CLINICAL CASE REPORT

Carbon monoxide poisoning while scuba diving: a rare event?

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ABSTRACT

Contamination of breathing gas is a risk for all divers. Some hydrocarbon contaminants will be sensed by the diver and the dive profile aborted. On the contrary, carbon monoxide may not be recognized by the diver and catastrophic consequences can result. Reported here is the fatal case of carbon monoxide poisoning while scuba diving, an event that has rarely been reported in the medical literature. A detailed review of other published cases of CO poisoning while scuba diving is included, attempting to identify causes in common and propose methods of prevention. ∎

INTRODUCTION

Carbon monoxide (CO) poisoning is a recognized risk of underwater diving with compressed-gas breathing. It results when breathing gas, delivered either from a scuba cylinder or surface-supplied from a compressor, is contaminated with CO. Despite the fact the condition is recognized, remarkably few cases of CO poisoning during scuba diving have been reported in the literature. A fatal case is reported here, along with a detailed review of the prior reported cases.

CASE REPORT

The patient was a 42-year-old previously healthy female who was scuba diving in salt water with a buddy and breathing compressed air. The dive profile was multilevel, with a maximum depth of 121 feet of seawater (fsw) and a total bottom time of 51 minutes. The divers ascended unremarkably and in a controlled fashion to 25 fsw, where they reportedly were completing or had just completed their decompression obligation when the afflicted diver developed a sudden change in mental status and dropped the regulator from her mouth. Unable to reinsert her mouthpiece, the dive companion inflated her buoyancy compensation device and they rose rapidly together to the surface.

They reached shore within five minutes, where the injured diver was described as unconscious and pulseless. Bystander CPR was immediately initiated and continued for 10 minutes until paramedics arrived. They found her pulseless with heart rate of 10 beats/minute on a monitor, responsive to atropine and epinephrine. Endotracheal intubation at the scene returned a copious amount of seawater.

Upon arrival in the emergency department at 13:15 hours the patient was described as a comatose, unresponsive, intubated, middle-aged female with systolic blood pressure 90 mmHg, pulse 110/minute (sinus rhythm), and temperature 31.4°C by esophageal probe. Initial laboratory results were notable for glucose 261 and arterial blood gas analysis with pH 7.14, partial pressure of carbon dioxide ($PaCO_2$) 26 mmHg, and partial pressure of oxygen in arterial blood (PaO_2) 321 mmHg while breathing 100% inspired oxygen. Chest radiograph demonstrated diffuse pulmonary infiltrates. Hypotension was treated with 7,600cc fluid resuscitation, and the patient rewarmed to 37.°C. She developed atrial fibrillation with rapid ventricular response and associated hypotension, refractory to medication and treated with cardioversion.

Carboxyhemoglobin (COHb) was 15.0% when measured at 14:40 hours, an estimated two hours after the dive accident and following 90 minutes of ventilation with 100% oxygen via endotracheal tube. Repeat level was 11.3% at 15:15 hours.

Arterial oxygenation deteriorated, with partial pressure of oxygen (PO_2) 35 mmHg on 100% oxygen plus 10 cm positive end-expiratory pressure (PEEP) and 29 mmHg on 15 cm PEEP. She was admitted to the critical care unit to attempt stabilization. Myoclonic jerking was noted upon her arrival there. The following day, neurologic status was not significantly changed. Head CT scan demonstrated cerebral edema and some abnormalities possibly representing cortical infarction. The patient was treated in a multiplace hyperbaric chamber using a U.S.

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Navy Treatment Table 6 profile, complicated by hypotension unresponsive to fluid administration and requiring initiation of vasopressors. Arterial blood gas analysis during mechanical ventilation in the chamber at 2.8 atmospheres absolute showed pH 7.42, PCO_2 33 mmHg, and PO_2 >800 mmHg.

The patient was unchanged following hyperbaric treatment. She was subsequently diagnosed to have severe, irreversible brain injury, and support was withdrawn following appropriate consultation with her family. The autopsy demonstrated diffuse cerebral edema, bilateral cerebellar tonsillar herniation and bilateral bronchopneumonia. COHb at autopsy was less than 5%. Analysis of breathing gas in the tanks of both divers demonstrated 1,320 ppm carbon monoxide. The tanks had been filled from the dive companion's combustion-powered air compressor.

Cause of death was determined to be hypoxic encephalopathy due to accidental carbon monoxide poisoning.

DISCUSSION

In a review of 451 diving fatalities occurring from 1993-1997 in the Divers Alert Network database, Caruso and co-workers found only two that were felt to have been caused by carbon monoxide poisoning [1]. Details of the two deaths were not provided outside of the fact that both had COHb levels of 32% at autopsy. While CO poisoning was therefore identified as a rare cause of recreational diving fatality, the authors speculated that cases of CO toxicity may be underdiagnosed. In that review, COHb levels were known to have been measured in only 15% of dive fatality cases.

In fact, there has been a paucity of cases of CO poisoning of any severity during scuba diving reported in the literature. The first was Jacques Cousteau, diving with companions in a cave in France in 1946, and reported in his book, *Silent World* [2]. The dive group experienced headache, confusion, weakness and near loss of consciousness at depth while breathing compressed air from tanks filled by a new diesel-powered compressor. Subsequent analysis of gas from their tanks showed 500 ppm CO, and examination of the compressor revealed that it drew exhaust into its air intake.

In 1972, Furgang described a diver who dove to 30 feet breathing air for 60 minutes [3]. Immediately upon surfacing he noted onset of headache, fatigue, incoordination and confusion. Symptoms cleared over two days. He refilled his dive tanks from his own gasoline-powered compressor and dove again to 30 feet. After 10 minutes at depth, he lost consciousness and was brought to the surface unconscious and pulseless. CPR was initiated and he regained consciousness in 30 minutes. He was combative, confused and hallucinating, and was believed to have suffered cerebral arterial gas embolism. Hyperbaric treatment was begun on U.S. Navy Treatment Table 6A. When he did not improve after 30 minutes at 165 fsw, treatment was continued on Table 4. Mental status improved after 12 hours and totally cleared after an additional six hours. Analysis of the gas in his tank revealed 4,180 ppm CO resulting from a faulty air compressor.

In 1973 Daenens reported a scuba diver in the Netherlands who made a dive to 25 meters while breathing compressed air. He immediately developed nausea and vomiting, followed by lower extremity weakness [4]. Within two to three minutes at depth, he signaled distress to his co-diver, then lost consciousness. His dive partner inflated a rescue buoy which carried the unconscious diver to the surface. He remained semiconscious for an hour, then regained consciousness spontaneously. Testing the air from his tank revealed 1.6 vol% CO (16,000 ppm), again traced to a faulty compressor.

In 2016 Smithius reported a second scuba diver from the Netherlands who made a dive for 30 minutes to a maximum depth of 17 meters, ascending when he felt lightheaded, confused and experienced loss of motor control [5]. Physical examination was unremarkable; COHb was 13.6%. He recovered after recompression therapy. Police investigation discovered a batch of CO-contaminated tanks.

In 2018, McDermott et al. reported an incident in England involving 10 boys taking a scuba lesson in an indoor pool [6]. Six developed symptoms consistent with CO poisoning and demonstrated COHb levels ranging from 6.2-32.8%. The most severely affected boy experienced transient loss of consciousness, pulmonary edema, troponin elevation and head CT scan changes of diffuse white matter hypodensity and cerebral edema. All the boys affected recovered completely with normobaric oxygen therapy. Eight of 15 air tanks collected from around the pool demonstrated greater than 1,000 ppm CO. Gas samples from the high-pressure bank at the dive center supplying the cylinders demonstrated 2,550 ppm CO resulting from a faulty compressor. Based upon serial COHb measurements, the authors back-extrapolated the most severe case to have had a peak level of 43.7%.

Back-extrapolating a CO-poisoned patient's carboxyhemoglobin level is a notoriously crude calculation, due in large part to errors in estimates of time breathing sup-

plemental oxygen and the fraction of oxygen actually delivered. In the present case the patient was intubated and ventilated with 100% oxygen for 90 of the estimated 120 minutes from dive accident to COHb measurement. Weaver and co-workers found the half-life of COHb to be 58 ± 32 minutes in 10 intubated CO-poisoned patients treated with normobaric 100% oxygen [7]. Applying that figure to the current patient would yield approximately 1.5 half-lives of known oxygen breathing and an initial COHb level of 40-50%.

Toxin contamination of breathing gas with resultant loss of consciousness underwater is a recognized complication of diving [8]. When the contaminant is a hydrocarbon, its presence may be noted by the diver and the profile aborted or gas supply changed before alteration in level of consciousness occurs. Conversely, since CO is insensible to humans, the scuba diver breathing contaminated gas unknowingly onloads CO throughout the dive. The toxic effects of CO during diving are often described as having their onset during ascent from depth [8], as was the case in this diver. The partial pressure of oxygen in blood and tissues is greatest at depth and decreases during ascent. Arterial oxygen delivery declines due to a reduction in dissolved and hemoglobin-bound oxygen, as does the antagonistic effect of oxygen on CO.

Cerebral arterial gas embolism due to expanding intra-alveolar gas is the first diagnosis to be considered when loss of consciousness occurs during ascent from depth. CO poisoning should also be considered when a diver loses consciousness while ascending, especially when it is in a controlled fashion. In two reported cases, dive-related CO poisoning was initially misdiagnosed as air embolism, one a scuba diver [3] and one a commercial diver breathing surface-supplied air [9]. Fortunately, hyperbaric oxygen is appropriate treatment for both conditions.

In the present case the patient did undergo hyperbaric oxygen therapy when sufficiently stabilized after a CT scan suggested the possibility of cortical infarction, potentially due to bubbles. It was considered unlikely that she had cerebral arterial gas embolism since she was not moving through the water column when her alteration in consciousness occurred and, in fact, cortical infarction was not later seen at autopsy.

There are two ways in which CO may contaminate scuba tanks. First, the compressor air intake hose can be placed too close to the exhaust from a combustion-powered compressor or generator, allowing entrainment of CO. In addition to distance, the intake should be upwind from the exhaust. This was felt to be the problem in one of the scuba cases [2] and a surface-supplied air case [9]. The second way that scuba tanks may become contaminated with CO is production of CO within the compressor itself. Incomplete combustion of lubricating fluid or debris resulting from defective piston washers or rings was found to be the problem in three of the scuba cases [3,4,6]. Note that this can occur even in electric compressors.

The obvious way for divers to avoid this problem is to test the breathing gas in their scuba tanks for the presence of CO prior to each dive. Personal gas testers are available but are not widely used because of expense. They cost in the range of $350-$400 US and have sensors that last 12-24 months. Having tanks filled at a "legitimate" dive shop does not guarantee elimination of risk because of the possible development of a compressor problem, as described repeatedly above. Filling tanks at a source that tests them for CO is a potential solution.

CONCLUSION

Despite the fact that CO poisoning is a recognized risk of scuba diving, it is rarely reported. Some evidence exists to suggest that it may not be considered as a diagnosis or may be misdiagnosed, just as it is in non-divers. Maintaining a high level of suspicion is important in the diagnosis of all forms of CO poisoning.

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Conflict of interest statement

The author has declared that no conflict of interest exists with this submission.

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