Case Report

Atypical magnetic resonance imaging findings in hypoxic brain injury

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ABSTRACT

Here, we present the case of hypoxic brain injury in a 50-year-old male patient who was found unconscious by his colleagues in the bathroom when he didn't return for some time. On magnetic resonance imaging (MRI), there were symmetrical T2/fluid-attenuated inversion recovery hyperintensities seen involving the cortical gray matter of bilateral temporal, parietal, and occipital lobes with thickening of the cortex and effacement of overlying sulci. These areas showed acute restriction of diffusion. The findings on MRI were suggestive of hypoxic brain injury. Hypoxic brain injury in adults can be due to drowning, asphyxiation, carbon monoxide poisoning, or cardiac arrest. The gas geyser syndrome causing hypoxic brain injury due to carbon monoxide poisoning is not a very uncommon occurrence in India as the use of gas geysers with poor ventilation in bathrooms is quite common.

Key words: Aphasia, Hypoxia, Magnetic resonance imaging

Hypoxic injuries to the brain are devastating and result in high mortality and morbidity in life in all age groups [1]. Hypoxic injuries to the brain are a common cause of loss of consciousness with many etiologies, such as drowning, hanging, cardiac conditions, or carbon monoxide poisoning [2]. Computerized tomography (CT) is the first modality of choice for diagnosing hypoxic injury. Hypoxia predominantly damages the grey matter so there is hypodensity seen within the grey matter on CT scan images with loss of grey-white matter differentiation [3]. Grey-white reversal can also be seen. Magnetic resonance imaging (MRI) is the most sensitive modality for evaluating these patterns of brain injury. Hypoxic brain injury due to carbon monoxide poisoning can happen due to the usage of coal-based heating appliances in winter months or using gas geysers in bathrooms to warm the water [4]. The loss of consciousness in the bathroom while using gas geysers is a known phenomenon in India and the MRI of the patient shows classical findings in the brain that include T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities in the deep gray nuclei and peri ventricular white matter [5].

Here, we present a case of hypoxic brain injury due to carbon monoxide poisoning secondary to a gas geyser with atypical MRI findings.

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CASE PRESENTATION

A 50-year-old previously healthy male patient without any comorbidities had gone to the bathroom in the morning and was found unconscious by his colleagues in the bathroom when he didn't return for some time. The patient was brought to the hospital in an unconscious state.

On examination, the patient was unconscious; however, the vitals were stable. His pupils were bilaterally symmetrical and reactive to light. The patient regained consciousness after arrival at the hospital while he was administered oxygen by a face mask; however, he had a neurological deficit in the form of motor aphasia. The rest of his sensory and motor examination was unremarkable. He had no sensory aphasia. He also had abnormal behavior in the form of disorientation and appeared confused.

He was evaluated in the hospital and his blood profile, electrocardiogram, and color Doppler for bilateral carotid arteries were within normal limits. Urgent non-contrast CT head was also done which showed no abnormality in the brain parenchyma. MRI was done after 4 days which revealed symmetrical T2/FLAIR hyperintensities involving the cortical gray matter of bilateral temporal, parietal, and occipital lobes (Fig. 1) with thickening of the cortex and effacement of overlying sulci. These areas are hyperintense on diffusion images and show acute restriction of diffusion on corresponding apparent diffusion coefficient (ADC) images (Fig. 2). No foci of blooming were seen on gradient recalled echo images. The white matter and deep gray nuclei were unremarkable. Based on the MRI findings and clinical scenario, a diagnosis of a hypoxic

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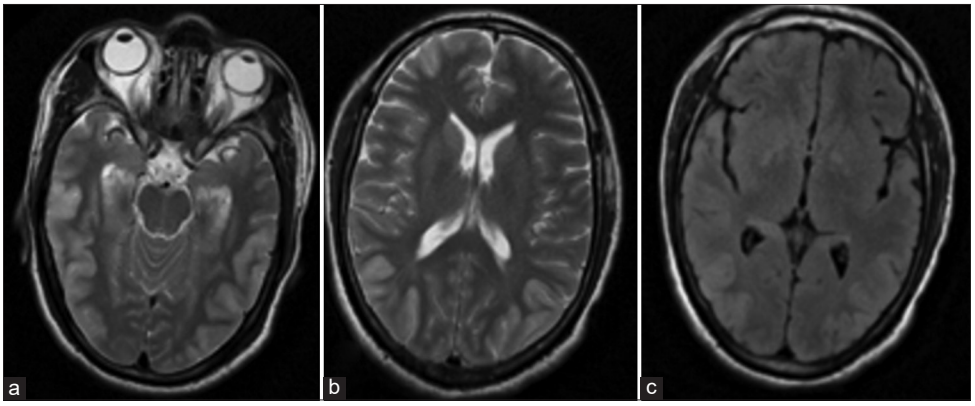

Figure 1: T2 axial (a and b) and fluid-attenuated inversion recovery (c) images show cortical hyperintensity involving the gray matter of bilateral temporal, parietal, and occipital lobes

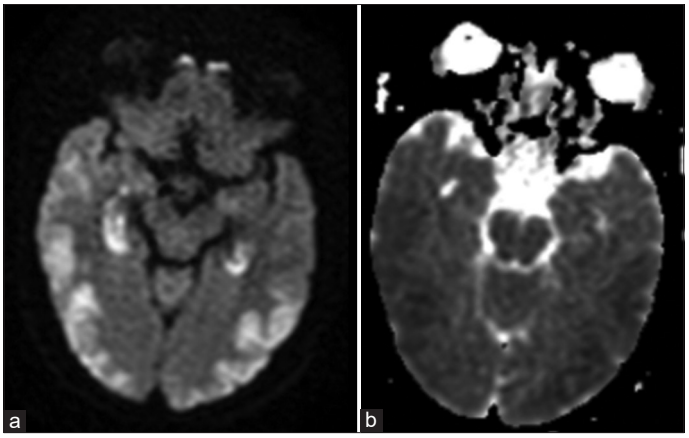

Figure 2: Diffusion-weighted imaging image (a) showing hyperintensity involving the gray matter of bilateral temporal and occipital lobes with corresponding apparent diffusion coefficient image (b) showing acute restriction of diffusion

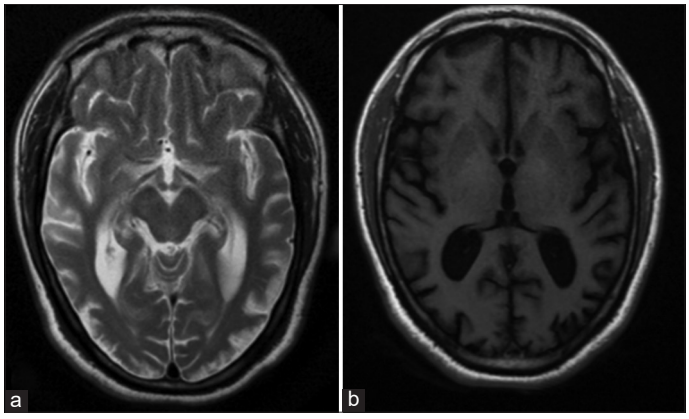

Figure 3: Repeat magnetic resonance imaging after 04 months reveals thinning of cortex (b) of the bilateral temporal and occipital lobes with T2 hyperintensity (a) consistent with encephalomalacia and gliosis

insult to the brain secondary to carbon monoxide poisoning due to a gas geyser was given.

The patient was managed conservatively and discharged after 7 days of admission. At discharge, the patient had motor aphasia; however, no other neurological deficit was there. The patient was reevaluated on follow-up after 4 months; however, there was no improvement in aphasia. Repeat MRI at follow-up had shown encephalomalacia with gliosis (Fig. 3) in the cortex of bilateral temporal, parietal, and occipital lobes.

DISCUSSION

Hypoxic brain injury in adults can be due to drowning, asphyxiation, carbon monoxide poisoning, or cardiac arrest. The gas geyser syndrome causing hypoxic brain injury due to carbon monoxide poisoning [1] is not a very uncommon occurrence in India as the use of gas geysers with poor ventilation in bathrooms is quite common in houses and offices [2]. Patients affected with gas geyser syndrome are often brought to the emergency department in an unconscious state.

The findings of hypoxic brain injury on MRI depend on the severity and duration of the insult and the interval between the insult and MRI. The typical findings on MRI in hypoxic brain injury show involvement of the deep grey nuclei (basal ganglia and thalami), peri-Rolandic cortex, occipital cortex, medial pre-frontal cortex, hippocampus, and cerebellum [2]. In carbon monoxide poisoning the classic findings involve the globus pallidus [3] with the involvement of the cerebral cortex reported much less commonly [4].

In the acute phase (<24 h), the involved areas show hyperintensity on diffusion-weighted images and restricted diffusion on corresponding ADC images due to cytotoxic edema. In the subacute stage (24 h–2 weeks), these areas are seen as hyperintense on T2 and FLAIR images and diffusion restriction starts decreasing over time. In the chronic stage, cortical laminar necrosis becomes prominent and is seen in the involved areas as gyriform T1 hyperintensity [4-6] and as time progresses encephalomalacia with gliosis develops.

In our case, the typical findings of basal ganglia involvement were not present. Our patient had atypical findings of symmetrical cortical involvement involving bilateral temporal, parietal, and occipital lobes that are hyperintense on diffusion-weighted images and show restriction of diffusion on corresponding ADC images. Similar findings have been reported earlier in a few case reports [7,8] but are not very commonly mentioned in the literature. MRI in our case was done after 4 days of incident and MRI revealed T2 and FLAIR hyperintensities in the cortex of involved areas.

A repeat MRI done after 4 months revealed T2 and FLAIR hyperintensity involving the cortex of bilateral temporal and occipital lobes consistent with encephalomalacia with gliosis. No T1 hyperintensity suggestive of cortical laminar necrosis is seen and there was no restriction of diffusion on diffusion-weighted imaging images. The diffusion restriction starts normalizing after 1 week and the same was seen in our case on repeat MRI after 4 months.

Carbon monoxide poisoning can have cognitive symptoms other than neurological symptoms. The cognitive symptoms can be general, such as confusion, disorientation, irritability, slow mental processing speed, or reduced intellectual function and symptoms can be specific, such as memory impairment, executive function impairment, apraxia, aphasia, and agnosia [9,10]. Our patient has presented with a loss of consciousness. Once the patient regained consciousness, he had aphasia that was persistent even after 4 months but no other cognitive or neurological symptoms. The actual reason for aphasia due to carbon monoxide poisoning is not clear on MRI images.

CONCLUSION

Hypoxic brain injury is a common entity seen in all age groups of patients due to varied causes. Liquid petroleum gas geysers are employed in various Indian home and office settings due to the lack of continuous electric supply in many areas of the country. This simple device may cause certain significantly disabling neurological events. Sudden loss of consciousness in the bathroom can be due to varied causes such as seizures, cardiac conditions, and syncopal episodes. The hypoxic ischemic injury caused by these various factors can happen at the time of insult or there may be continued damage even after oxygenation is established. The nature and extent of the damage appear to depend on the severity, time, course, and duration of oxygen deprivation and may have devastating neurological sequelae.

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