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CORRESPONDENCE

Reply to correspondence letter by A. Costa et al.: pediatric mercury poisoning, brain MRI, and white matter hyperintensities

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Dear Dr. Guzzi,
With pleasure we have read your comment on our case report of a 4-year-old girl with chronic intoxication of inorganic mercury secondary to the accidental use of an Hg_2Cl_2 and $HgCl_2$ containing skin whitening cream. Neurological deterioration occurred under treatment with dimercapto-1-propansulficacid and was made visible by brain magnet resonance imaging (MRI). We wish to thank you for your helpful contribution to the topic.

You cite an article describing the case of a 48-year-old thermometer factory worker who has been chronically exposed to inorganic mercury. MRI was described with mild central and cortical atrophy, as well as punctiform foci in both frontal regions (T2), but figures are not included in this publication. Furthermore, timing of the MRI is not documented, but seems to have been performed not earlier than 2 years after the end of exposition and normalization of mercury urine levels due to chelation therapy.

You are highlighting the role of neuron-specific enolase (NSE) in mercury-induced neuronal injury reporting that NSE is a quantitative marker of mercury neurotoxicity in vitro. This is an important detail for clinicians especially pediatricians, because mercury intoxication can mimic pheochromocytoma and NSE could be elevated in both situations. Thus, in case of elevation of NSE without obvious reason for brain damage, a screening for mercury in the urine and blood should be performed.

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