Case reports

Patent foramen ovale in scuba divers. A report of two cases and a brief review of the literature

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Scuba diving (diving with a self-contained underwater breathing apparatus) has become a popular sport. Decompression illness may be due to the formation of gas bubbles in various body tissues at an increased ambient pressure. The gas can pass from the systemic venous circulation into the arterial circulation as a result of either pulmonary barotrauma or intravascular shunting. Gas emboli may be the cause of an increased prevalence of brain lesions in sport divers.

The management of scuba divers (professionals and amateurs) with a patent foramen ovale is not clear.

We present the cases of 2 subjects with decompression illness and a patent foramen ovale and briefly review the literature on this combination.

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Introduction

Scuba diving (diving with a self-contained underwater breathing apparatus) has become a popular sport. There are about 3 million certified sport divers in the United States and about 1 million in Europe.

Most of the medical health problems in these athletes are due to decompression during the ascent of the diver. Decompression illness (DCI) (neurological injuries, inner ear DCI, and a widespread skin rash called cutaneous DCI) is suggestive of disorders that are consequent to gas bubble formation. These gas bubbles are thought to develop by expansion of preexisting gas nuclei found, at normal atmospheric pressure, in joints, the spine, sweat glands, and skin pores. These bubbles, if large enough, may cause pulmonary barotraumas and may escape into the systemic circulation through pulmonary arterio-venous shunts. Gas bubbles may also reach the systemic arterial side through a patent foramen ovale (PFO). Animal studies simulating deep dives yielding a high venous nitrogen bubble load showed that patency of the foramen ovale may be the cause of paradoxical arterial nitrogen bubble emboli, and thus of DCI[1,2].

DCI may occur even after uneventful dives, for which the diver has not committed any (reported) error in the standard accepted decompression procedures.

We report the cases of 2 patients with a history of DCI who were found to have a PFO and we review the literature on the management of such patients.

Description of cases

Case 1. A 39-year-old scuba diver (weight 72 kg, height 168 cm) was referred to our institution after two episodes of musculocutaneous DCI 15 min after surfacing from a dive (maximum 75 m in 20 min). The patient underwent clinical and transesophageal echocardiographic assessment with contrast medium; a PFO with right-to-left shunting was documented at rest and during the Valsalva maneuver.

The patient was informed and a decision was made to perform transcatheter closure of the PFO. The PFO was closed using a 23 mm StarFlex® (NMT Medical Inc., Boston, MA, USA). At the end of the procedure contrast transesophageal echocardiography was performed before and af-

ter a Valsalva maneuver, and no residual shunt was detected. The patient was discharged 24 hours after device implantation, on therapy with aspirin 325 mg/day for 6 months. He re-started diving 6 months after the procedure.

The patient underwent clinical and echocardiographic follow-ups at 1, 6, 12, and 24 months following the procedure: no thrombus was detected on the device and the patient had no further episodes of DCI during follow-up.

Case 2. A 48-year-old scuba diver (weight 75 kg, height 177 cm) with no remarkable medical or surgical history developed a transient ischemic attack after surfacing from a dive (maximum 35 m in 40 min). He had no vertigo, tinnitus, or nausea. Multiple intracranial ischemic lesions were identified at magnetic resonance imaging. The patient underwent clinical evaluation including complete screening for thrombophilic diseases; he was also submitted to contrast transesophageal echocardiography and a PFO with right-to-left shunting and a large aneurysm were documented at rest and during the Valsalva maneuver. No known causes for a thromboembolic event were found other than a PFO. The patient underwent transcatheter PFO closure using a 30 mm Cardia Intrasept device (Cardia Inc., Burnsville, MN, USA). Contrast transesophageal echocardiography was performed at the end of the procedure before and after a Valsalva maneuver and no residual shunt was detected. The patient was discharged 24 hours later on therapy with clopidogrel 85 mg for 6 months associated with aspirin 100 mg for 3 months in accordance with the specific indications of the Cardia Intrasept device.

The patient underwent clinical and echocardiographic follow-ups at 1 and 6 months after the procedure: no thrombus was detected on the device and the patient had no recurrence of transient ischemic attack during the follow-up.

Discussion

Diving carries the risk of neurological injuries and of cutaneous and embolic inner ear DCI. In case of neurological damage occurring in divers, the prime suspects are gas bubbles.

Reul et al.[3] suggested that gas emboli arising during or after surfacing are the cause of brain lesions in sport divers, but they did not address the question of how the gas emboli entered the arterial circulation.

The mechanism involved is probably related to the maneuvers used by divers to equalize the pressure in the middle ear (tympanic) cavity with the ambient hydrostatic pressure during their descent; these maneuvers may cause a similar increase in right atrial pressure and lead to permeabilization of a PFO if present. Many different maneuvers are available to achieve equalization of the pressure of the ear cavities; the most commonly used is a Valsalva maneuver. The Valsalva maneuver consists of manual blockage of the nostrils, followed by a forced expiration against the closed mouth and nose in order to increase the pressure in the nasopharyngeal cavity. Inevitably, this maneuver causes a rise in intrathoracic pressure.

There are six major steps in the Valsalva maneuver[4,5]: the initial inhalation phase, an exhalation phase, a strain phase, a releasing phase, and finally the second inhalation and exhalation phases[6]. Each phase is accompanied by changes in the airway and intrathoracic pressures. These pressure changes influence the right and left atrial pressure curves. During the deep inhalation initiating the cycle, there is a right atrial pressure predominance due to a decrease in intrathoracic pressure and an increased gradient between the extrathoracic veins and the right atrium. An increased inflow from the superior and inferior venae cavae to the right atrium, the increased filling capacity of the expanded lungs, as well as the ventricular interdependence cause a subsequent decrease in the left atrial return and pressure. During the exhalatory phase against resistance, the airway and intrathoracic pressures increase with a consequent predominance of the left atrial pressure. The increased intrathoracic pressure diminishes the systemic venous return to the heart. The peripheral venous flow first fills up the available venous capacity, which occurs at the expense of the flow through the central veins. This explains the already reported drop in right ventricular stroke volume[7,8]. De Lee et al.[7] observed that during the first few heartbeats following the release of the Valsalva maneuver, the right atrial pressure increases to levels above the pulmonary wedge pressure and therefore, presumably above the left atrial pressure.

Balestra et al.[4] suggested that the "quality" of the Valsalva maneuver during the dive is important. These authors enrolled 16 experienced divers and measured the rise and fall in intrathoracic pressure during various maneuvers. Intrathoracic pressures significantly higher than those of a standard maximal isometric effort were reached during maximal Valsalva maneuvers, whereas the "divers Valsalva maneuver" (a "gentle" Valsalva) produced intrathoracic pressures significantly lower than those of the standard maneuver ($25 \pm 6\%$, $p < 0.001$).

About 10-30% of the general population have a PFO or a right-to-left shunt as a potential route for venous bubbles to enter the arterial circulation[9-14]. This prevalence is roughly the same as that of divers with multiple brain lesions in the study by Reul et al.[3]. This led to the hypothesis most recently expressed by Wilmshurst et al.[15], that divers with a PFO may have a high risk of developing brain lesions.

Knauth et al.[16] showed that there is not only a correlation between PFO and neurological DCI, but also that PFO constitutes an important risk factor for the development of multiple brain lesions in sport divers.

These authors reported on a group of 87 divers (67 male), with a mean age of 35.7 ± 8.9 years who had performed an average of 565.3 scuba dives. A total of 41 brain lesions were detected in 11 of the 87 divers. Seven of the divers without a right-to-left shunt had 7 brain lesions (one each), while 4 of the divers with a right-to-left shunt had 34 lesions. Of these 4 divers, one, with a small PFO, had one lesion whereas the other 3 divers had multiple brain lesions and a PFO with a very significant hemodynamic relevance. The typical size of the lesions was 2-3 mm and all of them were located in the anterior cerebral circulation. The prevalence of brain lesions (single or multiple) was higher in the divers with a PFO than in those without.

These data have recently been confirmed by Torti et al.[17] who studied 230 divers and found that 27% of them had a PFO. The divers with more than 1 episode of DCI were exclusively in the PFO group. In all cases, the symptoms of major DCI began shortly before, or within 30 min of, surfacing. The only predictor of major DCI events in this study was the number of dives.

Paradoxical gas embolism could be also implied in the etiology of the widespread skin rash of cutaneous DCI. Wilmshurst et al.[15] studied 60 divers (57 amateur and 3 professional; 21 female) and one male caisson worker who, on one or more occasions, had presented with a skin rash after decompression following a dive or caisson exposure which was diagnosed by the attending physician as cutaneous DCI. These authors found that 47 (77.0%) of the 61 cases with cutaneous DCI had a shunt, whereas only 34 (27.6%) of the 123 control divers (i.e. divers who had never had cutaneous DCI) had this anomaly ($p < 0.001$). Nineteen of the control divers with shunts had small shunts (which the authors prospectively considered to be clinically irrelevant), but only one diver with cutaneous DCI had a small shunt. However, 30 (49.2%) of the 61 cases with a history of cutaneous DCI had a large shunt at rest. Cutaneous DCI occurred in only one of 29 divers (16 with large shunts, 3 with medium shunts, one with a small shunt, and 9 without a shunt), on two or three occasions in 16 divers (12 with large shunts, 2 with medium shunts, and 2 without a shunt), and on more than three occasions in the caisson worker and in 15 divers (13 with large shunts, and 2 without a shunt). These data confirm that most divers who present with cutaneous DCI also have a right-to-left shunt.

Klingmann et al.[18] showed that inner ear DCI (more common among amateur divers) may also, in some cases, be consequent to bubble emboli entering the labyrinthine artery. The authors presented 11 cases of inner ear dysfunction in 9 divers with inner ear DCI. All 9 divers had a significant right-to-left shunt as diagnosed at transcranial Doppler sonography.

Balestra et al.[4] concluded that divers should be taught not to perform forceful Valsalva maneuvers to equalize the middle ear pressures, and that any maneuver or exercise that is likely to cause a similar rise in intrathoracic pressure for a "prolonged" period should be discouraged in divers with a PFO, for a sufficiently long period after their dive. These divers (and maybe also those without a PFO) should be advised to refrain from strenuous leg or arm exercise (such as air tank handling and dive boat boarding with full equipment) after decompression dives.

We do not believe that it is so easy to avoid dive complications in divers with a PFO. Specific PFO anatomies (long tunnel and/or a floppy caudal portion of the septum primum on the left side), and the presence of an atrial septal aneurysm, may allow right-to-left shunting even at rest, clearly reducing the possible efficacy of these specific suggestions.

The absolute risk of suffering a major DCI event is quite low even in divers with a PFO. However, we still do not know the potential long-term neurological hazards[19] due to asymptomatic ischemic brain lesions which have also been found to be related to the presence of a PFO in divers[20], i.e., the effect of the cumulative cerebral ischemic burden associated with a PFO in divers is unknown.

Our 2 patients treated with transcatheter closure of the PFO experienced no new episodes of DCI after the procedure and they are still practicing scuba diving (although the follow-up is short).

Theoretically, percutaneous PFO closure could be effective in lowering the risk of DCI in divers, but this hypothesis has not yet been properly tested.

Clinical implications. In our clinical experience we find it difficult to manage divers casually found to have a PFO; should we advise them about the risks and suggest that they avoid diving or should we close the PFO? What is a cardiologist supposed to do when a diver asks for a certificate stating that he is fit to practice this sport? Should we ask all subjects who want to practice diving as a sport to undergo at least a transcranial sonography or contrast transesophageal echocardiography?

The COCIS (The Italian Organizing Cardiological Committee on Eligibility for Sports) does not provide specific suggestions about this subject. The UK Sport Diving Medical Committee suggests that sport divers known to have a right-to-left shunt should be allowed to dive to depths shallower than 15 m. If a diver with a shunt wishes to go deeper than 15 m the options include the use of nitrox with an air decompression table to result in little or no bubble nucleation or to perform transcatheter closure of the defect.

In conclusion, the clear association between PFO and DCI has led some diving schools to recommend screening professional divers or avid amateurs for the presence of a PFO. We believe that transcatheter closure of the PFO may be an option for these subjects. It is currently unclear whether occasional sport divers should undergo specific evaluation in order to exclude the presence of a PFO.

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