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## Case Report

# Clustered Carbon Monoxide Poisoning Cases: Accidental Poisoning In A Vehicle

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**Abstract:**

*Carbon monoxide (CO) is a product of combustion of organic matter in the presence of inadequate supply of oxygen. Common sources are burning fuel, engine exhaust, burning of animal dung, heater emissions and gas geyser. It is a toxic, clear, colorless and tasteless gas. The clinical presentation runs a spectrum, ranging from headache and dizziness to coma and death. Here we report three cases that presented to us in the month of November 2023 with history, sign & symptoms suggestive of CO poisoning.*

***Key words:*** *Carbon monoxide, carboxyhemoglobin, engine exhaust, toxic gas.*

### Introduction

CO poisoning can be both accidental and incidental and it continues to be a significant cause of morbidity. Exposure to CO is a serious health concern because individuals can be severely or fatally poisoned before even realizing that they have been exposed. Patients who survive acute poisoning are at risk of delayed neurologic sequelae[1]. The initial symptoms of CO poisoning are primarily nausea, fatigue, tachypnoea, headache, confusion and clumsiness, which are non-clinical effects that often lead to underdiagnosis or misdiagnosis of CO exposure[2]. Diagnosis is primarily based on history, circumstantial evidence and clinical examinations. Co-oximetry test is useful for detecting carboxyhemoglobin (COHb) level. Here we are reporting three cases of accidental carbon monoxide poisoning.

### Case report 1

A 42 years old male, a known case of diabetes mellitus, hypertension, known smoker, was found drowsy in his car along with 2 other people. Car engine was running and air conditioner was on. There was no evidence of associated convulsion, tongue bite, urinary incontinence, vomiting or trauma. He was brought to emergency department (ED). Patient was still drowsy. Pupils were normal in size, reacting to light. Plantar responses were flexor bilaterally and other systemic examinations were normal. Arterial blood gas (ABG) was done. Co-oximetry revealed raised carboxyhemoglobin (COHb) level (44.6%). Oxygen supplementation was started with a non rebreather mask (NRM) at 15L/min. Patient was shifted into ICU. Patient's consciousness level improved gradually. Follow up ABG was done after 5 hours which showed significantly reduced COHb level (6.7%). Another ABG was done in the next morning which showed normal carboxyhemoglobin level (0.9%). High Sensitive Troponin I (hsTnI) was raised (190 pg/ml in 1st sample and 887.9 pg/ml in 2nd sample). ECG was within normal limit. Echocardiography was normal as well. Patient took leave against medical advice in the following day.

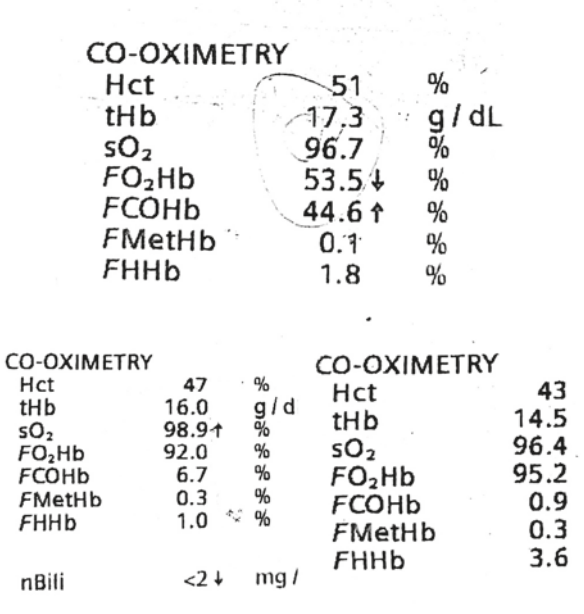


Figure 1: Serial ABG of Case 1 in emergency and ICU

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**Case report 2**

A 13 years old male was found unconscious in the front seat of same car. This boy had bladder incontinence and complained of headache, & palpitation prior to unconsciousness. Patient had no evidence of neck rigidity, convulsion, tongue bite or vomiting. Plantar response was withdrawal bilaterally. Pupils were constricted on both sides. His initial ABG showed raised COHb level (49.5%). Patient was given 15L/min oxygen supplementation via NRM. Patient was given admission in ICU and his consciousness level improved over next few hours. Another ABG was done after 2 hours and that showed normal COHb level (2%). His blood hsTnI was found raised in 2 blood samples taken 12 hours apart. 1st sample showed 446 pg/ml while 2nd sample revealed 1684 pg/ml. Echocardiography showed no regional wall motion abnormality and adequate left ventricular ejection fraction. Patient took leave against medical advice on the following day. They did consult a cardiologist later on and patient was asymptomatic.

| )-OXIMETRY | | |
|---|---|---|
| Hct | 45 | % |
| Hb | 15.4 | g / dL |
| $O_2$ | 97.2 | % |
| $O_2Hb$ | 48.8↓ | % |
| COHb | 49.5↑ | % |
| MetHb | 0.3 | % |
| HHb | 1.4 | % |
| Bili | 2.2 | mg / dL |

| CO-OXIMETRY | | |
|---|---|---|
| Hct | 44 | % |
| tHb | 15.1 | g / dL |
| $sO_2$ | 75.2↓ | % |
| $FO_2Hb$ | 73.7 | % |
| FCOHb | 2.0 | % |
| FMetHb | 0.0 | % |
| FHHb | 24.3 | % |
| nBili | <2↓ | mg / dL |

Figure 2: Serial ABG of Case 2

**Case report 3**

A 52 years old male was found drowsy in the back seat of same car mentioned above. He had no history of convulsion, tongue bite, bladder incontinence or vomiting. Patient's COHb level was 35.1% in ER. He also received 15L/min oxygen supplementation by NRM. His consciousness level improved rapidly and was admitted in ICU for further monitoring. Follow up co-oximetry showed normal COHb level at 1.2% His routine investigations revealed no other abnormality. Patient took leave against medical advice next day. Follow up calls were made and patient's family informed that he was asymptomatic.

| CO-OXIMETRY | | |
|---|---|---|
| Hct | 39 | % |
| tHb | 13.2 | g / dL |
| $sO_2$ | 96.7 | % |
| $FO_2Hb$ | 62.5↓ | % |
| FCOHb | 35.1↑ | % |
| FMetHb | 0.3 | % |
| FHHb | 2.1 | % |
| nBili | <2↓ | mg / dL |

| CO-OXIMETRY | | |
|---|---|---|
| Hct | 44 | % |
| tHb | 14.9 | g / dL |
| $sO_2$ | 97.9 | % |
| $FO_2Hb$ | 96.4 | % |
| FCOHb | 1.2 | % |
| FMetHb | 0.3 | % |
| FHHb | 2.1 | % |
| nBili | <2↓ | mg / dL |

Figure 3: Serial ABG of case 3 in ED and ICU

**Discussion**

Car exhaust fumes can contain up to 8% of carbon monoxide in enclosed spaces and is a very potent source of both accidental and suicidal cause of carbon monoxide poisoning [1]. It is generally considered that a COHb saturation level of 40% or more is fatal[5]. CO binds to hemoglobin with an affinity 200 to 250 times greater than that of oxygen. Toxicities result from impaired release of oxygen at the tissue level, causing cellular hypoxia and possibly direct CO mediated damage at the cellular level. Carboxyhemoglobin levels can be measured in either venous or arterial blood. Normal carboxyhemoglobin level is less than 5% but may be as high as 10% in smokers. Level is higher in urban regions compared to rural areas. CO poisoning can present with a wide spectrum of clinical manifestations, ranging from mild symptoms, including dizziness or headache, to very severe intoxications, which may result in coma, shock, or death [6]. CO poisoning can also lead to delayed neuropsychiatric complications and impaired cognitive function. Treatment includes immediate removal of the victim from the exposure and administration of high-flow or 100% oxygen by a nonrebreather reservoir oxygenmask[7]. Intubation may be necessary in patients exposed to CO from fire. Hyperbaric oxygen therapy shortens the half-life of carboxyhemoglobin to 15 to 30 minutes compared with 40 to 80 minutes when patients breathe 100% oxygen. Hyperbaric oxygen treatment may decrease postexposure cognitive deficits[7]. However, controversy exists over the specific criteria for instituting hyperbaric oxygen therapy in CO poisoning[8,9].

In our cases, diagnosis was made on the basis of history, circumstantial evidence, co-oximetry report and response to high flow oxygen therapy. Patients improved rapidly after oxygen supplementation and there was no residual neurological deficit. Some degree of myocardial injury has been reported in up to one-third of patients with moderate or severe CO poisoning, and those who survive appear to have an increased risk of mortality [10]. There have been reports of CO poisoning induced Takotsubo syndrome[11]. In a separate study, focusing only on CO-poisoned patients with high troponin I levels, the incidence of a Takotsubo-like pattern was around 23% [12]. Two of our patients had raised hsTnI level. Although their echocardiography showed normal findings. The cause of CO poisoning in these cases could be due to prolonged exposure to higher levels of carbon monoxide resulting from faulty exhaust system of their car.

**Conclusion**

These cases stress on the dangers of possible exposure to toxic levels of CO due to inadequate ventilation. Clinicians should suspect CO poisoning in cases of patients found in an unconscious state from an enclosed space. A high index of suspicion, early recognition and management can result in a favorable outcome. There is no specific antidote for this poisoning but high flow oxygen therapy is life saving.

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