Differentiating paralytic rabies from post antirabies vaccine polyradiculoneuropathy

Sir,

The recent report of flaccid paralysis following anti-rabies vaccine (ARV) was interesting, where Behari et al describe the diagnostic dilemma in a patient presenting with flaccid paralysis following administration of ARV. They mention that measurement of rabies antibody titer in the serum and cerebrospinal fluid could help in differentiating paralytic rabies from post-ARV polyradiculoneuropathy (Guillain-Barre syndrome, GBS). However, I would like to make certain observations.

Firstly, there are several features that could be useful in differentiating paralytic rabies from polyradiculoneuropathy, which could be summarized as follows:

1. History of dog bite: In a person who has not been bitten by a dog (as in the case reported by Behari et al), there is virtually no possibility of rabies and the diagnosis of GBS is straightforward.

2. Incubation period: The mean incubation period in paralytic rabies is 49 days, as compared to 14 days in case of post-ARV neurological syndromes.

3. Clinical involvement: Sphincter disturbances and sensory symptoms (in addition to ascending flaccid paralysis) are common in paralytic rabies, which is not the case with post-ARV polyradiculoneuropathy. This could be explained on the basis of direct involvement of brainstem and spinal cord by rabies virus, proven by autopsy studies.

4. Disease progression: Paralytic rabies progresses rapidly with early respiratory paralysis and death ensues within 7-11 days of symptom onset in all cases. On the other hand, post-ARV polyradiculoneuropathy has a better outcome with conservative management or immunotherapy and the mortality is less than 10%.

5. Magnetic resonance imaging (MRI): MRI of the brain in paralytic rabies shows exclusive involvement of the gray matter including the basal ganglia, thalami, pontine and midbrain nuclei. This is in contrast to the predominant white matter involvement in post-vaccinal acute disseminated encephalomyelitis. Moreover, in polyradiculoneuropathy; MRI is usually normal (as in the case reported by Behari et al).

Secondly, Behari et al treated their patient with steroids. However, significantly better therapeutic results have been shown with cyclophosphamide as compared to steroids. Moreover, patients treated with steroids have a higher incidence of relapse of GBS. Plasmapheresis or intravenous immunoglobulins are better options for treatment of these patients.

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References


Authors’ Reply

Sir,

We appreciate the comments and would like to clarify the issues raised:

1. History of dog bite: It is known that the most common cause of rabies is dog bite but in a small number rabies is caused by other animal bite as well. However, the dilemma arises in a patient bitten by dog and the question which arises is if the flaccid weakness is due to paralytic rabies or post antirabies vaccine polyradiculopathy.

2. Incubation period: Though the mean incubation period in paralytic rabies is 49 days, there are case in whom symptoms of paralytic rabies occur earlier.

3. Sphincter disturbance and sensory symptoms if occur are helpful in diagnosing a case as paralytic rabies. However, it may not be present in early stages in most cases.

4. We agree that disease progression is rather rapid and downhill in paralytic rabies but is not useful at the time of presentation.

5. Abnormality on magnetic resonance imaging is observed in paralytic rabies and was emphasized in the report. The point that we wished to raise were the difficulties en-
countered in such a situation especially in the first few days and tips to differentiate the two conditions.

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Satoyoshi syndrome:
Comments

Sir,

I read with interest a case report of Satoyoshi syndrome by Ashalatha et al.1 The similarity with a ‘jerking stiff-man syndrome’—a variant of stiff-man syndrome (SMS) is striking. Distal limb involvement, myoclonic jerking, board-like muscles, stimulus-evoked spasms, endocrinopathies, vitiligo, pernicious anemia and sicca syndrome can also be seen in jerking SMS.2 Continuous motor unit activity (CMUA) is necessary to diagnose this variant but can sometimes be elusive and manifest only in the paraspinal muscles.3 Earlier this year, anti-GAD antibodies were described with Satoyoshi syndrome.4 This validates the auto-immune nature of this disorder and suggests that it might be a variant of stiff-man syndrome. In both cases, a partial response to immunomodulating agents such as immuno-globulin or high dose steroids may be seen. I wonder if anti-GAD antibodies could be tested in the described case as well.

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References


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Neurosurgical training in India at the crossroads?

Sir,

The Neurosurgical Training in India at present seems to be in the crossroads. The problem seems to be many folds: 1) There does not seem to be any regulation of demand and supply, leading to underemployment and unemployment among qualified neurosurgeons! 2) There is a gross disparity between training imparted in various institutions, leading to gross difference in the quality of the Neurosurgeons produced. 3) There are the problems in training in non-teaching Private Hospitals offering neurosurgical training approved by the National Board of Examinations (NBE). These problems need to be addressed urgently by the Senior Neurosurgeons and Neurosurgical Teachers in the interest of the Profession and the Community at large.

On an average about 40 to 50 Neurosurgeons (both M.Ch. and D.N.B) are added every year in India. Though the number of neurosurgeons per population may still be low, compared to the developed countries, the non-availability of neurosurgical facilities precludes the migration of trained neurosurgeons to

Authors’ Reply

Sir,

We Dr. Varkey for the interest in our case report on Satoyoshi syndrome1. We are aware of the very recent publication of Drost et al., of anti-GAD antibodies in Satayoshi syndrome2. Interestingly, unlike our patient, the case reported by them did not have any of the typical features of Satayoshi syndrome such as short stature, bony deformities, glucose intolerance, jaw muscel spasms etc. which were reported by Satayoshi. But for the age of onset, the case could have easily been called stiff person syndrome, a condition where 65% patients could have antiGAD antibodies. We agree that if the test were positive in a more typical case such as ours, it would add more strength to the presumed autoimmune etiology of the condition as well as the suggestion of Drost et al., that Satayoshi syndrome and Stiff person syndrome could both be different clinical expressions of a spinal hyperexcitability state in different age groups. We will pursue the same.

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References


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