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INTERMEDIATE

CASE REPORT: CLINICAL CASE

Swimming-Induced Pulmonary Edema

An Underrecognized Cause of Triathlon-Associated Medical Emergencies

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ABSTRACT

Swimming-induced pulmonary edema is a leading cause of triathlon-associated emergencies and death. Cold water immersion, female sex, age>50, and wetsuit compression are associated risk factors. Pathophysiology is due to increased central blood pooling, leading to increased pulmonary capillary wedge pressure. Treatment is focused on prevention; however, recurrence is common. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:1094–1097)

HISTORY OF PRESENTATION

A 50-year-old triathlete with 11 years of racing experience developed subacute onset of dyspnea within 10 minutes of starting the swim portion of a triathlon. Dyspnea continued to progress even after she slowed down and stopped to rest several times. As she swam, she began to cough and heard crackles and wheezes with each breath.

She was unable to continue and was brought back to shore by water safety personnel. Emergency medical services were notified. On examination in the ambulance, vitals were blood pressure 130/88 mm Hg, pulse 118 beats/min, respiratory rate 30 breaths/min, oxygen saturation 92%. She was in moderate respiratory distress with tachypnea, sitting bolt upright, cough, and bilateral diffuse rales.

LEARNING OBJECTIVES

- To recognize that swimming-induced pulmonary edema is an underreported cause of medical emergencies in triathlon and open water swimming events.
- To summarize the appropriate workup for an athlete who has had respiratory symptoms in the water to reduce risk of recurrence and provide recommendations for prevention.

PAST MEDICAL HISTORY

Past medical history was significant for rheumatoid arthritis, with no history of cardiovascular or pulmonary disease and no traditional coronary risk factors.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis included swimming-induced pulmonary edema (SIPE), acute coronary syndrome, stress/Takotsubo cardiomyopathy, asthma, and panic attack.

INVESTIGATIONS

She was treated with oxygen and albuterol with mild improvement and transported to a local emergency

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department. On arrival, vital signs, chest X-ray, electrocardiogram, and bloodwork were normal, including normal troponin. She was diagnosed with "bronchospasm" and discharged. Rales and wheezing gradually resolved after 12 hours.

She saw her internist and a general cardiologist to rule out underlying cardiopulmonary pathology. Chest computed tomography done 48 hours later revealed normal lung parenchyma with residual pulmonary edema (Figure 1). Echocardiogram was normal, including diastolic parameters. Exercise treadmill testing showed no evidence for ischemia. Pulmonary function tests were normal without evidence for asthma.

She was referred to a sports cardiologist, who diagnosed her with SIPE. Cardiopulmonary exercise testing revealed an oxygen consumption of 49.4 mL/kg/min (144% predicted); however, ventilatory efficiency, as measured by minute ventilation/carbon dioxide production slope was elevated at 45 (normal <34). One month later, repeat cardiopulmonary exercise testing 60 minutes after premedication with 50 mg sildenafil, a pulmonary vasodilator, showed unchanged oxygen consumption of 49.0 mL/kg/min, but ventilatory efficiency that was significantly improved at 35 (Table 1).

MANAGEMENT

She was advised that there was a risk of recurrence; however, the risks could be mitigated by avoiding cold water, avoiding tight wetsuits, thorough in-water warm up, and premedication with sildenafil. She successfully completed 6 races in the following 2 years with no recurrence.

DISCUSSION

SIPE was first described in the 1980s in scuba divers and swimmers.[1] It is a rare but serious condition that typically occurs in otherwise healthy athletes competing in triathlon or open water swimming events.[2] SIPE is associated with cold water events and presents with rapid onset of shortness of breath, cough, and rales.[3] It can be fatal if the athlete continues to compete, but if caught early, symptoms usually resolve within 48 hours. It is significantly underdiagnosed and is a leading cause of triathlon-related swimming deaths.[2]

INCIDENCE AND RISK FACTORS. SIPE is more common than previously thought. A comprehensive review estimates the prevalence between 1.1% and 1.8% among triathletes and military combat swimmers.[4] Another study that assessed risk prospectively in a large open water swimming event found a risk of 0.44%.[5]

Risk factors include age >50, female sex, and presence of underlying cardiac disease, including left ventricular (LV) hypertrophy or hypertension.[4] Situational risk factors include cold water immersion, high level of effort, tight wetsuits with full-length leg coverage, overhydration or fluid retention, and inadequate warm up.[3] Unfortunately, recurrence is common and unpredictable; thus, the biggest risk factor for SIPE is having a prior episode, with recurrence rate of 13% to 22%.[2]

Many athletes with SIPE have no underlying cardiopulmonary pathology; however, 1 study of older athletes (mean age 48 years) had up to 72% incidence of at least 1 risk factor such as hypertension, subclinical coronary artery disease, or LV hypertrophy.[6] A case

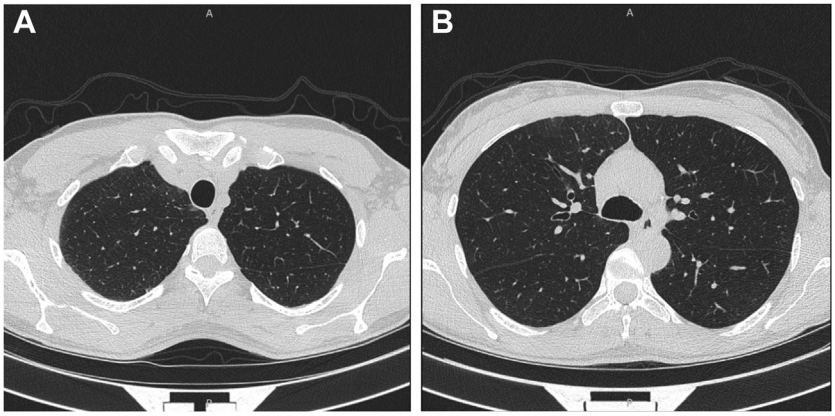
ABBREVIATIONS AND ACRONYMS

LV = left ventricle

RV = right ventricle

SIPE = swimming-induced pulmonary edema

FIGURE 1 High-Resolution Chest Computed Tomography



(A) Apical view demonstrating bilateral subpleural ground glass opacities; and **(B)** mid-upper lobe view demonstrating bilateral interlobular septal thickening.

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TABLE 1 Cardiopulmonary Exercise Testing Results

	Predicted	No Premedication	Sildenafil 50 mg
Exercise capacity			
Exercise time (min:s)		16:23	16:15
Peak respiratory exchange ratio	>1.0	1.09	1.07
Peak oxygen consumption (mL/kg/min)	34.3	49.4	49.0
Cardiovascular performance			
Peak heart rate (beats/min)	173	173	174
BP at rest (mm Hg)	<120/70	118/84	108/76
BP at peak (mm Hg)		162/74	148/70
Oxygen consumption at ventilatory threshold	13.72	43.7	35.3
Peak O_2 pulse (mL/beat)	11.2	16.2	15.9
Pulmonary vascular performance			
Spirometry: forced vital capacity (L)	3.46	3.96	4.07
Spirometry: FEV1 (L/min)	2.76	3.28	3.29
Spirometry: FEV1/forced vital capacity (%)	>70	83	81
Ventilatory efficiency: VE/VCO_2 slope	<34	**45**	**35**

Abnormal values are shown in **bold.**

BP = blood pressure; FEV1 = forced expiratory volume in 1 second; VE/VCO_2 = minute ventilation/carbon dioxide production.

series in divers found medical comorbidities in 68%, with 42% being cardiac, including valvular disease, Takotsubo cardiomyopathy and dysrhythmias.[7] It is unknown why there is a female predominance.

PATHOPHYSIOLOGY. The exact pathophysiology is not fully understood; however, a combination of factors that lead to a "perfect storm" of increased pulmonary capillary pressure ultimately overwhelms the alveolar air pressure, resulting in pulmonary edema.[3] Water immersion with supine positioning increases central blood pooling, augmented by peripheral vasoconstriction due to cold temperatures and leg compression from a wetsuit. This increases cardiac preload, left atrial pressure, and pulmonary capillary wedge pressure. Combined with increased cardiac output, these factors result in higher pulmonary artery pressure.[3] They are further exacerbated by any condition that increases preload, such as hypertension, or any condition that increases pulmonary vascular pressure.

TABLE 2 Swim Safety Guideline: Proposed Visual Safety Guideline for Athletes

Green Zone	Yellow Zone	Red Zone
The Green Zone is **GOOD**! If you are in this zone, KEEP GOING!	The Yellow Zone is **CAUTION**. SLOW DOWN or swim to a kayak and rest before continuing.	The Red Zone is **DANGER**. STOP racing. Float on your back and raise your arm to signal for help.
• Normal breathing • No cough • No chest pain	• Mild shortness of breath • Increased coughing • Minor chest tightness	• Shortness of breath worsening • Coughing up clear or bloody fluid • Chest pain that does not go away • Unable to think clearly • Dizziness or nearly passing out

Another proposed mechanism is that there may be a mismatch between the right ventricle (RV) and LV at the onset of exercise. Both LV and RV afterload increase with exertion, but RV afterload increases less due to pulmonary vasodilation, resulting in RV>LV stroke volume. If cardiac output is already near maximal, the LV cannot compensate by increasing work, leading to pulmonary congestion.[8]

RECOMMENDATIONS FOR PREVENTION. Shared decision-making with athletes is critical for safe return-to-sport. Emphasis should be on prevention of subsequent episodes and early recognition of symptoms. SIPE should be strongly considered in any athlete with cough, dyspnea, rales, or hemoptysis during or after open water swimming events.

SIPE is not correlated with level of experience or training. Thus, we recommend that athletes focus more on race day actions. Athletes should warm up by swimming for >15 minutes to allow the pulmonary vasculature to adapt before the race begins.[4] Start the race at an easy pace and speed up gradually. Avoid races with very cold water, wetsuits, and excessive pre-race hydration or salt loading.[4]

Pharmacologic management may include premedication with sildenafil 50 mg, 1 hour before racing.[9] Nifedipine has also been used in select cases.[10] For athletes with fluid retention, premedication with furosemide or other diuretic for 1 to 2 days before a race may be necessary. However, use of furosemide is not permitted in some sports at high levels of competition.

We propose popularizing a visual guideline for athletes to increase awareness and safety before race day (Table 2).

FOLLOW-UP

Shortly before a subsequent race, the triathlete developed a flare of rheumatoid arthritis and was prescribed prednisone and meloxicam, which caused fluid retention. Unfortunately, this led to a recurrence of SIPE, with severe 3 to 4 pillow orthopnea the night after the race. She was treated with furosemide 40 mg for 2 days with resolution of symptoms over 48 hours. Repeat bloodwork, echocardiogram, chest computed tomography, and pulmonary function tests were normal.

With the addition of furosemide premedication 1 to 2 days before races, the athlete continued competing in triathlons, including finishing an Ironman race. She has been without recurrence over 8 months of additional follow-up. This case illustrates the possible need for diuretics for prevention of SIPE in selected cases, a point that has, to our knowledge, not been made previously.

CONCLUSIONS

SIPE is a dangerous condition that is often underrecognized or misdiagnosed. It is critically important to increase awareness of SIPE among athletes, race directors, emergency medical personnel, and physicians. Better knowledge and more rapid recognition of this potentially fatal condition will save lives. Expert evaluation by a sports cardiologist, careful diagnostic testing, shared decision-making, and modification of race day activities can help mitigate the risk of recurrence.

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