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Case Report

Oxygen Relieved Carbon Monoxide Toxicity-Induced Coronary Vasospasm and Focal Atrial Tachycardia

Yasser Mohammed Hassanain Elsayed*

Critical Care Unit, Fraskour Central Hospital, Damietta Health Affairs, Egyptian Ministry of Health (MOH), Damietta, Egypt

***Corresponding Author:** Yasser Mohammed Hassanain Elsayed, Critical Care Unit, Fraskour Central Hospital, Damietta Health Affairs, Egyptian Ministry of Health (MOH), Damietta, Egypt.

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Abstract

Background: The current case was an adolescent patient presented with acute carbon monoxide poisoning. The case manifested with acute loss of consciousness with coronary vasospasm and atrial tachycardia. This had happened during bathing. The family had given a history of heater gas escaping. History and clinical data were the cornerstones for diagnosis. The case was treated only with 100% oxygen in the critical care unit. Lack of knowledge in the diagnosis and follow up was the absence of carboxyhemoglobin concentration assessment.

Aim of the Study: Aim of this case study was declaring the role of non-hyperbaric oxygen in the management of carbon monoxide intoxication. The current case can't compare to other studies due to absent of similar condition. A working hypothesis was symptomatic management for the case.

Methods: The study was an observational case study.

Results: Dramatic recovery of loss of consciousness, reversal of coronary artery spasm, and termination of focal atrial tachycardia had occurred with non-baric 100% oxygenation.

Conclusion: Non-baric oxygenation possesses a pivotal role in the reversal of brain and myocardial injury due to hypoxia of carbon monoxide toxicity.

Keywords: *Induced Coronary Vasospasm; Oxygen Relieved Carbon Monoxide Toxicity; Focal Atrial Tachycardia; Oxygen; Carbon Monoxide Toxicity*

Abbreviations

AT: Atrial Tachycardia; CAS: Coronary Artery Spasm; CO: Carbon Monoxide; COHb: Carboxyhemoglobin; ECG: Electrocardiograph; ED: Emergency Department; GCS: Glasgow Coma Scale; HBO: Hyperbaric Oxygen Therapy; IHD: Ischemic Heart Disease

Introduction

Coronary artery spasm (CAS) is a syndrome characterized by sudden chest pain due to epicardial coronary artery spasm which usually leads to transient myocardial ischemia, with chest pain and ECG changes [1]. Coronary artery spasm plays an important role in the pathogenesis of ischemic heart disease (IHD), including stable angina, unstable angina, myocardial infarction, and sudden death [2]. Coronary artery spasm is associated with ST-segment depression rather than ST-segment elevation on electrocardiography [3]. It may also occur

in angiographically normal coronary arteries as so-called 'variant of the variant [3]. Coronary angiography is the gold standard for the diagnosis of variant angina [4]. Calcium antagonists are the cornerstone of medical treatment [4].

The atrial tachycardia (AT) is a fast arrhythmia in which the electrical impulse originates in atrial tissue different than the sinoatrial node [5,6]. Atrial tachycardias can be classified as focal or macro-reentrant, depending on their mechanism, origin, and propagation of the electrical impulse [5]. Sudden onset and offset of a regular narrow complex (QRS < 120 ms) tachycardia coincidental with the presenting symptoms is very suggestive [7]. Focal atrial tachycardia, formerly known as paroxysmal atrial tachycardia is characterized as a fast rhythm from a discrete origin, discharging at a rate that is generally regular, and conducting in a centrifugal manner throughout the atrial tissue. Focal atrial tachycardia can be sustained or non-sustained [7]. The atrial rate during focal atrial tachycardia is usually between 100 and 250 bpm [7]. Focal atrial tachycardias are rather infrequent and most commonly seen in normal hearts and younger patients [5,6]. Macro-reentrant atrial tachycardias are more frequent among elderly patients with a history of structural heart disease [5,6]. The underlying mechanism of focal atrial tachycardia can be automatic, triggered activity, or micro-reentry [7]. The ECG is the cornerstone of diagnosis [7]. An estimate the origin of the focal AT from the P-wave morphology recorded on a standard 12-lead ECG [8]. P waves are visible before every QRS and are uniform in their appearance when looking at a single lead [7]. In general, a positive P wave in lead V1 and negative P waves in leads I and aVL are correlated to atrial tachycardias arising from the left atrium. Positive P waves in leads II, III, and aVF suggest that the origin of AT is from the cranial portion of either atrium. Shorter P-wave duration is correlated to atrial tachycardia arising from the paraseptal tissue versus the right or left atrial free wall [9]. Focal atrial tachycardia in the adult population is usually associated with a benign prognosis [7]. Non-sustained focal atrial tachycardia is common and often does not require treatment [7].

Carbon monoxide (CO) poisoning is one of the most common types of poisoning causing death all over the world [10-12]. Carbon monoxide is a non-irritating colorless, odorless gas produced by incomplete burning of carbon-containing fossil fuels [10-13]. Unrecognized CO exposure may lead to significant morbidity and mortality [13]. Carbon monoxide may be responsible for more than 50% of all fatal poisonings worldwide [13]. Average of 4% to 10% of all cases of poisoning occur during childhood, and 58% to 75% of deaths due to poisoning are caused by Carbon monoxide inhalation [10]. It causes thousands of uncalled for deaths every year [12]. About 50,000 visits in ED each year in the USA are attributed to CO poisoning [11,14]. Poisoning can be acute, subacute and chronic [12]. The normal concentration in the atmosphere is less than 0.001% and a concentration of 0.1% can be lethal [12]. Burning of charcoal, wood, kerosene, or natural gas for heating, and cooking produces carbon monoxide [12]. Unintentional deaths peak in the winter months, when heating systems are being used and windows are closed [13]. The amount of CO absorbed by the body depends on minute ventilation, duration of exposure and concentration of Carbon monoxide in the environment. Carbon monoxide quickly binds with hemoglobin with an affinity greater than that of oxygen to form COHb. COHb levels do not correlate well with symptoms, outcome or the phenomenon of delayed neurological sequelae [12]. Carbon monoxide intoxication causes tissue hypoxia and direct damage at the cellular level resulting in a headache, seizure, lethargy, and coma [11]. The pathophysiology of carbon monoxide poisoning was initially thought to be due exclusively to the cellular hypoxia imposed by replacing oxyhemoglobin with CO-Hgb and producing relative anemia [13]. Carbon monoxide binds to hemoglobin with an affinity more than 200 times that of oxygen [13]. It causes a leftward shift in the oxygen-hemoglobin dissociation curve, decreasing O_2 delivery to the tissues and resulting in tissue hypoxia [13]. The intracellular uptake of carbon monoxide is an important mechanism for neurological injury. When CO binds to cytochrome oxidase, it causes mitochondrial dysfunction resulting in oxidative stress-related damage [12]. The release of nitric oxide from platelets and endothelial cells, which forms the free radical peroxynitrite, can further inactivate mitochondrial enzymes and damage the vascular endothelium of the brain [12]. Clinical manifestations of acute carbon monoxide poisoning can be vague and may closely mimic various nonspecific viral illnesses [12]. Carbon monoxide has been called a "great mimicker" [13]. The clinical presentations associated with carbon monoxide toxicity may be diverse and nonspecific [13]. Carbon monoxide poisoning is typically diagnosed by a clinical triad: symptoms consistent with carbon monoxide poisoning, history of recent CO exposure, and elevated COHb levels [14]. Clinical suspicion is the most important step in diagnosis [10]. Early cardiovascular effects of carbon monoxide poisoning are manifested by tachycardia in response to hypoxia [14]. The acute symptoms of CO poisoning are reflected in the susceptibility of the brain and heart to hypoxia. Initially, patients may complain of a headache, dizziness, nausea, emotional lability, confusion, impaired

judgment, clumsiness, metabolic acidosis, and syncope [11,12]. Coma or seizures can occur in patients with prolonged CO exposure [12]. Elderly patients may have accompanying myocardial ischemia and myocardial infarction [12]. Myonecrosis rarely leads to compartment syndrome or renal failure [12]. Coma or altered mental status, cherry-red skin, retinal hemorrhages, hypotension, and pulmonary edema, lactic acidosis occur with severe CO poisoning [12]. Low-level experimental CO exposures producing CO-Hgb levels from 2% to 6% in patients who had documented IHD have produced arrhythmias [13]. Physicians should be alert for the symptoms of carbon monoxide poisoning, especially during the winter, when the risk of continued prolonged exposures may be greater [15]. The following are the criteria for hospitalization: Syncope and coma, neurological deficit at any time, clinical or electrocardiographic signs of cardiac compromise, refractory metabolic acidosis, abnormal chest radiograph, COHb level > 25%, COHb level > 15% with a history of cardiac disease or > 10% in a pregnant patient, PO < 60 mm Hg, rhabdomyolysis, signs and, symptoms persisting up to 4 hours after initiation of treatment [10,15].

Treatment of the CO-poisoned patient begins with supplemental oxygen and aggressive supportive care, including airway management, blood pressure support, and stabilization of cardiovascular status [14]. High-flow oxygen therapy should be administered immediately to treat hypoxia due to carbon monoxide poisoning and also to accelerate the elimination of CO from the body [13]. A non-re-breather mask supplies 100% oxygen to quickly clear COHb from the blood and this therapy reduces the elimination half-life of CO from 320 minutes in room air to 40 - 80 minutes versus HBO that can reduce the half-life of HbCO to 20 minutes [12,16]. Hyperbaric oxygen therapy (HBO) offers shortened symptom recovery time, decreased mortality rate, and the development of fewer neuropsychiatric symptoms [10]. Hyperbaric oxygen therapy consists of the delivery of 100% oxygen within a pressurized chamber, resulting in a manifold increase in the dissolved oxygen in the body (PaO_2 up to 2000 mm Hg) [13]. The following are the indications for Hyperbaric oxygen therapy in carbon monoxide poisoning: comatose patients, any period of unconsciousness, any abnormal score on the carbon monoxide neuropsychological screening battery, COHb levels > 40%, Cardiovascular involvement (chest pain, ECG changes arrhythmias), history of IHD and COHb levels > 15%, pregnant patients with COHb levels > 15%, patients who do not respond to 100% oxygen after 4 to 6h and patients with recurrent symptoms up to three weeks after exposure [15].

COHb blood level (%)	Duration of exposure	Possible clinical manifestations
0 - 10% (35 ppm)	6 - 8 hr	Headache and dizziness.
10 - 20% (200 ppm)	2 - 3 hr	Easy fatigability, tightness across the forehead, mild headache, loss of judgment, and dilatation of cutaneous vessels.
20 - 30% (400 ppm)	5 - 6 hr	Pounding frontal headache, impaired motor dexterity, blurring of vision, irritability, and throbbing in temples
30 - 40% (800 ppm)	4 - 5 hr	Severe headache, severe muscle weakness, nausea, vomiting, dimness of vision, mental confusion or delirium, dizziness, collapse, cherry-red color of lips, and skin.
40 - 50% (1600 ppm)	3 - 4 hr	As above, plus; tachycardia, cardiac irritability, syncope, increased pulse, and respiratory rate
50 - 60% (3200 ppm)	11/2 - 3 hr	Tachycardia, respiratory insufficiency, tachypnea, Cheyne-Strokes respiration, coma, and convulsion.
60 - 70% (6400 ppm)	1 - 11/2 hr	Coma, convulsion, severe acidosis, decreased heart action and respiration, respiratory insufficiency, and possibly death.
70 - 80% (12800 ppm)	1 - 2 min	Weak pulse, depressed respiration, respiratory fairly and death.

Table 1: *Level of COHb concentrations and clinical manifestations [10,12,15,17].*

Case Presentations

A fourteen-year-old Egyptian male adolescent student patient presented to the emergency room with sudden deep loss of consciousness during hot bathroom on closed space. The family gave no history of any diseases. They deny any drug abuse, substances, and special habits. His vital signs were as follows: blood pressure 100/70 mmHg, pulse rate 140 bpm, respiratory rate;12 bpm, and temperature

37.1°C. The flushed face was noted on examination. His GCS was: 9. No more relevant clinical data were noted during the clinical examination. The case was managed urgently and only with high concentration O_2 inhalation using the nasal mask (12 l/m) An initial emergency ECG which showed sinus tachycardia (140 bpm) with ST-segment depressions in leads II, III, aVF and I, V3-6 (Figure 1). ST-segment depressions indicate acute ischemic heart disease. Sinus tachycardia refers to reflection to hypoxic myocarditis. Serial ECG tracings were showed gradual and complete normalization of ST-segment depressions (Figure 2A-2D). Reversal of ST-segment depressions in the inferior and anterior leads after oxygenation is meaning the presence of coronary artery spasm. Non-sustained focal atrial tachycardia was seen in (Figure 2C) spontaneously disappeared (Figure 2D). Recovery of non-sustained focal atrial tachycardia with continuous oxygenation reflects myocardial hypoxia. Troponin T test was negative (< 0.001 ng/mL). His random blood sugar was: 163. Metabolic acidosis was seen on ABG. Unfortunately CO-HB test not available. Brain CT was normal. Later echocardiography was completely normal. Complete recovery had achieved and the patient was discharged within 12 hours of ICU admission with outpatient follow up.

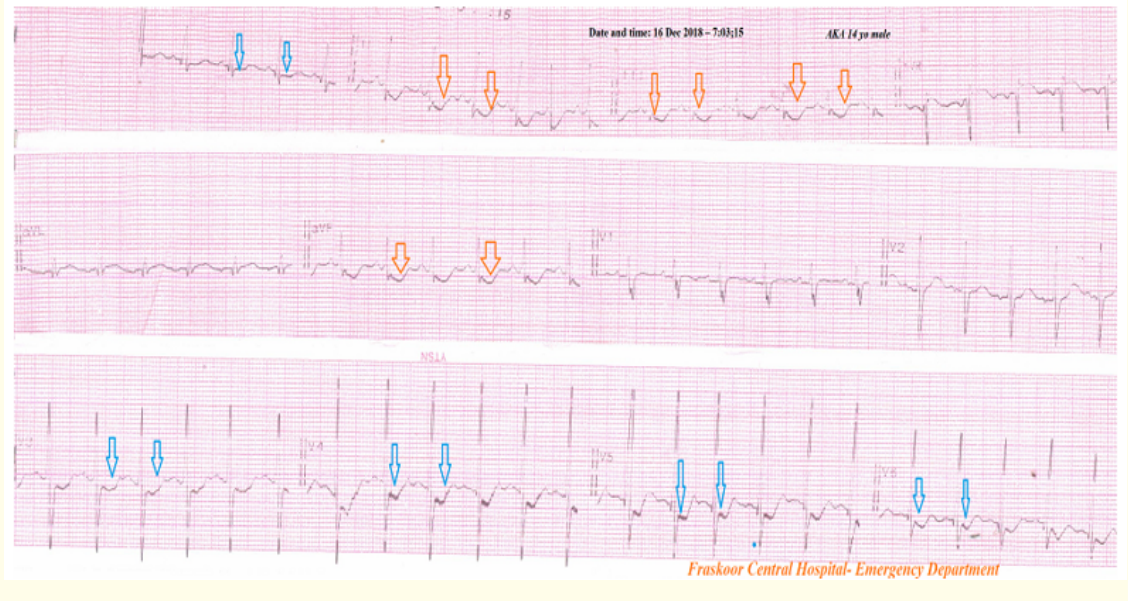

Figure 1: *ECG tracing of presentation (ED) showing marked sinus tachycardia (140 bpm) with ST-segment depressions in leads II, III, aVF (red arrows) and I, V3-6 (blue arrows).*

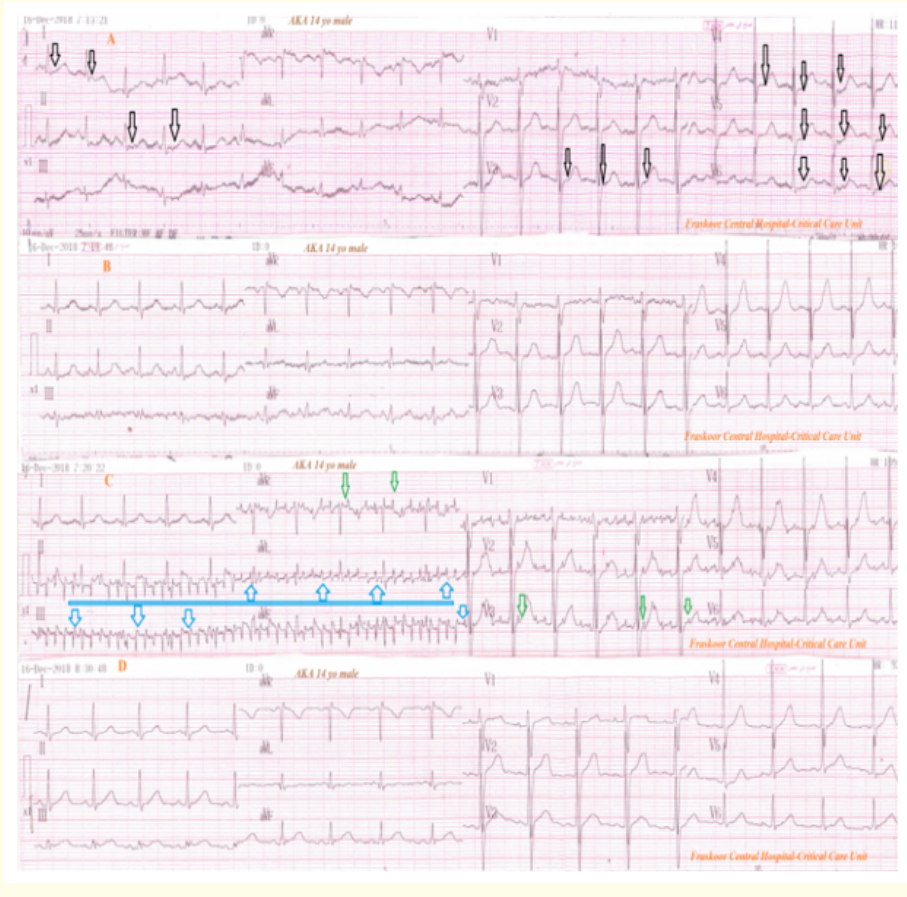

Figure 2: *Serial ECG tracings: A-tracing: After 5 minutes of ICU admission and O2 inhalation showing borderline sinus tachycardia (100 bpm) with ST-segment depressions in leads V3-6not in leads II, III, aVF and I (black arrows). B-tracing: After 12 minutes of O2 inhalation showing complete normalization of ST-segment depressions. C-tracing: After 13 minutes of admission showing non-sustained focal atrial tachycardia in leads II, III, aVF, aVR, and aVL with equal R-R interval (blue arrows) and multiple P-waves of the same morphology in leads aVR, and V1-3 (green arrows), aVL (blue arrows). D-tracing: After 77 minutes of O2 showing complete normalization of all the above changes (HR; bpm).*

Atrial flutter, atrial fibrillation, multifocal atrial tachycardia, atrioventricular nodal reentry tachycardia, sinus node dysfunction, and ventricular tachycardia, implicated in the case differential diagnoses.

Study method was an observational case study; the method was summarized as follows: there was evidence of myocardial and brain toxicity was noticed after carbon monoxide toxicity. The represented clinical manifestations were coronary artery spasm, focal atrial tachycardia, and loss of consciousness. Dramatic response of the above clinical manifestations only to 100% oxygen was the main result rather than the method of study.

Study question here; What does the evidence of brain and myocardial injuries in my case? How did the loss of consciousness recovery, coronary artery spasm reversal, and focal atrial tachycardia termination had happened with non-baric oxygenation? So, how the loss of consciousness, coronary artery spasm, and focal atrial tachycardia had happened? What was the role of non-baric oxygenation for the case?.

The primary objective for my case study was an appearance of the evidence of brain and myocardial injuries secondary to hypoxia due to carbon monoxide toxicity.

The secondary objective was the dramatic response of coronary artery spasm, focal atrial tachycardia, and loss of consciousness to 100% oxygen.

Discussion

Overview of my case results included in the occurrence of brain and myocardial injuries secondary to hypoxia due to carbon monoxide toxicity. Carbon monoxide toxicity implicated in the development of loss of consciousness, coronary artery spasm, and focal atrial tachycardia. ST-segment depressions in the inferior and anterior leads with sinus tachycardia in the initial ECG is evidence for ischemic heart disease due to hypoxia. Normalization of previous ST-segment depressions after O_2 supply means that changes were coronary artery vasospasm. History and clinical suspicion was the most important step in the diagnosis of the current case due to the absent assessment of CO-HB concentration. Despite clinical manifestations graduated with the level of COHb concentrations (Table 1), but COHb levels do not correlate well with symptoms, outcome or the phenomenon of delayed neurological sequelae [12].

Recommendations

The further broad study will be recommended.

Conclusions

- Non-baric oxygenation possesses a pivotal role in the reversal of brain injury due to hypoxia of carbon monoxide toxicity.
- Rapid response of loss of consciousness to O_2 deepen its efficacy base of emergency oxygenation for regaining the consciousness the cases in brain coma.
- Carbon monoxide toxicity induced-myocardial injuries with coronary vasospasm are best corrected with oxygen.
- Dramatic response of carbon monoxide toxicity induced-focal atrial tachycardia heightens the essential role of oxygenation in the management of arrhythmia.
- Non-baric oxygenation should take the priority for management of coma, coronary vasospasm, and focal atrial tachycardia due to carbon monoxide toxicity.
- The physician should not hurry to symptomatic management with multiple medications.
- Oxygenation in the management of arrhythmia is the future concept.

Conflicts of Interest

There are no conflicts of interest.

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