Recurrent decompression sickness in experienced divers treated with percutaneous patent foramen ovale closure

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INTRODUCTION

Decompression sickness (DCS) is a rare diving--related disease triggered by trapping nitrogen gas emboli in vessels and tissues as the ambient pressure surrounding the body pressure rapidly decreases during ascent and surfacing [1]. DCS may present with a wide range of acute clinical manifestations, from mild to severe, including persistent paralysis or even death [2]. The reported estimated prevalence of DCS is approximately up to 1.5%, depending on the diving environment and type of diving [1, 2].

The presence of patent foramen ovale (PFO) increases the risk of venous bubble transfer to the systemic circulation and of subsequent DCS by facilitating arterial air embolization (AAE). Therefore, PFO closure may play a role in reducing these complications. However, a treatment strategy for patients with DCS and PFO has not yet been established due to limited data. The guidelines indicate that PFO investigation and its closure should be conducted only in specific cases with high-risk and frequent activities [1, 3, 4]. Moreover, there are limited studies on diving habits and DCS incidence following PFO closure. The objective of this study was to summarize our experience in the field of device PFO closure in divers with a history of recurrent DCS.

METHODS

Among all 562 transcatheter PFO closures in our department, three procedures were performed for the secondary prevention of DCS between 2007 and 2022. Detailed data on the individual diving experience and DCS symptoms were collected. Indications for PFO closure and procedural characteristics were carefully analyzed in each case. The PFO with the right-to-left shunt was diagnosed with contrast transesophageal echocardiography (TEE) and the Valsalva maneuver and confirmed during the procedure. Each procedure was performed under sedation anesthesia via femoral venous access. In 2 patients, the PFO was closed with a 25-mm Amplatzer PFO Occluder (St. Jude Medical, St. Paul, MN, US), and in one patient with a 26-mm PFO Nit-Occlud device (PFM, Cologne, Germany). A telephone questionnaire was used to collect information about the current diving activities. The divers were asked if they were still diving and if they had changed their diving profile concentrating on the reduction of venous bubble load (restrictions on the depth of the dive, precise adherence to decompression guidelines, no repetitive dives during a single-day, reduced rate of ascent, use of nitrox), and if they had suffered any diving-related problems such as DCS. The study was approved by the local bioethics committee (approval no. 187/2016).

RESULTS AND DISCUSSION

We included one female and two male professional divers with at a median age of 45 (range 43–52) years on the procedure day. None of the patients had any concomitant chronic disease. Each patient experienced at least three episodes of DCS, with the symptom onset from half an hour to several hours after surfacing. All patients had cutaneous and musculoskeletal manifestations of DCS (Supplementary material, *Figure S1*), and the two male patients had also simultaneous neurological symptoms. Both patients had complete resolution of neurological symptoms without ischemic foci in neuroimaging.

There were no complications associated with the PFO closure. A follow-up assessment showed no residual shunts, device dislocation,

Table 1. Patients, PFO procedure, and follow-up characteristics

Characteristics	Patient 1	Patient 2	Patient 3
Sex	Female	Male	Male
Age, years	45	52	43
3MI, kg/m²	23.6	28.3	22.5
Co-morbidities	Migraine headache	None	None
Drugs taken chronically	None	None	None
Jsage of stimulants	No	No	No
Vicotinism	No	No	No
ype of diving (recreational or professional)	Professional	Professional/ currently recreational	Professional
lumber of DCS events before the intervention	3	3	4
lumber of dives before the intervention	800	2000	1600
Nax depth of diving before the intervention meters of seawater)	103	130	139
Nixture of breathing gas used during dives	Nitrox 28, 32, 36, 50 Trimix 18/35, 15/55, 12/60, 10/70 Pure oxygen during decom-	Nitrox 28, 32, 36 Trimix 18/35, 15/55, 12/60, 10/70 Heliox Pure oxygen during decompression	Nitrox 32, Pure oxygen during decompres sion
Symptoms of DCS (musculoskeletal, skin bends, cutis marmorata, neurological, vestibular, nemisensory loss, paresis, vomiting, visual disturbances)	pression musculoskeletal, skin bends, cutis marmorata	musculoskeletal, skin bends, cutis marmorata, visual disturbances and hemisensory loss, vestibular	musculoskeletal, skin bends, cutis marmorata, neurological, vomiting, visual disturbances, hemisensory loss
ime from diving to symptoms onset (hours)	1–2	1–2	up to half an hour
lyperbaric Oxygen Treatment of DCS (session number and duration)	After 2 nd DCS 1 session, 1.5 h	No	After 2 nd DCS 5 sessions, 1.5 h
schemia confirmed by neuroimaging	None	None	None
ime from DCS event/events to PFO diagnosis months)	1 st DCS – 11 3 rd DCS – 1	1^{st} DCS – 24 2^{nd} and 3^{rd} DCS 3 – several weeks	1 st DCS – 2 3 rd DCS – 1 week
Fime from DCS event/events to procedure months)	1 st DCS – 28 2 nd DCS – 18 3 rd DCS – 5	1^{st} DCS – 24 2^{nd} and 3^{rd} DCS – 2	$1^{st}DCS - 2$ 2^{nd} and $3^{rd}DCS - 1$
ype of implanted Occluder	Amplatzer PFO Occluder	Amplatzer PFO Occluder	PFO PFM Nit-Occlud
ize of implanted Occluder (mm)	25	25	26
ostprocedural thromboprophylaxis time months)	6	6	6
Prugs used for thromboprophylaxis	Aspirin+clopidogrel	Aspirin+clopidogrel	Asprin +clopidogrel
Complications after PFO closure (bleeding, amponade, occluder dislocation, occluder hrombosis, stroke)	None	None	None
24-hour follow-up echocardiography after :losure (residual shunt/no residual shunt)	No residual shunt	No residual shunt	No residual shunt
i-12 month follow-up echocardiography fter closure (residual shunt/no residual shunt)	No residual shunt	No residual shunt	No residual shunt
ime from the intervention to the next diving, months)	4	б	6
lumber of dives after the intervention	20	500	1600
lumber of dives per year	50	250	200
Nax depth of diving after the intervention meters of seawater)	65	90	130
lumber of DCS events after the intervention	0	0	0
Fime of follow-up from PFO closure to phone call (months)	12	58	96

Abbreviations: BMI, body mass index; DCS, decompression sickness; PFO, patent foramen ovale

or embolization. The median follow-up was 58 (range 12–96) months. All three patients continued diving. However, two patients performed shallower dives than before the procedure. None of the divers experienced a DCS recurrence after PFO closure. Details are presented in Table 1.

This study included only professional divers who followed safe decompression rules and were willing to continue diving. It was demonstrated previously that right-to-left shunt and a lack of changes in the way of diving after prior DCS were the only predictors of DCS recurrence, especially with regard to neurological manifestation [5]. In our group, each patient experienced at least three DCS episodes, but not after PFO closure. However, in qualification for PFO closure after DCS, potential complications of PFO closure must be considered, even though their occurrence is generally low (<2%) [6, 7]. In our study, there were no complications related to device implantation.

Our results demonstrated that PFO closure seems to be a feasible approach for the secondary prevention of DCS. Similar findings were shown by the DIVE-PFO registry, indicating catheter-based PFO closure as more effective in DCS prevention than conservative dive profile in divers with a high-grade PFO [8].

Moreover, PFO closure apart from decreasing the likelihood of DCS, may also alleviate such conditions as migraine with aura or cryptogenic stroke. Shunt closure will not prevent DCS caused by other mechanisms, including AAE resulting from pulmonary barotrauma or a provocative diving profile (rapid ascent or missed decompression stops) [2].

Our patient cohort was small, and the number of dives made calculations of DCS risk recurrence unreliable. Nonetheless, there is a paucity of data on these findings and our results could be important in future recommendations regarding diving for patients with a closed PFO.

Supplementary material

Supplementary material is available at https://journals. viamedica.pl/kardiologia_polska.

Article information

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REFERENCES

- Pristipino C, Germonpré P, Toni D, et al. European position paper on the management of patients with patent foramen ovale. Part II - Decompression sickness, migraine, arterial deoxygenation syndromes and select high-risk clinical conditions. EuroIntervention. 2021; 17(5): e367–e375, doi: 10.4244/EIJ-D-20-00785, indexed in Pubmed: 33506796.
- Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Engl J Med. 2022; 386(13): 1254–1264, doi: 10.1056/NE-JMra2116554, indexed in Pubmed: 35353963.
- Henzel J, Rudziński PN, Kłopotowski M, et al. Transcatheter closure of patent foramen ovale for the secondary prevention of decompression illness in professional divers: a single-centre experience with long-term follow-up. Kardiol Pol. 2018; 76(1): 153–157, doi: 10.5603/KP.a2017.0182, indexed in Pubmed: 28980295.
- Araszkiewicz A, Bartuś S, Demkow M, et al. Interventional closure of patent foramen ovale in prevention of thromboembolic events. Consensus document of the Association of Cardiovascular Interventions and the Section of Grownup Congenital Heart Disease of the Polish Cardiac Society. Kardiol Pol. 2019; 77(11): 1094–1105, doi: 10.33963/KP.15058, indexed in Pubmed: 31723115.
- Gempp E, Louge P, Blatteau JE, et al. Risks factors for recurrent neurological decompression sickness in recreational divers: a case-control study. J Sports Med Phys Fitness. 2012; 52(5): 530–536, indexed in Pubmed: 22976740.
- Wintzer-Wehekind J, Alperi A, Houde C, et al. Long-Term follow-up after closure of patent foramen ovale in patients with cryptogenic embolism. J Am Coll Cardiol. 2019; 73(3): 278–287, doi: 10.1016/j.jacc.2018.10.061, indexed in Pubmed: 30678757.
- Pristipino C, Filice FB. Long-term benefits and risks in patients after persistent foramen ovale closure: a contemporary approach to guide clinical decision making. Kardiol Pol. 2021; 79(3): 248–254, doi: 10.33963/KP.15817, indexed in Pubmed: 33599457.
- Honěk J, Šrámek M, Honěk T, et al. Patent foramen ovale closure is effective in Divers: long-term results from the DIVE-PFO registry. J Am Coll Cardiol. 2020; 76(9): 1149–1150, doi: 10.1016/j.jacc.2020.06.072, indexed in Pubmed: 32854848.