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Neurologic Deep Dive: A Simulation Case of Diagnosing and Treating Decompression Sickness for Emergency Medicine Residents

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Abstract

Introduction: Decompression sickness (DCS) is a rare and dangerous complication from a rapid decrease in environmental pressure, commonly seen in patients leaving a compressed-air environment, such as scuba divers, aviators, and deep tunnel workers. Failure to clinically diagnose and adequately treat DCS with hydration and supplemental oxygen before bridging to hyperbaric oxygen (HBO) therapy can result in permanent residual symptoms or, in rare cases, death. Despite the increasing incidence of DCS, there are limited published simulation case studies discussing this perilous environmental exposure. **Methods:** This fictional simulation case is written for emergency medicine residents to diagnose and manage DCS from a live-actor patient presenting with unilateral neurologic symptoms and concealed cyanotic mottling (cutis marmorata). This case ran for four separate iterations at a simulation center, with a resident, fellow, and attending acting as specific confederates for their respective roles. Following each case, the learners were debriefed at bedside, discussed a PowerPoint presentation, and underwent a question-and-answer session. **Results:** Based on postsimulation qualitative assessments, junior and senior residents correctly identified DCS, but junior residents alone were less likely to elicit pertinent social history or to fully physically examine the patient without the presence of senior residents. Both resident groups were able to verbally explain the fundamental DCS pathophysiology to the patient, but junior residents were unable to specifically direct oxygenation, hydration, and HBO protocols for DCS. After case completion and debriefing, all learners demonstrated achievement of primary learning objectives. **Discussion:** Overall, we noted this case worked well for junior EM residents with senior-resident backup. Both learner groups appreciated the concealed elements of case, including scuba diving history and exposed dermatologic findings, and reported that these were invaluable learning moments for all future patient encounters, not just those limited to DCS.

Keywords

Simulation, Decompression Sickness, Arterial Gas Embolism, Scuba Diving Complication, Hyperbaric Oxygen Therapy

Appendices

A. Simulation Case.docx
B. Decompression Sickness PowerPoint.pptx
C. Lab Values.docx

All appendices are peer reviewed as integral parts of the Original Publication.

Educational Objectives

By the end of the simulation, the learner will be able to:

1. Identify symptoms of decompression illness.
2. Maximize oxygenation therapies in decompression sickness.
3. Recognize arterial gas embolism as a life-threatening sequela of rapid ascension.

Introduction

The ocean, considered one of the Earth's greatest wonders, encompasses over 72% of the planet's surface and hosts thousands of known aquatic species while concealing millions of hidden marine marvels within its 1.35 billion cubic kilometers of salinic mystery.[1] As technology has advanced, previous primitive oceanic exploration methods such as the breath-holding technique have been replaced by the modern device known as the self-contained underwater breathing apparatus, also known as SCUBA or scuba.[2]

The first commercial scuba device was implemented in the 19th century, and there are currently over 9 million recreational scuba divers in the United States alone, with hundreds of thousands of new divers trained and certified annually.[3]

Unfortunately, the increase in scuba popularity has also resulted in increasing scuba diving–related injuries. While there are numerous diving-related hazards, including but not limited to the perilous environment, equipment failure, limited diver experience, gaseous toxicity (i.e., nitrogen narcosis and oxygen toxicity), and barotrauma, decompression illness (DCI) remains as one of the most feared and potentially life-threatening complications, occurring in over 1,000 U.S. scuba divers annually, with fatality nearing 10%.[4,5]

DCI occurs as a result of inadequate diffusion of inert gases when the partial pressure is rapidly reduced (e.g., rapid ascent), in which inert gas bubbles form in the diver's tissues and bloodstream and result in both localized and systemic symptoms based on the volume and location from the downstream hypoxic and inflammatory effects. While DCI can occur to any individual in a compressed-air environment (e.g., scuba divers, aviators, astronauts, caisson workers), common risk factors include rapid ascent, long or deep dives, cold ambient environment, and immediate air travel after scuba dives.[6,7] Passengers onboard commercial airlines are subjected to a greater change in partial pressure as the cabins are pressurized at 8,000 feet, as opposed to sea level, thus placing these passengers at a higher risk of DCI.[8]

DCI was first recognized as *caisson disease* in 1843, during the construction of the Brooklyn Bridge, when the affected tunnel (caisson) workers walked with a slight stoop, later described as the *bends,* after returning from the compressed environment of the caisson to atmospheric pressure.[9] DCI was later characterized into two categories: decompression sickness (DCS) and arterial gas embolism (AGE).

Historically, DCS was separated into Type I and Type II DCS due to the location of air bubbles and the severity of the symptoms. Type I DCS symptoms are often mild and involve the musculoskeletal, cutaneous, and lymphatic system, with symptoms including joint pains, pruritus, marbled or mottled skin around the shoulders, and lymphadenopathy.[10-12] Type II DCS symptoms are more severe and can lead to increased morbidity and mortality as they involve the neurologic and cardiopulmonary system. Concerning neurologic findings include altered mental status, paresthesia or paralysis, and seizures, while concerning cardiopulmonary symptoms include chest pain, shortness of breath, wheezing, and pharyngeal irritation (the chokes).[13-15]

Unlike DCS, AGE symptoms are much more severe and occur acutely. [16-19] AGE can occur due to an excessive *bottom time,* defined as the elapsed time from starting the descent to starting the final ascent to the surface, in which nitrogen oversaturation places the pulmonary parenchyma at a higher risk for tears and injuries.[20] During rapid ascent, the extra-alveolar gas enters the torn pulmonary venous blood vessels from the compromised pulmonary parenchyma, migrates to the left side of the heart, and is distributed systemically as air emboli.[21] In patients with patent foramen ovale, these symptomatic venous gas embolisms, occurring in about 5% of DCS, traverse the heart chambers and produce a fatal AGE, in which as little as 0.5 cc of air can lead to multiorgan failure such as cardiac arrest, strokes, apnea, and death.

Temporary treatment of both DCS and AGE includes IV hydration and oxygenation (via 100% oxygen); hyperbaric oxygen (HBO) therapy is considered the gold-standard therapy for all patients with DCS as delay in hyperbaric therapy, even by 12 hours, can decrease successful outcomes from 75% to 57%.[22] Hyperbaric therapy works by recreating the pressurized environment for the affected patient and accelerates the rate of bubble absorption while providing oxygenation by decreasing plasma nitrogen concentration and increasing the dissolved oxygen content of the arterial blood, respectively; recompression therapy should last for at least 4 hours, until symptoms abate. Additional treatments such as steroids, lidocaine, nonsteroidal anti-inflammatory drugs, and heparin have been used in conjunction with HBO for AGE with unclear efficacy.[23] Due to the rarity of this disease process, health care providers should consider contacting the Divers Alert Network (www.diversalertnetwork.org) for advice on DCI and AGE management, as well as coordinating patient transfers to receiving centers with hyperbaric chambers primarily fitted for wound healing or carbon monoxide exposures as opposed to diving complications.

While DCS is uncommon, the diagnosis of such a potentially life-threatening disease process is unique as it relies primarily on obtaining and synthesizing pertinent history and clinical exam findings. Despite DCS's medical significance, a MedEPORTAL review using the terms *scuba div-, decompression sic-, decompression ill-,* and *arterial gas embolism* did not reveal any relevant simulation cases, educational modules, or resources for the management of DCS. As a result, we feel that our case offers an invaluable learning opportunity to illustrate a systematic review and management of DCS requiring multiple levels of intervention and consultation, while fulfilling various sets of learning milestones per ACGME Emergency Medicine requirements, including (but not limited to) Performance of Focused History and Physical Exam, Diagnostic Studies, Diagnosis, Disposition, Medical Knowledge, Patient-Centered Communication, and Team Management.

Methods

Medical simulation has become an invaluable platform for medical education by allowing learners to be exposed to rare and fatal disease processes as well as practicing certain specialty skill sets in a targeted, simulated learning environment. This simulation case (Appendix A) is based on the fictional scenario of DCS in a patient who presents to the emergency department (ED) with persistent neurological symptoms and concealed dermatologic findings in the setting of air travel immediately after a scuba dive. The simulated case was designed by a panel consisting of three emergency medicine (EM) attendings and one simulation fellow to allow the learners to clinically diagnose an unusual but high-risk disease process, DCS, through a combination of insightful history taking and detailed physical exams, while learning how to communicate amongst team members and with consultants and the patient in a safe, simulated environment. The main goal of the simulation is to encourage learners to perform a systematic approach, emphasizing focused history taking and physical exam, to managing a patient with a neurologic complaint after a unique environmental exposure. A live actor was incorporated into the simulation case to carefully conceal certain revealing aspects of the history or exam findings (Appendix C) in order to provide an additional dimension of realism for the case.

Equipment

A simulation lab can be made to emulate an ED bay with a stretcher, IV pole, monitor, medication, ventilator, audiovisual room, and debriefing room/area. The patient can be prepped by using water-soluble body paint.

Personnel/Roles

The simulation case contains detailed roles for each actor.

1. Nurse: resident/nurse/attending.
2. Patient: resident/nurse/attending—we recommend wearing either a long-sleeved shirt or T-shirt just covering the cyanotic lesions.
3. Hyperbaric chamber consultant (phone): resident/nurse/attending.

Assessment

Learners are assessed based on their active participation in both the case scenario and case debriefing.

Debriefing

The debriefing is separated into three components. At the end of the case, the instructor should request and gather information about the delegation of roles while eliciting a case summary from the team leader. Instructors can also request supplemental case information, participant performance, and, specifically, strengths and opportunities for improvement from the team members or observers (if available). Instructors should review the critical action checklist with questions and an emphasis on the importance of pertinent information gathering and complete body exposure for full physical exams. A PowerPoint presentation (Appendix B) is available to help review DCS. Finally, learners are engaged to participate; they conclude with each offering one piece of a take-home message they have drawn from the simulation case that they intend to apply at work.

Results

Based on qualitative assessment, we found that both junior (PGY 1-2) and senior (PGY 3-4) EM residents were able to identify DCS after eliciting the pertinent histories (air travel and diving history) as well as exposing the patient to reveal the proximal upper-extremity cyanotic mottling. Groups consisting of junior residents required additional prompts from both the patient and the nurse to volunteer additional information (e.g., the nurse questioned the patient regarding his activities during the vacation, or the patient requested the learners to examine his body), while the mixed learner group (junior and senior residents) was able to reach the correct diagnosis without additional prompts from the actors. Both groups initiated supplemental oxygen therapies and contacted a receiving hyperbaric chamber facility for additional management; the junior residents elected to use two liters of nasal cannula, while the mixed group placed the patient on 15 liters of supplemental oxygen via face mask and initiated IV fluid repletion. Neither group elected to pursue additional imaging (radiograph or CT), but the mixed group requested a glucose level prior to making the diagnosis. The junior residents voiced more clinical uncertainty regarding specific transfer criteria for DCS but recognized the urgency due to persistent neurologic symptoms. Prior to patient transfer, both groups were able to correctly explain the basic pathophysiology for DCS to the patient in layperson terms.

During debriefing, open-ended questions, and take-home message summarization, learners demonstrated understanding of the learning objectives: identifying symptoms of DCI, maximizing oxygenation therapies in DCS, and recognizing AGE as a life-threatening sequela of rapid ascension.

Discussion

This resource is based on the fictional scenario for DCS of a young man presenting with unilateral sensation deficit, transient truncal pruritus, and concealed upper extremity cyanotic mottling in the setting of air travel immediately after a scuba dive. The simulation case requires learners to clinically diagnose, manage, and treat DCS in the absence of obvious volunteered history (i.e., recent scuba diving) or supportive radiographic or hematologic studies. Since DCS is both high risk and low incidence (especially in many landlocked states), this makes it an ideal simulation case study for EM residency education. After a thorough review of the MedEdPORTAL database, we did not find any cases or educational modules discussing DCS and created a simulation case to teach learners to identify, manage, and treat DCS and to recognize indications requiring HBO therapy.

After four separate case iterations, we noted that both junior and senior residents were able to identify DCS but that the junior residents had a greater knowledge gap in terms of acute management and transfer indication of DCS. We also noted that junior residents (without senior-resident presence) were less likely to elicit specific social history (aside from alcohol, smoking, and drug abuse) or fully expose the patient without scripted support from the actors. While junior residents demonstrated basic understanding and were able to verbally explain (to the patient) the fundamental pathophysiology of DCS, they struggled with offering specific therapeutic options. However, we noted that when paired with senior residents, the junior residents were more thorough with both history and physical exams and made the clinical diagnosis without additional volunteered information by the actors.

Overall, we noted this case worked well for junior EM residents with senior-resident backup. Both learner groups appreciated the concealed elements of case, including scuba diving history and exposed dermatologic findings, and reported that these were invaluable learning moments for all future patient encounters, not just those limited to DCS. The learners also appreciated brief discussions on other scuba diving complications, such as dysbarism, nitrogen narcosis, and oxygen toxicity. While we acknowledge that barotrauma is also a common form of diving-related injury, leading to potentially fatal complications such as pneumomediastinum, pneumothorax, and AGE, the majority of the learners surveyed after the simulation preferred the PowerPoint presentation to focus primarily on DCIs, while suggesting dedicating a future simulation case to AGE as a complication of barotrauma.

There were only a small number of participants from a single program. However, given the low incidence of scuba diving injuries and absence of an institutional hyperbaric chamber, this case was arguably more relevant and memorable in this particular learner population. The case was also presented as a training session; there were no control groups. Results were observational. It would be reasonable to expect that residents with personal diving experience would identify symptoms of DCI faster than residents without locational or personal exposure to scuba dives. Nonetheless, the active engagement of all participants suggests that elements of the case can apply to all EM residents, regardless of level of training.

As scuba dives become increasingly popular, this simulation case will become more relevant for management of DCS, as well as transport indications and policies for HBO therapy.

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References

1. Oceans & coasts. National Oceanic and Atmospheric Administration Web site. http://www.noaa.gov/oceans-coasts.
2. Brubakk AO, Neuman TS, eds. *Bennett and Elliott's Physiology and Medicine of Diving.* 5th ed. Philadelphia, PA: Saunders; 2003.
3. DeGorordo A, Vallejo-Manzur F, Chanin K, Varon J. Diving emergencies. *Resuscitation*. 2003;59(2):171-180. http://dx.doi.org/10.1016/S0300-9572(03)00236-3
4. Newton HB. Neurologic complications of scuba diving. *Am Fam Physician*. 2001;63(11):2211-2218.
5. Taylor DM, O'Toole KS, Ryan CM. Experienced scuba divers in Australia and the United States suffer considerable injury and morbidity. *Wilderness Environ Med.* 2003;14(2):83-88. http://dx.doi.org/10.1580/1080-6032(2003)014[0083:ESDIAA]2.0.CO;2
6. Barratt DM, Harch PG, Van Meter K. Decompression illness in divers: a review of the literature. *Neurologist*. 2002;8(3):186-202. http://dx.doi.org/10.1097/00127893-200205000-00005
7. Sheffield PJ. Flying after diving guidelines: a review. *Aviat Space Environ Med.* 1990;61(12):1130-1138.
8. Freiberger JJ, Denoble PJ, Pieper CF, et al. The relative risk of decompression sickness during and after air travel following diving. *Aviat Space Environ Med.* 2002;73(10):980-984.
9. Moon RE, Vann RD, Bennett PB. The physiology of decompression illness.*Sci Am*. 1995;273(2):70-77. http://dx.doi.org/10.1038/scientificamerican0895-70
10. Green RD, Leitch DR. Twenty years of treating decompression sickness.*Aviat Space Environ Med.* 1987;58(4):362-366.
11. Rivera JC. Decompression sickness among divers: an analysis of 935 cases. *Mil Med.* 1964;129:314-334.
12. Edmonds C, Thomas RL. Medical aspects of diving—4. *Med J Aust*. 1972;2(24):1367-1370.
13. Erde A, Edmonds C. Decompression sickness: a clinical series. *J Occup Med*. 1975;17(5):324-328.
14. Dick AP, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. *Neurology*. 1976;26(2):190-199.
15. Hallenbeck JM. Cinephotomicrography of dog spinal vessels during cord-damaging decompression sickness. *Neurology*. 1985;35(5):667-671.

16. Gerriets T, Tetzlaff K, Liceni T, et al. Arteriovenous bubbles following cold water sport dives: relation to right-to-left shunting. *Neurology*. 2000;55(11):1741-1743. http://dx.doi.org/10.1212/WNL.55.11.1741
17. Schwerzmann M, Seiler C, Lipp E, et al. Relation between directly detected patent foramen ovale and ischemic brain lesions in sport divers. *Ann Intern Med.* 2001;134(1):21-24. http://dx.doi.org/10.7326/0003-4819-134-1-200101020-00009
18. Gerriets T, Tetzlaff K, Hutzelmann A, et al. Association between right-to-left shunts and brain lesions in sport divers. *Aviat Space Environ Med*. 2003;74(10):1058-1060.
19. Knauth M, Ries S, Pohimann S, et al. Cohort study of multiple brain lesions in sport divers: role of a patent foramen ovale. *BMJ*. 1997;314(7082):701-705. http://dx.doi.org/10.1136/bmj.314.7082.701
20. Tetzlaff K, Bettinghausen E, Reuter M, Heller M, Leplow B. Risk factors for pulmonary barotrauma in divers. *Chest*. 1997;112(3):654-659. http://dx.doi.org/10.1378/chest.112.3.654
21. Leffler CT, White JC. Recompression treatments during the recovery of TWA Flight 800. *Undersea Hyperb Med*. 1997;24(4):301-308.
22. Melamed Y, Sherman D, Wiler-Ravell D, Kerem D. The transportable recompression rescue chamber as an alternative to delayed treatment in serious diving accidents. *Aviat Space Environ Med.* 1981;52(8):480-484.
23. Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness. *Cochrane Database Syst Rev*. 2012;16(5):CD005277. http://dx.doi.org/10.1002/14651858.cd005277.pub3

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