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Karaoke, power failure and carbon monoxide poisoning

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Abstract

Accidental carbon monoxide poisoning in countries with cold climates is commonly related to indoor heating. This condition appears to be relatively uncommon in tropical Asian countries and therefore the diagnosis may be unsuspected. We report a case of a Malaysian patient who presented with a severe, and ultimately fatal, delayed (biphasic) neuropsychiatric syndrome due to carbon monoxide poisoning. The diagnosis was made only when a history compatible with carbon monoxide poisoning subsequently surfaced, and neuroimaging demonstrated the typical pallidal lesions, associated with marked leukoencephalopathy, seen in this condition. Our case is unique because the poisoning occurred in the setting of indoor operation of a portable electricity generator in a karaoke centre because of power failure. Karaoke is a highly popular form of entertainment in many parts of Asia and we suggest that a high index of suspicion of carbon monoxide poisoning is required in this setting.

CASE REPORT

A previously well 58-year-old man was found unconscious in a karaoke lounge after an alcohol drinking session the night before. On being brought in to the emergency department, he was noted to be confused, without any focal neurological signs. Pulse oximeter (on supplemental oxygen) revealed 100% oxygen saturation. Blood investigations revealed only a slightly elevated level of serum alcohol; serum carboxyhaemoglobin and drug screen were normal / negative. Brain CT performed on presentation (Figure 1) showed bilateral hypodensity of the globus pallidus. His condition improved and he was discharged on the third hospital day. He was able to return to his usual activities, but was noted to be physically slow.

Two weeks later, he became confused and was unable to recognise family members. He subsequently became mute, was unable to walk and developed marked limb rigidity. There was no fever. Brain MRI performed at this time (Figure 2) showed hyperintense lesions of the globus pallidus on T2-weighted sequence (hypointense on T1 sequence). In addition, diffuse cerebral white matter signal change was evident, without contrast enhancement. MR angiography was normal.

He was brought to our hospital 6 weeks after the initial presentation, without a clear diagnosis, comatose and with marked extrapyramidal rigidity. Repeat brain MRI at this time (Figure 3) showed further worsening of the cerebral white matter changes, with restricted diffusion seen on DWI and ADC map. It emerged during further history taking from the family that he had been singing karaoke in a small enclosed room; because of a power failure, a diesel generator was operating in close proximity. At this time, a diagnosis of carbon monoxide poisoning with a severe delayed neuropsychiatric syndrome was

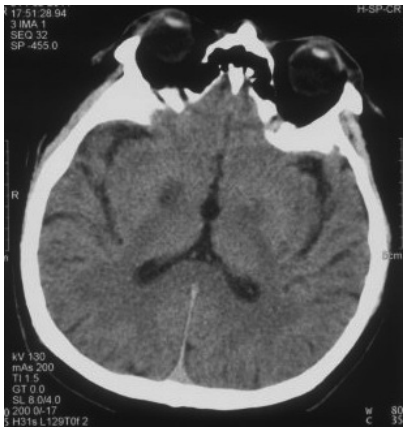

Figure 1. Non-contrasted axial brain CT, performed at initial presentation, showing bilateral hypodensity of the globus pallidus.

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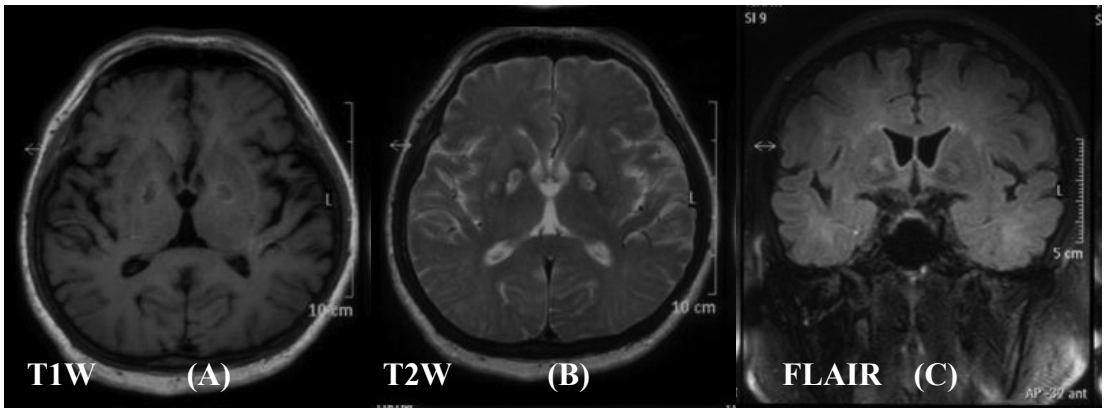

Figure 2. Non-contrasted 1.5-Tesla brain MRI axial T1 (A) and T2-weighted (B) sequences and coronal FLAIR (C) performed three weeks after the initial presentation, showing hyperintense lesions of the globus pallidus on T2-weighted sequence (hypointense on T1 sequence), and diffuse cerebral white matter signal change, without contrast enhancement.

made. He was given supportive treatment but did not show clinical improvement. He developed septicaemia due to hospital-acquired pneumonia and was ventilated for type 1 respiratory failure. Two weeks after admission to our hospital, the patient's family decided to take him home against medical advice. He passed away the following day.

DISCUSSION

The patient's clinical presentation and imaging findings were suggestive of carbon monoxide (CO) poisoning, but there was some delay before this diagnosis was reached, as the history of CO exposure (indoor operation of a portable generator in a poorly ventilated space)[1,2] was not available initially. Furthermore, accidental CO poisoning appears to be relatively uncommon in tropical Asian countries.[3] This may be because indoor heating (a common cause of accidental CO poisoning in countries with cold climates)[4,5] is not required and household windows are usually kept open.

The clinical presentation of CO poisoning is highly variable.[6,7] Patients may present with acute and/or delayed neurologic symptoms (the latter occurring days or even many months later). Clinical features of the delayed neuropsychiatric syndrome, which can be permanent, include impairment of mental status, mood changes, psychosis, movement disorders such as parkinsonism, and gait disturbance.[6,7] The reported incidence of delayed neuropsychiatric syndrome varies widely, but may be as high as 46% in CO-poisoned patients.[6,7] Predictors for the occurrence of delayed neuropsychiatric syndrome may include older age (≥36 years), longer duration of CO exposure (≥24 hours),

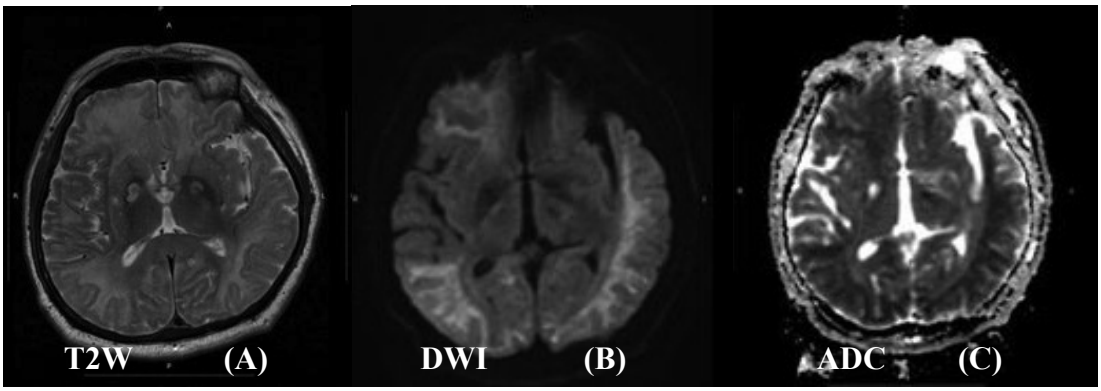

Figure 3. Axial T2-weighted 1.5-Tesla brain MRI (A) with DWI (B) and ADC map (C) performed six weeks after the initial presentation, showing further worsening of the cerebral white matter changes, with restricted diffusion on DWI and ADC map.

abnormal mental status at presentation or early during the hospital course, and more extensive brain imaging abnormalities.[7-9] On the other hand, carboxyhaemoglobin level at presentation does not appear to have predictive value[7,9]; measured levels may underestimate earlier levels because of carboxyhaemoglobin elimination over time, which is hastened by the administration of supplemental oxygen.[7] Of note, most pulse oximeters cannot distinguish carboxyhaemoglobin from oxyhaemoglobin.[6]

Our patient's illness took a biphasic course, with initial improvement of encephalopathy followed by marked worsening several weeks later. Brain CT performed during his initial presentation to hospital showed hypodensity of the globus pallidus, the commonest site of brain involvement in CO poisoning.[8,10,11] Brain MRI performed during the second phase of clinical worsening demonstrated diffuse cerebral white matter signal change without contrast enhancement, consistent with a delayed leukoencephalopathy / demyelination.[8,11]

Some randomised trial data suggest a potential benefit of treating acute CO poisoning (within 24 hours of presentation) with hyperbaric oxygen therapy, although this is controversial.[7] In our patient's case, hyperbaric oxygen was not administered due to the late presentation. There is currently no specific therapy for the delayed neuropsychiatric syndrome[7], but in view of the role of inflammation in CO–associated injury[7], anti-inflammatory therapies warrant further study.

One interesting aspect of our case is the environmental context in which the CO poisoning occurred. Karaoke, in which amateur singers sing along with a music video using a microphone, is a highly popular form of interactive entertainment in many parts of Asia. The most popular type of karaoke venue is a "karaoke box", a small or medium-sized booth or room that is typically enclosed for sound-proofing and privacy. This scenario of a poorly ventilated space may lend itself to accumulation of carbon monoxide, particularly when portable electricity generators are used.[1,2] Disruptions of electricity supply are also common in many developing regions in Asia. Although we could not find any other published cases of carbon monoxide poisoning occurring in this specific context, our case suggests that a high index of suspicion is required.

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