Letters to the Editor

Classic Picture of Lead Neuropathy

We read with interest the article by Mishra *et al.*(1). While going through the article without looking at the title, to me, it was a classic story of lead neuropathy in a 6½ -year-old girl, as also reported earlier, even in early infancy(2). The points in favor of lead neuropathy in the case are as follows:

1. Bilateral symmetric onset, progressive distal upper extremity weakness and wasting, involving the small muscles of the hand and forearm muscles with sparing of other limbs. There is no mention of power of individual muscles on examination. So one is not sure about wrist extensor power, whose weakness is usually an early feature along with weakness of extensor of ring and middle fingers in lead neuropathy(3).
2. Pure motor involvement without any sensory affection both from clinical and electrophysiologic angle (though no specific mention of sensory nerve conduction study is available in the report)(4).
3. Nerve conduction velocities of both median and ulnar nerves were within normal limits which is a typical feature of lead neuropathy. Though segmental demyelination is seen in the affected nerves pathologically, absolute conduction velocities remain within normal limits. However, the velocities are significantly reduced in the lead exposed patients when compared with those not exposed. When axonopathy is prominent, the weakness becomes more severe concomitant with a lowering of CMAP amplitude(4). In the case reported there is no mention of CMAP amplitude(1), which may be low.
4. Electromyography suggests that the patient had progressive neuropathy and not anterior horn cell disease. There were fibrillations and positive sharp waves ('sharp' word is however missing) without fasciculations (at least not mentioned). There was discrete single motor unit activity in the hand muscles. All the above parameters suggest active ongoing denervation changes without any significant re-innervation(4).
5. Muscle biopsy showing neurogenic atrophy without any regenerating or hypertrophic fibers or any pseudo-myopathic pattern may point to the progressing axonopathic involvement, rather than chronic anterior horn cell disease.
6. Serum lead level on two occasions, were high (91 mcg/dL and 76 mcg/dL). The upper allowable limit is 10 mcg/dL as per Center for disease control and World Health Organization (5,6). Those levels are more than adequate to produce neuropathy.

One wonders why this child did not have abdominal pain, raised intracranial pressure, cognitive abnormalities, anemia or basophilic stippling of RBC. But none of those clinical features are invariably present in a chronic setting(3,4). Erythrocytic Zn-protoporphyrin and urinary excretion of delta-aminolevulinic acid are more sensitive tests in detecting physiologic abnormalities produced by lead excess than basophilic stippling of RBCs(3).

There are several points against Hirayama's disease including the age, sex, onset and distribution of weekness. Also, there is no characteristic worsening of weakness when exposed to cold. MRI spine did not show any localized atrophy of the anterior aspect of C7, C8, T1 segment of the spinal cord. EMG and muscle biopsy findings were also not absolutely characteristic.

Overall the body of evidence suggests the diagnosis in the case to be lead neuropathy, and not Hirayama's disease. I request the authors now to contact the affected child and her parents to look for the factors for lead excess in her. May be in that process many others in the community can be detected to have lead toxicity.

Debabrata Ghosh,
Associate Professor, Pediatrics,
Advanced Pediatric Center,
PGIMER,
Chandigarh, India.
E-mail: ghoshdebabrata@hotmail.com

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Convulsions and Retinal Hemorrhages

Retinal hemorrhages in children are important clinical findings. They may be associated with trauma or systemic diseases(1). In the absence of bleeding disorders, retinal hemorrhage most often result from non - accidental injuries such as direct streaking of the head, severe shaking causing acceleration - deceleration and thoracic compressions. Sever contraction of chest muscles in convulsion, crying or vomiting may cause retinal hemorrhages(2,3).

The prevalence of retinal hemorrhage after convulsions is unclear, and there are few and small studies in this relation(2-4). So, we conducted this study to evaluate the incidence, nature and distribution of retinal hemorrhage in children after a convulsion which required hospitalization and to help clinicians how to deal with this problem.

Thirty one children aged 2 months to 5 years who hospitalized in pediatric ward (of Shahid Beheshti hospital) due to acute seizure attack, in 2002 were included in the study. All