**Scholars Journal of Medical Case Reports**
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

**Radiology**

# Role of Brain CT in the Early Diagnosis of Carbon Monoxide Poisoning: A Case Report

S. Aouaq[1]*, FZ. Daoui[1], Z. AIT SAID[1], Y. Bouktib[1], A. El Hajjami[1], B. Boutakioute[1], M. Ouali Idrissi[1], N. Cherif Idrissi El Guennouni[1]

[1]Department of Radiology, Errazi Hospital, Mohammed VI University Hospital Center, Cadi Ayyad University

**DOI:** https://doi.org/10.36347/sjmcr.2025.v13i10.095 | **Received:** 18.08.2025 | **Accepted:** 22.10.2025 | **Published:** 28.10.2025

***Corresponding author:** S. Aouaq
Department of Radiology, Errazi Hospital, Mohammed VI University Hospital Center, Cadi Ayyad University

**Abstract** | **Case Report**

***Introduction:*** Carbon monoxide (CO) poisoning represents a frequent medical emergency causing tissue hypoxia through its high affinity for hemoglobin. Clinical manifestations are often polymorphic and nonspecific, ranging from headaches and cognitive disorders to seizures, coma, and death, with risk of delayed neuropsychiatric sequelae. ***Case Report:*** We present the case of a 36-year-old patient with no significant medical history, admitted for acute accidental CO poisoning following prolonged exposure (over four hours) to fumes from a charcoal heater. The initial cerebral CT scan revealed symmetric nodular hypodensities in both globi pallidi, while biological parameters and chest X-ray were normal. ***Discussion:*** Radiologically, cerebral CT scan, often used as first-line imaging, may be normal or show diffuse edema and white matter hypodensities. Although less sensitive than MRI, it remains crucial in emergency settings for diagnostic orientation and severity assessment alongside clinical data. MRI, more sensitive, typically demonstrates characteristic lesions of the globi pallidi and white matter involvement, allowing better prognostic evaluation and follow-up. The topographic progression of lesions typically begins in the globi pallidi before extending to other basal ganglia structures. Pathophysiologically, the selective vulnerability of basal ganglia relates to their high energy metabolism and rich vascularization, making them particularly sensitive to hypoxic injury. ***Conclusion:*** This case illustrates the diagnostic value of cerebral imaging in CO poisoning. CT scanning maintains its role in initial assessment due to its accessibility, while MRI remains the gold standard for precise lesion characterization, prognostic evaluation, and follow-up of patients with CO intoxication.

**Keywords:** Carbon Monoxide Poisoning, Brain CT, MRI, Globus Pallidus, Delayed Encephalopathy.



## INTRODUCTION

Carbon monoxide (CO) poisoning is a common medical emergency that causes tissue hypoxia due to its high affinity for hemoglobin. Clinical manifestations are often polymorphic and nonspecific, ranging from headaches and cognitive disorders to seizures, coma, and death, with risk of delayed neuropsychiatric sequelae.

Radiologically, cerebral computed tomography (CT), often used as first-line imaging, may be normal or reveal diffuse edema and white matter hypodensities. Although less sensitive than MRI, it remains a key examination in emergency settings, contributing alongside clinical data to diagnostic orientation and severity assessment. MRI, being more sensitive, reveals typical lesions of the globi pallidi and white matter involvement, enabling better prognostic evaluation and follow-up.

We present a case of acute carbon monoxide poisoning complicated by severe coma and illustrated by suggestive CT abnormalities.

## CASE REPORT

A 36-year-old male patient with no significant medical history was admitted for acute accidental carbon monoxide poisoning secondary to prolonged exposure (over four hours) to fumes from a charcoal heater.

In the emergency department, the patient presented with severe coma (Glasgow 5/15), with eye opening only to pain and asymmetric withdrawal motor response to painful stimuli. Biological workup and chest radiography were normal, and carboxyhemoglobin levels were not available. Emergency orotracheal intubation was performed, followed by management in the intensive care unit.

Cerebral CT, performed a few hours after intoxication, revealed symmetric nodular hypodensities visible in projection of both globi pallidi.

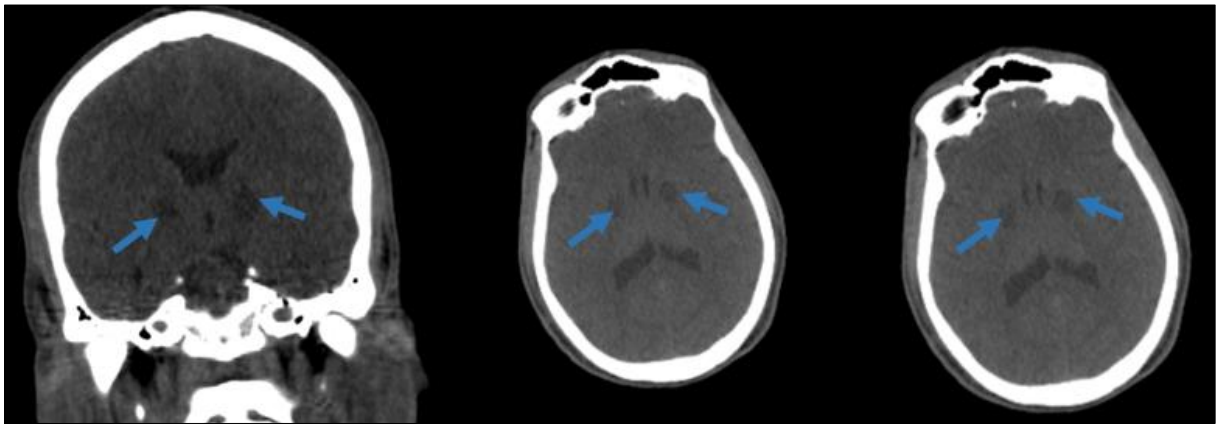

**Figure 1: A non-contrast brain scan reveals well-defined, spontaneously hypodense nodular lesions (blue arrows) symmetrically located in both globi palladi**

## DISCUSSION

Carbon monoxide (CO) poisoning represents a major public health concern, constituting the leading cause of death by intoxication in most Western countries [1]. As illustrated by our case, this intoxication occurs primarily in accidental domestic contexts, often related to defective heating devices or water heaters [2].

Clinically, the initial presentation is polymorphic, varying from non-specific symptoms (headaches, nausea, asthenia) to severe neurological manifestations (impaired consciousness, extrapyramidal syndrome) depending on carboxyhemoglobin concentration [3, 4]. Our case perfectly illustrates this clinical variability and underscores the importance of systematically considering this diagnosis in any unexplained acute neurological manifestation.

The contribution of cerebral imaging, particularly CT in our observation, proves crucial for positive diagnosis. In the acute phase, computed tomography may reveal symmetric hypodensities of the basal ganglia, particularly the globi pallidi, as observed in our patient [5]. However, it is important to note that initial examination by CT or MRI may appear normal despite the presence of deep coma [6]. Paradoxally, some white matter abnormalities detected on CT may be completely reversible, which limits the prognostic value of cerebral imaging in this context [6].

### Contribution of Brain MRI

Brain MRI, the reference examination, allows earlier and more sensitive detection of lesions [7, 8]. In the initial stage, it typically reveals predominant involvement of the globi pallidi manifesting as:

- T2/FLAIR hyperintensity in cases of edematous component
- T2 hypointensity in cases of hemorrhagic transformation [9, 10]

The initial neurological involvement shows characteristic topographic progression. Lesions typically begin in the globus pallidus before gradually extending to other basal ganglia structures, notably the putamen, caudate nucleus, thalamus, and substantia nigra [11]. This lesional expansion reflects the particular vulnerability of brain regions with high energy metabolism to toxic hypoxia [12].

White matter involvement, observed in a variable proportion (10-100% of patients depending on the series), manifests according to several distinct patterns [13]. Notably observed are:

- Multifocal necrotic lesions in the centrum semiovale
- Extensive periventricular necrotic lesions with possible extension to the corpus callosum
- Diffuse involvement of deep periventricular white matter [14]
- The contribution of advanced sequences proves decisive for fine characterization of lesions. Diffusion sequences demonstrate:
- Hyperintensity of periventricular white matter
- Restricted diffusion indicating cytotoxic edema
- Slight increase in diffusion coefficient in the globi pallidi, suggesting vasogenic edema [15]

The correlation between imaging findings and neurological prognosis remains imperfect. Only 12% of patients with long-term cognitive sequelae showed initial white matter abnormalities on MRI [13]. Late demyelinating lesions, once established, show limited reversibility [14]. The predictive value of early abnormalities detected by imaging thus remains relative, requiring cautious interpretation integrating all clinical and paraclinical parameters.

Pathophysiologically, the selective vulnerability of basal ganglia is explained by their high

energy metabolism and rich vascularization, making them particularly sensitive to hypoxia [16]. The lesional mechanism combines several factors: hypoxemia through competition with oxygen, mitochondrial dysfunction, activation of lipid peroxidation, and inflammatory phenomena [17]. These complex mechanisms explain the possible occurrence of delayed neurological manifestations, typically appearing after a free interval of several weeks [4].

Treatment primarily relies on normobaric or hyperbaric oxygen therapy, whose effectiveness is maximal when initiated early [18]. Imaging plays an increasing role in prognostic evaluation and follow-up. The persistence or worsening of lesions on imaging correlates with an increased risk of long-term neurological sequelae [19].

Nevertheless, the observed abnormalities are not specific and may be encountered in other contexts (cyanide poisoning, non-toxic hypoxia, metabolic disorders) [20]. Prudent interpretation, correlated with clinical and biological context, therefore remains essential.

## CONCLUSION

In conclusion, our observation highlights the value of cerebral imaging in the management of carbon monoxide poisoning. Computed tomography maintains its place in initial assessment due to its accessibility, while brain MRI, as the reference imaging method, provides crucial information for positive diagnosis, prognostic evaluation, and follow-up.

## REFERENCES


1. Rafai MA, Midafi N, Boulajaj FZ, El Moutawakkil B, Jidane M, Slassi I. Aspects cliniques et neuroradiologiques de l'intoxication aiguë au CO. Feuillets de Radiologie. 2010;50:237-40.
2. Hantson P, Duprez T. Imagerie cérébrale et intoxications. Réanimation. 2009;18:598-605.
3. Gados P, Conso F, Korach JM, Chevret S. Incidence and causes of monoxide intoxication: results of an epidemiologic survey in a french department. Arch Environ Health. 1991;46:373-6.
4. El Murr T, Tohme A, Chatad E. Surdité aiguë par intoxication au monoxyde de carbone. À propos d'un cas et revue de la littérature. Ann Med Interne. 2002;153:206-8.
5. Thom SR, Elmi K. Carbon monoxide poisoning: a review epidemiology, pathophysiology, clinical findings, and treatment options including hyperbaric oxygen therapy. J Toxicol Clin Toxicol. 1989;27:141-56.
6. O'Donnell P, Buxton PJ, Pitkin A, Jarvis LJ. The magnetic resonance imaging appearances of the brain in acute carbon monoxide poisoning. Clin Radiol. 2000;55:273-80.
7. Beppu T. The role of MR imaging in assessment of brain damage from carbon monoxide poisoning: a review of the literature. AJNR Am J Neuroradiol. 2014;35(5):1048-9.
8. Bianco F, Floris R. MRI appearances consistent with hemorrhagic infarction as an early manifestation of carbon monoxide poisoning. Neuroradiology. 1996;38:570-2.
9. Ferrier D, Wallace CJ, Fletcher WA, Fong TC. Magnetic resonance features in carbon monoxide poisoning. Can Assoc Radiol J. 1994;45:466-8.
10. Kawanami T, Kato T, Kurita K, Sasaki H. The pallidoreticular pattern of brain damage on MRI in a patient with carbon monoxide poisoning. J Neurol Neurosurg Psychiatry. 1998;64:282.
11. Lo CP, Chen SY. Neuroimaging of toxic and metabolic disorders. Clin Radiol. 2007;62:1153-62.
12. Parkinson RB, Hopkins RO, Cleavinger HB, Weaver LK, Victoroff J, Foley JF, et al. White matter hyperintensities and neuropsychological outcome following carbon monoxide poisoning. Neurology. 2002;58:1525-32.
13. Chang KH, Han MH, Kim HS, Wie BA, Han MC. Delayed encephalopathy after acute CO intoxication: MR imaging features and distribution of cerebral white matter lesions. Radiology. 1992;184:117-22.
14. Chu K, Jung KH, Kim HJ, Jeong SW, Kang DW, Roh JK. Diffusion-weighted MRI and 99mTc-HMPAO SPECT in delayed relapsing type of carbon monoxide poisoning: evidence of delayed cytotoxic edema. Eur Neurol. 2004;51:98-103.
15. Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med. 2002;347:1057-67.
16. Kim JH, Chang KH, Song IC, Kim KH, Kwon BJ, Kim HC, *et al.,* Delayed encephalopathy of acute carbon monoxide intoxication: diffusivity of cerebral white matter lesions. AJNR Am J Neuroradiol. 2003;24:1592-7.
17. Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. J Neurol Sci. 2007;262:122-30.
18. Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. Plast Reconstr Surg. 2011;127 Suppl 1:131S-141S.
19. Kanaya N, Imaizumi H, Nakayama M, Nagai H, Yamaya K, Namiki A. The utility of MRI in acute stage of carbon monoxide poisoning. Intensive Care Med. 1992;18:37-2.
20. Hantson P, Duprez T, Mahieu P. Neurotoxicity to the basal ganglia shown by brain magnetic resonance imaging (MRI) following poisoning by methanol and other substances. J Toxicol Clin Toxicol. 1997;35:151-61.