Neuroimage

Brain magnetic resonance imaging unveils the history of carbon monoxide poisoning

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A 64-year-old woman was referred to our hospital because of possible viral meningo-encephalitis. Two weeks prior to admission she developed a gait disturbance followed by a fever and headache. Neurological examination showed a small steppage gait, bradykinesia, and rigidity without tremor. However, deep tendon reflexes and cognitive function were normal. Cerebrospinal fluid was normal. A test using a nasopharyngeal swab indicated active infection of influenza A virus, prompting the introduction of oseltamivir that is anti-influenzaviral drug. Magnetic resonance imaging (MRI) using T2WI showed elliptical and diffuse hyperintense lesions in the bilateral globus pallidi [Figure 1A] and cerebral white matter [Figure 1B], respectively. The lesions in the globus pallidi revealed irregular hyperintensity using T1WI. A detailed interview with her disclosed that she had accidentally inhaled carbon monoxide inhalation by an oil heater four weeks earlier.

Discussion

CO is a well-known toxic substance that has high affinity for hemoglobin. Neuropathologic alterations of CO intoxication are characterized by hemorrhagic necrosis of the globus pallidus and demyelination of the cerebral white matter.[4] Except for acute fatal cases, individuals who recover from acute exposure of CO may develop a delayed neurological syndrome for up to 5 weeks after inhalation.[2,3] In some instances, it may be difficult to diagnose accurately this delayed neurological syndrome because clinical settings compromise the gathering of medical histories of patients. In as much as she completely recovered from the acute manifestations of the exposure, her parkinsonism was not considered in association with carbon monoxide. This case came as a fresh reminder of the worth of MRI and the presence of delayed neurologic sequela of carbon monoxide poisoning.

References


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