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## INTRODUCTION

Carbon monoxide (CO) poisoning is a life-threatening condition requiring prompt recognition and intervention to prevent irreversible neurological and cardiac damage. CO binds competitively to hemoglobin, forming carboxyhemoglobin (COHb), thereby impairing oxygen delivery and inducing cellular hypoxia. Additionally, CO disrupts mitochondrial function, promoting oxidative stress and neuronal injury.

## CASE DESCRIPTION

We report the case of a 32-year-old male found unconscious in the presence of smoke-filled enclosed space, presenting to the emergency department with a Glasgow Coma Scale (GCS) score of 5/15 and involuntary body movement. Initial brain computed tomography (CT) revealed bilateral acute putaminal infarcts. Laboratory investigations confirmed elevated COHb at 12.1%, establishing a diagnosis of acute CO poisoning. The patient underwent hyperbaric oxygen therapy (HBOT) with three sessions administered over 18 hours.

The first session, initiated five hours post-admission at 2.8 ATA for 120 minutes, improved GCS to 7. Subsequent sessions at 2.0 ATA for 90 minutes each led to progressive GCS improvements—9 and 11, respectively. COHb levels decreased to 1.1%, and the patient regained full consciousness, after which he was referred for psychiatric evaluation.



## DISCUSSION

This case underscores the critical importance of early HBOT in severe CO poisoning. HBOT accelerates CO elimination by reducing its half-life from approximately 300 minutes in ambient air to 30 minutes and enhances oxygenation of hypoxic tissues. It also mitigates oxidative injury and lowers the risk of delayed neurological sequelae (DNS). Notably, the bilateral symmetric putaminal infarcts seen on CT, though nonspecific, can serve as a radiological clue prompting consideration of CO toxicity, especially in unexplained unconsciousness.



## CONCLUSION

Clinicians should maintain a high index of suspicion when dealing with a case of history of exposure to smoke. Timely initiation of HBOT in acute CO poisoning is associated with significant neurological recovery and prevention of long-term complications. This case supports the growing evidence that early, protocol-driven HBOT can optimize outcomes in CO poisoning. Further research is warranted to define standardized treatment protocols and identify predictors of recovery.

## REFERENCE

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