Scuba Diving Safety: A Case Report of Diving Injury in the Red Sea

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FIGURE 1 The Red Sea

Case

A 28-year-old, previously healthy woman presented to the ED of Jacobi Medical Center, New York City, with bilateral shoulder and knee pain and shortness of breath. She had arrived at New York's JFK International Airport from Moscow, Russia, 3 days before presentation.

A recreational scuba diver born in the Ukraine and currently residing in New York, she had been training in underwater videography in the Red Sea off the coast of Egypt (Figure 1). Her dive profile consisted of multiple daily dives to 50 meters (approximately 150 feet), with computer-calculated decompression times from her dive instructor.

On Day 5 of her trip, the patient descended to depth with instruction that upon depletion of air, she would ascend via buddy breathing with her instructor. Unfortunately, the instructor was not in proximity of patient when her air depleted, and she experienced a panicked, uncontrolled ascent without any decompression stops.

Within 12 hours of this event, patient noted persistent severe joint pain but continued to dive. She finally sought medical attention on day 10 of her trip. After being informed that there were no emergency hyperbaric facilities on the Egyptian-Israeli border, and of an indefinite suspension of flights between Egypt and Israel, she flew to Moscow for treatment. (The patient was unaware that, in fact, she could have traveled by land to a nearby hyperbaric facility in Eilat, Israel.)

Her symptoms became worse during the flight to Moscow; upon arrival, she learned that her health insurance did not cover treatment abroad. She then contacted the US-based Divers Alert Network (DAN) and was referred to the Jacobi Hyperbaric Center in New York. During the flight to New York City, joint pain persisted, and she noted shortness of breath, confusion, and an unsteady gait.

In the Jacobi ED, the patient was in no acute distress. Vital signs were: temperature, 98.7 F; heart rate, 76 beats/min; blood pressure, 123/74 mm Hg; respiratory rate, 14

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breaths/min. Oxygen saturation was 99% on room air. Physical examination was essentially normal except for a marked left-leaning gait disturbance (patient could only walk by holding onto a wall). The rest of her neurologic, cardiovascular, and musculoskeletal examinations were normal. Although the patient's sensorium appeared intact, her speech subjectively seemed slow and hesitant to both the patient and a friend.

What is the pathophysiology of decompression sickness?

As in this case of uncontrolled ascent, diving injuries result from rapid changes in environmental pressure. Decompression sickness (DCS) occurs when nitrogen bubbles form in body tissues and the vascular system, leading to tissue hypoxia. A review of the gas laws of Boyle, Henry, and Dalton as they apply to scuba diving, helps explain the pathophysiology of DCS (see the box below).

The most important gases involved in DCS are nitrogen and, to a lesser degree, oxygen. In accordance with Henry and Dalton's laws, when one breathes compressed air in a pressurized environment, nitrogen gas dissolves in tissues. During ascent from depth, gas bubble-size increases as per Boyle's law. These changes in pressure lead to obstruction of blood flow and a cascade of inflammatory changes.

To further illustrate the effects of increased pressure and depth, it also helps to understand the units and conversions employed to describe pressure. With respect to pressure, 1 ATA (atmosphere absolute) equals 760 mm Hg of ambient pressure at sea level, which equals 33 feet of seawater ambient pressure. Therefore, there is an increase in pressure of 1 ATA for every 33 feet of depth.[1]

It's the Law: Gas and Pressure

Boyle's law: Pressure and volume are inversely proportional to each other, given a constant temperature.

Dalton's law: The total pressure of a mixture of different gases is the sum of all partial pressures of each individual gas.

Henry's law: Gas in a solution of liquid is proportional to the partial pressure of that gas.

Classically, DCS can be divided into three types. **Type I**, often referred to as "pain-only" bends, is the most common and least severe form of DCS. Patients typically experience pain in the large joints and extremities that is not affected by movement. Pain may be monoarticular or polyarticular, and is believed to be the result of tissue ischemia caused by the mechanical obstructive effects of nitrogen bubbles on joint tissue. In addition to pain, skin changes such as mottling can also occur in type I and are secondary to impaired lymphatic and venous drainage due to obstruction caused by nitrogen bubbles.

Type II DCS, or "serious" DCS, includes pulmonary, cardiovascular, and neurologic effects. Pulmonary DCS, called "the chokes," results from nitrogen bubbles in the pulmonary vasculature, causing chest pain, dyspnea, cough, and even hemoptysis. Severe pulmonary DCS can lead to cardiovascular collapse. Neurologic DCS includes sensory or motor deficits, or both, presenting in a patchy distribution, which reflects the effects of nitrogen-bubble obstruction on peripheral nerves. Patients may also experience vestibular DCS, which can result in vertigo, hearing loss, and gait disturbance. This form can be difficult to distinguish from inner ear barotrauma because symptoms are similar in both; treatment, however, is different for the latter.

Acute gas embolism (sometimes referred to as type III DCS) is a combination of acute arterial gas embolism (AGE) and DCS and is manifested by rapid onset of stroke-like or other neurologic symptoms—usually within 10 minutes after ascent. It typically results from a pneumothorax caused by the rupture of pulmonary alveoli in which intrathoracic nitrogen enters the vascular system either by crossing the pulmonary venous vessels to the arterial side or by passing through a patent foramen ovale ([PFO]; a congenital condition that can permit right to left intracardiac shunting of gas bubbles, increasing the risk of DCS). If these nitrogen bubbles occlude spinal arteries or cerebral vessels, the patient may develop a stroke-like syndrome with motor paralysis or symptoms of spinal cord dysfunction, including urinary or fecal incontinence or retention.[2] If computed tomography (CT) or magnetic resonance imaging of the brain is performed early (usually within 4 hours after injury), mac-

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Courtesy of Jacobi Medical Center, Bronx, New York.

FIGURE 2 The Jacobi multiplace hyperbaric chamber

roscopic air may be visualized; otherwise, imaging may be nondiagnostic. The sensitivity of radiographic imaging for the detection of bubbles is poor, however, and should not delay recompression therapy.

Case continued

Based on the patient's recent diving mishap, DCS was high on the differential. Pulmonary embolism was thought to be unlikely because symptom onset preceded air travel. The patient was placed on a cardiac monitor and given 100% oxygen. A chest radiograph ruled out pneumothorax and barotrauma, and brain CT, likewise, ruled out intracranial injury. Based on these findings, patient was immediately sent to the hyperbaric chamber for treatment of presumptive DCS (Figure 2).

What is the therapeutic benefit of hyperbaric oxygen in decompression sickness"?

The definitive treatment of DCS and AGE is recompression therapy in a hyperbaric chamber. Hyperbaric oxygen decreases the size of nitrogen bubbles, allowing oxygen to diffuse into tissues and displace nitrogen bubbles. This therapy improves oxygen delivery to ischemic tissues and decreases inflammatory neutrophil activity and tissue edema. Regarding treatment protocol, DAN recommends the US Navy Treatment Tables (USN TTs) for DCS.[3] In this case, USN TT 6 was employed, with a total treatment time of approximately 5 hours, beginning at 2.8 ATA with staged decompression (Figure 3).[4]

Is there any benefit to hyperbaric therapy for a patient with a delayed presentation of a diving injury?

The more severe DCS symptoms are at the time of injury, the more likely they are to persist. Although macroscopic nitrogen bubbles reabsorb within 24 hours and hyperbaric oxygen treatment benefits are optimal with early initiation, clinical benefits may be seen in patients who seek care after considerable delay. For example, delayed therapy may still resolve persistent ischemia as well as limit ongoing reperfusion injury. DAN recommends initiating emergent treatment to any diver presenting with signs and symptoms of DCS, regardless of delay, employing the appropriate USN TT.[3] This organization also recommends repetitive treatments until the patient's symptoms resolve or show no further resolution.

What are the complications of hyperbaric oxygen treatment?

Hyperbaric oxygen therapy is generally safe. Common complications include claustrophobia and middle ear barotrauma, which are usually not issues with scuba divers. Although rare, oxygen toxicity seizures can occur, as well as barotrauma of other anatomic structures, including the sinuses, lungs, and gastrointestinal tract. The only absolute contraindication to emergency hyperbaric therapy is untreated pneumothorax. Relative contraindications include pregnancy, epilepsy, and prior inner ear and sinus pathology.

What precautions should scuba divers take to avoid injury?

Novice divers should seek formal training by the Professional Association of Diving Instructors or the National Association of Underwater Instructors. Recreational divers should always dive with experienced and competent supervising divers, and all recreational divers are strongly recommended to use the US Navy decompression tables and not place too much trust in dive computers. This patient was ill-advised to rely on buddy breathing as a means of ascent, and she should have discontinued diving after her initial injury.

DAN is a non-profit organization that not only provides valuable information and education to divers, but also offers affordable supplemental health insurance. All recreational divers are strongly advised to purchase DAN supplemental insurance, which covers emergency transport back to the United States for medical care (in a pressurized aircraft if necessary). This transport is extremely costly (often more than $10,000) and is generally not covered by commercial insurance plans. Another precaution for divers is to confirm access to emergency hyperbaric oxygen treatment when planning an excursion.

Risk factors that contribute to DCS and should be avoided include fatigue, sleep deprivation, alcohol use, repetitive daily diving, and deep dives greater than 100 feet below sea water. Wreck and cave diving entail increased risk. Furthermore, injured patients should avoid flying in a commercial, semi-pressurized aircraft for at least 24 to 48 hours after an incident because exposure to a hypobaric environment may exacerbate symptoms of DCS.

Divers who have been injured despite heeding the above advice should consider evaluation for PFO.

Case conclusion

After initial hyperbaric treatment, patient's symptoms improved sufficiently to permit discharge home. Although gait and speech normalized, she experienced recurrent joint pain and received a total of six daily sessions of hyperbaric treatment the week following discharge, after which symptoms resolved. Because of the severity of her injury, the patient was advised against further scuba diving.

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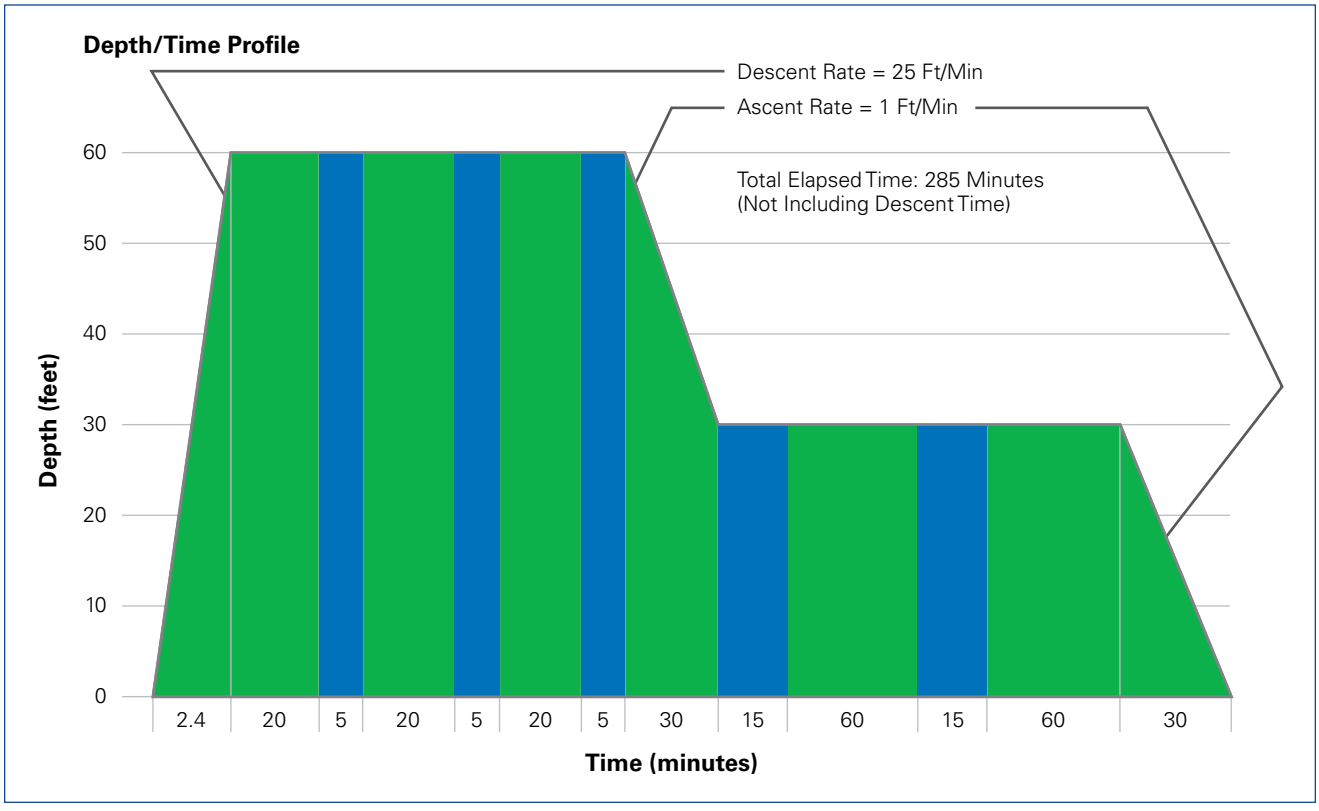

FIGURE 3 US Navy Treatment Table 6