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CASE REPORT

Medicine Science 2017;6(3):588-91

Medicine Science International Medical Journal

Distal symmetrical polyneuropathy due to organophosphate intoxication

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Received 20 March 2017; Accepted 27 April 2017
Available online 06.04.2017 with doi: 10.5455/medscience.2017.06.8634

Abstract
While a cholinergic crisis picture is onset in intoxication of organophosphate compounds; a severe polyneuropathy mainly affecting the motor fibers is viewed lately following weeks. Cholinergic crisis may require intubation meanwhile lately onset neuropathy may be characterized by cranial nerve palsies, a hazy vision, ataxia and skin paresthesia. Exposure to substance could be progressed by respiratory tract and skin. 20-year-old male patient with muscle weakness and thinning in the hands and legs was admitted to our outpatient clinic. In his story; he had suicidal drunk pesticide 6 months ago, hospitalized due to loss of consciousness, given respiratory support in the intensive care unit for following 11 days. During the following period, sensory disorders involving numbness and pain, and thinning in the arms and legs were onset. At nerve conduction study, axonal polyneuropathy was identified: sensory fibers were protected, motor conduction velocities and amplitudes were extremely decreased. At needle EMG studies widespread denervation potentials were recorded to be more pronounced in the distal muscle groups. In the discussed case; a cholinergic crisis picture occurred after exposure to toxic agents in the acute phase, the patient was observed as intubated for a while, in the subacute phase a polyneuropathy mostly located in the proximal muscles was onset and muscle atrophies have emerged in the late phase. In this writing, an organophosphate intoxication case in which cholinergic crisis was set in earlier days, polyneuropathy picture was seen on subacute stage and muscle atrophies were confirmed on following weeks was discussed.

Keywords: Organophosphate intoxication, cholinergic crisis, polyneuropathy

Introduction

Organophosphate compounds were first produced in early 1800's by Lassaigne as a result of a reaction of alcohol with phosphoric acid and in early 1900's chemists have manufactured pesticides that contain organophosphate compounds [1]. Currently, the main area of use for organophosphate compounds is pesticides and chronic exposure by inhalation or by skin can be a concern particularly in populations engaged in agricultural activities. Acute exposure usually occurs due to suicidal or accidental oral intake. Organophosphorus gases have caused many deaths as a weapon of mass destruction during First and Second World Wars and most recently in 1995 in Tokyo metro line, Japan [2]. Organophosphate compounds cause three well-defined neurological complications: a) Acute cholinergic crisis, b) Intermediate syndrome, c) Organophosphate-induced delayed polyneuropathy (OPIDP) [3]. Cholinergic crisis develops as a result of increased depolarization of nicotinic and muscarinic receptors at post-synaptic membrane.

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Usually occurs during the early phase. Intermediate form was described in 1974: proximal muscle weakness and respiratory distress develops within 24-96 hours. Distal muscles are mildly affected and cranial nerve palsies may accompany the clinical picture. Clinical manifestations may remain for 1-2 weeks and intubation may be required; 20-50% of the cases are in this group. Neuropathy is characterized by distal sensorimotor axonopathy predominantly involving motor fibers which typically occur 2-3 weeks following exposure [4]. Clinical features include weakness in limbs, moderate sensory impairment, muscle cramps, burning stinging pain. Blurred vision, balance disorders and weakness may accompany the clinical presentation. Spinal cord involvement with spasticity and increased reflexes can be observed in chronic exposure.

Case Report

Twenty years old male patient complaining of thinning and weakness of legs and arm had a history of pesticide ingestion (Metamidophos- Metakor 60 SL) in a suicidal attempt six months ago. According to the patient's statement, following ingesting approximately a tea glass of the pesticide he lost his conscious. He was told later that he

stayed in intensive care unit of Yeşilyurt State Hospital in a coma for 11 days with respiratory support.

After discharge from the intensive care unit, he was transferred to the ward. During the additional 6 days he was treated, he stated that his legs and arms were weak; he could not walk without help, although his complaints have reduced since then. He only described weakness limited to hands and distal parts of feet.

Neurological examination have revealed that cranial nerve involvement was absent, symmetrical atrophy of distal muscles was present in upper and lower extremities (Figures 1, 2 and 3) distal weakness was predominant in lower extremity (Table 1). Bilateral Achilles reflexes were negative, other reflexes were hypoactive. The patient described pain and reduced heat sensation with glove-stocking distribution while vibration and joint position sensations were normal.

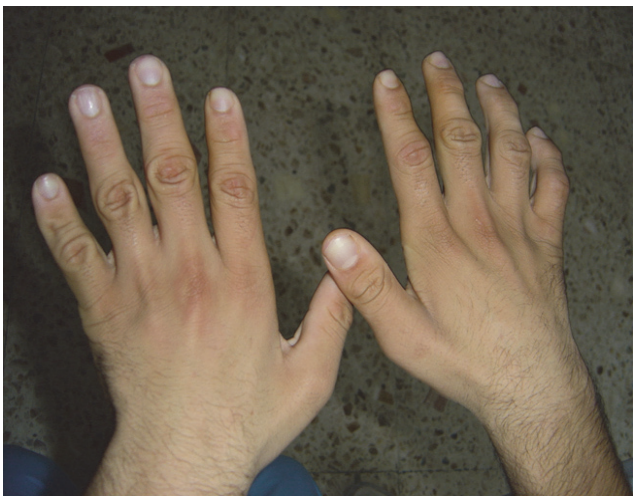

Figure 1. Interosseous muscle atrophy observed in the hands of the dorsal side

On electrophysiological assessment, sensory conductions were normal. Evaluation of motor conduction showed prolonged latencies, very low amplitudes and reduced velocities or loss of response. F responses were absent. Needle EMG evaluation revealed extensive denervation potentials of distal extremity muscles (Table 2, 3, 4,5).

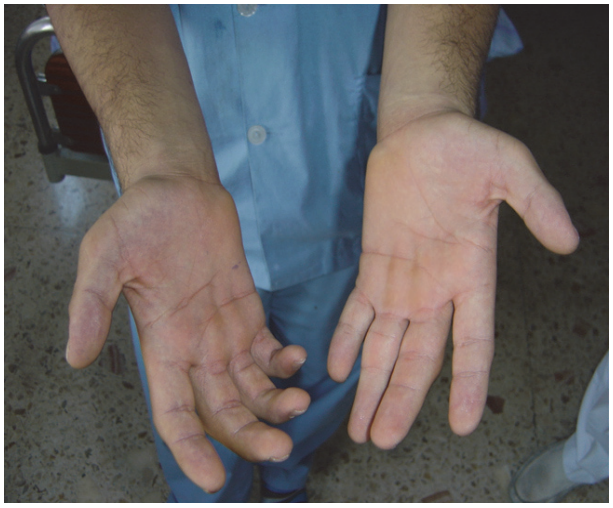

Figure 2. Tenar and hypothenar atrophy

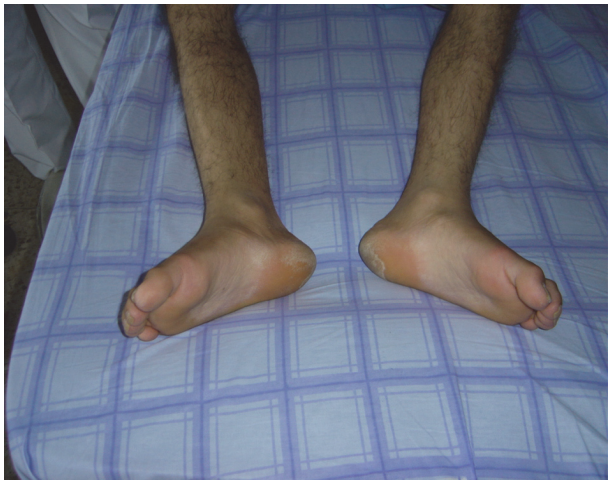

Figure 3. Atrophy is observed in the lower extremity distal muscle groups

Table 1. Muscle strength assessment

Muscle Evaluation	Left	Right
Deltoid	5/5	5/5
Biceps	5/5	5/5
Triceps	5/5	5/5
Upper Distal	3/5	3/5
Psoas	5/5	5/5
Quadriceps femoris	5/5	5/5
Ankle dorsiflexion	0-1/5	0-1/5
Ankle plantar flexion	0-1/5	0-1/5

Table 2. Values of sensory nerve conduction study

Nerve	Stimulus site	Record site	Distal latancy (msn)	Peak amp. (µV)	Distance (cm)	Conduction velocity (m/sn)
R – Median	Wrist	II. P.	2.90	9.7	14	56.0
R – Ulnar	Wrist	V. P.	2.90	7.3	12.5	52.1
R – Sural	Calf	Lat. Mal.	3.65	16.9	15	50.8

Table 3. Values of motor nerve conduction study

Nerve	Stimulus site	Record site	Distal latancy (msn)	Peak amp. (µV)	Distance (cm)	Conduction velocity (m/sn)
R – Median	Wrist	APB	No potential			
R – Ulnar	Wrist	ADM	3.90	0.3	5.5	
	Below elbow	ADM	9.90	0.1	20.5	34.2
	Above elbow	ADM	11.95	0.4	10	48.8
R – Radial	Elbow	EIP	3.75	4.2	13	
	Spiral groove	EIP	5.45	4.8	11	64.7
R – Peroneal	Ankle	EDB	No potential			
R – Tibial	Ankle	FHB	No potential			
R – Femoral	Ing. Canal	VM	7.8	1.4		
R – Peroneal	Cap. Fibula	TA	No potential			

Table 4. F response values

Nerve	Minimum F Latancy (msn)
R – Median	No potential
R – Ulnar	No potential
R – Comm. per.	No potential
R – Tibial	No potential

Table 5. Needle EMG study results

Muscle	Spontaneous Activity			MUAP			
	Fibrilation	PWD	Fasciculation	Amplitude	Duration	Polyphasia	Pattern
R – Deltoid	None	None	None	N	N	+1	N
R – Biceps	None	None	None	N	N	+1	N
R – EDB	None	None	None	N	+1	+2	Reduced
R – 1st DIO	+3	+3	None				Absent
R – ADM	+2	+2	None	-2	+1	+1	Discrete
R – APB	+1	None	None				Absent
R – EDB	+3	+3	None				Absent
R – TA	+2	+2	None				Absent
R – Med. GC	+3	+4	None	N	N	N	Discrete
R – AH	None	None	None	N	N	N	Absent
R – VM	None	None	None	+1	+1	+2	Reduced

Discussion

Organophosphate compounds are widely used as pesticides and in petroleum and plastic industry. Toxic exposure to organophosphate compounds occurs either through chronic skin exposure or by absorption from the respiratory-gastrointestinal systems. High fat solubility of these compounds allows their rapid delivery into the central and peripheral nervous system. Organophosphate compounds associated with neuropathy are tri-o-cresyl phosphate (TOCP), leptophos, mipafox, chlorphos, trichlorfon, malation, paration, metriphonate ve metamidophos [5].

Clinical features including probable acute cholinergic crisis, proximal muscle weakness recognized approximately 11 days later and subsequent distal atrophy and muscle weakness that occurred following pesticide ingestion containing metamidophos indicate our patient have suffered all clinical phases of organophosphate intoxication. This presentation usually occurs in cases intoxicated with excess amounts of the substance.

Occurrence of neuropathies approximately 1-2 weeks following acute phase is related to phosphorylation of certain proteins of the nervous system and associated Wallerian degeneration.

Motor neuropathies are the predominant clinical feature in the reported case. Such patients usually do not specify sensory complaints or describe paresthesia-hyperesthesia.

However, occasionally objective findings related to sensory loss may be observed on examination. In our patient, distal symmetric polyneuropathy with predominant motor component was detected which appeared at two weeks following acute cholinergic crisis and intermediate syndrome and persisted.

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