Chemical Injury Reports

Lead Poisoning in an Oil-Pipeline Maintenance Worker

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Background: Occupational lead poisoning has been documented in lead battery recycling workers and tile factory workers in Taiwan. Another potential source of lead poisoning occurs in oil-pipeline maintenance workers.

Problem: A 20-y-old male Taiwanese went to the emergency room and complained of abdominal fullness, diffuse abdominal pain, and postprandial vomiting for 3 d.

Exposure: The patient had been employed as a maintenance worker at a petroleum company for 9 mo. He used an acetylene torch to cut and connect the oil pipeline, and he used no respiratory protection apparatus. Red coatings were noted over the oil pipeline.

Objective measurements: Examination revealed diffuse abdominal tenderness. Laboratory tests revealed microcytic anemia (Hgb: 8.0 g/dl; Hct: 28.0; MCV = 85.7); and abnormal liver function (AST: 116; ALT: 339; GGT: 213). Amylase and lipase were within normal limits (i.e., 64 and 80 U/l). Basophilic stippling of RBCs was noted to be increased in the peripheral blood smear. KUB and lower GI series revealed dilatation of bowel loop. Echogram of liver showed no abnormality. The blood lead level was 105.3 µg/dl, and the urine δ-ALA was 33.6 mg/d.

Results: The patient was treated immediately with EDTA 1 500 mg i.v. for 1 d, after which the following therapy was administered: BAL 75 mg i.m. q 4 h for 2 d and EDTA 1 500 mg i.v. for 5 d. After 2 d of EDTA therapy, the blood lead level declined to 17.5 µg/dl. The blood lead level, after 5 d of EDTA treatment, was 37.5 µg/dl.

Collection of 24-h urine for measurement of lead excretion failed because the patient did not cooperate. Six mo after medical removal, the patient's blood lead level was 38 µg/dl. Blood hemoglobin returned to 16.1 g/dl. Normal liver function was noted. Motor nerve conduction velocity (NCV) showed delayed response and slight prolongation of distal latency on both median nerves. Sensory NCV examination showed low amplitude of the action potential of the right radial nerve.

Conclusion: Lead poisoning was confirmed in this pipeline thermal cutter. No sequela was noted after treatment, except for mild damage to the nervous system. Lead-containing red paints are still used widely as coating material in industries, especially in developing countries. Surveillance of lead poisoning in maintenance workers who burn or cut metal structures coated with lead-containing paints should not be neglected.

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Neuropsychiatric Sequelae Following Chemical Exposure

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Background: Patient GB worked at a large chemical company during which time (1969–1991) he developed and tested new chemical products. Beginning in 1986, he experienced onset of somatic, cognitive, and personality changes.

Problem: A neurobehavioral evaluation was requested following a diagnosis of hepatic injury with decreased cognitive function and psychiatric symptomatology (i.e., auditory and visual hallucinations).

Demographics: At the time of the evaluation, GB, a white right-handed male, was age 42 y. He was high-school educated, and transcripts revealed that he graduated third in a class of 45, with a GPA of 3.47.

Setting: Work setting was a wet-chemical lab of a large product-development plant located in southeastern Pennsylvania. The only protective equipment worn regularly was gloves.

Type of chemical: GB reported exposure to more than 100 chemicals, many of which were in the developmental phase. He noted the most frequent work occurred with toluene, as well as with additional chemicals that included allyl chloride, methyl chloride, acrylonitrile, and carbon tetrachloride.

Results: A neuropsychological evaluation was conducted in May 1991, with a follow-up evaluation in May 1992. At the first testing, GB was not taking any prescription medication, but at the second evaluation GB was taking Depakote (25 mg three times a day). Results revealed significant deficits across a number of cognitive domains; the majority of his scores on memory measures fell 2 standard deviations below the mean for a man of his age and education level. Impairments were also noted on measures of mental flexibility, attention/concentration, and psychomotor speed. Comparison of the two evaluations revealed minimal fluctuation in test performance. Auditory event-related potentials were obtained at the follow-up evaluation. Inspection of the wave-forms revealed morphologic abnormalities of the P300 component (the positive wave appearing approximately 300 msec after meaningful events). P300 latency was increased more than 2 standard deviations from that seen in normal controls. DSM-III-R Diagnosis of Organic Hallucinations (1991–1992), Organic Mood Syndrome (1991), and Organic Personality Syndrome were assigned on the basis of the Structured Clinical Interview for DSM-III-R.[1] Psychotic symptoms included visual and auditory hallucinations and overvalued referential and persecutory beliefs. Mood-related symptoms included depression, diminished interest and pleasure, appetite and weight loss, sleep disturbance, psychomotor retardation, feelings of worthlessness, recurrent thoughts of death, and concentration difficulty. Personality symptoms included uncharacteristic affective instability, outbursts of anger, suspiciousness, and social withdrawal. The Personality Disorders Examination[2] was administered to an informant, using a time frame from the age of 15 y until employment. The informant (GB's sister-in-law) attended school with GB and maintained close contact with him until his entry into the military after high school. No symptoms indicative of premorbid personality disorder were reported, and GB

was characterized as stable, was recognized for his scholastic ability, was well liked by his peers, was socially active and had close friendships, and was employed consistently.

Imaging: A SPECT scan performed in 1989 revealed decreased blood flow to the frontal regions, primarily the left posterior frontal and anterior temporal regions. In 1991, a CT and MRI were consistent with "mild frontal atrophy."

Summary: Whereas we cannot unequivocally eliminate other causes for the neuropsychiatric deficits noted in this case, the fact that no air samples were gathered at the worksite and no blood or urine measures were obtained at the time of exposure leads us to conclude that there is compelling evidence that this patient's workplace exposure eventuated in cerebral damage. The cognitive, neurophysiological, and imaging results were consistent with what has been reported in the literature for persons with chemical exposure.[3-5] The absence of a preexposure psychiatric history or personality disorder leads us to the assumption that the exposure also precipitated the hallucinatory experiences. Several reports in the literature have described hallucinations that follow workplace exposure to solvents, as well as solvent abuse.[6-8] GB stated that his exposure to toluene was frequent and chronic (i.e., "practically took baths in it"). Whereas we cannot determine which chemical(s) were responsible for the neuropsychiatric deficits noted in this case, the psychotic symptoms experienced by GB were consistent with what has been reported after chronic exposure to organic solvents.

Conclusion: The psychiatric features associated with chemical exposure may range from altered mood to full-blown psychoses. Structured psychiatric and personality interviews would appear warranted in cases of suspected chemical injury to document the extent of past and current psychiatric disturbance and to determine the proper treatment intervention.

References

1. Spitzer R, Williams J, Gibbon M, et al. Structured Clinical Interview for DSM-III-R (SCID). New York: Biometrics Research Unit, NYS Psychiatric Institute; 1989.
2. Loranger A. Personality Disorder Examination (PDE). Yonkers, NY: DV Communications, 1988.
3. Morrow L, Ryan C, Hodgson M, et al. Alterations in cognitive and psychological functioning after organic solvent exposure. J Occup Med 1990; 32:444–50.
4. Morrow L, Steinhauer S, Hodgson M. Delay in P300 latency in patients with organic solvent exposure. Arch Neurol 1992; 49:315–20.
5. Callender T, Morrow L, Subramanian K, et al. Three-dimensional brain metabolic imaging in patients with toxic encephalopathy. Environ Res 1993; 60:295–319.
6. Byrne A, Kirby B, Zibin T, et al. Psychiatric and neurological effects of chronic solvent abuse. Can J Psychiat 1991; 36:735–38.
7. Goldbloom D, Chouinard G. Schizophreniform psychosis associated with chronic industrial toluene exposure: case report. J Clin Psychiat 1985; 46:350–51.
8. Tarsh M. Schizophreniform psychosis caused by sniffing toluene. J Soc Occup Med 1979; 29:131–33.

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Editorial Comments

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One of the more controversial and intriguing issues in environmental health is the nature and etiology of neurological and/or psychiatric symptoms and syndromes that arise in individuals with chemical exposures.[1] Even when the exposure is within a toxic range, the appearance of behavioral change or mood instability often leads observers to shift set from a medical to a psychogenic interpretation, without a strong rationale. In contrast, in psychiatric practice outside the controversies surrounding chemicals, it is standard of care to assess a given patient for possible "organic" factors in any psychiatric presentation.[2,3] Elements of a good clinical history that raise suspicion of organic factors include good premorbid psychological and social functioning; lack of family history of similar problems; later age of onset of the condition; evidence of a potentially causative event preceding onset (e.g., head trauma with loss of consciousness); medical condition (e.g., multiple sclerosis, stroke, epilepsy); or medication/drugs (e.g., steroids, certain antihypertensive agents, stimulants, alcohol). Personality traits are normally stabilized by age 30 y; a dramatic change in behavior can signal a serious, nonpsychogenic etiology. The case report by Morrow et al. illustrates these points, as the authors have examined premorbid records and found evidence of marked alteration in behavior beginning relatively later in life. Finally, this case also highlights another important but generally neglected issue (i.e., the role of individual differences in susceptibility). Use of some of the neuroimaging tools employed in the above case may help us expand our understanding of how the same exposure may lead to clinical illness in one person, but not in another.

References

1. Committee on Neurotoxicology and Models for Assessing Risk. Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council. Environmental Neurotoxicology. Washington, DC: National Academy Press, 1992.
2. Yudofsky SC, Hales RE, Eds. Textbook of Neuropsychiatry. Washington, DC: American Psychiatric Press, 1992; 2nd ed.
3. Mesulam MM. Principles of Behavioral Neurology. Philadelphia, PA: F. A. Davis Co., 1985.

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Growing Up Exposed: Adult Manifestations of Protracted Childhood Insecticide Exposure

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Background: This woman grew up on a date and grape farm, which adjoined a mosquito abatement facility on the high California desert. At this location, insecticides were stored, mixed, and loaded into spray trucks, and the spray equipment and trucks were washed out into her living and play areas.

Problem: Adult recognition of symptoms consistent with chronic exposure to insecticides. Symptoms included impaired mental ability, especially for timed functions; impaired recall memory; extreme fatigue; irritability; and mood shifts.

Demographics: The 41-y-old college graduate—artist and mother—had never smoked.

Setting of exposure location: The family farm adjoined downwind and water the county mosquito control unit in Coachella Valley, California.

Type of chemicals: Insecticides—especially organophosphates, carbamates, and chlordane—were used in mosquito control.

Results: Key findings were adult onset or recognition of decreased mental ability, particularly speed of functions; difficulty recalling recently presented information; and extreme fatigue following minimal exertion. She also reported that irritability and mood shifts troubled her. Profile of Mood States score was 94 (expected: 23.3 ± 33.7). Physical findings revealed normal grip strength and vibration sensation.

Objective measurements were abnormal visual fields with bilateral scotomata and abnormally prolonged blink reflex latency (16.1 ms left and 15.5 ms right). Culture Fair, block design, and digit symbol scores were below those expected for a woman with 17 y of education. Delayed visual two-choice reaction time at 609 ms (500 ± 63), delayed trail-making A at 45 s and B at 132 s, and elevated numbers errors (12) on finger writing were abnormal.

Imaging: Normal chest x-rays.

Summary: Impaired performance of timed operations led to a diagnosis of neurobehavioral impairment, which, combined with the high frequency of symptoms and upsets of mood, is consistent with chemical encephalopathy. Severe memory impairment and difficulty