Latent Presentation of Decompression Sickness After Altitude Chamber Training in an Active Duty Flier

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BACKGROUND: Decompression sickness (DCS) is a potential danger and risk for both divers and aircrew alike. DCS is also a potential side effect of altitude (hypobaric) chamber training as well and can present long after training occurs. Literature review shows that altitude chamber induced DCS has approximately a 0.25% incidence.

CASE REPORT: A 32-yr-old, active duty military member developed symptoms of DCS 3 h after his hypobaric chamber training. Unfortunately, he did not seek treatment for DCS until 48 h after the exposure. His initial treatment included ground level oxygen therapy for 30 min at 12 L of oxygen per minute using a nonrebreathing mask. He achieved complete symptom resolution and was returned to duty. However, 12 d after his initial Flight Medicine evaluation, the patient returned complaining of a right temporal headache, multijoint pains, and fatigue. He was treated in the hyperbaric chamber and had complete resolution of symptoms. He was returned to flying status and 5 mo later denied any return of symptoms.

DISCUSSION: Hypobaric chamber familiarity training is a requirement for all military aircrew personnel to allow them assess their ability to identify symptoms of hypoxia. This training method is not only costly to maintain, but it also places aircrew and chamber technicians at risk for potential long-term side effects from failed recompression treatment of DCS. We are presenting a case of recurrent DCS symptoms 12 d after initial ground level oxygen therapy.

KEYWORDS: hypobaric chamber, hyperbaric therapy, dive table, air force.

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Decompression sickness (DCS) is caused by a change in barometric pressure from an area of high pressure to an area of lower pressure and is commonly seen in divers, aircrew in unpressurized aircraft, and with cabin decompression. DCS is also observed in well-controlled hypobaric training environments, i.e., altitude chambers.[2,3,7] Henry's Law states when the partial pressure of a gas over a liquid is decreased, the mass of that gas dissolved in that liquid will also decrease.[4] During decompression this results in an increased production of nitrogen bubbles into various tissues of the body, such as joints, the vascular system, and the spinal cord. Gas emboli in the vascular system can modify the endothelium through adhesion molecule-mediated endothelium activation and stimulate platelet formation.[1,8,10] Platelet aggregation leads to inflammatory pathways and can result in oxidative stress to organs. Mirzaii-Dizgah showed that military personnel who were exposed to hypoxia during altitude chamber exposures had a significant elevation in liver enzyme levels of aspartate aminotransferase and alanine aminotransferase caused by tissue damage.[9]

Risk factors for DCS include altitude exposure, time at altitude, exercise at altitude, dehydration, alcohol, and fatigue.[3,4,15] Based on research by Sheffield, around 75% of DCS symptoms present within the first hour and 90% of cases will present with symptoms within 12 h.[12] Some will present 24 h after the initial incident, but this is more commonly seen in divers that fly the following day.

Historically, DCS classification is separated based on symptomology. Type I DCS includes musculoskeletal symptoms (joint pain), skin manifestations (rash or pruritis), and lymphatic symptoms (edema or lymph node pain). The most common symptom of altitude induced DCS is musculoskeletal

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pain, being reported in 60–83% of cases.[3] Type II DCS affects the neurological system (paresthesia, paralysis, mental status change, and headaches), the inner ear (vertigo, dizziness, nausea), and the cardiopulmonary system (cough, pain with inspiration, increased breathing rate). Type II DCS is considered more concerning, but type I can also be disabling to personnel.

The purpose of hypobaric hypoxia training is to educate flyers on signs and symptoms associated with depressurization. Training exposes flyers to low pressure environments that can cause hypoxia in order to familiarize personnel with the physical and mental stress of depressurization. While in the hypobaric chamber, personnel are preoxygenated with 100% oxygen for 30 min at ground level. By breathing 100% oxygen, this allows for denitrogenation and reduces the risk of developing DCS.[15] Once preoxygenation is complete, the hypobaric chamber is gradually depressurized to 25,000 ft (7620 m), at which point aircrew are instructed to remove their masks to induce hypoxia and complete mental tasks. Training is important because from FY1983–2003, 1055 cases of cabin depressurization in military aircraft were reported. Of these, 876 (83%) were slow depressurization; the remaining 179 (17%) were rapid decompressions, and 350 (33.2%) incidents had adverse health effects and, of those, 83 developed DCS.[7]

CASE REPORT

A 32-yr-old Caucasian man, an Airborne Warning and Control Systems (AWACS) airborne surveillance technician, was performing requalification training for flying duties. Prior to his altitude chamber flight, he was preoxygenated for 30 min on 100% oxygen at ground level. He denied any difficulty with the equipment and believes that there was a complete seal around the mask without any air leaks. During the simulated ascent, he denied any complications. Starting from ground level, the hypobaric chamber was leveled to an altitude pressure of 25,000 ft (7620 m) at a rate of 5000 ft/min (1524 m/min). The descent rates were as follows: 25,000 ft to 18,000 ft (7620 m to 5486 m) at a rate of 5000 ft/min (1524 m/min) and 18,000 ft (5486 m) to ground level at a rate of 2500 ft/min (762 m/min). He was asked to remove his oxygen mask and assess his mental and physical functioning without the oxygen mask at 25,000 ft for no longer than 30 min. He was able to determine the appropriate symptoms of hypoxia in order to place his oxygen mask back on.

The second portion of an initial hypobaric chamber training profile requires rapid decompression simulation. He was moved from the main chamber and placed into a secondary locked chamber where rapid decompression training takes place. The secondary compartment of the hypobaric chamber was locked and leveled to 500 ft (152 m) above field level. The main chamber was pressurized to 4.5 psi and rapid decompression was conducted over a 1–2 s timeframe to an elevation of 12,000 ft (3658 m). He was able to place his mask on in 3 s, turned on his oxygen to 100%, and tolerated recompression over 30 s. He denied having any immediate effects from the rapid decompression, but stated that he just did not feel right. He stayed on oxygen an extra minute and then his hypobaric chamber training was complete.

It was not until 3 h after the hypobaric chamber training that he began to feel pressure behind his right eye. He noted at times having difficulty finding words and slurring his speech. However, he did not disclose these symptoms until he saw the hyperbaric specialist 12 d after his initial encounter at the Flight Medicine clinic. An hour after the initial symptoms of right eye pressure, he began to feel tired and started to notice joint pain in his knees and elbows. Unfortunately, he did not seek medical attention at this time. He went to sleep that night and came to the optometry clinic 48 h after the hypobaric chamber training. During an eye exam, the patient reported still having pressure behind his eye with a dull temporal headache. His pupils were dilated in the optometry clinic, but no significant pathology was noted on exam. The patient was then examined in the Flight Medicine clinic for further evaluation and workup that same day. Neurological exam was completely normal, with his only symptoms being eye pressure and diffuse joint aches, greater at the left elbow. He also complained of fatigue. He made no prior mention of slurred speech or word finding difficulties until he spoke with the hyperbaric specialist 12 d later.

The treating flight surgeon placed the patient on 100% oxygen at 12 $L \cdot min^{-1}$ for 30 min with a nonrebreather mask and the patient was reassessed. The patient reported that his symptoms had resolved completely and he was released to go home with follow-up 48 h or sooner if symptoms returned. Again, this patient did not seek attention following the return of symptoms within 2 h of leaving the flight medicine clinic. The patient later reported that he felt that his symptoms had resolved initially, but 2 h after the ground oxygen therapy, his symptoms returned. The patient decided that he would continue to watch his symptoms and follow up as needed.

The patient returned to the flight surgeon's office 3 d after his initial encounter at the flight medicine clinic to return to fly status. During his appointment, he denied any joint pain, slurred speech, fatigue, mental fogginess, or neurological symptoms within the previous 48 h. His physical exam did not show any abnormalities and, at this time, the Flight Surgeon felt that he could safely return the patient back to flying status.

The patient presented back to the clinic with a return of symptoms 14 d after his hypobaric chamber exposure and 12 d after his initial ground level oxygen treatment. He reported increased fatigue, a constant headache on the right side of his face, a dull ache in all of his joints (the left elbow being the worst), and had developed a diffuse pruritic rash on torso and arms.

We contacted the San Antonio hyperbaric facility and a local hyperbaric specialist to consult on this case because we felt that it was difficult to believe that the patient still showed signs of decompression sickness. The conclusion from all parties was that the safest course of action was to recompress the patient and reevaluate. During his examination with the local hyperbaric specialist, the patient admitted to previously having difficulty finding words, extreme fatigue, and joint pain. On physical exam, it was noted that the patient had 4/5 left arm muscle strength and a papular rash on his anterior torso. Our patient

did not have a history of an untreated pneumothorax, which is the only absolute contraindication to hyperbaric treatment. An MRI was not obtained at this time because the hyperbaric specialist felt it was more important to treat. The hyperbaric specialist conducted a 2-h dive and the patient again stated that all symptoms were completely resolved. After the dive treatment, he denied joint pain, fatigue, and headache.

An MRI was obtained after the hyperbaric treatment and it showed that the patient had a bilateral mucus retention cyst in his maxillary sinus. Mucous retention cysts can cause pain and discomfort during hyperbaric treatment. If mucous retention cysts are noted on an MRI prior to hyperbaric treatment, the patient can be given decongestants and slowly recompressed while being monitored by the provider for increased sinus pain. The patient denied any history of sinus pressure. The patient was admitted overnight for observation. If his symptoms returned, a treatment table 6 series would be completed the next day for an additional 5 h of treatment. He was released the next morning, asymptomatic, and followed up with the local flight surgeon.

A week after his treatment, he stated that his rash was gone, he denied all joint pains, he was no longer feeling fatigued, and his headache had not returned. The first day after the hyperbaric treatment, he was capable of playing a full game of basketball, and was able to return to his complete workout routine. He has since been returned to flying status without complication for 5 mo.

DISCUSSION

Hypobaric training is a requirement for all military personnel that are on active flying status. It is intended to expose personnel to scenarios of hypoxia and to identify individual symptoms of hypoxia as well as train for quick don procedures for rapid decompression. Unfortunately, this training is not without risk of DCS and potential long-term symptoms from failed recompression treatment of DCS. This brings into question the utility of hypobaric training for aircrew using an altitude chamber for requalification training. For years, some military installations have been approved to use reduced oxygen breathing devices (ROBD) to retest/recertify for hypoxia training. This technology decreases the cost needed to maintain an altitude chamber, decreases the funds needed to transport personnel to a chamber facility, and decreases medical cost associated with potential adverse side effects of an altitude chamber. ROBD can be used in combination with a more realistic simulation of flying and so demonstrate how hypoxia can inhibit or affect cognitive function in a more relevant environment than the hypobaric chamber. Most importantly, it prevents long-term side effects associated with failed recompression treatment of DCS and arterial gas embolism. A case report by Allan reported 3 of 17 personnel in the same hypobaric chamber, who were requalifying for altitude chamber training, had significant symptoms of DCS.[2] All three of the personnel flew home within 6 h of their hypobaric chamber training, they all required hyperbaric therapy, and one had residual symptoms requiring additional treatment.[2] This could have been prevented with the use of ROBD for requalification. A review of 133 patients by Wiriosemito showed that only 3–5% of DCS cases were caused by changes in pressurization while flying aircraft; the remaining were secondary to altitude chamber training.[16] These two articles are examples that there is an increased risk of DCS associated with hypobaric chamber training.

Based on DCS treatment guidelines, persons suspected of having DCS should be treated within 1 h of presentation. While awaiting consultation to hyperbarics, the patient can be started on 100% oxygen therapy with a nonrebreather mask, given fluids regardless of hydration status, and placed in a supine position.[14] For type I symptoms, ground level oxygen therapy can be administered for 2 h minimum, with a maximum of 3 h of treatment. If symptoms persist after 30–60 min, or if joint pain symptoms return after initial ground level oxygen treatment, the individual should be transferred to a recompression facility and treated on the appropriate treatment table.[14] During transportation, individuals should be kept on 100% oxygen.

The goal of hyperbaric oxygen treatment is to decrease the volume of air bubbles and provide oxygenated blood to areas of hypoxia. During management of DCS, hyperbaric oxygen should be initiated as early as possible even if there is a delay in presentation of DCS symptoms. A 2014 retrospective study compared 76 divers with delayed treatment of DCS greater than 48 h to 128 divers with DCS treated within 48 h after surfacing. Respectively, 76% and 78% had a full recovery, whereas 17.1% and 15.6% had a partial recovery, and 6.6% and 6.2% had no recovery after treatment.[8] In order to add value for late recompression, civilian divers diagnosed with DCS who underwent recompression therapy after 48 h of symptoms showed no difference in recovery compared to those treated before 48 h.[8] The percentage of those that had a partial recovery or no recovery did not show a statistical difference compared to patients who had early treatment.[8] Even if there is a delay in recompression therapy, it has been shown that the majority of patients will still get some symptom resolution, whether it is complete or partial resolution.[1,8] Improvement in symptoms have been noted in patients who have had delay times of up to 1 wk, and a long delay should not preclude follow-up treatments.[8] Once residual symptoms respond to additional recompression treatments, such treatments should be continued until no further benefit is noted.[14] An article from Workman in 1968 showed that out of 150 patients treated for DCS, 23 (15%) of those needed additional therapy. Of those re-treated, seven (30%) had complete symptom relief and nine (39%) had substantial relief, while the other seven (30%) had residual symptoms.[17] In Cianci's review article, 110 patient charts were screened from 1983–2002, with the average delay from initial symptoms to treatment being 93.5 h.[5] Even with a lengthy delay, they showed a 98% recovery rate for the entire group.[5] Although the average amount of treatments were 1.96 for 97 of the patients, the final 13 patients required around 10 treatments each for symptom resolution.[5] One case of optic neuropathy was observed in a patient 3 wk after exposure to an altitude chamber. On initial treatment, the

patient had resolution of symptoms, but symptoms reoccurred. After four treatments with hyperbaric therapy, the patient had complete resolution of symptoms.[13] Altitude DCS is not a common occurrence; because of this, there are limited studies directed toward the long-term effects of DCS from hypobaric chambers. However, residual symptoms have been documented in the diving community. In Xu's study of 5278 divers diagnosed with DCS, only 63.8% of the severe cases had complete recovery at the time of discharge. There were 11 cases of neurological sequelae, 9 spinal cord injuries, and 2 peripheral nerve paranesthesias.[18] Evidence is incomplete in the area of long-term side effects of altitude chamber induced DCS from failed treatment with hyperbaric therapy. We recommend further follow-up with patients diagnosed with DCS to better understand the long-term side effects of hypoxia chamber training.

When there is a clinical suspicion of DCS, it is better to err on the side of caution and treat conservatively with oxygen therapy and potentially hyperbaric treatment if symptoms persist. This was a difficult case given the time of presentation from the initial hypobaric chamber exposure and the hesitancy of the patient to honestly describe symptoms, resulting in the continuation of symptoms after 2 wk from the initial insult. In a 1988 study by the Naval Diving Unit, it was found that neurological symptoms can initially resolve following therapy and then reoccur following treatment.[6] Even when all central nervous system symptoms were relieved by recompression, some patients had symptoms return from a few hours to days after hyperbaric treatment.[6] If symptoms return, it is not uncommon to perform multiple table 6 dives for further treatment. Unfortunately, we must rely solely on the patient's subjective symptoms to make treatment decisions, making communication and education of utmost importance.

Unfortunately, there is stigma about personnel that are affected by DCS and whether or not crewmembers can be returned to flying. Because of this, personnel tend to wait to seek treatment. In regards to aeromedical decision making after DCS, per the U.S. Air Force medical standards directory, 2 February 2016, if symptoms of neurological DCS from an altitude chamber resolve within 14 d, no waiver is needed to return a patient to flying status. If neurological DCS occurs while flying an aircraft, a waiver will be required. Because of these changes to waiver protocol, it opens up communication from aircrew to seek medical attention earlier.

In conclusion, it is important to remember that as healthcare providers, we need to have a high index of suspicion and low threshold to treat DCS regardless of the time of presentation. It may also be pertinent to invest in safer means of exposing patients to hypoxic training environments via ROBD. Finally, patient education is imperative so they will seek early treatment for DCS and be guided to a faster recovery of symptoms.

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