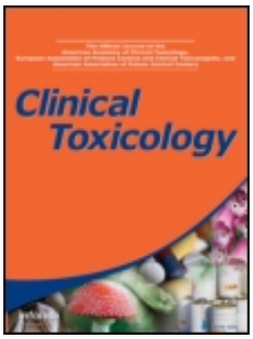

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CASE SERIES

Finding needles in a haystack: A case series of carbon monoxide poisoning detected using new technology in the emergency department

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Introduction. The diagnosis of carbon monoxide poisoning can be difficult because the symptoms are nonspecific and may mimic other illnesses. If carbon monoxide poisoning is suspected, the standard test at this time is venous or arterial carboxyhemoglobin levels. A new device, the Rad-57 pulse CO-oximeter (Masimo Inc.), can measure carboxyhemoglobin levels non-invasively at emergency department triage. *Methods*. The pulse CO-oximeter was utilized in our emergency department triage to measure carboxyhemoglobin levels on all patients. A retrospective chart review was then conducted to identify all patients with elevated levels. *Case Series*. Out of an estimated 74,880 patients who had their SpCO measured and documented at triage, seven patients who presented with vague complaints were diagnosed with occult carbon monoxide poisoning. Their diagnosis was facilitated by the non-invasive pulse CO-oximeter, which measured their carboxyhemoglobin levels when the standard vital signs were also documented at triage. *Conclusions*. The non-invasive pulse CO-oximeter could be a major triage tool for identifying unsuspected carbon monoxide poisoning among patients with nonspecific symptoms.

Keywords Carbon monoxide; Poisoning; Toxicity; Hyperbaric oxygen therapy

Introduction

Carbon monoxide (CO) poisoning is a significant health problem in the United States and can result in long-term neurologic or cardiovascular complications if unrecognized. Although the precise incidence of CO poisoning is unknown, the CDC has estimated that 15,200 patients were treated annually in emergency departments (EDs) during 2001–2003 for non-fire related unintentional CO exposure, and approximately 480 people died yearly from CO poisoning during that period (1). The incidence of CO poisoning increases during the winter months and after natural disasters when alternative heating and power sources are utilized. However, because CO is an odorless, colorless gas, most patients are not aware of their exposures.

The symptoms of CO poisoning are often vague and nonspecific and may mimic those of viral illnesses. Therefore, it is incumbent upon the clinician to have a broad differential diagnosis and screen for CO poisoning. The standard test for CO toxicity at this time is venous or arterial carboxyhemoglobin (COHb) levels, but there is now available a device to measure COHb levels non-invasively, the Rad-57 pulse CO-oximeter by Masimo, Inc. This portable device performs rapid spectroscopic COHb measurements with a fingertip probe similar to the standard pulse oximeter that measures the concentration of oxygenated hemoglobin. The sensor is placed usually either on the index or middle finger. The SpCO calculation performed by the device relies on a multi-wavelength calibration equation to estimate the concentration of COHb. The manufacturer determined the calibration range to be 0–40% and the relationship between SpCO and COHb as linear over that range with an error of 3%. This pulse CO-oximeter can be readily utilized at triage without additional time or effort to measure both oxygen saturation (SpO_2) and carboxyhemoglobin saturation (SpCO) on all patients who present to the ED. In this manuscript we use SpCO to denote measurements obtained by pulse CO-oximetry. We report multiple cases over a thirteen-month period of occult CO toxicity in patients who presented to our ED with vague complaints, for whom the diagnosis and treatment were facilitated by the initial SpCO measurement obtained at triage along with the vital signs as part of standard ED practice.

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Methods

Over the course of the thirteen-month period between November 30, 2005 and December 21, 2006, adult patients greater than 18 years of age presenting to our urban academic ED with an annual adult census of 95,000 patients had initial vital signs and oxygen saturation documented at triage as part of the standard ED triage practice. In addition, these patients also had carboxyhemoglobin levels measured non-invasively with the Rad-57 pulse CO-oximeter, which was made available in every triage area. All patients were eligible for inclusion in the study if they had a SpCO documented on the ED chart. Each day, two trained research assistants reviewed charts from the previous day and recorded the SpCO along with other variables into a database (Microsoft Access, Microsoft Corporation, Redmond, WA). Variables recorded in the database included: date, age, gender, mode of arrival to the ED, SpCO, venous COHb, SpO_2, heart rate, respiratory rate, systolic and diastolic blood pressure, and smoking history. One of the investigators verified accurate data entry by reviewing 1% of the entered charts. Patients with an elevated SpCO measurement documented on the triage sheet that was subsequently confirmed by a venous COHb level were then identified by two of the investigators. SpCO was considered elevated when it was beyond the normal range for a non-smoker, which was designated to be 10%. This cutoff value was established, as 10% was found to be two standard deviations from the mean value for all non-smoking patients using the CO-oximeter. Inter-rater reliability was not evaluated. Patients who had elevated SpCO levels and had an obvious exposure to CO, such as a house fire, were not considered to have had occult CO poisoning and were excluded.

During this 13 month time period, an estimated 104,000 adult patients were seen in our ED. Based on the sample of patients within the first three months after SpCO testing was initiated, approximately 72% of patients had an SpCO documented at triage. Critically ill patients who presented in distress and were taken directly to a resuscitation bay did not have vital signs checked at triage and therefore did not have an SpCO documented in their records. Twenty-eight patients were found to have an elevated SpCO that was subsequently confirmed by a venous COHb level: three were victims of a house fire, one had attempted suicide by running his car in a closed space, seven had occult CO poisoning and are presented in this case series (Table 1), and fifteen were smokers in whom it is unclear if they represent true cases of occult toxicity or if their elevated SpCO was due to smoking (Table 2). An additional two patients had mildly elevated SpCO values but are not formally presented here as details of the cases were not available upon chart review. This study received approval from the institutional review board for contacting patients by mail followed by a structured telephone interview to obtain follow up information. We did not attempt to follow up on the four patients with obvious sources for their CO toxicity. Forty-two percent of the remaining twenty-four patients were lost to follow up, as no valid contact information was available in their medical records.

Case series (Table 1)

Case 1

A 41 year-old male presented in February 2006 with a sudden-onset headache. He experienced intermittent palpitations while at work in the morning and then developed a severe headache with dizziness when he tried to stand up. His PMH was significant only for prostatitis, and he was employed as a mechanic. Initial vital signs were temperature 98.6°F, heart rate 93, blood pressure 160/82, respiratory rate 14, SpO_2 95% on room air, and SpCO 33%. His EKG showed a sinus arrhythmia with normal intervals and T wave inversions in III and aVF. The patient was alert and oriented to person, place and time, and his physical examination was normal. The venous COHb obtained 2 hours later was 25%. The repeat venous COHb at 5 hours on continuous NRB mask oxygen was 8%. The patient was discharged home. As no current contact information is available for this patient, it is unknown if a source was ultimately identified for his CO poisoning.

Case 2

A 54 year-old male with a PMH of insulin-dependent diabetes mellitus and hypertension presented in February 2006 with weakness, dizziness, nausea, and shortness of breath that had begun earlier that morning. He attributed his symptoms to a possible hypoglycemic episode, but as he had no resolution of symptoms after drinking some juice, he called emergency medical services (EMS). Paramedics reported an initial blood sugar of 120 mg/dL on their arrival. At triage, his initial vital signs were temperature 96.7°F, heart rate 80, blood pressure 154/71, respiratory rate 18, SpO_2 95% on room air, and SpCO 14%. His EKG demonstrated an old right bundle branch block with nonspecific ST-T changes in III with no signs of ischemia. The patient was noted to be lethargic but oriented to person, place, and time. Physical examination was otherwise unremarkable. Given his past medical history, the patient also received ASA 325mg and additional tests included cardiac enzymes, which revealed an elevated CPK of 484 IU/L with a normal troponin, and a chest x-ray that suggested possible lingular and left lower lobe air space disease. The venous COHb obtained 18 minutes after arrival was 28%. The patient was subsequently transferred to another hospital within two hours of his arrival for treatment with hyperbaric oxygen (HBO). His wife, who was present with him in the ED, was then advised to be evaluated as well. She reported feeling well when she woke up that morning but upon further questioning did recall developing a headache with nausea, dizziness, generalized body aches, and shortness

Table 1. Cases of occult CO poisoning (COP) in nonsmokers

	Chief complaint	Date of ED visit	SpCO (%)	Venous COHb (%)	Interval between SpCO & venous COHb (minutes)	Review of systems	Treatment	Disposition	Follow up
1	Headache and palpitations	2/21/06	33	25	120	Paresthesias in hands	100% O_2 NRB × 4 hrs	D/C and instructed to f/u with Fire Dept	No current contact information available, pt returned to ED 11 mo later with cough and congestion when no SpCO was documented
2	Weakness and dizziness	2/25/06	14	28	18	SOB, nausea	100% O_2 NRB × 1 hr 15 min then transferred for HBO	Transferred to another hospital for HBO	Fire Dept found elevated CO levels at home and COP was attributed to the use of a grill on the porch, Fire Dept returned the following day and found CO level had decreased to normal, pt purchased a CO detector for the home and has had no recurrent symptoms
	Headache and body aches, spouse of ED pt with occult CO poisoning	2/25/06	21	24	12	SOB, nausea, dizziness	100% O_2 NRB × 48 min then transferred for HBO	Transferred to another hospital for HBO	
3	Syncope	5/06/06	33	26	38	SOB, dizziness, confusion	100% O_2 NRB × 1 hr 20 min then transferred for HBO	Transferred to another hospital for HBO	Pt attributed COP to cement cutter and did not f/u with Fire Dept
4	Headache after argument with son	5/17/06	33	36	13	SOB, nausea, dizziness	100% O_2 NRB × 8.5 hrs then transferred for HBO	Transferred to another hospital for HBO	No current contact information available, pt never returned to ED but was seen in clinic 8 months later for shoulder pain and was found to have a negative ROS
5	Nausea, vomiting, and hip pain from sarcoidosis	10/27/06	19	17	43	Subjective fevers, abd pain, vomiting, diarrhea	100% O_2 NRB	Admitted for pain control and Fire Dept contacted by ED physician	Fire Dept found a faulty CO detector, elevated levels of CO in home, and an animal nesting in the chimney, Fire Dept returned 1 wk later and found CO levels were back to normal, pt has since purchased 2 new CO detectors, pt continues to experience bone pain but no other symptoms
	No complaints, spouse of ED pt with occult CO poisoning	10/27/06	10	9	93	Nausea, dizziness, headache	100% O_2 NRB × 2 hr 23 min	D/C and Fire Dept contacted by ED physician	

Table 2. Cases of CO poisoning (COP) in smokers

	Chief complaint	Date of ED visit	SpCO (%)	Venous COHb (%)	Interval between SpCO & venous COHb (minutes)	Review of systems	Treatment	Disposition	Follow up
1	Lost prescription	11/30/05	12	10	95	Cough, SOB, dizziness, lightheaded ness	100% O_2 NRB × 3 hr 12 min, Albuterol neb	D/C	Pt was homeless at the time of his ED visit and now lives in assisted living. He continues to smoke 1 PPD and has occasional SOB that is relieved with inhaler
2	Depression	12/07/05	17	12	27	Negative	100% O_2 NRB × 1 hr 45 min	Transferred for psychiatric admission	Pt attributed COP to smoking as he "smokes a lot just before going inside anywhere" and did not f/u with Fire Dept and continues to smoke 2 PPD
3	Suicidal thoughts	12/11/05	13	11	84	Headache		Admitted for suicidal ideation	No current contact information available, pt returned to ED 1 mo later for knee pain after a fall when SpCO = 3 and he continued to smoke per ED records
4	Mania	12/22/05	17	14	72	Negative		Admitted for mania	No current contact information available, pt never returned to ED
5	Toothache	12/22/05	16	14	59	Negative		D/C and instructed to check home for CO	No current contact information available, pt never returned to ED
6	Chest pain	12/31/05	17	11	50	SOB, cough	O_2 NC, Albuterol	Admitted for chest pain	Pt attributed COP to smoking and did not f/u with Fire Dept, pt has always had a CO detector that never alarmed, pt returned to ED 3 wks later with an MI when SpCO = 5, pt continues to smoke 1 PPD and experiences occasional CP for which she has seen her PCP

7	Seizure	1/06/06	14	11	25	Negative		D/C	No current contact information available, pt was homeless at time of ED visit and returned the following day with alcohol intoxication and SpCO = 5, pt returned to ED repeatedly with most recent visit 1 yr later when SpCO = 5
8	Back pain	1/08/06	13	14	39	Urinary hesitancy	100% O_2 NRB	D/C	Pt attributed COP to smoking, purchased a CO detector for home, did not f/u with Fire Dept, and continues to smoke 1 PPD, pt has had intermittent HA × 1yr for which he has followed up with PCP
9	Ankle injury	1/09/06	13	12	107	Negative		D/C	Pt attributed COP to smoking and did not f/u with Fire Dept as home "CO detector has not gone off," pt continues to smoke 1 PPD, pt has had intermittent HA × 3mo for which she has followed up with PCP and had outpt MRI
10	Back pain	1/28/06	14	13	46	Negative	100% O_2 NRB × 30 min	D/C and instructed to check CO detectors at home	Fire Dept found CO level at home to be 0, pt continues to smoke 1PPD and says she still plans on purchasing a CO detector for her home
11	Kidney stone	4/28/06	20	15	213	Hematuria, dysuria, left flank pain	100% O_2 NRB × 1 hr 30 min	D/C and instructed to f/u with Fire Dept	No current contact information available, pt returned to ED multiple times in the following month for abdominal pain with SpCO = 6 1 mo later, pt's most recent visit was 9 mo later when SpCO = 10 and he continues to smoke 1 PPD per ED records

(*Continued*)

Table 2. (*Continued*)

Chief complaint	Date of ED visit	SpCO (%)	Venous COHb (%)	Interval between SpCO & venous COHb (minutes)	Review of systems	Treatment	Disposition	Follow up
12 Headache	6/12/06	16	14	47	Nausea, vomiting, paresthesias	100% O_2 NRB × 2 hr 19 min	D/C and instructed to f/u with Fire Dept	No current contact information available, pt presented to ED for HA 3d later when SpCO = 5 and 5mo later when SpCO = 7, pt continued to smoke $^1/_2$ PPD per ED records
13 Fall	9/10/06	21	17	16	Back pain, abd pain	100% O_2 NRB × 2 hr 10 min	D/C and Fire Dept contacted by ED physician	No current contact information available, pt returned to ED 2 mo later with abd pain when SpCO = 8 and pt continues to smoke 1 PPD per ED records
14 Syncope	10/28/06	11	12	83	Nausea, vomiting, dizziness, anorexia, generalized weakness		D/C and instructed to f/u with Fire Dept	Fire Dept found CO level at home to be 0, CO detectors have never alarmed, pt continues to smoke 1 PPD, pt had another syncopal episode 2mo later and was found to have H. pylori and PUD
15 Headache	12/21/06	20	10	36	Dizziness, chest pain, generalized weakness	100% O_2 NRB × 1hr	D/C and instructed to f/u with Fire Dept	Pt attributed COP to smoking but did purchase a CO detector for her home and "has been meaning to call the Fire Dept" to check her home, pt continues to smoke 2 PPD and have intermittent HA

of breath. Initial vital signs were temperature 98.7°F, heart rate 88, blood pressure 129/63, respiratory rate 18, SpO_2 98% on room air, and SpCO 21%. The patient was alert and oriented to person, place and time, and her physical examination was normal. The venous COHb obtained 12 minutes after arrival was 24%. The patient was then transferred an hour after her arrival to the same hospital where her husband was sent for further treatment with HBO. The fire department found elevated concentrations of environmental CO in their home and attributed their poisoning to the use of a grill on the porch, which resulted in smoke drifting into their poorly ventilated house.

Case 3

A 57 year-old male presented in May 2006 with confusion after an unwitnessed syncopal episode. He was found unconscious by his family in the basement and was noted to be confused and diaphoretic when EMS arrived. He recalled feeling lightheaded prior to the episode when he was working with a concrete cutter and continued to complain of dyspnea in the ED. His PMH was remarkable for hypercholesterolemia, and he had a family history of CAD. Initial vital signs were temperature 97.2°F, heart rate 96, blood pressure 117/60, respiratory rate 18, SpO_2 91% on room air, and SpCO 33%. His EKG showed a normal sinus rhythm with normal intervals and nonspecific ST-T changes in III. The patient was alert and oriented to person, place and time, and his physical examination was unremarkable. The venous COHb obtained 38 minutes later was 26%. The patient was then transferred to another hospital within two hours of his arrival for treatment with HBO. The patient reported complete resolution of his lightheadedness, confusion, and shortness of breath after his treatment with HBO. Follow-up one year later revealed that the patient never contacted the Fire Department to have his home checked, never used the cutter again in the basement, and never had another syncopal episode.

Case 4

A 52 year-old female presented in May 2006 with a sudden-onset headache associated with nausea and dizziness. She appeared anxious and reported that she was upset because she had just had an argument with her son. She reported difficulty breathing during the altercation but denied any chest pain or dyspnea in the ED. Her PMH was significant for a recent vaginal biopsy two weeks prior. Initial vital signs were temperature 96.8°F, heart rate 103, blood pressure 139/88, respiratory rate 16, SpO_2 95% on room air, and SpCO 33%. Her EKG showed a sinus rhythm with normal intervals and no ST segment deviation. The patient was alert and oriented to person, place and time, and her physical examination was unremarkable. After CO toxicity was identified at triage, further directed questioning revealed the patient had been using a gas-powered generator in her basement because her electricity had been shut off. The venous COHb obtained 13 minutes later was 36%. Repeat venous COHb obtained 5 hours later was 7%. She was subsequently transferred to another hospital with a HBO chamber.

Case 5

A 44 year-old male presented in October 2006 with nausea, vomiting, and bilateral hip pain. He attributed his symptoms to the failure of his morphine pump, as he had similar symptoms earlier in the week and was found to have malposition of the intrathecal catheter of his pump. His PMH was remarkable for GERD and sarcoidosis with chronic bone pain due to lesions in his pelvis. The patient reported subjective fevers, abdominal pain, and diarrhea. Initial vital signs were temperature 98.2°F, heart rate 101, blood pressure 139/82, respiratory rate 22, SpO_2 99% on room air, and SpCO 19%. The patient was alert and oriented to person, place and time, and his physical exam was unremarkable except for mid-abdominal tenderness. The venous COHb obtained 43 minutes later was 17%. The fire department was contacted by the ED physician, and they found markedly elevated concentrations of environmental CO in the home. The patient recalled that he and his wife had turned up the heat in the house a few days earlier and had also been using the chimney. His wife, who was present with him in the ED, was then advised to be evaluated as well. She reported a mild headache with lightheadedness when she woke up that morning as well as some intermittent nausea. However, she stated that those symptoms were so mild that she had not planned to see a doctor. Initial vital signs were temperature 98.6°F, heart rate 94, blood pressure 121/77, respiratory rate 18, SpO_2 99% on room air, and SpCO 10%. The patient was alert and oriented to person, place and time, and her physical examination was normal. The venous COHb obtained 93 minutes after arrival was 9%. After treatment with 100% oxygen for approximately 2.5 hours, she was discharged home. Her husband's morphine pump was found to be working properly, and he was admitted for pain control. The fire department eventually discovered that their home had a faulty CO detector and that an animal had been nesting in the chimney, which was impeding proper ventilation.

Four of these patients were transferred to a nearby hospital for treatment with hyperbaric oxygen. While HBO therapy has been recommended to reduce cognitive sequelae in cases of severe CO poisoning characterized by changes in mental status or loss of consciousness, there are no firm guidelines that dictate when HBO must be used (2). In all of these cases, any decision to transfer for treatment with HBO was made by the treating physician in consultation with the poison control center.

There were an additional fifteen patients who had elevated SpCO levels at triage ranging from 11 to 21 during this study

period (Table 2). It is unclear if these represent cases of true occult CO toxicity as all of these patients reported a history of smoking and may have smoked just prior to their SpCO measurement. For those for whom we were able to contact for follow up, two followed up with the Fire Department, who found the CO levels in the home to be 0, six never followed up with the Fire Department, and all continue to smoke.

Discussion

Carbon monoxide poisoning continues to be a major public health concern, as the rates of calls to US poison control centers have not changed significantly from 1992 to 2002 (3). Epidemics of CO poisoning are known to occur after winter storms and other natural disasters. During a severe ice storm in North Carolina in 2002, more than 200 patients were evaluated at a single emergency department for CO exposure, of whom 18 received hyperbaric oxygen treatments (4). In the aftermath of Hurricane Katrina, a total of 51 cases of CO poisoning were reported by hyperbaric oxygen facilities in Alabama, Louisiana, and Mississippi from August 29 to September 24 (5). In such unique circumstances, it may be easier to diagnose CO poisoning because of heightened awareness by physicians of the use of alternative sources of heating or power. However, CO poisoning can occur year-round, as evidenced by the multiple cases of occult CO toxicity in this study.

Unintentional deaths from CO poisoning most commonly occur due to house fires. Other sources for CO poisoning include vehicle exhaust fumes and the use of indoor space heaters, charcoal grills, chimneys, and gasoline-powered generators in conjunction with inadequate ventilation. The symptoms of CO poisoning are diverse and can range from seemingly benign headaches, nausea and vomiting, and dizziness in mild toxicity to dysrhythmias, pulmonary edema, seizures, coma, and cardiac arrest in severe disease (6). In mild exposures, patients may incorrectly attribute their symptoms to a preexisting medical problem or to an insignificant viral illness, and some victims may even be asymptomatic. Two of our patients had already presumed a diagnosis of hypoglycemia and a malfunctioning morphine pump as the cause of their nausea and other coexisting symptoms because they had similar presentations in the past ascribed to those etiologies, and one of them had a presentation which warranted treatment with hyperbaric oxygen. Had the SpCO measurement not been available, the physician may have had premature closure and not been inclined to pursue CO poisoning in the differential. Another patient presented after an argument with her son with symptoms that were suggestive of an anxiety reaction. However, an elevated SpCO at triage prompted the physician to explore for potential sources for CO exposure, and it was discovered that she had been using a gas-powered generator in a closed space for days. Two of the patients were only evaluated in our ED for CO poisoning only after their spouses were found to have an elevated SpCO confirmed with a venous COHb level. These two women would not otherwise have been evaluated by a physician as they initially had no complaints and only recalled minor symptoms following a thorough review of systems.

Failure to diagnose CO poisoning can result in significant morbidity and mortality, as these patients may inadvertently be allowed to return to a dangerous environment. Four of the patients presented (2 married couples) did ultimately have a source identified for their occult toxicity when their homes were inspected by the fire department. If their SpCO had not been checked at triage, they could have been discharged home only to suffer continuous exposure to carbon monoxide with potentially dire consequences. Our intervention also resulted in one couple purchasing a CO detector for their home and in the other couple replacing a faulty CO detector that was identified by the fire department. All patients were instructed to follow up with the fire department, but the patient who presented after a syncopal episode was confident the etiology was the cement cutter he was using in the basement, so he did not consider any occult sources. Two patients were lost to follow up, but they both had potential sources for exposure after a thorough history was taken (one worked as a mechanic and the other was using a gas-powered generator).

Smokers are known to exhibit higher COHb levels than nonsmokers. A smoker is estimated to be exposed to 400 to 500 ppm of CO while actively smoking (6). Steady-state exposure for four hours to only 70 ppm can already result in equilibrium COHb levels in the blood of 10% (7). However, it is unclear how high levels can reach immediately after smoking cigarettes for a few minutes and how long elevated levels may be sustained in chronic smokers. We found fifteen patients who were known to be smokers to also have elevated SpCO levels. As we do not know the time elapsed since the last cigarette smoked or the extent of their tobacco use, it is unclear whether these patients had CO toxicity from other occult sources or if they had elevated SpCO levels from smoking. Six of these patients who continue to smoke were found to have SpCO levels within the normal range on subsequent unrelated visits to our ED.

The standard diagnostic test at this time for detecting CO poisoning measures COHb concentration in arterial or venous blood by multi-wavelength CO-oximetry. While this is a reasonable test to perform on victims of house fires in whom the diagnosis of CO poisoning is obviously considered, it would be impractical and inefficient to draw blood samples on every patient who presents with flu-like symptoms. Moreover, not all hospitals possess such equipment and must therefore send samples to outside facilities, which can significantly delay the diagnosis. Hampson et al. found that less than half of the hospitals they surveyed in the Pacific Northwest, where CO poisoning is common, have

the laboratory capability to measure COHb and the time required to obtain a result averaged 15 hours longer when samples were sent elsewhere (8). There are more rapid and less invasive diagnostic tests for carbon monoxide exposure, such as a breath analyzer. These breath analyzers require a cooperative subject capable of completing a 20 second breath hold prior to exhaling into the device, and normal measurements by breath analysis do not exclude poisoning (9). Alternatively, the pulse CO-oximeter (Rad-57 by Masimo, Inc.) is a non-invasive device which functions in a similar fashion to the standard pulse oximeter. It can quickly measure carboxyhemoglobin within seconds with an uncertainty of ± 2% within the range of 0–40(10). Mottram et al. also found the pulse CO-oximeter to measure carboxyhemoglobin accurately in 31 patients when compared to the standard of arterial sampling (11). The SpCO was recorded during blood sampling and the blood was analyzed within 15 minutes. They concluded that the pulse CO-oximeter measures SpCO accurately by analyzing the data using a student paired t-test with $p < 0.015$. However, the Bland-Altman method is the best statistical analysis for assessing agreement between different clinical measurements, so additional studies should be conducted with this method to fully gauge the accuracy of the non-invasive pulse CO-oximeter. Furthermore, while the manufacturer has indicated that skin pigmentation or presence of nail polish does not limit the pulse CO-oximeter's function, this has yet to be formally investigated and future studies are warranted.

It is difficult to assess the accuracy of the SpCO values for our patients given the time difference from when the venous COHb was obtained. The patient described in Case 2 did have a noticeable increase in the venous level that was drawn 18 minutes after the SpCO was measured. There is no clear explanation for this discrepancy, which speaks to the need for non-industry sponsored studies to compare the Rad-57 to standard venous or arterial sampling. We merely used the non-invasive pulse CO-oximeter as a screening tool and not as a precise measurement. By utilizing this tool at triage as part of our standard protocol, we were able to identify multiple cases of otherwise unsuspected carbon monoxide toxicity. Prior to the availability of this device, we may have had numerous cases of occult CO toxicity that went undiagnosed in the past. While screening for CO poisoning can be easily accomplished with this instrument, especially in areas where this diagnosis is prevalent, continued public education regarding the utility of CO detectors and the proper use of indoor heating sources remains a key component for prevention of CO poisoning.

Conclusions

We have presented cases of occult CO toxicity in which the diagnosis was significantly facilitated by non-invasive pulse CO-oximetry. While no conclusions can be drawn directly regarding the true accuracy of the device, given the various time delays between the non-invasive SpCO measurement and the COHb measurement from a venous blood draw, these cases suggest that non-invasive pulse CO-oximetry could be a major screening tool at ED triage for identifying unsuspected CO poisoning among patients with nonspecific symptoms. Future studies are warranted to evaluate and compare the accuracy and utility of non-invasive CO assessment to standard venous or arterial COHb measurements. While the advantage of such a device may be limited to certain geographical areas or seasonal situations where and when the incidence of CO poisoning is high, its value would certainly be appreciated when triaging patients in the wake of some natural disasters. Additionally, considering that many institutions presently do not have access to standard CO-oximetry, the non-invasive pulse CO-oximeter may be valuable in the management of patients with suspected CO exposure.

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