Delayed toxic−hypoxic leukoencephalopathy after posterior reversible encephalopathy syndrome

2022 Lifespan Research Day Abstract

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Abstract

Background & Aim: Delayed toxic−hypoxic leukoencephalopathy (DTHL) is a rare clinico−radiological syndrome characterized by white matter damage after hypoxic−ischemic brain injury, usually due to toxic exposure. We describe a patient who developed posterior reversible encephalopathy syndrome (PRES) followed by DTHL after two drug overdoses.

Methods: A man in his thirties with polysubstance use disorder suffered two drug overdoses after snorting fentanyl−laced cocaine. After the first overdose, he was treated with naloxone and returned to baseline. Less than 36 hours later, he had another overdose and presented with a saturation of 79% on ambient air and Glasgow Coma Scale of 8. Non−contrast head CT was normal. He was hospitalized for management of acute hypoxic respiratory failure (not requiring intubation), acute toxic−metabolic encephalopathy, aspiration pneumonitis, acute tubular necrosis, and rhabdomyolysis. On day #8, he had clinical seizures and non−contrast brain MRI was consistent with PRES. His clinical condition improved and cognition was near baseline at discharge. Briefly after discharge, he was brought back to the hospital for new onset aggression, disinhibition, and poor memory. Non−contrast head CT now demonstrated interval development of extensive subcortical white matter hypodensities. Non−contrast brain MRI (39 days after initial MRI) now showed extensive, symmetric, and confluent white matter hyperintensities on T2/FLAIR, consistent with DTHL. Follow−up MRI (75 days after initial MRI), revealed reduction in intensity of white matter lesions corresponding with improvement in neuropsychological testing.

Results:

Conclusion: Encephalopathy syndromes occur from exposure to toxins and some have characteristic neuroimaging findings. One of these is PRES, likely caused by endothelial toxicity or injury. PRES may occur with drugs, medications, uncontrolled hypertension, eclampsia, or sepsis. DTHL is a rare neuropsychiatric syndrome occurring after hypoxic−ischemic brain injury. DTHL is often described with carbon monoxide poisoning or drug overdose. Its pathophysiology may be related to delayed effects from activation of the apoptotic cascade or dysmyelination. DTHL typically follows a 'biphasic' presentation: an initial recovery and period of stability followed by an abrupt onset of cognitive impairment, upper motor neuron signs, gait disturbance, or psychosis. To our knowledge, cases of PRES followed by DTHL in the context of recreational drug overdoses have not been previously published.

Clinical Implications: