Electronic Journal of General Medicine
2023, 20(3), em467
e-ISSN: 2516-3507
https://www.ejgm.co.uk/

MODESTUM

Case Report **OPEN ACCESS**

A patient with toxic encephalopathy associated with acute carbon monoxide poisoning–A clinical case

Alexandra Yankova [1]*, Kristiyan Georgiev [2], Dimitrinka Dimitrova [1], Vladina Dimitrova-Kirilova [1], Veselinka Nestorova [1]

[1] Department of Optometry and Occupational Diseases, Faculty of Public Health, Medical University Varna, Varna, BULGARIA
[2] Department of Neurological Diseases and Neuroscience, Faculty of Medicine, Medical University Varna, Varna, BULGARIA
***Corresponding Author:** aleksandra.yankova@abv.bg

Citation: Yankova A, Georgiev K, Dimitrova D, Dimitrova-Kirilova V, Nestorova V. A patient with toxic encephalopathy associated with acute carbon monoxide poisoning–A clinical case. Electron J Gen Med. 2023;20(3):em467. https://doi.org/10.29333/ejgm/12934

ARTICLE INFO

Received: 06 Nov. 2022

Accepted: 01 Feb. 2023

ABSTRACT

Carbon monoxide (CO) is a colorless, odorless, and lacking in irritative properties gas. It has a higher affinity towards hemoglobin than oxygen and thus the complex carboxyhemoglobin develops. Acute CO poisoning leads to poly-organic insufficiency and the organs that suffer the most are the ones with higher oxygen needs- the brain, the heart and the kidneys. We present a clinical case of 49-year-old woman with a sudden appearance of complaints of dizziness, stiffness throughout the entire body, inability to move. Subsequently she was found by her relatives with disorientated and "foaming around her mouth". Earlier she had been using a gasoline generator because of a power outage. Her laboratory results came out with higher carboxyhemoglobin (40%), nitrogen waste products and mixed type acidosis. From the conducted brain MRI there were changes, correlating to toxic encephalopathy after acute CO poisoning.

Keywords: CO poisoning, intoxication, encephalopathy

INTRODUCTION

Carbon monoxide (CO) is a colorless, highly poisonous, odorless, tasteless, and flammable gas that is slightly less dense than air. The most common source is the partial combustion of carbon-containing compounds when insufficient oxygen or heat is present to produce carbon dioxide. In domestic environments the sources include malfunctioning fuel-burning appliances such as furnaces, ranges, water heaters, and gas and kerosene room heaters; engine-powered equipment such as portable generators (and cars left running in attached garages); fireplaces; and charcoal that is burned in homes and other enclosed areas. It is one of the most common causes for poisoning in a domestic environment and if not treated on time can have a high mortality rate or severe late complications.

METHODS AND RESULTS

A 49-year-old woman was admitted in a neurology department for complaints of dizziness, stiffness of the entire body, inability to move. Earlier during the day, due to a power outage, she needed to use a gasoline generator, which until that moment had not been in use. Afterwards she was found disorientated, confused and with foam around her mouth. Her relatives shared that the room smelled of gasoline and the pets, that were in the same room, were dead. The patient had no pre-existing conditions.

When she was hospitalized, the patient was conscious, disorientated, only answered with single words, with tachyarrhythmia heartbeat to 110 b/min. Her neurological status presented with a quadriparesis and rigid muscle tone for all of the limbs.

The laboratory results of the patient presented with high inflammatory markers (high white cell count-13.68×$10^{9,}$ and CRP-213.4 mg/l), high nitrogen waste products (urea-10.0 mmol/l, and creatinine-150 mcmol/l), mixed type acidosis (pH 7,046) and higher carboxyhemoglobin percentage (40%). A lumbar puncture was conducted, which produced a bloody and cloudy cerebrospinal fluid (CSF), which after centrifugation was colorless and clear. The biochemical analysis of the CSF revealed a high white cell count (11/mcl), high protein (0.77 g/l), positive Pandi and negative Rivalta. No bacterial or viral agents were found in the cerebrospinal fluid. The electrocardiogram at admission presented with tachyarrhythmia due to atrial fibrillation (140 b/min). The electroencephalogram presented with diffuse sharp waves. The brain computer tomography was normal, the chest X ray revealed pneumofibrosis. The abdominal ultrasound exam revealed bilateral nephrolithiasis. The brain magnetic resonance imaging presented with symmetrical pathological zones in the globus pallidus with characteristics of toxic encephalopathy, connected to CO poisoning (**Figure 1**).

When she was discharged the patient was conscious, oriented, without neurological symptoms, besides slight problems with concentration. The laboratory results were normal.

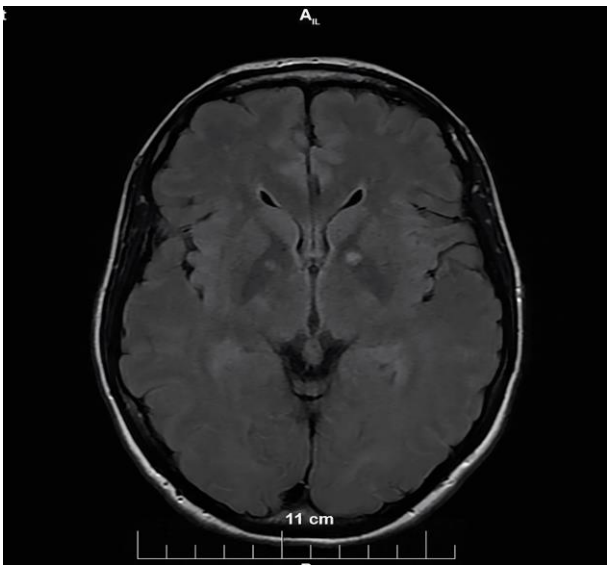

Figure 1. T1 brain MRI: Symmetrical hyperintense toxic changes in globus pallidum (reprinted with patient's permission)

DISCUSSION

CO is a colorless, odorless and lacking in irritative properties gas. It is produced in the partial burning of organic materials. The home is the most common place for CO poisoning to occur but is preventable if CO producing sources such as gas boilers, cookers and heaters are properly maintained and correctly used and by not using charcoal or wood stoves indoors. Because CO is colorless and odorless, most patients are unaware that they may have been exposed and therefore do not report this as a potential cause of their symptoms. This lack of detection leads to ongoing exposure to harmful effects of CO, with children, older people and pregnant women most susceptible to detrimental health effects [1].

The clinical manifestations of CO intoxication are often nonspecific and depend on several factors, including the concentration of inspired CO, the duration of exposure and the overall health status of the individual (pulmonary ventilation, physical condition, and the rate and efficiency of breathing) [2]. The average blood concentration of COHb varies among individuals. On the one hand, a healthy, non-smoking person has a COHb level of 2% or less. On the other hand, the level of COHb in heavy smokers is rarely above 10%. CO poisoning is asymptomatic at a blood concentration of less than 10%; at levels of 10% or greater, neurological symptoms such as nausea, headache and dizziness develop. When the average blood concentration of COHb reaches 30%-50%, increases in respiratory and heart rates, syncope, motor paralysis and confusion are observed. Beyond 50%, COHb is considered life-threatening [2].

It has a higher affinity towards hemoglobin than oxygen and thus carboxyhemoglobin develops. 90% of the absorbed CO connects to hemoglobin, while the other 10% connect to myoglobin and cytochrome C-oxidase. This leads to tissue hypoxia, oxidative stress and disorders of the mitochondrial breathing and production of ATP. Acute CO leads to polyorganic insufficiency, but the most affected organs are the most oxygen dependent ones- the brain, heart and kidneys [3, 4]. CO leads to platelet and neutrophile activation, which triggers an inflammatory cascade with interleukins, cytokines, and tumor-necrosis factor [5].

CO causes the release of nitrogen oxide from the endothelium and platelets, as well as the forming of free radicals. This leads to mitochondrial dysfunction, an increase to capillary permeability, leukocyte sequestration, and apoptosis. A reversible demyelination in the white brain matter occurs as a result of all of these changes known as Grinker's myelinopathy. It presents with cerebral oedema and bilateral necrosis of globus pallidus, the cerebellar hemispheres, the hippocampus and the cerebral cortex. It usually occurs during the recovery period and is the reason for the cognitive disorders and the extrapyramidal symptoms. Hyper proteinorachia and cerebral spinal fluid lymphocytic pleocytosis appears due to the increased capillary permeability and leukocyte infiltration through the blood-brain barrier [6, 7].

CO has a cardiotoxic effect caused by it blocking the myoglobin in the cardiac myocytes. Cardiovascular complications caused by CO poisoning have mostly been reported as left ventricular dysfunction, arrhythmias, and pulmonary edema, but it can also induce a coronary spasm and intracoronary thrombosis, leading to acute coronary syndrome even in healthy patients and in ones with non-critical atherosclerotic plaques. Some patients may have ST depressions or T wave inversions [8-11].

CO has a nephrotoxicity as a result of several mechanisms:

1. Tissue hypoxia and the free radicals.
2. The myoglobinemia due to rhabdomyolysis. Myoglobin itself is nephrotoxic.
3. The hypotension caused by the damaged heart [12].

In the beginning patients with acute CO poisoning have complaints mainly of a dull frontal headache, dizziness, nausea, fatigue. A longer exposure or a higher concentration may lead to an unstable gait, confusion, generalized tonic-clonic seizures, qualitative and quantitative changes in consciousness with visual and auditory hallucinations. In the worst cases CO poisoning can lead to coma, cardiac arrest and a quick lethal end. Arterial hypotension and a higher heart rate with episodes of atrial fibrillation are present from the cardiotoxicity of the gas. Less common manifestations caused by acute poisoning are myocardial infarction, pneumonia, pulmonary oedema, hyperglycemia, lactate acidosis, muscle necrosis, acute renal failure, skin lesions, and hearing and visual disorders [13-15].

Delayed encephalopathy after acute CO poisoning (DEACMP) is the most common and severe complication of CO poisoning. DEACMP patients suffer a series of neurological and psychiatric disorders, including cognitive dysfunction, motor impairment, extrapyramidal systems dysfunction, even consciousness disturbance after a brief lucid period, cortical blindness, depression. DEACMP may be caused by secondary brain tissue damage, due to CO-mediated brain tissue hypoxia, oxygen-based free radicals, and membrane peroxidation, instead of due to direct hypoxia-induced damage [16-19].

CASE DISCUSSION

The patient, which we have presented, exhibits a clinical picture, which can be associated to acute CO poisoning. Most CO poisonings are attributed to household accidents as is the

case here. The fact the patient was disoriented upon admission and had pyramidal and extrapyramidal symptomatic can be attributed to the presence of symmetrical pathological zones in the globus pallidus with characteristics of toxic encephalopathy in the brain MRI. The EEG can also be connected to the brain lesions. The increased inflammatory markers and spinal fluid changes can be attributed to the increased permeability due to the tissue hypoxia. The increased nitrogen waste products and the presence of atrial fibrillation can be connected to the tissue hypoxia. The high carboxyhemoglobin percentage can only be attributed to an acute CO poisoning.

In most published cases the MRI lesions in acute CO poisoning are located not only in the globus pallidus but are diffuse in the white matter [14, 20], the cerebellar hemispheres, the hippocampus and the cerebral cortex [21]. Most of the patients will also have some laboratory or ECG indicators for heart damage but in our case there was only atrial fibrillation, which was transient [22].

CONCLUSION

Acute CO poisoning is not always easy to diagnose when there is no suspicion for it, due to it mimicking many other diseases caused by the unspecific complaints and polyorganic insufficiency. The presented anamnesis of use of a gasoline generator with questionable functioning, the clinical picture, laboratory and imaging results confirmed our diagnosis of toxic encephalopathy in acute CO poisoning.

Author contributions: All authors have sufficiently contributed to the study and agreed with the results and conclusions.

Funding: No funding source is reported for this study.

Ethical statement: Authors stated that the participant received written informed consent before being admitted to the clinic. Authors further stated that the study has been approved by all the authors and the clinic which treated the patient.

Declaration of interest: No conflict of interest is declared by authors.

Data sharing statement: Data supporting the findings and conclusions are available upon request from the corresponding author.

REFERENCES

1. Jarman H, Atkinson RW, Babu A, Moss P. Cross-sectional study of carbon monoxide alarm use in patients attending the emergency department: A multicentre survey protocol. BMJ Open. 2022;12(11):e061202. https://doi.org/10.1136/bmjopen-2022-061202 PMid:36385037 PMCid:PMC9670950
2. Al-Matrouk A, Al-Hemoud A, Al-Hasan M, Alabouh Y, Dashti A, Bojbarah H. Carbon monoxide poisoning in Kuwait: A five-year, retrospective, epidemiological study. Int J Environ Res Public Health. 2021;18(16):8854. https://doi.org/10.3390/ijerph18168854 PMid:34444603 PMCid:PMC8394128
3. Rose JJ, Wang L, Xu Q, et al. Carbon monoxide poisoning: Pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med. 2017;195(5):596-606. https://doi.org/10.1164/rccm.201606-1275CI PMid:27753502 PMCid:PMC5363978
4. Hanley ME, Patel PH. Carbon monoxide toxicity. Treasure Islands, FL: StatPearls Publishing; 2022.
5. Kim SG, Woo J, Kang GW. A case report on the acute and late complications associated with carbon monoxide poisoning. Medicine (Baltimore). 2019;98(19):e15551. https://doi.org/10.1097/MD.0000000000015551 PMid:31083215 PMCid:PMC6531082
6. Oh S, Choi S-C. Acute carbon monoxide poisoning and delayed neurological sequelae: A potential neuroprotection bundle therapy. Neural Regen Res. 2015;10(1):36-8. https://doi.org/10.4103/1673-5374.150644 PMid:25788913 PMCid:PMC4357109
7. Lv Y, Zhang Y, Pan S. Brain stem and spinal cord demyelination due to acute carbon monoxide poisoning. Undersea Hyperb Med. 2021;48(3):247-53. https://doi.org/10.22462/05.06.2021.5 PMid:34390629
8. Garg J, Krishnamoorthy P, Palaniswamy C, et al. Cardiovascular abnormalities in carbon monoxide poisoning. Am J Ther. 2018;25(3):e339-48. https://doi.org/10.1097/MJT.0000000000000016 PMid:24518173
9. Ipek S, Gullu UU, Gungor S, Demiray S. The effect of full blood count and cardiac biomarkers on prognosis in carbon monoxide poisoning in children. Ir J Med Sci. 2022;1-10. https://doi.org/10.1007/s11845-022-03232-2 PMid:36445626 PMCid:PMC9707252
10. Haliga RE, Morărașu BC, Şorodoc V, etal. Rare causes of acute coronary syndrome: Carbon monoxide poisoning. Life (Basel). 2022;12(8):1158. https://doi.org/10.3390/life12081158 PMid:36013337 PMCid:PMC9410220
11. Olatunde O, Raj V, Tambe V, Szombathy T. Carbon monoxide poisoning and its effect on QTc prolongation. J Cardiol Cases. 2020;22(1):19-21. https://doi.org/10.1016/j.jccase.2020.03.009 PMid:32636963 PMCid:PMC7326656
12. Chiew AL, Buckley NA. Carbon monoxide poisoning in the 21st century. Crit Care. 2014;18(2):221. https://doi.org/10.1186/cc13846 PMCid:PMC4057251
13. Sonmez BM, Iscanli MD, Parlak S, Dogan Y, Ulubay HG, Temel E. Delayed neurologic sequelae of carbon monoxide intoxication. Turk J Emerg Med. 2018;18(4):167-9. https://doi.org/10.1016/j.tjem.2018.04.002 PMid:30533561 PMCid:PMC6261102
14. Tsai P-H, Chou M-C, Chiang S-W, et al. Early white matter injuries in patients with acute carbon monoxide intoxication: A tract-specific diffusion kurtosis imaging study and STROBE compliant article. Medicine (Baltimore). 2017;96(5):e5982. https://doi.org/10.1097/MD.0000000000005982 PMid:28151889 PMCid:PMC5293452
15. Halvorson TS, Stauffer SL, JonesKA. An elderly man with bilateral cavitary lesions in the globus pallidus. Brain Pathol. 2022;32(2):e13025. https://doi.org/10.1111/bpa.13025 PMid:34786782 PMCid:PMC8877725
16. Kumarihamy P, Kularatne SAM, Pathirage M, Gunaratne WMSN, Waduge R. A case of delayed neurological manifestation following carbon monoxide poisoning in Sri Lanka: Epidemiology of exposure and literature review. BMC Pharmacol Toxicol. 2019;20(1):17. https://doi.org/10.1186/s40360-019-0295-9 PMid:30953563 PMCid:PMC6451287
17. Huang F, Yang L, Tan Z, et al. Prognostic factors of hyperbaric oxygen therapy for patients with delayed encephalopathy after acute carbon monoxide poisoning. Heliyon. 2022;8(12):e12351. https://doi.org/10.1016/j.heliyon.2022.e12351 PMid:36582705 PMCid:PMC9792756

18. Zheng H, Liu H, Zhang G, Zhuang J, Li W, Zheng W. Abnormal brain functional network dynamics in acute CO poisoning. Front Neurosci. 2021;15:749887. https://doi.org/10.3389/fnins.2021.749887 PMid:34867160 PMCid:PMC8636030
19. Huang Y-Q, Peng Z-R, Huang F-L, Yang A-L. Mechanism of delayed encephalopathy after acute carbon monoxide poisoning. Neural Regen Res. 2020;15(12):2286-95. https://doi.org/10.4103/1673-5374.284995 PMid:32594050 PMCid:PMC7749483
20. Chou M-C, Li J-Y, Lai P-H. Longitudinal white matter changes following carbon monoxide poisoning: A 9-month follow-up voxelwise diffusional kurtosis imaging study. AJNR Am J Neuroradiol. 2019;40(3):478-82. https://doi.org/10.3174/ajnr.A5979 PMid:30765380 PMCid:PMC7028662
21. Jeon SB, Sohn CH, Seo D-W, et al. Acute brain lesions on magnetic resonance imaging and delayed neurological sequelae in carbon monoxide poisoning. JAMA Neurol. 2018;75(4):436-43. https://doi.org/10.1001/jamaneurol.2017.4618 PMid:29379952 PMCid:PMC5885203
22. Bahng Y, Baek K, Park J-T, Choi W-J, Kwak K. Carbon monoxide poisoning and developing ischemic heart disease: A nationwide population-based nested case-control study. Toxics. 2021;9(10):239. https://doi.org/10.3390/toxics9100239 PMid:34678935 PMCid:PMC8540068