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Case Report

Delayed neurological sequelae of carbon monoxide poisoning

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ABSTRACT

Carbon monoxide poisoning is a common presentation in cold countries and usually occurs in the setting of use of improperly vented fuel devices and fireplaces. It is a rare occurrence in a tropical country like India but should always be considered when a comatose patient is found in a confined area with poor ventilation. Patients who recover the effects of acute toxicity may develop delayed neurological sequelae in the form of a broad spectrum of neurological deficits, cognitive impairments and affective disorders. Magnetic resonance imaging of the brain typically shows involvement of the basal ganglia and cortical white matter.

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1. Introduction

Carbon monoxide is an odourless, colourless gas and accidental poisoning causes hypoxia, cell damage, and death resulting in acute symptoms of headache, fatigue, confusion, nausea, shortness of breath, seizures and coma. However, some patients develop delayed complications with neurological and cardiovascular involvement. A Delayed Neuropsychiatric Syndrome (DNS) develops within a few weeks after an initial complete clinical recovery from acute poisoning in 3–40% of patients.[1] The described sequelae include a broad spectrum of neurological deficits, cognitive impairments and affective disorders. These symptoms usually resolve spontaneously in a few months but could be permanent in 25% of the cases.[2] Though frequently reported from the cold countries, literature on carbon monoxide poisoning from India is scant with only one case of DNS being reported till date.[2] We present a case of a 43 year lady who developed delayed neurological complications in the form of pyramidal and extrapyramidal symptoms with cognitive impairment after an acute exposure to carbon monoxide.

2. Report of case

A 43 year old housewife from West Bengal was admitted with history of progressive decline in cognitive and executive functions, alterations in behaviour and slowness of movements of both upper and lower limbs and tremors in the right hand over the past 2 months. She was apparently well and independently performing all the activities of daily living till ten weeks before,

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when on a cold night she along with her husband and two children slept in a closed room with no ventilation and had burning firewood for warmth throughout the night. The next morning her relatives found them unconscious and took to a local hospital where they were all administered 100% oxygen via reservoir bag. The other family members regained consciousness within 6 h with no complications but the lady continued to be obtunded for 48 h. Computed Tomography (CT) imaging of the brain done at admission was normal. The exact treatment details during the acute stage in that hospital were not available. She then gradually regained consciousness and was discharged after 4 days after which she was able to resume her normal daily activities at home. Two weeks later she noticed progressive forgetfulness and difficulty in concentration. Soon she developed urinary incontinence. Subsequently she continued to have decline in cognitive functions and eventually became bed bound within one month, requiring assistance in almost all her self-care activities. She presented to our tertiary care centre in this stage and on examination, she had multiple superficial bed sores over the sacral and gluteal regions. Rest of the general physical examination was unremarkable. Examination of higher mental functions showed spontaneous but purposeless eye opening and she moved her limbs intermittently on verbal commands. She could localise painful stimuli and speech was restricted to a few inappropriate and incomprehensible words. The Glasgow Coma Scale (GCS) score was 9/15. There were no cranial nerve deficits to the extent that could be examined. Pupillary, corneal and conjunctival reflexes were intact. Motor system examination revealed hypertonia in both upper and lower limbs with cogwheel rigidity in the right upper limb and clasp knife spasticity in the left upper and lower limbs. There was also significant truncal and nuchal rigidity. All deep tendon reflexes were brisk. Plantar reflex was flexor bilaterally. She could localise pain to deep painful stimuli. Detailed sensory examination could not be performed. Cerebellar and gait testing could not be performed. Examination of other systems was normal.

In view of above history and physical examination, possibility of carbon monoxide mediated diffuse brain injury involving pyramidal and extra-pyramidal structures was considered. Magnetic resonance imaging (MRI) of the brain (Fig. 1) showed bilateral symmetrical long TR hyperintensities in the periventricular and deep white matter (Fig. 1a) sparing the subcortical U fibres which appear bright on diffusion-weighted image (DWI) and dark on apparent diffusion coefficient mapping (ADC). (Fig. 1b and c) There were also symmetrical hyperintensities in the medial aspect of the globus pallidus, which "bloomed" on SWI indicating haemorrhagic foci. These features were suggestive of delayed post-anoxic Leukoencephalopathy, which was consistent with the history of carbon monoxide poisoning. Electroencephalogram (EEG) showed bi-hemispheric diffuse slowing of waves.

Patient was provided supportive care for bedsore healing along with a progressive ambulation programme for cognitive and physical rehabilitation. She was given tizanidine and baclofen in gradually increasing doses for spasticity. She showed significant improvement while in the hospital and started obeying verbal commands and occasional gesturing for needs. She was discharged after 3 weeks of hospitalisation to follow up after 2 months.

3. Discussion

Incomplete combustion of carbonaceous products results in formation of carbon monoxide; and therefore western data show carbon monoxide poisoning, as a common occurrence during winters. It usually occurs in the setting of use of improperly vented fuel devices, like charcoal grills, camping stoves, kerosene heaters and gasoline powered generators. In this case there was a clear history of exposure to bio-fuel combustion in closed space and the subsequent clinical features and findings raise the possibility of acute central nervous system toxicity. Carbon monoxide rapidly diffuses across the pulmonary capillary endothelium and binds to the iron moiety of haem with 240 times as much affinity as oxygen. This causes a deformational change in the haemoglobin molecule and prevents release of oxygen at tissue level, thereby causing a leftward shift of the haemoglobin-oxygen dissociation curve. It also acts as a cytotoxin by inactivation of cytochrome oxidase through binding with cytochrome aa3, causing inhibition of cellular respiration and generation of free radicals, thereby causing lipid peroxidation and cell death by apoptosis. Lipid peroxidation results in formation of adducts, thereby resulting in the formation of a chemically altered myelin basic protein in the brain, which in turn triggers an immune response, which is thought to be responsible for the delayed neuropathology seen in cases of carbon monoxide poisoning.[3]

Carbon monoxide poisoning is known to affect the cardiovascular and neurological systems. Symptoms of acute poisoning include headache, fatigue, confusion, nausea, shortness of breath, seizures and coma. Myocardial ischaemia is seen in 33% of the affected patients with cardiomyopathy, atrial thrombi and giant cell arteritis being some rare presentations of cardiac involvement in carbon monoxide poisoning.[4] This patient did not have any evidence of myocardial ischaemia in the form of ECG changes or elevated cardiac biomarkers. Neurological manifestations of carbon monoxide intoxication commonly occur in the form of delayed neuropsychiatric syndrome (DNS) characterised by cognitive deficits, personality disturbances, movement disorders and focal neurological deficits. The incidence of DNS was found to be highly variable (3–40%) with the interval for appearance from the time of exposure to carbon monoxide being 2–40 days. The most common features are urinary and faecal incontinence, gait instability, mutism, extrapyramidal signs and mental deterioration; all of which were seen in the present patient.[5] Delayed movement disorders in the form of parkinsonism, dystonia, chorea and myoclonus have been reported in 13.2% of patients with carbon monoxide poisoning. The median latency between carbon monoxide poisoning and the onset of movement disorders was 4 weeks for parkinsonism, 51 weeks for dystonia, 4 weeks for chorea and 8 weeks for myoclonus.[5] Although, initial studies had linked the severity of exposure in the form of carboxy-haemoglobin levels to the development of this syndrome, recent studies have shown that there is no association between the two.[6] Serum S100B protein level was found to be independently associated with the development of delayed neurological toxicity.[7] A recent Italian study showed that the factors independently associated with the development of DNS were carbon monoxide exposure duration >6 h, low sensorium at presentation (GCS < 9), systolic blood

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