High-Altitude Decompression Sickness Treated with Hyperbaric Therapy and Extracorporeal Oxygenation

Jacek Siewiera; Przemysław Szałański; Dariusz Tomaszewski; Jacek Kot

BACKGROUND: High-altitude decompression sickness (HADCS) is a rare condition that has been associated with aircraft accidents. To the best of our knowledge, the present paper is the first case report of a patient treated for severe HADCS using recompression therapy and veno-venous extracorporeal oxygenation (VV-ECMO) with a complete recovery.

CASE REPORT: After depressurization of a cabin, the 51-yr-old jet pilot was admitted to the Military Institute of Medicine with a life-threatening HADCS approximately 6 h after landing from a high-altitude flight, in a dynamically deteriorating condition, with progressing dyspnea and edema, reporting increasing limb paresthesia, fluctuating consciousness, and right-sided paresis. Hyperbaric oxygen therapy in the intensive care mode was initiated. A therapeutic recompression with U.S. Navy Treatment Table 6 was performed with neurological improvement. Due to cardiovascular collapse, sedation, mechanical ventilation, and significant doses of catecholamines were started, followed by continuous veno-venous hemodialysis. In the face of disturbances in oxygenation, during the second day of treatment the patient was commenced on veno-venous extracorporeal oxygenation. Over the next 6 d, the patient's condition slowly improved. On day 7, VV-ECMO was discontinued. On day 19, the patient was discharged with no neurological deficits.

DISCUSSION: We observed two distinct stages during the acute phase of the disease. During the first stage, signs of hypoperfusion, neurological symptoms, and marbled skin were observed. During the second stage, multiple organ dysfunction dominated, including heart failure, pulmonary edema, acute kidney injury, and fluid overload, all of which can be attributed to extensive endothelial damage.

KEYWORDS: HBOT, hyperbaric oxygenation, high-altitude decompression sickness, extracorporeal oxygenation, decompression sickness.

Siewiera J, Szałański P, Tomaszewski D, Kot J. *High-altitude decompression sickness treated with hyperbaric therapy and extracorporeal oxygenation*. Aerosp Med Hum Perform. 2020; 91(2):106–109.

High-altitude decompression sickness (HADCS) is a relatively rare condition that has been associated with aircraft accidents and plane crashes for more than 50 yr.[3] There is a lack of information regarding the etiology of HADCS in scientific databases, and the disease may present with a wide range of symptoms and variable clinical course. The prognosis depends on HADCS severity.[9] The pathophysiology is the same as decompression sickness types I and II in divers; however, in HADCS, a decrease in atmospheric pressure below 1 ATA induces concomitant hypoxia, which aggravates the symptoms. Previously one case of successful, hyperbaric treatment and extracorporeal oxygenation in the case of a diver was described.[7] In the current literature, case reports regarding the management of severe decompression sickness caused by a fall in pressure below 1 ATA are relatively rare, and usually refer to hypobaric training,[13] accidental aircraft decompression, or extreme pressure fluctuations during flight.[1,6] Regardless of relatively limited pressure differences in the range up to 1 ATA, cases of severe DCS occurring with symptoms of gas embolism previously have been reported.[14] The present paper reports a case of severe life-threatening HADCS in a 51-yr-old jet pilot. Treatment included hyperbaric oxygenation (HBO) and extracorporeal membrane oxygenation, which resulted in

From the Departments of Hyperbaric Medicine, Cardiosurgery, and Anesthesiology and Intensive Care, Military Institute of Medicine, Warsaw, Poland; and the National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine, Medical University of Gdansk, Gdynia, Poland.

This manuscript was received for review in June 2019. It was accepted for publication in November 2019.

Address correspondence to: Jacek Siewiera, Ph.D., Military Institute of Medicine, Szserów 128, Warsaw, Mazowieckie 04-141, Poland; jacek.siewiera@gmail.com.

DOI: https://doi.org/10.3357/AMHP.5457.2020

total resolution of HADCS symptoms with no persistent organ damage or neurological deficits.

CASE REPORT

A 51-yr-old male jet pilot with a previous history of arterial hypertension and overweight was admitted to the Emergency Department of the Military Institute of Medicine in Warsaw 6 h after an emergency landing. During the flight, after dynamic ascent and depressurization of the cabin, the pilot began to experience dyspnea at an altitude of 28,000 ft (8534 m), followed by altered consciousness, sleepiness, tunnel vision, and intense weakness in the upper extremities.

Immediately after landing, the patient was breathing unassisted with 100% oxygen via a face mask. The patient was transferred from a district hospital to the ICU (Military Institute of Medicine) in a rapidly deteriorating condition, complaining of severe dyspnea, dizziness, numbness in the legs, and increasing right-side upper limb paresis and facial nerve palsy. Marbled skin symptom over the whole body was observed, especially on the limbs and back. Vital signs were S_pO_2 89%, HR 120 bpm, and BP 117/60 mmHg. Laboratory test results were P_aO_2 72 mmHg, HCT 68.9%, Hgb 22.6 $g \cdot dL^{-1}$; Lac 4.7 $mmol \cdot L^{-1}$; K+ 6.0 $mmol \cdot L^{-1}$; Na+ 133 $mmol \cdot L^{-1}$; and glucose 261 $mg \cdot dL^{-1}$. With impending respiratory failure, the patient spontaneously positioned himself in a prone position and reported persistent thirst despite 1500-mL crystalloid infusion. Extensive and rapidly growing facial and peripheral edema was observed, in addition to bluish livedo reticularis of the abdomen, buttocks, posterior thighs, and back. No gas bubbles or right-to-left shunting were identified in the ultrasound exam of the heart cavities and large vessels. On the head CT scan (**Fig. 1**), high-density small intracranial arteries were visualized, suggesting embolism or severe vasoconstriction without any focal lesions or brain edema symptoms.

Chest computed tomography was omitted due to possible delay in the implementation of treatment and the X-ray examination showed significant pulmonary edema later. At the time of admission, creatine kinase-MB, and troponin-T levels were at the upper limits of the norm. The patient received oxygen supplementation, in addition to 8 mg dexamethasone and 1 $mg \cdot kg^{-1}$ continuous infusion of 1% lidocaine. Lactate level reached 6.7 $mmol \cdot L^{-1}$ and pH dropped to 7.27. High-altitude decompression sickness was suspected and the decision was made to initiate hyperbaric therapy.

To confirm the diagnosis and optimize the hyperbaric oxygen therapy, we contacted the National Centre for Hyperbaric Medicine in Gdynia, Poland. The decision was made to start recompression therapy, with U.S. Navy Treatment Table 6 being prepared to extend the schedule.

Initially, the patient was breathing spontaneously and reporting a considerable reduction in respiratory symptoms. After 180 min of hyperbaric therapy, the neurological symptoms were completely resolved. Moreover, biochemical parameters improved (e.g., lactate dropped to 5.1 $mmol \cdot L^{-1}$); however, the edema intensified, with hematocrit persistently above 60%. During the hyperbaric session, the patient received 3500 mL crystalloids (a total of 6000 mL since the accident) and was still presenting ongoing features of fluid depletion, with co-occurring anuria. Since the neurological symptoms resolved, ventilation improved, and livedo reticularis diminished, hyperbaric oxygenation was conducted according to the standard U.S. Navy Table 6 schedule for 285 min. Over the next few hours, the ventilation parameters deteriorated, heart failure ensued, and livedo reticularis recurred. In the chest X-ray imaging, features of massive pulmonary edema were found. The patient required mechanical ventilation and high-dose norepinephrine and dobutamine infusion. Bluish reticular lesions were noted on the abdomen, buttocks, and back, accompanied by peripheral cyanosis. Within 24 h of admission, continuous veno-venous hemodialysis renal replacement therapy was initiated. Additionally, hypoxia persisted despite mechanical ventilation with F_IO_2 1.0 and high airway pressures.

Respiratory failure worsened on the third day following hyperbaric treatment. Despite the use of mechanical ventilation in BiPAP mode (24/12 cmH_2O), with $F_IO_2 = 1.0$, arterial blood saturation was 75–80%. The patient qualified for extracorporeal membrane oxygenation. In the aseptic conditions of the operating room, two percutaneous cannulas were placed under the control of transesophageal ultrasound in the left femoral vein (25F) and right jugular vein (17F). Heparinization under the control of APTT R was implemented. After commencing veno-venous extracorporeal oxygenation (VV-ECMO) therapy (Maquet Rotaflow®), arterial saturation improved instantly.

The patient's condition stabilized and multiple organ failure began to improve. On day 7, VV-ECMO was discontinued and the patient was extubated the following day. Facial and truncal edema gradually resolved. After sedation was stopped, the patient became fully alert. Neurologically, the patient complained only of weakness in the right upper extremity, which gradually improved. On day 9, an episode of delirium occurred, which responded well to antipsychotics. The ICU treatment is summarized in **Table I**. After 12 d of intensive care, the patient had uncompromised respiratory and cardiac function with no significant neurological deficits.

DISCUSSION

To the best of our knowledge, the present paper is the first case report of a patient treated for HADCS using recompression therapy, renal replacement therapy, and VV-ECMO with a complete recovery. We observed two distinct stages during the acute phase of the disease. The first phase lasted approximately 10 h from the accident until the recompression therapy was ended. During this stage, signs of hypoperfusion, neurological symptoms such as numbness and muscle weakness, and skin manifestations, including bluish livedo reticularis, were observed. During the second stage following recompression treatment, multiple organ dysfunction dominated, including heart failure, pulmonary edema, acute kidney injury, and fluid overload, all of which can be attributed to extensive endothelial damage.

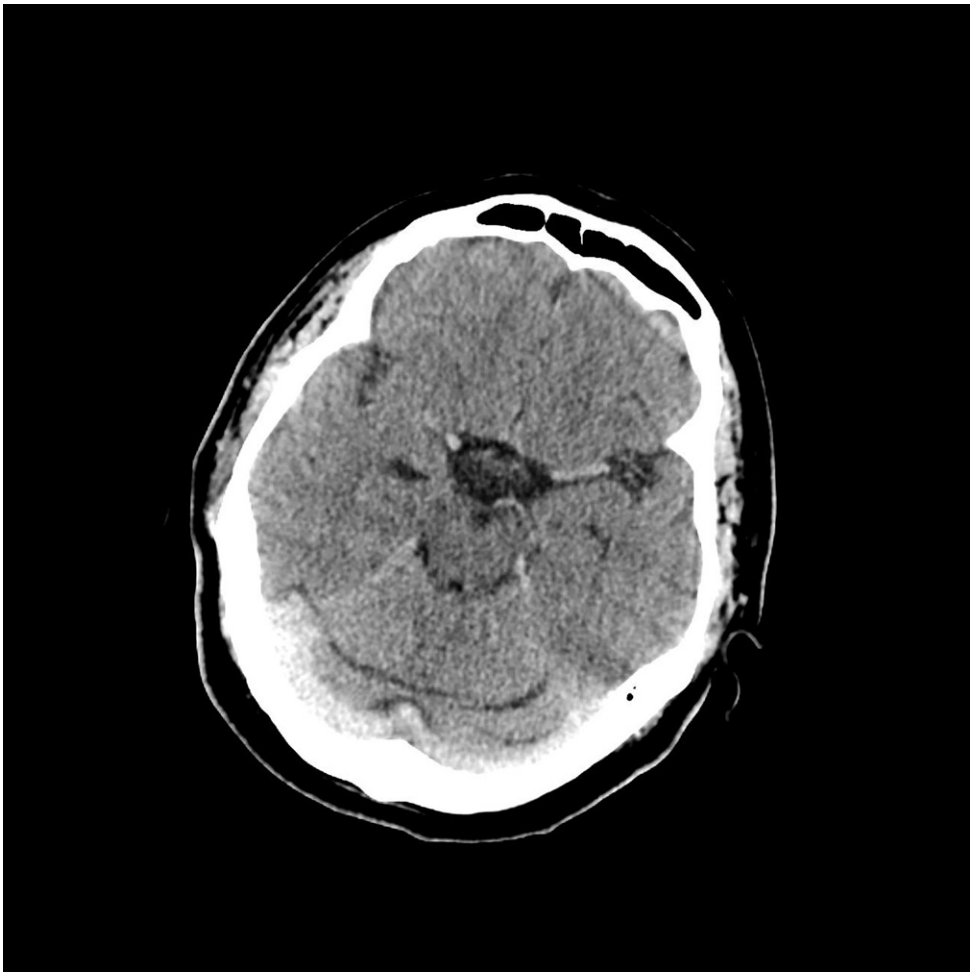

Fig. 1. Computed tomography of the brain with hypertensive image of the middle cerebral artery.

During the first stage of treatment, the choice of optimal recompression profile was difficult and remained controversial. We considered USN5, USN6, and Comex30. The presence of severe neurological symptoms advocated for prolonged therapy, considering single reports suggesting that recompression with more 'shallow' profiles in type II DCS may be ineffective.[12] We decided on hyperbaric oxygenation according to the U.S. Navy Table 6 profile with possible prolongation, since no free gas was identified on ultrasound or CT, which suggested the presence of small but numerous circular micro-bubbles. It was agreed between both hyperbaric facilities involved in the decision process that in the case of HADCS, unlike decompression sickness in divers, gas bubbles are less often grossly observed within blood vessels due to a lower relative pressure difference as the underlying cause. The improvement in ventilation, gas exchange, and complete resolution of neurological deficits upon physical examination directly after decompression supported the validity of our choice. The effect can be partially attributed to slow gas elimination from the central nervous system microcirculation, in addition to dose-dependent improvement in brain oxygenation,[8] despite a widely known mechanism underlying blood supply alterations to the brain limiting the effectiveness of hyperoxia in HBO therapy.[15] Attempts to improve the treatment effectiveness in severe DCS have become the cause of testing a wide spectrum of therapies, including perfluorocarbons in animal models, but without proven efficacy in humans.[2]

The second stage of the disease developed immediately following discontinuation of recompression therapy, which could have been due to cardiovascular failure following posthyperoxic blood vessel dilation and endothelial damage as the primary DCS mechanism.[5] These mechanisms resulted in a considerable fluid shift from blood vessels to the intercellular space, which was reflected in the biochemical tests (Hct around 65%). On the other hand, acute kidney injury is a previously reported complication of decompression sickness, which could be treated effectively with renal replacement therapy.[4]

We had serious difficulty sustaining organ function, including respiratory function. The impaired gas exchange

Table I. Treatment in Intensive Care.

	DAY OF TREATMENT											
	I	II	III	IV	V	VI	VII	VIII	IX	X	XI	XII
Hyperbaric treatment	x											
VV-ECMO			x	x	x	x	x	x	x			
Mechanical ventilation	x	x	x	x	x	x	x	x	x	x		
CVVHDF		x	x	x	x	x	x					
Vasoconstrictor infusion	x	x	x	x	x	x	x					
Sedation	x	x	x	x	x	x	x	x	x	x		
Delirium											x	x

VV-ECMO: veno-venous extracorporeal membrane oxygenation; CVVHDF: continous veno-venous hemodiafiltration.

was worsened by a massive pulmonary edema resulting from an aggressive fluid resuscitation. Based on clinical evaluation, it is impossible to point to a single cause of respiratory failure. The causes include increased endothelial leakage from the primary injury and increased transudate in the alveolar space due to increased capillary pressure. Pulmonary vessel constriction, responsible for increased pulmonary vascular resistance and pulmonary vascular pressure, can be induced either by hypoxic alveolar ventilation in different lung portions or an air embolism within the capillaries (or both).[11] In clinical evaluation of patients undergoing HBO therapy, oxygen toxicity by the Lorraine-Smith mechanism must be mentioned, which leads to alveolar-capillary membrane injury. It seems justified to suspect that oxygen toxicity not only overlaps with primary capillary injury, but also constitutes an independent risk factor increasing capillary dysfunction, as has been suggested by one article, in which a patient died during the second stage of DCS treatment as a result of pulmonary complications following long-term HBO therapy.[10] As depicted by our case, VV-ECMO applied in the second stage of DCS treatment following initial emergency recompression to eliminate free gas from the microcirculation can be more beneficial than continued hyperbaric therapy. We believe that it will be possible to simultaneously conduct both therapies in the future.

ACKNOWLEDGMENTS

The authors would like to thank Leszek Gryszko, Jarosław Gołowicz, Michał Pawlak, and Adam Machowicz for involvement and expertise in the treatment process.

Financial Disclosure Statement: The authors have no competing interests to declare.

Authors and affiliations: Jacek Siewiera, M.D., Ph.D., Department of Hyperbaric Medicine, Przemyslaw Szałański, M.D., Ph.D., Department of Cardiosurgery, and Dariusz Tomaszewski, M.D., Ph.D., Anesthesiology and Intensive Care Department, Military Institute of Medicine, Warsaw, Poland; and Jacek Kot, M.D., Ph.D., National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine, Medical University of Gdansk, Gdynia, Poland.

REFERENCES

1. Auten JD, Kuhne MA, Walker 2nd HM, Porter HO. Neurologic decompression sickness following cabin pressure fluctuations at high altitude. Aviat Space Environ Med. 2010; 81(4):427–430.
2. Cronin WA, Hall AA, Auker CR, Mahon RT. Perfluorocarbon in delayed recompression with a mixed gender swine model of decompression sickness. Aerosp Med Hum Perform. 2018; 89(1):14–18.
3. Gribble MD. A comparison of the "high-altitude" and "high-pressure" syndromes of decompression sickness. Br J Ind Med. 1960; 17(3):181–186.
4. Hibi A, Kamiya K, Kasugai T, Kamiya K, Kominato S, et al. Acute kidney injury caused by decompression illness successfully treated with hyperbaric oxygen therapy and temporary dialysis. CEN Case Rep. 2017; 6(2):200–205.
5. Imbert JP, Kiboub F, Balestra C. Measurement of the decompression stress during offshore saturation. Abstract and Conference Book, 42nd Annual Scientific Meeting of the European Underwater and Baromedical Society; Geneva, Switzerland; 13–16 September 2016. Stromness, Orkney (United Kingdom): European Underwater and Baromedical Society; 2016:33.
6. Johnston MJ. Loss of cabin pressure in a military transport: a mass casualty with decompression illnesses. Aviat Space Environ Med. 2008; 79(4):429–432.
7. Kondo Y, Fukami M, Kukita I. Extracorporeal membrane oxygenation therapy for pulmonary decompression illness. Crit Care. 2014; 18(3):438.
8. Kot J, Winklewski PJ, Sicko Z, Tkachenko Y. Effect of oxygen on neuronal excitability measured by critical flicker fusion frequency is dose dependent. J Clin Exp Neuropsychol. 2015; 37(3):276–284.
9. Lee VM, Hay AE. Altitude decompression illness - the operational risk at sustained altitudes up to 35,000 ft. Defense Technical Information Center Compilation Part Notice ADPO11091, Unclassified. Fort Belvoir (VA, USA): DTIC; 2001.
10. Louge P, Cantais E, Palmier B. Syndrome de détresse respiratoire aiguë après oxygénothérapie hyperbare prolongée: un cas d'intoxication pulmonaire à l'oxygène? Ann Fr Anesth Reanim. 2001; 20(6):559–562.
11. Mathieu D, Favory R, Collet F, Linke JC, Wattel F. Physiologic effects of hyperbaric oxygen on hemodynamics and microcirculation. In: Mathieu D. Handbook on hyperbaric medicine. Dordrecht: Springer; 2006:75–101.
12. Naqvi A, Clarence D. A case of decompression illness not responding to hyperbaric oxygen. J Intensive Care. 2018; 6(1):29.
13. Rice GM, Vacchiano CA, Moore JL Jr, Anderson DW. Incidence of decompression sickness in hypoxia training with and without 30-min O_2 prebreathe. Aviat Space Environ Med. 2003; 74(1):56–61.
14. Veronneau SJH, Pilmanis AA, Mohler SR. A case of mixed decompression illness manifestations in a commercial DC-8 cargo jet pilot. Oklahoma City (OK, USA): Federal Aviation Agency; 2009.
15. Winklewski PJ, Kot J, Frydrychowski AF, Nuckowska MK, Tkachenko Y. Effects of diving and oxygen on autonomic nervous system and cerebral blood flow. Diving Hyperb Med. 2013; 43(3):148–156.