The double chief factors in decompression illness: bubbles and patent foramen ovale

Dekompresyon hastalığında iki temel faktör: kabarcıklar ve "patent foramen ovale"

Dear Editor,

I read with great interest the recently published case report by Ozturk et al.(1) in the September issue of the Anadolu Kardiyoloji Dergisi. They presented a male pilot with gas embolism after hypobaric chamber training. It is known that some aviators and divers who have never experienced any decompression problems may show acute damages in multiple organs (2-5). The term decompression illness (DCI) is used for describing decompression disorders with gas bubbles as the initiator (2-5). In addition, formation of venous gas emboli as a result of decompression from hyperbaric pressures is well known. During sudden decrease of the pressure, nitrogen should be eliminated from tissues to venous circulation and then these gas bubbles may manifest by hemodynamic deterioration secondary to transmission into the left heart and coronary circulation (3, 4).

It is accepted that ultrasound is the best method for detecting gas, which is lead to increase the reflected intensity of the signal (2-5). However, it can often be difficult to differentiate bubbles and the movement of valves and vessel wall. Also, the placement of the Doppler signal in the blood flow reduces the possibility of bubble-like signals due to reflection on heart valve or vessels walls (2). Recently, the routine use of transesophageal echocardiography (TEE) in patient at high-risk for gas embolism have made early detection of micro- and macro-embolization (3). Furthermore, gas bubbles in the systemic circulation can be detected by a transthoracic echocardiography, though it is less sensitive than contrast TEE (4).

In the case report (1), the authors ignored and did not discuss the presence of a patent foramen ovale (PFO), which may cause paradoxical embolization. Patent foramen ovale also has been identified as a potential risk of decompression illness (3-5). Large volumes may cause pulmonary barotraumas and/or may escape into the systemic circulation via pulmonary arterio-venous shunts or patent foramen ovale (4). The PFO likely to be the most way for emboli because of PFO is detected in 25% to 30% of the general population (4). The recent study (the largest to date) in divers undergoing diagnostic testing for PFO revealed that the presence of TEE-detected PFO is related to an absolute risk that was found to be five times higher than that among divers without PFO (4).

Cartoni et al. (5) found that divers with PFO at rest had a higher risk of developing this form of DCI. They claimed that in presence of a PFO at rest, the exposure time permitting shunting could facilitate paradoxical gas embolism (5). They also recommended that if transthoracic echocardiography shows a positive contrast study or the diver had already developed a DCI episode TEE is required to assess the anatomic features associated with PFOs, like its diameter and fossa ovalis membrane mobility (5).

In conclusion, although echocardiography, especially TEE is not used routinely due to cost effective problems and also not a practical approach, it is important to evaluate both gas bubble and foramen ovale by TEE for planning preventative and therapeutic strategies in both aviators and divers who experienced DCI and have recurrent high risk due to duty.

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Author's Reply

The letter "The Double Chief Factors in Decompression Illness: Bubbles and Patent Foramen Ovale" is quite interesting with the concerns expressed and contributions to our previously published case report (1).

As mentioned in the letter, it is very well explained in the medical literature that bubble formation in decompression sickness (DCS) can be best detected by ultrasound. Spencer described the ultrasound findings and 5-grades reflecting bubble intensity in the circulating blood and heart (2). However, it is also known that there is no direct relation between the bubble intensity and clinical outcomes –which means there can be minor or no DCS

symptoms with Grade-5 (very high) bubble intensity, whereas Grade-1 or bubble intensity can cause severe DCS (3,4). Therefore, ultrasound assisted detection of bubbles is a research tool, rather than being a routine practice in training facilities.

On the other hand it is overemphasized that diagnostic procedures should not delay specific treatment in severe DCS cases (5).

Patent foramen ovale (PFO), which may cause paradoxical embolization is also a major concern, especially in the diving community because of the high prevalence and potential risks. Although PFO might have taken a role in our case it has not been possible to identify it. After acute treatment the patient has been discharged and continued further medical investigations at other institutions. Myocardial perfusion scintigraphy and coronary angiography reports showed normal results but the patient had twice transesophageal echocardiography which gave contradictory results about the presence of a PFO. The discussion regarding this contradiction intentionally left beyond the scope of our presentation.

In conclusion, on an operational point of view, instructors and flight surgeons should always be cautious about performing the specific treatment of DCS cases without a delay.

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