Case reports

Two fatal cases of immersion pulmonary oedema – using dive accident investigation to assist the forensic pathologist

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Abstract

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Immersion pulmonary oedema (IPE) is being increasingly recognized in swimmers, snorkellers and scuba divers presenting with acute symptoms of respiratory distress following immersion, but fatal case reports are uncommon. We report two fatal cases of probable IPE in middle-aged women, one whilst snorkelling and the other associated with a scuba dive. In the snorkeller's case, an episode of exercise-related chest tightness and shortness of breath that occurred 10 months previously was investigated but this proved negative, and she was on no medications. However, at autopsy, moderate left ventricular hypertrophy was noted. The scuba diver had suffered several previous episodes of severe shortness of breath following dives, one being so severe it led to cyanosis and impaired consciousness. At inquest, the pathologist's diagnosis was given as drowning and IPE was not mentioned. Expert input from doctors trained in diving medicine should be compulsory in the investigation of diving deaths, and forensic pathologists should be properly trained in and have guidelines for the conduct of post-immersion and post-diving autopsies.

Key words
Immersion, pulmonary oedema, deaths, snorkelling, scuba diving, autopsy, coroner's findings, case reports

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Introduction

Immersion pulmonary oedema (IPE) is being increasingly recognized in swimmers, snorkellers and scuba divers presenting with acute symptoms of respiratory distress following immersion, but fatal case reports are uncommon.[1–10] We report two fatal cases of probable IPE, one in a snorkeller and the other in a scuba diver, the latter case having similarities to one of the cases described by Edmonds et al.[1] These were referred to the coroner and, therefore, are in the public domain.

Case 1

A 56-year-old woman got into difficulties while snorkelling with dolphins and died the same day shortly after admission to hospital. She was described as a fit, healthy person on no medication. She was 1.68 m tall, weighing 71 kg (BMI 25.2 $kg \cdot m^{-2}$). About 10 months earlier she had had an episode of chest tightness and shortness of breath after strenuous exercise. She had a normal ECG at that time. Other investigations were negative and the episode resolved spontaneously over several days. More recently she had several shorter episodes of shortness of breath after strenuous exercise, but did not seek medical attention. She had some snorkelling experience, including three days on the Great Barrier Reef before travelling to New Zealand.

She was one of 14 snorkellers on a dolphin encounter trip, crewed by four staff. They were given a safety briefing, including a video, and provided with wetsuits. The victim completed a waiver form on which she did not list any medical problems. Water temperature was 15–16^{O}C and weather and sea conditions reasonable. They were instructed to go no further than 80 metres from the boat and occasionally, as they swam with the dolphin pod, the skipper moved the boat closer to the group. They had three swims, returning to the boat each time to move a short distance to where the dolphins were. It was during the third swim that the victim got into difficulty, the first two swims being apparently uneventful.

After about 10 minutes' interaction with the dolphins, a crewman noticed one snorkeller in distress with their fist raised (the prescribed signal for assistance). The vessel immediately drove towards her and the swimmer responded to calls with a thumbs-up signal. She then rolled over onto her back but had the snorkel in her mouth, and came up coughing and put her fist up again, along with the two people now with her, who assisted her back to the boat. She said "*I can't breathe*"; a small amount of froth was observed on her bottom lip. Her difficulty breathing worsened, and a friend helped her to remove the top of her wetsuit and sat her on the floor, raising her arms in the air and holding her head up. She was coughing up some foam from time to time. The swimmers were called back to the boat, an emergency call was made and the boat headed to shore. The maintenance crew and an ambulance were requested to meet them as the woman's condition had deteriorated. By the time the boat arrived at the jetty she was so weak she could no longer hold herself up.

The ambulance crew arrived as the vessel arrived, and they took oxygen, a defibrillator and their portable packs on board within a minute of the vessel mooring. The victim appeared to be in cardiac arrest, as she was deeply cyanosed and unresponsive. A rapid ABC assessment revealed an obstructed airway, with vomit and blood-stained sputum. The woman was turned on her side and her airway cleared with a finger sweep. The defibrillator showed "*sinus rhythm at about 80 beats per minute*", and a strong pulse was detected. An oropharyngeal airway was inserted after clearing her airway and she was placed on 100% oxygen. All the while, the victim continued to vomit and she had to be repeatedly rolled onto her side to clear her airway. She was placed on the stretcher, and started breathing for herself, at about eight breaths per minute, but "*quite distressed breaths*". She opened her eyes to command and became responsive and complained that she could not breathe.

On admission to the local hospital, the victim was breathing in a laboured fashion at about 40 breaths per minute, pulse rate 127 per min, blood pressure normal and temperature 35.4^{O}C. Pulse oximetry was not possible because of peripheral shutdown. An intensive care retrieval team was requested from the regional tertiary-level hospital but this could not be despatched as they had been sent to a mining accident. Because of repeated airway compromise from vomiting and further deterioration, it was decided to intubate the victim. This proved difficult though eventually successful. Despite this, ventilation was difficult and she continued to deteriorate. Cardiac arrest occurred about 30 minutes later. Despite continued resuscitation for approximately 35 minutes, the victim died.

Post-mortem examination by an experienced forensic pathologist (MS) demonstrated congestive heart failure in the context of cardiomegaly and exertion. There was no coronary artery disease and no circumstances indicating drowning or near drowning. The heart weighed 443 g, 140–160 g greater than expected for her body height and weight. The larynx, trachea and major bronchi were internally unobstructed but contained frothy sputum. There was no macroscopic evidence of aspiration of vomitus. The left lung weighed 869 g and the right 954 g (expected normal weight for body size would be 250–300 g each). Trace amounts of alcohol but no other drugs were detected. Microscopy of the myocardium showed mild regular hypertrophy only with no old or new ischaemic injuries. Her cardiomegaly might in part be the result of a training effect and/or mild hypertension. The cause of death was given as "*immersion pulmonary* [o]*edema syndrome*".

Case 2

In 2002, a 51-year-old, fit female (height 171 cm, weight 71 kg, BMI 24.3 kg·m^2) on no medications had done 20 dives over two years. She undertook a shore dive to a maximum depth of 11.9 metres in a sheltered bay (water temperature 17–18 ^{O}C). She experienced difficulty with breathing during the dive and over a 12-minute period made three ascents to the surface (accompanied by her buddy), from various depths, before finally returning to the surface. For the whole time, her regulator remained in her mouth. After the final ascent, she was noted to appear panicked and dyspnoeic, but mentally coherent and responding appropriately to her buddy. She was escorted to a mooring buoy to rest. Her respiratory condition deteriorated rapidly, with pink froth coming from her mouth, so she was towed to shore by her buddy, keeping her head above water. By arrival at the shore, she was unresponsive with no respirations or pulse. Basic life support (BLS) was commenced; ambulance officers assisted but the diver was unable to be resuscitated.

On examination all her diving gear was functioning normally except the diaphragm of the octopus regulator, which was not fitted correctly (this was not used at any stage). Calculations of air usage from her cylinder (11.4 L internal volume, 85 bar remaining) indicated she had breathed approximately 1,400 litres during her 12-minute dive.

Autopsy recorded pale pink, frothy fluid in the trachea, lower airways and bronchioles. There was no evidence of lung barotrauma, but hilar nodes showed histological features of sarcoidosis. There was minor mitral and tricuspid degeneration, normal coronary arteries, minor quantities of air in the brain, heart and liver consistent with post-mortem gas, and a degree of cerebral oedema. She had fractured ribs consistent with BLS. Post-mortem radiology was not performed. The cause of death (with a considerable degree of uncertainty) was given by the pathologist as drowning. However, witness reports stated that at no stage was the regulator out of her mouth underwater, and her head was always above water during rescue. These reports were confirmed by interview during the process of an independent investigation by one of the authors (DS).

Further information about her diving history was relevant, and obtained during the independent investigation. She had had three previous episodes of significant dyspnoea precipitated by scuba diving, one episode being so severe it led to cyanosis and impaired consciousness. She recovered fully from each of these episodes.

Discussion

For Case 1, it is not possible to completely rule out salt water aspiration; however, the post-mortem findings were consistent with congestive cardiac failure and cardiomegaly. In addition, despite continued emesis, there was no evidence of aspirated vomitus detected. The striking feature of this case is the rapidity of development of severe symptoms. This has recently been reported in other, non-fatal cases.[9]

On review of the records for Case 2, it was considered that this case was consistent with death due to scuba divers'

pulmonary oedema (SDPE – also known as immersion pulmonary oedema, IPE), and that the preceding non-fatal episodes were also consistent with SDPE. At the time of the coroner's hearing, a report was submitted to the coroner disputing the post-mortem conclusions. The dive buddy had indicated that at no stage was the regulator out of the diver's mouth, and ascents were controlled. Conditions on the day were calm, and the dive regulators were functioning normally, making salt water aspiration most unlikely. The 12-minute dive to 12 msw made pulmonary decompression illness unlikely.

SDPE appears to be under-reported and may not be recognised at postmortem because the pulmonary findings may be so similar to drowning.[1] IPE is an entity in which apparently fit adults develop sudden-onset pulmonary oedema while swimming, snorkelling or scuba diving, without any circumstantial evidence that this is part of a drowning or near-drowning event or necessarily related to any underlying cardiac disease.[1–10] There is an indication that older individuals with pre-existing hypertension or cardiovascular disease may be at higher risk than younger individuals. It is likely that Case 1 did have some degree of untreated hypertension.

The most common presentation is acute-onset coughing and shortness of breath while participating in the activity. The great majority of sufferers survive with symptoms resolving within 24 hours or less with or without supportive treatment.[3,4] Subsequent investigation of those affected often shows no or only minor underlying cardiac disease, which would be the alternative explanation for sudden onset pulmonary oedema. Initially there was a strong association seen between immersion in cold water and the onset of this condition, but it has been described in mild and even warm (swimming pool) conditions.[5,6] There has been a strong association with relatively extreme exertion (military diver training and triathlon sport) and prior overhydration, but cases have also been described in recreational settings.[1–10]

From a forensic pathology perspective, the appearance of the lungs cannot be reliably distinguished from classic drowning by autopsy findings alone, reinforcing the universal need to correlate the circumstances of death with the autopsy findings. In both these cases, the circumstances of death suggest drowning was unlikely, perhaps with slight uncertainty for Case 1 because of the brief episode of snorkel immersion. We believe that expert input from doctors trained in diving medicine should be compulsory during investigation of diving deaths, and forensic pathologists should be properly trained in and have guidelines for the conduct of post-immersion and post-diving autopsies.[11] Through a sequential analysis of events leading to diving fatalities a more comprehensive picture is constructed, which identifies causative triggers, disabling incidents and injuries and may be used for future prevention of accidents.[12] It may well be that IPE has been the mechanism of death in immersion in other cases attributed to drowning without such eye-witness observation to provide the distinction. Immersion deaths of victims dying alone, or sufficiently removed from their companions for them to give any reliable account of the precise circumstances, could well represent overlooked IPE.

Similar mechanisms may also explain sudden-onset pulmonary oedema in 'stress situations' not involving water immersion in the absence of structural heart disease, and is possibly related to the otherwise well-documented, stress-induced Takotsubo cardiomyopathy.[13] Pre-existing high vascular resistance and exaggerated vascular response to cold stress in divers who developed pulmonary oedema has been reported, although this was not a consistent finding in subsequent reports.[14–16] In one series, over a quarter of individuals sustaining IPE may have had reversible myocardial dysfunction.[17] Current research at Duke University Medical Centre is seeking volunteers who have suffered IPE to study whether there may be a genetic disposition in a small proportion of the population who carry a number of gene markers that may be associated with IPE.

Another issue is controversial. Should scuba divers be advised they are safe to return to diving after a non-fatal episode of IPE? When evaluating diver risk, the worst possible consequence is a fatality. Certainly all should be evaluated for manifest or occult cardiovascular disease.[18] IPE is not a benign condition. It also appears to be idiosyncratic, not occurring with every dive or immersion. Case 2 is remarkably similar to other reported cases, with significant periods of time between IPE episodes.[1,8] Unfortunately Edmond's case developed fatal IPE even after extensive land-based investigations had detected no abnormality.[1] Until a full epidemiological study is performed, or markers of IPE risk are identified, we recommend extreme caution when evaluating individuals seeking to return to diving after an episode of IPE.

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Conflict of interest:
FM Davis is Editor of *Diving and Hyperbaric Medicine*. Peer review and acceptance of this paper was entirely the responsibility of the European Editor, Dr Peter Müller.

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