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CASE REPORT

Carbon monoxide induced delayed post-hypoxic leukoencephalopathy in a morphine addict: a case report

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ABSTRACT

A 54-year-old male, marijuana smoker was found unconscious in his home because of a gas leak and gastric lavage was done, with aspirate of morphine in his stomach, after that he was hydrated. The case was then presented to us in January 2019 and non- contrast CT was done which showed multiple hypo-dense areas in white matter and basal ganglia, which was first thought to be secondary to CO poisoning and morphine. We discharged the patient. After a few days, he developed headache with associated nausea, and we gave supportive treatment. He remained cognitively healthy till he began to experience loss of consciousness with the development of aspiration pneumonia afterward. He was on mechanical ventilation. The MRI showed T2 hyperintensities in periventricular and subcortical regions, indicative of delayed post-hypoxic leukoencephalopathy, consistent with neuropsychiatric sequelae of anoxic injury because of CO poisoning.

Keywords: Morphine; Carbon Monoxide Poisoning; Unconsciousness; Leukoencephalopathy.

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INTRODUCTION

Delayed post-hypoxic leukoencephalopathy (DPHL) is a relatively rare and under-recognized condition that develops after prolonged periods of cerebral hypoxia. Because this is a not-so-common clinical entity, formal studies to find out the exact incidence are not known. However, the famous Korean prospective study of 2360 victims of Carbon Monoxide (CO) poisoning carried out in 1982 revealed that 5.5% victims developed neurologic sequelae, while 2.8% had a delayed onset. All patients who developed delayed neurologic sequelae were more than 30 years old.[1,2]

This syndrome may result from acute insults caused by benzodiazepines and methadone; however, the commonest cause is acute poisoning with CO.[1,3] There are well-documented cases of toxic leukoencephalopathies developing after exposure to anesthetic agents such as Fentanyl, intoxication with Oxycodone and Oxycontin, ICU-related, and following cardio-pulmonary arrest.[4]

Most of the patients following acute CO poisoning will present with symptoms persisting from acute to chronic phase, while only 10% will exhibit delayed neurologic sequelae after a mean lucid interval of 22 days following resolution of acute symptoms.[5] Most patients will have a preceding period of unconsciousness before the onset of neurologic symptoms except in the case of CO poisoning where 10% may not have an initial period of unconsciousness.[1] The presenting features of DPHL usually include altered cognition, incontinence, and behavioral changes in the background of typical Parkinsonian features (masked facies, rigidity, short-stepped gait, tremor), and akinetic mutism. Although cognitive symptoms may not allow performing a complete neurological examination, common examination findings may include signs of corticospinal tract involvement, and re-appearance of primitive reflexes.[6]

We present a case of delayed leukoencephalopathy following exposure to CO with an additive effect of morphine.

CASE REPORT

A 54 years old male jeweller with a history notable for depression and smoking marijuana for the last 20 years was found unconscious one morning in January 2019 with frothy discharge from the mouth and a distinguishable odor of heater gas leak in his room. He was rushed to a local emergency medical center where he was resuscitated and gastric lavage was done. The aspirate had a morphine-containing homeopathic remedy which he had been taking for hemorrhoids. He was then referred to our tertiary care hospital where the patient was hydrated, given 100% oxygen, and treated symptomatically. A non-contrast CT head examination revealed bilateral symmetric extensive hypodensities seen in the white matter and basal ganglia (putamen), possibly secondary to CO poisoning with an additive effect of morphine. The patient was discharged home in a normal cognitive and behavioral state.

The patient presented to the hospital 10 days later when he developed a gradual-onset headache associated with nausea; after receiving supportive therapy he was sent home in a normal mental status. His family reported that he had returned to his routine life self-sufficiently though becoming somewhat withdrawn. He remained cognitively fine till he began to have gradual loss of consciousness, later developing aspiration pneumonia as well.

He remained on mechanical ventilation for 10 days, and multidrug-resistant *Pseudomonas* and *Acinetobacter* were isolated from his blood. An MRI brain showed T2 hyperintensities in periventricular and subcortical regions, suggestive of DPHL, consistent with neuropsychiatric sequelae of anoxic injury due to CO poisoning. After remaining in the hospital for another few days with no improvement in the Glasgow Coma Scale, and continued supportive care, the patient was recommended to get shifted to the ward for further observation but he left the hospital because of financial problems.

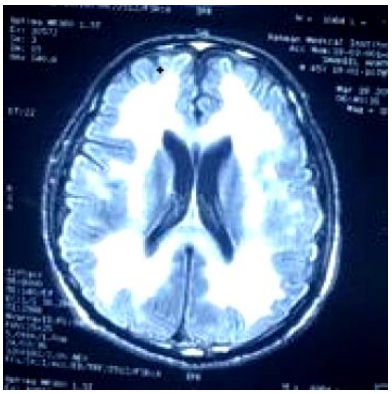

Fig (1a)

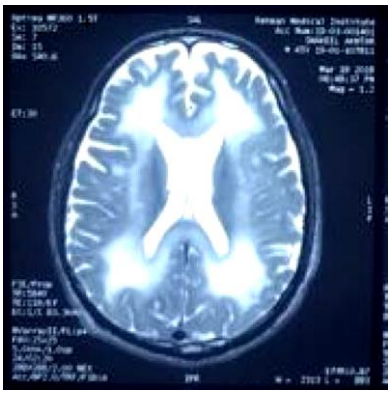

Fig (1b)

Figures 1a and 1b: MRI brain contrast images. Bilateral symmetrical white matter hyper-intensities in the periventricular and subcortical region consistent with delayed post-hypoxic leukoencephalopathy due to carbon monoxide poisoning.

DISCUSSION

Delayed post-hypoxic leukoencephalopathy (DPHL) is a syndrome symbolized by a complex blend of neurological and psychiatric symptoms days to weeks after an apparent recuperation from a coma after a lengthy interval of time in the midst of which there is cerebral hypo-oxygenation. It remains a diagnosis of exclusion where a clinical history is of utmost value, directing towards a particular cause and diagnosis; CO poisoning remains a rare yet underdiagnosed cause.[2] There have been multiple mechanisms proposed for DPHL, however, the exact mechanism still has to be deciphered. One of the mechanisms proposed is that it is mild to moderate hypoxia that causes DPHL; severe hypoxia as seen in acute hypoxic-ischemic injury damages the basal ganglia and hippocampal region, whereas in DPHL the damage is restricted to the white matter region. A similar pattern was followed in this case where there were widespread hyperintensities on T2/FLAIR involving the bilateral periventricular and subcortical white matter, with no restriction seen on DWI and no involvement of gray matter. Gottfried JA et al[7] reported a case of DPHL where the levels of enzyme arylsulfatase were decreased but instead of being completely deficient as seen in metachromatic leukodystrophy, the levels of arylsulfatase were reduced to about 27% of the normal value. Meyer MA et al[8] suggested another mechanism correlating the turnover rates of myelin-related proteins with the incidence of neurological symptoms.[8] The prognosis depends on the patient surviving the initial hospitalization period. Those who manage to survive the initial period show a good recovery. With frontal lobe-related neurological deficits, supportive care remains the treatment of choice.[9]

CONCLUSION

In DHPL, a lucid interval is followed by abrupt onset of neuropsychiatric symptoms, which progress, till within days the patient becomes unable to attend to typical daily needs. With early recognition, appropriate supportive care and rehabilitative services can be effectively utilized.

REFERENCES

1. Choi S. Delayed neurologic sequelae in carbon monoxide intoxication. Archives of Neurology. 1983 Jul 1;40(7):433-5.
2. Shprecher D, Mehta L. The syndrome of delayed post-hypoxic leukoencephalopathy. NeuroRehabilitation. 2010 Jan 1;26(1):65-72.
3. Tormoehlen LM. Toxic leukoencephalopathies. Neurol Clin. 2011 Aug;29(3):591-605.
4. Zamora CA, Nauen D, Hynecek R, Ilica AT, Izbudak I, Sair HI, et al. Delayed posthypoxic leukoencephalopathy: a case series and review of the literature. Brain Behav. 2015 Aug;5(8):e00364.
5. Beppu T. The role of MR imaging in assessment of brain damage from carbon monoxide poisoning: a review of the literature. Am J Neuroradiol. 2014 Apr 1;35(4):625- 31.
6. Shprecher DR, Flanigan KM, Smith AG, Smith SM, Schenkenberg T, Steffens J. Clinical and diagnostic features of delayed hypoxic leukoencephalopathy. J Neuropsychiatry Clin Neurosci. Fall 2008;20(4):473-7.
7. Gottfried JA, Mayer SA, Shungu DC, Chang Y, Duyn JH. Delayed posthypoxic demyelination: association with arylsulfatase A deficiency and lactic acidosis on proton MR spectroscopy. Neurology. 1997 Nov;49(5):1400-4.
8. Meyer MA. Delayed post-hypoxic leukoencephalopathy: case report with a review of disease pathophysiology. Neurol Int. 2013 Jul 22;5(3):e13.
9. Katyal N, Narula N, George P, Nattanamai P, Newey CR, Beary JM. Delayed Post-hypoxic Leukoencephalopathy: A case series and review of the literature. Cureus. 2018 Apr 15;10(4):e2481.