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Brown-Séquard syndrome due to Semple antirabies vaccine: case report

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We report a patient with a Brown-Séquard syndrome due to Semple type antirabies vaccine, with subclinical electrophysiological abnormalities, who recovered with immunosuppressants.

Key words: Brown-Séquard sydrome; antirabies vaccine; immunosuppressants; rabies; vaccination.

Introduction

Although neurological complications of the Semple antirabies vaccine are common[1–7], development of the Brown-Séquard syndrome has not previously been reported. We describe a patient with signs and symptoms resembling the Brown-Séquard syndrome following Semple antirabies vaccine and his complete recovery with immunosuppressive therapy.

Case report

A 28 year old man was admitted with neurological complications due to the Semple type of antirabies vaccine (ARV). He was bitten by a healthy dog one month before admission. The patient received 4 doses of vaccine on 4 consecutive days beginning on the day following the dog bite. One month after the first dose of ARV he developed pain around the thorax for 2 days. On the third day he had urinary retention, and a day later developed weakness of the right lower limb and inability to appreciate touch and pain sensation in the left half of the body below the middle part of the chest.

On examination, his higher mental function, cranial nerves and upper limbs were normal. There were pyramidal signs in right lower limb with 0–3/5 (MRC) weakness, spasticity, brisk deep tendon reflexes and an extensor plantar response. Pain, touch and temperature sensations were impaired over the trunk below T5 on left side and in left lower limb. Joint sensation was normal bilaterally. Other systems were normal.

Investigations

Haematological indices were normal as were estimation of blood sugar, urea, sodium, potassium and the liver function tests. Lumbar cerebrospinal fluid analysis, x-ray of the thoracic spine and spinal computerised tomography were also normal, as were the electroencephalogram, visual evoked potentials and brain stem auditory evoked responses. The somato sensory evoked potential of the posterior tibial nerve (N 39) was 39.5 m/sec bilaterally and the wave form was poorly defined. Electromyography was done with concentric needle electrode on 4 channel DISA 1500 model. The right abductor pollicis brevis, right quadriceps and right tibials anterior were sampled. Electromyography of the right abductor pollicis brevis was within normal limits. There was no spontaneous activity in the right quadriceps and the right tibialis anterior and the motor unit potentials were of normal amplitude and duration, but the firing rate was reduced to 4/second. Recruitment was incomplete in both these muscles, suggesting probably upper motor neuron lesion. Nerve conduction parameters are shown in Table I. In summary, abnormalities of nerve conduction were bilateral absence of F response from common peroneal nerve and absence of Hoffman's reflex from the left posterior tibial nerve suggesting radicular involvement. There was decreased amplitude of the left sural sensory action potential as compared to the right, with normal conduction velocity suggesting a mild postganglionic axon loss neuropathy.

Table I Nerve conduction parameters

Nerve	Site of stimulation	Site of record	Evoked motor response Latency ms	Amplitude mv	Velocity m/sec
Right median	Wrist	APB	2.4	7.5	52.71
		F = 24.0 (6/10)			
Right common peroneal	Ankle	EDB	6.4	5.0	42.11
		F = absent			
Left common peroneal	Ankle	EDB	5.3	6.0	46.10
		F = absent			
		'H' reflex not elicited left post tibial nerve			
			Sensory action potential Latency ms (onset)	*Amplitude v*	
Right median	Dig. II	Wrist	2.4	28.0	64.58
Right sural	Mid calf	Ankle	5.4	18.0	34.01
Left sural	Mid calf	Ankle	4.0	6.0	37.50

Treatment response

Injections of dexamethasone 16 mg daily were given from one month after the onset of illness for 15 days and tapered off over one month. Intravenous injections of cyclophosphamide 200 mg daily were given for 10 days from the same date. The patient gradually improved and was free from neurological deficits at the last follow up, 4 weeks later.

Discussion

Semple vaccine, a suspension of phenol inactivated rabies virus in the brain of sheep or higher mammals, is used in many developing countries. The neuroparalytic accidents due to this vaccine occur in about 1 in 220 cases and the failure rate is about 11%.[8,9] Suckling mice brain (SMB) vaccine containing inactivated rabies virus is used in Latin America and is associated with neuroparalytic accidents of 1/7865.[2]

Duck embryo vaccine (DEV), a non neural vaccine, was introduced during 1959. Label and Batts[3] reviewed literature in 1982 and found 24 cases of neurological complications due to this vaccine.

Human diploid cell (HDC) a non neural rabies vaccine was introduced during 1976. The neurological complications due to this vaccine are rare and it is highly immunogenic in contrast to Semple vaccine.[9]

The neurological complications are immunologically mediated and immunosuppressants are of great value as in the present case.[1,10–13]

With the present knowledge, hemisection of the spinal cord experimentally should produce loss of pain and thermal sensations on the opposite side of body and loss of proprioceptive sensation with pyramidal signs of the same side of lesion. However this is rarely seen clinically in complete form, partly because of the anatomy of blood supply, and in this case posterior column sensation was preserved on the side of the lesion.

The time has come to use human diploid cell vaccine exclusively even although stores of Semple vaccine may still be available and be much cheaper. The complication rate of Semple vaccine is so high that it hardly seems justifiable to use it nowadays.

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