Use of in-water recompression for decompression illness after deep freediving: a case series

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Abstract
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Introduction: There are increasing anecdotal reports of in-water recompression in freedivers who surface with neurological symptoms, likely suffering from decompression illness (DCI). Given the remote locations where many cases occurred, divers often struggled to access medical care, including the gold-standard hyperbaric oxygen treatment (HBOT), thus resorting to in-water recompression (IWR). Currently, IWR guidelines have only been discussed for scuba and surface supplied divers in specific scenarios, with protocols prescribing oxygen breathing at depths ≤ 9 metres maximum for around 1–3 hours.

Methods: We conducted detailed interviews with six competitive freedivers on signs, symptoms, management, and resolution of 13 cases of DCI. We additionally requested records of medical evaluation and treatment, with their consent.

Results: Three cases were suggestive of decompression sickness, six were consistent with arterial gas embolism, and four were ambiguous. Six cases were treated with IWR for 20–90 min at 5–25 metres with partial to complete resolution of symptoms. Four of these cases received HBOT afterwards. One diver reported significant permanent disability. Divers made several regimen changes after these incidents, including staying well-hydrated, reducing lung-packing, slowing their ascent rate, and/or employing prophylactic IWR when diving beyond a specified depth.

Conclusions: Given the remote locations of many incidents, freedivers often faced challenges in accessing HBOT. Self-treatment with IWR was widely used, either as a bridge to HBOT or as a standalone remedy. IWR poses potential risks, especially at the deeper depths reported in this study. This treatment modality is being utilised sometimes without medical oversight and recommended guidelines for IWR for freedivers should be developed.

Introduction

Severe debilitating neurologic symptoms after breath-hold diving (BHD), traditionally coined ‘Taravana syndrome’, have been well documented, and while thought to be linked to decompression illness (DCI), their pathophysiology has not been clearly delineated. DCI, a collective term encompassing decompression sickness (DCS) and arterial gas embolism (AGE), arises from evolution of bubbles from dissolved nitrogen gas (DCS) and/or air entering the arterial circulation after pulmonary barotrauma (AGE).[1] Theories of gas sources include de novo formation of bubbles in the arterial circulation with passage of bubbles through a patent foramen ovale, lung shunt, or frank lung barotrauma releasing alveolar gas.[2–6] DCI symptoms have also been attributed to endogenous cerebral ischaemia and blood-brain barrier disruption due to endothelial dysfunction.[7] Nonetheless, the pathophysiology underlying DCI particularly in BHD is still currently debated.[8]

The range of reported neurologic symptoms after BHD extends a wide spectrum from mild symptoms such as weakness, dizziness, and headaches to severe symptoms such as complete paralysis and loss of consciousness. Mild symptoms may be barely perceptible to divers and easily dismissed as post-dive fatigue or the lingering effects of hypoxaemia. Meanwhile, severe symptoms are rare with a recent systematic review of literature documenting 14 cases.[1,9] In addition, there are cases to suggest that this entity may not always be associated with depth given neurologic symptoms have been seen after shallow breath-hold diving and even lung-packing (glossopharyngeal insufflation).[10,11] Given the low incidence of severe neurologic symptoms, gathering data on such cases and

developing guidelines for the management of DCI in BHD has been difficult, with the added challenge of breath-hold depth diving often occurring in austere, remote locations often with no clinical medical support or oversight.

Given the rise of both recreational and competitive breath-hold diving, the medical community should have increased awareness of the possibility of DCI with severe symptoms, as well as a mechanism for gathering data that can inform medical management. Many hyperbaric practitioners may not be aware of injuries related to BHD. This case series challenges the historical dogma that DCI after BHD is not possible given the short time at depth.[12,13] In addition, many divers today are notably self-diagnosing and self-treating with in-water recompression (IWR). In-water recompression involves rapid re-immersion following the onset of symptoms, most often to shallow depths around 9 m, preferably while breathing pure oxygen.[14] Although this technique remains controversial and its actual benefits are difficult to assess due to the wide variety of protocols, it is increasingly being considered in various management guidelines under specific conditions.

Methods

This case series presents six divers with 13 cases of neurologic symptoms following BHD, documented through personal interviews conducted by two physician researchers after an initial survey of DCI in BHD. It is important to note that the symptoms and outcomes reported are anecdotal. We conducted detailed interviews after informed consent with six competitive freedivers on signs, symptoms, management, and resolution of 13 incidents that could represent DCI. We additionally requested medical records as available, with each subject's consent. This study was conducted with approval from the Institutional Review Board of University of California, San Diego, under protocol #810598 – Survey of Decompression Illness Incidents in Breath-Hold Divers. Each subject agreed to the publication of their case or cases in medical literature.

Results

Six divers reported 13 cases of suspected DCI summarised below (Table 1). Median depth of the deepest dive for each case was 100 m (interquartile range [IQR] 89−110 meters) with a median dive time of 3:08 minutes:seconds (IQR 2:15–3:16). Three cases were suggestive of DCS, six were consistent with AGE, and four were ambiguous. All reported incidents involved male divers, with suspected provoking factors being dehydration, lung packing, exhaustion, repetitive dives with short surface intervals, and lung barotrauma. Six cases were treated with IWR. The median maximum depth for IWR was 8.5 m (IQR 6.5−15.0) with a median time of 24.5 minutes (IQR 10.5–34.3). Four of the six cases received hyperbaric oxygen treatment (HBOT) afterwards. One diver reported significant residual deficits even after HBOT. A map of incident locations can be found in Figure 1. They have been unassigned from the diver and incident to protect their identity. The cases are described below. Following each case is our hypothesis of the cause for the reported signs and symptoms.

CASE 1 (DIVER A)

A male diver in his 30s had just completed a transatlantic flight and reportedly felt dehydrated. He performed four BHDs to 40, 60, 80, and 100 m and served as a safety diver for other divers as well. His final dive was a 100 m constant weight dive, with a dive time of three minutes 15 seconds. Upon reaching the shore around 10–15 minutes after the dive, he felt dizzy, had facial numbness, and was unable to walk due to muscle weakness. Around 45 minutes after symptom onset, he went to a local doctor who thought his symptoms were related to hypoglycaemia. He breathed around 10 minutes of surface oxygen at the clinic, and his symptoms were completely resolved. He attributes this incident to the number of dives, short surface intervals, exhaustion from travel, and dehydration with possible hypoglycaemia.

Analysis

We speculate his symptoms could represent DCS from repetitive dives successfully treated with surface oxygen. We believe the diagnosis of hypoglycaemia could be a red herring as the treatment for hypoglycaemia is glucose, not supplemental oxygen.

CASE 2 (DIVER A)

Diver A performed a competition constant weight dive with an announced depth of 125 m. At 90 m depth, he could no longer equalise and turned back to the surface. After this dive, he breathed prophylactic oxygen for five minutes on the surface. On his way back to shore around 3–5 minutes after the dive, he reported extreme vertigo and right-sided paralysis. After 30 minutes of breathing surface oxygen, his symptoms persisted. He proceeded to breathe 30 minutes more of surface oxygen, with symptoms resolving in the last 10 minutes of this oxygen administration. He attributes this incident to lung overpacking and competition nervousness. Of note, he reports that two days before this competition dive, he dove to 120 m twice, after which he experienced word-finding difficulties. He also reports that they were in phone contact with a local hyperbaric chamber who advised against transportation and to continue oxygen administration.

Analysis

We speculate that the patient's hemiplegia within minutes of surfacing could represent a cerebral AGE. The diver reported overpacking so he could have suffered asymptomatic

Table 1
Cases of severe neurologic symptoms and reported treatment; CPR – cardiopulmonary resuscitation; HBOT – hyperbaric oxygen treatment; IWR – in-water recompression; n/a – not available; Surf O_2 – surface oxygen

Diver	Case	Dive depth (m)	Dive time (min:sec)	Symptoms	Treatment	IWR depth (m)	IWR time (mins)
A	1	Multiple dives to 40, 60, 80 and 100	1:40	Dizziness, facial numbness, inability to walk	Surf O_2		
	2	125	n/a	Extreme vertigo, right-sided paralysis	Surf O_2		
B	3	Multiple dives to 40	2:00	Numbness and tingling in the right leg	IWR, Surf O_2	10	3
	4	127	6:38	Unconsciousness, paralysis to all extremities	CPR, IWR, Surf O_2	5–20	29
C	5	Multiple dives to 55	n/a	Severe headache, coughing fit, balance issues	Surf O_2		
	6	92	3:04	Left-side paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, shortness of breath	Surf O_2		
	7	100	3:20	Aphasia	IWR	15–25	60
	8	114	3:14	Left arm paralysis	IWR, Surf O_2	6–7	10
	9	105	3:10	Left arm paralysis, slurred speech, dizziness, fatigue, shortness of breathing	IWR, HBOT	5–7	12
D	10	89	3:15	Right arm tingling, difficulty breathing	Surf O_2		
	11	88	3:12	Abnormal behavior, facial drooping, right-sided paralysis	IWR, Surf O_2, HBOT	10	35
E	12	110	n/a	Aphasia, right arm, and leg paralysis	IWR, Surf O_2, HBOT	10–20	32
F	13	86	2:20	Right-hand numbness, tingling, right leg paralysis	IWR, HBOT	5	20

Figure 1
Map of incident locations, some of which are in remote, austere island locations

overexpansion barotrauma. Prophylactic surface oxygen did not prevent the incident, but it was successfully treated with surface oxygen.

CASE 3 (DIVER B)

A male diver in his 40s performed a 40 m constant weight dive with a 45 second hang (staying at depth) for a total dive time of two minutes. Of note, he had previously completed six dives to 40 m with a 45 second hang; therefore, this was not new or different from previous. He experienced numbness and tingling in the right leg. He did not have oxygen readily available and performed IWR on apnoea. The symptoms were not resolved. He was able to procure oxygen about one hour later, after which he re-entered the water for three minutes at 10 m. His symptoms partially resolved and dissipated over one month. He later received

a magnetic resonance imaging (MRI) scan which reportedly showed damage to the hypothalamus.

Analysis

We speculate this is a case of DCS from prolonged time at depth partially treated with IWR. The MRI findings of hypothalamus damage are likely unrelated to the case.

CASE 4 (DIVER B)

Around three years later, Diver B performed a 127 m personal best attempt free immersion dive with 50 lung packs. His previous personal best in this discipline was 126 m. The total dive time was six minutes and 42 seconds. On his ascent, he blacked out at 42 m. Per his dive computer, he sank back down to 76.8 m at 5:20. He was brought back to the surface at 6:42 unconscious. Cardio-pulmonary resuscitation (CPR) was performed for 30 seconds with a successful return of spontaneous circulation and consciousness. He had weakness over his extremities but could speak. He then self-administered IWR, descending to 20 m followed by a slow ascent breathing pure oxygen for eight minutes. He then conducted a second IWR, spending three minutes at 15 m, eight minutes at 10 m, and 10 minutes at 5 m. After this treatment, his symptoms were completely resolved.

Analysis

We speculate this is a case of drowning from a deep underwater blackout. The reason for the blackout is unclear but could include DCI.

CASE 5 (DIVER C)

A male diver in his late 30s performed breath-hold dives five times to around 55 m with a surface interval of around 20–30 minutes between each dive. At that time, his personal best was 80 m. He noted that he exerted himself after pulling up the bottom weight at the end of the dive session. As he was raising the bottom weight to the surface, he reports choking on some water that induced a coughing fit. When the cough subsided, he reports experiencing a sharp headache. After swimming to shore, he experienced balance issues, falling to the ground multiple times as he attempted to exit the water. Two hours later, he breathed surface oxygen for about 30 minutes, which partially resolved his symptoms. He noted that the episodes of dizziness persisted for about two weeks. He noted that perhaps decreasing the frequency of deep dives would have prevented this incident.

Analysis

We speculate this is a case of DCS from repetitive dives partially treated with delayed surface oxygen. His coughing is attributed to aspirating water and not pulmonary injury.

CASE 6 (DIVER C)

Around seven months later, Diver C performed a free immersion dive to a depth of 92 m, with a dive time of three minutes and four seconds. He packed approximately 10 times. At that time, his personal best in the discipline was 100 m. Of note, earlier in the month, he contracted COVID and was in quarantine before this dive. He reported nine days of rest before this dive. He noted that he also exerted himself after this dive by pulling up the bottom weight. Approximately 15–20 minutes after the dive, he experienced complete left-sided paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, and shortness of breath. He breathed pure oxygen at the surface for around 40 minutes. His symptoms began to gradually improve at around 25 minutes of using oxygen with full resolution by the end of the 40 minutes on oxygen. He reports that over the course of the next four months he would wake up each morning with hand tremors.

Analysis

We speculate this could be neurological DCS from the release of gas in tissues triggered by exertion after diving. However, cerebral AGE could also cause his unilateral neurological deficits. Given his recent pulmonary infection, glossopharyngeal insufflation, and concurrent shortness of breath, we suspect pulmonary barotrauma could have led to gas emboli. This case was successfully treated with surface oxygen. The hand tremors are likely unrelated to the case.

CASE 7 (DIVER C)

Around eight months later, Diver C performed a free-immersion training dive to 100 m, with a dive time of three minutes and 20 seconds. He noted that on this day he was dehydrated. Around 20 minutes after the dive, he experienced aphasia. He also struggled to get oxygen as it was on shore, which caused a delay. He self-administered IWR with pure oxygen at 15 m for 20 minutes, which completely resolved his symptoms. He completed a second IWR, where he breathed pure oxygen at 25 m while being slowly brought up to surface, which took a total time of 40 minutes. He had no residual symptoms after this incident.

Analysis

We speculate that this was a case of DCI successfully treated with IWR alone.

CASE 8 (DIVER C)

Around one year later, Diver C performed a constant weight mono-fin dive to 114 m over three minutes and 40 seconds. During prophylactic IWR at around 6–7 m, which he routinely performed immediately after the dive, he noticed weakness to his left arm. He stayed underwater for around 10 minutes, converting his prophylactic IWR to a treatment

session. Afterwards, he breathed oxygen at the surface for 10 minutes, which completely resolved his symptoms.

Analysis

Prophylactic IWR did not prevent this DCI incident, but the combination of IWR and surface oxygen successfully treated the case.

CASE 9 (DIVER C)

Around one year later, Diver C performed a constant weight bi-fin training dive to 105 m, with a dive time of three minutes 10 seconds with approximately 20 lung packs. Around two minutes after this dive, he experienced left arm paralysis, slurred speech, dizziness, fatigue, and shortness of breath. He immediately self-treated with IWR with pure oxygen at 5 m for around 6–7 minutes, which completely resolved his symptoms at the time. After surfacing, he finished the oxygen in the tank (the symptoms returned within 15 min after surfacing). Fifteen minutes after finishing the oxygen, his symptoms returned, notably his left arm deficits. About 1 hour and 30 minutes later, he was able to self-administer IWR again with pure oxygen at 7 m for five minutes, which resolved all his symptoms. However, once again, he ran out of oxygen, and 5–10 minutes after surfacing, he reported that all his symptoms were returning. He was then taken by ambulance to the nearest hospital and later transferred to a hyperbaric chamber, which took up to 72 hours. There, he underwent 11 sessions of hyperbaric treatment. Workup including complete blood count and basic metabolic panel, chest X-ray, and transthoracic echocardiogram was grossly normal. He has residual deficits in balance and weakness in his left arm.

Analysis

We speculate this is a case of cerebral AGE given the rapid onset of symptoms from surfacing. Given his concurrent shortness of breath along with focal neurological symptoms, we suspect he had a component of pulmonary barotrauma that could have led to arterial gas emboli. He was partially treated with IWR and HBOT.

CASE 10 (DIVER D)

A male diver in his 60s performed a free immersion dive to 89 m with a dive time of three minutes and 15 seconds. Within seconds after the dive, he experienced right arm tingling. He breathed surface oxygen for five minutes, which completely resolved his symptoms. He had increased breathing hours after the dive.

Analysis

This case was successfully treated with just five minutes of surface oxygen, suggesting that it was extremely mild DCI or transient hypoxemia after a dive.

CASE 11 (DIVER D)

Around two years later, Diver D performed a competition-free immersion dive to 88 m with a dive time of three minutes and 12 seconds. Of note, he noted that five days leading up to this competition dive, he dove to 93 m where he felt some chest pressure post-dive but otherwise recovered appropriately. He dove to 81 m the day before this dive without incident. Five to ten minutes after the competition dive, fellow competitors noticed abnormal behaviour and facial drooping. Competition staff noted that he could not follow directions or count fingers and experienced paralysis in his right leg and arm. Medical staff administered IWR with pure oxygen to 10 m with a slow ascent, with a total in-water time of 35 minutes. He had complete recovery after this treatment. He continued to breathe oxygen at the surface for 30 more minutes. Based on the recommendation of the medical staff, the diver underwent a second round of IWR breathing pure oxygen at 5 m with a slow ascent, for a total time of approximately 30 minutes. He continued to have no residual deficits. Three hours later, he was evacuated by helicopter to the nearest hyperbaric chamber. At this hospital, he was haemodynamically normal and neurologically intact, with a normal non-contrast cerebral computed tomography (CT) scan. His lung CT showed "*discrete areas of scattered ground-glass opacities in the pulmonary hemifields. Findings are nonspecific but suggestive of barotrauma-related lesions in the context of the incident*". He underwent intravenous hydration as well as two HBOT treatments (US Navy Table 5a). Months later, he underwent a transthoracic echocardiogram with bubble contrast which showed a small patent foramen ovale (PFO).

Analysis

We suspect this is a case of cerebral AGE given the rapid onset of unilateral neurological symptoms after surfacing. Even though the diver did not report any respiratory symptoms, he had cross-sectional lung imaging that demonstrated evidence of lung injury, which could have led to arterial gas emboli. The patient was also found to have a PFO, which is another potential pathway for gas entry into the arterial circulation. This case was successfully treated with IWR, surface oxygen, and HBOT.

CASE 12 (DIVER E)

A male diver in his late 40s performed a free immersion dive to a depth of 110 m. At that time his previous personal best was 120 m. Five minutes after the dive, he became aphasic with right arm and leg paralysis. Oxygen was procured from shore and brought out to the platform. He self-administered IWR with pure oxygen at around 13 m for around 15 minutes. At this time his paralysis was completely resolved but he still found it hard to find words and felt that his speech was not fully there. Several hours later, he self-administered IWR again following this protocol: 20 m for two minutes, 15 m for five minutes, 10 m for 10 minutes,

slow ascent to 5 m, and then to the surface. At this time, though his physical symptoms improved, he still could not do simple math and felt brain fog and sluggish. Two days later, he flew to a hyperbaric chamber where he completed five sessions of HBOT. Today, while he is not entirely sure, he feels that he has completely recovered, at least grossly his mental and physical function. He endorses plentiful rest and use of prophylactic oxygen (pure oxygen at 10 m for 5–6 minutes) for dives deeper than 105 m.

Analysis

We suspect this is a case of cerebral AGE successfully treated with IWR and HBOT.

CASE 13 (DIVER F)

A male diver in his 30s performed a training dive to 86 m with a dive time of two minutes and 20 seconds. He packed 40 times prior to this dive, exhaling prior to surfacing at a depth of less than 10 m on ascent. This was a personal best dive, where his previous personal best was 84 m. He notes that he experienced an increased urge to cough and 1–2 episodes of haemoptysis. Starting 30 seconds after this dive, he experienced right hand numbness and tingling and progressed to complete paralysis of his right leg. At that time, there was no oxygen readily available. He returned to shore and was unable to ambulate or feel his right leg. Around 30 minutes later, he self-administered IWR, where he breathed pure oxygen at 5 m for five minutes for four times with a surface interval of around five minutes. After the third round of IWR, his symptoms improved; however, he still could not move or feel his toes. He was taken to the nearest hyperbaric chamber and started HBOT around three hours after his symptoms started. He reported undergoing 10 treatments using US Navy Treatment Table 6 over the next 10 days. In this admission, he had a transthoracic echocardiogram with bubble contrast which showed no PFO. Laboratory tests were within normal limits. His symptoms significantly improved, although he has residual mild paresthesia in his right shin. He endorses packing less, hydrating well, slowing down on ascent in the last 30 m, exhaling before surfacing, and prophylactically breathing oxygen at depth after dives deeper than 85 m.

Analysis

We suspect this is a case of cerebral AGE from pulmonary barotrauma of ascent given the haemoptysis reported by the diver. This case was partially treated with IWR and HBOT.

Discussion

Of these cases, three cases were suggestive of DCS, six were consistent with AGE, and two were categorised as DCI given not enough data to distinguish between DCS and AGE, as outlined in Table 2. Reported risk factors included repetitive dives with short surface intervals, exhaustion, dehydration, and overpacking. There were no clear patterns or consensus of risk factors. There were also no patterns on timing of repetitive deep dives and surface intervals, with divers reporting up to one week of rest (cases 6 and 9) before the target dive that resulted in neurologic deficits. IWR was able to provide partial to complete resolution of symptoms in many of these cases, although there was no consistency between protocols used. Only one diver reported significant permanent neurological deficits, which likely resulted from the multi-day delay to HBOT.

Current prehospital management guidelines for DCI, including DCS and/or AGE, emphasise the importance of first aid and activating emergency services. While the DAN diving hotline provides global support, it cannot replace local emergency services. Initial management focuses on airway protection, administering oxygen, and maintaining normothermia. Oral hydration is recommended for conscious patients. Patients presenting with symptoms beyond mild DCS should receive definitive recompression therapy as soon as possible, as delays beyond six hours are associated with less complete recovery.[15,16] Air transport should be as close to sea-level atmospheric pressure as possible or conducted at the lowest feasible altitude (ideally below 150–300 m).

Given the remote island locations where many cases occurred, as shown in the map (Figure 1), divers struggled to access medical care, including hyperbaric chamber treatment. Thus, many self-administered IWR, either to temporize their symptoms as a bridge to hyperbaric treatment or in some cases, fully treat their physical impairment. Most divers in this series survived these incidents without permanent deficits. However, one case resulted in a moderate permanent neurologic deficit, highlighting the importance of immediate treatment. Divers must weigh the benefits and risks of seeking hyperbaric chamber treatment, which may not be readily accessible, versus conducting IWR in remote locations with no medical support or supervision. Only one case of IWR was administered by medical staff and all other incidents appear to be self-designed and self-administered.

Existing protocols for IWR have been designed for compressed air divers who have been supersaturated by breathing compressed gas, but it is unclear if the divers in this case series were aware of those protocols.[17] Recommended safety practices include using a full-face mask or mouthpiece retaining strap, tethering the diver to prevent sinking, limiting treatment depth to 9 m and having a buddy assist in case of a seizure related to oxygen toxicity.[14] Contraindications to IWR include hearing loss, vertigo, vomiting, altered mental status, shock, respiratory distress, or severe physical incapacitation.[14] The fact that many of these cases had severe symptoms that could be contraindications to IWR brings up safety concerns over the oversight and availability of oxygen. There is a need for these safety practices with the consideration for air or sea evacuation to be specific to the BHD environment to minimise improvisation during times of emergent need.

Table 2
Case symptomatology, presumed diagnosis, reported potential risk factors; AGE – arterial gas embolism; DCI – decompression illness; DCS – decompression sickness; PFO – patent foramen ovale

Diver	Case	Symptoms	Presumed diagnosis	Residual deficits	Potential risk factors
A	1	Dizziness, facial numbness, inability to walk	DCS	None	Repetitive dives, exhaustion, dehydration, overexpansion barotrauma
	2	Extreme vertigo, right-sided paralysis	AGE	None	
B	3	Numbness and tingling in the right leg	DCS	None	Prolonged time at depth
	4	Unconsciousness, paralysis to all extremities	Syncope, drowning	None	
C	5	Severe headache, coughing fit, balance issues	DCS	None	Repetitive dives (Case 5) Overexpansion barotrauma (cases 6 and 9)
	6	Left-side paralysis, blurry vision, slurred speech, impaired coordination, fatigue, dizziness, shortness of breath	AGE	None	
	7	Aphasia	DCI	None	
	8	Left arm paralysis	DCI	None	
	9	Left arm paralysis, slurred speech, dizziness, fatigue, shortness of breathing	AGE	Gait, balance, left upper extremity weakness	
D	10	Right arm tingling, difficulty breathing	DCI vs hypoxia	None	Overexpansion barotrauma, PFO
	11	Abnormal behavior, facial drooping, right-sided paralysis	AGE	None	
E	12	Aphasia, right arm, and leg paralysis	AGE	None	None obvious
F	13	Right-hand numbness, tingling, right leg paralysis	AGE	Paresthesia to right lower extremity	Overexpansion barotrauma

Conclusions

In conclusion, severe neurologic symptoms consistent with DCI can occur after breath-hold diving. The reported incidents occurred as shallow as 40 m, with several cases noted after repetitive dives with extended time at depth. All incidents reported in this case series occurred in male divers. Suspected contributors included dehydration, overpacking, exhaustion, and frequent deep dives, with two cases linked to lung barotrauma based on reported symptoms or imaging findings. Oxygen treatment, both normobaric and hyperbaric, was the most used treatment modality, with seven cases using self-administered IWR to achieve partial or full symptom resolution.

The incidents reported in this series underscore the importance of understanding DCI and the need for proper prehospital management and treatment protocols. Breath-hold divers are increasingly self-treating with IWR without seeking definitive hyperbaric oxygen therapy in a chamber under the supervision of a physician. IWR poses significant potential risks such as drowning and hyperoxia, especially at the depths reported in this study, and is currently not a recommended practice. In addition, after an incident has occurred and seemingly resolved, it is imperative that a diver seek definitive treatment and evaluation from a medical professional knowledgeable in hyperbaric medicine. As BHD practices continue to evolve, it remains critical to document and research these incidents to increase awareness of DCI incidents and develop best-practice guidelines for the management and prevention of these incidents.

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