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Epidemiologic Notes and Reports

Lead Intoxication Associated with Chewing Plastic Wire Coating — Ohio

In December 1991, a venous blood lead level (BLL) of 50 µg/dL was detected in a 46-year-old Ohio man during a routine pre-employment examination. He was referred to a university-based pharmacology and toxicology clinic for further evaluation; clinic physicians investigated the case. Although a repeat BLL obtained 1 month later was 51 µg/dL, he reported no exposure to known sources of lead during the interim. However, he reported numbness of his fingers and palms, tinnitus, and a possible decrease in his ability to perform basic arithmetical calculations.

A comprehensive occupational and environmental history obtained at the time of the second BLL test revealed no apparent source of his lead exposure. Although he had been employed for approximately 20 years as a microwave technician during military service and while employed at a television station, he reported no history of exposure to lead from soldering or welding. He had no activities or hobbies associated with exposure to lead or lead products, no previous bullet or birdshot wounds, and he denied drinking illicitly distilled alcohol or using lead additives in his car.

His residence was built in 1974 (after lead was banned from use in residential paint)*, and household water was obtained from a well. In January 1992, blood lead testing of family members revealed levels of 5 µg/dL for his wife and <5 µg/dL for his 17-year-old child. His only medication was ranitidine†, which he had used for the previous 1½ years for "indigestion." He reported occasional cigarette smoking.

Although results of a neurologic examination were normal, neuropsychiatric testing on March 13 demonstrated mild memory deficits, as evidenced by abnormalities on verbal and figural memory tests. Because of these abnormalities, beginning March 13, he was treated for 19 days with dimercaptosuccinic acid (DMSA), an oral chelating agent, and on April 4, his BLL had decreased to 13 µg/dL. However, BLLs on May 15 and July 23 were 49 µg/dL and 56 µg/dL, respectively.

*16 CFR §1303.2. Ban of lead-containing paint and certain consumer products bearing lead-containing paint.
†Ranitidine alters gastric acidity, which theoretically can influence gastrointestinal absorption of lead.

Lead Intoxication — Continued

During a July 1992 follow-up clinic visit, he mentioned that for approximately 20 years he had habitually chewed on the plastic insulation that he stripped off the ends of electrical wires. Samples of the copper wire with white, blue, and yellow plastic insulation were obtained and analyzed for lead content. The clear plastic outer coating (present on all colors of wire) and the copper wire contained no lead; however, the colored coatings contained 10,000–39,000 μg of lead per gram of coating.[§] On receipt of these results, he was instructed immediately to discontinue chewing the wire coating.

In January 1993, when his BLL was 24 μg/dL, he reported subjective improvement in his symptoms; follow-up neuropsychiatric testing is pending.

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Editorial Note: This report likely represents the first documented case of lead poisoning following ingestion of lead as a consequence of chewing on plastic wire coatings. Plastic coatings previously have been associated with lead exposure in the burning of lead-containing plastics during repair of a storage tank (*1*), the production of plastics (*2,3*), and the manufacture and use of stabilizers and pigments in the plastics industry (*4*). Although lead exposure also can occur among workers who burn the plastic coating off copper wire to recycle the copper, lead intoxication by this route has not been reported (*5*).

Lead compounds may be employed in the production of colored plastics (in which lead chromates are used as pigment) and in the manufacture of polyvinyl chloride (PVC) plastics (in which 2%–5% lead salts [including lead oxides, phthalate, sulfate, or carbonate, depending on the desired quality of the final product] are used as stabilizers). Although environmental regulation has reduced considerably the amount of lead used in the United States in the manufacture of PVC plastics, manufacturers of electrical wire and cable continue to produce PVC stabilized and/or pigmented with lead compounds (*6*).

More than 573,400 U.S. workers are employed in occupations involving electrical work. Among these workers, potential for excessive exposure to lead may result from inhalation of fumes generated during lead soldering (*7*). Because the plastic coating from wires is usually removed by mechanical stripping, ingestion of lead from these plastic coatings is probably uncommon. Nonetheless, the findings in this report remind occupational and other health-care providers of the need to be aware of this potential source of lead exposure. In addition, workers should be warned of the potential hazard of chewing plastic coatings or other plastic products that may contain lead.

References

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[§]Samples were analyzed using graphite furnace atomic absorption spectroscopy, following dissolution of the plastic coating in tetrahydrofuran.

Lead Intoxication — Continued

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Current Trends

Arboviral Diseases — United States, 1992

During 1992, health departments from 23 states reported to CDC 45 cases of arboviral encephalitis in humans and 97 in horses. An additional four states reported detection of arboviral activity in bird and mosquito populations. Unlike 1990 and 1991, when three St. Louis encephalitis (SLE) epidemics and an eastern equine encephalitis (EEE) epizootic occurred, during 1992, no focal outbreaks of arboviral disease were reported. This report summarizes information regarding arboviral encephalitis in the United States during 1992.

SLE. During 1992, 14 sporadic SLE cases occurred in Texas (12 cases) and California (two) (*1*)—a substantial decrease from 1990 and 1991 (247 and 78, respectively), when SLE cases were at their highest level since 1976.

LaCrosse encephalitis (LAC). During 1992, 29 cases of LAC encephalitis were reported from Illinois (seven cases), Ohio (six), West Virginia (six), Wisconsin (four), Minnesota (three), and North Carolina (three). This is the lowest number of LAC cases reported since surveillance began in 1964.

EEE and Western equine encephalitis (WEE). During 1992, Florida and Massachusetts each reported one case of EEE. Because of isolation of EEE virus from *Aedes albopictus* during 1991 in Florida, human case surveillance was intensified at five regional medical centers. From May through September 1992, 357 cerebrospinal fluid samples were collected from persons with symptoms suggestive of meningitis or encephalitis. None had EEE-specific immunoglobin M antibody. In 1992, 88 cases of EEE in horses were reported from Florida (54 cases), Georgia (nine), Virginia (nine), Mississippi (four), South Carolina (four), North Carolina (three), Texas (two), Arkansas (one), Kentucky (one), and Michigan (one). Although no cases of WEE were reported in humans, nine cases of WEE in horses were reported during 1992: Idaho (two cases), Missouri (two), Oklahoma (two), Colorado (one), South Dakota (one), and Utah (one).

Enzootic arbovirus activity. In 1992, 28 states conducted arboviral surveillance using virus isolation or antigen detection in captured mosquitoes or viral-specific antibody assays in sentinel or wild birds. Enzootic arboviral activity was reported from 16 states: EEE (Delaware, Florida, Georgia, Massachusetts, Michigan, New Jersey, North Carolina, Ohio, and South Carolina), SLE (Arizona, California, Illinois, Michigan, and Texas), WEE (Arizona, California, Colorado, Nevada, and Utah), and LAC (Illinois).

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