Scuba diving, swimming and pulmonary oedema

Diving-related 'immersion' pulmonary oedema is very uncommon and probably underreported.[1,2] Immersion in thermoneutral water augments cardiac preload and increases pulmonary arterial pressure as blood is redistributed from the periphery to the thoracic cavity. Intrathoracic blood volume may increase by half a litre, resulting in a 30% rise in cardiac output and increases in right atrial pressures of up to 16 mmHg.[2,3] Most cases

of immersion-related pulmonary oedema have been reported during dives in cold water conditions. Cold water immersion causes peripheral vasoconstriction with elevated systemic vascular resistance and increased ventricular wall stress that leads to increases in pulmonary capillary hydrostatic pressures and the potential for stress failure of blood vessel walls.[4–6] Rapid resolution can occur in diving-related pulmonary oedema and consistent with this observation, experimental disruption of the blood–gas barrier improves rapidly following reduction in pulmonary capillary pressures.[7]

We present a case of immersion pulmonary oedema occurring in a middle-aged male diving instructor with pre-existing chronic atrial fibrillation (AF) while diving in warm waters. Following the sentinel event, recurrence of symptoms occurred during a surface swim.

A 54-year-old lifelong non-smoker was transferred from Vanuatu to Tasmania for investigation of severe breathlessness during a scuba dive. He had been in chronic AF for 17 years and had occasional gout, but was otherwise fit and regularly scuba dived and swam in the sea for considerable distances. His medications included sotolol 80 mg b.i.d. and allopurinol 100 mg daily. He was an experienced diver and had dived to 45 m previously without any problems.

At the time of his initial presentation, he was undertaking a diving rescue course on a shipwreck off Vanuatu using compressed air. Water temperature was 28°C and the dive time was 35 min. At 20 m he developed acute severe breathlessness and central chest pain and required assistance to ascend. On surfacing, he coughed up a large amount of frothy fluid, but had no frank haemoptysis. He remained conscious and had no neurological symptoms. Oxygen was given on the boat and within 30 min his breathlessness had resolved, but some 'chest tightness' and a cough productive of clear sputum persisted for 2 days at which point he sought advice from a veterinarian. A chest X-ray carried out at that time by a veterinarian was reportedly normal and his symptoms resolved over the next few days.

One week later, he undertook a second dive with new equipment. After 35 min underwater he once again became acutely dyspnoeic and began ascent. At 6 m his symptoms again worsened and he surfaced rapidly at which point he coughed up a large volume of frothy fluid and had recurrence of chest tightness. His symptoms completely resolved with 30 min of oxygen therapy. Eight days later he had a third episode, but this time while swimming on the surface in rough waters without a wetsuit. After swimming 400 m, he developed identical symptoms and again coughed up a large amount of white frothy fluid. On this occasion, breathlessness resolved over 5 min without any intervention.

The patient was transferred on a commercial flight to Tasmania, his home State, for further investigation. On arrival, he was afebrile and normotensive (130/75 mmHg) and in controlled AF at a rate of 80 b.p.m. Heart sounds were normal. Oxygen saturation on breathing room air was 98% and neurological examination was unremarkable. He had scattered inspiratory crackles at both lung bases. He had a full range of painless motion of all joints and no skin rashes. Investigations showed a normal chest radiograph and high resolution computed tomography showed some minor atelectasis at the left lower lobe. Electrocardiogram (ECG) confirmed AF with a normal axis. Cardiac enzymes were all normal. Lung function tests showed normal ventilatory function and gas transfer. An echocardiogram was normal with no evidence of diastolic dysfunction. He reached stage III of the Bruce Protocol for a total exercise time of 9 min and achieved a maximal heart rate of 173/min with no symptoms or arrhythmias and no ischaemic ECG changes. Coronary angiography confirmed no significant coronary artery disease.

The patient's symptoms were considered most consistent with recurrent acute pulmonary oedema associated with water immersion. Unfortunately, his late presentation to medical services meant that the typical physical and radiological findings could not be confirmed. Other potential causes of his symptoms, such as barotrauma, were thought unlikely as the symptoms initially occurred at depth and quickly resolved on the surface. Acute decompression sickness was also unlikely as he had no neurological deficits and symptoms recurred during a surface swim. Aspiration was thought unlikely given he clearly reported no salt water aspiration before any of the events. Negative pressure pulmonary oedema developing during vigorous attempts to breathe against a faulty regulator was also considered unlikely as new equipment was used during the second dive and the original regulator had been thoroughly checked and no fault found.[8,9] Sotolol may have contributed to his symptoms by impairing the myocardial response to increased preload.[10]

In summary, acute pulmonary oedema during diving is a very uncommon problem, particularly in warm waters, and has been reported in association with AF only once before.[1] We have recommended that the patient should not return to professional diving, although previous reports suggest that a return to more conservative diving activities can occur without recurrence.[3] Although AF is considered an exclusion for recreational divers and a contraindication for professional divers, this is based on consensus opinion only.[11,12] Currently there is no way to identify those divers who may develop pulmonary oedema while underwater.

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