Severe Distributive Shock Due To Decompression Sickness

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I INTRODUCTION

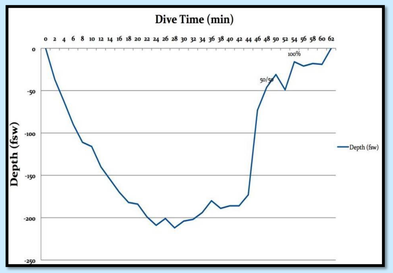
Distributive shock is a rare manifestation of decompression sickness. We present a case of severe distributive shock and acute respiratory failure due to decompression sickness.

II CASE

Pre-Hospital

A 50-year-old healthy technical diver made a single solo dive to maximum and average depths of 218 feet and 123 feet, respectively, with a total dive time of 63 minutes. Nitrogen narcosis led to an unexpected duration at depth resulting in inadequate gas supply and hours of omitted decompression.

Upon surfacing he developed respiratory distress and generalized weakness. He was removed from the water by nearby boaters, lost consciousness, and was subsequently intubated by emergency medical personnel. Evaluation in the emergency department was notable for cutis marmorata, pulmonary edema and hypoxemia requiring mechanical ventilation, and hypotension treated with norepinephrine infusion and isotonic crystalloid.



Dive profile from the patient's dive computer. Bottom gas was air. Decompression gases were 50/50 nitrox and 100% oxygen.

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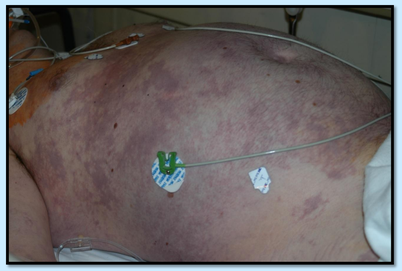
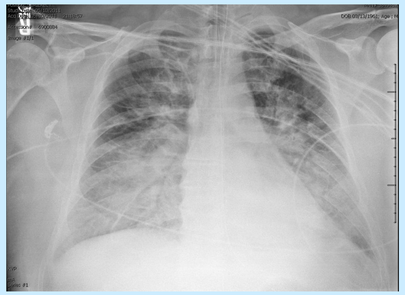
- Transfer to a hospital-based multiplace hyperbaric chamber from the emergency department of a nearby hospital.
- Treatment consisted of a US Navy Treatment Table 6 with omission of air-breaks and premature termination at 30 fsw due to worsening hypoxemia (initial PO_2 450 mm Hg; final PO_2 62 mm Hg during HBO_2) despite escalating PEEP.
- Vasopressin and epinephrine infusions were added due to maintain mean arterial pressures in the 50-60 range.
- Hypercapnea worsened despite increased minute ventilation, switch to pressure control ventilation, and administration of cisatracurium (maximum PCO_2 97 mm Hg).
- Intravenous boluses of sodium bicarbonate were given for progressive acidemia (pH 7.10 – 7.00).
- Additional hyperbaric treatments were not administered due to persistence of severe hypoxemia and shock over the next 48 hours.



Livedo reticularis (cutis marmorata) at presentation



Admission chest x-ray.

Hospital Course

Severe distributive shock consistent with capillary leak:

- Aggressive resuscitation with isotonic crystalloid: 21 Liters fluid given in the first 36 hours
- Severe hemoconcentration (initial hematocrit 58)
- Severe hypoalbuminemia (initial albumin 1.1 mg/dL)
- IV albumin boluses repeated in the first 36 hours
- Norepinephrine, vasopressin, epinephrine, bicarbonate infusions stopped within 48 hours

Acute lung injury due to pulmonary edema from decompression sickness ("chokes"):

- Severe hypoxemia requiring F_iO_2 1.0 and PEEP 20 for ~3 days
- Neuromsucular blocking agents administered to improve gas exchange, ventilator-patient synchrony.
- Mechanical ventilation discontinued on day 10.
- ICU discharge day 14
- Hospital discharge day 21 with normal cardiopulmonary function and without need for supplemental oxygen

Neurologic recovery:

- Brain MRI day 14: multifocal small bilateral subacute infarcts including occiputs
- Hospital discharge: no gross neurologic defects except visual loss
- Ophthalmologic exam day 30: bilateral ischemic optic neuropathy
- Day 55: Near normal vision right eye, finger counting left eye – improvement continues

III DISCUSSION

- Severe distributive shock due to decompression sickness ("bends shock") is rare in the modern era. We hypothesize that a large burden of undissolved intravascular gas mediates pan-endothelial injury and resultant transient capillary leak.
- Decades-old case reports in military personnel document a similar course: profound but transient capillary leak following omitted decompression. Patients typically developed distributive shock, pulmonary edema, and hemoconcentration that reversed within several days.
- Supportive critical care should include aggressive volume resuscitation to address profound intravascular hypovolemia and liberal use of colloid to correct low intravascular oncotic pressure.
- Physicians should weigh the risks and benefits of hyperbaric treatment carefully. Hyperbaric oxygen treatment risks may exceed benefits when: a. supra-physiologic arterial oxygen tensions cannot be achieved, b. inadequate ventilation is anticipated due to severe acute lung injury and hypercapnea may worsen an already critical acid-base status, c. the probability of cardiac arrest is high, d. the delay between insult and hyperbaric oxygen treatment is long.
- Modification of standard hyperbaric protocols are often necessary in such critically ill patients, including omission of air breaks and premature termination of treatments.