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Selective Cerebellar Degeneration Following Carbon Monoxide Poisoning

Carbon monoxide (CO) is a toxic gas produced by the incomplete combustion of carbon-containing compounds.¹ Until recently, coal contributed a significant portion of the fuel used for heating and cooking in Korea, and most cases of CO poisoning were due to defective exhaust tubes in old coal-heating facilities. To our knowledge, selective degeneration of the cerebellar cortex and white matter on MR imaging following CO exposure has not been described in the literature.²

A 39-year-old woman presented with chronic limb ataxia, titubation, scanning dysarthria, and balance problems for 13 years after CO intoxication. At 26 years of age, she was found unconscious at home after midnight, following exposure to a malfunctioning coal-heating system in a poorly ventilated room. A diagnosis of CO poisoning was made, and hyperbaric oxygen therapy was administered. She regained consciousness after a few days. However, she had difficulty with articulation, walking, and manual dexterity. Head titubation was apparent. The patient was discharged following an improvement in her general condition, but the ataxic symptoms noted above persisted.

Brain MR imaging performed in January 2007 revealed diffuse severe cerebellar atrophy (Fig 1). The basal ganglia, thalamus, and hippocampus were not affected. There was no diffuse change in the cerebral deep white matter or cortex. We were unable to obtain a copy of the brain imaging that was performed just after her poisoning 13 years earlier. The patient's cognition was normal, and her head titubation improved with clonazepam medication. However, her limb ataxia and gait disturbance were unchanged at the 1-year follow-up visit.

The most common sites for pathologic findings on MR imaging of

patients with CO exposure include the globus pallidus, cerebral deep white matter, cerebral cortex, putamen, thalamus, and hippocampus. Cerebellar involvement is relatively infrequent and is usually accompanied by other brain lesions in cases of severe intoxication.^{3,4} Mascalchi et al² are the only authors to have reported an extensive bilateral cerebellar white matter signal-intensity change with sparing of the overlying cerebellar cortex and basal ganglia by using MR imaging. In their case, a 12-year-old boy who had CO poisoning at 6 years of age was examined in the chronic phase, like our patient. They ascribed the unusual cerebellar involvement to the long delay between exposure and MR imaging.²

Our report suggests that selective cerebellar involvement is rare but may occur after CO poisoning.

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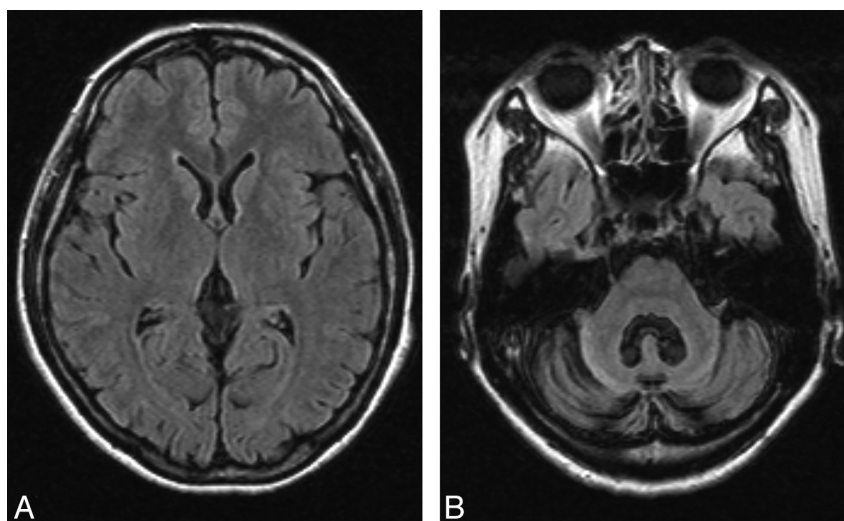


Fig 1. A, Axial MR image of the patient by using fluid-attenuated inversion recovery (FLAIR) sequences shows no diffuse change in the basal ganglia, subcortical white matter, or thalamus. B, Axial FLAIR image demonstrates selective degeneration of the cerebellar cortex and cerebellar white matter.