Takotsubo cardiomyopathy findings on cardiac magnetic resonance imaging following immersion pulmonary oedema

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Abstract

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Immersion pulmonary oedema (IPO) can affect sea swimmers, snorkelers, and scuba divers. It can be fatal and cases are often mistaken for drowning. There has been an association between IPO and the development of takotsubo cardiomyopathy. We present a case study of a diver rescued from the water with IPO, who was subsequently found to have takotsubo cardiomyopathy on cardiac magnetic resonance imaging (CMR). This case demonstrates CMR findings as well as follow-up investigation results. The diver's and instructor's perspective during the initial dive incident are also described.

Introduction

Immersion pulmonary oedema (IPO) has been implicated in several serious diving incidents over the past few years.[1–2] Although most divers recover fully once out of the water, there are some where impaired left ventricular function is identified, a feature not typically associated with IPO, but likely linked to a concurrent stress cardiomyopathy.[3–6] Takotsubo cardiomyopathy is a transient myocardial dysfunction that mimics an acute coronary syndrome, presenting with similar symptoms (chest pain, shortness of breath), ischaemic electrocardiograph (ECG) changes, and a troponin rise. Characteristically, there is a reversible left ventricular regional wall abnormality disassociated from the coronary arteries. It is most common in post-menopausal women and is sometimes associated with a physical or emotional trigger.[7]

Whilst the link between takotsubo cardiomyopathy and IPO is well documented, no reports have presented cardiac magnetic resonance imaging (CMR) findings. CMR provides the gold standard for functional imaging in suspected takotsubo cardiomyopathy providing assessment of left ventricular (LV) and right ventricular (RV) volume and function, regional wall motion abnormalities, and uniquely assessing myocardial tissue characterisation.[7]

Case report

The patient consented to the reporting of their case.

A 54-year-old female was diving off the south coast of England in 12°C water, wearing a 7 mm wetsuit and breathing air on open-circuit scuba. She was previously well and taking no regular medications. She descended to 9.4 metres of seawater for 36 minutes before experiencing difficulty inhaling from her regulator. She signalled out of air (despite having adequate gas supply), inducing panic and triggering ascent to the surface. At the surface she vomited, became cyanosed and voiced continuous fearful screaming. She was evacuated via helicopter to the nearest emergency department. On arrival, she was dyspnoeic with pink frothy sputum and widespread inspiratory crepitations throughout both lung fields. Chest X-ray showed changes consistent with pulmonary congestion (Figure 1).

Arterial blood gas analysis while breathing on simple face mask with delivery of 15 $L \cdot min^{-1}$ of oxygen showed (normal values in brackets): pH 7.31 (7.35–7.45); P_aO_2 9.7 (11.0–14.4) kPa; P_aCO_2 5.3 (4.6–6.4) kPa; HCO_3 19.4 (21–28) $mmol \cdot L^{-1}$; base excess -2.7 (-3–3) $mmol \cdot L^{-1}$; lactate 2.7 (< 2) $mmol \cdot L^{-1}$.

An initial ECG identified atrial fibrillation with a fast ventricular response (120 beats per minute), although the patient subsequently spontaneously reverted to sinus rhythm. Initial troponins were raised but improved the following day (Table 1).

A diagnosis of IPO was made, and intravenous diuretics were started to good effect. On day two a coronary angiogram

Figure 1
Day 1 chest X-ray showing pulmonary oedema

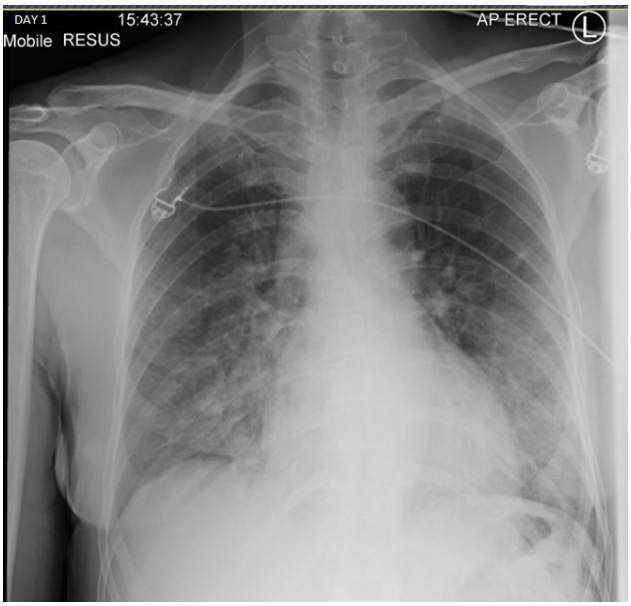


Table 1
Blood test results; CRP – C-reactive protein; GFR – glomerular filtration rate; Hb – haemoglobin

Parameter (normal values)	Day 1 02:37	Day 1 13:21	Day 2 08:12	Day 3 08:56
CRP (< 5 mg·L^{-1})			69	
GFR (> 90)				> 90
Sodium (135–145 mmol·L^{-1})				139
Potassium (3.5–5 mmol·L^{-1})				3.7
Urea (2.5–6.7 mmol·L^{-1})				3.1
Creatinine (62–106 µmol·L^{-1})				64
Troponin (0–16 ng·L^{-1})	1,879	2,082	786	
White cell count (4–11 x 10^{9}·L^{-1})				8.9
Hb (> 120 g·L^{-1})				147
Platelets (150–400 x 10^{9}·L^{-1})				200

identified normal coronary arteries. A transthoracic echocardiogram showed a LV at the upper limit of normal size, with mildly impaired systolic function (ejection fraction [EF] = 45%) and abnormal apical function.

A CMR scan on day six identified a LV at the upper limits of normal size and volume (left ventricular end diastolic volume index 60 ml·m^{-2}, EF 46%), with circumferential akinesia in the mid-apical segments, and some focal dyskinetic motion in the true apex. Myocardial oedema and subtle, patchy, low intensity, non-ischaemic late gadolinium enhancement was observed in the mid to apical segments; in keeping with takotsubo cardiomyopathy (Figure 2).

The diver was discharged on day seven on bisoprolol and ramipril. An implantable loop recorder was inserted to capture any further episodes of arrhythmia.

Two months later the diver had made a good recovery, running 5–10 km three times per week with no recurrence of symptoms. The ECG showed sinus rhythm with corrected QT interval at the upper limit of normal and deep T-wave inversion in leads I, II, aVL and V3–V6. Blood pressure monitoring revealed a range of 146–163 mmHg (systolic)/ 79–88 mmHg (diastolic). She reported occasional short palpitations, but her implanted loop recorder showed no further episodes of arrhythmia. Repeat echo showed normal LV systolic function (EF 63%) with no hypertrophy or dilatation. Her ramipril was stopped and switched over to losartan and amlodipine.

Seven months after the incident a repeat CMR was performed that showed a mildly dilated LV with normal systolic function and a RV at the upper limits of normal size. There was no late gadolinium enhancement, no residual wall motion abnormalities and no oedema; all in keeping with a resolved episode of takotsubo cardiomyopathy (Figure 2).

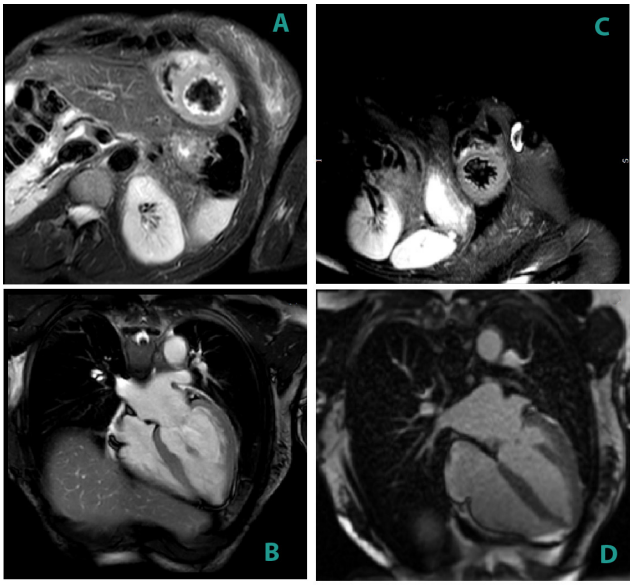
INSTRUCTOR'S PERSPECTIVE

We were 30 minutes into the dive at 8 msw when I gave the signal to ascend for a safety stop. I passed my surface marker buoy and she started to reel it in but forgot to deflate her BCD. I assisted her with this and we prepared to ascend, but she signalled out of air. I gave her my octopus and she signalled OK but was unable to link arms appropriately; she fell backwards, straining the octopus hose.

I helped her into an upright position, took the reel back, and checked her pressure gauge that said she had 90 bar. I tested her regulator and signalled to her to switch back. She made the switch and signalled that she was OK. She then got a blank look in her eyes and did not respond to my signal to ascend.

I performed a controlled ascent and inflated her BCD on the surface. She vomited, laid on her back and became unresponsive, her facial colour looked purple. I removed her equipment, and she was lifted onto the boat. She was placed into the recovery position, vomited further and then started screaming. She appeared to be responding to her husband, and so I asked if she was in pain. She did not reply verbally but she shook her head to indicate no. I administered oxygen and noticed that there were some bloody droplets in the mask. She became calm, and her facial colour improved. She was transferred to the emergency services.

Figure 2
Initial cardiac MRI images (A+B), along with images (C+D) from the follow-up cardiac MRI seven months later that showed full resolution; A shows T2/STIR image demonstrating circumferential high signal in the mid to apical segments in keeping with myocardial oedema; B shows subtle patchy low intensity late gadolinium enhancement in the mid lateral wall



DIVER'S PERSPECTIVE

I am PADI advanced open water qualified and have completed 15 dives in cold water. The dive was uneventful until I suddenly could not breathe, and my heart started racing. I thought I had a faulty regulator so I grabbed my instructor's octopus and from there on I cannot remember much until I was airlifted in the helicopter. I think I was in bad state when I first arrived in the hospital but, again, I cannot remember much of the first couple of hours.

I was short of breath the first two days after the accident but then it cleared up completely. I would say that I was fully recovered within two weeks. I am feeling good now and have resumed running and cycling. I still feel anxious going swimming as it brings back bad memories of the accident.

I previously worked as an airline pilot and had a medical every six months so I know that I was medically well before the accident. I am a non-smoker and drink minimal alcohol. I do not suffer from low mood or stress and did not take any medications. My father had a heart attack when he was 62 and suffered from high blood pressure and my mother has a normal heart and blood pressure. My fitness is good which I believe really helped my fast recovery.

I feel very grateful to the Royal National Lifeboat Institution, coastguard, doctors and nurses at the hospital.

Discussion

In IPO, the increased intrathoracic blood volume during immersion leads to a rise in preload. When combined with exertion and cold water-mediated peripheral vasoconstriction (increased afterload), the hydrostatic pressure gradient from pulmonary vessels to alveoli causes alveolar oedema, leading to symptoms such as shortness of breath and frothy haemoptysis.[3] Hypertension, cold water, beta-blockers, physical exertion and negative pressure breathing (rebreathers) have all been implicated as risk factors.[3,4]

The physical stress may, in some cases, trigger takotsubo cardiomyopathy as two-thirds of these cardiomyopathies are precipitated by extreme acute emotional or physical stress.[8] Over 50% of patients in the International Takotsubo Registry have a prior history of a psychiatric illness or a chronic neurological disorder.[9] Patients with such pathologies are less likely to be diving, and indeed, our patient had no history of these.

Takotsubo typically presents in post-menopausal women, although any gender and age group can be affected. Despite normal or non-obstructing coronary artery disease on angiography, ECG changes resemble myocardial ischemia with ST-elevation, T-wave inversion and QT prolongation with risk of ventricular arrhythmias. There is often a corresponding rise in cardiac biomarkers including troponins and cardiac brain natriuretic peptide.[10]

Postulated mechanisms include direct myocardial stunning induced by catecholamine release and vasoconstriction mediated ischaemia. Stress cardiomyopathies have been triggered by the direct administration of catecholamines and circulating levels in the acute phase have been found to be 10–20 x normal.[11]

The CMR findings here align with T2- short-tau inversion recovery (STIR) imaging seen in takotsubo cardiomyopathy patients; high signal, diffuse ventricular oedema distributed in the mid-apical planes of the LV and dissociated from the coronary arterial distribution.[12–14] Late gadolinium enhancement has been linked to takotsubo cardiomyopathy in more recent studies, thus the patchy low intensity enhancement correlates with this.[13] There was no basal hyperkinesia (octopus pot appearance) normally seen in 75–80% of Takotsubo cases, or mitral valve dysfunction, seen in 25% of Takotsubo cases.[13]

Resolution of the CMR findings, with no detectable late enhancement and return of normal ventricular function after the seven month follow up period, was anticipated. Reversibility is the hallmark of takotsubo cardiomyopathy as demonstrated previously with CMR.[12,13,15]

We postulate that this diver likely had essential primary hypertension predisposing to IPO during her dive which, in turn, resulted in the development of a takotsubo cardiomyopathy with associated atrial fibrillation. There has been much speculation as to whether cardiomyopathies in divers develop pre-dive or during the dive itself.[3] The description of events that the diver provided indicates that it occurred during the dive, in line with other case reports.[4,16]

Regarding medical management, an angiotensin receptor blocker was chosen as antihypertensive therapy over an angiotensin converting enzyme inhibitor to avoid the potential side effect of a cough. A calcium channel blocker was also chosen as, anecdotally, it has been shown reduce recurrence of IPO. This is thought to be due to a reduction in vasoconstrictive responses to physiological stimuli.[3,17,18]

The typical advice for divers who develop IPO is to avoid further diving due to risk of recurrence, although opinion is divided on this. If divers choose to return they would need to ensure that their hypertension is controlled. Other recommendations include; the use of open circuit equipment, diving with an experienced buddy, ensuring surface support (including oxygen) is available and a dive profile that would avoid obligatory decompression stops.

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