Neurological Complications after Acute Organophosphorus Poisoning

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Study was done in medical unit 1 BV hospital Bahawalpur to evaluate the neurological complications in surviving patients of acute organophosphorus poisoning.There were 38 patients of acute poisoning admitted in unit 1 from 1st Jan 1994 to 31st Dec 1995.5 patients died due to poisoning.Out of thitrythree surviving patients five developed pure motor polyneuropathy which was axonal type.The mean duration for appearance of axonopathy clinically following poisoning was 31.8 days.There was no sensory neuropathy and other neurobehavioural changes.On follow up visits for one year there was no improvement.

Key words: Neurological complications, organophosphorous poisoning

Organophosphorus insecticides include chlorpyrifos, malathion, parathion, methyl parathion and fenthion. They irreversibly inhibit acetylcholinesterase, resulting in an over abundance of acetylcholine at synapsis and myoncural junction which initially excites then paralyze the CNS[1]. So organophosphate insecticides produce clinical effects by acting on the autonomic nervous system, on skeletal muscle and on cholinergic sites within CNS. There are three recognized types, of toxicity syndromes: acute which is cholinergic crises and chance of death is high, intermediate (24 hours to 96 hours) vocal cord paralysis and muscle weakness, delayed type-syndrome weeks to months later including polyneuropathy, pyramidal signs and chronic neurobehaviural effects[2].

A study is conducted in medical unit 1 BV hospital Bahawalpur to evaluate the late neurological complication in surviving patients of acute organophosphorus poisoning due to oral ingestion for suicide, inhalation and through skin during spray on crops.

Patients and Methods

This study included thirty eight patients who were admitted in medical unit-I B.V hospital Bahawalpur with acute poisoning of organophosphorus compounds from Ist Jan 1994 to 31 Dec 1995. These patients were treated as an acute emergency appropriately. The patients who survived were followed clinically every 3-months for a period of one year. Those who developed muscular weakness and wasting of limbs were examined on each visit. Bulk of muscles of limbs, power of each group of muscles, reflexes and planters were checked. Sensations transmitted by spinothalamic tracts and dorsal columns were checked. All these findings were recorded on each visit. Investigations of these patients were carried out blood complete examination, urine complete examination, X-ray chest, serum urea/creatinine, LFTs and Blood Sugar, Nerve conduction and EMG.

Results

Number of patients = 38
Male = 22
Female = 16
Age range of patients = 15 – 40 years.

Table – I

Organo phosphorus poisoning	No. of patients
By Oral ingestion	10
Through skin and inhalation	28

Table – II

Number of patients died	5
Number of patients alive	33

Table – III

Patients died by oral ingestion of compounds	4
Patients died by poisoning through inhalation and skin during spray	1
Total number of patients developing weakness and wasting of distal muscles of limbs.	5

Table – IV Duration of onset of muscular wasting, weakness following poisoning

Patient No	Duration
1	18 days
2	25days
3	40 days
5	28 days
6	48 days

Table – 5

Muscular weakness and wasting following oral ingestion of poison	5 patients
Muscular weakness and wasting by inhalation and through skin	Nil

Motor deficit in five patients at the time of presentation. Bulk: Wasting of short muscles of hands including thenar, hypothenar, dorsal/palmer interossei, muscles of fore arm muscles of feet and legs.

Table 6 Clinical Finding-Higher Mental Functions. Normal

Tone	Decreased
Power	Decreased in distal groups of muscular
Reflexes	Areflexia
Planters	Downgoing
Sensations	Dorsal Column And Spinothalamic Intact

Table 7 Nerve Conduction Study (Motor)

Nerve	DML	Velocity	Amplitude	F Wave
Median	Normal 5	Normal 5	Reduced 5	Normal 5
Ulner	Normal 5	Normal 5	Reduced 5	Normal 5
Common peroneal	Normal 5	Normal 5	Reduced 5	Normal 5
Tibial	Normal 5	Normal 5	Reduced 5	Normal 5

EMG showed extensive denervation.

Table 8 Follow up clinically 3 monthly for a year. Higher mental functions -Normal

	3 months	6 months	9 months	12 months
Bulk	-ve	-ve	-ve	-ve
Power	Decreased	Decreased	Static	Static
Bulk	-ve	-ve	-ve	-ve
Power	Static	Decreased	Static	Static
Bulk	-ve	-ve	-ve	-ve
Power	Decreased	Static	Static	Static
Bulk	-ve	-ve	-ve	-ve
Power	Static	Static	Static	Static
Bulk	-ve	-ve	-ve	-ve
Power	Decreased	Decreased	Static	Static

Discussion

After poisoning of organophosphorus insectisides onset of symptoms occurs within minutes to hours. The latency depends on the dose absorbed and on the route of exposure. The earliest symptoms of mild toxicity are headache, blurred vision, weakness, and tachycardia. In severe cases sweating, salivation and generalized weakness occurs. In more severe cases the patient presents with coma, loss of pupillary reflex, tendon reflexes, flaccid paralysis, which may progress to respiratory arrest[3]. Recovery from acute effects of organophosphorus poisoning occurs over several days and is complete in most cases.

In this study five patients out of thirty three who survived, developed pure motor polyneuropathy, clinically and was confirmed by nerve conduction studies. These five patients were those who ingested the poison orally. None of the patients who inhaled or absorbed the poison through skin during spray developed this complication. Most probably it depends upon the amount of poisonous compound which is taken orally for suicide.

Delayed onset polyoneuropathy occurring with organophosphorus compounds are due to inhibition of a protein known as neuropathy target esterase (NTE)[4].

The mean intervening period of poisoning and onset of motor polyneuropathy is 31.8 days in this study varing from 18 to 48 days. Lottie et at (1986) have found that NTE inhibition occurs within hours following exposure but clinical onset of neuropathy is not until 2 –4 weeks later[5]. They also have demonstrated that inhibition of NTE present in peripheral lymphocytes can predict the development of neuropathy days before the clinical onset.

In our study nerve conduction has shown that it was axonopathy that is why on follow up there was no improvement in weakness and wasting up to a year, and there was no sensory impairment as well. Moretto A and Lotti-M have shown that motor neuropathy is axonal type and sensory component may present but very mild only when large doses of certain organophosphates are ingested[6]

On review of literature long term toxic effects affecting behavior as well as mental and visual functions are observed after exposure to high doses of poison with repeated acute intoxications[7] In this study no such behavioural changes or mental, visual dysfunctions are observed. In our patients perhaps it is because of only one acute episode not repeated as Korsank and Sato in 1977 have mentioned forgetfulness, difficulty in thinking, fatigue with varying degrees of chronic exposure to organophosphates[8].

Conclusion

- Most common neurological complication following acute organophosphorus poisoning is delayed type of pure motor polyneuropathy which is axonopathy.
- The mean duration for appearance of axonopathy clinically following acute poisoning is 31.8 days.
- This axonopathy is related with high amount of insecticide poisoning.

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