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Case report

Monsoon fasiculation paralysis syndrome: revisited

Apeksha Pokalkar*

Neuro Care, Hyderabad, Telangana, India

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***Correspondence:**
Dr. Apeksha Pokalkar,
E-mail: mdneurocare@gmail.com

ABSTRACT

Cases of sudden onset external ophthalmoplegia with bulbar and skeletal muscle weakness and fasciculations are noted in this region during the monsoon season. The neurological deficit peaks within 2-3 days with potential risk of respiratory paralysis. Total recovery takes place recovery within 4-8 days if respiratory crises is taken care of. Two such cases who presented with acute onset of flaccid quadriplegia with relative preservation deep tendon jerks are reported here. There was no history of any bites or exposure to known toxins. The patients made spontaneous recovery in one week time.

Keywords: Quadriplegia, Ophthalmoplegia, Bulbar palsy, Fasciculations, Monsoon season

INTRODUCTION

Interesting cases of an acute onset, external ophthalmoplegia with bulbar and skeletal muscle weakness with fasciculations occurring particularly during monsoon season in this geographical area of unknown etiology.[1] The patients can land up in life threatening respiratory paralysis during the peak of weakness. There is total recovery of neurological deficit in 4-8 days once the crisis subsides. These cases need to be differentiated from cholinergic crisis and snake bite for proper management.

CASE REPORTS

Case 1

In August 2019, a 26 years old homemaker from a suburban area presented with overnight onset of inability to open eyes, difficulty in swallowing and weakness of both upper and lower limbs of one day duration. Around 3am she developed abdominal pain and a bout of vomiting and went back to sleep.

On awakening she noticed inability to move her upper and lower limbs, difficulty in swallowing and drooping of both eyelids. No history of diplopia, blurring of vision, increased salivation or diarrhoea. Physical examination did not reveal any bite marks. No evidence of toxin ingestion. Pupils normal, extraocular movements were restricted in all directions without diplopia. Muscles of mastication were weak. Bilateral ptosis with bifacial and bulbar weakness present. Other cranial nerves were normal. Proximal muscle weakness of shoulder and hip girdle and axial muscles of grade 3/5.

Distal muscles normal. There were fasiculations in the calf and arm muscles. Sensory system, deep tendon and plantar reflexes were normal. Other system examination was normal. Weakness progressed for two days. She needed Ryles tube feeding, but could maintain oxygen saturation on her own. The fasiculations waned on day 3 and the patient made steady improvement in her ocular, bulbar and skeletal muscle weakness. Her respiratory function was monitored by respiratory rate (RR), single breath count test (SBCT) and oxygen saturation.[2] In five days, she made a complete recovery.

No bladder or bowel dysfunction. There were no cramps, increased salivation, lacrimation, diarrhoea or blurry vision. She denies snake bite or ingestion of stale food, high carbohydrate food or any pesticide. The illness was not preceded by febrile illness or gastroenteritis.

Investigations

Routine biochemical tests including serum potassium and CPK were normal. Nerve conduction studies (NCS) were normal. Repetitive nerve stimulation test (RNS) test showed no decrement. Neostigmine and ice pack test negative. CSF, MRI-brain and spine, CECT-abdomen normal.

Diagnosis

Monsoon fasciculation paralysis syndrome.

Treatment

Symptomatic treatment with neostigmine and atropine was given. The patient did not receive any polyvalent anti snake venom (ASV) serum.

Case 2

In July 2023, a 42 years old man farmer by occupation from neighbouring district developed overnight onset of oculobulbar palsy with quadriplegia when he was sleeping indoors. He had external ophthalmoplegia, bifacial weakness, masticatory and bulbar weakness. He also had axial, proximal muscle weakness of grade 4/5 with extensive fasiculations and myokymia over thigh, calf and arm muscles with preserved reflexes.

Weakness progressed to 2/5 on day-2 when he transiently developed tachypnoea, SBCT of 20/minute, however he maintained oxygen saturation. Ventilatory support was not needed however the patient required nasogastric tube feeding for four days. Fasiculations subsided on day 2 and patient made steady and complete recovery on day 6. No significant antecedent illness prior to this illness. There was no exposure to any toxins. None of his family members had similar illness.

He was evaluated on similar line as described above. His routine blood chemistry was normal. His NCS and RNS were normal. Needle EMG showed repetitive discharges and fasiculation potentials with full recruitment. Ultrasound abdomen was normal. Imaging of brain and spine were not done. The patient did not receive atropine and neostigmine or polyvalent ASV serum.

DISCUSSION

Both the patients were from rural background, had overnight onset of neurological illness during monsoon season. Characteristic muscle involvement of ocular, facial, masticatory, bulbar and skeletal muscle weakness mimicking snake envenomation or cholinergic crisis.[3,4] The interesting feature was both patients had fasiculations and myokymia. Both of them needed nasogastric feeding during peak period of muscle weakness and had potential risk of respiratory failure. But both patients described neither had myasthenia gravis nor were bitten by any poisonous snake. They were not exposed to organophosphates. No other family members were affected. In both cases polyvalent anti-snake venom serum was avoided as there was no clear clinical evidence of snake bite.

Spontaneous total recovery within one weak time indicate that whatever toxin involved is a reversible one by an unknown animal which comes to human dwellings in monsoon season and is small enough to go unnoticed. Both patients could not recollect having bitten by any animal.

Interestingly such cases were not reported in other seasons. Snake envenomation occurs all through the year with marginal increase during monsoon season. Detailed differential diagnoses and pathophysiological mechanisms are described in literature.[1]

The clinical clue for the diagnosis of this syndrome is presence of fasciculations in first 2-3 days of onset. Personal communication with the departments of medicine and neurology of two premier government institutions of Hyderabad indicate that number of cases were decreasing in recent years. This might be due rapid urbanisation of the surrounding villages which changes the habitat of the animal. Such cases must be occurring elsewhere too, but easily mistaken as snake bite treated accordingly and go unreported.[5,6]

Other animals were incriminated for neuroparalytic syndrome and criticised as well in the literature.[7] There is paucity of literature on the toxins which could cause such characteristic involvement with fasiculatons and repetitive discharges on needle EMG. Detailed epidemiological studies might identify the animal involved, then toxicological studies can be carried out in future.

CONCLUSION

Awareness of monsoon fasciculation paralysis syndrome is important for its detection in emergency ward to prevent death due to respiratory paralysis and to avoid inadvertent use of anti-snake venom sera.

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