

A case of severe multi-system decompression sickness requiring critical care, aggressive fluid resuscitation and hyperbaric oxygen therapy

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Abstract

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An experienced recreational diver developed a severe multisystem life-threatening decompression sickness following a series of eight dives over four days, the profiles of which were all within acceptable limits. The diver had marked haemoconcentration and required large volume intravenous fluid replacement during initial treatment. Similar presentations have previously only been described following deep dives, fast ascents or where mandatory decompression had been missed. The management of this case, including the involvement of a hyperbaric unit co-located to an intensive care department, and the underlying pathophysiology are discussed.

Introduction

Severe multisystemic decompression sickness (DCS) involving cardiopulmonary compromise is rare, affecting < 1% of DCS cases.¹ It typically presents with combinations of hypoxia, hypotension, quadriplegia, rash, shock, coagulopathy and haemoconcentration. DCS of this severity is typically a consequence of deep air dives, rapid uncontrolled ascents, or technical multiday decompression diving.¹

This report describes a recreational diving incident following an unprovocative dive profile resulting in severe multisystemic DCS associated with capillary leak syndrome. This case highlights the requirement for large volume fluid resuscitation, early recompression, and the ability of the hyperbaric unit to care for an intubated patient in such cases.

Case summary

The patient has fully consented to the reporting of their case.

Diver-X (47-year-old male) was an experienced recreational diver with 355 lifetime dives and instructor qualification. He had a raised body mass index (30 kg·m⁻²) but no underlying medical conditions or previous diving incidents.

He dived on open circuit using gradient factors of 55/85 over four consecutive days (Table 1). On the final day, he

completed two boat dives with a 1.5-hour surface interval. The first (Dive 7) was to a maximum depth of 33 metres of seawater (msw) for 45 minutes breathing Nitrox 31%. A three-minute decompression stop was undertaken at 6 msw and 3 msw. The second dive (Dive 8 – incident dive) was to a maximum depth of 27 msw (average 21 msw) for 35 minutes with a safety stop at 6 msw, resulting in a total dive time of 42 minutes (Figure 1). He was breathing Nitrox 28%.

The dive went as planned with no unusual events. He surfaced with his buddy in a controlled manner, appeared well and was able to exit the water without assistance. Five minutes after this, he reported tingling in his hands and feet. This rapidly progressed, and within 15 minutes he was unresponsive to pain stimuli with laboured breathing, dilated pupils and shaking legs. Surface oxygen was administered by boat crew.

The coastguard facilitated evacuation to the nearest emergency department, arriving 1.5 hours after leaving the water. During transfer, intravenous fluids and antiemetics were administered. Despite normobaric oxygen he continued to deteriorate, developing complete paralysis below the cervical spine by the time of arrival in the emergency department. A provisional diagnosis of severe neurological DCS was made.

Examination by the duty diving physician found truncal cutis marmorata, quadriplegia, focal seizures, and pupillary

Table 1

Summary of eight dives over four consecutive days

Day	Dive	Max depth (msw)	Dive duration (minutes)	Pre-dive surface interval
1	1	21.5	53	—
	2	23.1	48	1 hour 19 mins
2	3	25.0	49	21 hours 12 mins
	4	11.5	49	1 hours 47 mins
3	5	31.8	35	20 hours 41 mins
	6	22.7	53	1 hour 57 mins
4	7	33	45	20 hours 43 mins
	8	27.1	42	1 hour 30 mins

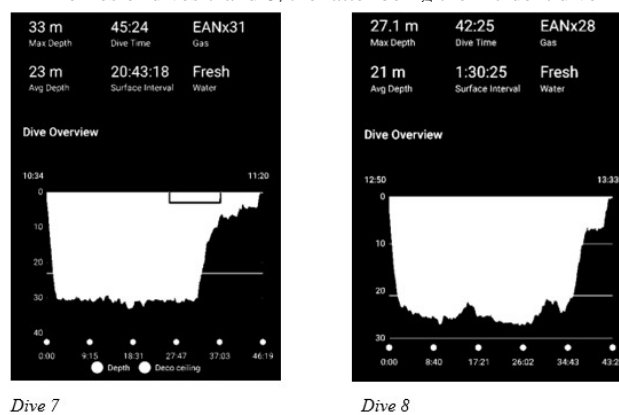
nystagmus bilaterally with right sided pupillary deviation. His airway was patent, achieving saturations of 93% whilst breathing oxygen delivered at 15 L·min⁻¹ via a non-rebreather mask with a respiratory rate of 20–25 breaths·min⁻¹. It should be noted that whilst the oxygen delivery was set at 100%, the non-rebreather mask would have provided the patient with an FiO₂ (fraction of inspired oxygen) of around 60–80%. This was the highest percentage that was able to be given, as it is unlikely that with the tachypnoea that a demand valve device would have been tolerated or effective. Consideration of non-invasive ventilation would have likely delayed the requirement for intubation and ventilation, which was ultimately the most effective method of providing 100% oxygen in this patient.

Diver-X was cold peripherally, displaying signs of shock with a heart rate of 125 beats·min⁻¹ and blood pressure of 129/107 mmHg. A 12-lead ECG showed sinus tachycardia. A chest x-ray demonstrated bilateral lower-lobe reticular infiltrates but no pneumothorax or mediastinal gas. Further intravenous access was gained, and fluid resuscitation continued. Seizure management involved lorazepam and a levetiracetam infusion. Initial Glasgow Coma Scale (GCS) was 14/15; he was able to identify his location, follow commands and open his eyes to voice. There was no movement below the cervical spine to painful stimuli.

Serial arterial blood gas (ABG) measurements via an arterial line showed significant haemoconcentration with a haematocrit of 68%, haemoglobin (Hb) of 222 g·L⁻¹ and a developing lactic acidosis (Table 2). A computed

Figure 1

Profiles of dives 7 and 8, the latter being the incident dive



tomography (CT) head and thorax showed significant portal venous gas and mild interstitial oedema at lung bases but no acute intracranial abnormality or evidence of pulmonary barotrauma.

Diver-X continued to deteriorate with progressing bulbar weakness, reduced GCS, tachycardia, hypotension, tachypnoea, and hypoxia with saturations of 89% on high flow oxygen via non-rebreather mask. Due to reduced consciousness, focal seizure activity and risk of airway compromise he was intubated in the emergency department and early intensive care support was sought. Immediately following intubation, he became increasingly tachycardic, hypoxic, and hypotensive, requiring vasopressor support and high FiO₂. He was transferred to the hyperbaric facility by a critical care transfer team. This was a 5-minute journey to an adjacent building via ambulance.

Recompression with a fully extended Royal Navy (RN) Table 62 to 2.8 atmospheres absolute (atm abs) started 5.5 hours after surfacing. The table included 127 minutes at 2.8 atm abs and 300 minutes at 1.9 atm abs. A RN62 table was chosen as it is a treatment table deemed to be effective for neurological decompression sickness whilst reducing the risk to the chamber personnel of mandatory decompression if the patient became unstable and the recompression had to be aborted (for example when compared a heliox table like a Comex 30).

During recompression, Diver-X was significantly fluid deplete and oliguric requiring 9 L of crystalloid fluid resuscitation. His ABG showed only a marginal improvement in haematocrit and Hb levels with a stable lactate (Table 2).

Diver-X was transferred to the intensive care unit (ICU). During the first 72 hours, he was increasingly hypoxic despite a high FiO₂, accompanied by an initial increase in noradrenaline requirement which then stabilised. He was laid prone for 48 hours to optimise ventilation and oxygenation. Further hyperbaric treatment was considered within this time however it was deemed to be too high risk given the transfer required by land ambulance (albeit short) and the relative

Table 2

Haematological and biochemical parameters within 12 hours of surfacing; ^^^above detectable limits; atm abs – pressure in atmospheres absolute BE – base excess; Hb –haemoglobin; Hct –haematocrit

Parameter	Hours after surfacing							
	2	4	6	8	9	10	11	12
FiO ₂			1.0	1.0	1.0	1.0	1.0	1.0
Atm abs	1.0	1.0	2.8	2.8	1.9	1.9	1.9	1.9
pH	7.35	7.32	7.26	7.30	7.32	7.32	7.37	7.34
PaO ₂ kPa mmHg			^^^ ^^^	39.6 297	53.9 404	42.5 319		
PaCO ₂ kPa mmHg	6.66 50	5.24 39	5.96 45	5.84 44	5.21 39	4.85 36	5.2	
HCO ₃ ⁻ (mmol·L ⁻¹)	27.3	20.2	20.1	21.4	20.2	18.6	22	
BE (mmol·L ⁻¹)			-7	-5	-5.9	-7.5		
Hct	68.0%	65.3%	63.0%	66.0%	63.9%	61.9%		
Hb (g·L ⁻¹)	222	213	206	215	208	202	201	212
Lactate (mmol·L ⁻¹)	2.6	2.3	3.0	2.4	2.4	2.6	2.4	2.4

remoteness of a hyperbaric facility off-site from ICU. Whilst it may have been achievable it was felt to be unsafe until the patient had improved clinically.

During early sedation holds Diver-X exhibited poor neurological recovery (only able to stick out tongue on command), prompting re-sedation. A CT head scan showed evolving ischaemic changes, and chest imaging demonstrated the development of aspiration pneumonia with patchy airspace consolidation. He was started on intravenous antibiotics.

After five days in ICU, Diver-X was stable enough to be transferred to the hyperbaric-chamber. He had three repeat treatments with un-extended RN62 tables over days 5, 7 and 9 while sedated and ventilated. Sedation holds during this time showed neurological improvement with some new leg movements.

Successful extubation was achieved on Day 12. Significant neurology persisted with hyperreflexia, hypotonia and low power throughout. Over days 14, 15 and 16 he had three further treatments with RN66 tables. Neurological assessment after each treatment showed improvement until a plateau following seven treatments. At this stage Diver-X was alert and orientated with marked improvement in global power and spontaneous movement in all four limbs. Some residual weakness remained, predominantly in the left lower limb. He was transferred to a stroke rehabilitation ward where physiotherapy continued for 10 weeks.

Magnetic resonance imaging revealed an overall improvement in intracranial appearances and no convincing abnormal cord signal. His clinical improvement mirrored this with increasing strength in all limbs and improving sensation.

He began to mobilise using a wheelchair and continued to improve following transfer to a hospital local to his family with intensive physiotherapy input. He was discharged home able to walk independently.

Diver-X was advised to have bubble contrast echocardiogram to screen for a patent foramen ovale (PFO) to investigate underlying risk factors for severe DCS. Although the cardiac centre he attended did not use full diving protocol, they were able to detect a Grade 2 PFO on Valsalva. He does not wish to return to diving.

PATIENT PERSPECTIVE

"I only remember snippets between getting back on the dive-boat and the emergency room. The dive went to plan, and I didn't feel unwell until I was back on the boat. It took around a week for me to grasp the timeline of events. I still find all a bit surreal with hindsight I really had no idea what was going for the first few days.

I had no movement on my left and was extremely weak on my right. After a stay on the stroke ward, I was transferred to a neuro-centre closer to family, still being hoisted. I was discharged two weeks later, walking independently, and followed five weeks of daily physiotherapy and rehabilitation at home. I now walk relatively long distances, commute on the tube, regularly swim 500 m and row 2 km daily alongside my daily regime and I'm largely independent. My left side is almost on par with the right, I have not regained full sensation in some of my fingers. I started back at work in the city nine weeks later, I found some of the more complex topics hard to conceptualise to begin with. Cognitively as far as I'm aware there aren't any issues. All in all, I'm in a good place.

Table 3

A summary of haemoconcentration and precipitating incident dives reported in current literature;^{5–10} Hb – haemoglobin; Hct – haematocrit; msw – metres of seawater

Reference	Hct	Hb g·L ⁻¹	Dive features
5		254	Depth: 110 msw, uncontrolled ascent from 24 msw
6	58%		Depth: 66 msw, 6.5 h omitted decompression
7	63%	209	Depth: 55 msw, uncontrolled ascent from 55msw
8	58%	206	Depth: 50 msw + five deep dives within four days prior
9	65%	221	Dive 1: 54 msw, dive 2: 48 msw, partial omitted decompression
10	55%		Dive 1: 60 msw, dive 2: 40 msw

I think about what happened a lot and what could have been. I owe so much to the staff in ICU and the hyperbaric chamber”.

Discussion

In this case, a benign dive profile led to severe, life threatening, multisystem DCS. Multiday diving to within table/computer limits is common practice and yet there are no similar reports of such severe DCS.

The presence of a patent foramen ovale (PFO) was presumably the major risk factor in this case allowing a arterialised venous bubbles to contact saturated tissues, creating a bubble cascade effect throughout the vascular endothelium. Diver-X reported no unusual activity such as excessive Valsalva or exertion during the dive therefore it is unclear why he had such a large bubble cascade on this occasion. Widespread endothelial injury compromising endothelial integrity, and or activation of inflammatory cascades leading to capillary leak syndrome would provide an explanation for the huge fluid depletion seen.

Haemoconcentration demonstrated by raised haematocrit, has been well described in divers and animal research models secondary to vascular leakage due to increased permeability across damaged vascular endothelium.^{2–5} In this case the haematocrit and haemoglobin values were high compared to the literature. Cases of DCS of similar severity involving dives to depths of at least 40 msw and including omitted decompression or rapid ascents had an average haematocrit of 59.8% and haemoglobin of 222 g·L⁻¹ (Table 3).^{6–10}

During the initial treatment, Diver-X received 9 L of crystalloid fluid. Despite the large volume of fluid resuscitation there was only marginal improvement in haematocrit (Table 2), demonstrating potential for a larger fluid resuscitation requirement, a feature consistent with severe DCS described in case studies^{6–8,10} and summative literature.^{1,11}

Conclusions

This case report demonstrates that life-threatening severe multisystem DCI can occur following benign dive profiles,

likely a result of an underlying contributing factor, such as a PFO. The facility to provide early recompression and aggressive fluid resuscitation in ventilated patients is critical. A co-located ICU and hyperbaric unit equipped with suitably trained staff is vital and it is likely that this case of DCI would have proved fatal if this had not been available.

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