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IMAGING DIAGNOSIS OF CARBON MONOXIDE POISONING

Radio-Diagnosis

Prishaa Bhurrji Research Intern, Nanavati Max Super Speciality Hospital
Dr. Ritvik Thakrar Neuro-radiology Fellow, Nanavati Max Super Speciality Hospital
Sanjana Sanzgiri* Research Intern, Nanavati Max Super Speciality Hospital *Corresponding Author
Dr. Gayatri Harshe Consultant Radiologist, Nanavati Max Super Speciality Hospital
Dr. Mitusha Verma Consultant Radiologist, Nanavati Max Super Speciality Hospital
Dr. Deepak Patkar Head of Department of Radiology, Nanavati Max Super Speciality Hospital

ABSTRACT

Carbon monoxide, a colourless, odourless and tasteless gas causes toxicity when excessive amounts of the gas is inhaled. Individuals usually become unconscious before they realise, they are getting poisoned. Severe tissue hypoxia can occur if this condition is not treated immediately, therefore, appropriate diagnosis using imaging and management is crucial.

KEYWORDS

CO poisoning, carbon monoxide poisoning, radiological findings

INTRODUCTION

CO poisoning (incidence of 16 cases per 100,000) is a fatal condition that occurs as a result of breathing in fumes of CO from sources including gas boilers, portable generators, car engines etc. (Rose et al., 2017). Individuals suffering from CO poisoning usually experience symptoms such as headache, weakness, nausea and shortness of breath immediately after exposure and can be lethal. As the condition worsens, the brain and heart are affected causing loss of muscle control and eventually leading to loss of consciousness (*Carbon Monoxide Poisoning - NHS*, n.d.). High amount of exposure can lead to death in less than five minutes. CO is an odourless and colourless gas which makes it difficult to detect. It also strongly binds to haemoglobin in the bloodstream and can cause severe hypoxic injury if not treated immediately. The high binding affinity of CO for haemoglobin (~240 times greater than that of oxygen) makes it a highly toxic molecule, causing tissue hypoxia in the body (Megas et al., 2021). A cascade of toxic events follows tissue hypoxia leading to a disruption in the metabolism of cells and in turn, disrupting their normal functioning. This could have life-threatening consequences in patients if neglected and not reversed urgently. Thus, appropriate diagnosis and timely treatment of the condition is essential.

Imaging modalities, including CT scans and MRI are used for the diagnosis of this condition. However, imaging findings in early stages of CO poisoning can be detected with more sensitivity and specificity on MRI as compared to CT. Radiographic features demonstrate bilateral changes with the globus pallidus which is the most commonly affected region (Bell & Weerakkody, 2010).

Case Report

History Of Presentation

A 35-year-old female was admitted to our hospital after collapsing suddenly in her bathroom. She was found by her relatives lying unconscious whilst frothing from her mouth. She was intubated as a result of being unresponsive, elsewhere. CT scan of the brain performed prior to admission at our hospital, did not reveal any significant abnormalities. On clinical examination at our hospital, all her limbs were moving. However, there was a reduction in power (Grade – 3/5 in both upper and lower limbs). Patient was subsequently admitted to the ICU and was advised to undergo an MRI examination of the brain; findings of which are explained in detail further.

Past Medical History

There was no known history of any comorbidities. No history of any exertional symptoms and no evidence of the intake of any unusual substance was found.

Investigations

An MRI (3 Tesla) scan of the brain was performed using T1 and T2-weighted sequences in multiple planes, using a quadrature head coil. The scan revealed symmetric, large, patchy areas of restricted diffusion seen in the bilateral fronto-parietal, temporal and occipital cortex and subcortical white matter. Areas of restricted diffusion (**Fig. 1A**) were also observed in both hippocampi, bilateral globus palladi, both cerebellar hemispheres and the septum pellucidum. These showed drop in signal intensity on

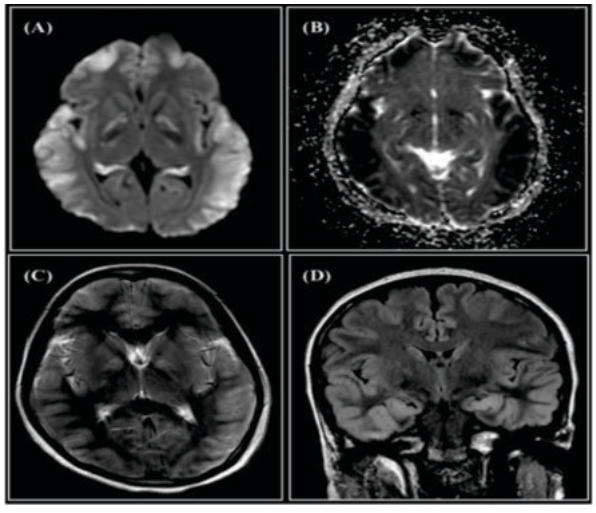

Figure 1 *MRI scan of the brain. (A) Diffusion restriction seen in bilateral frontal and parietal cortex, subcortical white matter and hippocampi; (B) Low signal intensity on ADC; (C) T2 hyperintensities in same regions as in DWI; (D) FLAIR hyperintensities in bilateral temporal cortex, both hippocampi and in subcortical white matter.*

ADC maps (**Fig. 1B**) and appeared slightly hyperintense on FLAIR images (**Fig. 1C** and **Fig. 1D**). On the basis of these findings, a diagnosis of CO poisoning, with associated severe hypoxia, was made.

Medical Management

Hyperbaric oxygen therapy along with medication and IV fluid is given to patients suffering from CO poisoning. This therapy comprises of breathing pure oxygen in a pressurised environment.

Follow-up

At discharge, the patient was hemodynamically and vitally stable.

Differential Diagnosis

CO poisoning (**Fig. 2A**) may sometimes be misdiagnosed for toxic encephalopathies, prion encephalopathies, mitochondrial encephalopathies or metabolic disorders as they manifest similar radiological findings, affecting the basal ganglia (primarily, the globus pallidus) and the white matter

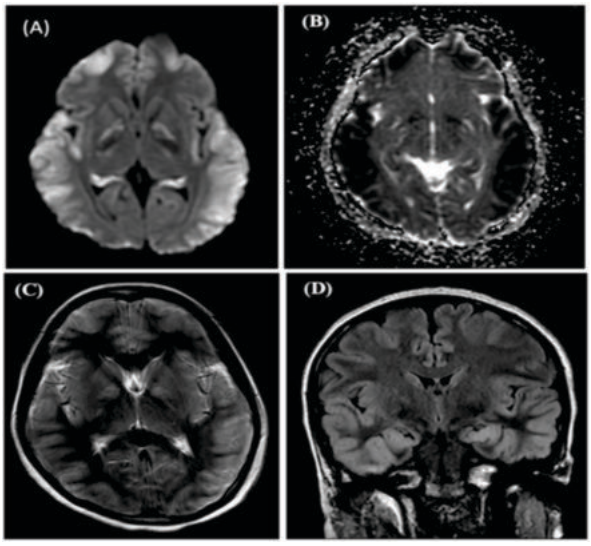

Figure 2 *Differential diagnosis for CO poisoning. (A) Diffusion restriction in the subcortical white matter in CO poisoning; (B) Diffusion restriction seen in the head of caudate nucleus, bilaterally and in putamina in CJD; (C) Diffusion restriction seen in the head of caudate nucleus, putamina, bilaterally and thalami in Leigh disease; (D) Symmetric T2 hyperintensities seen along the posterior limb of the internal capsule, bilaterally in Wilson's disease. Diffusion does not show any significant abnormality.*

(Bell & Weerakkody, 2010). Toxic encephalopathy can be used to describe brain dysfunction resulting from toxic exposure of substances including heroin, opioids, cocaine, ethanol etc. (Hacking & Deng, 2020). CJD (**Fig. 2B**) is a form of prion disease and is a rare neurodegenerative disorder caused by the accumulation of an abnormal glycoprotein called prion protein (*Creutzfeldt-Jakob Disease, Classic (CJD) | Prion Diseases | CDC*, n.d.). Leigh disease (**Fig. 2C**) can be classified as a mitochondrial encephalopathy and can be characterized by the progressive formation of symmetrical lesions in the brainstem or the basal ganglia (Alemao et al., 2022). Wilson's disease (**Fig. 2D**), also called hepatolenticular degeneration, is a rare multisystem disorder of copper metabolism. The central nervous system findings of this condition often include abnormal copper accumulation in the midbrain and basal ganglia (Islam & Knipe, 2015). The clinical profile and MRI findings for each of these differential diagnoses and CO poisoning is shown below in **Table 1**.

Table 1 *Differential Diagnosis. The clinical profile and MRI findings for CO poisoning, toxic encephalopathy, CJD, Leigh disease and Wilson's disease.*

Condition	CO Poisoning	Toxic Encephalopathy	CJD	Leigh Disease	Wilson's Disease
Clinical Profile	• Headache, dilation of cutaneous vasculature • Weakness, confusion • Cardiac dysthymias, tachycardia	• Changes in cognitive function, seizures, hydrocephalus • Disturbed visual, olfactory, auditory functions	• Subacute dementia and intractable myoclonus • Global aphasia, right hemiparesis and severe gait disorder	• Progressive neurological deterioration • Loss of motor skills and appetite • Vomiting, irritability and seizure activity	• Hepatic manifestations leading to jaundice • Neuropsychiatric symptoms like dementia and depression
MRI Findings	• Areas of restricted diffusion seen in acute phase • T2/FLAIR - hyperintense • High signal intensity on T2-weighted FLAIR images	• DWI restriction involving white matter, may also involve the corpus callosum • FLAIR images showing confluent and symmetric white matter abnormalities	• DWI restriction more pronounced than T2/FLAIR hyperintensity • T1-weighted images show high signal changes in globus pallidus	• DWI restriction may be seen in acute phase • T2-weighted images show high signal in brainstem, medulla, grey matter, etc. • T1 images usually demonstrate reduced signal in T2 abnormal areas	• Abnormal T2 hyperintensity in the putamina • Other areas of T2 signal abnormalities predominantly affecting the deep grey nuclei • Axial T2 MRI at pons showing a "double panda sign" • DWI restriction may/may not be seen in the affected areas

DISCUSSION

CO poisoning in organisms requiring oxygen to survive has existed since the presence of fire and its smoke, however, the first case of poisoning reported in humans occurred in 1945 (Megas et al., 2021). One of the first diagnostic devices to detect and measure CO concentration in expired breath was the Dräger test tube. Overtime, cases of CO intoxication decreased drastically due to industrial transformations (including a switch from coal to natural gas) and increasing numbers of different approaches to committing suicide (Megas et al., 2021). However, one should be wary of the radiological findings, with MRI being the most sensitive as demonstrated in this case. Clinical suspicion of CO poisoning also plays a major role.

Learning Objectives

- Although low in incidence now, CO poisoning can be detrimental and may lead to death.
- History is extremely important. Clinical suspicion for CO poisoning leads to early imaging. Early imaging aids confirming diagnosis and thus, help in early treatment.
- DWI & FLAIR images are the most important sequences. CT does not play a significant role.
- Radiologists should be well versed with findings of CO poisoning and its differentials.

REFERENCES:

1. Alemao, N. N. G., Gowda, S., Jain, A., Singh, K., Piplani, S., Shetty, P. D., Dhawan, S., Arya, S., Chugh, Y., & Piplani, S. (2022). Leigh's disease, a fatal finding in the common world: A case report. *Radiology Case Reports*, *17*(9), 3321–3325. https://doi.org/10.1016/J.RADCR.2022.06.060
2. Bell, D., & Weerakkody, Y. (2010). Carbon monoxide poisoning. *Radiopaedia.Org*. https://doi.org/10.53347/RID-11020
3. *Carbon monoxide poisoning - NHS*. (n.d.). Retrieved August 24, 2023, from https://www.nhs.uk/conditions/carbon-monoxide-poisoning/
4. *Creutzfeldt-Jakob Disease, Classic (CJD) | Prion Diseases | CDC*. (n.d.). Retrieved August 24, 2023, from https://www.cdc.gov/prions/cjd/index.html
5. Hacking, C., & Deng, F. (2020). Toxic leukoencephalopathy (mnemonic). *Radiopaedia.Org*. https://doi.org/10.53347/RID-74476
6. Islam, D., & Knipe, H. (2015). Wilson disease (CNS manifestations). *Radiopaedia.Org*. https://doi.org/10.53347/RID-39667
7. Megas, I. F., Beier, J. P., & Grieb, G. (2021). The History of Carbon Monoxide Intoxication. *Medicina*, *57*(5). https://doi.org/10.3390/MEDICINA57050400
8. Rose, J. J., Wang, L., Xu, Q., McTiernan, C. F., Shiva, S., Tejero, J., & Gladwin, M. T. (2017). Carbon monoxide poisoning: Pathogenesis, management, and future directions of therapy. *American Journal of Respiratory and Critical Care Medicine*, *195*(5), 596–606. https://doi.org/10.1164/RCCM.201606-1275CI/SUPPL_ FILE/DISCLOSURES.PDF