Figure 1: T1-weighted (a) and T2-weighted (b) MR imaging demonstrates heterogeneously hypointense and hyperintense pattern respectively. Coronal MR imaging (c) noted 2.5 cm nodular lesion connected to the upper part of the main lesion.

Figure 2: DSA image shows the displacement of anterior and posterior vascular territory of the brain and no aneurysmal dilatation. There is avascular mass effect.

Figure 3: Histopathological section of the lesion discloses the typical lamellar structures of dermoid cysts.

Organo-phosphate induced delayed neuropathy: Report of two cases

Sir,

Organophosphates are the most common cause of acute poisoning in India. Organophosphate induced delayed neuropathy (OPIDN) is common following exposure to OPC’s, which have weak cholinergic activity, little insecticidal activity and are of use in chemical warfare. The presently used organophosphates have potent cholinergic activity and are...

References


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being widely used as insecticides. However OPIDN is distinctly uncommon following exposure to these OPC’s.¹

A 35-year-old farmer consumed about 200 ml of dichlorvos with a suicidal intent. He was treated with atropine and pralidoxime and required assisted ventilation for 7 days. Four days after his discharge, he developed weakness of all the four limbs and fever. Neurological examination revealed generalized hypotonia with power 3/5 in proximal muscles and 5/5 in distal muscles of upper limbs and 3/5 and 4/5 in lower limbs respectively. Sensory examination was normal. However there was complete areflexia and plantars were mute. Hematological, biochemical and CSF examinations did not reveal significant abnormalities. Nerve conduction revealed normal distal latency (right median 3.2 ms, 7.3 ms wrist elbow), (ulnar 2.9 ms, 7.4 ms wrist below elbow), (peroneal 2.3 ms, 2.0 ms ankle fibular head); amplitude (11.0 mV, 8.3 mV wrist elbow; 5.9, 6.3 mV; 2.3, 2.0 mV) and conduction velocity (54 ms, 51 ms, 37 ms) respectively. However F-waves were absent. Sural nerve was not stimulable. Sensory nerve conduction in the right ulnar and median nerves was peak latency 2.6, 2.7 ms; amplitude 14 mV, 38 mV; conduction velocity 51 ms and 62 ms respectively. These findings were considered to be consistent with mild polyradiculoneuropathy with mild peripheral neuropathy. The nerve conduction was repeated 5 days later and it revealed decreased amplitude (1.4 mV and 1.1 mV); distal latency (7.5 ms) and conduction velocity (43 ms) in the right peroneal nerve. The right sural nerve was not stimulable. However in the median and ulnar nerves, amplitude, distal latency and conduction velocity were normal. EMG of right vastus lateralis revealed normal insertional activity, minimal spontaneous activity, a few polyphasic MUP’s with increased duration and amplitude with decreased recruitment and discrete pattern and less than normal interference consistent with neurogenic EMG. Repetitive nerve stimulation (RNS) did not reveal increment-decrement phenomenon. He was followed up for 6 months during which he recovered completely and the nerve conduction done at 6 months was normal except F-waves were absent (right median nerve distal latency 4.2 ms, 8.7 ms; amplitude 12.6 uV, 9.7 mV; conduction velocity 47 ms and amplitude 4.1,4.7 mV).

A 19-year-old female had ingested 200-300 ml of monocrotophos with a suicidal intent 23 days ago and was treated with atropine and pralidoxime (total 3 gm). For the next 3 weeks, she continued to remain in altered sensorium and was brought to the institute. On examination her pulse was 100/min and BP 130/70 mmHg. Pupils were 3 mm in size with normal reaction to light. Neurological examination revealed spontaneous eye opening and movements, generalized hypotonia, areflexia and power grade 0-1 in all the four limbs. Nerve conduction revealed absent F waves but normal latency (3.2 ms, 7.4 ms wrist elbow), amplitude (4.0 m V, 2.3 m V and conduction velocity (57 ms) in right median motor nerve. The sensory median conduction revealed normal peak latency (2.9 ms), amplitude (12 uV) and conduction velocity (55 ms). However the peroneal nerve was not stimulable. The impression was predominant distal motor neuropathy more marked in the lower limbs than the upper limbs. EMG of right vastus lateralis revealed normal insertional activity but minimal spontaneous activity, a few polyphasic MUP’s with increased duration and amplitude with decreased recruitment and discrete pattern with less than normal interference as in first case. RNS could not be carried out. Her altered sensorium was thought to be due to hypoxic brain damage. However MRI of brain was normal. She was treated with antibiotics and became afebrile after about 2 weeks. Her neurological status had not changed at discharge and she did not return for follow up.

Organophosphate induced delayed neuropathy is a well-recognized complication of organophosphate poisoning.² The later appears 2-4 weeks after poisoning and leads to motor paralysis affecting the distal muscles of limbs, minimal sensory involvement and calf pain which precedes its onset. It has been reported following poisoning with compounds like TOCP, mipafox, leptophos, chlorophos etc which have weak cholinergic activity and are not being used as insecticides at present.² Senanayake and Jhonson reported 10 cases of OPIDN following poisoning with methomidaphos.³ All these cases had acute cholinergic crisis preceding its development. Das and Jena⁴ have reported a young female who developed OPIDN following phorate ingestion. Both our patients had acute cholinergic crisis and 2-3 weeks later developed OPIDN.

In patients reported by Senanayake and Jhonson, 50% inhibition of neuropathy target esterase (NTE) was found.¹ However we have not been able to estimate this in our patients. NTE is present in abundance in the axons of nervous system and > 70% of its phosphorylation and ageing leads to development of neuropathy in experimental studies.²

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