



Brief Report

Swimming-induced pulmonary edema in triathletes^{☆,☆☆}

Charles C. Miller III PhD^{a,b,*}, Katherine Calder-Becker^c, Francois Modave PhD^d

^aDepartment of Biomedical Sciences, Texas Tech University Health Sciences Center at El Paso Paul L. Foster School of Medicine, El Paso, TX, USA

^bDepartment of Cardiothoracic and Vascular Surgery, University of Texas Medical School at Houston, Houston, TX, USA

^cCap Cities Triathlon, Montreal, Quebec, Canada

^dDepartment of Computer Science, University of Texas at El Paso, Central Washington University, Ellensburg, WA, USA

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Abstract

Background: Pulmonary edema related to water immersion has been reported in military trainees and scuba and breath-hold divers, but rarely in the community. To date, no risk factors for this phenomenon have been identified by epidemiological methods. Recently, sporadic reports of swimming-induced pulmonary edema (SIPE) have emerged in the triathlon community. We surveyed the population of a national North American triathlon organization (USA Triathlon) to determine prevalence of and risk factors for symptoms compatible with SIPE.

Methods: We surveyed the population of USA Triathlon through the organization's monthly newsletter distribution channel. We evaluated prevalence of symptoms compatible with pulmonary edema, and then followed up with a case-control study that included additional cases we had identified previously, to identify risk factors for this condition among triathletes.

Results: Symptom history compatible with SIPE was identified in 1.4% of the population. Associated factors identified in multivariable analysis included history of hypertension, course length of half-Ironman distance or greater, female gender and use of fish oil supplements. Of the 31 cases reported, only 4 occurred in the absence of any associated factors.

Conclusions: The identification of hypertension and fish oil in particular as risk factors raise questions about the role of cardiac diastolic function in the setting of water-immersion cardiac preload, as well as the hematologic effects of fish oil. Mechanistic studies of these risk factors in a directly observed prospective cohort are indicated.

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1. Background

Pulmonary edema of immersion has been reported in select populations such as submersion divers [1–4] and combat swimmers [5,6], but only sporadically in the community setting [7]. Aside from the studies of combat swimmers, who are typically subjected to intense swimming trials, often after massive hydration, no reports of more than a dozen community-based cases have appeared in the literature. Termed swimming induced pulmonary edema

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* Corresponding author. Department of Biomedical Sciences, Texas Tech University Health Sciences Center at El Paso Paul L. Foster School of Medicine, El Paso, TX 79905, USA. Tel.: +1 915 783 5227; fax: +1 915 783 5223.

E-mail addresses: charles.miller@ttuhsc.edu (C.C. Miller), triathlonteam@gmail.com (K. Calder-Becker), francois.modave@gmail.com (F. Modave).

(SIPE), the phenomenon has been difficult to characterize with respect to risk factors [3,6,8], although it has been reported to be at least temporally associated with cold-water swims, wearing a wetsuit and even massive hydration of up to five liters in two hours prior to immersion [5].

Recently, reports of symptoms consistent with SIPE have begun to emerge anecdotally among groups of triathletes and on internet forums in the triathlon community. These reports have been linked, at least through speculation in the popular press [9], to the unusually large number of swim-related deaths that occurred in triathlons in the 2008 North American season [10]. Since the reports of SIPE-compatible symptoms in the triathlon swim have been both infrequent and widely geographically distributed, it has not been practical to perform direct examination of all the people who have reported these complaints. Such examinations, even if feasible, would likely be of minimal value at times remote from the acute events, since the events appear to be largely self-limiting [6,8]. Therefore, we developed a survey of symptom history and potentially associated risk factors in order to better characterize the phenomenon, and to assist in generation of hypotheses for future research. In this article, we report the findings of a prevalence survey and a follow up case-control study administered via the internet to North American triathletes through a large national triathlon organization.

2. Methods

No validated, community-based pulmonary edema questionnaire has appeared in the literature, so it was necessary for us to develop a survey for this purpose. We reviewed the symptom criteria described previously by Weiler-Ravell et al [5], but since we did not have the benefit of direct examination of our participants as Weiler-Ravell's group had, we limited our case definition to "cough productive of pink frothy or blood-tinged secretions" for the analysis. This description is both pathognomonic for pulmonary edema and sufficiently alarming that it would be difficult for a survey respondent to miss. We also captured demographic information, detailed personal and family medical history, training, hydration and wetsuit wearing habits, and swim conditions on the day of first episode. We titled the survey "swim-related breathing problems survey" so as not to lead respondents to think of pulmonary edema when completing it. We performed pilot testing with three small groups of triathletes to identify ambiguities in questions and to evaluate completeness of responses. We also were careful to place the "pink frothy or blood-tinged secretions" response variable in the context of questions of breathing problems in the swim, to differentiate it from bleeding due to injury (a crowded triathlon swim can involve a lot of contact between athletes).

With the cooperation of the USA Triathlon organization (USAT), we distributed a link to the online survey to their entire membership of 104,887 triathletes, through their

monthly e-mail newsletter. We distributed the link each month for three consecutive months between August and October 2008, and programmed our online survey tool (surveymonkey.com) to accept only one response per IP address to avoid duplicate responses. The survey was conducted anonymously, collected no personally identifiable information, and was approved by our Institutional Review Board for Human Research. We conducted the study in two phases. 1) we sent out a link to the survey through the USAT electronic newsletter as described previously, and 2) we sent it to a group of cases who had identified themselves through an online triathlon discussion forum (slowtwitch.com). The goal of the two-phase approach was to develop a population-based estimate of the prevalence of SIPE-compatible symptoms, and then to follow that up with a case-control study of all cases known to us, which included cases identified in the discussion forum in addition to the new cases identified through the population survey. The IP address filter used on the prevalence survey (USAT respondents) was retained for the internet forum survey (slowtwitch respondents) to avoid duplicate responses, and we specifically asked the respondents not to complete the survey more than once.

USAT has a large "juniors" population of young triathletes under age 18. Many long-course triathlons do not allow juniors to participate, and this would put them outside the complete risk set for triathlon-related SIPE events. We therefore restricted the sample population to athletes in the 20-and above age groups and to those who reported their age group membership. Prevalence was estimated as the ratio of cases reported in the population survey to the total number of respondents, with an exact binomial 95% confidence interval computed for the point estimate. The case-control study involved responses of all known cases, with all non-cases serving as controls. Univariate data were analyzed by contingency table methods with Fisher's exact p values, and by univariate logistic regression for continuous independent variables. Multivariable analyses were conducted using stratified contingency table methods and multiple logistic regression modeling, with interactions evaluated up to first-order. Analyses were conducted using SAS 9.1.3, Service Pack 4 (SAS Institute, Inc., Cary, NC).

3. Results

The age distribution of the overall USA Triathlon population, excluding juniors, is shown in Fig. 1A. USAT statistics on newsletter reading rates indicate that approximately one percent (about 1,400) of the emailed newsletters are opened at each mailing. USAT surveys were returned by 1423 respondents over three cycles of distribution, which indicates that we received responses from 1.3% of the total population, but closer to 1/3 of the people who actually opened the newsletter. Of the 1423 respondents, 17 were

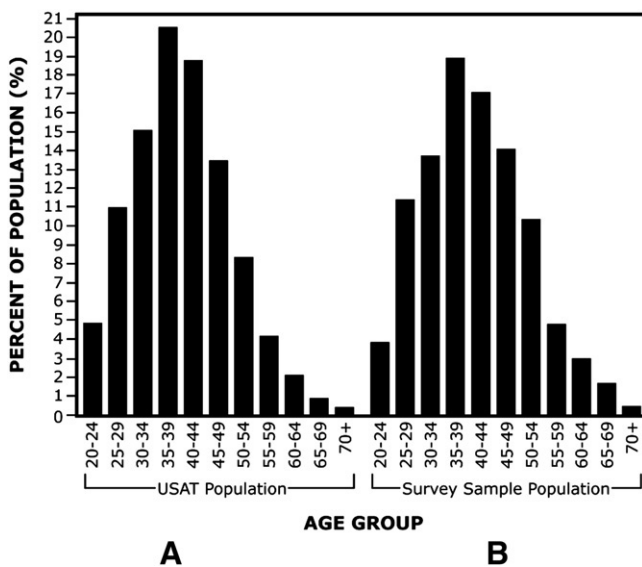


Fig. 1 Age distribution of USAT population (left panel) and survey sample population (right panel). Chi square for difference $P = .21$.

juniors and 6 had incomplete responses. After exclusion of the juniors and the incomplete responses, the population prevalence sample consisted of 1400 surveys, and none of the surveys that were excluded reported SIPE-related symptoms. Fig. 1B shows the age distribution among the survey respondents. Age group relative frequencies between the population and the sample were not different by statistical comparison.

In the population study, 20/1400 patients reported having experienced cough productive of pink, frothy or bloody secretions during a swim, for an estimated population prevalence of 1.4% (95% confidence interval 0.9-2.2%). As an expansion of the population survey, the case-control study included an additional 11 cases that had previously come to our attention through slowtwitch.com, which brought the total number of cases available for analysis to 31, and the denominator for the case-control study to 1411, which is the population described in the remainder of this article.

Statistically significant univariate risk factors were history of hypertension, consumption of fish oil supplements, wetsuit use, and long-course (half-Ironman or greater) event distance, as shown in Table 1. Although somewhat more prevalent in athletes over 40, SIPE symptoms were not significantly associated with participant age. Self-reported swimming skill also was unrelated to SIPE symptoms. Failure of athletes to warm-up before swimming and excessive pre-swim hydration have been described previously as potential risk factors, and though the point estimates went in the anticipated directions in our data, we were unable to demonstrate statistically significant associations with these independent variables. We did ask whether sports drink or water was used to hydrate, but we were unable to detect and associations with fluid consumed.

In adjusted analysis, hypertension, long course distance and fish oil consumption remained in the model. Female gender was entered, and wetsuit use dropped out of the model (Table 2). No interactions were observed in stratified or regression analyses. Of the 31 cases reported, only four occurred in the absence of any of these independent variables.

4. Discussion

This is the first study to report a population-based estimate of SIPE prevalence in community triathletes, and is the first to report statistical risk factor associations in any population. Symptoms compatible with swimming-induced pulmonary edema are estimated to occur in 1.4 percent of triathlon participants in North America, as represented by the membership of the USA Triathlon organization. This is consistent with and well within statistical error of the prevalence of 1.8% reported by Adir et al [8] in their directly observed combat swimmers, as well as the 1.1% prevalence reported by Pons et al in scuba divers and swimmers [3]. Our findings identify strong associations of SIPE symptoms with self-reported history of hypertension, fish oil use and long-course event distance. Adjusted analysis suggests that women may also be at higher risk. Wetsuit use does not appear to be independently associated with SIPE-related symptoms once adjustments are made for gender, fish oil consumption, hypertension and course length.

Immersion in water increases cardiac output for a given oxygen consumption (VO_2), mainly through increased preload as indicated by increased left ventricular end-diastolic volume [11]. Pulmonary artery pressure is roughly doubled by immersion both at rest and during exercise, and the fractional increase in right atrial pressure is even greater than that in the pulmonary artery [12]. In hypertensive athletes, diastolic dysfunction due to hypertension could plausibly interact with the increased PA pressure and left ventricular end-diastolic volume of immersion to produce capillary breach leading to pulmonary edema. Diastolic dysfunction is known to be associated with hypertension [13], and is also a well-known cause of pulmonary edema, the contribution of which has been separated clearly from systolic heart failure in experimental studies [14]. The emergence of hypertension as the strongest of the independent risk factors is consistent with the hypothesis that diastolic dysfunction is involved in the pathogenesis of swimming induced pulmonary edema in triathletes. Some additional insult to the capillaries is clearly required for SIPE to manifest, since pulmonary artery pressure elevation alone is not sufficient to cause pulmonary edema in similar circumstances [15]. Indeed, the vast majority of swimmers never have a problem.

Increased preload in swimmers from immersion may be enhanced by wearing of tight-fitting wetsuits, or by peripheral vasoconstriction due to cold, all leading to

Table 1 Case-control study SIPE risk factors

Variable	No. patients (%)	No. Cases (%)	Odds Ratio	95% CI **	P ***
Overall	1411 (100.0)	31 (2.2)			
Age					
20 – 29	216 (15.3)	3 (1.4)	1		
30 – 39	461 (32.7)	7 (1.5)	1.09	0.28 – 4.27	1.0
40 – 49	440 (31.2)	11 (2.5)	1.82	0.50 – 6.59	.41
50 – 59	222 (15.7)	10 (4.5)	3.35	0.91 – 12.34	.09
60 – 69	64 (4.6)	0 (0.0)	x		
70 – 79	8 (0.6)	0 (0.0)	x		
Female	662 (46.9)	20 (3.0)	2.08	1.0 – 4.38	.07
Male	749 (53.1)	11 (1.5)	1		
Hypertension	85 (6.0)	7 (8.2)	4.87	2.03 – 11.65	.002
Normal BP	1326 (94.0)	24 (1.8)	1		
Diabetes	7 (0.5)	1 (14.3)	7.63	0.89 – 65.38	.14
No Diabetes	1404 (99.5)	30 (2.1)	1		
Multivitamin	750 (51.0)	18 (2.5)	1.34	0.65 – 2.75	.47
No Multivitamin	691 (49.0)	13 (1.9)	1		
Vitamin C	224 (15.9)	9 (4.0)	2.22	1.00 – 4.88	.07
No Vitamin C	1187 (84.1)	22 (1.9)	1		
Vitamin E	127 (9.0)	4 (3.2)	1.51	0.52 – 4.40	.52
No Vitamin E	1284 (91.0)	27 (2.1)	1		
Fish Oil	304 (21.6)	14 (4.6)	3.10	1.51 – 6.35	.003
No Fish Oil	1107 (78.5)	17 (1.5)	1		
Flax Oil	128 (9.1)	5 (3.9)	1.97	0.74 – 5.21	.19
No Flax Oil	1283 (90.9)	26 (2.0)	1		
Swimming Skill					
Strong	364 (25.8)	8 (2.2)	1.0	0.44 – 2.25	1.0
Not strong	1047 (74.2)	23 (2.2)	1		
Pre-Swim					
Warm Up	402 (28.5)	5 (1.2)	0.48	0.18 – 1.25	.16
No Warm Up	1009 (71.5)	26 (2.6)	1		
Pre-Swim Hydration					
>1L	227 (16.1)	9 (3.9)	2.18	0.99 – 4.80	.08
<1L	1184 (83.9)	22 (1.9)	1		
Open Water	843 (59.7)	22 (2.6)	1.66	0.76 – 3.64	.27
Pool	568 (40.3)	9 (1.6)	1		
Wetsuit	482 (34.2)	18 (3.7)	2.73	1.33 – 5.63	.007
No Wetsuit	929 (65.8)	13 (1.4)	1		
Long Course	183 (13.0)	10 (5.5)	3.32	1.54 – 7.17	.004
Short Course	1228 (87.0)	21 (1.7)	1		
Climate Trained In					
Hot	659 (46.7)	12 (1.8)	0.72	0.35 – 1.49	.47
Not Hot	752 (53.3)	19 (2.5)	1		

Hypertension is self-reported history of hypertension.

Diabetes is self-reported Type 1 or Type 2 diabetes.

Long course is half-Ironman distance or greater.

Hot climate is self reported perception of climate.

x= zero cell – effect not estimable.

** 95% CI= 95% confidence interval. Confidence intervals are test-based.

*** p= probability of Type I statistical error. Values without parentheses are Fisher's Exact (when calculable) or Pearson Chi-square probabilities.

central shift of the blood volume. This mechanism has been hypothesized to be a contributor to SIPE in the literature [5,16], as has the increased negative intrathoracic pressure generated in competition, which could alter the transcapillary pressure differential in favor of alveolar flooding [4] We were unable to separate a multivariate effect of wetsuit use, though this may be due to the large overlap between

long-course distance and wetsuit use, especially given the limited statistical power arising from the relatively small number of events.

The independence of fish oil consumption as a risk factor may implicate this agent's antiplatelet and vasodilatory activity in the pathogenesis of pulmonary capillary leak under increased pulmonary artery pressure. Antiplatelet

Table 2 Multiple logistic regression risk factors for SIPE

Variable	Parameter Estimate	Adjusted Odds Ratio	95% C.I.	P
Intercept	-5.1506			
Hypertension	1.6821	5.38	2.15-13.48	.0003
Female Gender	1.0114	2.75	1.26-6.02	.02
Long Course	1.1938	3.30	1.50-7.27	.003
Fish Oil	0.9792	2.66	1.28-5.54	.009

Hypertension is self-reported history of hypertension.

Diabetes is self-reported Type 1 or Type 2 diabetes.

Long course is half-Ironman distance or greater.

Hot climate is self reported perception of climate.

medications have been linked to isolated cases of pulmonary edema [17], and aspirin has been specifically implicated in pulmonary edema in breath-hold divers [18]. Ascent to altitude has been reported to decrease platelet counts, and this has been described as a potential mechanism of high altitude pulmonary edema [19].

Several reports in the literature have considered massive pre-swim hydration – of as much as 5 liters consumed 2 hours prior to swimming – as risk factors [5,16]. We saw some increase in the odds of SIPE in triathletes reporting pre-swim hydration of one liter or more, but this was not statistically significant in univariate or multivariable analyses. We explored interactions with other variables, but were not able to identify any differential effects of these variables, or any additional contribution of sports drinks containing electrolytes.

Limitations of our study include self-reported symptoms and risk factors, and lack of an independently validated measurement tool for swimming induced pulmonary edema. Self-reported hypertension, in particular, is troublesome, as undetected disease in the population may underestimate the true prevalence of this risk factor. We did not attempt to measure water temperature, which most triathletes would not be expected to be able to report reliably at a survey time remote from competition, and we also did not differentiate between salt and fresh water swims. Further, we were unable to arrive at a good definition for “hot” competition conditions, and the varied personal perceptual interpretations make this variable difficult to standardize. These are practical limitations inherent to early studies of unusual events that occur over a widely dispersed geographical area. Despite the lack of a validated survey instrument, self-limiting community-acquired pulmonary edema is exceptional, and our single question involving self-report of the pathognomonic signs of pulmonary edema is a reasonable metric under the circumstances. To be conservative in our case definition, we limited positive findings to reports of pink frothy or bloody secretions during or after a swim. This symptom is hard to miss, and is pathognomonic for pulmonary edema. If anything, this stringent case criterion would be expected to result in under-reporting of cases. We were careful to segregate coughed-up blood that was associated with

significant shortness of breath from any other source of bleeding such as a cut lip in our questionnaire. Our pilot test groups clearly understood this, but how this translated to the broader population is less clear. The fact that our estimates were within a half percentage point of other series derived from directly observed populations gives us some reassurance that the question was answered correctly in general.

The major new findings of this study are that pulmonary edema is an unusual but not rare event, which occurs in approximately 1.4% of community triathletes. It is associated with hypertension, fish oil use, long course triathlons and female gender. Replication of these findings in a prospectively evaluated, directly observed cohort would be helpful in determining the reproducibility of the risk factor associations. Mechanistic studies to evaluate the roles of these risk factors would also be of significant interest for future research.

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The three authors collaboratively designed the survey. Ms. Calder-Becker maintained the case directory. Dr. Miller and Dr. Modave analyzed the data. Dr. Miller wrote the manuscript. All three authors critically revised the manuscript.

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