Case report

Immersion pulmonary oedema and Takotsubo cardiomyopathy

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Abstract

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A 67-year-old female scuba diver developed a typical immersion pulmonary oedema (IPE), but investigations strongly indicated Takotsubo cardiomyopathy (TC). The cardiac abnormalities included increased cardiac enzymes, electrocardiographic anomalies and echocardiographic changes, all reverting to normal within days. This case demonstrates a similarity and association between IPE and TC, and the importance of prompt cardiac investigations both in the investigation of IPE and in making the diagnosis of TC.

Key words

Diving incidents; echocardiography; immersion; pulmonary oedema; cardiovascular; case reports

Introduction

Immersion pulmonary oedema (IPE) has been increasingly reported in the literature since the 1980s.[1] It can occur with surface swimming and scuba diving. The diagnosis is initially based on clinical criteria (i.e., onset of dyspnoea during diving, cough, frothy sputum, hypoxia and bilateral rales), confirmed by chest X-ray (CXR) or computed tomography (CT). Death or recovery occurs within hours. Recurrences are common.[2]

Takotsubo cardiomyopathy (TC) is an acute, reversible disorder of the heart characterised by left ventricular dysfunction. It is identified by echocardiographic evidence of transient apical ballooning, acute reversible apical ventricular dysfunction, transient electrocardiographic (ECG) changes and increased cardiac enzymes and markers despite normal angiography findings.[3,4,]

Case report

A 67-year-old woman developed respiratory distress whilst scuba diving near Wooli, NSW, Australia. She was an experienced diver with more than 1,800 dives over a 12-year period. She had never suffered a diving incident. Medically, she had spinal dysraphism and hypercholesterolaemia, both of which were well controlled on pregabalin and rosuvastatin. There was no evidence of hypertension, cardiac disease, arrhythmia or pulmonary disease. An echocardiogram during a routine medical check-up a year previously was normal. Cardiac CT calcium score at that time was 100 (1–10 = small plaque; 11–100 = mild plaque present; 101–400 moderate plaque; > 400 = extensive plaque).

On the day prior to the incident, she had two boat dives using compressed air, one to a maximum depth of 15 metres' sea water (msw) for 56 minutes and another to 18 msw for 48 minutes. Water temperature was 19^{O}C, which was colder than expected. There was a surge with a mild current and she was using hired equipment as she felt her own regulator had developed some resistance. All these factors made her more stressed than usual but she completed both dives with no symptoms. The next day, after a 21-hour surface interval (no appreciable nitrogen load), she performed a boat dive at 8:57 a.m. to 20 msw using her own regulator (the problem with resistance had been rectified). As the water temperature was still 19^{O}C, she added a wetsuit vest and an extra kg of weight (total 5.5 kg). The extra vest was described as tight but not overtly so. Sea conditions were mild, with minimal current and good visibility.

She had some initial difficulty with buoyancy and required a head-down descent. The dive was otherwise uneventful for 15 minutes. At that time and at a depth of 19–20 msw, she became aware of "*not feeling right*" and felt some difficulty in breathing. She indicated this to her companion and they ascended over 8 minutes to 5–6 msw where they performed a 5-minute safety stop. The dyspnoea worsened during this ascent and they then ascended rapidly to the surface, where she started coughing and expectorated pink, frothy sputum. She also felt a rattling sensation in her chest. There was no chest pain. At no time did she feel excessive resistance to breathing from her regulator and she did not remove her regulator until she reached the surface. There was no aspiration of sea water or bubbling in the regulator at any stage. Dive computer recordings revealed a surface-corrected respiratory minute volume of 12.4 L·min^{-1} compared to 16.2 L·min^{-1} the previous day. Her total dive time was 28 minutes.

From the surface, she was assisted into the boat, laid supine and given 100% oxygen by mask. On reaching the shore after approximately 40 minutes, she managed to walk from the jetty before being assessed by the ambulance and helicopter

retrieval crew. She was medivaced and admitted to Coffs Harbour Hospital within two hours of arriving on shore.

On admission, clinically she was in acute pulmonary oedema with crepitations to the mid zones bilaterally. Oxygen saturation on air was 90%. She was given 100% oxygen combined with continuous positive airway pressure, intravenous frusemide, aspirin, and clopidogrel. CXR showed acute pulmonary oedema. ECG revealed sinus rhythm, premature ventricular complexes, borderline left axis deviation and non-specific *t*-wave abnormalities on the lateral leads. Troponin T was elevated to 4,052 $ng \cdot L^{-1}$.

Transthoracic echocardiogram (TTE) was performed an hour after admission and showed normal left ventricular (LV) size with moderate segmental impairment of systolic function; left ventricular ejection fraction (LVEF) was 38%. There was extensive anterolateral, lateral and posterior hypokinesis and mild mitral incompetence. Four hours after admission, she underwent coronary angiography which revealed moderate segmental LV dysfunction (mid-anterior, lateral and inferoposterior hypokinesis with sparing of apex and basal walls). The left main coronary artery was widely patent, whilst the anterior descending, circumflex, intermediate and right coronary arteries had mild irregularities only. It was concluded she had minor diffuse coronary artery disease, not requiring any intervention. Haematological and biochemical results were normal. Natriuretic peptides were not performed.

She was well within six hours of admission and was discharged home the next day with a diagnosis of Takotsubo cardiomyopathy. She was taken off aspirin, frusemide and clopidogrel and was started on bisiprolol. A repeat TTE performed six days after the incident demonstrated normal LV and RV size and function; LVEF of 62%; Grade 1 (abnormal relaxation) diastolic dysfunction with normal estimated filling pressures with a normal estimated right heart pressures (RVSP = 34%) and mild (grade 1/4) mitral and tricuspid regurgitation.

She presented a week later for advice on returning to diving. On review of her clinical presentation, it was thought the likely diagnosis was IPE with TC. Pulmonary function testing was normal, with a forced expiratory volume in 1 sec (FEV_1) of 2.32 L, forced vital capacity (FVC) of 2.99 L and FEV_1/FVC ratio of 77%. Hypertonic saline provocation testing was negative. She was taken off bisiprolol and remained normotensive. On advice, she has refrained from further scuba diving and will take precautions while swimming/snorkelling.

Discussion

We describe a case of an elderly woman presenting with pulmonary oedema whilst scuba diving. Clinically she had typical IPE, but the cardiac investigations revealed characteristic evidence of TC. The important features that this case demonstrates are the similarity between the two disorders, the importance of early investigation if such cases are to be identified and the potential for false presumption of cardiac normality if these investigations are delayed.

There have been a number of proposed aetiologies for IPE, including cold-induced pulmonary hypertension, immersion, hydrostatic pressures, negative inspiratory pressures, exertion, stress, aspiration, genetic predisposition and underlying cardiac pathology. Other than immersion and possibly stress, most of the postulated causes for IPE were not evident in this patient. The better known diving disorders of aspiration, pulmonary barotrauma and decompression sickness were also not evident from the dive description or profile. There was no evidence, clinically or on investigation, of any respiratory or cardiac disorder preceding the dive or a week subsequently. The presence of known cardiac pathology in IPE cases has been noted previously.[5–9] More recently, there has been an association of IPE with transient cardiac pathology. Some divers with IPE present with findings of a reversible myocardial dysfunction (RMD); in at least 28% of cases in one series.[10]

Takotsubu cardiomyopathy is characterised by reversible left ventricular dysfunction.[3] It is more common in postmenopausal women. There is usually a trigger in the form of physical or psychological stress. Diagnostic criteria for TC require:

- transient LV wall motion abnormalities involving the apical and/or mid-ventricular myocardial segments with wall motion abnormalities extending beyond a single epicardial coronary distribution;
- absence of obstructive epicardial coronary artery disease or angiographic evidence of acute plaque rupture that could be responsible for the observed wall motion abnormality;
- new ECG abnormalities such as transient ST-segment elevation and/or diffuse T-wave inversions.[4]

There is typically a slight increase in the creatine kinase, troponins I and T and brain natriuretic peptide levels. The ECG, echocardiographic and ventriculographic changes resolve spontaneously. Reported mortality from TC has varied in different case series from 0 to 12%; overall in-hospital mortality was 1.1%.[11]

The abnormal investigations with RMD are similar to those of TC. The distinction between RMD and TC is ill defined and some regard these as interchangeable and expressions of stress cardiomyopathies.[3,4] Because the cardiac abnormalities are transient with TC/RMD, investigations need to be instituted promptly following the incident if such cases are to be correctly assessed.

Other cases of TC in scuba divers and swimmers have been reported.[12,13] Discussions on the appropriate first aid,

treatment and assessment for diving fitness are described elsewhere.[2] There are four possibilities to be considered when there is a co-existence of TC or RMD and IPE; either may lead to the other, IPE may be TC/RMD triggered by immersion or the association may be coincidental.

Conclusions

All cases of IPE should be investigated for possible TC/RMD as early as possible after treatment has been instituted. This includes cardiac enzymes and markers, ECGs and echocardiography. If any of these are abnormal, they should be repeated. Presumption of cardiac normality should not be made unless such investigations have been performed promptly. Normal investigations days or weeks later do not necessarily imply cardiac normality.

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