Occupational Medicine Forum

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Occupational Medicine Physician's Guide to Neuropathy in the Workplace Part 3: Case Presentation

This is the last of three Forum articles on Neuropathy in the Workplace, by Jonathan S. Rutchik, MD, MPH. Part I focused on the History and Physical, along with the differential diagnosis and laboratory and risk assessment; Part II focused on EMG, Crytogenic, and Toxic Neuropathy. Dr Rutchik is a private practitioner affiliated with the University of California at San Francisco Medical School in the Division of Occupational Medicine. He is a board certified physician in both neurology and occupational medicine. Comments may be sent to jsrutch@neoma.com.

A 52-year-old auto mechanic presents with burning feet for 5 years. He did report a medical history that included mild hypothyroidism and a 12 pack of beer every 3 weeks for 10 years. He has a coworker with a similar problem. He reported worsening for 3 years but complained of little motor weakness. Examination revealed normal motor power with sensory deficits to pin prick, vibration, and temperature in his lower extremities. Reflexes were diminished but symmetrical. Hair was present on the toes and no nail changes were apparent. Nerve conduction velocity testing revealed absent sensory amplitudes and the needle electromyography (EMG) revealed denervation in lower extremities.

The patient worked in a large automotive open building with 17 coworkers; he was the worker with the most tenure. He used a solvent from small spray cans, to clean and degrease engine parts for his initial 10 years. At some point, his company switched to using 55 gallon drums for the solvent along with a hand pump. This would often leak and spill. He often used the solvent on his clothes to remove grease. He was in an open work area that abutted where workers would dispense this product. There was a nearby fan that ventilated the room. A coworker was identified as having a neuropathy that was associated with this solvent chemical. At that point, the company changed back to aerosol cans to dispense the solvent. He had been using this method for the last 3 years.

He reported that there were two lunchrooms, one inside building. He did acknowledge using personal protective equipment that included latex glove and then Vicryl gloves.

Symptoms began while the 55 gallon drum was being used, but worsened when drum changed to aerosol. The solvent was noted to be a spray that quickly dried to become a powder, was odorous, and had made him dizzy on occasion.

The industrial mechanic presented above was exposed to n-hexane. His medical history is complicated by both alcohol and thyroid illness, both of which can contribute to peripheral neuropathy.

Twenty percent of n-hexane inhaled is absorbed in alveoli. There is both GI and dermal absorption and metabolism in both is hepatic. Initially, the metabolite, 2-hexanol leads to euphoric and narcotic effects. Its biological exposure index is 2,5 hexanedione in urine which is known to cross links axonal filaments and interrupts transport.

N-hexane excretion is 10% unchanged in the lung and 90% in urine. Sensorimotor polyneuropathy from prolonged and repeated exposures, characterized by coasting and associated optic neuropathy have been described in the literature. Central nervous system damage and Parkinsonism have been reported.

Unfortunately, he had not been tested and was examined by this neurologist 6 months postremoval from work. Industrial hygiene assessment was not performed by the employer.

For this patient, the plan was to remove him from work and observe. His EMG nerve conduction velocity results (symmetrical reduction of sensory amplitudes) were consistent with distal symmetrical neuropathy with possible contributions from thyroiditis, alcohol, and n-hexane exposure. The fact that his colleague had been diagnosed also with n-hexane induced neuropathy further supported the contribution from the industrial agent. All other testing was normal. Repeat testing in 6 months to 1 year was not performed. Treatment has included capsaicin ointment, tricyclic antidepressants and anti-inflammatory medication, as well as alcohol abstinence and thyroid medications. He continued to report burning in his feet and could not work.

Conclusion

Toxic peripheral neuropathy is rare compared to more common causes. Removal from the offending agent leads mainly to improvement. It is important to note, however, that exposure does not prove causation. Synthesis of an entire medical history is required along with assessment by laboratory testing for chronic medical illness that may contribute or be the underlying cause. As a significant number of neuropathies are inherited, family history must be considered. Once neuropathy is confirmed by electrodiagnostic testing, exposure must be supported by body burden assessment, if possible, and a biologically plausible argument considering conditions of exposure, chronology of symptom presentation, consistency of physiologic mechanism, dose response, other organ involvement and improvement or lack of worsening with removal or exposure over time. As many neurotoxic agents involve the central nervous system, this must be considered also. Case reports, series, and epidemiologic studies as well as animal studies can be helpful to match exposure dose and duration with an agent that may be responsible. As prevention is most important, employee health practices must be routinely updated and openly discussed in the workplace.

References

1. Albers JW, Bromberg MB. Chemically induced toxic neuropathy. In: Rosenberg NL, ed. *Occupational and Environmental Neurology*. Chapter 8. Boston: Butterworth Heinmann; 1995.
2. Albers JW. *Toxic Neuropathies. Clinical EMG*. AANEM; 1994: AANEM Course 441.
3. Aminoff MJ, Greenberg DA, Simon RP. Disorders of somatic sensation. In: *Clinical Neurology*. Chapter 6. New York: Lange Medical Books; 2005.
4. Berger AR, Schaumberg HH. Disorders of the peripheral nervous system. In: Rosenstock L, Cullen MR, eds. *Textbook of Clinical Occupational and Environmental Medicine*. Philadelphia: Saunders; 1994:482–513.
5. 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.
6. Bleeker ML, Albers JW, Aminoff MJ, Ford DP, Lindgren KN. Continuum: lifelong learning in neurology. *Neurotoxicology*. 1999;20:9–50.
7. Feldman RG, White RF. Role of the neurologist in hazard identification and risk assessment. *Environ Health Perspect.* 1996;104(Suppl 2):227–237.
8. Feldman RG. Effect of toxins and physical agents on the nervous system. In: Bradley WG, Daroff RB, Fenichel GM, et al., eds. *Neurology in Clinical Practice*. Boston: Butterworth-Heinemann; 1991:1185–1207.
9. Feldman RG. Neurotoxic effects of trichloroethylene in drinking water. In: Isaacson RL, Jensen KF, eds. The *Vulnerable Brain and Environmental Risks: Toxins in Air and Water*. Vol 3. New York: Plenum Press; 1994.
10. Feldman RG. *Occupational and Environmental Neurotoxicology*. Philadelphia: Lippincott-Raven;1999.
11. Feldman RG. The recognition and differentiation of neurotoxic and non-neurotoxic syndromes. In: Chang LW, Slikker W, eds. *Neurotoxicology: Approaches and Methods*. San Diego: Academic Press; 1995:689–694.
12. Frumkin H, McCunney RJ, Barbanel C. Health effects of common substances. In: MccCunney RJ, ed. *A Practical Approach to Occupational and Enviromental Medicine*. 2nd ed. Boston: Little Brown; 1994.
13. Grogan PM, Katz JS. Toxic neuropathies. *Neurol Clin.* 2005;23:377–396.
14. Hoitsma E, Reulen JP, de Baets M, Drent M, Spaans F, Faber CG. Small fiber neuropathy: a common and important clinical disorder. *J Neurol Sci.* 2004;227: 119–130.
15. Kimura J. Polyneuropathies. In: *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 2nd ed. Philadelphia: FA Davis;1989:462–481.
16. Kinsella LJ, Berger AR. Neurotoxicology in the Workplace: How to Evaluate Patients with Suspected Neurotoxic Peripheral Nerve Disease. AANEM. Issues and Controversies in the Injured Worker. AANEM; 2005: AANEM Course C.
17. Overbeek BU, van Alfer N, Boc J, Zwarts MJ. Sural radial amplitude ratio: reference values in healthy subjects. *Muscle Nerve*. 2005;32:613–618.
18. Rowland LP, ed. *Merritt's Textbook of Neurology*. 8th ed. Philadelphia: Lea & Febiger; 1989.
19. Rutchik J. Toxic neuropathy. In: *Emedicine, Neurology.* (An internet medical textbook). 2001 and 2005. Available at: www.emedicine.com. Accessed December 1, 2008.
20. Rutchik J, Wittman R. Solvent neurotoxicity. In: Phillips S, Krieger G, eds. *Clinics of Occupational and Environmental Medicine*. Philadelphia: WB Saunders; 2004.
21. Rutkove SB, Kothari MJ, Raynor EM, Levy M, Fadic R, Nardin R. Sural radial amplitude ratio in the diagnosis of mild axonal plyneuropathy. *Muscle Nerve*. 1997;20:1236–1241.
22. Schaumberg HH, Spencer PS. Clinical and experimental studies of distal axonopathy—a frequent form of brain and nerve damage produced by environmental chemical hazards. *Ann N Y Acad Sci.* 1979;329:14–29.
23. Schaumberg HH. Human neurotoxic disease. In: Spencer P, Schaumberg HH, eds. *Experimental and Clinical Neurotoxicology*. 2nd ed. New York: Oxford University Press; 2000.
24. Sullivan JP, Logigian EL, Kochanan N, Herrmann DN. Yield of the sural/radial amplitude ratio versus the medial plantar nerve in sensory neuropathies with a normal sural response. *J Clin Neurophysiol*. 2008;25:111–114.