A Case of Recurrent Swimming induced Pulmonary Oedema in a Triathlete: The Need for awareness

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Abstract

This report discusses a rare case of a 55-year-old female triathlete who developed recurrent episodes of swimming-induced pulmonary oedema (SIPE). She had two hospital admissions with pulmonary oedema after developing breathlessness whilst swimming, including a near-drowning experience in an open-water swim. With increasing popularity of triathlon and open water sports, this case highlights the importance of a greater awareness of SIPE amongst health professionals, event organisers and athletes. This report explores the previous reported cases in triathletes and those who have suffered recurrent episodes. It is paramount that an accurate diagnosis is made as these individuals may be at an increased risk of future life-threatening episodes.

Word Count: 104

Key Word: Swimming induced pulmonary oedema, Immersion induced pulmonary oedema, Triathlon

Introduction:

Triathlon is one of the fastest growing sports in the world. The swim component of a triathlon is usually held in open water (fresh water lakes, rivers or sea-based). Swimming-induced pulmonary oedema (SIPE) is a rare cause of acute breathlessness in water-based events. It was first described in the 1980s in healthy scuba divers (Wilmshurst et al., 1989). More recently it has been described in open-water swimmers (Beinart et al., 2007), triathletes (Deady et al., 2006; Spiteri et al., 2011; Casey et al., 2014), military trainees (Mahon et al., 2002; Adir et al., 2004; Knutson 2010) and in an aqua-jogger (Wenger & Russi, 2007). The incidence has been estimated as 1.1% amongst divers (Pons et al., 1995) and 1.8% amongst combat swimmers (Adir et al., 2004).

With the growing popularity of these water-based events, it is important that medical professionals involved in the care of triathletes and swimmers are aware of this specific problem. This case highlights the need for awareness of SIPE amongst health professionals, event organisers and athletes. It is important that this condition is recognised early and accurately diagnosed with appropriate advice given as these individuals may be at increased risk of further life-threatening episodes in the future.

Method

Informed written consent was gained from the subject.

Case Report:

A fifty-five year old female amateur triathlete presented with a cough and shortness of breath to the event medical team following the swim leg of a sprint distance triathlon. She had started the 750m swim in a fresh-water lake (water temperature 17℃) feeling well and without any symptoms. Her chosen stroke was breaststroke and she wore a well-fitted wetsuit, which she had used during training. Within five minutes (approximately 150 metres from the start), she became increasingly breathless and was struggling to keep her usual pace. Her breathing worsened but continued although she sought assistance several times from the safety canoe. Eventually, she completed the swim after approximately 40 minutes. On shore, she was unable to start the bike leg of the event due to increasing breathlessness and sought assistance from the triathlon medical team in the bike transition area.

She was physically fit, with good exercise reserve. She had a history of depression for which she took 30mg of escitalopram. She had started open water swimming one year previously and had trained well over the course of the year. Eight weeks prior to the triathlon she had successfully completed a standard 42.2km marathon in a time of 4hr 45 minutes and a week prior to the triathlon she had completed a 1500m open water training swim uneventfully in around 40 minutes. In the weeks leading up to the marathon she had experienced an occasional irregularity of her heartbeat at rest. Her general practitioner performed an Electrocardiogram (ECG) and in light of her physical activity level referred her to the local cardiology team who requested an echocardiogram, 24-hour ECG monitoring and subsequent myocardial perfusion scan (results are shown in table 1). Although she had these tests performed before the triathlon she was yet to be fully informed of the results. She had been advised to avoid vigorous exercise whilst awaiting a follow-up cardiology appointment however she had chosen to ignore this advice.

On assessment at the triathlon medical tent she denied swallowing or aspirating water and had no chest pain. She was comfortable at rest, her oxygen saturations (SpO_2) were 88% on air, respiratory rate (RR) was 18 breaths/minute, blood pressure (BP) was 150/90 mm Hg and pulse (HR) 88bpm. Jugular venous pressure was not raised, and there was no pedal oedema. On chest auscultation there were bilateral coarse crackles to the mid-zones. Cardiovascular examination was normal with no murmurs heard. She was started on 4L of supplementary oxygen via nasal cannulae, which improved her SpO_2 to 97%. She was transferred by ambulance to the local emergency department (ED).

On arrival in the ED her observations were similar, with SpO_2 of 93% on air. Arterial blood gas (ABG) showed type I respiratory failure with a PO2 of 10.2KPa (normal range:11.1-12.2KPa). ECG exhibited T-wave inversion in lead III (figure 1). Chest radiograph (CXR) showed a normal heart size and clear lung fields. In light of the raised troponin of 0.39 (normal less than 0.04; see table 1), she was referred to the cardiology team with possible acute coronary syndrome (ACS). She received 40mg of intravenous furosemide in the meantime. Overnight her symptoms resolved and SpO_2 rose to 99% on air.

The next day, in light of the hypoxia on ABG and raised D-dimer, a CT pulmonary angiogram (CTPA) was performed. This excluded a pulmonary embolism and showed a ground glass appearance which probably represented pulmonary edema (figure 2). An echocardiogram was reported as showing left ventricular (LV) hypertrophy (LVH) with estimated antero-septal wall thickness of 18 mm and LV ejection fraction of >55%. There was appearance of a focal basal aneurysm of LV inferior free wall a similar previous echocardiogram (see table 1). Coronary angiography demonstrated normal coronary arteries. Although the diagnosis was unclear at this point, with possibilities including ACS, stress

cardiomyopathy or other causes of cardiomyopathy, since she had made a remarkable recovery she was discharged with instructions to follow up to her local cardiology team for further investigation. She was advised to avoid exercise for the next month.

Six weeks after this episode, she was reviewed by her consultant cardiologist with the results of her 24 hour tape and a stress myocardial perfusion scan which showed no evidence of inducible ischaemia. Following this, a cardiac Magnetic Resonance Imaging scan (cMRI), MRI Renal angiogram and a 24-hour urinary collection for catecholamines were organised to search for rare causes of flash pulmonary oedema. She was advised to refrain from strenuous exercise during this period. However, she still continued moderate intensity exercise without any symptoms.

Approximately two months following the triathlon, she attempted an open-water swim in a wetsuit. Five minutes after entering the water she became breathless and describes coughing up pink frothy sputum. She was struggling to swim and had to be assisted to the shore by other swimmers whereupon she was driven to the closest local hospital where she was found to be in pulmonary oedema. Once again she responded symptomatically well to intravenous furosemide. A battery of investigation was conducted because of the repeat presentation, including repeat coronary angiography, which was normal (A summary of investigations during the five day admission are shown in table 1). Subsequently the cMRI showed good cardiac function with no evidence of cardiomyopathy or myocardial infarction (results and cardiac measurements are shown in table 1). In light of recurrent presentation and investigations the diagnosis of Swimming Induced Pulmonary Oedema (SIPE) was made. She has not returned to open water swimming.

Discussion:

Most cases of SIPE are reported during or after water based events like diving or swimming, without any history of water aspiration. It is characterized by an acute onset of dyspnoea, cough and expectoration of frothy sputum, with evidence of pulmonary oedema on physical examination (Adir et al., 2004). There may also be radiographic findings of pulmonary oedema. Symptoms tend to rapidly resolve over the subsequent 24-48 hours with no apparent residual damage. Further investigation usually demonstrates normal underlying cardiac and pulmonary function (Slade et al 2001), although this may be less likely in older swimmer/divers as an episode of SIPE may unmask other subclinical disease (Peacher et al., 2014). In their study of a group of 36 subjects (mean age of 48 years) with a history of SIPE, Peacher et al (2014) found that 72% had at least one significant concurrent medical condition (e.g. hypertension, cardiac dysrhythmia/dysfunction/structural abnormality, asthma, diabetes mellitus, or overweight.) Other potential risk factors identified by Miller et al (2010) include the use of fish oils, longer races, female swimmers, wetsuit use and over-hydration. They hypothesised that the fish oil had anti-platelet and vasodilatory action which may promote pulmonary capillary leakage resulting in pulmonary edema. Over-hydration has previously been linked to SIPE. Weiler-Ravell et al (1995) reported a series of eight military swimmers in a group of thirty who developed pulmonary oedema and haemoptysis during a single 2.4km time trial in open sea. In the two hours preceding exercise each swimmer was instructed to drink approximately five litres of water to avoid dehydration. This larger circulating plasma volume was thought to be a contributing factor to the development of SIPE through increased cardiac pre-load in this report.

Pathophysiology:

Due to the sporadic nature of SIPE and the inability to reproduce it under experimental conditions, the pathophysiology still remains elusive (West et al., 1991). Predominant theories include cold water immersion leading to peripheral vasoconstriction and central blood pooling (Wester et al., 2001), which in turn increases cardiac preload (Edmonds, 2012). In combination with an increased cardiac output from strenuous exercise, this results in elevated pulmonary artery pressure. Increased hydrostatic pressure results in alveolar oedema and breakdown of the capillary-alveolar barrier (Tsukimoto et al., 1991; West et al., 1991). Casey et al (2014) suggest a modified explanation, describing that there is a limited increase in left ventricular (LV) stroke volume (SV) compared to a relatively greater rise in the right ventricular SV leading to an imbalance and subsequently fluid accumulation.

Interestingly wearing a wetsuit may increases the odds ratio of developing pulmonary oedema (Miller et al 2010). Carter et al (2011) suggested the wetsuit was a likely contributing factor in their case series as it was worn in all subjects, with one subject specifically reporting that an overly tight wetsuit had added to the sensation of dyspnoea. Tight-fitting wetsuits may themselves exacerbate increases in preload through compression of peripheral vessels, leading to increasing central blood pooling (Lungdren & Miller, 1999). Furthermore, chest constriction from the wetsuit may decrease lung volumes and cause relatively negative intra-thoracic pressures (Slade et al., 2001). Nevertheless, there are several cases of SIPE where a wetsuit was not worn (Weiler-Ravell et al. 1995; Adir et al., 2004).

Cases in Triathletes:

Numerous cases of SIPE have been described in open water swimmers and divers (Adir et al., 2004; Peacher et al., 2014), but there have been only sixteen) reported amongst triathletes

(Biswas et al., 2004; Boggio-Alarco et al., 2006; Deady et al., 2006; Stefanko et al., 2009; Carter et al., 2011; Spiteri et al., 2011; Casey et al., 2014, Yamanashi et al., 2015). However, Miller et al (2010) found an incidence rate of 1.4%, in a survey of 1400 triathletes who had self-reported symptoms suggestive of SIPE. These reports suggest under-reporting among athletes and that many triathletes may have suffered a minor episode of SIPE, which resolve quickly after exiting the water. Among the 16 cases reported, mean age was 41 years old, two-thirds were male and two had other co-morbidities of asthma and type I DM). Four cases had raised cardiac enzymes immediately post-event, thought to be related to cardiac ischaemia. Two-thirds had an abnormal chest radiograph on admission. In our current case, the clinical picture was less clear due to the initial echocardiogram findings of a small basal aneurysmal segment in the LV inferior free wall and a subsequent echocardiogram also showing an estimated antero-septal wall thickness of 18 mm to suggest the possibility of an intrinsic cardiac abnormality. Subsequently this finding was determined to be physiological on cMRI with no suggestive features of hypertrophic cardiomyopathy. On reflection, the wall thickness measurement of 18mm was felt to be an overestimate, as it did not correlate with the initial echocardiogram nor cMRI measurements.

Recurrent SIPE has been reported among triathletes previously (Carter et al., 2011; Spiteri et al., 2011) albeit predominantly among women. Carter et al (2011) highlights that two out of the three female triathletes in their group suffered with recurrence of SIPE. Among one, a 58-year-old female, suffered at least four episodes with symptoms starting within 15 minutes of swimming in each episode. However, not all of these episodes required hospitalization and between episodes she successfully completed two open water triathlons and multiple lake training sessions further confirming the variable nature of this condition. Spiteri et al (2011)

reports that their athlete subsequently returned to swimming with no further episodes and found starting swimming at a slower pace a useful aid.

Recurrence of SIPE appears to be unpredictable. Recurrence rates have been repoted between 17%-22% among scuba divers and swimmers (table 2) (Weiler-Ravell et al., 1995; Shupak et al., 2000; Adir et al 2004, Peacher et al., 2014). Table 2 highlights that recurrence appears not to be limited by age group, sport or conditions. Adir et al (2004) found among 70 military swimmers, 16 had a recurrence of SIPE. In all these cases, this took place at least three months after the first incident. It is not clear what predisposed them to recurrence. It is also very variable and dormant, Edmonds et al. (2010) describes a fatal case of a 51-year-old female recreational diver who logged 54 dives over the course of a year between episodes of SIPE. A post-mortem diagnosis of acute pulmonary oedema was made. This highlights the potential severity of the condition and recurrence is not uncommon.

Management for Clinicians:

It is essential that clinicians responding to calls at such events are aware of this condition and it's potential seriousness. In the pre-hospital setting, it is vital that appropriate resources are available for a safe water evacuation to prevent drowning. Immediate removal from water is vital and can resolve symptoms (Carter et al., 2011). After initial assessment including measurement of oxygen saturations and supplementary oxygen (if clinically indicated) patients are likely to require transfer to hospital for further investigations and management. Communication regarding the potential diagnosis of SIPE should be made when transferring patients, to alert colleagues of this relatively rare condition. The symptoms usually resolve spontaneously within 24-48 hours of presentation with a conservative approach of supplementary oxygen. Intravenous or oral diuretics may be required. It is recommended that

those who suffer with an episode of SIPE should have prompt evaluation of their cardiac and pulmonary physiology (Peacher et al., 2014).

In the acute setting, a CXR, ECG, echocardiogram, cardiac enzymes and possible coronary angiogram should be performed to rule out coronary disease causing pulmonary oedema. In this case the patient was extensively investigated including cardiac MRI due to possible structural abnormality seen on initial echocardiogram. Lack of awareness amongst the admitting team meant a delay in diagnosis of SIPE. Therefore this patient was not advised about the risk of potential recurrences. Patients should be made aware of the potential of recurrence after the initial diagnosis of SIPE in order that future swimming can be planned in a controlled environment to reduce the risk of drowning if a repeat SIPE episode were to occur, although it is acknowledged that episodes of recurrence are unpredictable.

Clinicians should suspect SIPE in athletes who are unusually short of breath during or after the swimming stage of a triathlon or open water swim race. Race organisers and medical staff should be educated in the recognition of SIPE and its management considering the increased participation in this sport.

Word count: 2341

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Table 1: Summary of investigations:

Investigation prior to Triathlon		
12 lead Electrocardiogram (Initial ECG at General practice)	Sinus rhythm 72 bpm. QRS minor notching and intra-ventricular conduction delay. Nil other abnormalities	
Echocardiogram	Focal basal aneurysm of LV inferior free wall. Inter-ventricular septal thickness of 11 mm, Ejection fraction of 57%.	
Myocardial Perfusion Scan	Normal with no evidence of inducible ischemia	
24hr Tape Monitor	Sinus rhythm ranging from 49 bpm to 123 bpm. Short PR interval of 109 milliseconds	
1st Admission to Hospital		
Observations on admission	Temp 35.6 BP 135/68 RR15 Sats: 93% on air BM;5.3 pulse 68	
Relevant Bloods test	Haemoglobin, 146 g/l; white blood cell count, 700 × 109/l; platelet count, 197 000 × 109/l. D-Dimer 1740 ng/ml (normal <250 ng/ml).	Sodium, 144 mmol/l (normal range 135–145); potassium, 3.4 mmol/l (3.4–5.1); urea 5.0 mmol/l (2.5–7.5); creatinine, 52 mmol/l (70–130). Troponin I – 0.38 (normal <0.04) 12 Hour Troponin - 0.82
Arterial bloods gas	pH: 7.47 (7.35-7.45) PCO_2 4.7 kPa (4.27-6.0) $P0_2$ -10.2 (11.1-14.4) HCO_3 – 27.0 cBase $(B)_c$ -3.0mmol/lL	
Chest Radiograph	Chest radiograph showed a normal heart size and clear lung fields	
Coronary Angiogram	Normal coronary arteries. LV gram: EDP 5mmHg. LV normal in function and size with mild basal inferior hypokinesia	
2nd Admission to Hospital (approximately 2 months post 1st admission)		
Observations on admission	Temp 36.8 pulse 57 BP 120/9- RR15 Sats:93% on air BM;5.3	
Chest Radiograph	There is increased shadowing in the right lower zone with loss of translucency. Rest of the lungs are clear.	
ECG	Sinus Rhythm with normal QT interval.	
Relevant Blood test	CRP 21 Troponin 194, 12 hr Troponin 103	
Exercise ECG	Good work load, Some ST depression in Inferior leads	
Coronary Angiogram	Normal Coronary arteries	
Investigation Post Discharge from 2nd Admission		
Cardiac MRI with Gadolinium	Mild thinning of the inferior wall, no evidence of aneurysm of inferior wall, no evidence of infarct on late gadolinium sequence and the appearances were deemed physiological. Ventricular measurements: Basal Septal wall (diastole) 7.6mm, Basal Anterior wall(diastole):6.5mm, Basal lateral wall(diastole):6.5mm.	
MRI Renal tract	MRA of the renal arteries was normal with no evidence of stenosis.	
72 hrs Urinary collection	Catecholamines were normal	

Table 2: Recurrent cases of SIPE documented in the literature

Study	No. Cases of SIPE described in report	Mean Age	N=Subjects with Recurrent episodes	Context	Comments PMH= Past medical history E= episode
Edmonds 2012	3 (F)	49	1(51F)	Scuba diving	Death prior to hospital admission (n=3) Previous episode of immersion pulmonary oedema (n=1)
Spiteri 2011	1(F)	37	1(37F)	Triathlete	Recurrent episode did not require hospitalization. E1= at half Ironman distance, Wetsuit worn, open sea, water temperature 18°C E2 = sprint distance, no wetsuit worn, calm open sea. Temperature 22°C
Carter 2011	3 (F)	48.7	2 (58,43F)	Triathletes	Further episodes of dyspnoea/haemoptysis during triathlon. Both athletes each had a total of 4 recurring episodes. Recurrent episode did not require hospitalization
Dwyer 2007	1 (M)	54	1(54M)	Scuba diving	PMH: chronic atrial fibrillation, gout
Glanvill 2006	1 (F)	48	1(48M)	Scuba diving	PMH: Rheumatic fever
Adir 2004	70 (M)	18.5	16 (18M)	Surface swimming	Recurrent episodes all occurred at least 3 months following 1st episode
Cochard 2005	5(4M,1F)	49	2(55,46M)	Scuba diving	Case 1: 55 male – 2 episodes 8 months part, 2nd episode died of cerebral oedema 72 hours after a cardiac arrest sustained while swimming on the surface to shore. Case 2: 46 male – reported similar symptoms shortly after surfacing 3 years prior to 2nd episode.
Slade 2001	8 (3M, 5F)	52	3(52M 53,55F)	Scuba diving	3 divers reported similar symptoms of cough and breathlessness during/post dive
Shupak 2000	21 (M)	18.5	6 (18.5 M)	Surface swimming	6 episodes were recurrent.
Hampson 1997	6(2M, 4F)	43.3	1(42M)	Scuba diving	Total of six episodes reported from diver. One admission to hospital. All occurred at greater than 35ft.
Weiler-Ravell 1995	8 (M)	18.3	2 (18M)	Surface Swimming	In recurrent episodes – athletes did not volume load as in initial study
Pons 1995	5 (4M, 1F)	27.8	1 (39F)	Multiple	Recurrent episodes (n=5) :2 episode whilst scuba diving and x3 surface swimming (lake swimming, 2 episode in a swimming pool). Required one admission to hospital post scuba diving

Figure Legends:

Figure 1: 12 Lead Electrocardiogram of 1st admission to hospital shows Normal Axis, T wave inversion in Lead III.

Figure 2: CT pulmonary angiogram on 1st admission show no pulmonary embolism and a mild ground glass appearance consistent with infection/oedema.