Myocardial oedema in the setting of immersion pulmonary oedema - Cause or effect?

James Oldman,1 Sarah Morwood,1 James Willis,1 Daniel Xavier Augustine1,2

SUMMARY
Immersion pulmonary oedema (IPE) is an under-reported and poorly understood phenomenon thought to be related to exercise-induced haemodynamic changes while submersed in water. Previous work has demonstrated reversible myocardial dysfunction during acute episodes. We present a case of IPE with concomitant, transient, left ventricular myocardial oedema characterised via MRI. This is a novel finding and may be evidence of left ventricular strain due to pressure overload or secondary to a subclinical myocarditis.

BACKGROUND
Open water swimming is a popular sport, with more than three million people participating in England in 2021.1 However, there is a growing body of evidence of an association with swimming-induced pulmonary oedema (SIPE).2–5 A subtype of immersion pulmonary oedema (IPE), it is characterised by fluid accumulation in the lungs in the absence of water aspiration during swimming, resulting in acute dyspnoea, hypoxia and a productive cough.6 First reported in 1989, its incidence is likely under-reported and has been estimated between 1.1% and 1.8%,2 and frequently occurs in those who are otherwise fit and healthy. Risk factors for development of SIPE include advanced age,2 female sex,3 hypertension,7 long course distances,4 cooler water temperature,5 and pre-existing heart disease.8 Furthermore, lower lung volumes and expiratory flow 12 months earlier,9 higher mean pulmonary arterial (PAP) and pulmonary arterial wedge pressures (PAWP) at rest,10 have all been correlated with the development of SIPE.

The precise pathophysiology remains elusive but likely involves increases in pulmonary arterial pressures related to centralisation of blood volume and an exaggerated pulmonary vasoconstrictive response to cold in the setting of increased cardiac output during exercise.10 When combined with peripheral vasoconstriction, increasing left ventricular afterload in cold water, together with negative pulmonary pressures during inspiration, there is stretch of the pulmonary capillary basement membrane which leads to alveolar oedema.5 Individuals with left ventricular hypertrophy, hypertension or structural heart disease typically have higher atrial pressures and are therefore less likely to tolerate further increases in preload, predisposing to SIPE.11

Clinical examination typically demonstrates bi-basal crepitations, and radiological investigations frequently display changes consistent with alveolar oedema. It is recommended that those presenting with SIPE undergo detailed evaluation of their cardiovascular physiology. In divers, elevated troponin, natriuretic peptides and electrocardiogram (ECG) changes consistent with myocardial strain and transient hypokinesia on echocardiography have been reported.12 Typically, coronary artery abnormalities are ruled out using CT angiography and patients often undergo cardiac MRI to define cardiac structure and function further. Other investigations such as MRI renal angiography and 24-hour urinary

Figure 1 Twelve-lead ECG on admission demonstrating sinus rhythm and T wave inversion in leads I, AvL and V2.
catecholamines may be undertaken to rule out uncommon causes of pulmonary oedema.

Treatment is primarily supportive and involves immediate removal from water and cessation of exercise while sitting upright. Oxygen therapy is frequently required. Diuresis, bronchodilators and continuous positive airway pressure may be necessary in some instances, while invasive mechanical ventilation with haemodynamic support is reserved for the most severe cases. Episodes typically resolve within 24–48 hours of leaving the water, but autopsy findings have been suggestive of IPE in approximately 10% of scuba-diving deaths. Notably, left ventricular hypertrophy was confirmed in all such cases.

Recurrence is common and has been reported between 13% and 22% among scuba divers and swimmers. Episodes vary in severity and frequently do not require hospitalisation; however, patients should be appropriately informed regarding the high risk of recurrence, and following an initial episode, be assumed to have a predisposition. Swimming at a slower pace, accompanied, in warmer water without a tight-fitting wetsuit are appropriate first steps following recovery. Non-steroidal anti-inflammatory drugs should be avoided and any comorbidity that worsens diastolic function should be optimised. Prophylactic sildenafil has been shown to lower PAP and PAWP, and this appears to be an effective preventative measure in those with a history of recurrent SIPE. Sufficiently powered randomised controlled data are needed for formal validation of this approach.

CASE PRESENTATION
A fit and well female in her 50s presented with significant dyspnoea and haemoptysis following a night open-water swimming event in water of around 17°C while wearing a wetsuit. She had no past medical history of note.

A keen competitive long-distance swimmer and triathlete, she had experienced significant dyspnoea during an open-water event 2 weeks prior and was forced to abandon, before feeling breathless for some days afterwards. The following weekend, the patient undertook an uneventful 3 km pool swim.

A week later, a repeat attempt during a night swimming event led to significant dyspnoea and haemoptysis within 300 metres and precipitated presentation to hospital. She had coincidentally had her Pfizer COVID-19 booster vaccination 6 hours before the swim. There was no chest discomfort, palpitation, pre-syncpe or preceding viral symptoms.

On admission, she was tachycardic (100 beats/min (bpm)) and breathless with an ongoing cough. Oxygen saturations were 96% on air. Blood pressure was 138/69 mm Hg and her temperature was 36.5°C. Her symptoms and tachycardia spontaneously settled within 2 hours of presentation. On clinical examination, there were fine bi-basal crepitations but her cardiovascular system was otherwise normal. Admission ECG demonstrated sinus rhythm with T wave inversion in leads I, AvL and V2 with a normal cardiac axis and corrected QT interval (figure 1).

INVESTIGATIONS
Plain chest radiography on admission demonstrated mild pulmonary oedema. CT of the thorax ruled out large vessel pulmonary embolism and demonstrated perihilar ground-glass consolidation bilaterally, with more significant changes in the lower lobes (figure 2). Transthoracic echocardiography demonstrated normal left and right ventricular size and function, with normal diastolic parameters. Initial blood test results demonstrated a neutrophilia with white cell count $14.7 \times 10^9/L$ (normal range $3.5-11 \times 10^9/L$) and neutrophils $12.6 \times 10^9/L$ (normal range $2.0-7 \times 10^9/L$), with an elevated troponin T ($23$ and $32$ ng/L) (normal range < $14$ ng/L) and N-terminal pro b-type natriuretic peptide (NT-Pro BNP) ($974$ ng/L) (normal range < $150$ ng/L).

Interestingly, cardiac MRI demonstrated prolonged T2 relaxation time of the basal to mid anterior, anterolateral and inferior-lateral segments, indicative of myocardial oedema (figure 3). There was also basal inferolateral non-ischaemic mid-wall late...
gadolinium enhancement, potentially indicative of fibrosis (figure 4). Left ventricular dimensions and systolic and diastolic function were normal. CT coronary angiography 8 weeks later demonstrated conventional coronary anatomy with a minor burden of non-flow-limiting atheromatous plaque, along with resolution of pulmonary oedema.

DIFFERENTIAL DIAGNOSIS
The hallmarks of IPE are: (1) acute onset of dyspnoea and/or haemoptysis during or immediately after swimming in the absence of water aspiration, laryngospasm or preceding infection; (2) hypoxia; (3) radiological evidence of pulmonary oedema; (4) rapid resolution within 48 hours. Nevertheless, it is important to keep an open mind when assessing patients with acute pulmonary oedema.

Causes can be broadly divided into cardiogenic (structural, arrhythmia) and non-cardiogenic causes. Significant structural heart disease was ruled out via transthoracic echocardiography. Mild structural cardiac abnormalities should not be overestimated when IPE is suspected, as significant pressure changes can exacerbate mild underlying cardiovascular abnormalities. Cardiac MRI demonstrated localised myocardial oedema in the setting of normal right and left ventricular size and function. A non-specific indicator of myocardial injury, this pattern could be consistent with ischaemia, pressure overload or a subclinical myocarditis. Alternative pathologies can also be excluded using cardiac MRI. Myocardial infarction is often characterised by a sub-endocardial distribution of late gadolinium enhancement, while typical morphological appearances and apical oedema may be appreciated in Takotsubo cardiomyopathy. Arrhythmia was deemed less likely and subsequent 48-hour monitoring was reassuring.

Non-cardiogenic causes are broad and, in the absence of clear neurogenic pathology, pulmonary embolism should be considered. In this case, CT pulmonary angiography ruled out thromboembolic disease and demonstrated ground-glass changes

Figure 5 Twelve-lead ECG on follow-up demonstrating sinus rhythm and T wave inversion in leads I, AvL and V6.

Figure 6 Twelve-lead ECG 2 months later demonstrating sinus rhythm and resolution of T wave inversion.
throughout both lungs consistent with pulmonary oedema. In circumstances where aetiology is unclear, renal artery stenosis should be considered and investigated via appropriate imaging.

In this case, a history of acute dyspnoea occurring in cold water, on exertion, while wearing a wetsuit, accompanied by pulmonary oedema with spontaneous resolution in the absence of aspiration or structural heart disease, was felt to be diagnostic of IPE. The time course suggests that the recent COVID-19 booster vaccination was unrelated.

**TREATMENT**

Symptoms settled within 2 hours of presentation to hospital. A treatment dose of dalteparin was administered while CT pulmonary angiography was awaited and the patient was discharged the next morning.

**OUTCOME AND FOLLOW-UP**

Following a period of monitoring, the patient was discharged the following day. Four days post admission, troponin T had fallen to 8 ng/L (normal range <14 ng/L) and NT-Pro BNP to 288 ng/L (normal range <150 ng/L). On clinic review, the ECG demonstrated sinus rhythm, and T wave inversion in leads I, AVL and V6 with a normal cardiac axis and corrected QT interval (figure 4).

Interval cardiac MRI demonstrated resolution of myocardial oedema as well as basal cavity inferior lateral fibrosis (figure 4). There was some residual basal inferior myocardial fibrosis present and again, left ventricular size and function were normal.

Forty-eight-hour ambulatory ECG demonstrated sinus rhythm throughout with heart rates between 49 and 117 bpm and rare atrial ectopy. An exercise test was completed 3 months post admission and demonstrated an appropriate rise in blood pressure without undue symptoms or arrhythmia. ECG at the time demonstrated resolution of T wave inversion (figure 6). Hypertension (169/75 mm Hg) was noted on clinic review and felt to be situational given a normal blood pressure during hospital admission and subsequently normal measurements when attending for the exercise test (110/70 mm Hg) and on ambulatory monitoring.

The patient currently remains well in herself and has been appropriately advised regarding precautions if wishing to continue with open water swimming. She will be reviewed with a surveillance cardiac MRI in early 2023.

**DISCUSSION**

To the best of our knowledge, this is the first published evidence of myocardial oedema characterised using MRI in the setting of IPE. While it is conceivable that this represents a pre-existing inflammatory process such as myocarditis which contributed towards IPE, it is also potentially a consequence of the acute episode. This is an interesting finding and complements historic data demonstrating reversible and frequently regional myocardial dysfunction in the setting of IPE. Importantly, echocardiography was normal in this case, although 2 days following the acute episode, therefore any dysfunction is unlikely to have been fully appreciated.

The aetiology of myocardial dysfunction in these scenarios is unclear, but authors have proposed a stress cardiomyopathy, akin to Takotsubo, as the underlying cause—a syndrome in which localised myocardial oedema on MRI is frequently observed. There are other possible explanations for localised myocardial oedema in this setting. Ischaemia due to cold-induced coronary vasospasm in the setting of increased myocardial oxygen demand or ventricular pressure overload as suggested in this case by an elevation in NT-Pro-BNP. In addition, the slight troponin elevation in combination with localised lateral repolarisation abnormalities, together with the myocardial oedema and fibrosis seen on the initial MRI, support a degree of myocardial injury. The ECG in particular adds to existing data which appears to demonstrate a tendency toward lateral repolarisation and ST segment disturbances.

In this case, hypertension at clinic review may allude to a predisposition to increased vascular tone in the setting of environmental stimuli. Indeed, those with a history of IPE have been shown to be more likely to demonstrate pathological vasoconstriction when exposed to cold and to have an increased likelihood of subsequently developing hypertension.

The UK Diving Medical Committee has published guidance for divers. However, at present, there are no formal national medical guidelines concerning the recognition and management of this complex condition. Detection is therefore dependent on

**Patient’s perspective**

While swimming in a quarry at a night swim I started to hyperventilate and realised I couldn’t swim any further. Luckily, I was able to call for help and got guided back to the quay by a paddleboard. When I got out, I undid my wetsuit and immediately felt the sensation of my lungs filling with fluid. I started to cough and had a metallic taste in my mouth. When I got into the light, I could see my sputum was pink and frothy.

I was very lucky to be surrounded by a great team at the quay who all knew I had SIPE. My husband drove me to the ED [emergency department] where I underwent an ECG, CXR [chest X-ray], bloods and CTPA [CT pulmonary angiogram]. Throughout the evening I passed urine frequently for about 10 hours until the cough stopped and I was discharged home.

Two weeks prior to this incident I had experienced a much milder event while open water swimming in the sea that I hadn’t attributed to SIPE at the time, but had also experienced difficulty in my normal running and pool swimming training. I had just assumed I was a bit under the weather.

Other than this I have had no other symptoms and am now fully recovered and back to full training.

**Learning points**

- Immersion pulmonary oedema is under-recognised and should be considered in any patient presenting with dyspnoea following immersion.
- Thorough investigation of the cardiovascular system should be undertaken and is frequently normal.
- Some patients may demonstrate transient myocardial dysfunction during an acute episode. This may be identified via demonstration of hypokinesia on echocardiography or hypokinesia or myocardial oedema on cardiac MRI.
- The precise aetiology of this condition is yet to be established. Possibilities include a stress cardiomyopathy, coronary vasospasm, ventricular pressure overload, or an inflammatory process.
- Recurrence is common and following an initial episode, patients should be presumed to have a predisposition. Patients should therefore be counselled appropriately.
a low index of suspicion when reviewing breathless patients who have been immersed.

As cardiac MRI becomes mainstream globally, it is likely that more data from IPE cases will emerge, enabling us to characterise myocardial pathology better in this under-diagnosed syndrome and potentially contribute toward risk stratification algorithms. This may facilitate individualised guidance regarding continued participation in water activities.

Contributors J.O collected the clinical information and authored the initial draft of the case report. SM and JW provided input into subsequent drafts. DXA oversaw the process and provided input into the final draft of the case report.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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