Diffuse white matter lesions in carbon disulfide intoxication: microangiopathy or demyelination

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摘要

Abstract

Long-term exposure to carbon disulfide (CS(2)) may induce diffuse encephalopathy with parkinsonism, pyramidal signs, cerebellar ataxia, and cognitive impairments, as well as axonal polyneuropathy. The pathogenic mechanisms of diffuse encephalopathy are unclear, although vasculopathy and toxic demyelination have been proposed. Recently, we have encountered a patient who developed headache, limb tremors, gait disturbance, dysarthria, memory impairment, and emotional lability after long-term exposure to CS(2). The brain magnetic resonance images (MRI) showed diffuse hyperintensity lesions in T(2)-weighted images in the subcortical white matter, basal ganglia, and brain stem. The brain computed tomography perfusion study revealed a diffusely decreased regional cerebral blood flow and prolonged regional mean transit time in the subcortical white matter and basal ganglion. To our knowledge, there have been few reports demonstrating diffuse white matter lesions in chronic CS(2) encephalopathy using brain MRI. In addition, the (99m)Tc-TRODAT-1 single photon emission computed tomography showed a normal uptake of the dopamine transporter, indicating a normal presynaptic dopaminergic pathway. We conclude that diffuse white matter lesions may develop after chronic exposure to CS(2), possibly through microangiopathy. In addition, CS(2) poisoning can be considered as one of the causes of chronic leukoencephalopathy.