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Case Report

The mystery of the vision and its silent killer: A case report of optic neuropathy due to carbon monoxide poisoning

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ABSTRACT

We report a case of a young, healthy male with sudden neurological collapse in the bathroom, presenting 3 weeks later with bilateral progressive dimunition of vision. Patient's initial investigations including computed tomography (CT) brain was normal. At the time of presentation, he had severe visual impairment with defective colour vision and disc pallor bilaterally. Magnetic resonance imaging (MRI) brain was suggestive of carbon monoxide (CO) poisoning. A visual evoked potential (VEP) showed characteristic positive-negative-positive (PNP) waveform. Retrospective history confirmed the use of gas geysers in his bathroom. Patient was treated with intravenous steroids for 3 days followed by oral steroids to which he responded with improvement in visual acuity. Parenteral vitamin B12 was also administered. Optic neuropathy is a rare presentation of CO poisoning with very few cases reported in literature.

1. Introduction

Sudden loss of consciousness in the bathroom could be due to a number of causes ranging from seizures, head injury, cardiac events, stroke, syncopal attacks due to various poisonings and toxin exposure.[1] With the use of Liquified Petroleum Gas (LPG)- based water heaters, popularly known as gas geysers, in ill-ventilated small bathrooms, there has been an increase in the number of such incidents reported.[2]

We report a case of sudden neurological collapse in the bathroom presenting three weeks later with bilateral dimunition of vision, due to optic neuropathy, an extremely rarely reported occurrence according to literature.

2. Case Report

A 28-year old healthy male, accountant, was found unconscious in his bathroom after a hot water bath, without any tonic movements of the limbs or frothing at the mouth. He was rushed to a nearby hospital and given oxygen therapy, intravenous fluids and provisionally treated with antiepileptics. He regained consciousness after 30 minutes. On the day of admission, CT Brain was done and reported to be normal. All other routine blood tests, ECG and 2D echocardiography were normal. 3 days later, he developed blurring of vision. He presented to our hospital 3 weeks after the incident with blurring of vision. On examination, his visual acuity was affected (bilaterally, counting fingers at 2 metres), colour vision was severely impaired in both the eyes. Contrast sensitivity was impaired bilaterally. Pupils were sluggishly reactive to light bilaterally. Fundus examination revealed bilateral temporal disc pallor. The remainder of his general and systemic examination was normal.

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His routine blood investigations (including hemogram, liver and renal function tests) were normal. MRI brain revealed symmetrical hyperintensities in bilateral globi pallidi on T2W and FLAIR images and also showed restricted diffusion. Mild post contrast enhancement was seen in these areas. These findings are typical for carbon monoxide poisoning.[3,4] MRI orbit revealed bulky appearing left optic nerve with post contrast enhancement and hyperintense signal on diffusion weighted sequence suggestive of left optic neuritis. On the right side, thinning of the optic nerve at the intraorbital, intracanalicular and cisternal segments was noted suggestive of right optic atrophy. Visual evoked potentials (VEP) were of abnormal morphology to both flash and pattern stimulation, consisting of positive-negative-positive (PNP) waveshape and slight delay of PVEP was noted.

In retrospect, it was found that the patient had a gas geyser at his house and it was postulated that his clinical syndrome was due to acute carbon monoxide poisoning.

Due to the clinical and radiological impression of optic neuropathy, as per the advice of the neurologist, the patient was given a trial of intravenous methylprednisolone (IVMP) 1 g/day for 3 days followed by oral steroids 1 mg/kg body weight for 11 days and then tapered and stopped. Patient was also started on intramuscular vitamin B12 injection 1 mg/day for 2 weeks, followed by twice weekly and then once a month. An improvement in visual acuity was noted following 1 dose of IVMP to 6/60 and thereafter remained stable. However, colour vision, contrast sensitivity remained severely impaired. Visual field obtained prior to and after steroid therapy was not reliable due to high false positive result despite repeated attempts.

3. Discussion

Carbon monoxide (CO) is a colourless and odourless toxic gas produced as a result of incomplete combustion of carbon-based fuels. The affinity of CO for heme protein is approximately 250 times that of oxygen, and the formation of carboxyhemoglobin reduces the oxygen carrying capacity of the blood leading to tissue hypoxia. The delayed effects of CO poisoning are a result of inhibition of mitochondrial electron transport system and activation of polymorphonuclear leucocytes which undergo diapedesis and cause brain lipid peroxidation. A number of patterns of brain injury may be seen in CO poisoning.[4] The brain areas usually involved are globi pallidi, corpus callosum, thalamus, hippocampus, periventricular white matter and cortex.

White matter demyelination is responsible for delayed neuropsychiatric symptoms in these patients.[4] Memory deficits, personality changes and specific symptoms from parietal lobe involvement has been described. Visual impairment due to cortical blindness which may be transient with normal to near normal recovery has been noted in both children and elderly.[5]

It has been suggested that CO poisoning causes toxic neuropathy.[6] Animal studies have shown that CO poisoning and tobacco amblyopia produce similar abnormalities in the evoked potentials.[7] Therefore, it has been postulated that there may be a common final pathway between CO and cyanide in producing optic neuropathy. There is evidence from a previous study that the optic neuropathy caused by CO poisoning is atleast partly reversible and as in the case of tobacco amblyopia, the patient may benefit from treatment with hydroxocobalamine. Steroid for the treatment of CO poisoning may not be completely beneficial. However, a trial of steroid may not be disastrous in these cases which have poor prognosis otherwise. The improvement in visual acuity in our case cannot be solely attributed to steroid administration as spontaneous resolution may also have occurred, the two being indistinguishable clinically. Given that our case was a young patient belonging to the working class, there was a strong need to visually rehabilitate him to the greatest possible extent. As a result, with the available evidence from previous literature, a course of steroids was indicated and administered along with a course of intramuscular vitamin B12. The above trial seemed propitious in our case. However, further insight through research is needed to deem it as a standard treatment.

From the ophthalmology perspective, it may be difficult to diagnose CO poisoning in cases presenting with dimunition of vision and loss of consciousness, unless a history of gas geyser usage is evident or obtained on probing. This highlights the paramount importance of history in the diagnostic method.

4. Conclusion

The key objective of this paper is to increase the awareness regarding long-lasting and distressing threat to vision that may occur due to gas geyser associated carbon monoxide poisoning. This calls for a need for stringent policies concerning gas geyser installations.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

References

1. Singh P, Lamba A, Bansal R, Singh G. Unexplained neurological events during bathing in young people: Possible association with the use of gas geysers. *Ann Indian Acad Neurol*. 2008;11:103–5.
2. Correia P, Agrawal C, Ranjan R. Gas geyser syndrome: An important preventable cause of disabling neurological events. *Ann Indian Acad Neurol*. 2013;16(2):245–8.
3. Patil VA, Lovhale NS, Jaggi ST, Tandel SV. A Case of Gas Geyser Syndrome with Peculiar Findings on MRI Brain. *Neurol India*.

2021;69(3):772–3.
4. Lo CP, Chen SY, Lee KW, Chen WL, Chen CY, Hsueh CJ, et al. Brain injury after acute carbon monoxide poisoning: Early and late complications. *AJR Am J Roentgenol*. 2007;189(4):205–11.
5. Katafuchi Y, Nishimi T, Matsuishi T, Kimura Y, Otaki E, Yamashita Y. Cortical blindness in acute carbon monoxide poisoning. *Brain Dev*. 1985;7(5):516–9.
6. Simmons IG, Good PA. Carbon monoxide poisoning causes optic neuropathy. *Eye (Lond)*. 1998;12(Pt 5):809–14.
7. Oku H, Fukushima K, Miyata M, Wakakura M, Ishikawa S. Cyanide with vitamin B12 deficiency as the cause of experimental tobacco amblyopia. *Nippon Ganka Gakkai Zasshi*. 1991;95(2):158–64.

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