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EM CASE OF THE WEEK

BROWARD HEALTH MEDICAL CENTER DEPARTMENT OF EMERGENCY MEDICINE

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Figure 1. The author diving off the Florida Keys

SCUBA Diving is an increasingly popular recreational sport and a major attraction for tourists to South Florida and the Caribbean. While most injuries that can occur during a dive are relatively benign, decompression syndrome, aka "the bends," can be a life threatening emergency that must be diagnosed and treated urgently. This case discusses the presentation, evaluation, and treatment of DCS.

EM CASE OF THE WEEK

EM Case of the Week is a weekly "pop quiz" for ED staff. The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

The Injured Diver

A 32 year old previously healthy male presents to the ED complaining of recent onset fatigue, joint pain, and a tingling sensation in his right hand. His symptoms started 1 hour ago. No inciting incident or trauma is noted; however, the patient states that he used compressed air (SCUBA) to make several dives for lobster 6 hours ago. In triage, vital signs were as follows: T 98.9, HR 110, RR 14, BP 130/90, O_2 sat 98%. Physical exam is unremarkable except for decreased sensation in the right hand over the radial nerve distribution and diffuse joint pain not exacerbated by movement. Decompression sickness (DCS) is suspected and orders are placed to treat the patient. Which of the following is the correct pathophysiology and treatment of acute DCS?

A. The patient is hypercoaguable. Administer TPA, 100% O_2 @ 4L/min via nasal cannula, place the patient in the Trendelenburg position, and consult vascular surgery.

B. The patient is coagulopathic. Administer 2 units of fresh frozen plasma, give 100% O_2 @ 6L/min via nasal cannula, and transfer to the nearest hyperbaric chamber.

C. The patient has nitrogen gas bubble emboli in the vasculature and tissues. Administer 100% 0_2 @ 12L/min via non-rebreather, administer isotonic fluids, and transfer to the nearest hyperbaric chamber.

D. The patient is myelopathic. Order NSAIDS for pain, give 100% 0_2 @ 12L/min via non-rebreather mask, and consult neurology for EMG/NCS.

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Figure 2.

Take Home Points

- DCS is a serious medical emergency that needs to be treated urgently and vigorously. Long term sequelae or death are probable with treatment delay.
- Symptoms are most commonly present in the first few hours following a dive, but can occur as much as 48 hours after a dive.
- Symptoms are variable and commonly include musculoskeletal pain, skin rash and/or pain, focal neurologic deficits, altered mental status, vertigo, and/or difficulty breathing.
- Initial treatment should consist of high flow 100% 0_2 in a non-rebreather mask, isotonic fluid replacement, and transfer to a hyperbaric chamber. ABC's should always be monitored as patients can rapidly deteriorate.

Decompression Sickness (DCS)

The correct answer is C. Initial treatment for suspected DCS in a stable diver is rapid administration of 100% O_2 at a minimum of 12L/min via a non-rebreather mask, rapid isotonic fluid resuscitation to a urine output of 1-2ml/kg per hour, and urgent transfer to a hyperbaric recompression chamber. The patient evaluation should include a thorough neurological assessment and evaluation for pressure related injuries such as a pneumothorax or ruptured tympanic membrane. No specific pharmacologic intervention or imaging modality has proven effective in the diagnosis or treatment of acute DCS. Detailed information about the dive profile(s) should be recorded.

Discussion:

Decompression sickness is one of the most serious medical complications of sport diving. The condition occurs when diatomic nitrogen (N_2) that has dissolved into plasma and tissues under the increased pressure at depth comes out of solution as the ambient pressure is decreased upon ascent. As the N_2 comes out of solution, it forms gaseous bubble emboli that can cause tissue damage or ischemia. N_2, the major component of atmospheric air and thus a recreational diver's compressed breathing gas, is an inert molecule in human metabolism. Unlike oxygen, N_2 is not utilized by our bodies. This property allows it to follow predicable physical principles of diffusion in response to pressure gradients (Henry's Law). With the human body consisting of ~2/3 fluid, N_2 has a large medium with which to equilibrate as pressure changes with depth.

While the rate of N_2 diffusion can be predicted in vitro, the human body introduces difficult to predict variability of the N_2 diffusion rate and tissue loading. In the diving community, the terms "on gassing" and "off gassing" refer to the accumulation and release of N_2 from the body, respectively. The variable pressure gradient during a dive, duration of pressure exposure, mix of inspired breathing gas, total volume of inspired gas, rate of tissue perfusion, and the rate of tissue cellular metabolism must all be taken into account when creating safe exposure limit tables for human diving. *(cont'd on next page)*

The risk of DCS increases with the depth, duration, and number of dives completed within a given time period. The greater the depth of the dive, the larger the pressure gradient is, which increases the rate of on gassing. On the other hand, increased dive duration allows more time for on gassing. Since not all N_2 is removed from solution during ascent, off gassing, and thus susceptibility to DCS, takes place following a dive for up to 24-48 hours. Therefore, repetitive dives within this time frame increase exposure and tissue loading. Anatomic abnormalities that move blood from venous to arterial circulation, such as a patent foramen ovale (PFO), also increase the risk of developing DCS.

With a well-planned dive according to established tables, a slow rate of ascent, and appropriate stops for decompression, a large and rapid change in the pressure gradient can be avoided, allowing for dissolved N_2 to be off gassed through the lungs. It is not uncommon, however, for divers to develop microbubbles in the venous vasculature upon ascent. These microbubbles are typically expelled by the lungs before reaching arterial circulation. If the bubble formation occurs at a rate greater than can be expelled with respiration or within fixed tissue components, gaseous emboli can form. The location and size of gaseous emboli influence the clinical manifestations. Emboli occurring within cutaneous tissue, joints, or bone typically cause musculoskeletal pain or skin manifestations. Emboli developing in the brain or spinal cord can cause focal neurologic deficits commensurate with embolus location. Emboli occurring in the pulmonary vasculature can cause coughing and difficulty breathing. Emboli developing in the microvasculature can impede microcirculation and cause local ischemia.

Patient symptoms are variable in intensity and presentation. When DCS is suspected, it is important to ascertain, if possible, the dive profile used, including number of dives, depth, duration, breathing mixture used, and any abnormal events such as rapid ascents or missed safety (decompression) stops. In the stable diver, a thorough history and physical with particular attention to the neurological exam is vital for planning treatment.

Treatment.

Initial evaluation in the unstable diver should focus on the ABC's, (Airway, Breathing, and Circulation). In the stable diver, 100% high flow oxygen (≥ 12L/min) via a non-rebreather mask should be administered to decrease the N_2 gradient between inspired gas and blood, thus promoting off gassing at atmospheric pressure. Isotonic fluid replacement increases tissue perfusion and oxygenation while aiding in the resolution of microvascular bubbles. Flow rate should be sufficient enough to maintain urine output at 1-2ml/kg per hour. Management of associated symptoms prior to recompression in a hyperbaric chamber is necessary for patient comfort and to reduce physiologic stress. Treatment of nausea and vomiting associated with vestibular DCS can be aided with ondansetron and diazepam.

Definitive treatment for DCS is hyperbaric oxygen therapy (HBOT) in a recompression chamber (Figure 3). HBOT consists of recompressing the patient to typically three times the atmospheric pressure at sea level (equivalent dive depth of 66ft/20m). The patient will breathe 100% oxygen at this pressure for several hours. This combination of recompression with 100% inspired O_2 aids in reducing bubble size while increasing the diffusion gradient in favor of off gassing N_2. Various recompression algorithms have been developed to treat DCS and can be implemented depending on the severity of symptoms and response to HBOT therapy.

One should consult the Divers Alert Network (DAN) for all suspected dive injuries (Figure 2). DAN, based out of Duke University, is available to assist in dive emergencies 24 hours a day. DAN maintains the most up to date list of available hyperbaric chambers and can arrange specialized air transport to hyperbaric facilities, if necessary.

Figure 3. A hyperbaric chamber

http://www.fpc.qinetiq.com/who-we-are/publishingimages/the-hyperbaric-chamber-large.jpg

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