Original Article

Ice Storm-related Carbon Monoxide Poisonings in North Carolina: A Reminder

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Abstract: Severe winter weather, such as ice storms, that results in loss of electrical power, is frequently mentioned as a contributing factor in acute carbon monoxide (CO) poisoning. However, in our literature review, such events are infrequently reported. This article reports on such an event in which more than 200 patients were evaluated and treated at a single facility because of the crippling effects of an ice storm leading to prolonged loss of power and subsequent catastrophes with alternative heating and cooking sources. One hundred seventy-six patients were treated and subsequently released after Emergency Department-based treatment for CO exposure, and three patients were admitted. Eighteen patients were treated with hyperbaric treatments and discharged. Three others left before treatment was completed. Three cases representing varying levels of severity at presentation leading to differing treatment algorithms are discussed to demonstrate a suggested clinical decision pathway in the treatment of unintentional CO poisoning.

Key Words: carbon monoxide, charcoal grills, hyperbaric medicine, ice/winter storms, poisoning

During the first week in December 2002, more than 1 million residents in North Carolina as well as South Carolina found themselves without electrical power during what was reported by the leading electrical power provider in the area to be the worst ice storm in their history of operation ("More than 725,000 customers have power restored following worst ice storm in Duke Power history" Duke Power Media, December 8, 2002. http://www.dukepower.com/content/news/jic/2002/dec/2002120803.html). Power outages began on December 4, 2002, and power was not completely restored in the leading provider's service territory until approximately December 14. With temperatures well below freezing during this period, especially at night, persons who used electricity-dependent heating sources resorted to alternatives such as kerosene-, gasoline-, or propane-powered space heaters and even charcoal grills placed inside their homes. In many cases, adequate ventilation was not provided or even recognized as necessary. Others used gasoline-powered electrical generators. However, because of a failure to recognize the need for adequate ventilation or secondary to concerns about possible theft, in many instances the generator was placed inside or partially inside the home, or within connecting structures such as garages or enclosed porches, leading to carbon monoxide (CO) exposure. These actions resulted in many cases of obvious or occult exposure to CO.[1–7]

Duke University Medical Center (DUMC), a tertiary-care referral and level-one trauma center located in Durham, NC, was significantly affected by this ice storm. The institution serves as the primary hospital for the county and surrounding area's population. During the week of December 5 through December 12, 2002, the DUMC Emergency Department (ED) documented more than 200 individuals who were known or suspected to have had toxic exposure to CO, based on historic and physical signs and/or symptoms. The vast majority of patients were represented by the growing Latino population. Many patients who had minor symptoms of nausea or headache were treated with high-flow oxygen and later released.

Materials and Methods

Because of the large and continuous influx of CO-exposed persons over several days, a patient care pathway was developed for these patients after review of currently available literature, discussions by ED staff with representatives

Key Points

- A North Carolina ice storm of 2002 resulted in the presentation of more than 200 patients with carbon monoxide poisoning to the Duke University Medical Center Emergency Department.
- Three cases illustrating the varying severities of presentations and differing treatments are presented.
- A potential algorithm for triage and treatment of carbon monoxide poisoning using current recommendations is discussed.

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from Hyperbaric Medicine, hospital administration, and others. Standing orders were initiated at triage for any patient thought to be exposed to CO by history or signs and symptoms (Table 1). Carboxyhemoglobin levels, a standard part of blood gas analysis at this institution, were readily available to the ED staff; therefore a venous blood gas (VBG) was obtained on patients identified by the standing orders. Initial arterial blood gas sampling (ABG) was reserved for patients who had acute respiratory symptoms on presentation. The institution has an in-house hyperbaric facility, and hyperbaric consultants were available throughout the course of this event. Spanish language translators were available to the ED throughout the course of this event to assist in communication.

Previous reports exist in the literature, documenting the occurrence of CO exposures resulting from natural disasters. However, when reviewing the available literature, only four studies were found by the authors specifically describing ice storm-related CO exposures.[2,5–7] The current case series was initiated to review the disruptive effects particular to winter ice storms that can lead to CO poisoning, and to discuss CO poisoning presentations, both overt and occult.

Results

The electronic coding and patient tracking system used in the DUMC ED was reviewed for the period of time from December 4 through December 14, 2002. All patients with the International Classification of Diseases (ICD) code and di-

Table. Standing orders for immediate-care carbon monoxide inhalation

Criteria
Patient with history of or suspected of having been in an enclosed or poorly ventilated environment where they inhaled smoke, automobile exhaust, charcoal fumes, kerosene, or gas stove fumes.
Depending on the extent of the exposure, patients may complain of a variety of symptoms: headache, dizziness, nausea, and/or vomiting, as well as breathing difficulties, mental status changes, syncope, seizures, or coma.
Standing orders
1. Obtain a baseline pulse oximeter reading.
Note: CO itself will not lower the pulse oximeter reading. A low pulse oximeter reading is suggestive of another possibly concomitant problem.
2. Administer 100% oxygen by nonrebreather mask or endotrachael tube immediately and continue for a minimum of 4 hours.
3. Notify Respiratory Therapy.
4. Question the patient about possibility of concurrent trauma if, for example, the patient fell while passing out.
5. Obtain a venous (preferably) or arterial (only if other associated respiratory findings are present) carboxyhemoglobin level on all patients with a history or suspicion of exposure, even if they are asymptomatic.
6. If patient has any of the following, start an IV and obtain a carboxyhemoglobin level. In addition, obtain a dextrose stick on patients with an altered mental status. In pregnant patients, obtain fetal heart tones.
• History of LOC—Was the patient difficult to arouse? (not simply a fainting spell or fell asleep).
• Any subjective neurologic findings—memory difficulties, altered mental status, disorientation, or ataxia.
• Infants $<$ 3 mo of age.
• Pregnant.
• Patient with a history of equivalent exposure time to a household member or other immediate contact with a documented significant CO inhalation.
7. For patients complaining of chest pain, obtain electrocardiogram and place patient on continuous cardiac monitoring.
8. Vital signs every 60 minutes or more frequently if indicated, especially in patients with severe neurologic complaints or respiratory symptoms.
Hyperbaric consultation
1. Consult Hyperbarics for any patient with
• History of LOC—Was the patient difficult to arouse? (not simply a fainting spell or fell asleep).
• Neurologic findings—memory difficulties, altered mental status, disorientation, or ataxia.
• Pediatric or pregnant patients with CO $\geq$ 20% or signs of fetal distress.
• Adult patients with CO $\geq$ 25%.
• Symptoms that persist despite 4 hours of oxygen therapy.
Immediate consultation with ed physician
1. Neurologic findings other than headache.
2. LOC.
3. Oxygen saturation $<$90%.
4. Abnormal vital signs.
5. History or signs of trauma.
6. Pregnant woman with fetal heart tones $\leq$ 120 beats per minute.

CO, carbon monoxide; LOC, loss of consciousness.

agnosis for CO were retrospectively reviewed. Three sample cases that best illustrate the range of severity of CO poisoning were subsequently selected by the authors for discussion.

Case studies

Patient 1. A 6-year-old twin Latino male who, along with multiple family members, was exposed to a charcoal grill brought indoors to provide heat because of the lack of electricity. The patient was exposed for approximately 2 hours, with development of symptoms of nausea with vomiting, shortness of breath, and headache. He was pallid and eventually lost consciousness for an unknown period of time, according to the mother. The patient was difficult to arouse.

Upon examination, the boy was found to have normal vital signs with a normal physical examination, except that he was drowsy. His neurologic and psychiatric assessments were appropriate for his age. His ABG on room air showed a pH of 7.36, P_{CO_2} 48, P_{O_2} 29, CO_2 total 28, $\%O_2$ saturation of 63.4%, and %CO saturation of 19.6. Although by record this was reported as an ABG, this was obviously a VBG given the PO_2 of 29.

After completion of one hyperbaric oxygen treatment, the patient was returned to the ED, was reevaluated and observed for a short period, and then subsequently discharged to home in stable condition. Reasons for CO exposure and home safety were reviewed with the patient and family by the ED staff at the time of discharge, and alternative temporary living facilities were arranged before discharge.

Patient 2. A 26-year-old Latino man lost electrical power and was using charcoal briquettes in a grill that had been brought inside the home to provide heat. He and his wife had gone to bed and were asleep for an undetermined amount of time. They eventually awoke and felt nauseated and dizzy. The patient had one episode of emesis. His wife was so dizzy that he had to drag her outside. Outside, he slipped and fell on a patch of ice, striking his chin on the cement. He had no loss of consciousness or neck pain. His complaints at arrival were nausea and a small chin laceration.

After initial evaluation, the patient was placed on a 100% nonrebreather face mask. A peripheral intravenous line was placed, and a VBG revealed a pH of 7.32, P_{CO_2} 40, P_{O_2} 25, and %CO saturation of 21.8. The patient's chin was sutured, and C-spine films were obtained and interpreted as negative for clinically significant acute injury. Despite the elevated CO level on VBG, the patient denied loss of consciousness or chest pain and did not display any neurologic abnormalities on initial and follow-up evaluations in the ED. The patient was therefore triaged to a 100% nonrebreather face mask protocol, with observation and follow-up examinations. After 4 hours of treatment on high-flow oxygen, the patient remained stable and asymptomatic. He was discharged in stable condition. The cause of the elevated CO level was explored with the patient and his wife before discharge, and alternative shelter options were provided.

Patient 3. A 23-year-old black man was brought in by Emergency Medical Services. He was accompanied by his family, who supplied most of the history. Members of the family found the patient unresponsive, on the floor of his garage, with a gasoline generator running. All of the doors and windows were closed. The family thought the patient fell, but the presumed fall was not witnessed. It was unclear how long the patient had been unconscious. The patient was transported emergently to the ED. At arrival, the patient was combative and confused and had to be pharmacologically sedated to examine and treat him. The patient's ABG on 100% oxygen revealed a pH of 7.22, P_{CO_2} of 47, P_{O_2} 259, and %CO saturation of 28. A head computed tomographic scan did not reveal intracranial hemorrhage or skull fracture.

Due to severe CO intoxication, as evidenced by the patient's history, neurologic status, and elevated CO level, he was stabilized and evaluated by our hyperbaric consultants and transferred to the hyperbaric center for treatment. After receiving hyperbaric treatment, he was admitted to the medical intensive care unit. The patient underwent a total of three successive hyperbaric treatments. The patient was ultimately diagnosed with severe encephalopathy believed to be consistent with prolonged CO poisoning as well as minor barotraumas to his ears, from which he recovered with supportive therapy. The patient subsequently underwent aggressive physical, occupational, and speech rehabilitation therapy and was thought to regain much of his preexposure abilities, though at last follow-up was still receiving continued rehabilitation.

Discussion

Presentation

During the CO disaster period from December 5 through 12, large numbers of patients presented to the ED for a variety of symptoms. Unlike the surge of victims from a disaster such as an explosion, patient arrivals were episodic, based on transportation difficulties, but generally continuous. They tapered off as power and other essential utilities began to be restored. Record review indicates that more than 200 patients were evaluated during this period for complaints suggestive of CO exposure. One hundred seventy-six patients were treated in the ED, including ED-based treatment for CO exposure and ED observation, and were subsequently discharged after determination of a safe home or temporary living facility. Eighteen patients were transferred from the ED to the in-house Hyperbaric Center for HBO treatments, were returned to the ED, observed, and subsequently discharged after similar determination of a safe home or temporary living environment. Three patients were admitted (one to the medical intensive care unit). At least three others left before treatment was completed.

Pathophysiology

Carbon monoxide is an odorless, colorless, nonirritating gas produced through incomplete combustion of virtually all carbon-containing products. Acute exposure results in 3,000 accidental or suicidal deaths each year and 10,000 episodes of illness.[8] Exposure often results from structure fires (eg, wood), clogged vents for home heating units (eg, methane), use of gasoline-powered generators indoors or in inadequately ventilated areas such as partially enclosed garages or porches,[9] automobile exhaust, gas or kerosene heaters and stoves, and open and unventilated fires in closed spaces.[10] Another source of exposure is cigarette smoking; however, because of the dilutional effects of air, the toxic effects are greatly reduced. CO interacts with deoxyhemoglobin and forms carboxyhemoglobin. The absorption of carbon monoxide also causes a leftward shift in the oxygen-hemoglobin dissociation curve, which results in decreased oxygen-carrying capacity and impaired release of oxygen to the tissues. Carbon monoxide binds to hemoglobin approximately 225 to 240 times more tightly than oxygen. Once bound, it is very difficult to displace the CO, and it does so very slowly, at a half-life of 4 to 6 hours on room air. High tissue levels of oxygen hasten the half-life of CO dissociation. With a 100% nonrebreather face mask, the half-life is reduced to 90 minutes; 100% oxygen given through multiple sessions at 3 atm in a hyperbaric oxygen (HBO) chamber can reduce the half-life to 20 to 30 minutes.

Symptoms of exposure are often very nonspecific, with common complaints being headache, irritability, nausea, dizziness, myalgias, lethargy, or other symptoms related to hypoxia. Severe toxicity can present with altered mental status, including coma and seizures, and/or abnormal vital signs, including hypotension, chest pain with ischemia, syncope, cardiac arrest, and metabolic acidosis[11] probably resulting from tissue hypoxia. Clinical suspicion is necessary, as many such patients may present without an obvious cause. Often the patient and family or friends are not aware of exposure to CO. Therefore, one must consider the possibility of CO poisoning in the differential diagnosis whenever a patient presents with otherwise unexplained headache, nausea, confusion, or coma, especially if other family members or coworkers have similar symptoms.

ED Treatment

Emergent treatment begins with 100% high-flow oxygen, which should be instituted as soon as the diagnosis is suspected. A good history is necessary to note the method of exposure, as well as signs and symptoms of severe toxicity such as syncope or chest pain. Attention to the airway, breathing, and circulation are of prime importance. All presenting patients should have laboratory samples drawn, beginning with either an arterial or venous blood gas to determine the carboxyhemoglobin (COHb) level,[12] although the COHb numerical value in itself does not necessarily correlate clinically. Likewise, O_2 levels on ABG samples may be falsely elevated and may not display the true extent of hypoxia in CO-exposed individuals, but should be considered for those with acute respiratory symptoms. VBG sampling is an excellent and minimally invasive technique for determination of carboxyhemoglobin values,[12] especially in a disaster situation, in which time constraints become a factor, as was the case during this event. It is recommended that women of childbearing age should be tested for possible pregnancy, as fetal toxicity from CO exposure is frequently discussed as a special concern in the CO literature.[13]

Patients should be screened for trauma and other acute or destabilized medical conditions. CO-exposed patients who were entrapped in fire-related events are at particular risk for traumatic injuries as well as inhalational injuries from multiple inhaled toxins.[13] Patients who are CO-exposed from winter storm or other loss-of-power situations may not recognize the relation between their symptoms and exposure to open-flame heating sources, and thus may not offer such historic information. Therefore, clinical suspicion is critical in such presentations.

Specific treatment interventions for acute CO poisoning are generally divided into either HBO therapy or use of 100% normobaric oxygen (NBO) through a nonrebreather mask or other delivery system, such as an endotracheal tube for those with an unstable airway. Clinical judgment is required in the decision tree for treatment, for there is no true consensus in the literature on when, how long, or how often each treatment regime should be used. For example, the Undersea and Hyperbaric Medical Society reported in 1986 that if a hyperbaric chamber is locally available, a person should undergo hyperbaric treatment if his or her blood CO level is greater than 25%, regardless of symptoms. If a chamber is not available, patients with carboxyhemoglobin levels of 40% or greater should be sent to a hyperbaric chamber.[14] More recently, the Undersea and Hyperbaric Medical Society has recommended that patients who present with a history of severe symptoms—such as seizure, syncope, or chest pain suggestive of cardiac ischemia[13]—receive hyperbaric treatment regardless of COHb value, especially since time may have elapsed between exposure and presentation, lowering the carboxyhemoglobin value. COHb levels do not necessarily demonstrate or correlate to the length of time that the patient may have been exposed to CO.

The treatment of pregnant women acutely CO-intoxicated is an area of controversy. It has been generally recommended that pregnant women should undergo hyperbaric treatment either regardless of CO level or at lower levels (often noted as approximately 15%) than nonpregnant patients.[15] The rationale for such a treatment strategy includes the argument that the fetus is already under greater stress for oxygen delivery from maternal hemoglobin and thus has a lower P_{O_2} to begin with. However, there are few quantifiable

data in human models that support such contentions, and no prospective, case-control studies with human models exist.

Neuropsychiatric testing such as that described by Messier and Myers[16] has been suggested to alert the clinician to subtle symptoms of neurologic ischemia not evident by gross neurologic testing, and current treatment recommendations generally include performance of such screening testing. The literature describes neuropsychiatric symptoms that may be transient or persistent and may appear immediately or days to weeks after exposure.[13] Behavioral symptoms such as irritability, violence, personality disturbances, euphoria, confusion, and impaired judgment have been described.[17] Sequelae include difficulties with visual and verbal memory, spatial deficits, and declines in cognitive efficiency and flexibility.[18]

Recently, Weaver and colleagues[19] found that three HBO treatments within a 24-hour period appeared to reduce the risk of cognitive sequelae at 6 weeks and 12 months' follow up for those patients presenting with significant signs and symptoms of CO poisoning, including loss of consciousness, confusion, headache, malaise, fatigue, forgetfulness, dizziness, visual disturbances, nausea, vomiting, cardiac ischemia, or metabolic acidosis. However, in another recent controlled, prospective, randomized human trial, Scheinkestel et al[20] found little difference between HBO and normobaric groups, and noted that no patients in the normobaric-treated group had delayed neurologic sequelae, versus 4.8% of those in the HBO group.

Current treatment guidelines are therefore generalized recommendations that may vary from center to center. For the patient who displays only mild to moderate symptoms, such as no loss of consciousness without cardiac, neurologic, or metabolic instability, 100% NBO by nonrebreather face mask is commonly recommended until the patient clinically improves, along with a period of observation for approximately 4 to 6 hours in the ED while on at least low-flow oxygen (<50%) to ensure that symptoms do not recur and oxygen toxicity is avoided.[13] These patients may then be discharged with appropriate primary care follow-up. If patients in this group continue to display symptoms of poisoning, however, an overnight period of observation or admission to an inpatient service would be appropriate.

Guidelines for patients with significant symptomology, including prolonged loss of consciousness, generally include a recommendation for HBO treatments. There are, however, no absolute criteria establishing what symptoms are considered "significant," or what length of time a loss of consciousness would have to have persisted to qualify the patient for HBO therapy. Loss of consciousness is even more difficult to quantify because the information is often anecdotal and/or estimated.

At our institution, HBO consultation and a treatment center is readily available. In patient 1 above, the patient underwent hyperbaric treatment for the history of loss of consciousness and the fact that he was a pediatric patient. In patient 2, although the patient displayed symptoms of intermediate CO poisoning, he did not display signs or symptoms of cardiovascular and neurologic instability, and was thus treated with high-flow 100% NBO by nonrebreather face mask for 6 hours. He was then observed for residual symptoms before being discharged home. In patient 3, the patient clearly displayed active cardiovascular and neurologic instability and therefore initial treatment included HBO therapy. He continued to display neurologic symptoms after initial HBO treatments and thus was admitted to the intensive care unit for continued treatment.

Criteria for transfer of CO-exposed patients to another facility for HBO therapy, if it is not locally available, currently are not standardized. When considering such a transfer, one must consider multiple factors, including whether the patient is stable from all traumatic injuries and medical conditions, whether the patient can receive adequate care for all acute medical conditions and/or traumatic injuries at the receiving facility, and the mechanism of transfer, such as ground versus air. In any case that is transferred, it is necessary to follow all transfer requirements and regulations, including appropriate documentation.

Conclusion

Carbon monoxide poisoning can often present with vague, nonspecific symptoms such as headache, chest pain, dizziness, and other symptoms of end-organ hypoxia. During the period from December 5 through December 12, 2002, the DUMC ED saw more than 200 cases of CO poisoning. This phenomenon was related to a severe power outage occurring as a direct result of an ice storm that weighed down trees and power lines. Because of the lack of alternate heating sources, many residents chose to bring in gasoline- and propane-powered generators and heaters and even charcoal grills into parts of their home that were not adequately ventilated. The facility developed various strategies including the institution of standing orders and rapid availability of medical consultants such as hyperbaric physicians, as well as other consultants such as translators for communication assistance, and social workers to assist in the safe disposition of patients whose homes had been unsafely heated.

It is of paramount importance that emergency and urgent care facilities remain vigilant for presentations of CO poisoning in the event of natural disasters or significant power outages in the setting of cold temperatures, and be prepared to respond in coordinated fashion to such emergencies. As in any potential disaster scenario, a steady influx of patients ought to warn providers of a possible sentinel event having arisen, at which time a plan and even a preset algorithm should be available for such an emergency.

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Be nice to people on your way up because you meet them on your way down.

—Jimmy Durante