Case report

Scuba divers' pulmonary oedema: recurrences and fatalities

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Abstract

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Scuba divers' pulmonary oedema (SDPE) is an increasingly recognised disorder in divers. We report three fatal cases of SDPE, demonstrating its potentially serious nature even in the absence of underlying cardiac disease demonstrable clinically or at autopsy. This, together with the frequency of recurrences, has implications on assessing fitness for subsequent diving, snorkelling and swimming. The differential diagnosis of this disorder is also considered, as is its possible inducement by salt water aspiration and its relationship to drowning.

Key words
Scuba diving, pulmonary oedema, salt water aspiration, deaths, case reports

Introduction

Scuba divers' pulmonary oedema (SDPE) was first recorded in 1981.[1] Comprehensive reviews have been prepared since by various authors.[2–6] In these reviews, the physiological bases of the disorder have been canvassed; it is one type of immersion pulmonary oedema (IPE). SDPE presents with scuba divers developing fast shallow respirations, dyspnoea, fatigue, cough and white or sometimes blood-stained frothy expectoration. The signs include hypoxia and the auscultatory evidence of pulmonary oedema. Investigations reveal impaired spirometry and reduced lung compliance, hypoxaemia and characteristic radiological (plain chest X-ray or CT scan) abnormalities.

SDPE is said to be more frequent in older divers and those with cardiovascular pathology.[1–6] It tends to recur in at least 30% of cases.[5] Exertion during the dive is often not excessive and frequently the condition becomes more evident during ascent or surface swimming. Spontaneous resolution is often prompt after leaving the water. Only one death has been reported in the traditional medical literature and this was based on significant pre-existing cardiac pathology.[5] The latter is characteristic of some of these SDPE cases and is one aetiological feature that may be amenable to correction.

These three case histories illustrate the difficulty in predicting the development of non-cardiac based SDPE, the significance of recurrences and the possibility of death from this disorder. They have implications regarding appropriate advice that is given to affected divers.

Case history 1

Incident 1: A 51-year-old female nurse had no significant past medical history other than a mild allergic diathesis in early life, presenting with eczema and hay fever. She was an experienced scuba diver, logging over 900 dives without incident and possessing open-water and deep-diving qualifications. She was considered a conservative but enthusiastic club diver. The day before the incident she had completed two non-decompression, computer-assisted dives in an area well known to her. The first was to 24 metres' sea water (msw) for 50 minutes, followed by a surface interval of three hours; the second to 7 msw for 10 minutes, aborted due to currents and poor visibility. That afternoon and night she consumed 70 grams of alcohol, together with other fluids.

The following day, she felt well, although a little fatigued. At 0800 h she commenced a dive profile that she had undertaken on other occasions without difficulty. This involved a 30-metre surface swim, fully equipped but finning on her back and with the regulator out. The conditions were described as perfect, and the current was considered "*moderate at the worst*". Although she reported that she did not experience any aspiration, she did state that the wash from a boat splashed over her head once, causing her to cough and swallow some sea water. Later, during this four-minute swim, she became dyspnoeic. Her companion observed that it was a "*tough swim*" and that her lips appeared cyanotic and her breathing rate was rapid during the minute she spent resting on the marker buoy. In subsequent interrogations, she denied any salt water aspiration, chest discomfort, palpitations or syncopal sensation at that time.

Because they thought there could be less current at depth, they commenced the dive but only reached about 12 msw in one to two minutes. They aborted the dive after three minutes, due to her progressive dyspnoea and feeling fatigued. They ascended slowly, over about five minutes, before surfacing near the shore. She was then assisted in walking and removing her equipment.

Her coughing was frequent with expectoration initially whitish but becoming pink and frothy and she was aware of fluid rattling in her chest. She was dyspnoeic and cyanotic, with a grey appearance. She improved somewhat over the next quarter of an hour and was then able to walk unassisted. Ambulance paramedics administered high-concentration oxygen, until the medevac helicopter arrived. In telephone discussion with the DAN diving emergency service (DES), the clinician heard her wheezing and noted her complaints of dyspnoea and a "*rattling*" in her chest. She was transferred to the metropolitan hospital, breathing oxygen administered via a simple face mask.

Her vital signs on admission at 1045 h were not grossly abnormal, with a heart rate of 100 beats per minute and a respiratory rate of 24 breaths per minute, but she still had a persistent, non-productive cough with wheezing and crepitations at both lung bases. She was continued on oxygen and bronchodilators were administered. The chest X-ray showed minor linear basal densities, more on the right, consistent with interstitial oedema. All other investigations (ECG, lung function, electrolytes, biochemistry, liver function, oxygen saturation) were normal. The respiratory difficulty settled by 1500 hours and she was discharged the following day, for later review. Then, her lung function tests showed improvements of 18% in forced vital capacity, increasing to 26% following administration of a bronchodilator. The original impairment was considered to be consistent with increased airway reactivity associated with lung damage. A mild neutrophil leucocytosis was similarly explained. There were no other symptoms or signs suggestive of decompression sickness or pulmonary barotrauma and the dive profile was not indicative of these disorders.

A month later, a specialist cardiologist consultation included clinical assessment, ECG, stress testing and transthoracic echocardiograms, without any abnormality being detected. He concluded that the episode of pulmonary oedema was non-cardiogenic and that the patient had normal cardiac function. Repeat lung function testing at the same laboratory showed normal lung values and an asthma provocation test was negative. There was an improvement in lung volumes compared to the previous tests.

Her enthusiasm to return to diving and to re-establish her DAN diving insurance for future overseas diving trips led to consultations with at least six diving medical specialists. The diagnoses were divided between SDPE and the salt water aspiration syndrome (SWAS), and advice varied from unfitness for any diving (snorkel or scuba) to approval for unrestricted diving. She considered the conflicting advice available and also attempted her own research on this subject, and then resumed diving.

Incident 2: Almost a year later, now with another 54 logged dives, and with no further medical history apart from the incident above, she died whilst diving. She was participating in a night dive from shore. There was a moderate wind and the surface was choppy. Surface water temperature was about 22^{O}C reducing to 19^{O}C at depth and was described as comfortable. She was wearing a semi-dry suit.

The victim was with three others, in two buddy pairs. They swam on the surface for about 30 metres before descending and working along the sloping bottom to a maximum depth of 18 msw. For most of the dive the victim appeared to be fine and responded affirmatively to the buddy's regular 'OK?' signals. However, after about 25 minutes, at a depth of 14 msw, she signalled that she was 'not OK'. They decided to return and they swam underwater up the slope and towards the shore. Each time the buddy enquired if she was OK she responded in the negative. On reaching a depth of 7 msw, the buddy held her hand and they slowly ascended and surfaced in a sheltered area, with a dive time of 37 minutes.

At the surface, she vomited a brown, lumpy liquid. She was trying to cough and had an audible wheeze. She stated faintly that she could not breathe and she continued to vomit. Her BCD was inflated and she rolled over onto her back as the buddy towed her towards the shore. The buddy could hear her wheezing and struggling to breathe. She was still conscious and complained that she could not breathe, but tried to kick her legs to assist the buddy towing her. The buddy towed her approximately 100 metres to thigh-deep water beside rocks. She was assisted onto the rocks. It was believed that she did not inhale any water during the rescue.

She then became unconscious and apnoeic, and her buddy commenced basic life support. This produced regurgitation of stomach contents and some bloody sputum. Others assisted until the paramedics arrived about 15 minutes later. They implemented advanced life support but she failed to respond.

At autopsy the lungs were oedematous, weighing over 1.4 kg, and did not appear unduly hyperexpanded. There was no pathological evidence to indicate other causes of death, including previous or recent cardiovascular disease. The heart weighed 310 g. Toxicology was negative. The pathological diagnosis of acute pulmonary oedema was made.

Case history 2

This 45-year-old woman was apparently healthy and had become certified as an Open Water Diver one week earlier, having completed four open-water training dives. She was then participating in an Advanced Open Water course and had completed three uneventful dives on the previous day to a maximum depth of 7 msw, with a surface interval of 8 hours between the last two dives.

On the day, the water was calm and clear with visibility of 10–15 msw, and the dive was at slack water. The victim was

with a group of six students, accompanied by an instructor and a divemaster. They descended to a depth of 26 msw and knelt on the sea bed while answering questions on a slate. The duration of the dive was 16 minutes and she had completed other narcosis tasks. The victim then gave a low-on-air hand signal. The instructor noted that her contents gauge read 120 bar and gave her his 'octopus' regulator to breathe on briefly while he breathed on her demand valve, to check that it was 'OK'; it appeared to be functioning normally. She then took back her own regulator. However, a short time later, she again signalled she was low on air before starting to ascend. The instructor indicated to the others to remain on the sea bed with the divemaster and caught hold of the victim by her buoyancy compensator. They then ascended together while using his buoyancy to control their ascent rate. Soon after departure he noticed she seemed to be having some difficulty with her breathing, taking rapid, short, shallow breaths. However, she refused the offer of his secondary regulator. She then ceased to respond to his signals. The ascent was described as controlled and at a rate of around 15 msw per minute. On surfacing, the instructor asked if she was 'OK' to which she replied "*No, I don't feel good*" before rolling onto her side, unconscious. Shortly afterwards, white froth began to flow from her mouth.

The instructor then towed the victim some 30 metres to shore, intermittently providing rescue breaths, despite the continued flow of frothy sputum. Another diver assisted the victim onto the shore where she was assessed as unconscious and apnoeic. Basic life support was commenced and was complicated by vomitus, water, bile and froth obstructing her airway. After about ten minutes, another diver arrived with an AED defibrillator which indicated that no shock be given. At this time the victim had fixed, dilated pupils.

Paramedics arrived soon after and commenced advanced life support. A shockable cardiac rhythm was briefly created although subsequent defibrillation failed to restore sinus rhythm. There was continued difficulty ventilating the victim as the airway appeared to be obstructed by fluid.

An equipment check on the beach showed the remaining air at 90 bar. Examination of her equipment by the police diving branch subsequently showed no abnormality in equipment or gas, except for the hose to her primary regulator. This was kinked (longstanding) and this kink may have restricted the air flow. However, a subsequent test dive with the equipment failed to elicit this restriction, despite using various activities, positions and depths up to 29 msw.

The victim had passed a fit-to-dive medical but had omitted to mention that she had taken dexamphetamine (25–30 mg daily) for adult onset attention deficit hyperactivity disorder and also suffered from migraine, though rarely. She may have discontinued this medication before diving as no drugs were detected by toxicology at autopsy.

Autopsy X-ray two days after death showed generalized air distribution throughout the body and all the vascular system. This was attributed to post-mortem decompression artifact possibly aggravated by the resuscitative attempts. She was slightly overweight (height 176 cm; weight 84 kg; BMI 27). The heart weighed 360 g and was normal with minor degrees of atheroma and up to 20% narrowing of the coronary arteries. No evidence of infarction or fibrosis was seen, but there was fine patchy replacement fibrosis in the heart on histology, which is not explained. The right and left lungs weighed 915 g and 740 g respectively and were well-expanded and the parenchyma showed extensive pulmonary oedema but no congestion. There were gastric contents in the upper airways.

The pathological diagnosis of acute pulmonary oedema was made. As the symptoms commenced and progressed at maximum depth and as there was no preceding ascent, both decompression sickness and pulmonary barotrauma diagnoses were dismissed.

Case history 3

Another death was mentioned as an unreferenced addition in a previous review of SDPE.[3] This case probably originated from a DAN report of a fatality in 1996.[7] This was followed up with the original source and the following information was elicited.

A 51-year-old experienced, female diver undertook an uneventful, short, shallow dive with her husband. On surfacing she became dyspnoeic. She was towed with her buoyancy compensator inflated and allegedly with her head above water. She was then brought on board the diving boat where she lost consciousness and died despite resuscitation efforts. Autopsy revealed no evidence of pulmonary barotrauma, air embolism or decompression sickness. The lungs were extremely oedematous and frothy pink fluid filled the airways. There was some evidence of arteriosclerosis – the left anterior descending coronary artery had a stenosis of over 50% – but the coronaries were still patent. There was no evidence of previous or recent cardiac disease.

The pathological diagnosis of acute pulmonary oedema was made.

Discussion

Pons et al described SDPE as a rare event in healthy individuals.[8] The actual incidence is unknown, but it is likely to be under-diagnosed.[3–6,8] Deaths from SDPE are probably under-reported because the disease is not a high profile one (even amongst diving clinicians) and pathological findings are similar to those of drowning.[9,10] The latter diagnosis is often the default one for those who die in the ocean and have heavy, fluid-filled lungs. Differentiating drowning from SDPE pathology is a complex and questionable procedure,

not achieved at most autopsies. Also, a diver incapacitated by acute pulmonary oedema is then susceptible to superimposed water aspiration, with drowning obliterating the original pathology. The identification/distribution of diatoms is unlikely to be of value, as both can occur with immersion deaths. There is no single pathognomonic discriminator. It is possible that *emphysema aquosum* may be more typical of drowning pathology, but its aetiology is presumed to be associated with bronchoconstriction and this occurs also with SDPE.

Recurrences of SDPE have been reported in up to 30% of cases. This is likely to be a considerable underestimate of the actual risk, as treating clinicians usually do not perform long-term reviews on successfully treated cases. Also, contact may not be possible with this itinerant group and some divers affected by SDPE may avoid the risk of a recurrence by avoiding exposure to the cause – scuba diving or snorkelling. Recurrences may occur in both surface swimming and diving activities; the real recurrence rate is unknown.

The one death from SDPE that has been reported in the traditional medical literature was associated with significant cardiac pathology – in a diver with hypertension, hyperlipidaemia and arteriopathy and who sustained a cardiac arrest whilst swimming back to shore. He died 72 hours later from cerebral oedema.[5] He had suffered a SDPE episode that had been well documented, eight months previously. The problems of cardiac-based SDPE have already been canvassed and warnings given regarding the risk of subsequent immersion and diving.[6]

Other causes of pulmonary oedema that may occur with scuba diving should be considered in the differential diagnosis of SDPE. These include existing cardiac disease and diving- or immersion-induced diseases, e.g., salt water aspiration and the drowning syndromes, gas-induced pulmonary toxicity, dysbaric lung disease and pulmonary decompression sickness. Certain marine envenomations, especially the Irukandji syndrome, cold urticaria, asthma and other medical disorders may produce or simulate pulmonary oedema and be aggravated by the diving environment and equipment.[11]

Most differential diagnoses to explain the initial incident in Case 1 had been excluded by the dive profile or by subsequent medical assessments and investigations. The remaining differential diagnosis is what has been termed the salt water aspiration syndrome (SWAS), which is described in detail elsewhere.[11] Distinguishing between SDPE and SWAS is a difficult diagnostic conundrum. It is possible that sea water aspiration may precede or even induce the development of SDPE in some cases (as may be so in Case 1) by damaging pulmonary capillaries and then exposing them to the increased negative inspiratory pressures experienced with scuba diving, snorkelling and immersion.

SWAS has many clinical features similar to SDPE.[12,13] The dyspnoea, cough and expectoration are common to both, as are reduced lung volumes, arterial hypoxia and rapidly changing radiological signs in the lungs. The clinical manifestations of SWAS, such as fever and rigors, nausea, headache, muscular pain and mild leucocytosis are probably due to the combination of the lung pathology of aspiration and associated cold exposure, in the original series. The main differentiation, clinically, is that SWAS tends to develop soon after the dive whereas SDPE develops during the immersion, and is aggravated with the ascent.

Cases of both SDPE and SWAS have a rapid improvement with oxygen supplementation, and so the initial rescue from the water and conventional diver first aid treatments are applicable to both.

Subsequent management of the SDPE cases is hampered by the relatively few case histories documented. The medical advice to be given to victims of SDPE, even those without cardiac pathology, should probably be based on the high risk of recurrences, the possibility of death and our failure to clarify what environmental conditions, apart from immersion, precipitate the event.

Conclusions

SDPE is a serious illness amongst scuba divers. It tends to recur, even without known predisposing factors (other than age and immersion). Cardiac pathology may be influential in some cases and salt water aspiration in others. However, it is potentially lethal even in those without pre-existing clinical or demonstrable cardiac disease and without significant cardiac pathology, as detected at autopsy.

We present, for the first time to our knowledge, evidence of fatal consequences of SDPE without any significant demonstrable cardiovascular pathology.

Advice against further immersion (e.g., snorkelling, scuba diving) exposure in those victims who survive the first episode, is probably warranted. The illness and fatality rates are not known, but are probably underestimated in the diving medical literature.

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References

1 Wilmshurst P, Nuri M, Crowther A, Betts I, Webb-Peploe MM. Forearm vascular response in subjects who develop recurrent pulmonary oedema when scuba diving: a new syndrome. *Br Heart J.* 1981;45:349.

2 Lundgren CE, Miller JN. *The lung at depth. Lung biology in health and disease*. Vol 132. New York: Marcel Dekker Inc; 1999.
3 Slade JB, Hattori T, Ray CS, Bove AA, Cianci P. Pulmonary oedema associated with scuba diving. *Chest.* 2001;120:1686-94.
4 Koehle MS, Lepawsky M, McKenzie DC. Pulmonary oedema of immersion. *Sports Med*. 2005;35:183-90.
5 Cochard G, Arvieux J, Lacour J-M, Madouas G, Mongredien H, Arvieux CC. Pulmonary edema in scuba divers. Recurrence and fatal outcome. *Undersea Hyperb Med*. 2005;32:39-44.
6 Edmonds C. Scuba divers' pulmonary oedema – a review. *Diving Hyperb Med*. 2009;39:226-31.
7 Diver Alert Network. *Report on decompression illness and diving fatalities*. 1998 Edition. Durham, NC: Divers Alert Network; 2000. Record No: 3696. p. 101.
8 Pons M, Blickenstorfer D, Oechslin E, Hold G, Greminger P. Pulmonary oedema in healthy persons during scuba diving and swimming. *Eur Respir*. 1995;8:762-7.
9 Christe A, Aghayev E, Jackowski C, Thali MJ, Vock P. Drowning – post–mortem imaging findings by computed tomography. *Eur Radiol*. 2008;18:283-90.
10 Lorin de la Grandmaison G, Paraire F. Place of pathology in the forensic diagnosis of drowning. *Ann Pathol*. 2003; 23:400-7.
11 Edmonds C, Lowry C, Pennefather J, Walker R. *Diving and subaquatic medicine*. 4th ed. London: Arnold; 2002.
12 Coulange M, Rossi P, Gargne O, Gole Y, Bessereau J, et al. Pulmonary oedema in healthy SCUBA divers: new physiopathological pathways. *Clin Physiol Funct Imaging*. 2010;30:181-6.
13 Edmonds C. A salt water aspiration syndrome. *Mil Med*. 1970;135:779-85.

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