Research Article

The Gradient Perfusion Model Part 3: An extraordinary case of decompression sickness

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ABSTRACT

Introduction: Decompression sickness (DCS) has been associated with unusual circumstances such as breath-hold diving, shallow depths, and short bottom times. We report a case of DCS with an extraordinary cause and course.

Materials and Methods: A 72-year-old healthy Hispanic female was referred to our 24/7 Hyperbaric Medicine Unit for emergency hyperbaric oxygen recompression treatment (HBO_2 RCT) after developing lower-extremity paralysis following a hyperbaric air exposure in a homemade hyperbaric chamber.

Findings/Case report: After an uneventful exposure to hyperbaric air at a maximum 72-foot depth (3.2 ATA, 32.3 psig), the patient had the delayed onset of abdominal pain and paraplegia after eating a meal. After HBO_2 RCT in accordance with our management algorithm, the patient had a full recovery.

Conclusions: This patient's presentation and course corresponded to what we label as "disordered decompression" and conformed to our Gradient Perfusion Model. With a finite blood volume and the need to perfuse two "intermediate" tissues simultaneously, we postulate that a "steal" syndrome arose to cause the abdominal and paralysis symptoms.

INTRODUCTION

Unique cases of decompression sickness (DCS) that occur under unusual circumstances not typically considered to cause DCS warrant reporting. Such conditions include breath-hold diving, shallow dives and/or brief bottom times. Although our case report involves none of the above, it is extraordinary for other reasons. A notable feature is that this DCS case did not occur as a result of scuba diving. Its seemingly unexplained presentation may be best explained as disordered decompression in accordance with our Gradient Perfusion Model [1,2]. In addition, there are possible legal ramifications as well as the opportunity to offer measures that should be taken to prevent future events of this type.

CASE REPORT

Background and transfer

A healthy 72-year-old Hispanic female with a history of treated breast cancer was transferred by ambulance from her home area about 180 miles from our facility due to suspected DCS/gas embolism. Delayed onset symptoms included lower-extremity paralysis and abdominal pain, which occurred about four hours after an unusual hyperbaric exposure. Although her symptoms partially remitted with breathing surface oxygen, she was transferred to our 24/7 hyperbaric medicine facility for evaluation and management.

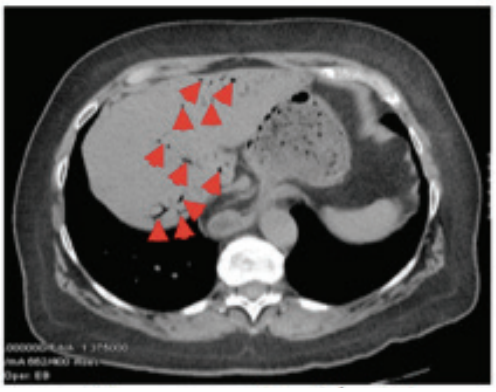
Hyperbaric exposure

The patient decided to undergo a "pep-up" hyperbaric exposure in a personally designed chamber constructed in a family member's garage. The family member learned about hyperbaric chambers from reading about athletes using them to speed recovery from injuries and fatigue.

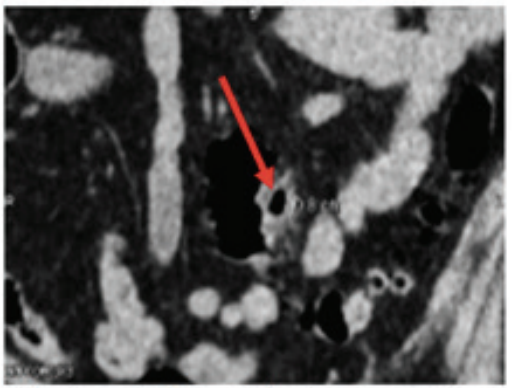
A steel-hulled chamber manufactured in Mexico was fitted to an air compressor. There was no provision for breathing pure oxygen. The chamber, pressurized with air, was being used on friends and neighbors to alleviate aches and pains and for increasing energy levels. The woman underwent an early evening hyperbaric air exposure at an equivalent

KEYWORDS: decompression sickness; hyperbaric chamber; lower-extremity paralysis

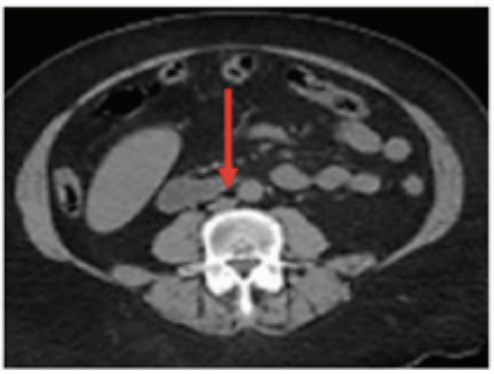
FIGURE 1. COMPUTERIZED TOMOGRAPHY SCANS DEMONSTRATING ABDOMINAL AND PELVIC VEINS



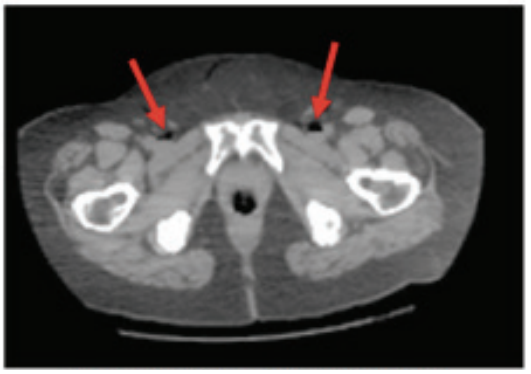
a. Transverse CT with air in portal veins (red arrows)



b. Coronal CT with air in superior mesenteric vein (red arrow)



c. Transverse CT with air-fluid level in inferior vena cava (red arrow)



d. Transverse CT with air in femoral veins (red arrows)

depth of 72 feet of seawater (3.2 ATA, 32.3 psig) for about an hour. An atmosphere pressure gauge was used in lieu of a depth gauge. The descent took 30 minutes because of the small compressor and because the patient had difficulty equilibrating pressures in her middle ear spaces. The bottom time was reported as 30 minutes.

The ascent to the surface was done over a 20-minute period. This was the protocol used for prior treatments of friends and neighbors. The exposure occurred without an incident. The patient said she felt "great" afterward.

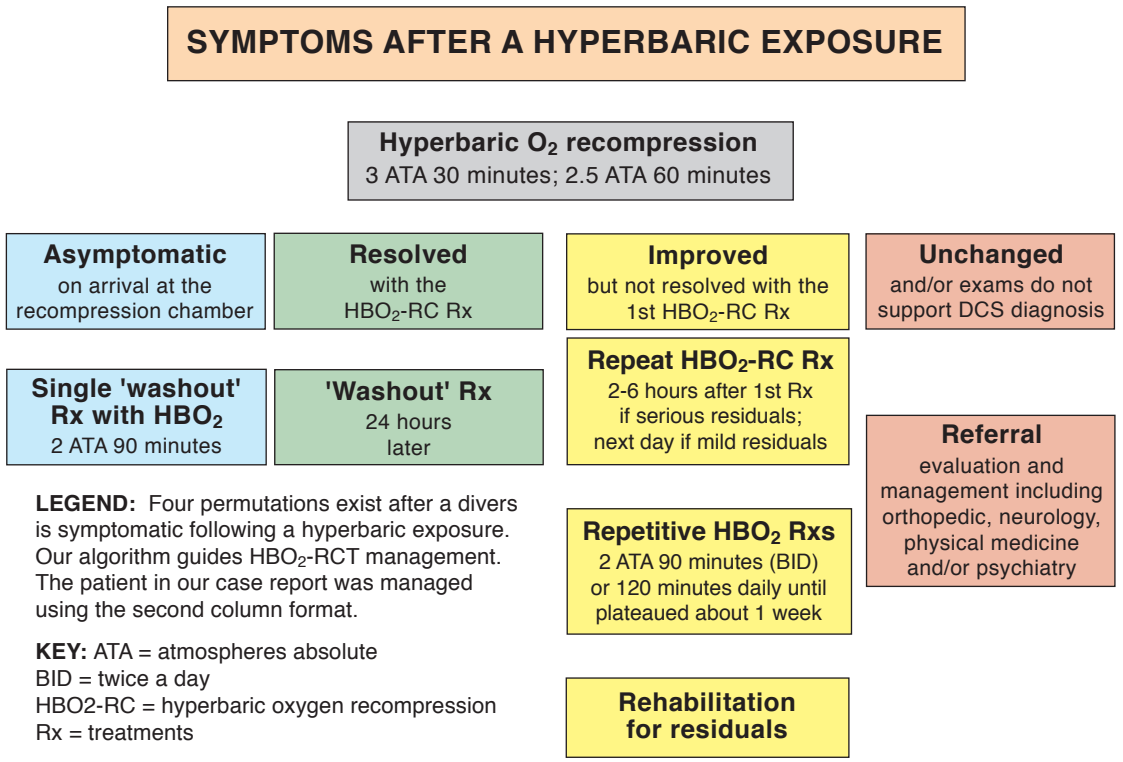
Symptom presentations

After returning home and eating a "hearty" meal, the patient experienced abdominal pain and was unable to move or feel sensation in her lower extremities. The symptoms appeared approximately four hours after the exposure. The Emergency Medical System (911) was activated, and the patient was transferred to a local hospital by ambulance. A computerized tomography (CT) scan of the abdomen and pelvis showed gas in the hepatic portal veins, superior mesenteric vein, inferior vena cava, and femoral veins (Figure 1).

Initial management

Over the next couple of hours of breathing oxygen, the patient's abdominal pain cleared completely. Gradually, sensation and active movement improved to her lower extremities. Because of the potential seriousness of the problems, the patient was transferred by

FIGURE 2. ALGORITHM FOR HYPERBARIC OXYGEN RECOMPRESSION MANAGEMENT OF SUSPECTED DECOMPRESSION SICKNESS



ambulance, a four-hour ride, to our facility, the nearest one available for an emergency hyperbaric oxygen recompression treatment (HBO_2 RCT). She arrived at our facility about 12 hours after finishing the "dive," with mild weakness and decreased lower-extremity sensation.

Our management

Initial screening in our emergency department showed mild dehydration, tachycardia and hypoxemia while breathing room air. Her white blood cell count was minimally elevated. The patient was treated according to our management algorithm using the "residual symptoms after a hyperbaric exposure" protocol (Figure 2). After completing the first HBO_2 RCT, the patient's hypoxemia cleared, and all residual weakness and sensation losses resolved. A "washout" treatment was given the next day according to our protocol, and the patient was discharged. The patient was advised not to undergo "perk-up" hyperbaric pressurizations again.

Follow-up

A check with the patient two weeks later revealed that she had remained asymptomatic and felt well. She told us that she would be pleased to have us share her experience with colleagues.

DISCUSSION

Although at first evaluation the patient's disparate symptoms in two markedly different tissue types would be labeled a case of unexplained DCS, it meets the criteria for disordered decompression. Our Gradient Perfusion Model provides an explanation for the patient's course of events [3,4]. This explanation is based on the findings that two different tissue types – gastrointestinal and spinal cord – were affected. They fit the category of intermediate tissues with regard to perfusion and are under sympathetic nervous system control. After the hyperbaric air exposure and subsequent "hearty" meal, the patient was not able to safely offload the inert gas in these two tissues simultaneously.

With a finite blood volume and possibly impaired cardiac function, steal syndromes occurred in the spinal cord and visceral blood flow. With shared and resultant impaired perfusion, gradients were large enough for autochthonous bubbles to form in the slow-moving venous blood of these structures.

Continuous outflow of a stream from a frozen lake with subfreezing water temperatures is a good analogy to explain the propensity for bubbles to form in slower-moving venous blood. Areas of slow-moving blood flow and/or stasis are notorious for bubble formation, much like the freezing of stagnant lake water. In fast-moving stream water, freezing does not occur. This is analagous to arterial blood flow with microbubbles being carried to the lungs and offloaded. As a consequence of slow flow, bubbles on the venous side can coalesce. With ascent they can enlarge to a point at which they cause symptoms.

Areas of slow-moving blood flow and/or stasis are associated with bubble formation. Examples include the Babson plexus of veins in the spinal cord circulation, decreased arterial inflow as in steal syndromes, impairment of venous return due to external compression, and disordered perfusion sites after injuries. The former two (i.e., Babson plexus and decreased perfusion) are likely to have occurred in this patient's case.

The diagnosis of DCS was confirmed not only by the temporal relationships between the hyperbaric air pressurization plus "hearty" meal consumption but the responses to medical management as well. The initial improvement with breathing pure oxygen and complete resolution with an HBO_2 RCT suggest that bubble formation in the visceral veins and spinal cord minimally exceeded the threshold for symptoms to occur. The CT scan confirmed the presence of gas in the slow-moving veins of the lower abdomen, pelvis and lower extremities. The responses to treatment coupled with the patient's history did not justify obtaining a magnetic resonance imaging study of the spine, although it could have been of academic interest. Of note, a CT scan of the chest did not show evidence of extra-alveolar air syndromes.

We managed the patient according to our DCS algorithm (Figure 2). The lower-extremity weakness and sensory deficits justified the initial HBO_2 RCT, even though the patient's symptoms were improving with oxygen breathing, and it had been more than 12 hours since the patient's exposure to hyperbaric air. The next day washout treatment was given, in accordance with our protocol, to remove any residual nitrogen in slow tissues such as bones, ligaments and other connective tissues. These tissues could possibly have become symptomatic in a delayed fashion with the mild altitude elevation that would occur with the patient's return to her home.

In addition, we sought legal advice as to how we should deal with the family member who constructed the chamber and managed the exposure. Based on this advice the individual was advised to learn about chamber safety, chamber standards, and indications for hyperbaric oxygen therapy. References such as the National Fire Protection Association and the Undersea and Hyperbaric Medicine (UHMS) safety manuals were recommended to the family member. Shortly thereafter, at a chapter UHMS meeting, the family member was in attendance.

CONCLUSION

We report a case of decompression sickness that occurred under unusual circumstances. The patient's history and response to management confirmed the diagnosis. The signs and symptoms conformed to our Gradient Perfusion Model for explaining why the patient became symptomatic. Symptoms occurred in intermediate tissues with respect to ongassing and offgassing, with perfusion highly regulated by the sympathetic nervous system. Advice was provided with the goal of preventing further occurrences of this type.

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Conflict of interest statement

The authors declare that no conflicts of interest exist with this submission.

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