Spastic paraplegia and ataxia in a welder

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51-year-old welder (non-smoker) developed subacute lower paresthesias and progressive spastic-ataxic paraplegia. Symptoms appeared ten days after first zinc exposure on yacht construction site (8-hours-day/40 days in confined space, face-shield only). Examination revealed loss of vibratory-sensation, spasticity, hyperreflexia, sensory ataxia. Serum heavy metal (Cu 1 mg/L, Pb 4.22 µg/L), B-complex vitamins, paraneoplastic on serum and CSF were unremarkable. Elevated zinc level (137 µg/dL) was found. Spinal MRI showed combined degeneration (Figure 1). Myelopathy in zinc smelter workers was reported in 19th century (1) and was recently rediscovered in excessive zinc-ingestion. Copper secondary-deficiency is hypothesized even if normal copper serum-level is also reported (2). Two-months later copper became low (0.8 mg/L) a time-dependent process might explain late copper deficiency in our case. Symptoms ameliorated four-months later with normalization of serum zinc/copper levels.


2. Rembold K, Small J, Tilem M. A Subacute Combined Degeneration-Like Syndrome in the Setting of Zinc Toxicity with Normal Serum Copper Neurology Feb 2013, 80 (7 Supplement) P07.203;
Figure 1. Axial MRI of the dorsal spine (at the D10 level) with a bilateral symmetrical T2 hyperintense signal within the posterior and lateral columns of spine demonstrating an “inverted V sign,” (A,) and creating a 3-point sign as “pair of binoculars sign” or a “dot-sign” or “dumbbell” on the axial plane of dorsal spinal cord (B see arrow). Sagittal MRI spine showing a longitudinally extensive spinal cord lesion with T2 hyperintense signal in the dorsal columns extending from D2 to D10 (white arrows) (C,D). Medullary cone was not affected. There was no cord expansion or atrophy. T1 weighted images showed no abnormalities. No contrast enhancement was found.
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