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MR in Trichloroethane Poisoning

Montserrat del Amo, Juan Berenguer, Teresa Pujol, and J. M. Mercader

Summary: We present a case of acute trichloroethane intoxication caused by inhalation of typewriter correction fluid. CT and MR findings revealed lesions in the basal ganglia and cortex similar to those observed in patients with methanol and carbon monoxide poisoning.

Index term: Brain, effect of toxic substances on

Toxic encephalopathy caused by methanol ingestion is characterized by cytotoxic edema, necrosis, cerebral hemorrhage, and brain atrophy (1, 2). The consequences to the putamen result from the direct toxic effects of the methanol metabolites and metabolic acidosis on the basal ganglia (2). We present the CT and MR findings in a case of an acute toxic reaction to 1,1,1-trichloroethane after inhalation of correction fluid.

Case Report

A 17-year-old man was admitted with an acute toxic reaction to 1,1,1-trichloroethane following inhalation of typewriter correction fluid in a suicide attempt. His medical history was unremarkable and he had no history of drug abuse. Physical examination revealed the patient to be in a profound comatose state with reactive miosis.

Results of serum laboratory tests were normal and urine determination of trichloroacetic acid was positive. The patient had a Glasgow coma scale score of 6 and tonicoclonic convulsions that were treated with phenobarbital. The electroencephalogram revealed epileptiform activity. A computed tomographic (CT) scan revealed bilateral hypodensities in the basal ganglia and occipital sulci.

Twelve days after the patient was admitted, a magnetic resonance (MR) examination, performed on a 1.5-T unit, showed poor occipital gray–white matter differentiation on the T1-weighted spin-echo sequences (570/15 [repetition time/echo time]) and a mild bilateral hyperintense area in the globus pallidus on all T1-weighted (Fig 1A) and T2-weighted images. There was an increased signal in the putamen and caudate nucleus and in both occipital hemispheres on T2-weighted spin-echo sequences (2200/20–80) (Fig 1B). Fifty-six days after admission a second MR examination showed an increase in the hyperintense signal in both lenticular nuclei on the T1-weighted spin-echo images (608/14) and hyperintensity, particularly in the putamen and bilateral parasaggital occipital area, on T2-weighted spin-echo images (3500/19–93). Cerebral atrophy, predominantly supratentorial, was present (Fig 1C–E).

The patient remained in a comatose state and there was no clinical improvement 6 months after inhalation.

Discussion

A common industrial solvent, 1,1,1-trichloroethane, or methylchloroform, is found in typewriter correction fluid. It is also used as a recreational drug. Several clinical responses to accidental or voluntary inhalation of 1,1,1-trichloroethane have been described, including acute eosinophilic pneumonia (3), peripheral sensory neuropathy (4), and sudden death caused by ventricular fibrillation or myocardial depression (5–7). In a study using an animal model (8), cessation after 4 days of continuous inhalation of trichloroethane resulted in convulsions, suggesting that trichloroethane may produce a physical chemical dependence. These convulsions disappeared with anticonvulsive treatment.

A study of the long-term effects of a moderate or high level of exposure to 1,1,1,-trichloroethane showed mild encephalopathy (problems with short-term memory, a decrease in attention span and ability to concentrate, moodiness, and disequilibrium). These investigators found no lesions on CT or MR studies that could be attributed to exposure to trichloroethane (9).

In our case, the patient was admitted to the hospital in a coma resulting from acute trichloroethane intoxication after voluntary inhalation of correction fluid. MR images showed bilateral

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From the Service of Radiology, Department of Neuroradiology, Hospital Clínic i provincial de Barcelona (Spain).

Address reprint requests to Montserrat del Amo, MD, Department of Radiology, Hospital Clínic i Provincial de Barcelona, Villarroel, 170, 08036 Barcelona, Spain.

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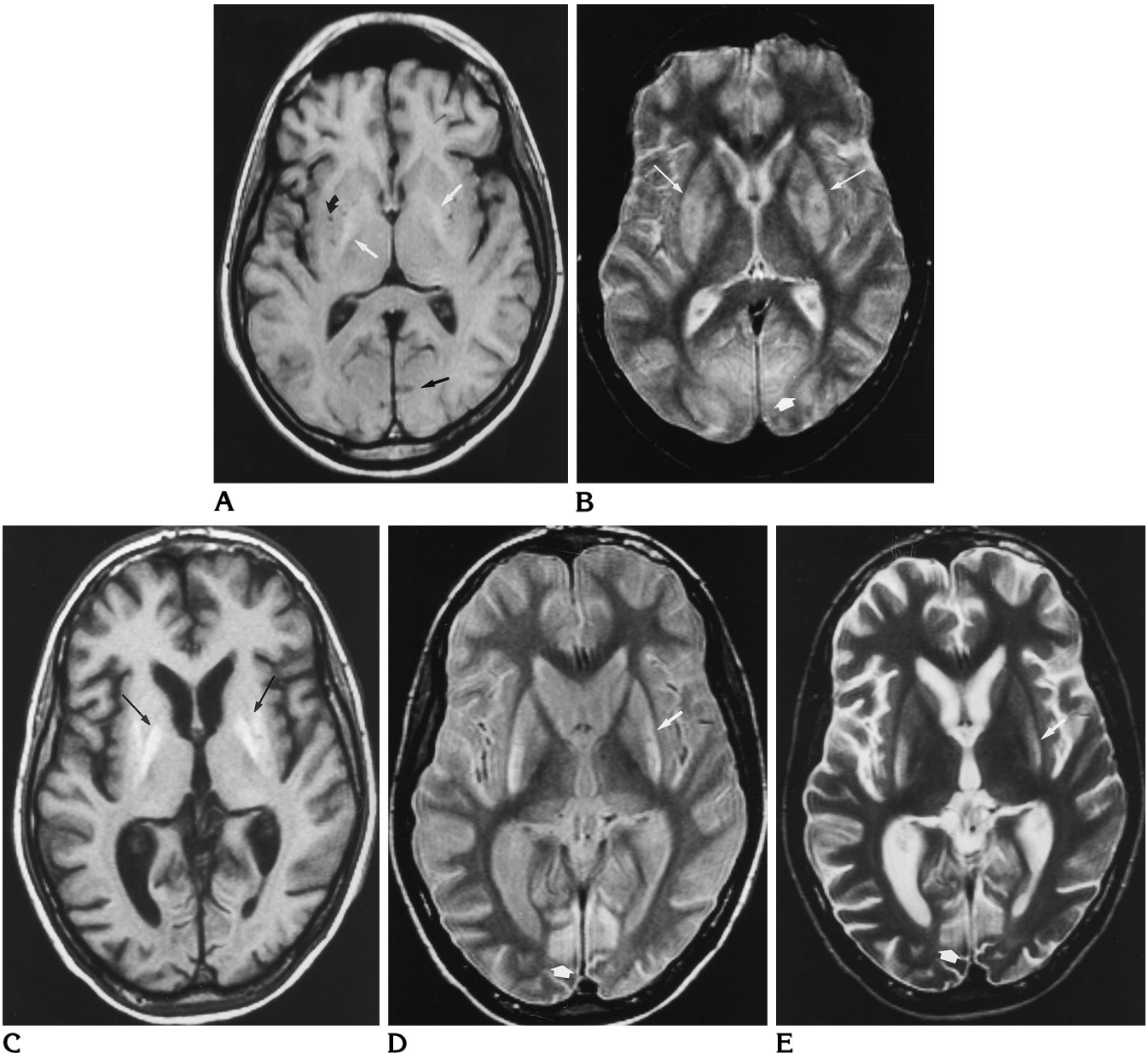

Fig 1. Seventeen-year-old boy with an acute toxic reaction to 1,1,1-trichloroethane.

A, Unenhanced axial T1-weighted spin-echo MR image (570/15) obtained 12 days after admittance shows poor differentiation between gray and white matter bilaterally in the occipital lobe (*straight black arrow*) and increased signal in both pallidus nuclei (*white arrows*). Note prominent perivascular (Virchow-Robin) spaces (*curved black arrow*).

B, Axial T2-weighted spin-echo MR image (2200/80) obtained at the same time as *A* shows high signal intensity in caudate and lenticular nuclei (*long arrows*) with increased signal bilaterally in the occipital lobes involving the cortex and white mater (*short arrow*).

C, Unenhanced axial T1-weighted spin-echo MR image (608/14) obtained 56 days after admission shows marked high signal in both lenticular nuclei (*arrows*) along with cerebral atrophy.

Axial proton density–weighted (*D*) and T2-weighted spin-echo (*E*) MR images (3500/19–93) obtained at the same time as *C* show increased signal in both putamena (*long arrow*) and in the occipital lobes (*short arrow*).

pallidal hyperintensity on T1-weighted and T2-weighted spin-echo images that later involved the entire lenticular nucleus. A repeat MR examination done 6 months later showed significant global atrophy. Signal abnormalities in the basal ganglia corresponded to necrosis, hemorrhage, and atrophy, resulting from the toxic effects of the trichloroethane. These findings are similar to those described in encephalopathies related to other toxins, such as methanol (1), heroin (10), and carbon monoxide, and to hypoxic-anoxic insults (1), in which involvement of the cortical and basal ganglia is seen. This pattern of involvement has also been observed in Wilson disease, Leigh disease, Kearn-Sayre syndrome, and Leber optic atrophy (2, 11).

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