Severe Decompression Sickness Associated with Shock and Acute Respiratory Failure

Abdullah Arjomand, James R. Holm, and Anthony J. Gerbino

1Sections of Graduate Medical Education, Virginia Mason Medical Center, Seattle, WA, USA
2Undersea and Hyperbaric Medicine, Virginia Mason Medical Center, Seattle, WA, USA
3Pulmonary Medicine, Virginia Mason Medical Center, Seattle, WA, USA
4Critical Care Medicine, Virginia Mason Medical Center, Seattle, WA, USA

Correspondence should be addressed to Anthony J. Gerbino; tgerbino@comcast.net

Received 23 April 2020; Revised 15 September 2020; Accepted 22 October 2020; Published 5 November 2020

Academic Editor: Zsolt Molnár

Decompression sickness (DCS) is a well-recognized complication of diving but rarely results in shock or respiratory failure. We report a case of severe DCS in a diver associated with shock and respiratory failure requiring mechanical ventilation. A healthy 50-year-old male diver dove to a depth of 218 feet for 43 minutes while breathing air but omitted 6.5 hours of required air decompression due to diver error. The clinical presentation was remarkable for loss of consciousness, hypotension, cutis marmorata, peripheral edema, and severe hypoxia requiring mechanical ventilation with diffuse lung opacities on chest radiograph. Laboratories were significant for polycythemia and hypoalbuminemia. A single hyperbaric oxygen treatment was provided on the day of admission during which shock worsened requiring aggressive volume resuscitation and three vasopressors. In the first 37 hours of hospitalization, 22 liters of crystalloid and multiple albumin boluses were administered for refractory hypotension by which time all vasopressors had been discontinued and blood pressure had normalized. He required 10 days of mechanical ventilation and was discharged on day 21 with mild DCS-related neurologic deficits. This clinical course is characteristic of DCS-related shock wherein bubble-endothelial interactions cause a transient capillary leak syndrome associated with plasma extravasation, hemoconcentration, and hypovolemia. The pathophysiology and typical clinical course of DCS-related shock suggest the need for aggressive but time-limited administration of crystalloid and albumin. Because hyperbaric oxygen is the primary treatment for DCS, treatment with hyperbaric oxygen should be strongly considered even in the face of extreme critical illness.

1. Introduction

Decompression sickness (DCS) is a well-recognized complication of diving that occurs when inert gas breathed at depth leaves solution and forms injurious bubbles. Symptoms are most often musculoskeletal and neurologic and are effectively treated with hyperbaric oxygen using a standard protocol administered over roughly five to eight hours.

Shock is a rare manifestation of DCS that is typically associated with a large, omitted decompression obligation. These patients often present with severe illness, challenging the clinical team to provide aggressive critical care that transitions from the emergency department, to the hyperbaric chamber, and then to the intensive care unit. We report a case of DCS causing severe shock associated with plasma extravasation, hemoconcentration, and respiratory failure requiring mechanical ventilation.

2. Case

A healthy 50-year-old experienced SCUBA diver dove to a maximum depth of 218 feet for 43 minutes while breathing air. He became confused due to nitrogen narcosis leading to a longer, deeper dive than originally planned. He ascended from depth slowly according to the staged decompression suggested by his dive computer but ultimately exhausted his supply of breathing gas and was forced to surface having omitted 6.5 hours of required air decompression. Upon
surfacing, he was conscious and aware that he had a ma-
sive unfulfilled decompression obligation, hailed a nearby
boat, and requested that emergency medical services be
summoned. Shortly thereafter, he lost consciousness but
remained at the surface due to his buoyancy control device.
He was rescued from the water, intubated, and mecha-
nically ventilated.
Upon arrival in the emergency room, he was hypoten-
sive requiring norepinephrine infusion. Peripheral edema
was present. He developed cutis marmorata (Figure 1)
[1], a rash typically associated with severe DCS. The initial
chest radiograph (after 2 L intravenous crystalloid) dem-
onstrated diffuse opacities. Initial labs showed hypoalbuminemia
(1.1 g/dL), polycythemia (hematocrit 58), lactic acidosis, and
impaired gas exchange (pH 7.18, P_{O_2} 120 mm Hg, P_{CO_2}
50 mm Hg on 100% oxygen).
He was transported to a hospital-based multiplace hyper-
baric chamber with critical care capabilities. The standard
hyperbaric treatment protocol for decompression sickness,
a United States Navy Treatment Table 6, was begun. Shock
worsened during hyperbaric treatment prompting repeated
boluses of intravenous crystalloid and albumin and addition
of vasopressin and epinephrine infusions to norepineph-
rine. Similarly, progressive hypoxia, acidemia, and ventila-
tor dysynchrony during hyperbaric treatment prompted
increases in positive end-expiratory pressure and initiation
of bicarbonate and cisatracurium infusions. Despite this
management, shock and hypoxia worsened and the decision
was made to terminate the hyperbaric treatment after 230
minutes of a planned 260-minute treatment. Subsequent
hyperbaric treatments were not provided due to cardiopul-
monary instability.
During the initial 37 hours of hospitalization, 22 liters of
crystalloid and multiple albumin boluses were administered
for refractory hypotension at which time blood pressure
normalized and all vasopressors had been discontinued
(Figure 2). Despite aggressive diuresis over the next few days
until euvolemic, he required low tidal volume mechanical
ventilation for 10 days. He was discharged on day 21 without
neurologic deficits included mild ischemic optic neuropathy and cognitive dysfunction
with brain MRI demonstrating small, multifocal infarcts
consistent with severe DCS.

3. Discussion
We present a case of severe DCS due to a massive omitted
decompression requirement associated with shock, acute
respiratory failure, and neurologic injury. This case high-
lights two rare manifestations of DCS—shock and respira-
tory failure—treated with hyperbaric oxygen therapy in the
setting of cardiopulmonary collapse.
Only three cases of shock due to DCS have been reported
in the last 45 years [2–4] but case series of DCS reported prior
to that time in divers, aviators, and animal models reveal a
characteristic clinical course. These accounts [5–8] describe
hypovolemic shock with plasma extravasation associated
with hemoconcentration and hypoalbuminemia, typically
resolving within 48–72 hours if the subject survived. The rar-
ity of these clinical reports and their publication outside of
the critical care literature may make recognition of DCS-
related shock challenging for the intensivist, especially when
not associated with an exceptional dive profile.
The pathophysiology of shock due to DCS involves the
interaction of undissolved inert gas with vascular endothel-
ium. Inert gas breathed at high ambient pressure (i.e., at
depth) forms bubbles in tissue and vascular spaces if, upon
diver ascent, the pressure decreases too quickly relative to the
amount of dissolved gas. In the present case, the omitted
decompression was massive, resulting in an uncommonly
large burden of bubbles. Bubble-endothelial interactions lead
to endothelial dysfunction and an inflammatory response [9–
12] that increases vascular permeability, resulting in plasma
extravasation and subsequent intravascular volume depletion
[7]. Whether there is a component of distributive shock
related to endothelial dysfunction that compounds hypovole-
mic shock is unclear. This pathophysiology and characteristic
clinical course suggest the need for aggressive, early but time-limited administration of crystalloid and albumin to correct intravascular hypovolemia and oncotropic pressure defects.

We advocate for a trial of hyperbaric oxygen even in the most unstable of patients with DCS or arterial gas embolism because hyperbaric oxygen is the primary treatment for these diseases and rapid improvement in typical DCS-related symptoms is common [13]. Although shock did not improve during hyperbaric treatment in the present case, we speculate that clinical outcome would have been worse without such treatment.

There are several caveats to the aggressive use of hyperbaric oxygen in the critically ill DCS patient. The patient should be first transported to the closest emergency department for evaluation and stabilization, even if that center does not have hyperbaric capabilities. Second, the ability to comfortably manage severely ill patients is variable, even among hyperbaric centers with critical care capabilities [14, 15]. Finally, the decision to treat severely ill patients with hyperbaric oxygen requires close collaboration between the intensivist and hyperbaricist throughout the clinical course to repeatedly weigh the risks and benefits of treatment [14].

This patient’s respiratory failure likely was multifactorial including water aspiration, lung DCS, extravascular fluid overload, and possibly DCS-related fat emboli. Water aspiration is likely because the patient lost consciousness at the surface prior to rescue. While hypotension predominates in DCS-related shock, varying degrees of lung injury are often reported [3, 5, 6] and interactions between pulmonary endothelium and bubbles result in vascular leak [10, 11]. DCS-related pulmonary edema is suggested in the present case by dense, homogeneous lung opacities on the admission chest radiograph (rather than patchy infiltrates anticipated from aspiration at the water’s surface) and the massive systemic capillary leak that suggests similar injury to the pulmonary endothelium. Aggressive volume resuscitation undoubtedly contributed to respiratory failure, with effects likely amplified by increased permeability of the alveolar-capillary membrane. Although there is no evidence of fat emboli in the present case, fat emboli have been found in the lung and other organs in fatal cases of DCS [6, 8, 16], presumably due to bubble-mediated infarction of long bones, and thus may also play a role in DCS-related lung injury.

We have presented a case of DCS-related shock with acute respiratory failure requiring prolonged mechanical ventilation. This case illustrates the characteristic clinical course of time-limited shock with plasma extravasation caused by severe DCS. We recommend aggressive, early resuscitation with crystalloid and albumin to correct intravascular volume deficits and strong consideration of hyperbaric oxygen treatment even in extreme critical illness.

Data Availability

Data are not applicable (all data related to the presented case is available in the medical record).

Consent

The patient has provided signed informed consent for publication of his clinical case.

Conflicts of Interest

The authors have no conflicts of interest to disclose.

References

[1] M. Mutluoglu, H. Ay, and G. Uzun, “Cutaneous manifestations of decompression sickness: cutis marmorata,” The New Zealand Medical Journal, vol. 124, no. 1340, pp. 87-88, 2011.
[2] B. Trytiko and S. Mitchell, “Extreme survival: a serious technical diving accident,” South Pacific Underwater Medicine Society (SPUMS) Journal, vol. 35, pp. 23-27, 2005.
[3] E. Gempp, G. Lacroix, J. M. Cournac, and P. Louge, “Severe capillary leak syndrome after inner ear decompression sickness in a recreational scuba diver,” The Journal of Emergency Medicine, vol. 45, no. 1, pp. 70–73, 2013.
[4] S. Klapa, J. Meyne, W. Kähler et al., “Decompression illness with hypovolemic shock and neurological failure symptoms after two risky dives: a case report,” Physiological Reports, vol. 5, no. 6, article e13094, 2017.
[5] W. G. Malette, J. Fitzgerald, and A. Cockett, “Dysbarism. A review of thirty-five cases with suggestion for therapy,” Aerospace Medicine, vol. 33, pp. 1132–1139, 1962.
[6] W. Haymaker, A. D. Johnston, and V. M. Downey, “Fatal decompression sickness during jet aircraft flight: a clinicopathological study of two cases,” The Journal of Aviation Medicine, vol. 27, no. 1, pp. 2–17, 1956.
[7] F. P. Brunner, P. G. Frick, and A. A. Bühlmann, “Post-decompression shock due to extravasation of plasma,” The Lancet, vol. 283, no. 7342, pp. 1071–1073, 1964.
[8] A. T. K. Cockett, “Pathophysiology of shock secondary to underwater decompression sickness,” Bulletin de la Société Internationale de Chirurgie, vol. 32, no. 2, pp. 229–237, 1973.
[9] K. Zhang, D. Wang, Z. Jiang, X. Ning, P. Buzzacott, and W. Xu, “Endothelial dysfunction correlates with decompression bubbles in rats,” Scientific Reports, vol. 6, no. 1, article 33390, 2016.
[10] V. Nossum, A. Hjelde, and A. O. Brubakk, “Small amounts of venous gas embolism cause delayed impairment of endothelial function and increase polymorphonuclear neutrophil infiltration,” European Journal of Applied Physiology, vol. 86, no. 3, pp. 209–214, 2002.
[11] M. Barak and Y. Katz, “Microbubbles: pathophysiology and clinical implications,” Chest, vol. 128, no. 4, pp. 2918–2932, 2005.
[12] J. D. Martin and S. R. Thom, “Vascular leukocyte sequestration in decompression sickness and prophylactic hyperbaric oxygen therapy in rats,” Aviation, Space, and Environmental Medicine, vol. 73, no. 6, pp. 565–569, 2002.
[13] R. D. Vann, F. K. Butler, S. J. Mitchell, and R. E. Moon, “Decompression illness,” Lancet, vol. 377, no. 9760, pp. 153–164, 2011.
[14] D. Mathieu, B. Ratzenhofer-Komenda, and J. Kot, “Hyperbaric oxygen therapy for intensive care patients: position statement by the European Committee for Hyperbaric Medicine,” Diving and Hyperbaric Medicine, vol. 45, no. 1, pp. 42–46, 2015.
[15] J. Kot, “Staffing and training issues in critical care hyperbaric medicine,” *Diving and Hyperbaric Medicine*, vol. 45, no. 1, pp. 47–50, 2015.

[16] W. Rait, “The etiology of postdecompression shock in aircrew-men,” *United States Armed Forces Medical Journal*, vol. 10, no. 7, pp. 790–805, 1959.