A retrospective review of divers treated for inner ear decompression sickness at Fiona Stanley Hospital hyperbaric medicine unit 2014–2020

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Abstract
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Introduction: Inner ear decompression sickness (IEDCS) is increasingly recognised in recreational diving, with the inner ear particularly vulnerable to decompression sickness in divers with a right-to-left shunt, such as is possible through a persistent (patent) foramen ovale (PFO). A review of patients treated for IEDCS at Fiona Stanley Hospital Hyperbaric Medicine Unit (FSH HMU) in Western Australia was performed to examine the epidemiology, risk factors for developing this condition, the treatment administered and the outcomes of this patient population.

Methods: A retrospective review of all divers treated for IEDCS from the opening of the FSH HMU on 17 November 2014 to 31 December 2020 was performed. Patients were included if presenting with vestibular or cochlear dysfunction within 24 hours of surfacing from a dive, and excluded if demonstrating features of inner ear barotrauma.

Results: There were a total of 23 IEDCS patients and 24 cases of IEDCS included for analysis, with 88% experiencing vestibular manifestations and 38% cochlear. Median dive time was 40 minutes and median maximum depth was 24.5 metres. The median time from surfacing to hyperbaric oxygen treatment (HBOT) was 22 hours. Vestibulocochlear symptoms fully resolved in 67% and complete symptom recovery was achieved in 58%. A PFO was found in 6 of 10 patients who subsequently underwent investigation with bubble contrast echocardiography upon follow-up.

Conclusions: IEDCS occurred predominantly after non-technical repetitive air dives and ongoing symptoms and signs were often observed after HBOT. Appropriate follow-up is required given the high prevalence of PFO in these patients.

Introduction

Inner ear decompression sickness (IEDCS) has traditionally been associated with deep, mixed gas diving.[1] Bubble formation in the vestibulocochlear system was partly attributed to gas counter-diffusion, where bubbles formed as a result of transient super-saturation when the breathing gas was switched from helium to nitrogen during ascent.[2] It is however becoming increasingly recognised that IEDCS may also occur in non-technical air diving, demonstrating that there are other mechanisms by which the inner ear structures are susceptible to damage from bubble formation.[3–6] Previous research has suggested the association of a right-to-left shunt as a predisposing factor, whereby arterialised gas emboli can potentially grow due to slower inert gas washout from tissues of the inner ear.[6,7]

Inner ear decompression sickness is characterised by bubble mediated injury to the vestibulocochlear system. Vestibular manifestations including vertigo, nystagmus, nausea and vomiting have been found to present more commonly than those of the cochlear system, encompassing hearing loss and tinnitus. The propensity for vestibular symptoms to predominate is thought to be due to lower perfusion and hence slower inert gas washout of the vestibular system compared with the cochlea.[3]

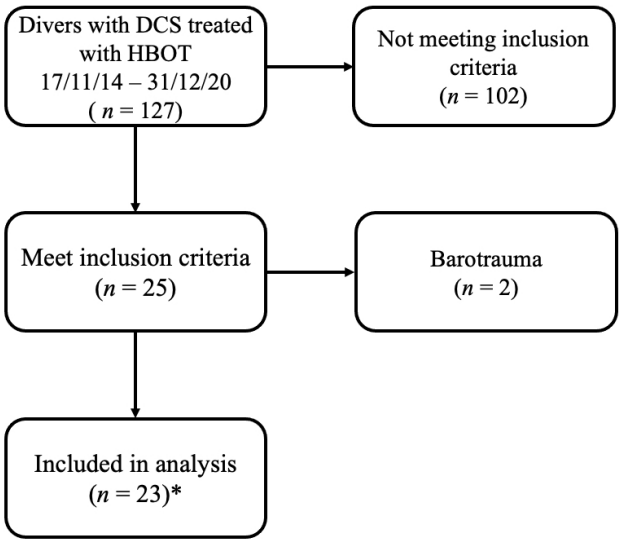
A universally accepted definition of IEDCS is lacking and studies to date vary in their inclusion criteria. Other dive related injuries such as cerebellar decompression sickness (DCS) or inner ear barotrauma (IEBt) can display symptoms similar to IEDCS and the diver with symptoms of vestibulocochlear dysfunction can present a diagnostic challenge to the clinician.[8] A review has recently described the most useful variables in differentiating IEBt from IEDCS, including dive type (breath-hold versus scuba), dive gas (compressed air versus mixed gas), dive profile (mean depth 13 versus 43 metres), symptom onset (upon descent

versus ascent or after surfacing), symptom distribution (vestibular versus cochlear) and absence or presence of other DCS symptoms.[9] It is important to perform a thorough assessment to attempt to differentiate IEDCS from IEBt, although case data has shown that a trial of hyperbaric oxygen treatment (HBOT) may not worsen IEBt if it cannot be excluded, provided the diver can equalise their ears.[10]

The aim of this study was to investigate the epidemiology of patients with IEDCS treated at Fiona Stanley Hospital Hyperbaric Medicine Unit (FSH HMU) in Western Australia (WA) and provide a description of risk factors for developing the condition, in addition to investigating treatment outcomes for this population. The FSH HMU is the WA State Referral Service for diving and hyperbaric medicine and takes referrals from throughout the state, as well as Australia's Indian Ocean territories (Cocos-Keeling and Christmas Islands). Western Australia has the longest coastline of any Australian state, providing a vast area for dive activities and potential challenges in the retrieval of injured divers.[11] The time to HBOT was compared with other case series from around the world to determine if this unique geography may affect treatment times and outcomes.

Figure 1

Flow diagram of data acquisition; *one diver was treated twice during the study period giving a total 24 episodes of IEDCS; DCS – decompression sickness; HBOT – hyperbaric oxygen treatment



Methods

Written approval was obtained for data review and extraction by Governance, Evidence, Knowledge and Outcomes (GEKO) at FSH (Approval Number 39297), and by the Human Research Ethics Committee of Curtin University (Approval Number HRE2021-0029).

PATIENT SELECTION

A retrospective review of the electronic medical record of all patients with a diagnosis of DCS presenting to FSH HMU from its opening on 17 November 2014 to 31 December 2020 was performed. The medical records were reviewed to identify those with symptoms suggestive of IEDCS. Patients were included if presenting with vestibular dysfunction (vertigo, nystagmus) and/or cochlear dysfunction (hearing loss, tinnitus) occurring within 24 hours (h) of surfacing from a dive. Patients with symptoms or signs of aural barotrauma (history of ear clearing difficulty, tympanic membrane erythema or haemorrhage), suggesting the alternative diagnosis of IEBt, were excluded.

DATA COLLECTION

Data were collected in an electronic spreadsheet and the following parameters were recorded: patient demographics including age and sex, diving experience, risk factors for DCS including dive time, maximum depth, rapid ascent, omitted decompression obligation, dehydration, thermal comfort, exertion during the dive, and repetitive diving within 24 h. Also recorded were breathing gas and delivery system used, plus DCS symptomatology including time from surfacing to onset of symptoms, vestibulocochlear symptoms reported, and associated DCS symptoms. Data regarding treatment and outcomes including time to first HBOT, initial treatment table used, total number HBOT and outcome following treatment including residual symptoms at discharge and follow-up were collected. Data were also collected regarding recommendations made for investigation of a right-to-left shunt, with results of these tests documented where available.

STATISTICAL ANALYSIS

Data were compiled from a manual review of FSH HMU electronic patient records, stored in MS Excel® and analysed using SAS version 9.4 (SAS, Cary NC, USA). One patient was treated twice for IEDCS, two years apart, and was counted separately (weighting per case = 1) when describing number of treatments received and dive conditions, but counted just once (given a weighting of 0.5 per IEDCS) when describing age, proportion male, and other demographic factors. Frequency counts are reported with percentages. Weighted age and unweighted dive time in minutes (min), maximum depth in metres (m), time to symptom onset from surfacing (min) and delay to recompression (h) are each reported as medians with interquartile ranges (IQR).

Results

There were 23 IEDCS patients, comprising 18% of patients treated with HBOT for DCS over the study period, and 24

cases of IEDCS included for analysis. A flow diagram of patient record identification is shown in Figure 1. Of the 23 patients, 21 (91%) were male, with median age 44 years (IQR 15).

Seven (30%) divers reported having less than 100 dives experience, another seven (30%) between 100–500 dives, and nine (39%) > 500 dives. Three (13%) divers reported previous DCS (one musculoskeletal treated with HBOT with details of the other two not documented). During the 24 dives that resulted in IEDCS, 20 (83%) divers were breathing air, three (13%) enriched air nitrox (32% oxygen (O_2)) and one was using a rebreather. There were 22 (92%) divers using scuba and two (8%) were using surface supply. Median dive time was 40 min (IQR 11) and median maximum depth was 24.5 m (IQR 10).

Substantial exertion underwater and being underweighted were reported by two (8%) divers, whilst being cold during the dive was reported by one (4%). One diver self-reported dehydration and drinking alcohol to excess on the evening prior to the incident dive. Nineteen (79%) were making repetitive dives within 24 h. Four (17%) reported a rapid ascent and four (17%) reported omitting a decompression obligation. Half the divers reported making a safety stop and half did not. Five (21%) divers reported substantial exertion after the dive.

Median delay to symptom onset after surfacing was 20 min (IQR 41) and median delay between surfacing and treatment with HBOT was 22 h (IQR 59.5). Eleven (46%) cases were treated initially at our HMU, while 50% of patients were given first-aid at another hospital prior to transfer, and one (4%) patient had received prior initial recompression in Bali, Indonesia.

Of the 24 IEDCS cases, 21 (88%) showed vestibular involvement and nine (38%) cochlear involvement, with six (25%) having both vestibular and cochlear symptoms. Other manifestations of DCS were present in 18 (75%), which included constitutional (n = 13, 54%), musculoskeletal (n = 9, 38%), neurological (n = 6, 25%), cutaneous (n = 5, 21%), and spinal (n = 1, 4%). Hearing tests were documented for 11 (46%) patients and three (13%) were prescribed corticosteroids for hearing loss. The initial HBOT table used was the United States Navy Treatment Table 6 (USN TT6) for 23 (96%) patients, with one (4%) receiving a 18:90:60 treatment table (284 kPa / 2.8 atmospheres absolute for 90 min with a 60 min decompression). The total number of HBOT received ranged from 1–15 (median 6, IQR 6.5), with 10 (42%) cases reporting residual symptoms after their final treatment. Adjunctive therapy with intravenous (IV) fluids was administered in 17 (71%) cases. Of the 23 patients, 13 (57%) were advised to undergo bubble contrast transthoracic echocardiography to investigate for a right-to-left shunt, with 10 (77%) having this performed. This confirmed the presence of a persistent foramen ovale (PFO) and hence an intracardiac shunt in six (60%) of the 10 tested patients. No intrapulmonary shunts were identified. Table 1 shows patient and incident dive demographics, symptoms, treatment administered and outcomes.

Discussion

The aims of this study were to describe the epidemiology and risk factors for IEDCS cases treated at FSH HMU and to describe their treatment outcomes. Patients with IEDCS comprised 18% of divers treated for DCS during the study period. This is similar to other case series, which have reported the incidence of IEDCS to be between 16% and 24% of treated DCS cases.[4,5] A study from Malta has shown that the incidence of IEDCS had increased between 1987–2017, with the cause for this hypothesised to be due to changes in the practice of recreational diving, with more divers diving deeper, repetitively, and with reverse profiles.[12]

The median maximum depth of 24.5 m for the incident dive is shallower than previously reported case series, yet the median dive time of 40 min is similar.[4] This may reflect a difference in recreational diving practices or the coastal topography in Western Australia. Two patients had symptom onset after 10 m incident dives, without a history of rapid ascent to suggest cerebral artery gas embolism. One made a repetitive dive with significant physical exertion at depth and multiple freedives during the preceeding surface interval, with subsequent bubble contrast echocardiography negative for a PFO. Another presented with unilateral sensorineural hearing loss with tinnitus, without clinical evidence of peri-lymph fistula upon otolaryngologist review. Right-to-left shunt testing was not performed in this patient. The vast majority of divers were breathing scuba air, with three using nitrox and one using a rebreather. This supports the hypothesis that IEDCS occurs via mechanisms other than supersaturation caused by gas switching, as occurs in deep technical diving.[1]

The most common risk factor for IEDCS in this study was repetitive diving within 24 h, with 19 (79%) of divers reporting this. This is consistent with recent case series where predisposing factors related to inert gas uptake, or on-gassing, were found to be the predominant risk factors for IEDCS.[5,12]

The kinetics of inert gas in the inner ear has been described through the use of a three compartment model.[2] The inner ear comprises the membranous labyrinth, the perilymph and the endolymph. Only the membranous labyrinth has a vascular supply, and hence is the location for inert gas washout. It has been described that there is a prolonged period of supersaturation of inert gas in the membranous labyrinth immediately after surfacing from a dive, when compared with brain tissue.[13] This suggests how bubbles,

Table 1

Patient demographics, incident dive characteristics, symptomology, treatment and outcomes; *one diver was treated twice during the study period; C – cochlear; DCS – decompression sickness; F – female; HBOT – hyperbaric oxygen treatment; M – male; NP – not performed; PFO – persistent (patent) foramen ovale; V – vestibular; VC – vestibulocochlear

Age	Sex	Dive time (min)	Maximum depth (m)	Symptom onset from surfacing (min)	Vestibulocochlear symptoms	Other DCS symptoms	Delay to recompression (h)	Total number HBOT	Residual symptoms at HBOT completion	Result of transthoracic bubble contrast echocardiography
25	F	60	18	30	V	Yes	88	2	Nil	NP
27	M	44	30	15	V	Yes	20	7	Ataxia	PFO
27	M	40	12	240	V	Yes	24	3	Nil	NP
30	M	46	32	5	V	Yes	5	8	Nil	NP
34	M	42	15	120	C	Yes	28	2	Nil	NP
36	M	60	10	0	V	Yes	216	2	Nil	Normal
39	M	20	28	0	V	Yes	7	3	Musculoskeletal	NP
41	M	43	10	1140	C	No	90	10	Nil	NP
41	M	29	37	3	VC	Yes	11	15	Tinnitus	NP
42	M	125	18	90	V	No	9	10	Ataxia	PFO
43	M	37	28	60	V	No	9	8	Nil	PFO
44	M	58	19	0	V	Yes	24	2	Nil	NP
44	M	35	30	15	VC	No	8	6	SNHL	NP
45	M	36	20	20	V	Yes	20	2	Nil	PFO
47	M	15	24	15	VC	No	164	7	SNHL	NP
48	M	60	28	30	V	Yes	4	6	Nil	PFO
50	F	35	18	240	V	Yes	48	2	Nil	NP
51	M	40	25	30	V	Yes	5	7	Ataxia	Normal
52	M	?	62	0	VC	Yes	115	9	Nil	Normal
55	M	40	15	0	C	Yes	192	1	Headache	NP
55	M	40	20	20	V	No	20	10	Nil	Normal
70 *	M	25	25	10	V	Yes	30	3	Nil	PFO
72 *	M	25	25	30	VC	Yes	6	6	Ataxia, tinnitus	PFO
72	M	45	25	20	VC	Yes	30	9	Ataxia	NP

entering the arterial circulation through a PFO, can grow in the membranous labyrinth due to the localised increased partial pressure of gas. This is postulated to be a mechanism by which isolated IEDCS can occur.

The second most common risk factor for IEDCS was exertion and heavy lifting after the dive, present in five (21%) divers, with three of those being recommended to have bubble contrast echocardiography. Of those, two were tested, confirming the presence of a PFO in both. Increased right atrial pressure, as occurs after the release phase of the Valsalva manoeuvre, or after heavy lifting with a closed glottis, may shunt blood from the venous to the arterial circulation in those with a PFO.[14] It has been shown that bubbles may be detected in the venous system after surfacing from a dive, even when diving within no-decompression limits and they are predominantly filtered by the lungs.[15] When the bubbles become arterialised through a PFO, divers are at increased risk of certain subtypes of DCS, including cerebral, spinal, cutaneous and inner ear.[16,17]

A safety stop was not documented in half of the cases reviewed, which may be a contributory risk factor for developing DCS, however there could be limitations in documentation. Nevertheless, the performance of a safety stop when diving is recommended for reducing the risk of DCS and this may reflect the need for improving diver education. More pertinent to the risk of developing DCS was the omission of a decompression obligation. This was present in four (17%) cases, increasing the risk of bubble formation in this cohort. The time from surfacing to onset of symptoms varied from zero to 1,140 min, with a median of 20 min (IQR 41). This is similar to previously reported onset times for IEDCS.[4] Symptoms developed within two hours of surfacing in 88% of divers, with two divers developing symptoms at four hours and one patient developing isolated severe unilateral sensorineural hearing loss with tinnitus at 19 h. This outlier made a complete recovery with normal hearing thresholds on audiometry after 10 HBOT sessions with adjunctive oral corticosteroids.

The majority (88%) of patients had vestibular symptoms, with fewer (38%) showing cochlear involvement, which is a consistent finding in other reports.[4,5] The predisposition for vestibular symptoms is thought to be due to the vestibular apparatus having a quarter of the blood flow than that of the cochlea with an increased tissue volume, causing slower gas washout and prolonged supersaturation and therefore propensity to bubble growth within the vestibule.[7] Isolated IEDCS was present in 25% of patients, while the majority (75%) had other systems involved. This trend is similar to findings from a recent report.[5] The predominant associated DCS symptoms were constitutional (54%), followed by musculoskeletal (38%), neurological (25%), cutaneous (21%), and spinal (4%).

The time to first HBOT varied from four to 216 h (median 22, IQR 59.5). The median time to recompression was longer than in previous studies of IEDCS.[4] This could be due to the vastness of WA and distances involved in transporting injured divers for treatment in Perth, where our HMU has the only hyperbaric chambers in WA available for civilian use. Twelve patients were recompressed within 24 h, with seven (58%) of those having ongoing symptoms at completion of HBOT. Twelve patients had a delay to recompression of greater than or equal to 24 h, however only three (25%) of these had ongoing symptoms at discharge. Patients who presented earlier could have been sicker, which could account for the increase in residual symptoms seen in this cohort. However, our numbers are small and a recent study found that patients with decompression illness that present with tinnitus or hearing loss have worse outcomes with increased residual symptoms when recompressed beyond 48 h.[18]

Thirteen patients initially presented to healthcare facilities other than our own. Two of these were outside of Australia; in Bali Indonesia where HBOT was administered to one patient, and in Fiji where normobaric O_2 was given, with both of these patients flying on standard commercial flights to Perth where they were subsequently reviewed. Of the 11 patients who presented to other facilities within WA, all were given normobaric O_2 and IV fluids at the initial receiving facility. Five of these arrived to our HMU by private vehicle, with distances driven ranging from 160 km (Bunbury, WA) to 1,200 km (Exmouth, WA). Four were transferred by road ambulance with ongoing normobaric O_2 and IV fluids, of which three were from local metropolitan hospitals and one was from Bunbury, WA. Two patients were transported by fixed wing air ambulance, by the Royal Flying Doctor Service, in cabins pressurised to sea level, with face mask O_2 and IV fluids during transfer. Decisions regarding the transport method to our unit depend on clinical stability, response to first aid, distance involved and the capacity of retrieval services.

The United States Navy Table 6 was the initial treatment table for all but one patient, who received treatment table 18:90:60. This patient had been diving in Fiji three days prior to presentation to our HMU and developed a rash 30 min after surfacing, with associated limb pain and headache, which was treated there as a presumed allergic reaction with corticosteroids and antihistamines. A few hours later they subsequently developed worsening musculoskeletal pain with vestibular symptoms, which fully resolved after two HBOT sessions upon return to Perth. They were advised to have investigation for a PFO, however they had not had this performed upon follow-up.

Residual symptoms were present in 10 (42%) patients at discharge, with the majority (58%) being symptom free post-HBOT. Ongoing symptoms were isolated vestibular for

four patients, isolated cochlear for three, vestibulocochlear for one and other DCS manifestations for two (neurological and musculoskeletal). This demonstrates that IEDCS may be refractory to treatment and symptoms may persist despite treatment. Of the 10 with ongoing symptoms after completion of HBOT, documentation of subsequent follow-up was available for four patients. Two reported persistent tinnitus, one had ongoing severe sensorineural hearing loss, and one patient had complete resolution of shoulder pain. The long-term consequences of IEDCS could not be fully explored due to limitations in the data, however we have shown that a select group of patients had ongoing deficits to both the vestibular and cochlear systems.

A previous study on IEDCS reported ongoing vestibulocochlear deficits upon follow-up in 91% of patients.[19] Of eight patients with ongoing vestibular deficits, only one (12.5%) was symptomatic. This could be explained by the more extensive otoneurologic assessment used in that study, which included electronystagmography, alternate bi-thermal caloric testing, sinusoidal harmonic acceleration testing and computerised dynamic posturography. It is important to consider the implications regarding return to diving for patients with a history of vestibular IEDCS, as a high proportion may have ongoing underlying vestibular dysfunction despite apparent resolution of symptoms. An unfamiliar underwater environment could overwhelm the brain's compensatory mechanisms that develop after an insult to the vestibular system with potentially serious consequences.[19] A more recent study found 69% of patients recovered completely after treatment for IEDCS, which is more reflective of our cohort.[5] A thorough assessment and follow-up is recommended for all patients presenting with IEDCS, as ongoing deficits can be subtle, with audiometry a minimum for testing on discharge.

Investigation for a right-to-left shunt with bubble contrast echocardiography was advised for 13 patients, of which 10 had the investigation, with six (60%) of these being positive. This supports the hypothesis that IEDCS is associated with a right-to-left shunt as the prevalence of a PFO in the general population is approximately 25%.[20] The reason for not investigating the remaining 10 patients with bubble contrast echocardiography was not documented. Once bubbles pass into the arterial circulation, they can cause damage to the inner ear through a number of mechanisms. The vestibular vascular supply is an end-artery, thus occlusion or endothelial bubble stripping can cause localised inflammation and ischaemia.[21] Bubbles can increase in size due to localised supersaturation of the inner ear, causing selective vulnerability of the inner ear to DCS in those with a right-to-left shunt.[13] Due to the increased risk of certain subtypes of DCS in those with a PFO, it is advised to consider PFO testing with trans-thoracic bubble contrast echocardiography, including provocative testing with Valsalva and sniffing, for patients presenting with IEDCS, as per the South Pacific Underwater Medicine Society and United Kingdom Sports Diving Medical Committee joint position statement.[22]

A disproportionate number (91%) of the patients in this IEDCS study were male, although our dataset is too small to infer an association between gender and IEDCS. Historically, there have been three surveys of recreational divers in WA; the first in 2000 (n = 540) found 72% of divers were male, the second in 2006 (n = 499) 75%, and the third in 2014 (n = 139) 73%.[23–25] Of 24 WA recreational diving fatalities described in 2009, 16 (75%) were male.[26] Of the 83 divers treated for decompression illness at our HMU reported in 2020, 80% were male.[27] Much larger numbers would be needed to determine whether any gender related pre-disposition exists.

Two patients were excluded from the study due to signs of middle ear barotrauma (MEBt), suggesting a likely alternate diagnosis of IEBt, however no patients reported difficulty equalising. A case series of 50 patients with IEBt found 38% had no evidence of MEBt at otoscopy, hence the absence of erythema of the tympanic membrane does not exclude IEBt.[28] Features suggestive of IEBt include vestibulocochlear symptom onset during ear clearing manoeuvres or straining, a previous history of aural barotrauma and co-existent MEBt. IEDCS is increasingly likely in divers with a provocative dive profile, history of rapid ascent, omitted decompression obligation, coexistent symptoms of DCS or when symptoms occur after surfacing. Several investigations have been used to investigate suspected cases of IEBt including pure tone audiometry (+/- positional testing), the fistula test, high resolution temporal bone computed tomography scanning, electronystagmography, caloric testing, the Tulio phenomenon and otoacoustic emission testing.[10] These tests may be useful in helping differentiate the cause of vestibulocochlear symptoms in divers who present a diagnostic dilemma to the clinician.

LIMITATIONS

The study design is retrospective and reliant upon data collected from the medical records. Some data were missing and the diagnosis of IEDCS relied on assessments that had been documented for the purpose of treatment, not research. Also, dive profile computer downloads were not available so maximum tissue super-saturation pressures could not be factored in, nor ascent rates. Information on vestibular testing and hearing tests were limited. Long-term outcomes were difficult to assess given follow-up was documented for a minority (43%) of patients. Ten patients were not documented to have been recommended a PFO test, although guidelines suggest that all patients with IEDCS should have this considered, or else modify their diving practices.[22] This could be due to limitations in the documentation, however a new assessment form for divers is being developed in

our HMU to more accurately record details of clinical assessment, dive demographics, risk factors for DCS and follow-up.

Conclusions

This study presents data for all IEDCS cases treated at FSH HMU from 2014 to 2020. Divers were predominantly using scuba equipment breathing air, with a median maximum depth of 24.5 m, supporting previous literature that this disease is not isolated to deep technical diving. The main risk factor was repetitive diving and the vestibular system was found to be more vulnerable to DCS than the cochlear system. It is important to perform a comprehensive otoneurologic assessment for patients presenting with potential IEDCS, and counselling and follow-up is important given the high prevalence in this cohort of a PFO and right-to-left shunt, with implications for future diving practices and safety.

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