Pediatrics Research Directory

Open Air Carbon Monoxide Poisoning in a Child Swimming Behind a Boat

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Abstract and Introduction

Abstract

Carbon monoxide (CO) poisoning is the most common fatal poisoning in the United States. The circumstances often involve an unsuspected increase of CO in an enclosed environment. Victims often are unaware that their activity or environment placed them at risk for CO poisoning. The possibility of open air CO poisonings was first reported in 1987. We present a case of open air CO poisoning resulting in neurologic depression and a markedly elevated carboxyhemoglobin level in a child who had been swimming behind a house boat. Emergency physicians and pediatricians should be aware of the possibility of accidental open air CO poisoning in children and adults who swim around recreational boat

Introduction

Carbon monoxide (CO) Poisonings are responsible for approximately 3,500 deaths in the United States each year.[1] Unintentional or accidental CO poisonings account for 30% of these deaths. Of unintentional deaths from CO poisoning, 5% to 14% occur in infants and children, who are often the victims of enclosed CO poisoning in motor vehicle and residential locations.[1,2] Previous reports have described open air CO poisonings in adults.[3-5] Carbon monoxide poisonings of adults and children during recreational boating have also been reported.[6] but these were related to enclosed not open air exposures. We report a case of severe CO poisoning that occurred while swimming behind a boat. This common recreational activity might pose a serious unrecognized health risk to both children and adults. Physician awareness that open air CO poisonings can occur may lead to a better definition of the frequency and severity of this problem and to improved diagnosis, treatment, and prevention.

Case Report

A 4-year-old, 14-kg girl was brought to an outside emergency department after a period of unresponsiveness and near drowning while swimming in a fresh water lake. She had previously been healthy, without a medical history of trauma, harmful ingestion, or seizures. Her family history was unremarkable. On the day of presentation, she had been without complaint. Weather conditions described by the National Weather Service for that day in June were temperature of 66° to 87°F, wind speed of 8 miles per hour, and trace precipitation. The water was calm and warm.

The boat was anchored in a cove with 3 to 4 other non-operating boats. The main boat behind which she had been swimming was a privately owned recreational vehicle (1989 model house boat, 50 feet in length). All propeller engines were shut down, and the boat had been anchored for approximately 3 to 5 hours. The only operating engine was a factory installed, gasoline fueled, electrical generator that vented 1 to 1 1/2 inches above the water surface at the stern of the boat. The boat was equipped with two CO detectors with digital displays and auditory alarms; one in the main cabin and one 15 feet from the stern (swim deck) of the boat. Neither detector had shown a CO level or alarmed that particular day.
The patient, along with a group of other children, was swimming behind the stern at a distance of 15 to 20 feet. The patient was swimming in a ski jacket and with direct adult supervision. The only variance noted in her activity from the other swimming children was that she came to the swim deck of the boat more often for reappllication of sunblock. The first indication of a problem occurred just moments after she had reapplied lotion. She began floating supine in the water and was unconscious and rigid. She was quickly brought onto the boat and appeared "pale and stiff" by parental report. She was unresponsive with poor respiratory effort. Emergency medical services were immediately notified. After 2 to 3 minutes of aggressive stimulation, she began responding with grunts but was described as "disoriented and sleepy." Paramedics arrived in 10 to 15 minutes, administered oxygen (15 L/min) by nonbreather mask, and transported the child to the nearest emergency department (ED) within 30 to 45 minutes after the episode.

Her appearance was described as "pale with cherry red lips." She was appropriately responsive to person and place. Vital signs were temperature 36.6°C, blood pressure 90/56 mm Hg, respiratory rate 32/min, and heart rate 119/min. Her pupils were equal and briskly reactive to light. The remainder of the physical examination was unremarkable. Serum electrolyte measurements, blood count, toxicology screens and a venous carboxyhemoglobin (COHb) value were obtained. The COHb determination was done because the ED physician suspected CO poisoning. Electrolyte values, including calcium and magnesium, and complete blood count were normal. Arterial blood gas values were normal except for a venous COHb level of 22.2% (normal, 0.1% to 2.0%). Normobaric O2 therapy at 15 L/min with a nonbreather mask was continuous from the time of her assessment in the field. On the basis of the high COHb level and the clinical symptoms, a diagnosis of CO poisoning was made, and the patient was referred to University of Missouri Children's Hospital for hyperbaric oxygen therapy. No other members of the group, adults or children, had headache or other symptoms of CO poisoning.

On arrival to our facility 3 hours after presentation, the patient remained alert and interactive. Physical examination continued to show the "cherry red" lips. Vital signs were temperature 36.4°C, blood pressure 101/52 mm Hg, respiratory rate 24/min, and heart rate 82/min. Normobaric O2 therapy at 15 L/min was continued during preparation for hyperbaric therapy. Venous blood gas values with co-oximetry were pH 7.37, PCO2 40 mm Hg, PO2 69 mm Hg, HCO3 - 23 mEq/mL, base excess -1.1 mEq/mL, hemoglobin 11.5 g/dL, COHb 1.1%, and measured arterial oxygen saturation (SaO2) 94.8%. Hyperbaric oxygen therapy was given at 45 feet of sea water, or 2.4 atmospheres absolute, for 2.5 hours. Venous blood gas values after treatment (6 hours after presentation) were pH 7.4, PCO2 37 mm Hg, PO2 86 mm Hg, HCO3 - 23 mEq/mL, base excess 0.8 mEq/mL, hemoglobin 11 g/dL, COHb 0.4%, and measured SaO2 97.2%. Creatine kinase level was normal. At 18 hours after CO intoxication, the patient was discharged to parental care, and 2 months later she remained without adverse neurologic sequelae.

Discussion

Carbon monoxide is a colorless and odorless gas that can accumulate rapidly in the air, lungs, and blood. It binds to hemoglobin with 200 times the affinity of O2, producing a "biochemical" asphyxia to brain, heart, and other body tissues.[7,8] Signs of toxicity include headache, nausea, dizziness, confusion, vomiting, and lethargy. Unless severe, these symptoms are difficult to attribute to CO toxicity and may be misdiagnosed as other more benign processes, such as seasickness, sun exposure, or a viral illness.[6,9,10]

The yearly incidence of nonfatal CO poisonings is unclear. The Centers for Disease Control and Prevention estimates that CO poisoning is diagnosed in 10,000 people per year. This source also suggests that many more children and others are unknowingly exposed, improperly diagnosed, or choose not to seek medical attention.[11] Some may die of unrecognized CO poisoning, making the exact number of cases difficult to establish. Neurologic sequelae of CO toxicity, the most commonly identified acute and late morbidity, occurs in 14% to 40% of patients.[7,12] Other reported complications include cerebral edema, cardiac ischemia, rhabdomyolysis, and thrombosis.[13-15]
Diagnosis is made by considering, identifying, and recognizing symptoms of CO poisoning. Clinical suspicion then leads to a laboratory measurement of COHb in arterial or venous blood (0.1% to 2.0% is normal).[7] During acute CO intoxication, the physical examination can be remarkable for "cherry red" mucous membranes, tachypnea, tachycardia, and hypotension. Unfortunately, these symptoms may be absent in many children with CO poisoning.[9,16] Studies of children with CO intoxication report that syncope/unconsciousness occurs in 40% to 50%, headache in 56%, lethargy in 25% to 68%, and nausea in 75%. These are more prevalent features of CO toxicity in children and may be more predictive of severe CO poisoning.[10]

Pulse oximetry is inaccurate because COHb absorbs light within the wavelengths monitored by the device. This results in the pulse oximeter interpreting the wavelength of COHb as oxygenated hemoglobin and then reporting a falsely elevated reading for oxygen saturation of hemoglobin (SpO2).[17] Other laboratory tests are nonspecific, and no correlation exists between measured COHb levels and risk for adverse outcomes, especially in children.[9]

The difficulty in recognizing CO toxicity in patients during recreational boating has been previously reported. In a retrospective study, Silvers and Hampson[6] identified enclosable cabins and age of the boat (more than 10 years) as risk factors for CO poisoning. They did not report or recognize any cases of open air poisoning in their study. Given that other passengers were without symptoms and that there was no detection by CO monitors on the boat, it is unlikely that our patient's CO poisoning occurred in the enclosed boat.

Cases of open air CO poisonings have been reported. DiMaio and Dana[3] identified 5 suicide cases involving intentional open air CO poisonings from car exhaust. In each instance, the suicide was committed in an outdoor setting, but the decedents placed their face in close proximity to the exhaust pipe of a running automobile engine. In a case similar to ours, Jumberic[4] reported a case of drowning from an unintentional CO poisoning in a teenager who was swimming behind an idling boat. The COHb value was 62% on postmortem examination. Uncontrolled duplication of the situation using the same boat engine revealed a CO level of 100 parts per million at water level. Additional cases of unintentional CO poisoning in open air settings have occurred in adults during operation of machinery in outdoor settings.[4,5,18] The CO source in our case appeared to be the gasoline fueled electrical generator that vented above the swim platform. Ours is the first reported case of a child with diagnosed CO poisoning occurring in an open air environment.

Treatment of CO poisoning should begin in the field by identifying patients at risk for CO poisoning, removing the patient from the suspected site/source of exposure, and providing fresh, outdoor air.[19] Normobaric treatment with an FIO2 of 1.0 via a high flow or a nonrebreathing system should be instituted in all cases of suspected CO poisoning.[13,19] Normobaric treatment alone can decrease the elimination half-life of COHb from 5 hours to 1 hour.[16] This explains the decrease in COHb levels from 22% to 1.1% in our patient from initial presentation at the outside ED to arrival in our hospital.

Hyperbaric oxygen therapy is a treatment for severe CO poisoning (ie, in patients with COHb of 15% to 20% and/or syncope, seizures, coma, cerebral edema). Because of the limited availability of hyperbaric oxygen therapy, and the rapid reduction in measured COHb with normobaric treatment alone, multiple studies have looked for benefits of hyperbaric over normobaric treatment in preventing long-term neurologic sequelae.[19-23] Until more definitive studies are done, hyperbaric treatment when available should be strongly considered for use in patients with findings of severe CO poisoning. We believe our patient's symptoms, the elevated COHb level at the outside hospital, and the observation that neurologic damage can continue even after COHb levels normalize warranted hyperbaric therapy.

The greatest risk factor for accidental CO poisoning is "unrecognized" exposure. Our case represents a common activity -- swimming behind a boat that has a generator running -- that unknowingly placed this child at risk for CO poisoning. With the number of families participating in recreational boating steadily increasing every year, physicians should consider CO poisoning when a child or an adult has unexplained mental status changes, seizures, or lethargy while on a boat or near recreational boating. Carbon
monoxide poisoning should also be in the differential diagnosis of unexplained drowning or near-drowning events during recreational boating in both children and adults. Until more definitive studies are available, physician education and increased public awareness are the most effective means of improving diagnosis, treatment, and prevention of CO poisoning in children during recreational swimming and boating activities.

References

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