Delayed neurological sequelae of carbon monoxide poisoning: a case report

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ABSTRACT

Delayed neurological sequelae of Carbon Monoxide poisoning is commonest neurological complication of CO poisoning. Presentation can be confusing with other differentials like stroke and delayed neuropathy of OP poisoning. Imaging like MRI head can be helpful for diagnosis. Different predicting tools are available for the development of DNS. Early detection and prompt management of CO poisoning with normobaric oxygen can prevent DNS. Some conditions requires Hyperbaric oxygen too.

Keywords: Carbon monoxide, delayed neurological sequelae, hyperbaric oxygen therapy (HBOT)

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INTRODUCTION

Delayed Neurological Sequelae (DNS) is a serious complication that occurs within several days to 6 weeks after a complete or partial recovery from carbon monoxide (CO) intoxication1. They are recurrent–transient neuropsychiatric consequences of carbon monoxide (CO) intoxication. They manifest with alternating periods of exacerbation and remission that cause diffuse white matter or gray matter injury2. Presentation mimics with other common differentials frequently visiting to our Emergency department. However early recognition and prompt treatment of CO poisoning can prevent its neurological sequelae. Here we present a case presented in our ER.

CASE REPORT

A couple in their 40s presented in our ER with a month long history of altered sensorium, abnormal behavior and not able to feed. They gave history suggestive of prolonged exposure to smoke from burning coal. They were farmer by occupation and gave history of exposure to carbendazim (fungicide), Dieldrin (organochloride), Kitnashak (organophosphate).

Couple was found unconscious by the neighbor in their home one month back. At that time, they were taken to nearby hospital where they were managed in the line of CO poisoning and later discharged. Husband regained consciousness after 18 hours and wife after 6 hours. Few days later both developed weakness and fatigability, which was progressive. After two weeks developed abnormal behavior, roaming with no reason, short-term memory, vacant stare, tremors in hand and feet, and disorientation.

At the time of presentation to our ER both had vacant Stare, abnormal behavior, disorientation, does not speak and had abnormal body movements. Features were associated with urinary incontinence, altered consciousness, lethargy, cognitive disturbances, bowel and bladder incontinence and disturbed sleep. However, there was no history of seizures, headache, fever, and tingling sensation of limbs, shortness of breath, chest pain, apraxia or agnosia.

On Examination

GCS was E4V1M5; RR - 18 bpm; BP - 130/80 mm of Hg; heart rate - 88 bpm and SpO_2 95 percentage under room air. There was no pallor, jaundice, lymphadenopathy, clubbing, cyanosis and edema. Their hydration status was within normal limit. Systemic examination revealed normal respiratory, cardiovascular and gastrointestinal findings. CNS examination could not be done in detail since they were unresponsive. However, some objective findings were present. All four limbs were rigid to move. Reflexes: Deep tendon reflexes were exaggerated, no signs of meningeal irritations.

With all the above history and examinations, we delineate some differentials as - Delayed Neurological Sequelae of Carbon Monoxide poisoning, OP poisoning (OPIDN), Stroke, Parkinson disease, Meningoencephalitis, Dyselectrolytaemia. We did NCCT head, which showed normal scan (Figure 1). We did MRI head (Figure 2-6), which gave following report:

- Deep and subcortical T2 and FLAIR white matter hyper intensities in:
  - bilateral occipital lobe,
  - frontal lobe,
  - corona radiate and
  - centrum semiovale and
  - anterior aspect of external capsule bilaterally

- Feature are likely of delayed onset cytotoxic edema as a sequel of CO poisoning

Both were managed with multivitamins 1 cap PO OD for 1 month, Tab Thiamine 100mg TDS for one month along with Tab Haloperidol 0.25mg PO HS for 1 month and with Levodopa/carbidopa (25mg/100mg) half tablet TDS for 1 month. Both patient were discharged after 2 weeks of conservative management. Currently both are back in their hometown, Dang, Nepal. The wife has slowly improved, after the 6th week. Now can walk herself, go to the toilet herself. There was no follow up at hospital. But we followed up the patients on the phone. We talked to their son on the phone. Husband's condition is the same. He can open eyes, has subtle involuntary toe movements with the same vacant blank stare. However, he does not talk, cannot walk, has involuntary micturition and defecation.

DISCUSSION

Patient presenting with altered level of consciousness and/or with neurological deficits needs full workup, diagnosis, early intervention to prevent complication. Some differential diagnosis in such cases could be stroke, meningoencephalitis, dyselectrolytemia, Carbon Monoxide poisoning, etc. Among these, Carbon Monoxide poisoning is one of the commonest, especially during the winter season. Acute carbon

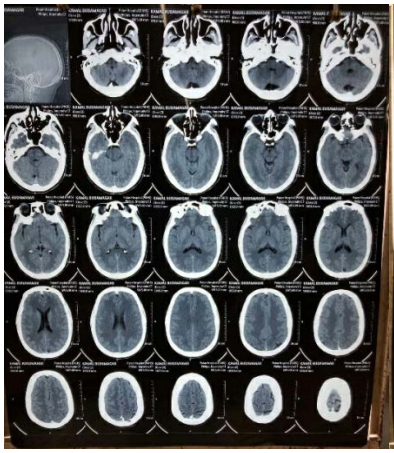

Figure 1. NCCT head

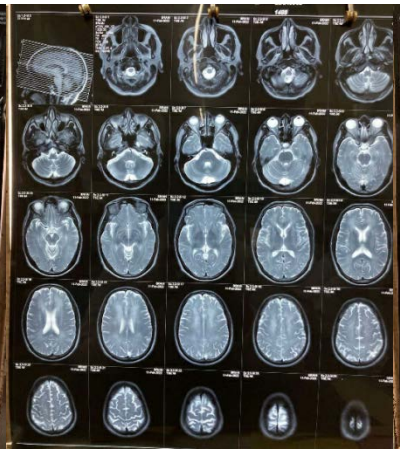

Figure 2. MRI of head

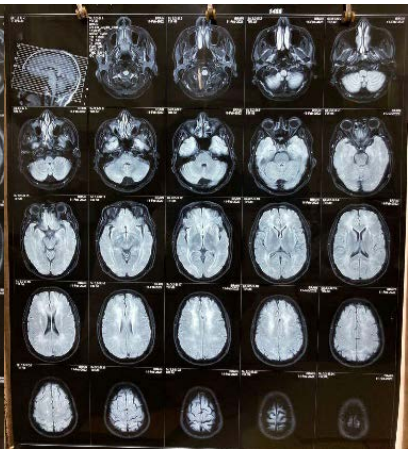

Figure 3. MRI of head

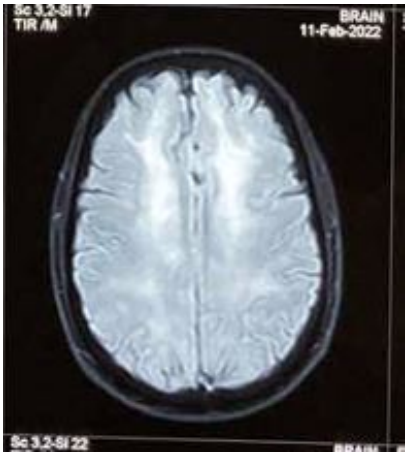

Figure 4. MRI of head

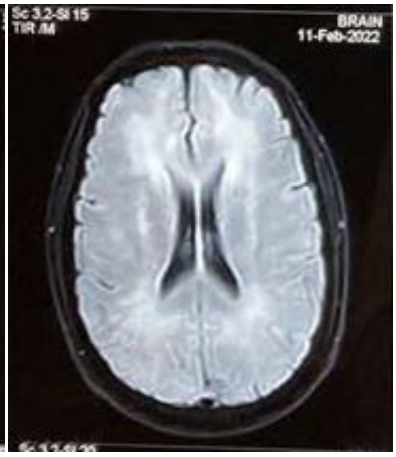

Figure 5. MRI of head

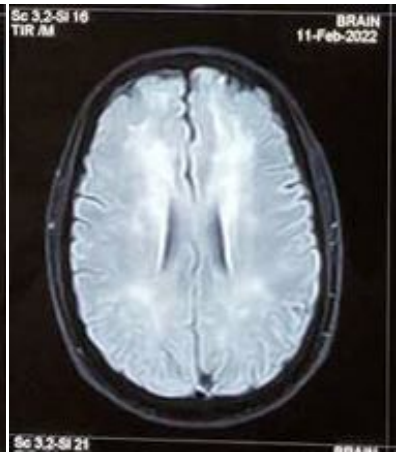

Figure 6. MRI of head

monoxide (CO) poisoning can occur due to malfunctions in heating systems or water heaters, vehicle emissions, or fires and can sometimes lead to delayed neurologic sequelae (DNS) or even death. DNS is reported in 5% to 26% of patients after CO poisoning. It is characterized by the presence of clinically silent or lucid intervals, lasting about 2 to 6 weeks after exposure, and followed by the development of DNS, including a broad spectrum of neurological deficits, cognitive impairments, and affective disorders.[1] The pathophysiology of DNS is unclear, but it is thought to be caused by brain damage associated with excitatory amino acid and oxidative stress.[2]

Many risk factors would predispose an intoxication to DNS. Some are- number of days in which walking was impossible during the acute stage (WALK) (8.5 days), attempted to commit suicide, older age, higher body mass index, hypertension, loss of consciousness, longer CO exposure, lower Glasgow Coma Scale (GCS) on-site/at emergency room, elevation of lactate, peak LDH, peak CK, high CK(>1603 U/L) on arrival, neutrophil–lymphocyte ratio (NLR) at presentation acute lesion on DWI, etc.[3-5] A predictive formula for calculating the possibility of developing delayed neurological sequelae (DNS): Risk score = −4.54 + 3.35 * (Abnormal findings on MRI = yes) - 0.51 * (Initial GCS score) + 0.65 * (Duration of exposure) + 0.01 * (CK). Then, the probability of developing DNS could be calculated: Probability of DNS = $1/(1 + e^{\text{Risk score}})$.[6]

Different tools have been studied to measure the level of CO in blood. One of the non-invasive tool is CO- oximeter. Another available but invasive technique would be blood gas analysis. Therefore, whenever we encounter a case suspicious of CO intoxication an ABG and ECG is must. Different therapeutic models have been studied for the treatment of acute CO intoxication and to prevent DNS like Hyperbaric Oxygen Therapy (HBOT), Normo-baric oxygen therapy (NBO2) with not much difference,[7] intravascular laser irradiation of blood (ILIB) therapy,[8] combine therapy of dexamethasone–mannitol–HBO2 is better in treating DNS than HBO2 alone and HBO2 combination with dexamethasone.[9] However, some conditions that necessitates HOBT are: CO level > 25 percent; CO level > 20 percent in pregnant patient; Loss of consciousness; severe

metabolic acidosis (pH <7.1); evidences of end organ ischaemia.

Regarding the prognosis and neuro-protective different tools have been studied. Higher ethanol level and higher initial GCS score has been found associated with lower incidence of DNS. Ethanol could have a neuroprotective effect on the occurrence of DNS in CO poisoning patients.[10] Some studies and case reports have mentioned about the use of dopamine agonists like levodopa either alone or in combination with other drugs like carbidopa and bromocriptine in cases of parkinsonism after CO poisoning, as in our case.[11] At the same time, other studies do not suggest for the same in the milder form of Parkinsonism after CO poisoning.[12]

CONCLUSION

CO poisoning is a silent killer. Most of the residence living are in high risk zone of CO poisoning. Among many other complications, delayed neurological sequelae is common and may remain undetected. Early detection and prompt treatment can prevent such complication.

Informed Consent

An informed consent was obtained from the patients and patients' family on sharing above information.

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