dogs, cats, bats and other wild animals. Other modes of transmission are through inhalation, by contact of infected saliva with an open wound or mucous membrane and via infected corneal transplants (2-4).

The incubation period of rabies is typically 2 to 8 weeks(6,7), although it varies with the type of infecting strain and is found to be inversely related both to the size of the inoculum and to the proximity of the site of the bite to the CNS. The viruses are usually introduced deep in to the soft tissues by an animal bite and infect the muscle, possibly through an affinity for nicotinic cholinergic receptors. The replicate in the muscle before reaching the CNS, either through the neuromuscular spindles or the motor end plates (6). Alternatively the virus may directly affect the sensory nerve endings of the superficial soft tissue. The passage of the virus to the CNS occurs axonally through retrograde axoplasmic flow of approximately 12 to 24 mm per day until the virus reaches the next neuronal cell body. In paralytic rabies the medulla and the spinal cord are mainly involved by extensive neuronal damages and inflammation, whereas in the encephalitic form it is the brain stem and the cerebrum, particularly the limbic system. Clinical illness begins upon arrival of the virus in to the CNS. Human rabies may present in one of two forms. Encephalitic and paralytic.

(7) In encephalitic rabies the initial symptoms are nonspecific such as fever, malaise, anorexia, cough and pain or paraesthesia at the bite site. Subsequently patient develop hydrophobia, aerophobia, hypersalivation, hyperirritability, hyperactivity and priapism. Neurologic symptoms such as seizures, agitation and alternating mood swings, often occur. Paralytic rabies encephalitis causes more diagnostic problems. As the clinical symptoms of hydrophobia and aerophobia are present in only half these patients. Both form of the disease are invariably fatal and most patient die within 10 days of the onset of neurologic symptoms. Patient usually succumbs to failure of basic central vegetative functions. Although death may also be due to concomitant rabies myocarditis.

4. Conclusion

As the patient was in the incubation period of rabies was our first differential diagnosis with post vaccinal encephalomyelitis being the 2nd. The CSF report of the rabies antibodies is still awaited. The question of post exposure active and passive immunization failure still exists.