

Case reports

Acute coronary syndrome and decompression illness: a challenge for the diving physician

Marco Brauzzi, Fabio Andreozzi, Laura De Fina, Paolo Tanasi and Stefano Falini

Abstract

(Brauzzi M, Andreozzi F, De Fina L, Tanasi P, Falini S. Acute coronary syndrome and decompression illness: a challenge for the diving physician. *Diving and Hyperbaric Medicine*. 2013 December;43(4):229-231.)

Decompression illness (DCI) is a syndrome with diverse clinical manifestations but in which cardiac symptoms are rare. In the presence of cardiac symptoms, the necessity to rule out an acute coronary syndrome (ACS) which requires prompt treatment may result in delay to appropriate recompression treatment. We describe three cases with cardiologic symptoms referred to our centre by the Emergency Department (ED) of our facility. The first was a 48-year-old woman who lost consciousness during a dive and required cardiopulmonary resuscitation. The final diagnosis was acute myocardial infarction and the patient did not undergo recompression treatment. The second case was that of a 27-year-old man who complained of tachycardia, dyspnoea and vertigo soon after a dive. He was referred by helicopter ambulance and in the ED was diagnosed with new-onset atrial fibrillation. Recompression resulted in disappearance of his vertigo, and sinus rhythm was restored pharmacologically. The third case was a 43-year-old man, with a history of coronary artery disease, who had undergone coronary artery bypass grafting three years previously. After a repetitive dive without adequate decompression, he complained of crushing retrosternal pain and numbness in the upper left arm. All cardiovascular examinations were negative and the patient was recompressed, with resolution of his symptoms. Features to consider in arriving at the correct differential diagnosis in divers presenting with cardiac symptoms are discussed in the light of these three illustrative cases.

Key words

Decompression illness, decompression sickness, cardiovascular, diving accidents, case reports

Introduction

Decompression illness (DCI) is caused by the formation of air bubbles in blood or in tissues, during or after a reduction in environmental pressure (decompression). It includes two pathophysiological syndromes: arterial gas embolism (AGE) and the more common decompression sickness (DCS).¹ Recompression should occur as soon as possible to avoid deterioration and late recurrence.² In case of cardiac symptoms, distinguishing DCI from an acute coronary syndrome (ACS) is a potential challenge for the diving physician, given that both conditions require urgent but different treatments neither of which should be delayed.^{3,4} We describe three patients referred to our centre by the emergency department (ED) of our hospital. In all cases the dominant symptoms were cardiac.

Case reports

CASE 1

A 48-year-old female, with no risk factors for coronary artery disease (CAD), had passed a diving medical examination three months earlier. Weather conditions on the day of the incident were described as “nice”. At the beginning of the bottom phase of a scuba dive to 25 metres’ of sea water (msw), she lost consciousness without any prodromal symptoms.

Her dive buddy conducted an emergency controlled ascent to bring her to the surface. The helicopter ambulance arrived on the scene within 20 minutes. Oro-tracheal intubation was performed during medical evacuation to hospital. In the ED, blood analysis showed raised cardiac enzymes and a heart ultrasonography was performed. She suffered a cardiac arrest and advanced life support was initiated. Initially the team was unaware that the patient had lost consciousness early in the bottom phase of the dive, until a few minutes later the victim’s buddy described by phone the history of the accident. She was successfully resuscitated and admitted to the intensive care unit (ICU) on mechanical ventilation.

In the ICU, she had a large P_aO_2/F_iO_2 difference, with mild metabolic acidosis. She was haemodynamically unstable, requiring dopamine $10 \mu\text{g kg}^{-1} \text{min}^{-1}$; iv furosemide was administered. The first chest X-ray in the ICU showed pulmonary oedema with widespread alveolar involvement. Transthoracic echocardiography showed “left ventricle globally hypokinetic with normal size and a small pericardial effusion”. She was commenced on a heparin infusion. On the second day, a transoesophageal echo (TEE) showed an enlarged, akinetic left ventricular apex, aneurysm of the interatrial septum with a patent foramen ovale (PFO) and an ejection fraction of about 30%. Over the following days the patient rapidly improved, and two days after weaning from mechanical ventilation she was discharged from the

ICU. She was fully recovered in about 10 days. The final diagnosis was “*acute anterior myocardial infarction with acute pulmonary oedema in a patient with aneurysm of the interatrial septum and hypertrophic cardiac disease*”.

CASE 2

This diver was a 27-year-old male, referred by heliambulance for tachyarrhythmia and onset of intense vertigo about 30 minutes after a scuba dive to 56 msw for 18 min. Initially the patient complained of tachycardia and dyspnoea followed by the vertigo and nausea and vomiting. Personal history revealed thyroid disease and gastro-oesophageal reflux. The dive was performed within the limits of his diving computer profile. On arrival in the ED, new-onset atrial fibrillation with an average ventricular rate of 95 beats per minute (bpm) was diagnosed. Echocardiography revealed an aneurysm of the atrial septum. The patient was treated with a US Navy Treatment Table 6. During the treatment all neurological symptoms resolved. In the cardiology ward, after propafenone failed to convert his rhythm, amiodarone and heparin were administered, with reversal to sinus rhythm at a rate of 72 bpm. Follow-up TEE showed a type I Hanley interatrial aneurysm, with almost fixed prolapsing in the right atrium and a PFO with right-to-left shunting during a Valsalva manoeuvre. He received two further US Navy Treatment Table 5 (USN TT5) and five HBOT at 253 kPa for 90 min over the next seven days. Brain MRI (magnetic resonance imaging) was normal and he was asymptomatic when discharged.

CASE 3

A 43-year-old male presented himself to the ER in the evening after two dives, the first in the morning to 49 msw for 41 min with decompression, and the second in the afternoon also to 49 msw for 31 min with omitted decompression. He presented because of the onset of crushing retrosternal chest pain, tachyarrhythmia and left upper-arm paraesthesia, which recovered after a period of normobaric oxygen breathing. The patient's history revealed an episode of unstable angina, treated three years earlier with coronary artery bypass grafting (CABG), with subsequent complete recovery and good cardiac performance. Blood analysis and echocardiography showed no evidence of ACS. However, gas bubbles were seen in the right heart chambers on the echo. The patient was treated with a USN TT5 and discharged the following day after complete regression of his symptoms. He was advised to consult his cardiologist before resuming diving.

Discussion

CASE 1

Many cases of sudden death from cardiovascular disease with no prior history have been reported whilst diving,

cardiovascular events causing 20–30% of all deaths that occur while scuba diving.⁵ For many people, the real problem is that the first sign of CAD is a heart attack. In this case, the diagnosis was difficult because of the clinical condition of the patient and the initial lack of information on the dynamics of the accident. Therefore, the most probable diagnosis in the early phase was DCI. Luckily the phone call received from the diving centre explained the situation and described the onset of symptoms. Otherwise we were ready to treat her in the hyperbaric chamber.

CASE 2

Atrial fibrillation is the most common cardiac dysrhythmia. It may be asymptomatic, but is often associated with palpitations, fainting, chest pain or congestive heart failure. However, in some people, atrial fibrillation is caused by either idiopathic or benign conditions. Paradoxical embolization occurs when gas that has entered the venous circulation migrates to the systemic arterial circulation, leading to signs and symptoms of AGE.⁶ The two mechanisms by which this can occur are migration of gas through a right-to-left shunt, as in patents with PFO, or overwhelming the pulmonary capillary filtration system. These bubbles may embolize to the whole body, including the coronary circulation. The clinical manifestations are dependent on the volume of gas that enters the vasculature and the rate of entrainment of gas. Generally from a cardiological viewpoint, symptoms of right heart strain may develop or, as cardiac output declines, tachyarrhythmia and hypotension may develop, as happened in this case. Cardiac symptoms of DCS do not receive enough consideration. It is suggested that, whenever possible, an electrocardiogram should be performed in the basic evaluation of suspected DCS.

CASE 3

Severe DCI is characterised by central nervous system and pulmonary symptoms and circulatory problems such as hypovolaemic shock.¹ Pain is reported in only about 30% of cases. Because of the anatomical complexity of the central and peripheral nervous systems, signs and symptoms are variable and diverse. Symptom onset is usually rapid, especially in severe cases of DCI, but may be delayed for up to 36 hours after the dive.

In the 2005 Diver Alert Network Report, more than 14% of the fatalities reported had a chronic history of high blood pressure and/or heart disease.⁷ Obesity, another factor reported in 55% of fatalities, is connected to heart disease and hypertension, with resulting links to poor health and poor exercise tolerance. In combination with other contributing factors, cardiopathy can increase the risk of a severe or fatal dive incident.

An individual who has undergone CABG or angioplasty may have suffered significant cardiac damage before the surgery.

The post-operative cardiac function of individuals dictates their fitness for diving. In patients who have had open-chest surgery, after a period of stabilization and healing (6–12 months is usually recommended), a thorough cardiovascular evaluation is needed prior to being cleared to dive. Patients should be free of chest pain and have normal exercise tolerance, as evidenced by a normal stress test (13 mets or stage 4 of the Bruce protocol). If there is any doubt about the success of the procedure or about the condition of the coronary arteries, the individual should refrain from diving.

In Case 3 the problem was differential diagnosis between myocardial infarction and DCI. The blood analysis and cardiac evaluation, which proved negative, resulted in a delay of about three hours in starting recompression treatment. This diver resumed diving activity after his CABG surgery without prior clearance from a diving physician.

The onset of cardiac symptoms after a dive, particularly in patients with a clinical history of CAD does not automatically rule out the diagnosis of DCI, just as neurological symptoms do not rule out an ACS. Therefore, the assessing physician needs to take into consideration:^{3–7}

- timing of the onset of symptoms after the dive;
- the realistic possibility of DCI (based on the time and depth of the dive, the necessity for decompression, how the decompression was performed);
- physical activity at the time of onset of symptoms;
- severity of symptomatology and its development (i.e., improving, stable or deteriorating);
- the diver's previous medical history;
- whether similar or the same symptoms have occurred in the past after dives.

In some cases, a differential diagnosis may be difficult, as in these cases. Whilst the predominant symptoms were cardiac, only in one case was there no need for recompression.

Conclusions

So we may conclude that:

- DCI may sometimes onset with cardiac symptoms;
- it is not true that all cardiac symptoms at the end of a dive are the result of DCI;
- a patient with a previous CABG must be carefully evaluated before starting recompression treatment;
- it is mandatory that a thorough cardiological assessment is performed on all patients presenting with symptoms of DCI, and maintained for at least 48 hours.

References

- 1 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377:153-64.
- 2 Moon RE, Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Environ Med*. 1997;68:234-43.
- 3 Bove AA. Cardiovascular problems and diving. *SPUMS Journal*. 1996;26:178-86.
- 4 Lowry C. Cardiac problems and sudden death. In: Edmonds C, Lowry C, Pennefather J, Walker R, editors. *Diving and subaquatic medicine*, 4th ed. London: Hodder Arnold; 2002. p. 399-408.
- 5 Denoble PJ, Caruso JL, Dear G de L, Pieper CF, Vann RD. Common causes of open circuit recreational diving fatalities. *Undersea Hyperb Med*. 2008;35:393-406.
- 6 Bousages A. Interatrial right-to-left shunting after SCUBA diving. *Int J Sports Med*. 2006;27:508; author reply 509.
- 7 Vann R, Denoble P, Dovenbarger J, Freiburger J, Pollock NW, Caruso JL, Ugucioni DM. *DAN Report on decompression illness, diving fatalities and Project Dive Exploration*, 2005 Edition. Durham, NC: Divers Alert Network; 2005.

Patient consent

All three patients, one being a foreign national, were lost to follow-up, and all attempts to contact them have failed.

Submitted: 28 May 2013

Accepted: 30 October 2013

Marco Brauzzi¹, Fabio Andreozzi¹, Laura De Fina², Paolo Tanasi² and Stefano Falini²

¹ Hyperbaric Medicine Unit, Niguarda Hospital, Milan, Italy.

² Hyperbaric Medicine Unit, Misericordia Hospital, Grosseto, Italy.

Address for correspondence:

Marco Brauzzi, MD

Via del Commendone 42

58100 Grosseto, Italy

Phone and Fax: +39-(0)5-6445-0434

E-mail: <marco.brauzzi@ospedaleniguarda.it>