EDITORIAL COMMENTARY

Immersion pulmonary edema: drowning from the inside

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Immersion pulmonary (IPE, also known as swimming-induced pulmonary edema, SIPE) is a condition in which pulmonary edema develops rapidly during a dive or vigorous swim. Symptoms include dyspnea and hemoptysis. Physical exam reveals typical signs of bilateral pulmonary edema, which can be confirmed radiographically or with bedside ultrasound [1-3].

IPE tends to recur in susceptible individuals. Often there are cardiac or pulmonary comorbidities, but IPE can occur in highly fit individuals such as triathletes and military trainees: IPE has been reported among special forces units from both the United States and Israel. Why pulmonary edema, a condition typically associated with heart failure, should occur in normal individuals remains somewhat of a mystery.

Evidence supports the notion that IPE is a form of hemodynamic pulmonary edema. Immersion in water results in redistribution of blood from the legs and splanchnic vessels into the thorax, causing engorgement and increased pressure in pulmonary vessels, which is augmented by exercise. IPE was initially reported by Dr. Peter Wilmshurst, who observed that many victims subsequently developed hypertension [4]. Testing in his laboratory revealed an exaggerated increase in forearm vascular resistance in response to stimuli such as cold or oxygen breathing. He hypothesized that IPE is caused by the combination of exaggeratedly high afterload due to cold and increased preload due to immersion, which induces transient heart failure. Indeed, hypertension continues to be the most commonly described predisposing factor [1,3,5,6]. Besides hypertension, authors have described important comorbidities that include myocardial ischemia, cardiomyopathy including stress cardiomyopathy (SCM, including Takotsubo cardiomyopathy), cardiac valve disease, arrhythmias, small lungs, lung disease and excess fluid consumption. The cause of IPE in highly fit triathletes and military special forces trainees who have been screened medically has not been completely elucidated. However, hemodynamic studies during submerged exercise in cold water indicate that, compared to non-susceptible individuals, IPE susceptibility is associated with higher pulmonary artery and wedge pressures [7], thus predisposing to classic hemodynamic pulmonary edema. One possible risk factor in such cases may be reduced ventricular diastolic compliance, resulting in higher left ventricular filling pressures. Clinical evaluation of some IPE-susceptible individuals by the author has revealed an exaggerated hypertensive response to cold-water immersion. In such cases the resulting increase in circulatory afterload during cold-water exposure could provide a mechanism for transient heart failure as proposed by Wilmshurst [4].

Treatment of IPE is supportive: Once an IPE victim is removed from the water and warmed up, the hemodynamic conditions that predispose to IPE quickly reverse. Thus traditional treatments for hemodynamic pulmonary edema such as diuretics and vasodilators are not usually necessary. Supplemental oxygen is often required; inhaled β2-adrenergic agonists may also be helpful to accelerate expulsion of water from pulmonary air spaces [8].

In Wilmshurst's initial publication he provided evidence that cold water was an essential component of the pathophysiology. Indeed, a significant proportion of the now hundreds of reported cases occurred in cold water. To this list there are two new case series. Dr. Edmonds and colleagues have added 29 individuals from Oceania (warmer water) who experienced 41 IPE cases, of which 31 are described in detail [9]. His case series is accompanied by a thoughtful analysis [10]. These cases occurred from 2002-2018; median age was 55 years (range 21-72 years); females made up 55% of the cases; there were six deaths. Unlike other reported series in which surface swimmers constituted a large fraction [1], most of Edmonds' cases were in scuba divers. Cold water was implicated in only a small number of cases. Different from previous reports [1], the majority of Edmonds' cases were female.

Dr. Henckes and colleagues have reported a relatively large controlled study of divers experiencing IPE and controls. They collected data from a questionnaire received from 88 IPE victims and 392 controls. Mean age of the IPE victims was 50.9 years (vs. 45.3 years in controls); 47% were female (vs. 29.3% of controls).

What have we learned from these new case series? Edmonds' paper confirms observations of others that IPE can be fatal [11-14]. His case series also includes a large number of individuals with other underlying cardiac pathology, which emphasizes the benefit of investigation of IPE cases at the time of the event, especially with echocardiography. One particularly interesting observation in Edmonds' series is the high prevalence of SCM (26%), confirming the observations of Gempp [15], who in a series of IPE cases described reversible wall motion abnormalities, presumably a form of SCM. Edmonds and colleagues point out that the percentage of SCM cases in their recent series is probably an underestimate due to lack of detailed medical investigation of IPE cases in earlier years, while recently echocardiography has become fairly routine. An open question is whether SCM pre-existed the swim or dive, or did it occur as a result? It seems unlikely that someone with left ventricular impairment sufficient to induce heart failure (and therefore probably symptomatic) would entertain the notion of jumping in the water, especially for a scuba dive. The logical conclusion is that SCM is more likely to be triggered by the swim or dive rather than the reverse [16]. Evidence for this explanation is that exogenous epinephrine administration can induce Takotsubo cardiomyopathy [17-19]; thus the explanation for SCM in swimmers or divers is likely to be the effects on the heart of a rise in endogenous catecholamine levels during cold-water immersion [20-22].

In Dr. Henckes' series multivariable analysis was used to identify factors that may represent higher risk of IPE in divers. Her series nicely confirms risk factors previously published, including older age, female gender, hypertension and physical exertion [1,5,23,24]. While NSAID use has previously been suggested as a possible risk factor in a single case report [25], Henckes and colleagues have observed a statistically significant association (odds ratio 24.32). Whether this represents a specific risk of the medication or of the condition for which it was taken is a question requiring further investigation. Although not statistically significant, it is interesting that of the IPE group 4.5% reported a history of cardiomyopathy versus 2.3% of the controls.

A careful search for predisposing conditions is good practice in all IPE victims who have not already been medically screened. Hypertension remains the most common medical predisposing factor, most likely not only due to the increase in afterload, but also because accompanying left ventricular hypertrophy (LVH) may adversely affect diastolic function, causing elevated left ventricular end-diastolic pressure. For patients in whom hypertension is not evident during a clinic visit, ambulatory blood pressure monitoring may be helpful to detect masked hypertension [26], especially if LVH is observed on echocardiography.

Follow-up investigations that should also be considered in selected patients include examining for coronary artery disease and lung pathology. Recurrence is frequent in IPE, and good practice includes counseling IPE victims not to return to diving or competitive swimming until relevant medical conditions are treated.

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REFERENCES

1. Peacher DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc. 2015; 47(6): 1128-1134.

2. Grunig H, Nikolaidis PT, Moon RE, Knechtle B. Diagnosis of swimming induced pulmonary edema-a review. Front Physiol. 2017;8:652.

3. Wilmshurst PT. Immersion pulmonary oedema: a cardiological perspective. Diving Hyperb Med. 2019;49(1):30-40.

4. Wilmshurst PT, Nuri M, Crowther A, Webb-Peploe MM. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. Lancet. 1989;1: 62-65.

5. Gempp E, Demaistre S, Louge P. Hypertension is predictive of recurrent immersion pulmonary edema in scuba divers. Int J Cardiol. 2014;172(2):528-529.

6. Spencer S, Dickinson J, Forbes L. Occurrence, risk factors, prognosis and prevention of swimming-induced pulmonary oedema: a systematic review. Sports Med Open. 2018;4(1):43.

7. Moon RE, Martina SD, Peacher DF, Potter JF, Wester TE, Cherry AD, Natoli MJ, Otteni CE, Kernagis DN, White WD, Freiberger JJ. Swimming-induced pulmonary edema: pathophysiology and risk reduction with sildenafil. Circulation. 2016; 133(10):988-996.

8. Sakuma T, Okaniwa G, Nakada T, Nishimura T, Fujimura S, Matthay MA. Alveolar fluid clearance in the resected human lung. Am J Respir Crit Care Med. 1994;150(2):305-310.

9. Edmonds C, Lippmann J, Fock A. Immersion pulmonary edema-case reports from Oceania. Undersea Hyperb Med. 2019; 46(4):581-600.

10. Edmonds C, Lippmann J, Bove AA. Immersion pulmonary edema. An analysis of 31 cases from Oceana. Undersea Hyperb Med. 2019; 46(4):601-609.

11. Cochard G, Arvieux J, Lacour JM, Madouas G, Mongredien H, Arvieux CC. Pulmonary edema in scuba divers: recurrence and fatal outcome. Undersea Hyperb Med. 2005;32(1):39-44.

12. Henckes A, Lion F, Cochard G, Arvieux J, Arvieux CC. [Pulmonary oedema in scuba-diving: frequency and seriousness about a series of 19 cases]. Ann Fr Anesth Reanim. 2008; 27(9): 694-699.

13. Smart DR, Sage M, Davis FM. Two fatal cases of immersion pulmonary oedema - using dive accident investigation to assist the forensic pathologist. Diving Hyperb Med. 2014;44(2):97-100.

14. Moon RE, Martina SD, Peacher DF, Kraus WE. Deaths in triathletes: immersion pulmonary oedema as a possible cause. BMJ Open Sport Exerc Med. 2016;2(1):e000146.

15. Gempp E, Louge P, Henckes A, Demaistre S, Heno P, Blatteau JE. Reversible myocardial dysfunction and clinical outcome in scuba divers with immersion pulmonary edema. Am J Cardiol. 2013;111(11):1655-1659.

16. Baber A, Nair SU, Duggal S, Bhatti S, Sundlof DW. Stress cardiomyopathy caused by diving: case report and review of the literature. J Emerg Med. 2016;50(2):277-280.

17. Madias JE. Epinephrine administration and Takotsubo syndrome: Lessons from past experiences. Int J Cardiol. 2016; 207:100-102.

18. Nazir S, Lohani S, Tachamo N, Ghimire S, Poudel DR, Donato A. Takotsubo cardiomyopathy associated with epinephrine use: A systematic review and meta-analysis. Int J Cardiol. 2017; 229:67-70.

19. Maes S, Dhooghe N, Schotte H, Cattoir S, Jacobs TF, Van Landuyt K. Takotsubo cardiomyopathy induced by epinephrine infiltration for liposuction: broken heart syndrome. Aesthet Surg J. 2019 Sep 13;39(10):NP431-NP436.

20. Weiss M, Hack F, Stehle R, Pollert R, Weicker H. Effects of temperature and water immersion on plasma catecholamines and circulation. Int J Sports Med. 1988;9 Suppl 2:S113-S117.

21. Smith DJ, Deuster PA, Ryan CJ, Doubt TJ. Prolonged whole body immersion in cold water: hormonal and metabolic changes. Undersea Biomed Res. 1990;17(2):139-147.

22. Sramek P, Simeckova M, Jansky L, Savlikova J, Vybiral S. Human physiological responses to immersion into water of different temperatures. Eur J Appl Physiol. 2000;81(5):436-442.

23. Koehle MS, Lepawsky M, McKenzie DC. Pulmonary oedema of immersion. Sports Med. 2005;35(3):183-190.

24. Miller CC, 3rd, Calder-Becker K, Modave F. Swimming-induced pulmonary edema in triathletes. Am J Emerg Med. 2010; 28(8):941-946.

25. Van Renterghem D, Depuydt C. Hemoptysis and pulmonary edema in a scuba diver using diclofenac. Pharmacology. 2012; 89(1-2):103-104.

26. Gorostidi M, Vinyoles E, Banegas JR, de la Sierra A. Prevalence of white-coat and masked hypertension in national and international registries. Hypertens Res. 2015;38(1):1-7.