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LETTER TO THE EDITOR

Lead poisoning in two children with Landau-Kleffner syndrome

To the Editor:

Landau-Kleffner syndrome (LKS) or acquired epileptic aphasia (AEA) is a rare childhood neurological disorder described in 1957 as a regression in language development usually by 3 years of age, accompanied by a convulsive disorder with a typical pattern of continuous slow wave disturbances during sleep (CSWS) in the peri-sylvian fissure region.^{1–3} The medical literature does not mention a vulnerability of children with LKS to environmental toxins. We describe here two cases of lead poisoning in children with LKS.

Case 1

A 4-year-old boy with mild speech regression was referred to the Pediatric Environmental Health Center (PEHC) at Children's Hospital Boston for an elevated blood lead level (BLL) to 22 mcg/dL discovered during a neurological evaluation for new onset, tonic/clonic seizures. The mother observed that he was hyperactive and irritable with a low frustration threshold. Preschool teachers reported outbursts of anger – including hitting, spitting, snorting, throwing objects, and screaming – making other children afraid to be around him. He constantly put objects in his mouth, including bark, mulch, rocks, sand, dirt, and toys. An electroencephalogram (EEG) showed a left occipital spike abnormality; a brain electrical activity mapping (BEAM) study showed frequent left occipital spikes and spike slow-wave tracings during sleep and right mid-temporal and central paroxysmal theta in waking, consistent with LKS. The family's apartment had lead paint near windows and on a porch; his day care center also had lead violations. Despite subsequent lead abatement the BLL rose as high as 35 mcg/dL.

Case 2

A 5-year-old girl with LKS, hyperactivity and pica was referred to the PEHC for a BLL of 38.4 mcg/dL. Over her first 3 years, she experienced language regression, became less engaged, and developed hyperactivity, tantrums, parasomnias, and pica. There was no obvious seizure activity, though occasionally parents noticed brief staring episodes. At 3 years, EEG and BEAM studies showed frequent hemispheric discharges with sleep potentiation in left lateral frontal and left lateral parietal-temporal regions, giving her a diagnosis of LKS and electrical status epilepticus of sleep (ESES). Examination showed a difficult to engage child who did not make eye contact, made nonsensical noises and occasional words, and mouthed multiple items in the exam room. The family's 1880s vintage home had multiple areas of chipping original leaded paint and peeling wallpaper.

Developmental implications of having both LKS and lead poisoning are unknown; one could speculate an added risk of poor language and adverse developmental and cognitive outcomes. Older children with pervasive developmental disorders (PDDs) or autism can have lead poisoning.⁴ It may be that children with LKS retain oral exploratory behaviors and pica well into their school-aged years, also placing them at continued risk. We believe that childhood lead poisoning screening recommendations for state health departments should be expanded to include older children who have developmental disabilities and persistent oral behaviors. We recommend that physicians caring for children with LKS living in residential areas known to have endemic lead contamination monitor their BLL periodically as long as they retain pica habits.

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Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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