

Organophosphate Induced Neuropathy - An Epidemic Case Report at Civil Hospital, Ahmedabad

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ABSTRACT:

Introduction: Organophosphates are the important biochemical substance, generally used as insecticides, nematocides, fungicides, solvents, plasticizers, drugs, herbicides and chemical warfare nerve agents. The most common route of entry of OPs in to body, is through insecticides, whether suicidal or accidental. Acute poisoning of OP is frequently observed in developing countries. Organophosphates cause inhibition of acetylcholinesterase enzyme in the body which leads to accumulation of acetylcholine. Acetylcholine is very important for nerve function. The Prevalence of OP poisoning is around 1 million per year. **Aim:** Aim of this study was to do an epidemic case study the effect of Organophosphet on peripheral nerves. **Material and method:** 17 (9 male and 8 female) Patients with organophosphate consumptions were clinically assessed and were tested by NCV study at Physiotherapy Department of Civil Hospital, Ahmedabad. **Results and Conclusions:** The Study of 17 organophosphate poison consumption revealed that there was Electrophysiological evidence of demyelinating and axonal type of pure motor polyneuropathy affecting LL >U1 with normal sensory nerve conduction.

Key words: Organophosphate, NCV, Neuropathy.

Introduction:

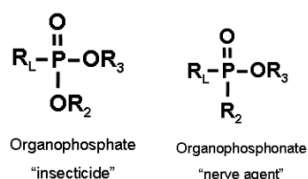
Organophosphates are the important biochemical substance, generally used as insecticides, nematocides, fungicides, solvents, plasticizers, drugs, herbicides and chemical warfare nerve agents. The most common use in the world is as insecticides¹. Organophosphate compounds are used as pesticides since 1941. Organophosphate poisoning occurs as a result of exposure to organophosphates (OPs). The most common route of entry of OPs in to body, is through insecticides, whether suicidal or accidental^{2, 3}. Organophosphates cause inhibition of acetylcholinesterase enzyme in the body which leads to accumulation of acetylcholine. Acetylcholine is very important for nerve function. Prevalence of OP poisoning is around 1 million per year.



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Molecular structure (Ecobichon 1996)

Acute poisoning of OP is frequently observed in developing countries. It was estimated that during 2002-2005 around 3,000,000 human beings were poisoned by OP pesticides in the world⁴. The total fatality rate has been estimated 20%⁵. OP pesticide mass poisoning was reported from Sierra Leon, West Africa in 1986. This epidemic poisoning affected 49 persons with 14 deaths. The toxicant identified was parathion, a highly toxic OP pesticide. The hypothesis of this epidemiological poisoning was that the bread was made from contaminated flour and that the flour was contaminated with parathion during truck shipment⁶. A similar poisoning event was noted in India in 2008 where 15 people who consumed a meal during social ceremony developed signs and symptoms of OP poisoning⁷. This epidemic study is done on 17 Organophosphate exposure patients referred to Civil hospital, Ahmedabad (Male 9, female 8) with history of consuming wheat with castor oil.

Materials and Methodology:

For present Case study 17 both male and female patients, having History of consuming organophosphate through food admitted at Civil Hospital ,Ahmedabad were taken. The duration of Study was One week. All Patients were Examined Clinically and Motor and Sensory NCV study was done of Bilateral Tibial nerves, Bilateral Peroneal Nerve, Bilateral Ulnar and Bilateral Median Nerves on RMS EMG/NCV machine.

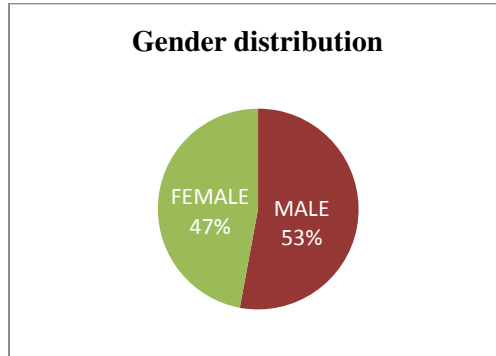
Observations & results:

17 patients were referred to Civil Hospital, Ahmedabad between 5th September to 12th September 2015.

- 1) All patients were living in same locality (Dahegam).
- 2) All patients gave history of consuming roti made from wheat flour, wheat was stored by applying castor oil and/or insecticides spray on it to prevent rotting.
- 3) Most of members of the family are affected to a more or lesser extent.
- 4) All have symptoms from 10 days to 2 months. (Only 1 patient had affection since 2 days)
- 5) All had difficulty in walking as the first symptom.
- 6) No patient gave history of fever, infection.

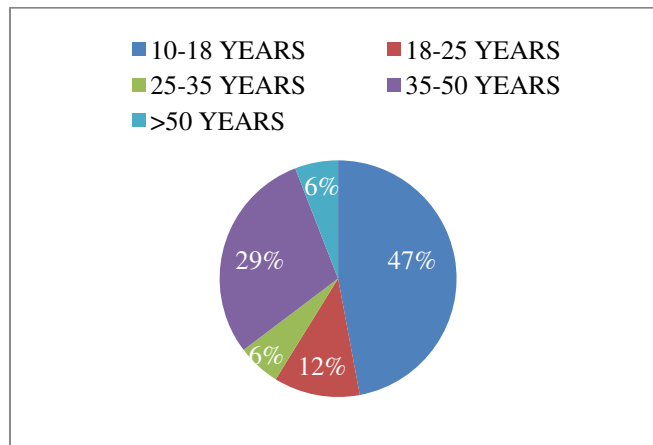
2] Gender distribution:

Image-1 Pie Chart- Gender Distribution



3] Age distribution:

Image-2 Pie chart showing Age distribution.



4] Motor examination:

a) Muscle power:

Table- 1 Lower limb muscle power of 17 patients

MUSCLE	Muscle Power $\geq 4/5$	Muscle Power: $>2/5$	Muscle Power: $\leq 2/5$
Hip	17 (100%)	---	---
Knee	17(100%)	---	---
Dorsiflexors	---	2 (18%)	15 (82%)
Plantarflexors	---	4 (24%)	13 (76%)
Evertors	---	3 (18%)	14 (82%)
Invertors	---	3 (18%)	14 (82%)

Image-3 Pie chart showing Muscle power of Ankle muscles

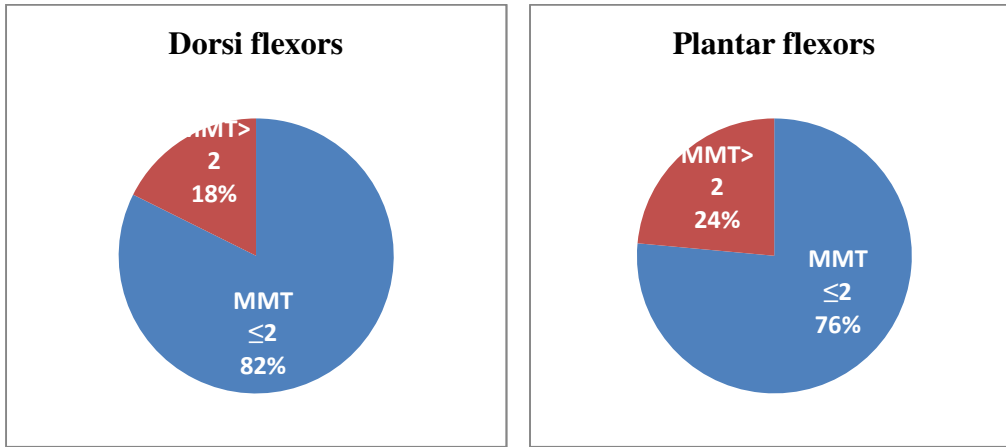


Image-4 Pie chart Showing Muscle power of Subtalar joint muscles

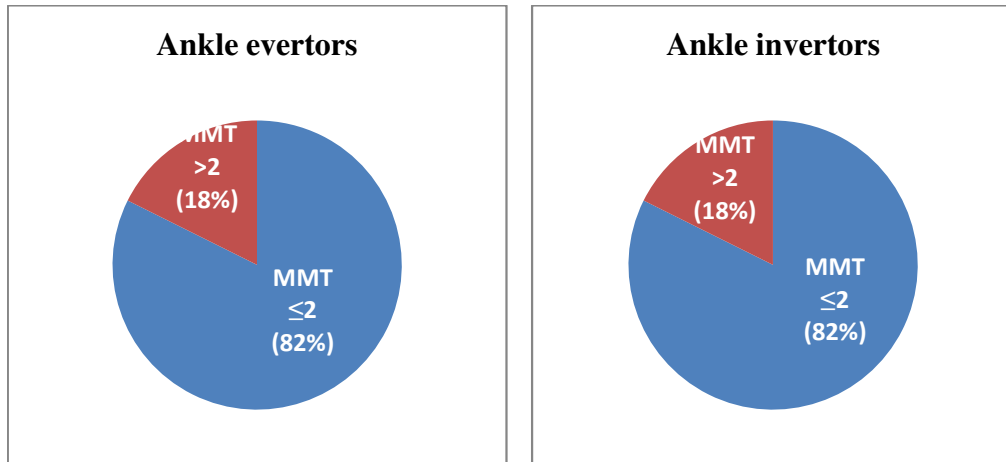
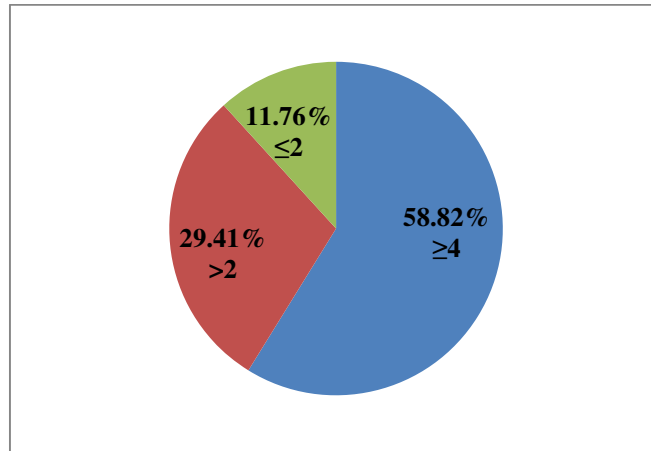


Table- 2 Upper limb muscle power of 17 patients

MUSCLE	Muscle Power ≥4/5	Muscle Power: >2/5	Muscle Power: ≤2/5
Shoulder	17(100%)	-----	---
Elbow	17(100%)	---	---
Wrist flexors	10(58.82%)	05(29.41%)	02(11.76%)
Wrist extensor	10(58.82%)	05(29.41%)	02(11.76%)
Figer flexors	10(58.82%)	05(29.41%)	02(11.76%)
Finger extensors	10(58.82%)	05(29.41%)	02(11.76%)

Image-5 Pie chart Showing Muscle power distribution of Upper limb**b) Reflexes:****Elbow: ++****Knee: ++****Ankle: +/-.****c) Wasting:**7 out of 17 patients had **wasting** in **thenar** and **hypothenar eminence****Image: 6 Wasting of Thenar & Hypothenar Muscles****d) Deformity:****12 out of 17** patients had foot drop present.**5] Sensory examination:**

Deep and Superficial sensations were normal in all patients.

6] Electrophysiological examination:

a) MNCV:

Image: 7 Shows MNCV

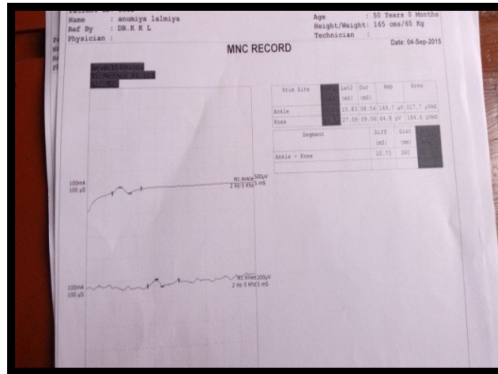


Table – 3 Motor Nerve Conduction Velocity Study:

Nerve affected (mncv)	Demyelinating type	Axonal type	Demyelinating and axonal	Normal	Total patients
Tibial nerve	0(0%)	2(12%)	13(76%)	2(12%)	17
Peroneal nerve	0(0%)	1(6%)	14(82%)	2(12%)	17
Median nerve	2(12%)	2(12%)	7(41%)	6(35%)	17
Ulnar nerve	1(6%)	1(6%)	3(18%)	12(70%)	17

Image-8 Pie Chart Showing NCV Affection of Tibial and Peroneal Nerve

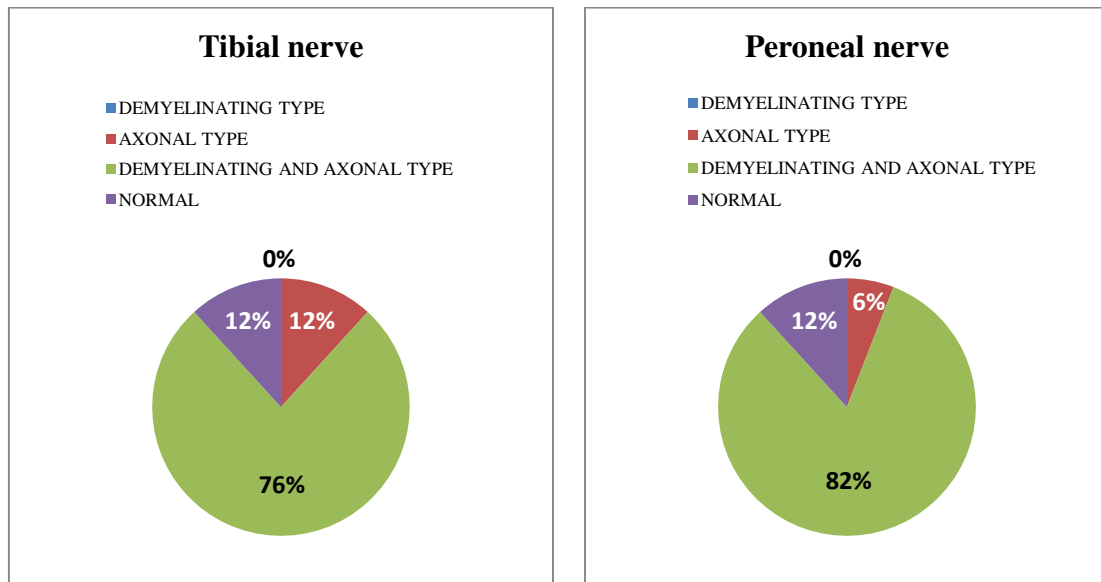
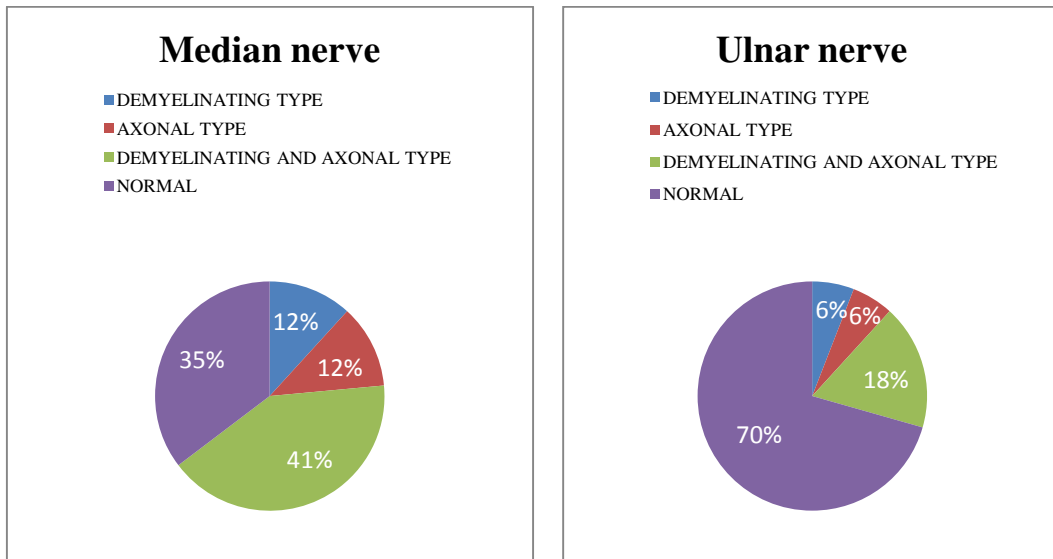
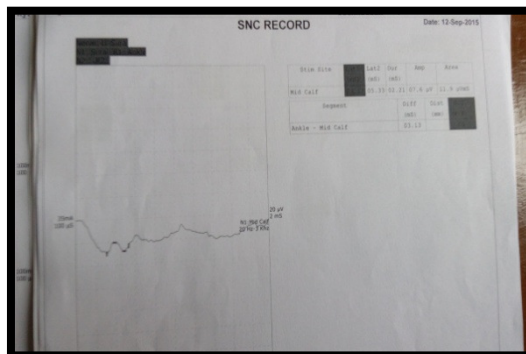


Image-9 Pie Chart showing NCV Affection of Median and Ulnar Nerve**b) SNCV:**

SNCV study of all the patients (Upper limb and Lower limb) is found to be within normal limits. Also, clinically sensory examination of all patients was normal.

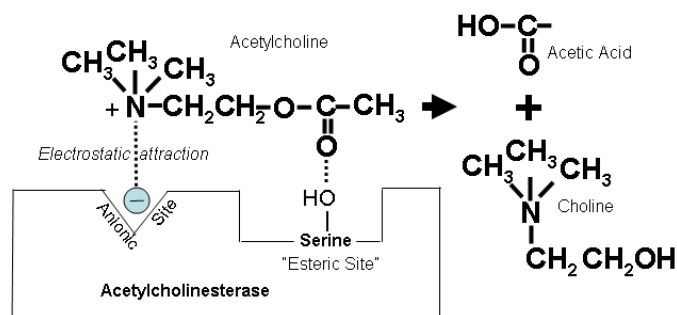
Image-10 Shows SNCV

From Clinical Examinations and Electrophysiological studies of OP induced patients, it is evident that:

1. A total of 17 patients included 9 males and 8 females. **Gender difference was not observed** in disease affection.
2. Electrophysiological evidence showed affection of **Peripheral nerves**.
3. Pure **Motor Polyneuropathy** was **observed** affecting all four limbs (**Lower limb more than Upper limb**).
4. Electrophysiological evidence showed **Demyelinating and Axonal type of Pure Motor Polyneuropathy**.
5. Organophosphate affects **Small and Distal muscles**. (**Foot >Hand**).
6. The **Sensations were not affected**.
7. Electrophysiological evidence showed **SNCV within normal limits**.

Discussion:

Acute pesticide poisoning is a significant cause for morbidity and mortality worldwide, especially in developing countries⁸. Prevention and control measures of non-intentional OP poisoning are different from those required for suicidal poisonings. Therefore, it is important to accurately determine the importance of the problems arising due to OP poisoning, identification of the cases and the morbidity resulting from exposure. Hence this study attempts to figure out clinical and electrophysiological features of patients affected with OP.



OP affects by inhibition of enzyme acetylcholinesterase (AChE) which leads to accumulation of Acetylcholine (Ach) in the body. Most common cause of OP poisoning is exposure to insecticides or pesticides. OP inactivates AChE by phosphorylating the serine hydroxyl residue on AChE. AChE is critical for nerve function, so the irreversible blockage of this enzyme leads to accumulation of Ach and results in muscle overstimulation. Paraoxonase (PON1) is a key enzyme involved in OP poisoning and is found to be important in determining individual's sensitivity of OP exposure.

Signs and symptoms of OP poisoning are: muscle weakness, fatigue, cramps, fasciculation, and peripheral nerve affection. Muscarinic acetylcholine receptors when affected, cause visual disturbances, bronchoconstriction, increased salivation, lacrimation, sweating, peristalsis, and tightness in the chest^{9, 10}.

Here we have considered the effect of OP on peripheral nerves. MNCV study was evident of Symmetrical Axonal and demyelinating type of polyneuropathy. Axonal polyneuropathy was more common than demyelinating type of polyneuropathy. This result is similar to the study carried out by Mohamed B and Abou-Donia, in Organophosphorus ester-induced delayed neurotoxicity, and they found that the lesions were characterized by degeneration of the axons with subsequent secondary degeneration of myelin.

Conclusion:

Organophosphate affects peripheral nerves. It can be concluded from this Electrophysiological study on 17 patients that Organophosphorus poisoning leads to Axonal and Demyelinating affection of Peripheral Motor nerves.

Acknowledgements:

We would sincerely like to thank Dr. Asha Murthy Shah, Dr. B.C. Kaji, Physiotherapy Staff and P.G. Students for helping us in this study.

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