Triage and Treatment of Mass Casualty Decompression Sickness After Depressurization at 6400 m

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BACKGROUND: Decompression sickness is a condition that results from an abrupt change from a higher to a lower pressure. It is described most commonly in divers; however, it can occur in aviation incidents, which this case report will discuss.

CASE REPORT: Following an acute cabin depressurization incident, 36 patients presented to a small outpatient clinic with multiple symptoms, including fatigue, headache, nausea, vomiting, and dizziness. These patients were evaluated, triaged, and some were able to be successfully treated with supplemental oxygen in clinic. Eight of the patients had symptoms that were either persistent or concerning enough that they were referred to the dive medical clinic, where the dive medical team diagnosed six of the patients with Type II decompression sickness and referred them for hyperbaric oxygen chamber therapy. All patients who received hyperbaric therapy experienced at least some relief of symptoms, with most reporting some residual fatigue after the therapy.

DISCUSSION: This case provided both lessons in triage and management of multiple patients in a small outpatient clinic, as well as the challenges in making the diagnosis of decompression sickness.

KEYWORDS: altitude, arterial gas embolism, cabin pressure, hyperbaric, decompression illness.

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Decompression sickness (DCS) encompasses both decompression illness and arterial gas embolisms that result from gas bubble formation in the setting of abrupt change from higher to lower atmospheric pressures. DCS can be further categorized into two types based on general clinical manifestations, but this classification is not strictly applied in clinical diagnosis and treatment. Symptoms of DCS can range from mild, including joint pain and pruritus, to severe, including stroke and pulmonary embolism. DCS is typically identified after dive and flight activities, more commonly associated with diving due to the nature of participants being exposed to steeper gradients of atmospheric pressure as well as other factors contributing to the solubility of inert gases.[7] The symptoms are sequelae of the formation of bubbles of inert gas, namely nitrogen, within the body due to decreased solubility at lower atmospheric pressures as described in Boyle's law.[4]

For the purposes of this case, there were no patients who developed arterial gas embolisms and it will focus on aviation-induced DCS. The mechanism of DCS development in flight occurs typically during the beginning of the exercise in the initial ascent or a cabin depressurization event. This contrasts with diving, during which the potential for DCS development occurs at the end of the activity with ascension. There have been case reports demonstrating these mechanisms in aviation concerning pilots operating high altitude aircraft with rapid ascent or multiple flights in a day leading to the development of DCS.[1,3,6] Another case report of aviation-related DCS involved several patients in a military flight that lost cabin pressure while at cruising altitude.[5] Aircraft are typically pressurized to 2000 m (6500 ft) altitude and sudden loss of pressurization exposes passengers to the ambient pressure of the aircraft's cruising altitude, typically two to threefold higher elevation. In this case the cabin was pressurized at approximately 1500 m (4921.3 ft), roughly 0.83 atm, and the depressurization occurred at 6400 m (20,997.4 ft) elevation, which corresponds to 0.46 atm.

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The evaluation for a patient with concern for DCS should involve a history with corresponding mechanism such as recent dive, flight with rapid ascension or sudden loss of cabin pressure, noncommercial air travel, altitude chamber use, or a combination of these. Thorough ear, nose, throat, skin, neurological, musculoskeletal, cardiopulmonary, and mental status examinations are essential in the evaluation of a possible DCS patient. Examination findings may include altered mentation, weakness, ataxia, pruritic rash, wheezing, and others. As there is no diagnostic testing to confirm DCS, workup for differential diagnoses such as hypoxemia, myocardial ischemia, and cerebral ischemia may be necessary to rule out these life-threatening pathologies that DCS presentations can mimic.

The treatment for DCS is hyper-oxygenation to facilitate diffusion of nitrogen out of the patient's blood into the air spaces of the lungs, where it can be expelled. Patients with mild symptoms and a concern for DCS should receive 100% supplemental oxygen for 2 h. Patients with more severe presentations, as well as less severe cases that do not resolve with 100% supplemental oxygen, should be referred for hyperbaric oxygen therapy.[5] The hyperbaric chamber uses the same principle with the added effect of increased pressure to deliver 100% oxygen and maximize the gradient.[2]

Due to rapid blood flow through certain organs such as the lungs, heart, and brain, typically the symptoms associated with these are the first to resolve with therapy.[2,7] The symptoms that take longer to resolve are generally those associated with tissues that have poorer perfusion, such as cartilage and joints. Adipose can absorb much higher amounts of nitrogen and symptoms may persist longer or presentation may be delayed, depending on the body fat composition of the patient.[7]

CASE REPORT

There were 49 military service members who were passengers aboard a military aircraft at 6400 m (21,000 ft) above sea level when the aircraft experienced a rapid cabin depressurization, previously controlled at approximately 1500 to 1800 m (5000–6000 ft). The aircraft then descended to 3000 m (10,000 ft) within 5 min following the rapid depressurization. The aircraft was not outfitted to supply the passengers with oxygen and only the flight crew received supplemental oxygen during the incident. The aircraft then turned back to the nearest base, which was the base from which they had departed. Upon landing, they were briefly evaluated at the flight line by crash-fire responders and an enlisted medical technician who was also a passenger on the flight. The initial medical evaluation was a blood pressure and $S_{p}O_2$, which was normal at that time for all the passengers. All 49 military service members were sent home to be re-evaluated the next day because they all were thought to be doing well and were fatigued from a 14-h day. They were provided emergency room precautions for headaches, joint pains, nausea, or otherwise feeling ill (see **Fig. 1** for graphical representation of timeline of events).

The 49 passengers were all men, age range 18–43 yr old, in good overall health with no comorbid conditions. Several passengers experienced symptoms both immediately at the time of depressurization as well as after the incident. The passengers reported seeing a dense fog form in the cabin, some describing it as if it was snowing, and that the temperature dropped to intolerable levels. Passengers reported experiencing narrowed field of vision and there was a mix in reports on timelines and perceptions of where they were in the flight, as if some people became confused. One passenger, a 43-yr-old man, had a witnessed brief syncopal event with some extremity convulsions in flight during

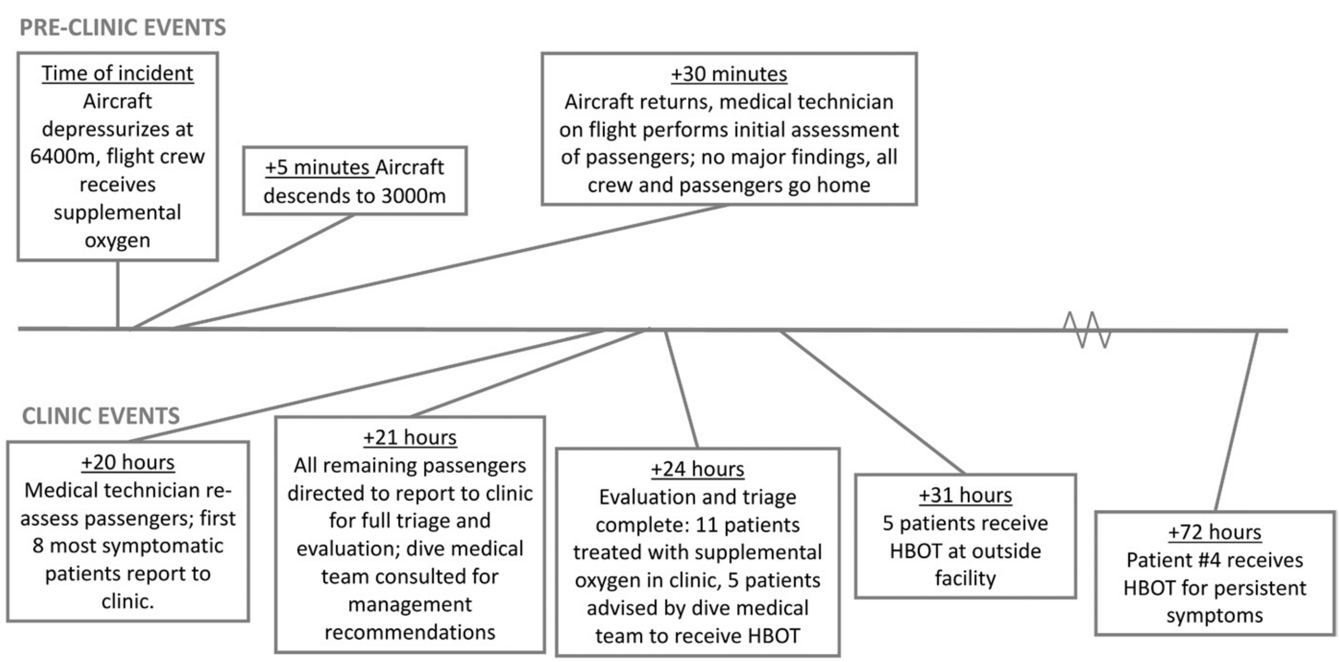

Fig. 1. Timeline of events.

the descent and another passenger, a 20-yr-old man, experienced sudden sharp right knee pain, which self-resolved 4 h after the incident. The flight crew had access to oxygen during the incident and did not report any symptoms during or after the flight.

The following morning, any individuals who were experiencing symptoms were asked to come to the military medical clinic to be evaluated. There was a steady flow of patients with symptoms and one that had also reported to the emergency room for nausea, vomiting, and a headache overnight. All 49 individuals were then asked to come to the clinic to be triaged, and 36 out of the 49 passengers were reporting symptoms that had persisted since the incident (see **Table I**). Eight passengers had more severe symptoms and/or exam findings and thus were evaluated first (see **Table II**).

Of the patients with more severe presentations, two were found to have perforated tympanic membranes. A total of 11 patients, including 7 of the most severely symptomatic patients, received 2 h of 4 L · min^{-1} supplemental oxygen via simple face mask in clinic. Four of the patients who received oxygen in clinic had complete resolution of their symptoms, were discharged from the clinic, and were instructed to return to the clinic or emergency department if they developed any symptoms again. This same day, the patients with severe symptoms who did not improve with oxygen were presented to the local dive medical officer on base. The dive medical team performed an evaluation, including a detailed neurological exam, and recommended hyperbaric chamber therapy for five of the patients that same day. Another patient was followed closely for 3 d with continued mild symptoms, which the dive medical team ultimately elected to treat with hyperbaric therapy as well. The time from clinical presentation in the outpatient clinic to the hyperbaric chamber was several hours, as it involved the medical evaluations and initial treatments as well as arranging the hyperbaric treatment and getting to the hyperbaric chamber, which was approximately a 1-h drive away. The hyperbaric chamber therapy involved treatment table 6 with one extension for the initial five patients, and treatment table 6 for the patient who was sent 3 d later. All patients who received hyperbaric therapy experienced at least some relief of symptoms and most reported residual fatigue after the therapy.

The dive medical officer recommended follow-up with Otolaryngology and Neurology for all six patients who received hyperbaric therapy. Of the six patients, patients, 1, 2, 6, and 7 followed up with the specialists; the rest were lost to follow-up. Neurology ordered a brain MRI and MRA on patient 2 for continued mild headache after the incident to rule out barotrauma or vascular trauma, and a brain MRI on patient 7 for a mild abnormality noted on tandem gait. Patient 1's imaging came back with nonspecific white matter changes which were determined to not be clinically significant and likely related to tobacco use. Patient 7 was lost to follow-up and the MRI was never obtained. After a thorough evaluation by an Otolaryngologist for the referred patients, nothing significant was found and there were no additional recommendations.

Table I. Symptoms Persisting in 36 Passengers.

SYMPTOMS	# OF PATIENTS	% OF TOTAL PASSENGERS
Fatigue	9	18.36
Headache	23	46.93
Nausea	5	10.20
Vomiting	2	4.08
Dizziness	6	12.24
Lack of concentration/confusion	2	4.08
Extremity paresthesias	1	2.04
Chest pain	2	4.08

Table II. Severe Symptoms in 8 Passengers.

PASSENGER	SYMPTOMS
Patient 1	headache, vomiting, lightheadedness
Patient 2	headache, nausea, confusion
Patient 3	knee and back pain, resolved 4 h after incident
Patient 4	headache, fatigue
Patient 5	nausea, vomiting, extremity paresthesias, headache, fatigue
Patient 6	fatigue, dizziness, confusion
Patient 7	headache, dizziness
Patient 8	chest pain

DISCUSSION

This case presented several clinical learning points regarding making the diagnoses and resource management with the large number of patients. As previously mentioned, the presentation of DCS can overlap with several other plausible diagnoses, including inner and middle ear barotrauma, cerebrovascular accident, and hypoxemia. A quick method for triaging such a large ambulatory group was developed on the spot, which included drafting up a DCS symptomatology form given to all patients. Any patients who answered yes to any of the symptoms were triaged up. In addition, patients who had difficulty filling the form out, which indicated possible cognitive deficits, were triaged up. There were several patients with obvious signs of barotrauma and patients with more severe symptoms who later received neuroimaging to evaluate for cerebrovascular insult. While these patients with more severe symptoms had no pathological findings on imaging, they likely had superimposed barotrauma of the inner vs. middle ear in the setting of DCS.

The number of patients and their presentation to a small outpatient clinic with limited available staff and resources also presented unique challenges in appropriately responding to and managing these patients. By definition it was a mass casualty scenario; thus, while all patients were effectively triaged and evaluated, we were not able to fully treat all symptomatic patients. The eight patients with more severe presentation received appropriate referral to higher care and treatment as stated previously. There was insufficient supplemental oxygen in the clinic for the remaining 26 symptomatic patients to all receive the recommended 2 h of oxygen.

There are several factors that were outside of the control of the evaluating clinic which are likely to have been very impactful in the overall clinical course. It is unclear how likely that these patients would have benefited from earlier, more thorough evaluation immediately after the incident as opposed to 24 h after, when they presented to clinic. It is also unclear how likely in-flight oxygen for the passengers would have affected the number of patients and severity of symptoms. The immediate adjustment of lowering altitude by the flight crew was likely beneficial for mitigating patients' symptoms while in flight. In general, this is recommended for any patients with onboard medical emergencies suspected of having a component of DCS or respiratory distress.

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