Swimming-induced pulmonary edema

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ABSTRACT

Swimming-induced pulmonary edema occurs when fluid accumulates in the lungs in the absence of water aspiration during swimming and produces acute shortness of breath and a cough with blood-tinged sputum. We report a case of a 58-year-old female athlete presenting with acute dyspnea during the swimming portion of a half-triathlon competition. She had complete resolution within 24 h of presentation.

KEYWORDS Athlete; cold water; immersion pulmonary edema; swimming; triathlon

Swimming-induced pulmonary edema (SIPE) is a type of pulmonary edema that occurs during surface or underwater swimming and causes the accumulation of fluid in the lungs without water aspiration and consequently symptoms of pulmonary edema.^{1,2} It has an estimated prevalence of 1.4% among triathletes.¹ We report a patient with acute dyspnea during the swimming portion of a half-triathlon competition.

CASE DESCRIPTION

A 58-year-old white woman presented with sudden dyspnea associated with cough productive of frothy pink sputum that began while swimming during a half-triathlon competition. After being pulled out of the water, she received bronchodilator treatments without improvement. She denied water aspiration. Her blood pressure was 133/87 mm Hg; heart rate, 102 beats per minute; respiratory rate, 24 breaths per minute; temperature, 97°F; and oxygen saturation, 83% on room air. She was in moderate respiratory distress but had no laryngospasm. She had scant bibasilar crackles and no lower limb edema. The chest radiograph showed bilateral alveolar infiltrates consistent with pulmonary edema (*Figure 1a*). Her cardiac enzymes were normal, and the brain natriuretic peptide level was 126 pg/mL (normal < 124 pg/mL). A transthoracic echocardiogram showed normal systolic and diastolic function with trace mitral and tricuspid regurgitation.

She required respiratory support with bilevel positive airway pressure ventilation and was given 40 mg of furosemide. After 4 h, her oxygen saturation improved, and she was switched to nasal cannula for oxygenation. After 24 h, a second chest radiograph revealed clear lung fields (*Figure 1b*), she no longer required oxygen, and her symptoms had resolved.

DISCUSSION

SIPE is an unusual complication that can occur during heavy exercise in water. It is often misdiagnosed and can rapidly deteriorate into life-threatening situations.³ Moon et al identified 58 deaths between October 2008 and November 2015 in the United States and Canada in triathletes during training or competition; 42 deaths occurred during swimming.² Wilmschurst et al suggested that swimming in cold water and hypertension are the most important factors that predispose athletes to develop SIPE.⁴ Other important factors during the event include ambient air temperature, exertion level, and anxiety.^{4,5} Potential risk factors include older age, female sex, left ventricular hypertrophy, and obesity.⁶ Postmortem studies have reported increased heart mass (9 of 20 available autopsies) and left ventricular free wall thickness (6 of 14 autopsies) in some triathletes.²

In 2006, Ludwig et al suggested the following criteria for SIPE: (1) acute onset of dyspnea or hemoptysis during or immediately after swimming; (2) no history of water aspiration, laryngospasm, or preceding infectious process; (3) hypoxemia, defined by an oxygen saturation <92% by pulse

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Table 1. Characteristics of patients with swimming-induced pulmonary edema in published reports

First, author, year, ref	Cases	Gender	Age (years)	Comorbidities	Water temp. (°C)	Presentation	Time of resolution (h)
Melau, 2019 ¹⁰	3	M = 2	30–40	None	14–17.5	Dyspnea (3) Chest tightness (1) Hemoptysis (3) SO ₂ < 92% (2)	<24
Shah, 2018 ¹¹	1	F	60	Repaired coarctation of aorta, bicuspid aortic valve	10	Dyspnea Cough Chest tightness	<48
Smith, 2017 ¹²	1	F	55	None	17	Dyspnea Cough SO ₂ < 92%	<48
Yamanashi, 2015 ¹³	1	M	38	None	21.4	Dyspnea SO ₂ < 92%	<24
Casey, 2014 ¹⁴	2	F = 1	55–60	None	13	Dyspnea (2) Chest tightness (1) Hemoptysis (1) SO ₂ < 92% (1)	<24
Carter, 2011 ¹⁵	3	F = 3	43–58	Asthma (1), seasonal allergies (2)	15–22	Dyspnea (3) Hemoptysis (3) Chest tightness (3) SO ₂ < 92% (2)	<24 (2) N/A (1)
Noti, 2009 ⁸	1	F	23	None	N/A	Dyspnea Hemoptysis SO ₂ < 92%	<24
Wenger, 2007 ¹⁶	1	M	43	None	20	Dyspnea Hemoptysis Chest pain	<24
Beinart, 2007 ¹⁷	1	F	54	Hypertension	22	Dyspnea SO ₂ < 92%	>48
Deady, 2006 ¹⁸	1	F	38	None	15	Dyspnea Hemoptysis SO ₂ < 92%	<24
Adir, 2004 ⁹	70	M = 70	18–19	None	16–22	Dyspnea (70) Cough (67) Hemoptysis (63) Mean SO ₂ 88% ± 6.6	<24
Biswas, 2004 ¹⁹	1	M	36	Diabetes mellitus	22	Dyspnea Hemoptysis	<24
Lund, 2003 ²⁰	3	M = 3	21–27	None	19	Dyspnea (3) Cough (3) Hemoptysis (1) SO ₂ < 92% (1)	<48
Shupak, 2000 ²¹	21	M = 21	18–19	None	16–18	Dyspnea Cough Hemoptysis SO ₂ 91% ± 6.5	N/A
Weiler-Ravell, 1995 ²²	8	M = 8	18–19	N/A	23	Cough (7) Hemoptysis (8) SO ₂ < 92% (5)	<24

N/A indicates not available; SO₂, oxygen saturation.

oximetry or an alveolar-arterial gradient >30 mm Hg; and (4) radiographic opacities consistent with an alveolar filling process and/or interstitial pulmonary edema that resolve within 48 h.⁷ Other potential cardiac causes of pulmonary edema need to be excluded.⁸ Our patient presented with a history and radiographic findings highly suggestive of SIPE; her clinical and radiological improvement within 24 h confirmed the diagnosis. On the day our patient swam, the water temperature was 21°C and the air temperature was 24°C. She did not have a history of hypertension.

Adir et al studied 70 naval trainees who developed SIPE during swimming time trials over 2.4 to 3.6 km in open seas.⁹ These trials took between 30 and 45 min with mean water temperatures during training of 19.6°C. All trainees developed dyspnea, 67 developed cough, and 39 had hemoptysis. On physical examination, 64 trainees had crackles, and 6 had wheezes. The mean oxygen saturation after exercise was 88.4%. Chest radiographs taken 12 to 18 h after presentation were within normal limits. Sixteen trainees had recurrent episodes of SIPE. *Table 1* summarizes other case reports and studies.^{8–22}

Figure 1. Chest radiographs (a) on admission, showing bilateral alveolar infiltrates suggestive of pulmonary edema and (b) after treatment, showing reduction in alveolar infiltrates.

The pathophysiological events during SIPE remain incompletely understood. There is a moderate association between water temperature and the incidence of SIPE,²³ and the temperatures associated with SIPE have ranged from 13°C to 23°C.⁸ Subjects with a history of SIPE have significantly elevated mean pulmonary artery pressures (34.0 vs 22.5 mm Hg) and pulmonary artery wedge pressures (18.8 vs 11.0 mm Hg) compared to controls during exercise in 20°C water.²⁴ Immersion in cold temperatures causes redistribution of blood from the extremities to the thorax due to vasoconstriction, with increased blood volume in central vessels and increased pulmonary artery pressures.²⁰ In addition, the increase in the pulmonary artery wedge pressure suggests decreased left ventricular diastolic compliance, which increases pressures in pulmonary capillaries. The increased pulmonary blood flow associated with exercise and increased hydrostatic pressure in capillaries could increase vascular permeability. Ludwig et al studied five combat swimmers who developed acute respiratory symptoms following swimming 300 to 1600 m in open water with water temperatures ranging from 14°C to 18°C.²⁵ All subjects had airspace consolidation on chest radiographs. These subjects underwent bronchoscopy and had bronchoalveolar lavage fluid containing increased numbers of erythrocytes and increased albumin and immunoglobulin G levels, which cleared on subsequent bronchoscopies after symptom resolution. These results suggest that stress failure occurs in pulmonary capillaries secondary to increased pressures in patients with SIPE. In summary, SIPE should be considered in patients with acute respiratory symptoms during and after swimming events.

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