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Multimodal pharmacologic approach in delayed neuropsychiatric syndrome after carbon monoxide intoxication

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Background

Delayed neuropsychiatric syndrome (DNS) is a well-recognized complication of carbon monoxide (CO) intoxication that develops after an apparent recovery from the acute event. Patients typically present with cognitive decline, parkinsonian features, gait disturbance, and urinary incontinence after a lucid interval. Neuroimaging findings such as lesions in the globus pallidus and deep white matter have been reported to be associated with the development of DNS. Despite its poor prognosis, no established treatment exists, although several case reports have suggested potential benefits of high-dose corticosteroid therapy.

Case Description

A 60-year-old man was found unconscious in a closed vehicle after burning charcoal on September 17, 2025 and was treated for hypoxic brain injury at another hospital. Hyperbaric oxygen therapy was not administered. Brain MRI performed on September 22, 2025 demonstrated T2/FLAIR hyperintensities in the bilateral basal ganglia centered on the globus pallidus, consistent with CO-related hypoxic injury. He recovered and was discharged; however, several days later he developed progressive cognitive decline, loss of communication, urinary incontinence, and gait disturbance, compatible with DNS.

He was admitted to the Department of Rehabilitation Medicine on October 17, 2025. Initial evaluation on October 22, 2025 revealed severe cognitive impairment (MMSE 1) with total dependence in activities of daily living (K-MBI 4). Clinically, he showed minimal verbal output, poor initiation, and resting tremor.
Follow-up brain MRI performed on November 12, 2025 revealed confluent T2 high signal changes in both periventricular white matter with persistent basal ganglia involvement.

Based on previous reports suggesting inflammatory mechanisms in DNS, high-dose corticosteroid therapy was administered (prednisolone 60 mg/day for 11 days with tapering). Donepezil was titrated from 5 mg to 10 mg and subsequently to 23 mg daily. Levodopa was administered at 100 mg three times daily, increased to 200 mg three times daily; however, it was discontinued due to worsening symptoms including diaphoresis, tremor, and cervical–trunk extension stiffness.

Follow-up evaluation on December 2, 2025 showed persistent severe impairment on cognitive assessments (MMSE 1) with limited functional improvement (K-MBI 2). Although these objective findings and MRI lesions (Figure 1, 2) suggested a poor prognosis, caregivers and clinicians observed subtle improvements in arousal, initiation, and verbal responsiveness, including occasional spontaneous name calling and increased interaction with family members.

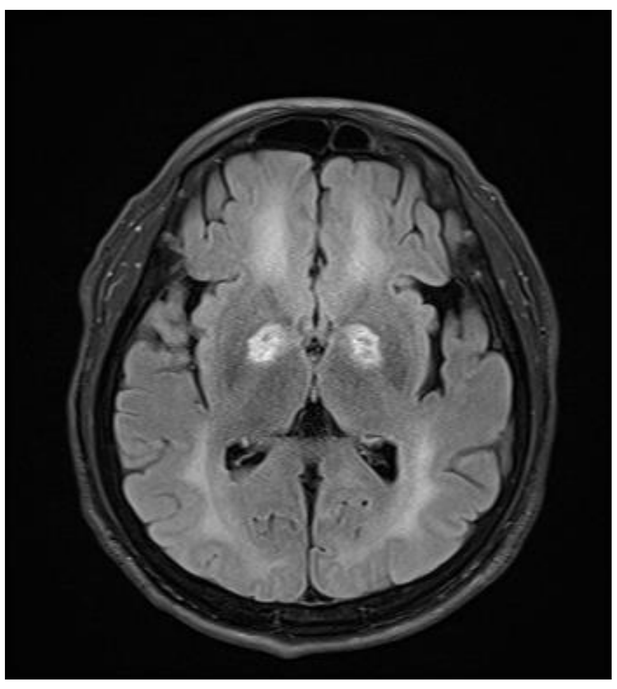

Figure 1. Brain MRI showing T2/FLAIR hyperintensities in the bilateral globus pallidus consistent with CO-related hypoxic injury.

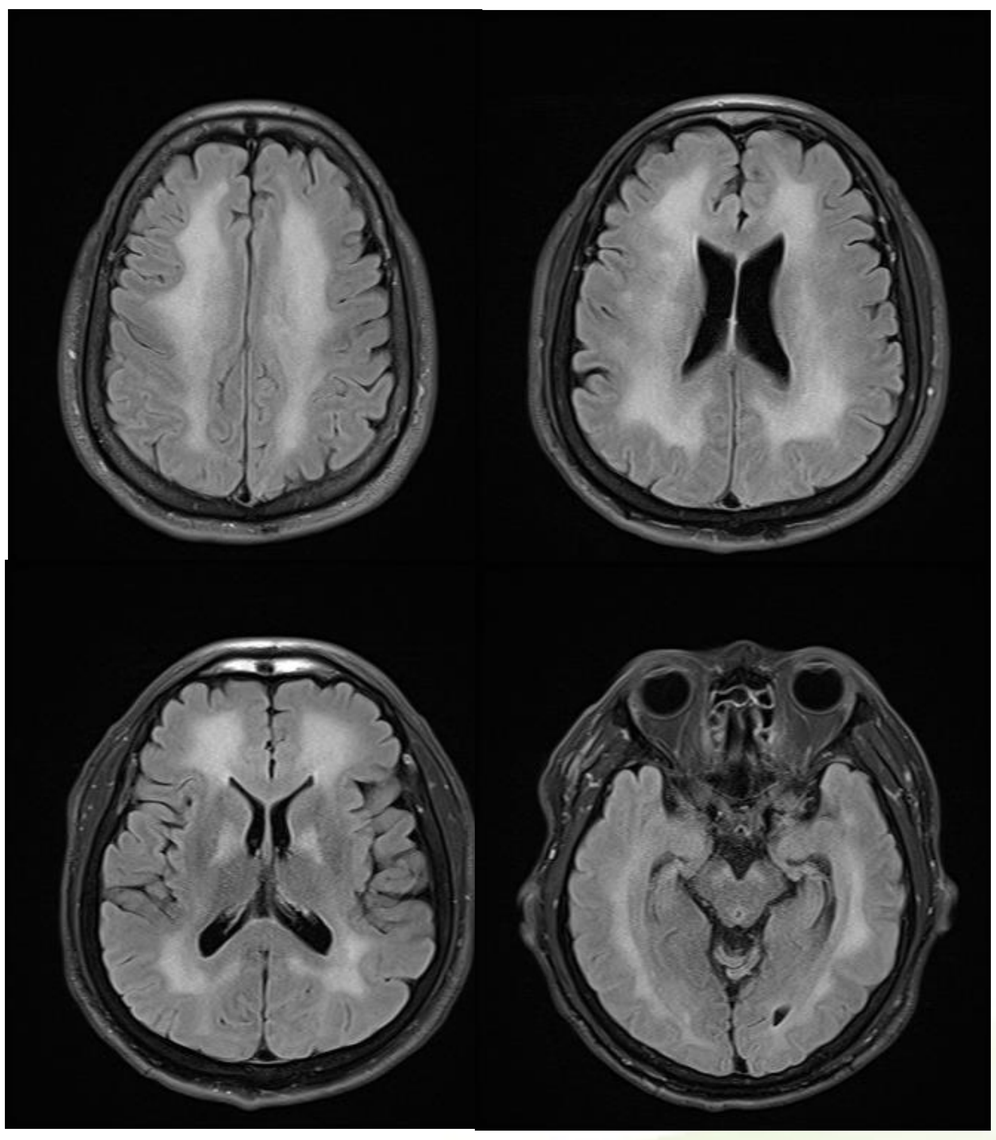

Figure 2. Follow-up T2 FLAIR MRI demonstrating confluent T2 high signal changes in the bilateral periventricular white matter and basal ganglia.

Conclusion

This case demonstrates the impact of a multimodal pharmacologic approach on the clinical course of DNS following CO intoxication. Although objective cognitive scores did not show significant changes during follow-up, qualitative improvements in responsiveness were observed after multimodal pharmacologic management.