

Case report:

Toxin induced neuropathy presenting as acute inflammatory demyelinating polyneuropathy

Manthappa M, Raviraja V, Nishant N, Lorraine D

Department of Medicine, Kasturba Medical College,
Manipal University, Karnataka, India

Abstract

An adult male patient presented to us with bilateral symmetric polyneuropathy resembling acute inflammatory demyelinating neuropathy (AIDP). On further questioning, patient gave history of exposure to organophosphate insecticides. Sural nerve biopsy revealed features consistent with toxin induced neuropathy. We are highlighting this case because of its AIDP like presentation which is very rare and also to stress the importance of recognizing exposure to insecticides or other toxic compounds. Detection of toxin or medication induced neuropathy can be an important diagnosis that impacts quality of life by preventing further exposure to the agent and worsening of neuropathy.

Case report

A 32 year old male patient presented to us with insidious onset bilateral lower limb weakness which gradually progressed to involve both upper limbs and neck. The total duration of weakness was 25 days. There was no involvement of cranial nerves, bowel or bladder. He also reported tingling and dragging sensation in the distal parts of both lower and upper limbs. Examination showed findings consistent with bilateral symmetric polyneuropathy. There was universal areflexia. Nerve conduction studies showed bilateral symmetrical motor sensory demyelinating axonal neuropathy. CSF analysis showed albuminocytological dissociation. A diagnosis of acute inflammatory demyelinating polyneuropathy (Guillaine Barre syndrome) was made based on clinical and laboratory findings.

However, the absence of sensory nerve action potential (SNAP) in NCV study was against the diagnosis of AIDP. On further questioning the patient gave history of spraying insecticides (Gyphosate and Dichlorophenoxyacetic acid).

Sural nerve biopsy was done and histopathological study showed predominant axonal pathology, focal neovascularization, with no evidence of vasculitis, and with patchy loss of myelinated fibres. All these findings were highly suggestive of toxin induced neuropathy. Patient was started on supportive measures and was asked to avoid further exposure to insecticides to which he responded slowly with improvement in the power of all four limbs.

Discussion

A variety of drugs and industrial chemicals can cause peripheral neuropathy^[1-3]. Human studies infrequently have associated exposure to environmental sources with peripheral neuropathy. Insecticides have been reported to cause damage to nervous system including peripheral neuropathy like presentation^[4,5]. Unintentional exposure to insecticides may occur while mixing or applying the pesticide or through dermal exposure from those working in the fields shortly after spraying⁶. Organosphates are known to cause nervous system damage including organophosphorous agent induced delayed neuropathy (OPIDN). OPIDN usually starts one to three weeks after exposure and presents usually with distal muscle weakness. Severe cases may progress to involve proximal muscles and upper limbs resembling AIDP which was present in this case. Reports of AIDP after organophosphate exposure have been rare. There is one report of an acute demyelinating motor neuropathy which developed after exposure to merphos⁷. Another study noted clustering of Guillaine Barre Syndrome cases in the area of organophosphate exposure⁸. The mechanism of nerve damage by organophosphates involves inhibition of neuropathy target esterase (NTE) by phosphorylation⁹.

In patients who present with typical features of AIDP, other etiologies with similar presentation should be kept in mind as the treatment differs for AIDP and toxin induced neuropathies. Treatment for AIDP involves use of intravenous immunoglobulin or plasmapheresis whereas the treatment for toxin induced neuropathies involves withdrawal of the offending agent and supportive therapy.

Conclusion

Patients who present with AIDP should be routinely questioned about contact with insecticides or other toxic compounds as the management of these two conditions are different. Detection of toxin or medication induced neuropathy can impact quality of life by preventing further exposure to the agent and worsening of neuropathy.

References

1. Schroder JM, Hoheneck M, Weis J, Deist H. Ethylene oxide polyneuropathy: clinical follow-up study with morphometric and electron microscopic findings in a sural nerve biopsy. *J Neurol.* 1985;232:83-90.
2. Kuzuhara S, Kanazawa I, Nakanishi T, Egashira T. Ethylene oxide polyneuropathy. *Neurology*1983;33:377-80.
3. Bleeker ML. Clinical presentations of selected neurotoxic compounds. In: Bleeker ML, Hansen JA, eds. *Occupational Neurology and Clinical Neurotoxicology.* Baltimore: Williams & Wilkins; 1994:207-234.
4. Schaumburg HH, Spencer PS. Clinical and experimental studies of distal axonopathy- A frequent form of brain and nerve damage produced by environmental chemical hazards. *Ann NY Acad Sci.* 1979;14-29.
5. Schaumburg HH, Spencer PS. The neurology and neuropathology of the occupational neuropathies. *J Occup Med.* 1976;18:739-42.
6. Metcalf, D.R., Holmes, J.H. VII: Toxicology and physiology. EEG, psychological and neurological alterations in humans with organophosphorous exposure. *Ann. NY Acad. Sci.*160:357,1969.
7. Fisher, J.R.: Guillain-Barre syndrome following organophosphate poisoning. *JAMA,* 238:1950, 1977.
8. London L, Bourne D, Sayed R, Eastman R. Guillain-Barre syndrome in a rural farming district in South Africa: a possible relationship to environmental organophosphate exposure. *Arch Environ Health.* 2004 ;59:575-80.
9. Johnson, M.K.: Receptor or enzyme: the puzzle of NTE and organophosphate-induced delayed polyneuropathy. *Trends Pharmacol. Sci.,* 1987;8:174

Corresponding author:

Manthappa M, Associate professor, Department of Medicine,

Kasturba Medical College, Manipal, Manipal University, Karnataka-India

Email : manthappa@yahoo.com