ORIGINAL SCIENTIFIC ARTICLES

Acute Carbon Monoxide Poisoning in a Filipino Household

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ABSTRACT

There have been multiple reports already regarding acute carbon monoxide/CO poisoning in the Philippines secondary to misuse of portable generators, especially during the times of typhoon. We present a case of unintentional carbon monoxide poisoning in a Filipino household wherein three of its members were found dead and leaving other five unconscious before they were rushed to the hospital. The index patient had increased serum fraction percentage of carboxyhemoglobin level and presented with rhabdomyolysis during admission. Neuroimaging confirmed a hypoxic-ischemic encephalopathy secondary to carbon monoxide intoxication. Even without hyperbaric oxygen therapy, patient improved with adequate hydration, early rehabilitation and trauma-focused psychotherapy.

Keywords: *carbon monoxide poisoning, carbon monoxide, household*

INTRODUCTION

The Philippines' geographical location makes it highly susceptible to natural disasters, thereby producing numerous calamity-related damages and casualties.[1] In times of post-typhoon power shortages, these diesel-powered portable generators have been used by many in the far-flung provinces.[2] However, the use of these generators has caused several accidental and unintentional carbon monoxide poisoning cases and deaths worldwide.[3,4] These pieces of equipment emit carbon monoxide and, when placed in an enclosed space, could cause buildup of CO [5] and ultimately wreak havoc to patients.

We now present a case report of acute carbon monoxide intoxication in a Filipino household due to portable generator. We specifically highlighted an index patient who was fully worked up during his admission.

Case Presentation

A 34-year old male was brought to emergency room due to altered mental status. He was seen by neighbors lying unconscious, together with his wife, their 3 children and 3 other helpers. Upon investigation, they found that the portable generator was accidentally placed inside their house, after being ran over by a strong typhoon a day prior. Three family members were already dead but the remaining unconscious members were brought to various hospitals for further management. Our emergency room had received three of them, but the index patient was referred to different specialties, including the Neurology service. Upon examination, patient had normal vital signs and was hooked to 10LPM face mask for oxygen support since there was no available hyperbaric therapy in our institution. Patient

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was encephalopathic as was seen drowsy with unsustained eye opening, converses incomprehensibly and doesn't follow commands. No lateralizing signs of weakness or numbness, and pathologic reflexes seen.

Complete blood count (shown in Table 1) showed increased white blood cell count with neutrophilic shift.

Table 1. Complete blood count

	Result	Reference
Hemoglobin	16.9	13.5-18.0 g/dL
Hematocrit	48	42-54%
Red blood cell (RBC)	5.6	4.7-6.0 x10^{12}/L
Mean corpuscular hemoglobin concentration (MCHC)	35	32-36 g/dL
Mean corpuscular hemoglobin (MCH)	30	27-31 pg
Mean corpuscular volume (MCV)	86	78-100 fL
Red cell distribution width (RDW)	12.6	11.5-15.0 %
White blood cell count (WBC)	16.82	5.0-10.0 x10^{9}/L
Neutrophils	85	37-72 %
Lymphocytes	6	20-50 %
Monocytes	9	2-9 %
Eosinophils	0	0-4 %
Basophils	0	0-1 %
Platelet	433	150-440 x10^{9}/L

Apart from the increased creatinine levels, the patient also presented with muscle tenderness and slightly darker urine output, to which the attending physicians entertained rhabdomyolysis. Table 3 shows increased urine red blood cell count and slight proteinuria. Moreover, muscle enzymes were also markedly elevated at first, but had decreasing trends as aggressive hydration continued (shown in Table 4).

Due to unavailability of a pulse carbon monoxide oximetry, a serial determination of blood gas was employed (shown in Table 5). An increasing trend of fraction percentage of carboxyhemoglobin was seen but stabilized after 4th hospital day. Apart from the carboxyhemoglobin level, patient's metabolic acidosis also resolved eventually. During this time, the patient was seen to have drastic improvement from neurological standpoint as he had already better sensorium and cognition.

He also underwent cranial magnetic resonance imaging/MRI (seen in Figure 1), which showed restricted diffusion in bilateral globus pallidus area on Diffusion Weighted Imaging/DWI sequence with associated dropout signal in Apparent diffusion coefficient/ADC map. Moreover, there is a low signal intensity in T1 weighted image with hyperintense signals on T2-weighted and Fluid-attenuated inversion recovery/FLAIR images. On gradient echo/GRE imaging, there is noted tiny foci of increased susceptibility, which may represent microhemorrhages. All of the findings represented changes seen in post-hypoxic encephalopathy, consistent with patient's exposure to carbon monoxide.

Patient was eventually started gradual rehabilitation for continuous and steady recovery. Moreover, he was referred to Psychiatry service for trauma-specialized psychotherapy. He was also started with an antidepressant prior to discharge. The two other family members admitted at our institution were also discharged well and stable.

DISCUSSION

Carbon monoxide/CO is a colorless, non-irritating gas which is a byproduct of combustion. Fire-related smoke inhalation is responsible for majority of CO poisoning cases. However, it is also seen in fuel-burning

Table 2. Serial blood workups during admission

	Reference	**1st Hospital Day**	**2nd Hospital Day**	**3rd Hospital Day**	**4th Hospital Day**	**5th Hospital Day**	**6th Hospital Day**
Creatinine	64-104 umol/L	198.3	356.1	576.30	552.80	449.30	340.70
Aspartate Transferase/AST	5-34 U/L	1,135	897	357	157	Not extracted	Not extracted
Alanine Transferase/ALT	5-55 U/L	122	158	97	61	Not extracted	Not extracted

Table 3. Urinalysis

	Result	Reference
Color	Yellow	Yellow
Appearance	Slightly hazy	Clear
Specific Gravity	1.025	1.005-1.025
pH	5.5	4.5-8
Sugar	Trace	Negative
Ketones	Negative	Negative
Blood	3	0
Nitrite	Negative	Negative
Protein	2	0
Bilirubin	Negative	Negative
Urobilinogen	Negative	Negative
Leucocytes	Trace	Negative
Urine RBC	24	0-2/hpf
Urine WBC	5	0-2/hpf
Urine Epithelial Cells	0	0-10/hpf
Bacteria	2	0

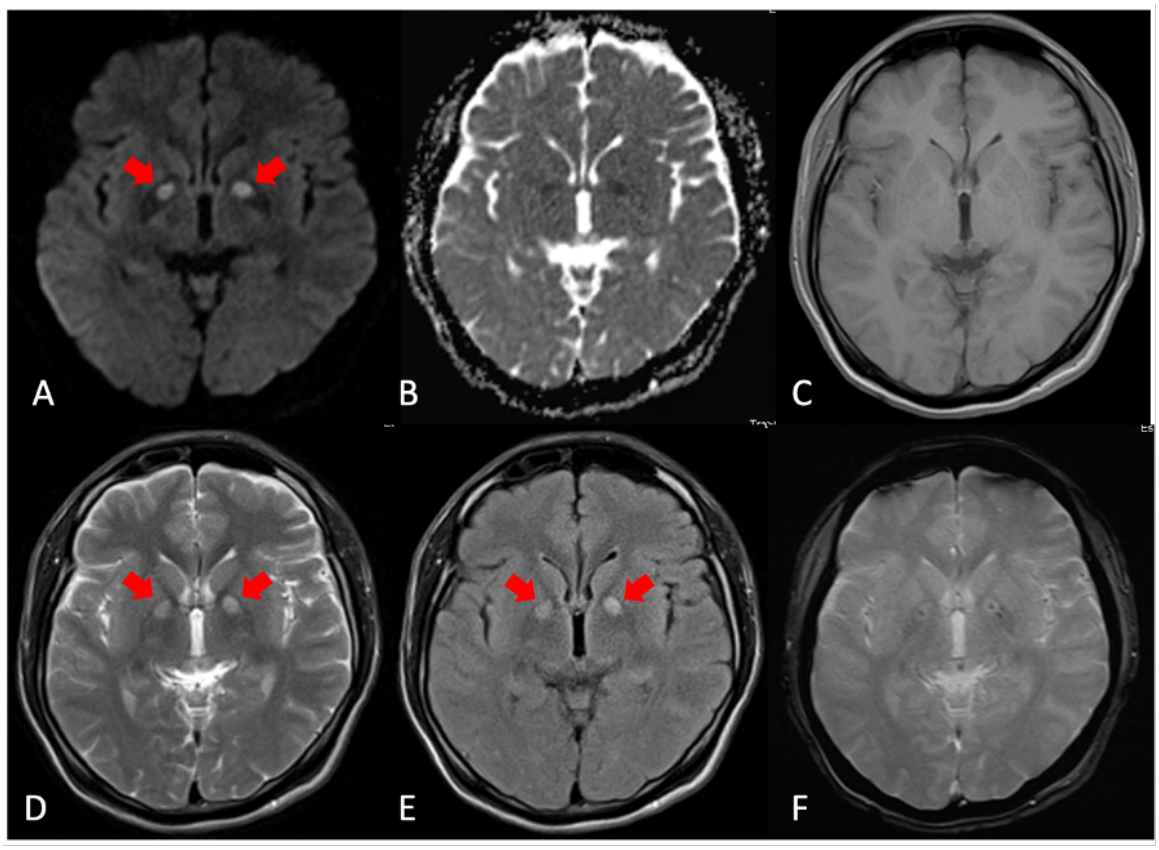
Table 4. Muscle Enzymes

	Reference	**1st Hospital Day**	**2nd Hospital Day**	**3rd Hospital Day**	**4th Hospital Day**	**5th Hospital Day**	**6th Hospital Day**
Total Creatine Kinase/CK	30-200 U/L	14,089	4,267	6,708	4,187	3,127	1,830
CK MM (skeletal muscle)	27-175 U/L	13,519	3,916	6,499	4,049	3,006	1,762
CK MB (heart)	3-25 U/L	570	351	209	138	121	68

Table 5. Serial Arterial Blood Gas Determination

Arterial Blood Gas	**1st Hospital Day**	**2nd Hospital Day**	**3rd Hospital Day**	**4th Hospital Day**	**5th Hospital Day**	**6th Hospital Day**
pH	7.312	7.284	7.338	7.453	7.406	7.472
pCO2	36.5	35.3	37	35	40.4	33.3
HCO3	18.7	17.2	20	25.2	24.9	25.5
pO2	312	287	246	75.5	75.4	87.2
Saturated O2 (%)	97.7	97.8	98.1	95.1	94.6	96.6
Fraction of inspired oxygen/FiO2 (%)	60	40	60	40	40	40
Fraction of oxygenated hemoglobin/FO2Hb (%)	93.6	94	94.4	91	90.6	92.1
Fraction of carboxyhemoglobin/ FCOHb (%)	0.2	0.3	0.4	0.9	0.8	0.7
Fraction of methemoglobin/ FMetHb (%)	4	3.6	3.4	3.4	3.4	3.2

Figure 1. MRI axial, cross sectional view a) Diffusion weighted imaging/DWI B) Apparent diffusion coefficient/ADC map C) T1-weighted D) T2-weighted E) T2-Fluid attenuated inversion recovery/FLAIR F) Gradient echo/GRE.
*Red arrows pointing to lesions situated around bilateral globus pallidus



devices, gasoline-powered generators, and motor vehicles operating in poorly ventilated spaces.[3,6]

As the carbon monoxide gets inhaled, it binds to the iron moiety of heme with significant affinity compared to oxygen. This then results in decreased oxygen delivery to tissues, leading to subsequent damage.[7] A wide range of symptoms, from non-specific constitutional symptoms like headache, drowsiness, fatigue, etc. to altered mental status leading to coma, can be seen. However, it all depends on the fraction inspired percentage of carbon monoxide, patient's baseline co-morbidities, and duration of exposure to the toxin.[8]

Studies have also demonstrated carbon monoxide intoxication producing delayed neurologic sequalae/DNS which appear around 2-40 days after a lucid interval post-intoxication followed by recurrent intermittent neuropsychiatric symptoms.[6,9] The MRI findings of such showed lesions in basal ganglia, white matter, or globus pallidus[9,] which were present in our index patient.

Rhabdomyolysis as a complication of carbon monoxide poisoning has been reported in literature. Muscle necrosis secondary to CO poisoning leads to release of myoglobin from skeletal muscle, which in turn causes acute kidney injury. Although the increased muscle tenderness correlated with increased muscle enzymes didn't lead to an acute compartment syndrome, which is also a common cause of rhabdomyolysis[10], patient was managed conservatively through aggressive hydration and continuous surveillance.

Hyperbaric oxygen therapy has been regarded as initial therapy for acute carbon monoxide poisoning. However, this alone as a standard therapy is still unclear and remains controversial.[6,7,9] Also, this therapy still has

undetermined effects to the carbon monoxide-associated delayed neurologic sequelae.[9]

CONCLUSION

Early recognition of a patient's clinical exposure to carbon monoxide or to any chemicals, correlated with signs and symptoms, can aid a clinician in the treatment and management. Furthermore, this provides an avenue for prompt response from government and healthcare officials to attend to these certain situations.

ACKNOWLEDGEMENTS

None

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