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Dr. Nayer Jamshed is currently working as Assistant Professor in the Department of Emergency Medicine at All India Institute of Medical Sciences, New Delhi. He is interest in Toxicology, Emergency Cardiology, Emergency Ultrasound, High Fidelity Simulation Training Program and Extracorporeal Membrane Oxygenation. He is an active member of the proposed Institute fellowship program in Toxicology. He has published widely in Emergency Medicine, mainly in Toxicology, Infectious diseases, Respiratory, Endocrine and Cardiac Emergencies and has also written chapters on common poisoning for standard post graduate textbooks.


### Diagnosis and Management of PNS involvement in Acute OP Poisoning

Organophosphorous compound (OP) is the most common poisoning in India and Asia-Pacific region. It can manifest as acute cholinergic syndrome, intermediate syndrome and organophosphate induced delayed neuropathy (OPIDN). OPIDN is a neglected and under-reported complication of (OP) poisoning. It is predominantly a motor axonal neuropathy, characterized by wrist and foot drop with minimal sensory loss. Involvement of spinal cord tracts with distal axonopathy can simulate as early presentation of amyotrophic lateral sclerosis. It manifests days to weeks after exposure to an OP agent. OPIDN usually occur after acute cholinergic toxicity, but it can occur in isolation.

OPIDN results because of the inhibition of the enzyme neuropathy target esterase (NTE) also known as lysophospholipase. OPIDN occur when >70% of NTE is inhibited. Aging of phosphorylated enzyme complex is reported to have high likelihood to produce this neurotoxic effect. NTE catalyses breakdown of phosphatidylcholine, which is the major phospholipid of eukaryotic cell membrane. Loss of NTE perturbs membrane phospholipid homeostasis, axonal transport and glial-axonal interaction. Depletion of ornithine decarboxylase was also hypothesized for OPIDN.

Clinical presentation may include muscle cramp, tingling, ataxia, distal numbness, paraesthesia, muscle weakness (lower limb > upper limb) and depressed deep tendon reflexes. High index of suspicion, focussed history, clinical examination and investigations will help in making the diagnosis of OPIDN.

Electrophysiological studies may show reduced amplitude of compound muscle action potential with normal or slightly reduced sensory nerve action potential. Prolonged distal latency with reduced nerve conduction velocity will be seen in most cases of OPIDN. Harvey-Masland test at 3 Hz will not show any waning phenomenon which suggests intact neuromuscular transmission. Needle EMG may reveal denervation potential in muscles like tibialis anterior and quadricep femoris. Nerve biopsy in OPIDN will be suggestive of axonal degeneration with demyelination. NTE assay will show reduced enzyme level.

No specific treatment is available, atropine and oximes have no role in OPIDN. NSAIDS, opioids, amytriptilline, capsaicin, duloxetine and ketotifen can reduce pain. Anticonvulsants like pregabalin, gabapentin and carbamazepine reduce the neurogenic pain and hyperaesthesia. Thiamine, methylprednisolone, vitamin $B_1$, $B_2$, $B_6$, $B_{12}$ were used with variable results. Recently TRPA-1 antagonist and Lapatinib have shown promising results in experimental animals. Physiotherapy plays a substantial role in improving the motor symptoms of OPIDN. Personal care should be taken to prevent injuries and pressure sores. Most of the mild to moderate symptoms of OPIDN improves with time, while prognosis of severe symptoms is guarded.