

## Mass Carbon monoxide poisoning in a family: A Case Report from Nepalgunj

Rajesh Kumar Mandal,<sup>1</sup> Nirmal Shakya,<sup>1</sup> Sanket Kumar Risal,<sup>1</sup> Rajani KC Shah<sup>1</sup>

<sup>1</sup>Department of Internal Medicine, Bheri Hospital, Nepalgunj, Nepal.

### ABSTRACT

Carbon monoxide (CO) is a colorless, odorless gas produced as a by-product of complete combustion of hydrocarbons. CO binds rapidly to Haemoglobin (Hb), leading to the formation of carboxyhemoglobin (COHb), so the oxygen carrying capacity of the blood will be decreased and causing tissue hypoxia. We present the case of accidental carbon monoxide poisoning in a family where four members were reported dead and three unconscious in the morning after sleeping in the room with burning briquettes. Carbon monoxide poisoning can be fatal. Public awareness about such poisoning is essential for prevention.

**Keywords:** Carbon monoxide poisoning; carboxyhemoglobin; hypoxia.

### INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless gas produced as a by-product of incomplete combustion.<sup>1</sup> Industrial workers at pulp mills, steel foundries, and plants producing formaldehyde or coke are at risk for exposure, as are personnel at fire scenes and individuals working indoors with combustion engines or combustible gases.<sup>3</sup> The diagnosis of carbon monoxide poisoning is frequently made obvious by the patients history of exposure.<sup>2</sup> Carbon monoxide has been called “the disease of a thousand faces” and its symptoms range from vague malaise or flu like symptoms to profound central nervous dysfunction with overt psychiatric manifestations.<sup>4</sup>

### CASE REPORT

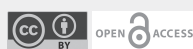
A extended family with seven members along with two children, one of 3 years and other of 3 months happened to sleep in the same room with doors and

window closed during the winter months of 29th December 2021 with burning briquettes so as to keep the room warm. Husband 26 year male was a caretaker of a government office at Nepalgunj. At 6 am in the morning, there was power cut and one of his fellow mate called him on telephone for generator room’s key, in spite being ringed several times there was no response. Then he decided to reach his room and knocked several times. Caretaker’s 24 year wife somehow opened the room. He tried to wake him up, but could not. He shouted help and others gathered. They were rushed to the hospital which was nearby. Caretaker, his 53 year old mother, 3 month male child, and 29yr female (relative) was declared brought dead at Emergency department of Bheri Hospital at 6:15 am. His younger brother aged 18 was intubated for severe respiratory distress. Caretaker’s wife and 3 year child was kept in ICU for observation with oxygen face mask. The lady and the child gained consciousness shortly. Here we describe the presentation and management of the 18 year male patient who developed a severe cardiac dysfunction, leading to profound cardiogenic shock and respiratory insufficiency after CO poisoning.

The patient was rushed to Emergency Department of Bheri Hospital at 6:15 am. The patient was agitated

#### Correspondence

Dr. Rajesh Kumar Mandal.  
Email: rkmandal338@gmail.com



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**Figure 1: Patient under Ventilator support in ICU.**

and was in severe respiratory distress. His saturation in room air was 25% at presentation. His blood pressure was 90/60, pulse was 110 per minutes. He was disoriented and agitated. There was severe cyanosis which could be seen on lips and digits of fingers. His face was congested and conjunctiva was suffused. There was cherry red frothing from his mouth. There were basal crepts in his bilateral lungs.

His spontaneous breathing was shallow and weak with high flow O<sub>2</sub> mask. The saturation hardly rose to 60%. His ABG was sent. He was intubated and supplied with 100% oxygen and shifted to Intensive Care Unit (ICU) Figure 1. During intubation cherry red froth was suctioned out. His ABG showed: pH: 7.310, pO<sub>2</sub>: 20, pCO<sub>2</sub>: 42.3 mmHg, HCO<sub>3</sub>: 18.4, sO<sub>2</sub>: 27% and SBE: -5.4. His blood pressure dropped and dopamine was started at lower dose of 5 mcg/kg. Chest X-ray was done and routine blood investigations were sent. The chest X-ray revealed multiple patchy infiltrations over bilateral lungs field. Figure 2. Echocardiography showed an ejection fraction of 45 %. Four hours after admission, the ABG was repeated at 10:30am. The reports were pH: 7.374, pO<sub>2</sub>: 311, pCO<sub>2</sub>: 31.6 mmHg, HCO<sub>3</sub>: 18.1, sO<sub>2</sub>: 100%, SBE: -7.2.

Meanwhile the patient was managed with iv fluids and dextrose. The patient gained consciousness at



**Figure 2: Chest X-ray of patient.**

4pm in the same day. He could follow the command. The patient was kept on CPAP on spontaneous mode. The patient's cardiovascular and respiratory status kept improving. The blood pressure also became stable 120/80 mmHg. The dopamine was titrated down and was stopped at morning 6am next day. The urine output was 1400ml over 24 hours. The patient was extubated on the second day at 9 am. His neurological findings were normal after extubation. He was transferred from the ICU to ward on the 3<sup>rd</sup> day and he no longer required oxygen. He was observed for few days. The chest X-ray and routine blood reports were repeated on the 5<sup>th</sup> day. All were within the normal limits. He was then discharged in accompany with his visitors. Figure 3



**Figure 3: Patient at discharge.**

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

## DISCUSSION

Carbon monoxide (CO) is a gas resulting from incomplete combustion. While almost certainly under-diagnosed, there are many documented serious poisonings every year most commonly from faulty gas heaters, burning briquettes, and household fire.<sup>5</sup> In this case burning briquettes were used. The room was small around 120 feet square and poorly ventilated, more over due to cold the doors and windows were closed, which leads to carbon monoxide poisoning. A study revealed that people using firewood for cooking in comparison with natural gas were more vulnerable to CO poisoning. Firewood generates more CO emission as compared to gas (52.8 to 14.2 gm/kg).<sup>6</sup> Carbon monoxide binds to hemoglobin reversibly with an affinity approximately 240 times greater than that of oxygen, thereby reducing the total oxygen-carrying capacity of the hemoglobin. This competitive binding shifts the oxygen-hemoglobin dissociation curve to the left, resulting in the impaired release of oxygen at the tissue level and cellular hypoxia.<sup>7</sup>

Carbon monoxide poisoning has been associated with amnesia, encephalopathy, dysarthria, parkinsonism, peripheral neuropathy, bullous skin lesions, supranuclear gaze palsy, cerebral haemorrhage, cardiotoxicity and muscle necrosis with renal failure.<sup>8</sup> CO binds to myocardial myoglobin more slowly than it does to hemoglobin, but the bond is stronger and the release slower.<sup>7</sup> CO-related cardiovascular dysfunction includes angina, myocardial infarction, arrhythmia, left ventricular dysfunction, transient

myocardial stunning, cardiogenic shock, and sudden death.<sup>8</sup>

This patient developed pulmonary edema which was evident by cherry red frothy secretions during intubation, basal crepts in bilateral lungs and acute respiratory distress with saturation dropping to 25% in room air. The possible causes of pulmonary edema include toxic effects of CO on the alveolar membranes, myocardial damage leading to left ventricular failure, the aspiration of gastric contents after loss of consciousness, and neurogenic pulmonary edema.<sup>7,9</sup> In room air the half-life of COHb is up to 5 hours; with high-flow oxygen and a reservoir mask this reduces to approximately 70 minutes; and hyperbaric oxygen reduces this further to about 25 minutes.<sup>10</sup> In this case the repeat ABG at 10:30 am showed pH:7.374, pO<sub>2</sub>: 311, pCO<sub>2</sub>: 31.6 mmHg, HCO<sub>3</sub>: 18.1, sO<sub>2</sub>: 100%, SBE: -7.2 which denoted improvement from hypoxia. The patient recovered successfully with aggressive cardiopulmonary resuscitation, including inotropic agents and ventilator support.

## SUMMARY

Burning fire woods, briquettes, gas heaters in a poorly ventilated room or spaces are potential and common source of accidental poisoning which can be fatal. Public awareness and health education regarding the sources and dangers of CO poisoning can be helpful in decreasing the morbidity and mortality from CO poisoning in Nepal.

**Conflict of interest:** None

NJHS

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