

Carbon Monoxide and the Eye: A Teaching Case Report

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Abstract

Both acute and chronic exposure to carbon monoxide (CO) gas can have serious and permanent effects on a person's health and vision, especially if left untreated. The brain and eyes are at risk upon exposure to this clear, odorless gas due to the large oxygen demands of these structures. This teaching case report highlights the need for eyecare professionals to recognize the possible ocular, systemic and neurological effects of CO poisoning and the impending long-term risk factors. This report also reviews the body's response to acute hypoxic events, including ocular and systemic symptomatology, the testing and procedures used for the differential diagnosis of CO poisoning, the events and risk factors leading to CO poisoning, the pathophysiology of CO on the body, and the treatment options for CO poisoning from the perspective of an eyecare provider.

Key Words: carbon monoxide, carbon monoxide poisoning, CO poisoning, CO exposure, acute carbon monoxide poisoning, carboxyhemoglobin, ocular CO poisoning, diplopia, double vision

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Background

It is well-documented that both acute and chronic exposure to carbon monoxide (CO) gas can have serious and permanent effects on a person's health and vision, especially if left untreated. Carbon monoxide is poisonous to the human body specifically because it binds to hemoglobin 225 to 240 times more tightly than oxygen. This results in a reduced oxygen-carrying capacity and the inability of oxygen to be distributed throughout tissues.¹ The extent of this damage depends on the concentration of the inhaled poisonous gas and the length of exposure. The parts of the body most affected by CO poisoning are those most susceptible to hypoxia.² Naturally, the brain and eyes are at risk upon exposure to this clear, odorless gas due to the large oxygen demands of these structures. This teaching case report provides a comprehensive evaluation of a unique case of systemic etiology that presented in an optometry clinic with both systemic and ocular symptoms. Specifically, it addresses the clinical techniques and diagnostic tests needed to come to the right diagnosis, and it evaluates the ancillary tests required to rule out other possible causes.

This teaching case report is appropriate for all levels of learners. For first- and second-year students, the recommended emphasis is an application of basic science to explain clinical presentation, elements of a thorough case history, and test selection. For third-year students, fourth-year students and residents, the same concepts can be emphasized, and the additional concepts of clinical application, assessment and management, and the optometrist's role on the healthcare team can be discussed.

Student Discussion Guide

Case description

Patient AK, a 24-year-old Caucasian female, presented at the Atwater Eye Care Clinic for a comprehensive ocular examination. She complained of a sudden onset of double vision, blurred vision, nausea, headaches and malaise on multiple occasions in the few days prior to the exam. She stated that she was in excellent health. She first noticed her blurry vision lasting 10-20

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minutes when running on the Friday prior to the exam and noted multiple recurrences throughout the next few days. Upon further questioning, the patient noted that symptoms were worse when at home. When asked about the conditions at home, she explained that her furnace had made a loud sound and malfunctioned in the middle of the night prior to initiation of symptoms. She reported that due to the temperature in the house, she had limited her time at home when she could. In fact, she did not spend the night before the examination at home and had only been in her home for an hour several hours before the exam.

At the time of the examination, she was not experiencing any symptoms that she had intermittently experienced. Her medical and ocular history were unremarkable, and she denied having any surgeries, environmental allergies or drug allergies. Her current medications included a daily multi-vitamin, fish oil, and Apri (desogestrel and ethinyl estradiol tablets). She stated that she had smoked socially in the past but was not a current smoker. She was oriented to time, place and person, and her mood and affect were normal.

The patient's pupils were equal, round and reactive to light and accommodation without afferent pupillary defects OU. Extraocular muscle versions were full and smooth OU without pain or diplopia, and Humphrey visual field 30-2 Sita-Standard testing was unremarkable OD and OS. Cover test revealed orthophoria at distance and a small (2 prism diopter) esophoria at near with no vertical deviation. Worth 4 dot testing was within normal limits at all distances under normal illumination. Fixation disparity testing with Saladin card revealed orthophoria at distance and near. Her habitual spectacle prescription was -0.75D OU, but she did not wear glasses regularly. Refraction yielded a best-corrected visual acuity of 20/20 OD and OS with a prescription of -0.75D OD and -1.25D OS. Negative relative accommodation (NRA), positive relative accommodation (PRA) and fused cross cylinder (FCC) were measured at +2.00, -1.50 and +1.25 respectively. Blood pressure was taken from her right arm while she was in a seated position and measured

110/78.

Slit lamp examination of the anterior segment was unremarkable. Her intraocular pressures were measured at 13 mmHg OU with Goldmann Applanation tonometry. Dilated fundus evaluation of the posterior segment using a +90D lens revealed healthy optic nerves with a cup to disc ratio of approximately 0.35/0.35 OU, healthy retinal vasculature and a healthy macula OU. Binocular indirect ophthalmoscopy using a +20D fundus lens revealed a flat and intact retina 360° OU with unremarkable findings.

Lab testing

Subsequent to the ocular examination, the patient was sent to the Indiana University Student Health Center for blood testing. Complete Blood Count (CBC), arterial blood gases and carbon monoxide blood testing were completed at 4:46 p.m. the same day. Arterial pH was found to be 7.52 (normal range = 7.35-7.45), HGB arterial blood gases were 14.8 G/dL (normal range = 12.0-17.0), arterial carbon monoxide was 0.8% (normal range = 0.0-3.0) and CBC was within normal ranges. Additional results were also unremarkable.

Follow-up call #1: two days after initial presentation

The patient reported that she had not experienced similar symptoms since her examination. Since then her heater/furnace had been turned off pending a replacement unit, and she had purchased and installed a CO detector. No extreme levels of CO had been detected since installation.

Follow-up call #2: three weeks after initial presentation

The patient reported no visual disturbances of any kind since the examination. She also noted that the heating unit issues had been fixed and that her CO detector had not indicated any abnormal levels. The patient was asked to call in if any symptoms or issues developed.

Educator's Guide

Literature review

Although carbon monoxide poisoning does not commonly present to an ophthalmic clinic, it is a significant issue that could have severe conse-

quences if not recognized early by the clinician. Nearly 70,000 cases of CO poisoning were reported in the United States between 2000 and 2009, with headaches and nausea presenting as the most common symptoms.³ Other studies have shown that blurred vision, photophobia and diplopia can also be associated with CO exposure.⁴⁻⁶ Males and females are equally susceptible, and CO exposures occur most commonly during the winter months with 77.6% of cases at residences.³ Most frequently, exposure to poisonous amounts of carbon monoxide occurs due to a faulty heating unit within a building. Other sources of high levels of CO exposure are automobile accidents and suicide attempts.

Many studies have attempted to identify the threshold value of CO exposure needed to cause harmful effects on the human visual system. It is known that large amounts of CO exposure will cause visual dysfunction and is made evident by high carboxyhemoglobin (COHb) levels.⁵ However, the CO exposure threshold that causes visual symptoms is highly controversial. Hudnell and Benignus concluded that COHb levels at 17% or lower are not detrimental to the visual luminance and contrast detection in young, healthy males.⁵ They came to this conclusion by observing the susceptibility of the human contrast sensitivity threshold and critical frequency flicker, which is the highest light frequency at which flicker is observed. This study showed that neither the contrast sensitivity threshold nor the critical frequency flicker were adversely affected at this COHb level.⁴ Other reports show that "slight" adverse effects were observed at a COHb level of 18% after exercise.⁵

Systemic symptoms of exposure are considered non-specific and include headaches, irritability, nausea, dizziness, myalgias, lethargy and other symptoms associated with hypoxia.² The literature describes signs associated with CO poisoning to be visual field loss with normal pupillary responses and papilledema at the nasal margin, but these signs are considered rare.⁷ Symptoms can present abruptly upon exposure or have a delayed onset. A recent report indicated that 10-30% of patients without any signs or symptoms

upon exposure actually experienced a delayed onset of neurological or psychiatric symptoms.⁴ If present, these delayed symptoms have been noted immediately or within days, weeks or even three years after exposure. They may manifest as amnesia, confusion, cognitive dysfunction characterized by attention and working memory deficits, emotional and personality disorders (depression or apathy), incontinence, or motor deficits similar to Parkinsonian symptoms.⁴ Research indicates the presence of these late-onset symptoms can be attributed to focal edema or demyelination within the cerebral white matter.^{4,8,9} However, it is important to note that many presentations of these late-onset neurological and psychiatric symptoms are based on the location of the defect, and therefore can be very subtle or even subclinical.⁴ This report also highlights the potential for saccadic dysfunction as a long-term adverse effect of CO poisoning, but saccadic function can also be disrupted in other neurological disorders and perhaps can only be confirmed as a non-specific sign of neurologic dysfunction.⁴ It would be pertinent for an eyecare professional to educate the patient about these possibilities and potentially recommend a referral to a neuro-ophthalmologist for a comprehensive ocular motor assessment (evaluation of saccadic function) or a comprehensive neurologic examination evaluating higher-order cognitive processing, including working memory, response inhibition and attention.⁴ With early recognition, the patient can benefit from the application of rehabilitative strategies to address these subtle defects that could be overlooked.⁴

Carbon monoxide is toxic to the human body because it binds to hemoglobin approximately 225 to 240 times more tightly than oxygen to form COHb.² Intake of carbon monoxide into the bloodstream causes a leftward shift in the oxygen-hemoglobin dissociation curve resulting in decreased oxygen-carrying capacity. When COHb is formed, oxygen transportation to different organs and tissues in the body is impaired.^{2,10} Once a person is removed from the source, carbon monoxide is eliminated from the body during exhalation and has a variable half-life from

two to five hours.^{2,11,12} Hypoxia and associated symptoms have been reported to occur at carbon monoxide levels greater than 100 ppm or at a COHb concentration greater than 30% in the bloodstream.¹⁰ Surprisingly, inter-individual differences in lung capacity, history of loss of consciousness, gender, age or duration of exposure have not been shown to affect the rate of CO elimination.¹³ One study shows that the only factor capable of influencing the rate of CO elimination from the body is the amount of 100% oxygen administered in a treatment setting, which allows a reduced COHb half-life with an increase in administered oxygen therapy.¹³

Parts of the body most susceptible to CO poisoning are those requiring large amounts of oxygen to function, such as the brain and heart.¹ Reports show that CO exposure can cause myocardial dysfunction in the presence of healthy cardiac tissue.¹⁴ The globus pallidus is the part of the brain most commonly affected, but reports also show that other basal ganglia nuclei, the thalamus, brainstem, cerebellum and cerebral cortex can also be involved.⁴ The eyes are also highly susceptible to hypoxia, and ischemic changes occur in the retina and optic nerves of patients exposed to CO for more than 12 hours.^{15,16} Specifically, the reported signs of ischemic retinopathy and neuropathy are as follows: superficial, flame-shaped retinal hemorrhages, venous tortuosity and engorgement, cotton wool spots, bilateral swollen discs, optic atrophy and retinal edema.^{15,10} Gass describes retinopathy due to CO exposure as multiple intraretinal and subretinal hemorrhages resembling those seen in Terson's syndrome.¹⁷ Specifically, Terson's syndrome is characterized by retinal and vitreal hemorrhages associated with subarachnoid or subdural hemorrhages.¹⁷ These hemorrhages associated with both systemic etiologies are caused by a sudden increase in venous pressure that causes peripapillary capillaries to rupture.¹⁷ Other fundus changes seen in CO poisoning are papilledema, venous engorgement and vessel tortuosity.¹⁷ It is uncertain whether ischemic injury to the vascular endothelium is the direct cause of this retinopathy, or if the physical compression of venous vessels by

the optic nerve edema plays a more significant role.¹⁷ In general, tissue damage associated with ischemia is characteristically manifested as increased capillary permeability and higher susceptibility to chronic damage.¹⁰

Due to the susceptibility of the central nervous system to hypoxia, debilitating effects on vision generally have a retrochiasmal origin due to responses stimulated by the neurological system rather than ischemic events within the ocular structures themselves.^{4,11} Vision loss and other adverse visual effects due to CO poisoning are considered rare, but neuropsychiatric effects such as alteration of mental state, amnesia, apraxia, Parkinsonism and other conditions listed above are more common manifestations. These effects are considered to have transient or permanent cortical involvement depending on the extent of the exposure.⁷ Within the brain tissue, the CO replacement of oxygen on hemoglobin will have an immediate effect and a delayed response. The immediate cellular injury due to hypoxia causes perivascular oxidative stress, which initiates the activation of reactive oxygen species such as NMDA (N-methyl-D-aspartate) and nNOS (neuronal nitric oxide synthase).¹⁸ This oxidative stress cascade and lack of anti-oxidant protection has been shown to promote the neuropathology associated with CO poisoning.¹⁸ This toxic damage and associated hypoxia disrupts the neurovascular autoregulation mechanism and causes endothelial damage to central nervous system (CNS) capillaries.⁷ This neuropathology and subsequent hypotension is exaggerated in "watershed" areas of the CNS due to limited anastomoses and their characteristic susceptibility to hypoxia.⁷ Delayed damage is caused by extensive lipid peroxidation in the neurons, which depletes overall ATP and leads to a cascade of cellular apoptosis and cerebral demyelination of the white matter in the brain tissue.^{7,18} This neuropathy will affect vision if oxidative damage occurs along any part of the visual pathway, specifically in the occipital lobe.¹⁹

It is important to rule out other potential causes of these symptoms (diplopia, headaches, blurred vision and nausea) such as trauma, extraocular muscle (EOM) palsy, butane poisoning, mul-

tiple sclerosis, fatigue and migraines. (Table 1)

- Trauma is generally indicated as a differential diagnosis in blurred vision and diplopia. For cases with possible orbital damage, an X-ray is warranted to rule out any fractures that may cause muscle entrapment leading to diplopia. For other cases involving traumatic brain injury (TBI), computed tomography (CT) or magnetic resonance imaging (MRI) may be necessary to rule out any inflammation or structural damage causing the patient's symptoms. Nearly 75% of severe TBI cases present with acute neurological deficits and are most commonly seen with automotive injuries. Though specific numbers are unknown, it is also possible to see delayed diplopia and other mild visual disturbances following less severe trauma as well.²⁰ This usually is a secondary result of causes such as damage to intraocular structures (lens dislocation, vitreous tears or hemorrhages), damage to the visual pathway, or cranial nerve palsies.^{20,21} In all cases of trauma, a detailed patient history is essential to help rule out or differentiate traumatic events and help guide management and treatment. Our patient reported no history of trauma, which quickly ruled this out.
- EOM palsy or a decompensated phoria can also be considered when patients present with vague symptoms of diplopia and headaches. While an EOM palsy presents slightly more often than a decompensated phoria (10% vs. 8%), proper testing can distinguish between the two differentials.²² To differentiate a decompensated phoria, an assessment of vergence ranges and a binocular vision workup would be warranted. An EOM palsy can be caused by a mechanical blockage within the orbit secondary to thyroid eye disease or orbital tumors. These patients typically present with associated proptosis or exophthalmos, which would highlight the underlying cause, but an MRI or CT scan would be needed to differentiate between these conditions. It can also be

Table 1
Differential Diagnosis

	Age Range	Gender	Symptoms	Additional Tests
Trauma	Any	Any	Diplopia, visual disturbances, blurred vision, proptosis	X-rays/CT for orbit fractures, CT or MRI for inflammation or other structural damage
EOM Palsy	Any	Any	Diplopia, headaches, proptosis, exophthalmos	Forced duction, Parks 3-step, red lens, MRI, CT, X-ray
Butane Poisoning	Any	Any	Dizziness, headache, neurological depression	Arterial blood gases, n-butane blood screen
Multiple Sclerosis	20-45	Women	Visual impairment, optic neuritis, uveitis, papillitis	Visual field testing, MRI, CT, SPECT
Giant Cell Arteritis	50+	Men	Headaches, visual disturbances, reduced visual acuity, malaise	Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), physical examination, temporal artery biopsy
Migraines	18-59	Women	Unilateral, extreme headaches, nausea, vomiting, and/or visual aura. Fatigue, dizziness	Physical examination

caused by a neurological defect owing to complete or partial paresis of the extraocular muscles. The cause can be differentiated by the forced duction test or other tests, specifically the Parks 3-step or the red lens test can be used to identify the defective muscle(s). Because binocular vision testing and motility were within normal limits for the patient, this was ruled out as a differential.

- Butane poisoning exhibits similar symptoms to CO poisoning, including dizziness, headache and neurological depression. Symptoms vary depending on the severity and duration of intoxication.²³ It is important to differentiate from this due to the vast number of households that use gas as a heat source. According to the Centers for Disease Control, more than 50% of homes in the United States use a mixture of natural gas, butane and propane as the primary source of home energy.²⁴ In addition, butane is commonly used as a propellant for spray deodorant. A characteristic pungent odor can be associated with these gases to easily identify a leak. However, misuse and intentional abuse are very common causes of this poisoning and should also be considered during examination.²³ It is important to note that CO results from the burning of butane gas, and thus most cases of butane poisoning are

differentiated from a thorough case history. Additional testing includes arterial blood gas and n-butane blood screens.

- Multiple sclerosis (MS) is an inflammatory, demyelinating central nervous system disease that typically presents in women age 20-45. It is associated with periods of remission between exacerbations. The signs and symptoms vary depending on the area affected in the brain or optic nerve and can often cause optic neuritis or ocular motility dysfunction.²⁵ Ocular signs and symptoms include visual impairment, optic neuritis, uveitis, papillitis, etc. A thorough history to evaluate any acute episodes of onset and remission of pain, visual field testing to determine any unilateral visual defects, and motility testing are helpful in differentiating early MS in patients. Areas of cortical blindness caused by CO toxicity can appear as occipital injury, white matter lesions and cerebral ischemia. These can sometimes be apparent through imaging techniques such as CT, MRI and single photon emission computed tomography (SPECT).^{19,26} While the patient fits the age range for this particular differential, it was ruled out based on the unremarkable results of the tests performed on the patient.
- Giant cell arteritis (GCA) and

other ischemic optic neuropathies have been falsely diagnosed in the presentation of carbon monoxide poisoning. Both problems have similar symptoms, such as headaches and visual disturbances with grey patches, reduced visual acuity and malaise.²⁷ GCA testing includes physical examination, temporal artery biopsy, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) blood tests. GCA typically presents in Scandinavian men older than 50, with incidence rising greatly with age. This very rarely affects people under the age of 50 and was ruled out for our patient.²⁸

- Migraines are typically unilateral, extreme headaches and usually accompanied by nausea, vomiting and/or an occasional visual aura. Migraines can present with a prodrome that includes irritability, fatigue, dizziness, stiff muscles and other symptoms followed by subsequent auras, pain and headaches. They affect nearly 18% of women and 6% of men. Symptoms range from mild to disabling. Peak incidence of migraines is seen in women 30-39 years of age, but general high prevalence is reported between 18-59 years of age.²⁹ According to the International Headache Society Classification of Headaches, diagnosis of a migraine requires symptoms of nausea, vomiting or photophobia in addition to two of the following features: unilateral location, throbbing, worsening with routine activity or moderate to severe intensity.³⁰ A thorough history is required to classify a migraine, but was ruled out for our patient due to the negative history.

The literature describes several tests that can be used to diagnose CO poisoning. COHb levels can be tested to determine whether the patient has been exposed to carbon monoxide, but this test cannot be used to determine the severity of exposure or dictate prognosis. COHb levels also should not be used to dictate the extent of necessary treatment.¹ Other blood components and arterial blood gases are used to further illustrate the current status of CO poisoning within the bloodstream. Specifi-

cally, low arterial pH has been directly associated with increased mortality in carbon monoxide poisoning with a 30% mortality rate linked to an arterial pH of 7.25 or lower.³¹ Arterial pH is lowered by COHb formation and deterioration in intracellular energy metabolism.³¹ A calculated oxidative stress index (OSI) can also be very helpful in predicting the potential for delayed neurologic damage, so it is a good test to determine prognosis and dictate a specific treatment plan.³² Finally, the literature also supports the use of positron emission tomography (PET) and SPECT scanning to determine which areas of the brain have become damaged or will show adverse effects even when MRI and CT imaging are within normal limits.¹⁹ Unlike MRI or CT scans, these imaging studies both use radio-labeled, molecular components of a normal physiologic process that emit positrons or gamma rays when they decay. This allows quantitative imaging of a particular physiologic process, such as glucose metabolism in PET or cerebral blood flow in SPECT.¹⁹ SPECT imaging with [^{99m}Tc]HMPAO of patients with visual loss secondary to carbon monoxide poisoning showed defects in cerebral blood flow within structures along the visual pathway.⁹ Additionally, in patients with visual loss who were exposed to CO, fluorodeoxyglucose PET imaging showed deficits within the occipital region when MRIs were considered normal.¹⁹ PET and SPECT imaging can be clinically valuable in determining the prognosis of patients affected by carbon monoxide because most MRI imaging fails to highlight damaged tissue.¹⁹

The standard treatment for CO exposure is considered to be oxygen therapy administered with different efficacies in different vehicles. The elimination of carbon monoxide from the body is accelerated by allowing oxygen to compete more effectively at hemoglobin binding sites. Using a face mask to administer high-flow, 100% oxygen, the half-life of carbon monoxide in the body can be decreased to 60-90 minutes.² A newer, more expensive alternative called hyperbaric oxygen therapy (HBO) has been found to reduce the half-life to 20-30 minutes if administered through multiple sessions at 3 atm.² This therapy in-

volves a pressurized chamber that contains increased oxygen levels compared to normal atmospheric pressures.² Like other oxygen therapies, it is designed to administer higher levels of oxygen for inspiration, and its higher efficacy results in carbon monoxide being displaced more easily.² It is recommended to continue high-concentration oxygen therapy until the COHb level is below 5%.¹³ Oxygen therapy also increases the oxygen reserve in the bloodstream and supplements the hypoxic parts of the body until equilibrium is reached.²

Intended audience for this teaching case report

This teaching case report is appropriate for all levels of learners. Recommended emphasis for first- and second-year students: application of basic science to explain clinical presentation, elements of thorough case history, test selection. Recommended emphasis for third-year students, fourth-year students and residents: the same as for first- and second-year students, with the addition of clinical application, assessment and management, and the optometrist's role on the healthcare team.

Learning objectives

- To describe common and uncommon ocular presentations found with acute, excess CO exposure
- To describe long-term effects of carbon monoxide poisoning
- To describe the general effect that carbon monoxide has on the body, eye and visual pathways, both immediate and delayed
- To describe retinal hypoxia and associated signs and symptoms
- To describe the physiological mechanism causing immediate vs. delayed damage and associated structures
- To identify key diagnostic tests for CO poisoning
- To describe treatment options for CO poisoning
- To apply critical thinking skills to the care of a patient (i.e., know how to develop differential diagnoses and know what tests can rule out each possible cause)
- To understand the role and re-

sponsibilities of the primary care optometrist in the management of a patient with symptoms suggestive of carbon monoxide poisoning (i.e., know how to take a good case history and know what questions to ask relevant to CO poisoning and how to provide proper patient education)

- To know which healthcare professionals to make a referral to depending on cause and extent of CO exposure

Key concepts

- The body's response to acute hypoxic events, including ocular and systemic symptomatology
- Testing and procedures used for the differential diagnosis of CO poisoning
- Events and risk factors leading to carbon monoxide poisoning
- The pathophysiology of carbon monoxide on the body/eye/visual system
- Treatment of CO poisoning

Discussion topics

- Ocular anatomy:
 - o retinal layers
 - o retinal blood supply
 - o sources of energy
 - metabolism of the retinal layers
- Neurological anatomy:
 - o occipital lobe
 - anatomy of the occipital lobe including blood supply, visual pathways, possible visual field defects
 - function of the occipital lobe
- Regarding carbon monoxide poisoning
 - o environmental, social and demographic factors and the risk for CO poisoning
 - o common sources of carbon monoxide exposure
 - o carbon monoxide poisoning vs. other gas poisoning
 - o ocular findings associated with acute hypoxic events

- o ocular and medical history (i.e., how to take a history on a patient exposed to CO)
- o ocular findings
- o differential diagnosis
 - trauma
 - EOM palsy
 - butane poisoning
 - multiple sclerosis
 - giant cell arteritis
 - migraines
- o visual fields (i.e., differentiate between a pre-chiasmal, chiasmal and post-chiasmal defect)
- o laboratory testing
- o treatment options
 - standard treatment options available
 - patient education on indications and complications of treatment
 - devices for detection of carbon monoxide
 - long-term effects of carbon monoxide poisoning
 - necessity and type of referrals
- o patient education on long-term effects (i.e., population at greatest risk, long-term sequelae, proper referral for long-term management)

Discussion

Case history

Upon initial presentation, the patient complained of vague and general symptoms of blurriness and double vision that had resolved by the time she arrived at the clinic. She also mentioned episodes of headaches, malaise and nausea in correspondence with her ocular symptoms. The most common symptom associated with CO poisoning is a headache, but other visual symptoms such as photophobia, blurred vision and diplopia have been documented as well.³³ With cases of carbon monoxide exposure, it is important to take a detailed and thorough ocular and medical history in order to properly diagnose and identify the cause of exposure. The clinician should ask questions regard-

ing the specific time and duration of the symptoms in addition to identifying any specific palliative or provocative factors involved. In this case, the symptoms were exacerbated while the patient was in her apartment and improved after she would leave for some time. It is also warranted to ask what the patient was doing when she noticed the symptoms. She stated that her symptoms peaked when she went running despite the fact that she was physically fit and a perpetual runner. It is shown that carbon monoxide poisoning can manifest at lower levels of exposure during exercise.⁵ Particularly when symptoms are seemingly unusual or vague, it also is important to ask if anything has changed in the patient's personal, social or medical history within the past few months. In this case, the patient was able to remember that the furnace in her apartment complex had malfunctioned within the past week. The symptoms of vision loss associated with CO poisoning are either absent or very vague in most cases, but without a proper diagnosis and treatment the long-term effects to vision can be catastrophic. The clinician must rely on thorough history-taking and diagnostic tests to reach a diagnosis.

Confirmation of diagnosis

There are several diagnostic tests that can be used with a suggestive case history or with vague symptoms where a diagnosis is unclear. Measurement of COHb levels is the standard of care when carbon monoxide exposure is suspected. This helps to determine whether the individual has been exposed to carbon monoxide, but it is unable to determine severity of exposure or prognosis.¹ It only indicates whether the patient has been exposed within the time frame it takes to eliminate carbon monoxide (COHb) from the bloodstream, and any concentration level above 2-3% is considered abnormally elevated.^{13,11} The COHb level in this patient was 0.8%, which is within the normal range and not enough to cause symptoms. However, the time frame between testing and exposure must be taken into consideration. This patient was initially seen at 1:10 p.m., and blood work was performed at 4:46 p.m. Additionally, she had not spent a considerable amount of time near the

source of exposure prior to blood testing because she had not slept at her apartment for more than 24 hours. This measurement can give a false negative if there is a delay of more than 12 hours between testing and the last exposure as in this case because CO levels in the blood decrease exponentially after the patient is removed from the source.^{13,32} The improvement in symptoms after exposure is ceased is an obvious indication of CO poisoning, and the delay in COHb testing should be considered as the reason for a false negative in this case.

However, carbon monoxide toxicity is also considered a diagnosis of exclusion. Other testing must be administered to rule out differentials and other systemic etiologies. In this case, the patient presented with previous episodes of blurry vision and diplopia. Subjective refraction revealed a mild amount of uncorrected refractive error, but the fact that the patient was familiar with being habitually uncorrected implies that her episode of blurry vision was acutely severe and had resolved by the time of the exam. The patient's intermittent blurry vision was also assessed by performing NRA, PRA and FCC. This accommodative testing was performed to determine the potential for latent hyperopia and confirm accuracy of the subjective refraction. Humphrey visual field testing was utilized to further analyze the patient's unspecified loss of vision. Specifically, it is important to know that the patient was not experiencing any peripheral vision loss or focal loss of sensitivity within her visual field.

When a patient presents with episodes of double vision, it is important to perform a preliminary binocular vision workup and extraocular muscle assessment to rule out any ocular cause of diplopia. In this case, the patient's binocular vision status was considered within normal limits, as defined by fixation disparity and phoria testing. Also, no palsy or muscle restriction was noted. If the diplopia had been unresolved at the time of the exam, it would have been prudent to determine comitancy and severity. This additional testing sufficed to rule out an ocular cause for the complaint of episodic, intermittent diplopia.

It is also important to order other blood and ancillary testing to aid in evaluating possible systemic etiologies. In this case, CBC with differential and arterial blood gases were ordered to give normal values with the exception of an elevated arterial pH. This systemic alkalosis could be an indication of the body's adaption mechanism to reduce the adverse effects of carbon monoxide within the body. Measuring other blood components or ordering an electrocardiogram (ECG) or chest X-ray would be other ways to monitor the cardiovascular side effects associated with carbon monoxide exposure. Other testing that could have provided more information about prognosis would be a calculated OSI value and PET and SPECT imaging. These tests would have been required if other neurological symptoms had persisted because their results indicate the extent of any delayed neurologic injury.

Management and treatment

The initial treatment for acute CO poisoning is identifying and quickly removing the source of exposure. Because CO is removed primarily via pulmonary circulation, treatment options are directed toward increasing oxygenation to decrease the half-life of CO within the body. Mild to moderate exposure can be treated using high-flow, 100% oxygen in isobaric conditions. This has been shown to decrease COHb half-life from 300 to 90 minutes.² In more severe cases without improvement, hospitalization and HBO may be required. This method can decrease COHb half-life to just 30 minutes within the body, but facilities are limited within the country.² Special attention must also be given for children and pregnant women due to their higher oxygen requirements and greater risk for hypoxic damage. Because CO bound to fetal hemoglobin has a longer half-life, it is essential to initiate treatment immediately for these patients.¹¹ In patients at risk for heart disease, it is also advisable to obtain an ECG to monitor for any possible cardiac ischemia.³⁴

Long-term prognosis following CO poisoning can vary depending on the severity and duration of intoxication. Once maximal therapy is implemented, a physical assessment of overall health should be performed. Though uncom-

mon, it is possible to see delayed neurocognitive function in certain patients, so a neurological evaluation should also be done.³⁵ Our patient presented with very unspecific symptoms associated with her CO exposure, so it was important to educate her on the possibility of these adverse effects in neurocognitive and psychological function.^{4,35} COHb levels should be monitored regularly over time, if possible, until levels are normal. Patients should be thoroughly educated on risk factors, common sources contributing to CO poisoning, and early clinical findings to ensure they do not have a repeat event. Preventative measures including CO detectors and routine furnace maintenance should be taken.

Cases of intentional poisoning, though not as common, should be handled very cautiously. Most suicide attempts by carbon monoxide are seen in middle-aged men and typically in rural regions.³⁶ A variety of social problems have also been correlated with the incidence of suicide by CO poisoning, so the support of family and friends is essential during the recovery period.⁽³⁶⁾ These patients along with their support network of family and friends should be educated on the symptomatology, and patients should undergo psychological evaluation as soon as possible to determine the potential risk for suicide. They should be monitored very closely by their primary care physician and psychiatrist in order to receive proper treatment.³⁶

Conclusion

This case demonstrates the importance of a thorough patient history and comprehensive examination in diagnosing cases of an uncommon etiology. Rather than diagnosing from a particular finding, this particular case has shown that diligently ruling out differential etiologies and asking certain questions during an exam can lead to a proper and successful diagnosis. Cases such as this are rarely seen in an optometric setting, as they generally are caught in an emergency room or by a family doctor. However, this case highlights the need for eyecare professionals to recognize the possible ocular, systemic and neurological effects of carbon monoxide poisoning and the impending, long-

term risk factors. Mild, acute CO poisoning presents with general headache, malaise and blurred vision, and rarely diplopia. These particular symptoms must be correlated and diagnosed early in order to prevent chronic problems for patients. Proper education about long-term effects, elimination of the cause, and integration of preventative measures for future incidences must be implemented for patient safety.

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