The Journal of Emergency Medicine, Vol. 56, No. 3, pp. 332–336, 2019

0736-4679/$ - see front matter

Case Presentations of the Harvard Affiliated Emergency Medicine Residencies

CASE PRESENTATIONS OF THE HARVARD AFFILIATED EMERGENCY MEDICINE RESIDENCIES: WEAKNESS AFTER DIVING

Michael DeLuca, MD,*†‡ Daniel Barkhuff, MD,§ Nicole M. Duggan, MD,*†‡ Emily S. Miller, MD,*‡ Kathleen Wittels, MD,*† and Susan R. Wilcox, MD, FACEP*‡

*Department of Emergency Medicine, Harvard Medical School, Boston, Massachusetts, †Department of Emergency Medicine, Brigham and Women's Hospital, Boston, Massachusetts, ‡Department of Emergency Medicine, Massachusetts General Hospital, Boston, Massachusetts, and §Division of Emergency Medicine, University of Vermont Medical Center, Burlington, Vermont

Reprint Address: Susan R. Wilcox, MD, Department of Emergency Medicine, Massachusetts General Hospital, 55 Fruit Street, Boston, MA 02114

Dr. Daniel Barkhuff: Today's case is a 67-year-old man who presented to the University of Vermont via emergency medical services (EMS) for diffuse pain and weakness immediately after ascension while diving to an approximate depth of 135 feet in Lake Champlain. The patient had been in his usual state of health prior to the dive and was a seasoned scuba diver with decades of experience. He reported a longer-than-expected time spent at maximum depth (12 min instead of the planned 4 min based on dive tables) secondary to an entanglement. He spent a total dive time of approximately 1 h below the surface. Upon beginning his ascent, he observed an initial safety stop at 15–20 feet and then surfaced. Almost immediately he noted a feeling of lightheadedness and weakness in his right arm. He then dove back down to a depth of 15 feet for approximately 10 min prior to surfacing again and calling EMS.

Dr. Emily Miller: What was his initial presentation to the emergency department (ED)?

Dr. Barkhuff: Upon arrival to the ED, the patient was alert, oriented, and protecting his airway. Vital signs were: heart rate 66 beats/min, temperature 36.5°C, blood pressure 134/97 mm Hg, and oxygen saturation 100% on 1 L of supplemental oxygen by nasal cannula. He initially had multiple complaints of pain in his hips bilaterally, pain in his right hand, and right leg "spasm," but had no headache. He complained of weakness and numbness most pronounced in his right leg and in his right upper extremity. On physical examination, cranial nerves 2–12 were intact. There was no facial droop. There was decreased sensation to light touch in the right upper and lower extremities in all peripheral nerve distributions. The strength was initially 4/5 in the bilateral upper and lower extremities, but the strength continued to decline during the ED stay, worsening to 2+/5 in the right upper extremity. The cerebellar function, tested with finger to nose, was intact. Gait testing was not performed. The remainder of the physical examination was unremarkable.

Dr. Kathleen Wittels: After your initial assessment, what diagnoses were you considering?

Dr. Barkhuff: The patient's presentation was concerning for an acute neurological process. Given his history, the most likely pathology was a dive injury, known as decompression illness. Decompression illness includes two diseases: acute arterial gas embolism (AGE) and decompression sickness (DCS). Barotrauma occurs when there is a mismatch between the pressure inside and outside of an air-filled structure. Whereas injuries from barotrauma can occur both when ascending and descending, pulmonary barotrauma, which is the cause of AGE, occurs only with ascent. The most dreaded complication of AGE is neurological injury (1). AGE can result from entry of air bubbles directly into the pulmonary veins, venous gas emboli that are not filtered by the pulmonary capillary system, or via a right-to-left shunt. Once in the arterial system, gas emboli can elicit an inflammatory cascade and cause tissue ischemia, as with any other type of emboli (2,3).

DCS occurs when air bubbles are released by the oversaturation of tissues by nitrogen caused by higher relative pressure at depth. There are mechanical impacts associated with bubble formation leading to vascular and tissue injury. There are likely more significant effects caused by the subsequent inflammatory cascade initiated by the interaction of air bubbles with tissue and blood elements, including leukocytes, endothelium, and platelets (1,3,4). Air bubbles may interact with endothelium, setting off an inflammatory process that contributes to greater vascular permeability (3). These systemic inflammatory responses lead to tissue injury and symptoms of DCS.

DCS is rare, occurring in approximately one of every 5000 dives, and causes tissue injury via bubble development within the venous and arterial systems, through patent foramen ovale, as well as within the affected tissue itself (1,2). Type 1 decompression sickness includes joint and muscle aches and skin manifestations such as pain or itchiness, whereas Type 2 includes more serious manifestations such as central nervous system—often in the spinal cord—and cardiac insults (2). Specific to this case, spinal venous outflow tracks are susceptible to bubble formation in DCS (2,3,5). It is important to note that, as the spinal injury is related to vascular congestion caused by bubbles in the venous outflow and subsequent inflammatory cascade, clinical presentations may not follow an expected dermatomal or anatomic distribution, as in spinal cord injury or cerebral vascular accident (3).

One factor differentiating AGE from DCS is time to onset of symptoms. AGE often presents within minutes due to its pathophysiology of mechanical obstruction of an arterial vessel. With DCS there is typically a latency period, as the inflammatory cascade is an essential contributor to injury (3). There may be overlap in presentation of these two entities, complicating diagnosis.

Also considered were a central nervous system hemorrhage or ischemic stroke, though this was considered less likely. Metabolic and toxicological processes, such as hypo- and hypernatremia, hyperammonemia, and hypoglycemia were considered, however, these were thought to be unlikely given his history and abnormal neurological examination.

Dr. Miller: What was your approach to stabilizing this patient and arranging definitive care?

Dr. Barkhuff: Throughout his ED stay, the patient was complaining of lightheadedness but was awake, alert, and protecting his airway. The vital signs remained normal and he had no evidence of traumatic injury. The decision was made to transfer the patient to a facility with hyperbaric medicine therapy almost immediately, based on the likelihood of this being a dive injury requiring hyperbaric treatment. An immediate search for a hyperbaric chamber was initiated through the Divers Alert Network (DAN). DAN advised that the closest chamber to the patient's location was in Montreal, followed by Portland, Maine, followed by the Massachusetts General Hospital (MGH) in Boston. DAN advised that the first two locations were in use, and recommended transfer to MGH. Emergent air transport was immediately arranged. While awaiting transfer, a noncontrasted computed tomography (CT) scan of the head was performed to look for bleeding or an acute ischemic infarct, as these diagnoses remained on the differential, though the likelihood of these injuries was low. Of note, an air embolism injury to the brain is unlikely to be seen on CT scan (2).

The head CT did not show any acute pathology, and by the time the patient had returned from the CT scan, the transport team had arrived.

Dr. Susan Wilcox: What factors did you consider in transfer and mode of transfer?

Dr. Barkhuff: Ground transport to a hyperbaric chamber would have likely taken approximately 4 h. Air transport was far faster, but also carried the risk of worsening the suspected dive injury as the helicopter is not pressurized, and a gain in elevation could potentially worsen the patient's condition. DCS, one of the dive injuries that the treatment team suspected, occurs because nitrogen is inert, and becomes dissolved in body tissues until saturation, in accordance with Henry's Law. Once the diver is back at normal atmospheric pressure, the dissolved nitrogen will come out of suspension and form bubbles, often in the central nervous system. Air travel is problematic because any gain in elevation will reduce atmospheric pressure and, according to Boyle's Law, gas volumes will increase (1). This reduced pressure, leading to more gas coming out of solution, becomes especially problematic when considering the risks of worsening central nervous system disease.

In this case, DAN recommended transfer as quickly as possible, and the ED team recommended the flight crew to maintain the lowest possible altitude that would be safe during transport. The transport crew agreed to attempt to stay below 1000 feet during their transport, which is generally recommended in unpressurized air transport (3). Per DAN protocols, all patients with suspected decompression sickness or arterial air embolism should get a fluid bolus given possible intravascular hypovolemia from increased vascular permeability, be placed on a nonrebreather oxygen mask and air transported, with the flight crew staying as close to ground level as possible, as DAN recommends 800 feet. DAN does not provide a specific recommendation as to what transport time should be considered to require air transport vs. ground, but the organization does note that the longer the delay, the greater the likelihood of permanent injury. The amount of time after which recompression will not help is unknown. Another recommendation from DAN was to transport the patient in the left lateral decubitus position, as vomiting occurs frequently in DCS patients. Though there are limited data to support their use, antiplatelet agents are often utilized as adjunct treatments in DCS and AGE (6,7). Aspirin may be given while transporting the patient to a hyperbaric-capable facility as a means to reduce the activation of platelets caused by intravascular air (1). A Cochrane Review from 2012 found that treatment with both nonsteroidal antiinflammatory medications and heliox may reduce the number of hyperbaric sessions, but neither improved clinical outcomes (8). Aspirin was not given prior to transfer in this case.

Dr. Wittels: What was the patient's physical examination on arrival to MGH?

Dr. Michael DeLuca: During transport, the patient was started on high-flow oxygen via nonrebreather mask by the critical care transport team as a temporizing measure until he reached the hyperbaric chamber. Treatment with 100% oxygen while transporting to a hyperbaric chamber is critical to create a gradient for oxygen to displace the air and nitrogen bubbles formed in DCS and AGE (1,2). Despite this intervention, the weakness of his right arm progressed during transport. On arrival to MGH he was alert, oriented, and protecting his airway, with a Glasgow Coma Scale score of 15. The heart rate was 56 beats/min, temperature was 36.7°C, blood pressure was 108/58 mm Hg, and oxygen saturation on nonrebreather mask was 99%. On examination there was diffuse weakness in the bilateral arms and legs. There was 2/5 strength in his right arm proximally and decreased grip strength compared with the left. There was 2/5 strength in bilateral leg raise and 3/5 strength in his left upper extremity. Sensation to light touch was slightly diminished over both lower and upper extremities. Cranial nerves 2 through 12 were intact and symmetric.

Dr. DaMarcus Baymon: What were the highest priority interventions upon arrival?

Dr. Nicole Duggan: Upon arrival, the treatment team's principal concern was for a diving-related injury, specifically DCS or AGE. His history of a recent prolonged, relatively deep dive without head or spine trauma, acute onset of progressive bilateral extremity motor and sensory deficits, and normal head CT and chemistries were all consistent with this diagnosis. Given the lack of any symptoms specific to the brain, such as altered mental status, speech difficulties, neglect, or cranial nerve abnormalities, and given bilateral limb involvement, the treatment team strongly suspected a spinal cord lesion. The immediate onset of symptoms was concerning for AGE, however, the progressive, diffuse neurological findings were more consistent with DCS. Regardless, the definitive management of spinal cord injury in the setting of both DCS and AGE is hyperbaric oxygen (HBO) therapy (called "dives").

Evidence suggests that time to initiation of HBO therapy should be minimized in cases of suspected AGE and DCS, where successful recompression within the first 5 min of injury (which our patient tried to achieve by diving back down when he first felt symptoms back at the lake) can lead to significantly improved outcomes, including mortality as low as 5% and very low morbidity, whereas recompression delay of up to 5 h can lead to 10% mortality and 50% morbidity, depending on the severity of injury (9). Although the data also point to an inverse relationship between time to HBO initiation and rates of complete symptom resolution in cases of DCS, recent evidence suggests there may be some benefit of HBO therapy up to several days beyond the initial injury. However, a firm time point beyond which HBO is considered ineffective has yet to be defined (3–5). For our patient, the Hyperbaric Medicine team was consulted immediately upon arrival, and he was transferred directly from the ED to the hyperbaric chamber. His time from injury to initiation of his first HBO dive was approximately 7.5 h.

Dr. Anna Condella: What are potential complications and contraindications of HBO therapy?

Dr. DeLuca: Complications from HBO therapy typically fall into two categories: barotrauma from pressure changes, which can occur in any enclosed body cavity, and complications from oxygen toxicity. Middle ear barotrauma is one of the most common complications of hyperbaric oxygen therapy (10,11). Our patient was unable to tolerate increasing middle ear pressure during his first HBO dive and required bilateral myringotomy. Oxygen toxicity from HBO therapy most commonly manifests as central nervous system affects, which can include symptoms such as seizures, myopia (typically reversible), tinnitus, nausea, facial twitching, and dizziness, however, respiratory complications of acute oxygen toxicity have also been noted (12). It is essential that any pneumothorax be treated prior to initiation of HBO (13). Relative contraindications to HBO therapy include conditions such as obstructive lung pathology, pulmonary blebs, active sinus or ear infections, or decreased seizure threshold given risk of convulsions from oxygen toxicity while in the chamber (11). Fortunately, our patient had no known contraindications.

Dr. Mariam Fofana: What were your considerations for airway protection and hemodynamic stabilization prior to and during HBO dives?

Dr. Duggan: The ability to monitor and resuscitate critically ill patients in an HBO chamber can be limited and vary greatly with the type of chamber. During HBO dives, "air breaks" during which the patient breathes 21% oxygen from a designated source, as opposed to 100% oxygen from the ambient chamber, are often included during the dive. These air breaks reduce the likelihood of neurologic complications from oxygen toxicity (13). Administration of air breaks requires a patient to intermittently position a diver's mask on his or her face and obtain a tight seal on command. If a patient is unable to do this, he or she may need to be intubated for precise oxygen delivery.

At the time of transfer from the ED to the HBO chamber, the patient was hemodynamically stable and protecting his airway. Although initially he was able to hold a mask to his face with his left hand, throughout the course of the first HBO dive, his motor deficits progressed. With 1 h and 30 min left of the first scheduled dive, he was no longer able to reliably position his mask. Throughout the course of this dive, he became increasingly bradycardic, with a heart rate in the low 30s beats/min, as well as hypotensive with a systolic blood pressure in the 70s. At this point the HBO dive was halted prematurely, and the patient was transferred back to the ED for stabilization. Out of concern for neurogenic shock, he was started on norepinephrine, which improved his blood pressure. After extensive discussion with both the patient and family, the decision was made for elective intubation to improve his chances of tolerating subsequent HBO therapy over the next several days. Of note, inflation of any balloons that will be entering the HBO chamber, such as endotracheal tubes or Foley catheters, must be filled with fluid such as sterile water, as liquids are less compressible than gases during high pressure fluctuations. After intubation and stabilization, he was transferred from the ED to the medical intensive care unit (MICU) for ongoing management.

Dr. Farah Dadabhoy: What was the remainder of the patient's hospital course?

Dr. Duggan: Over the course of the next week, the patient underwent a total of five HBO dives. On arrival to the MICU, he continued to have diffuse, profound weakness in all four extremities, and his motor deficits did not improve after two HBO treatments, so Neurology was consulted. Brain, cervical-, and thoracic-spine magnetic resonance imaging studies showed spinal cord infarct with edema involving the cervical cord from C1–C7, as well as an additional focus of restricted diffusion in the central cord at the T11–T12 level, concerning for cord infarct. A repeat noncontrast head CT scan did not show any acute intracranial hemorrhage or infarction. As mentioned above, clinical suspicion was highest for the patient suffering severe neurological injury, likely from spinal DCS or AGE. During his clinical work-up, the treatment team considered the possibility of pulmonary barotrauma leading to AGE from entry of air into the systemic circulation. However, on further evaluation, there was no evidence of lung injury or patent foramen ovale.

Dr. Christopher Nash: What adjunctive therapies for DCS or AGE were considered during his hospital stay?

Dr. DeLuca: Given his nonimproving neurologic status after initial sessions of HBO therapy, adjunctive therapies including heparin, glucocorticoids, and antiplatelet therapy (aspirin) were again considered (1). These therapies have all been trialed in patients with DCS, however, evidence for their efficacy is lacking (1–3). Ultimately, after joint discussions between MICU, DAN, and the Neurology teams, the patient was started on a trial of glucocorticoids given the spinal cord edema seen on magnetic resonance imaging, 81 mg aspirin daily, and prophylactic heparin dosing to protect against venous thromboembolism in the setting of prolonged immobilization secondary to his injury.

Dr. Melanie Molina: What was his final examination on discharge from MGH and ultimate disposition?

Dr. DeLuca: The patient was admitted for a total of 11 days and received five HBO sessions. He was ultimately discharged to a rehabilitation center with a diagnosis of tetraplegia at the level of C4 with neurogenic bowel and bladder. At the time of discharge from rehabilitation facility to a skilled nursing facility approximately 1 month later, with the help of intensive physical therapy, he had achieved moderate recovery of major muscle groups, primarily on his right side.

Dr. Miller: What are the take-home learning points from this case?

Dr. DeLuca: DCS and AGE are two of the most dreaded complications of diving, can lead to severe neurological injury, and should be strongly considered in any patient with neurologic symptoms after a dive. Patients with DCS or AGE should be treated promptly with hyperbaric therapy. The DAN can facilitate locating a hyperbaric chamber, and they provide guidelines recommending a fluid bolus and placing the patient on a nonrebreather oxygen mask prior to initiating hyperbaric treatment. When patients have to be transported, the risks of flight should be weighed against the risks of delaying hyperbaric therapy. Ideally, if a patient must be flown, staying at an altitude of under 800 feet is ideal.

Complications of HBO treatment include barotrauma from pressure changes, with middle ear barotrauma being the most common, and complications from oxygen toxicity. Oxygen toxicity often manifests as central nervous system effects. Although HBO is the definitive treatment for AGE and DCS, the patient may become unstable during the dive, and the ability to monitor and resuscitate critically ill patients in an HBO chamber is limited. Unstable patients should be stabilized prior to being placed in the chamber.

REFERENCES

1. Bove AA. Diving medicine. Am J Respir Crit Care Med 2014;189:1479–86.
2. Hawes J, Massey EW. Neurologic injuries from scuba diving. Neurol Clin 2008;26:297–308.
3. Pollock NW, Buteau D. Updates in decompression illness. Emerg Med Clin North Am 2017;35:301–19.
4. Hadanny A, Fishlev G, Bechor Y, et al. Delayed recompression for decompression sickness: retrospective analysis. PLoS One 2015;10:e0124919.
5. Gempp E, Blatteau J-E. Risk factors and treatment outcome in scuba divers with spinal cord decompression sickness. J Crit Care 2010;25:236–42.
6. Bessereau J, Genotelle N, Brun P-M, et al. Decompression sickness in urban divers in France. Int Marit Health 2012;63:170–3.
7. Bessereau J, Coulange M, Genotelle N, et al. [Aspirin in decompression sickness]. Therapie 2008;63:419–23. [in French].
8. Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness. Cochrane Database Syst Rev 2012;5:CD005277.
9. Byyny RL, Shockley LW. Scuba diving and dysbarism. In: Walls R, Hockberger R, Gausche-Hill M, eds. Rosen's emergency medicine: concepts and clinical practice: Volume 1 & 2. 9th edn. Philadelphia, PA: Elsevier; 2017:1773–86.
10. Bessereau J, Tabah A, Genotelle N, et al. Middle-ear barotrauma after hyperbaric oxygen therapy. Undersea Hyperb Med 2010;37:203–8.
11. Foster JH. Hyperbaric oxygen therapy: contraindications and complications. J Oral Maxillofac Surg 1992;50:1081–6.
12. Warchol JM, Cooper JS, Diesing TS. Hyperbaric oxygen-associated seizure leading to stroke. Diving Hyperb Med 2017;47:260–2.
13. Leach RM, Rees PJ, Wilmshurst P. Hyperbaric oxygen therapy. BMJ 1998;317:1140–3.