Case reports

A diver with immersion pulmonary oedema and prolonged respiratory symptoms

Ryo Morishima[1,2,3], Kei Nakashima[2], Shinya Suzuki[4], Nobuo Yamami[5], Masahiro Aoshima[2]

[1] *Department of Neurology, Tokyo Metropolitan Neurological Hospital, Fuchu, Tokyo, Japan*
[2] *Department of Pulmonology, Kameda Medical Centre, Kamogawa, Chiba, Japan*
[3] *Department of Medicine, Awa Regional Medical Centre, Tateyama, Chiba*
[4] *Department of Emergency Medicine, Kameda Medical Centre*
[5] *Yamami Clinic, Nichinan, Miyazaki, Japan*

Corresponding author: *Ryo Morishima, 183-0042 Musashidai 2-6-1, Fuchu city, Tokyo, 183-0042, Japan*
riyou_morishima@tmhp.jp

Key words
Ascent; Barotrauma; Breath-hold diving; Scuba diving; Hyperbaric oxygen therapy; Inflammation; Case reports

Abstract

(Morishima R, Nakashima K, Suzuki S, Yamami N, Aoshima M. A diver with immersion pulmonary oedema and prolonged respiratory symptoms. Diving and Hyperbaric Medicine. 2018 December 24;48(4):259–261. doi: 10.28920/dhm48.3.259-261. PMID: 30517959.)

Immersion pulmonary oedema (IPE) is particularly associated with an excessive reaction to exercise and/or cold stress. IPE usually resolves without recompression therapy within a day or two. Herein we report a diver diagnosed with IPE, in whom symptoms persisted for five days. A 58-year-old man presented with sudden onset of dyspnoea, cough and haemoptysis after surfacing. He was an experienced diving instructor with a history of moderate mitral valve regurgitation. While IPE was diagnosed and oxygen administered, respiratory symptoms deteriorated, and serum C-reactive protein elevated. No evidence of infection was seen. Three hyperbaric oxygen treatments were given on the basis of suspected decompression sickness, and symptoms subsequently resolved. The recently diagnosed mitral valve regurgitation and inflammatory response were considered to have contributed to the prolongation of symptoms.

Introduction

The concept of immersion pulmonary oedema (IPE) was established in recent years following a first report in 1981.[1] The condition is especially associated with an excessive reaction to exercise or cold stress.[2] IPE usually resolves quickly with rest and oxygen administration, according to previous reports.[2–4] We present a case of IPE in which respiratory symptoms lasted five days, and which improved after three hyperbaric oxygen treatments (HBOT), along with administration of antibiotics and diuretics. Such protraction of respiratory manifestations in a patient with IPE does not appear to have been reported previously. We consider the possible pathophysiological mechanisms underlying this unusual clinical situation.

Case report

The patient was a 58-year-old man with 20 years' experience as a scuba diving instructor. He had been diagnosed with moderate mitral valve regurgitation (MR) six months before the current event, but had not been prohibited from diving. Cough-variant asthma had once been suspected when he complained of chronic cough after an upper respiratory tract infection. He had a 20-pack-year history of smoking, but had quit 10 years earlier. He had no history of allergy and was not on any medications.

He went air scuba diving on an autumn morning using appropriately prepared equipment and wearing a wetsuit. He had been aware of mild dyspnoea and cough for two days before diving. He had felt substantial dyspnoea after swimming to a boat but this resolved after a short rest. The sea temperature was 20.4°C and the wave height was 2 m. His first dive of the day reached a maximum depth of 18.7 metres' sea water (msw) and lasted 56 minutes (min), including a three-minute safety stop at 5 msw. He felt the temperature was a bit cold and exercise intensity was light. He changed his ordinary air regulator to an oxygen regulator on the decompression stop in 5 msw, to demonstrate oxygen decompression for educational purposes. Just after restarting the ascent, he experienced intense shortness of breath that worsened on surfacing. Although he took off his wetsuit as it felt tight, symptoms did not resolve. He rested while inhaling pure oxygen but symptoms deteriorated and became accompanied by irresistible coughing and haemoptysis, and he was transported to a local hospital. He denied experiencing any marine animal stings or aspiration during

Figure 1
Chest X-ray on admission reveals bilateral lung base-dominant infiltrations

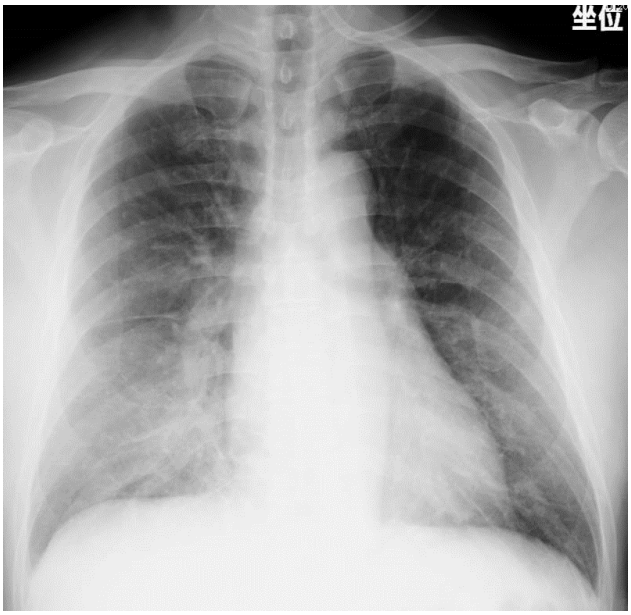
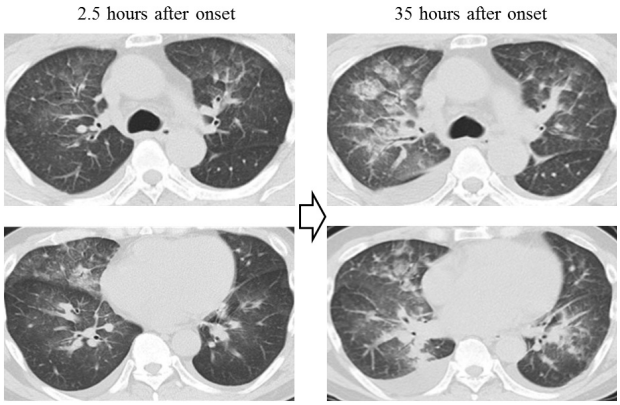


Figure 2
Chest CT on admission (2.5 h after onset) shows ground glass opacity in a central peribronchial-vascular distribution and smooth thickening of the interlobular septae in the lung bases bilaterally, compatible with pulmonary oedema Two days later (right, 35 h after onset), Chest CT findings worsened, revealing bilateral consolidation and pleural effusions



the dive, even when he exchanged his regulator.

On admission, heart rate was 138 beats per min, respiratory rate 24 breaths per min and oxygen saturation was 96% on oxygen at 6 L·min^{-1}. Vesicular breath sounds were diminished in both lung fields on auscultation. Cardiac examination revealed a systolic murmur (Levine 3/6) at the apex radiating to the axilla. No peripheral oedema was evident and jugular venous distention was not seen. Electrocardiography showed sinus tachycardia. Chest X-ray showed bilateral dependent oedema in the lower lung fields (Figure 1). Chest computed tomography (CT) revealed ground glass opacities with a central peribronchial-vascular distribution and smooth thickening of the interlobular septae in bilateral lung bases and apices (Figure 2). Laboratory studies showed a serum troponin I concentration of 0.020 ng·mL^{-1} and a brain natriuretic peptide (BNP) level of 150.8 pg·mL^{-1}. He was admitted to hospital to ensure rest and to administer oxygen, on suspicion of IPE. Ceftriaxone and azithromycin were administered from the day of admission because community-acquired pneumonia (CAP) was also being considered.

Hypoxia worsened, accompanied by productive cough during the night on the second hospital day. Transthoracic echocardiography (TTE) performed at this time revealed severe MR with tendon rupture, normal left ventricular wall motion and no evidence of right heart strain. Repeat chest CT revealed progression of pulmonary oedema and bilateral pleural effusions (Figure 2). Diuretics were administered and hypoxia improved slightly. However, fever increased to 39°C and laboratory studies revealed a strong inflammatory response (C-reactive protein (CRP) 20.7 mg·dL^{-1}). As the clinical course was atypical for IPE, heart failure and CAP, the patient was transported to our hospital on suspicion of decompression sickness (DCS).

On admission, heart rate was 114 beats per min, respiratory rate 25 breaths per min, blood pressure 113/68 mmHg, body temperature 38.4°C and oxygen saturation 95% on oxygen at 3 L·min^{-1}. Chest auscultation revealed fine crackles in the basilar regions bilaterally. No peripheral oedema or cyanosis were noted. Laboratory studies showed a CRP level of 22 mg·dL^{-1}, a BNP level of 134 pg·mL^{-1}, and otherwise normal results, including liver enzymes. Negative results were obtained for sputum and blood cultures, urine pneumococcal antigen, legionella antigen and pharyngeal mycoplasma antigen. Sputum cytology showed a markedly increased eosinophil count.

We suspected respiratory DCS and started recompression therapy according to United States Navy treatment table 6 (USN TT6).[5] Hypoxia, tachycardia, and tachypnea improved during the first recompression therapy, but response was incomplete. This first HBOT was extended. Two further USN TT6 resolved all symptoms, CT findings, fever and inflammatory response. Ceftriaxone was discontinued on day five as elevated liver enzymes were noted, ampicillin/sulbactam was administered instead. The patient was discharged without any further complications and subsequently underwent mitral valve replacement.

Discussion

We initially diagnosed IPE based on the typical presentation and radiological findings. However, despite appropriate initial therapy for IPE, symptoms deteriorated and we needed to consider other potential diagnoses. In addition to the use of antibiotics and diuretics, three HBOT were performed on the suspicion of DCS and symptoms had resolved by the completion of these.

The pathophysiological mechanism leading to IPE is elevation of lung capillary pressure, caused by a cold- or exertion-induced hypertensive response.[1,6] The pre-existing MR in our patient represents a known risk factor for pulmonary oedema. During immersion, the increased preload caused by the cold environment may have contributed to the development of pulmonary oedema.[2] On the other hand, shortness of breath just after swimming but before diving could have been an early manifestation of IPE, as in a previous report.[3]

The protraction of IPE into a five-day pulmonary disorder and pronounced inflammatory response were prominent features in the present case. Most reported cases of IPE resolved faster, without antibiotics or a need for recompression therapy.[3,4] The possibility of an infectious pathogenesis contributing to the high fever and inflammatory response could not be excluded in this case. On the other hand, the findings were sufficient to look for aetiologies other than infection because antibiotic administration appeared ineffective and no evidence of infectious pathogens was detected from microbiological examinations.

Another possible diagnosis was heart failure from reversible stress cardiomyopathy (Takotsubo cardiomyopathy), acute myocarditis or acute exacerbation of known MR.[8,9] However, there was little possibility of myocarditis or Takotsubo cardiomyopathy given the lack of myocardial strain findings on ECG, the normal wall motion seen on TTE on hospital day two in the previous institute and the negative results for serum troponin I.

Pre-existing MR could plausibly have contributed to this prolongation of respiratory symptoms. MR contributes to elevation of pulmonary artery pressure and subsequent pulmonary oedema. When his MR had deteriorated owing to tendon rupture was unclear, although the rupture had occurred at least since his first visit to the previous institute. According to his cardiologist, the pulmonary oedema could not have been owing only to MR because there was no evidence of right heart strain or hypotension.

This diver had the possibility of having respiratory DCS. Respiratory DCS has been described anecdotally as "*the chokes*". This is a rare presentation of DCS and case reports of respiratory DCS are limited.[10] Respiratory DCS has been considered to arise from the formation of a large quantity of gas bubbles and subsequent congestion of the pulmonary circulation.[5] The patient's diving log indicated a bottom time of 48 min and a maximum depth of 18.7 m. This suggests his safety stop was made within the no-decompression limit,[5] and the probability of profuse gas bubbles in pulmonary vessels seemed low. Consideration of this condition as respiratory DCS was thus difficult in the classical context, although one report has described a case thought to represent respiratory DCS with insufficient inert gas loading to form gas bubbles.[10] The clinical course of our patient, employing oxygen during decompression, post-dive, during transport and three USN TT, also reduces the likelihood of DCS as an explanation.

In conclusion, pre-existing MR could have contributed to protraction of respiratory symptoms and the slow response to recompression therapy in this case. Simultaneously, IPE itself may sometimes be more protracted than we have previously assumed in such situations. The inflammatory response could have been evoked by oxygen, infection or lung overinflation.

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Acknowledgements

We would like to thank the patient for his permission to publish this report. We would also like to thank Drs Kenji Yoshioka and Takayuki Ando for their useful discussions and for providing clinical data.

Conflicts of interest and funding: nil

Submitted: 05 February; revised 27 May and 05 July 2018
Accepted: 17 November 2018