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CASE REPORT

ADVANCED

CLINICAL CASE

Recurrent Decompression Illness Even After the Closure of Patent Foramen Ovale in a Diver

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ABSTRACT

Patent foramen ovale (PFO) is a risk factor for the development of decompression illness (DCI) and a therapeutic target for preventing the recurrence of DCI because nitrogen bubbles generated during diving can be paradoxically embolized through the PFO. Here, we report the case of a diver who experienced recurrent DCI even after a successful PFO closure. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2023;5:101687) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 41-year-old man, a diving instructor using a self-contained underwater breathing apparatus (SCUBA) presented with arthralgia, headache, and a pruritic rash in both feet immediately after diving. He had experienced 4 episodes of decompression illness (DCI) after recreational diving during the 14 years before his clinic visit and had received hyperbaric oxygen chamber therapy for 2 episodes. The patient was a smoker and had dyslipidemia. His blood pressure and heart rates were 139/85 mm Hg and 81 beats/min, respectively. There were no abnormal neurologic signs or specific findings on physical examination. He had visited our diver clinic 3 months earlier for the evaluation of his patent foramen ovale (PFO). A transesophageal echocardiogram (TEE) with a saline bubble test showed a high-risk, grade 3 PFO with a hypermobile interatrial septum (Video 1). We performed a PFO closure to prevent future DCI events resulting from arterial embolization of venous nitrogen bubbles that occur during ascent in the course of SCUBA diving. With the patient under general anesthesia, the right femoral vein was punctured. A pulmonary angiogram showed no remarkable arteriovenous fistula (Video 2). The 0.35-inch hydrophilic wire and 5-F multipurpose catheter easily crossed the PFO channel. The diameter of the PFO channel was 11 mm by echocardiographic measurement while the sizing balloon was inflated (Video 3). Without vascular complications, a 25-mm Amplatzer PFO occluder was implanted into the PFO channel (**Figure 1**). Six months after PFO closure, a follow-up TEE examination using saline bubbles showed that

LEARNING OBJECTIVES

- To understand 3 major pathophysiologic features involved in the development of PFO-related DCI in the underwater environment.
- To counsel patients who have had DCI on the treatment strategies to prevent a recurrence.

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ABBREVIATIONS AND ACRONYMS

DCI = decompression illness

DCS = decompression sickness

LA = left atrium

PFO = patent foramen ovale

RA = right atrium

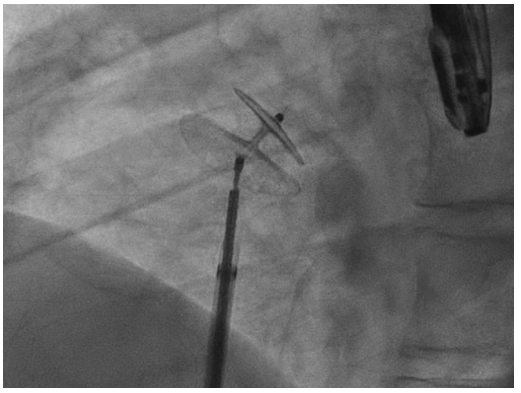
RAP = right atrial pressure

SCUBA = self-contained underwater breathing apparatus

the PFO occluder was well seated in the interatrial septum without a peridevice thrombus.

However, >20 saline bubbles still crossed the remnant PFO channel during the Valsalva maneuver (Video 4). We advised him to refrain from diving, but he continued doing so against our advice. After 15 consecutive recreational dives for 4 days (8 months after PFO closure), he experienced recurrent DCI symptoms such as headache, painful sensation in the hands and feet, and skin rash and pruritus in both feet (**Figure 2**). During the physical examination, several round rashes with itching were observed on the sole of his left foot. Small pruritic rashes with mild tenderness were also observed on the dorsum of his left second and third toes. There were no significant findings on neurologic examination. On his last dives, he frequently descended and ascended for underwater photography. He also did not follow the safety stop at 5 meters. His symptoms were immediately resolved by hyperbaric oxygen chamber therapy.

FIGURE 1 Successful Implantation of PFO Occluder



A 25-mm PFO occluder was implanted into a patent foramen ovale (PFO) channel without vascular complications under transesophageal echocardiogram guidance.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for the cause of recurrent DCI included the following: incomplete closure of the PFO owing to insufficient endothelialization around the closure device, DCS resulting from excessive nitrogen bubble expansion at the tissue level, and arterial gas embolism through the pulmonary circulation.

INVESTIGATIONS

We performed bilateral pulmonary angiography again. Selective angiography of each lobar artery showed no arteriovenous malformation (Video 5). Fifteen months after PFO closure, we performed transthoracic echocardiography (TTE) after nonprovocative boat diving around the beach to evaluate the real risk for the development of DCI after diving. He dove for 28 minutes at a water temperature of 14 °C and a maximum depth of 34 meters. It took him 24 minutes to undergo the TTE examination after surfacing on the boat. The TTE revealed that multiple venous nitrogen bubbles were released from the tissue, which entered the right atrium (RA) and right ventricle. On release of abdominal strain to increase the right atrial pressure (RAP), venous nitrogen bubbles in the RA did not move to the LA (Video 6).

MANAGEMENT

The patient was advised to refrain from diving, but given his determination to keep diving, shared decision making was implemented. We recommended that he dive after thoroughly following a conservative protocol with a safety stop at a depth of 5 meters to allow release of excess nitrogen accumulated in the tissues and blood out of the body while resurfacing. The recommendations were as follows: 1) diving with a buddy diver or group at the top; 2) ascent speed not exceeding 30 feet/min; 3) avoiding diving near no-decompression limits without diving for more than >30 meters; and 4) taking a day off from diving during a multiday diving trip. We specifically advised him not to descend again and perform the Valsalva maneuver while ascending after diving.

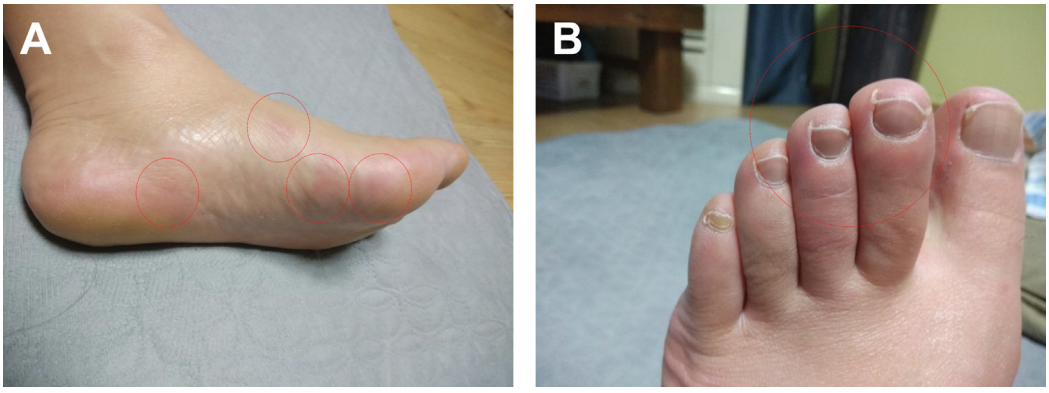
FOLLOW-UP

Two years after the PFO closure, a follow-up TEE examination showed that saline bubbles no longer crossed the interatrial septum during the Valsalva maneuver (Video 7). The patient has not reported DCI symptoms in at least a year.

DISCUSSION

DCS is a condition in which inhaled nitrogen is dissolved in the tissues or blood under high pressure during a diver's descent and forms gas bubbles while they rise, mechanically affecting tissue or inhibiting blood flow. Arterial gas embolism refers to the introduction of alveolar or venous gas emboli via cardiac shunts or pulmonary vessels into the arterial circulation. Because it may be difficult to clinically differentiate DCS from an arterial gas embolism in divers and the treatment protocols for the 2 disorders

FIGURE 2 **Pruritic Rash on Left Foot After Diving**



Immediately after diving, the patient experienced a pruritic rash on the medial aspect of his left sole **(A)** and the dorsum of the second and third toes **(B)**.

are the same, the term "decompression illness" is sometimes used to indicate DCS, arterial gas embolism, or both.[1] The following 3 conditions must occur simultaneously before the development of PFO-related DCI after SCUBA diving: 1) moderate to large PFO sizes; 2) venous nitrogen bubbles during ascent or surfacing after diving; and 3) timely embolization and subsequent passage of venous nitrogen bubbles while the PFO is opened. This is a major reason for the low incidence of PFO-related DCI compared with the high prevalence of PFO.[2] Arterial embolization of venous nitrogen bubbles was reported in divers without PFO even when the number of venous nitrogen bubbles overwhelmed the ability of the pulmonary circuit to trap and eliminate them.[3] However, this is not the main mechanism of arterial embolization of venous nitrogen bubbles. Honěk et al[4] compared the occurrence of arterial bubbles after 2 simulated dives in divers with PFO versus those treated with a catheter-based PFO closure. The arterial bubbles were observed in 32% of divers after the 18-meter dive and in 88% after the 50-meter dive in the PFO group. By contrast, no arterial bubbles were detected in the closure group. Therefore, those authors suggested that PFO plays a key role in the paradoxical embolization of venous nitrogen bubbles after SCUBA diving. Previous retrospective studies have reported a higher prevalence of PFO in divers who experienced DCI than in those who did not.[2,5] However, the relationship between PFO and DCI remains uncertain because there have been no well-designed prospective studies with objective adjudication of DCI-like symptoms to demonstrate the causal inference between them. Catheter-based PFO closure has been performed in divers with high risk for PFO who have experienced DCI since the late 1990s;[6] however, no randomized trial has demonstrated the efficacy of PFO closure on the secondary prevention of DCI. This case showed that even if PFO closure is successfully performed in a diver with DCI, the PFO channel may be patent for a considerable period, and unconstrained diving during this period can lead to DCS. Moon et al[7] reported that residual shunts were present in 26% of patients with PFO closure to prevent cryptogenic stroke 9 months after the procedure. Divers must perform the Valsalva maneuver to equalize the middle-ear pressure to the ambient pressure during the descent. It should be repeated approximately every 2 to 3 feet. We hypothesize that the repeated stretching of the connective tissue around a PFO delays the complete endothelization of the closure device in divers. A consensus statement from the South Pacific Underwater Medicine Society and the United Kingdom Sports Diving Medical Committee recommends that divers require a repeated bubble contrast echocardiogram demonstrating complete blockage of the right-to-left-shunt before returning to diving, a minimum of 3 months after the closure.[8] Even after a complete PFO closure, other mechanisms, such as DCS resulting from the excessive expansion of nitrogen bubbles at the tissue level or arterial gas embolism through the pulmonary circulation, can cause DCI. Therefore, it is necessary to adhere to a conservative protocol that minimizes the generation of venous nitrogen bubbles to prevent recurrent DCI in

divers with PFO closure because bubble formation is the primary process in all DCI types. Valsalva maneuvers, abdominal strain, sneezing, and prolonged breath-holding can increase RAP and open the PFO in divers. Even getting on a boat with heavy diving gear would increase the RAP of highly susceptible divers, such as those having a PFO with a hypermobile interatrial septum or aneurysm. It is also important to educate divers on preventing an increase in RAP so that venous nitrogen bubbles entering the RA will not pass to the LA while the divers ascend.

CONCLUSIONS

This case indicates that the risk of DCI may remain even after PFO closure. The incomplete closure of PFO due to delayed endothelialization around the device, DCS, and arterial gas embolism through the pulmonary circulation should be considered. Divers should be educated on maintaining a conservative diving protocol to decrease nitrogen gas load, and avoiding some measures to promote arterial embolization of nitrogen bubbles by increasing RAP during ascent.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS decompression sickness, diving, patent foramen ovale

APPENDIX For supplemental videos, please see the online version of this paper.