A 41-year-old man was found unconscious at his home by a friend. He was immediately admitted to the hospital through the emergency department. At the emergency department he regained consciousness. He mentioned he had a few alcoholic beverages the night before but otherwise clinical history was negative. On physical examination swelling of the right orbital area was evident. Further clinical history and laboratory tests were unremarkable. CT scan of the brain showed the right periorbital swelling. Also noted was marked hypodensity of the globus pallidus bilaterally. These areas of hypodensity showed no contrast enhancement. Next MRI of the brain was performed. On T2 weighted images symmetric hyperintensity of the globus pallidus was seen bilaterally. On diffusion weighted images strong hyperintensity was evident due to a reduction in apparent diffusion coefficient reflecting cytotoxic edema. Due to the unusual nature of these lesions further clinical history was obtained and the patient admitted to the single use of ecstasy the night before he was admitted. Follow up MRI after 3 weeks showed a decrease of signal intensity on T2 weighted images of the central area of the globi pallidi consistent with the development of liquefaction necrosis. After contrast administration there was marked enhancement of T1 weighted images likely due to passage through the blood-brain barrier. On diffusion weighted images loss of hyperintensity was evident and an increase in apparent diffusion coefficient was seen. Further clinical evolution was unremarkable.

Comment

Acute parenchymal injuries of the basal ganglia have been reported in a number of conditions. These conditions consist of anoxic-ischemic lesions and metabolic conditions including metabolic acidosis, hypoglycemia and exposure to toxic substances. Although these lesions are often symmetrical, they tend to involve the striatum (putamen and caudate nucleus). These structures have a higher rate of oxygen and glucose consumption. Lesions restricted to the globi pallidi are less commonly reported. An important cause corresponds to the acute phase of carbon monoxide poisoning. Other conditions that have been reported include heroin, ecstasy and cocaine. The mechanism of injury with ecstasy is believed to be a local release of serotonin resulting in prolonged vasospasm and downstream ischemia. The globus pallidus is the area of the brain most vulnerable to the effects of ecstasy because it is very rich in serotonin nerve terminals. Imaging findings on CT include a hypointense appearance of the globi pallidi without evidence of contrast enhancement. On T2 weighted MR images symmetric hyperintensity of the globi pallidi may be evident. T1 weighted images are normal and there is no contrast enhancement. On diffusion weighted images strong hyperintensity may be apparent related to a reduction in apparent diffusion coefficient related to cytotoxic edema. After a few weeks MR findings change. There may be a decrease in hyperintensity on T2 weighted images especially centrally in the lesions due to liquefaction necrosis. On T1 weighted images contrast enhancement may now be seen likely related to a deficiency in the blood-brain barrier. On T2 * sequences a hypointense appearance may be noted because of hemorrhagic transformation. On diffusion weighted images there is a loss of hyperintensity and an increase in apparent diffusion coefficient. In conclusion, acute injuries of the basal ganglia usually involve the striatum. Lesions restricted to the globi pallidi are less common and may be seen with acute carbon monoxide poisoning and drug abuse (heroin, ecstasy, and cocaine).

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