Pulmonary edema of scuba divers

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Hampson NB, Dunford RG. Pulmonary edema of scuba divers. Undersea Hyperbaric Med 1997; 24(1):29–33.—A syndrome of acute pulmonary edema has been previously reported among scuba divers in cold, European waters. Because of the temperatures involved, the name "cold-induced pulmonary edema" was coined in the original 1989 description. We report six individuals who developed the identical syndrome, five while diving in Puget Sound and one in the Gulf of Mexico. The four women and two men ranged in age from 24 to 60 yr. They experienced one to six episodes apiece, each with the development severe dyspnea at depth without excessive exertion. Associated symptoms included cough, weakness, expectoration of froth, chest discomfort, orthopnea, wheezing, hemoptysis, and dizziness. Emergency medical evaluation of four divers revealed rales on examination and pulmonary edema on chest radiograph. In one diver with pulmonary edema on chest radiograph, pulmonary capillary wedge pressure was normal when measured acutely. Symptoms resolved either spontaneously over 1–2 days or with standard medical treatment for pulmonary edema. Prior history of cardiovascular disease was negative except for hypertension and mitral valve prolapse in one diver. Cardiac evaluations following recovery from the acute episodes were normal. Episodes in the cold waters of Puget Sound sometimes occurred despite the use of dry suits. Furthermore, one diver developed recurrent episodes in 27°C water off Cozumel, Mexico. Development of pulmonary edema while scuba diving constitutes a distinct clinical entity which may occur in either "cold" or "warm" water. It is not associated with a decompression mechanism. Personnel caring for divers should be aware of the syndrome in order to provide optimal medical management.

pulmonary edema, diving, underwater

A variety of disorders may cause respiratory symptoms among recreational scuba divers. These include pulmonary barotrauma, water aspiration, breathing of contaminated gas, and pulmonary decompression sickness (the "chokes"), among others (1). A syndrome comprised of the development of pulmonary edema while diving has been previously described in a small number of European divers (2,3). Among the limited number of cases reported to date, all have occurred in cold water. We report here our experience with six individuals experiencing this syndrome, including one who developed it while diving in relatively warm water. We believe that the syndrome may be more common than has been reported in the medical literature, due in part to lack of awareness of the condition and a resultant failure of diagnosis by medical personnel.

METHODS

Information on individuals referred to Virginia Mason Medical Center in Seattle, Washington, for evaluation of pulmonary edema developing while scuba diving was collected from 1989 to 1995. Data regarding patient medical history, diving experience, details of incident dives, and results of subsequent medical evaluation for the condition were tabulated. Relevant medical records from evaluations performed outside our institution were requested and reviewed with patient consent.

RESULTS

During the 7-yr study, six individuals were referred to our institution for evaluation of symptoms consistent with the syndrome. Their case characteristics are as follows:

Diver 1: The patient was a healthy, 34-yr-old woman with 4 yr of uneventful recreational scuba diving experience. She dove to 75 ft in Puget Sound (Washington State) wearing a dry suit where she worked for 15 min pouring concrete. She began to cough uncontrollably at the end of that time, prompting her ascent. She stopped several times during ascent to cough, then continued in a controlled fashion to the surface. She swam 200 ft to shore, arriving there tachypneic and coughing up frothy white fluid. She experienced dyspnea with minimal exertion for the remainder of the day, in addition to cough. She slept in a chair that night due to orthopnea. The cough had resolved by the following morning. Because dyspnea persisted, she came for medical evaluation. She was taking no medications. Physical examination was notable for the presence of bibasilar rales on chest auscultation. Resting electrocardiogram was normal with the exception of sinus bradycardia. Chest radiograph demonstrated prominent lung markings at both bases, and arterial blood gas analysis revealed pHa 7.43, Pa_{CO_2} 34 mmHg, and Pa_{O_2} 87 mmHg (calculated alveolar-to-arterial oxygen difference, 20 mmHg). Resting surface echocardiogram demonstrated normal left ventricu-

lar systolic and diastolic function. She denied water aspiration or equipment malfunction during the episode. Dyspnea resolved spontaneously and she had no recurrence of symptoms during 5 subsequent yr of active diving.

Diver 2: The patient was a 60-yr-old woman with a medical history notable for essential hypertension treated with diuretic and calcium channel blocker medications, as well as a cerebrovascular accident 10 yr earlier which manifested as transient aphasia without sequellae. She was certified as a diver 4 mo. earlier but did not dive in the interval. She developed symptoms during 3 days of diving off the island of Cozumel (Mexico). On Day 1 she dove to 40 ft for 35 min without difficulty. On Day 2 she dove to 80 ft and was at depth for 15 min when she noted the onset of chilling, weakness, chest congestion, and dyspnea. She ascended in a controlled fashion with her instructor. On board the boat, she was weak and expectorating frothy fluid. Oxygen was administered and symptoms resolved over 4 h. On Day 3 she dove to 80 ft and developed identical symptoms after 20 min at depth. She was taken to the surface, administered O_2, and transported to a medical center. Physical examination was notable for tachypnea. A chest radiograph revealed bilateral perihilar alveolar infiltrates consistent with pulmonary edema. She was treated with diuretics, digoxin, and antibiotics and showed clinical improvement over several hours. Repeat chest radiograph the following morning showed total resolution of the infiltrates. Medical evaluation upon return to Seattle included echocardiography demonstrating mitral valve prolapse with mild regurgitation and normal left ventricular function. In addition, exercise treadmill testing and coronary arteriography were normal. She experienced no recurrence during seven subsequent dives in Australia to depths of 60 ft in 25°C water wearing a wet suit.

Diver 3: The patient was a 42-yr-old male with a 2-yr history of active scuba diving. At the time of his evaluation he was diving 50 times annually. While diving recreationally in a wet suit at a depth of 77 ft in Puget Sound he experienced the sudden onset of severe dyspnea, associated with dizziness and a sensation of flushing. He ascended rapidly and was still dyspneic upon reaching the surface. Shortness of breath improved within minutes on board the boat, but chest tightness and a nonproductive cough persisted for 1–2 days. He was not moving vertically through the water at the time symptoms developed, did not believe he aspirated, had no prior history of cardiopulmonary disease, and was taking no medication.

He subsequently developed an identical syndrome on five occasions after the initial episode. All occurred at depths greater than 35 ft and typically at 60–70 ft. Despite diving in a wet suit in cold water, he did not feel chilled before the onset of symptoms. His equipment was professionally inspected twice and found to be functioning normally.

Medical evaluation performed weeks after his final episode included a normal chest radiograph, normal spirometry, and radionuclide cardiac scan without evidence for ischemic heart disease.

Diver 4: The patient was a 48-yr-old female who developed symptoms while diving in Puget Sound as part of her basic scuba certification course. On Day 1 she dove to a depth of 22 ft for 30 min in a dry suit without difficulty. On Day 2 she dove to 30 ft, where she developed gradually progressive dyspnea. After 10 min at depth, she was gasping and panting to breathe. She was rapidly assisted to the surface by her instructor, possibly experiencing transient loss of consciousness while ascending. Upon arriving at the surface, she was awake and noted severe dyspnea. She described a "gurgling" sound when she breathed and the feeling that she "had water in my lungs." She was expectorating blood-tinged frothy sputum. Oxygen was administered onboard the boat without symptomatic improvement. Upon reaching shore, she was evaluated by paramedics and intubated for severe respiratory distress. She was transported to our emergency department where physical examination revealed rales and rhonchi over all lung fields. A chest radiograph demonstrated interstitial pulmonary edema. A pulmonary artery catheter was placed with demonstration of a pulmonary capillary wedge pressure of 10 mmHg. Diuretics were administered and extubation subsequently performed. Because of possible loss of consciousness during ascent and limitations on neurologic evaluation resulting from administration of sedatives in the field, she was treated in the hyperbaric chamber on a US Navy treatment table VI for possible arterial gas embolism. She was discharged from the hospital the following morning feeling well and with a normal neurologic evaluation.

She had no significant past medical history and was very athletically active before the episode. Medications taken by the patient included estrogen and progesterone. Diving equipment utilized was rented from a commercial diveshop and found to be functioning normally when tested after the episode. She did not dive again.

Diver 5: The patient was a 52-yr-old female who had completed 20 uneventful scuba diving exposures since basic certification 3 mo. earlier. At the time of her symptoms she was diving in Puget Sound in a wet suit for the purpose of advanced certification. On Day 1 she dove 3 times. The first two exposures were uneventful excursions to 35 ft for 30 min. After an 8-h surface interval she dove to 40 ft for 40 min. She became separated from her buddy and ascended in a controlled fashion. Upon reaching the

surface she noted severe shortness of breath and wheezing. She was aided to shore where she was markedly fatigued. Wheezing and dyspnea persisted for the 2 h it took to arrive home. Symptoms were unrelieved by the use of an over-the-counter inhaled adrenergic agonist medication. She went to bed and slept propped upright because of orthopnea.

She awoke the next morning feeling entirely well. She returned to the training site where she dove to 100 ft maximum depth. Shortly after reaching that depth she developed profound dyspnea. She was aided by her instructor in a slow ascent to the surface, then to shore. Because symptoms of dyspnea and wheezing persisted she was taken to a nearby emergency department. She was found to be hypothermic with tympanic temperature 32.9°C and oral temperature 34.3°C. Her pulse was 64/min, respiratory rate 20/min, and blood pressure 90/60 mmHg. Arterial blood gas analysis on room air revealed pHa 7.39, Pa_{CO_2} 41 mmHg, and Pa_{O_2} 35 mmHg (alveolar-to-arterial O_2 difference, 65 mmHg). Chest radiograph demonstrated pulmonary edema. She was admitted to the intensive care unit and treated with O_2, diuretics, and continuous positive airway pressure by mask. Symptoms entirely resolved overnight, as did the pulmonary edema on chest radiograph. Arterial hemoglobin saturation measured by pulse oximetry improved to 98% with the patient breathing room air.

The patient had no prior history of cardiopulmonary disease. She was taking propranolol and amitriptyline for migraine headaches, as well as estrogen replacement therapy.

She had no recurrence of symptoms with further diving in Fiji or Mexico wearing a wet suit. She has decided not to dive in colder waters.

Diver 6: The patient was a healthy, 24-yr-old male with a history of 130 uncomplicated diving profiles in Puget Sound over the preceding year. He typically dove in a dry suit but had worn a wet suit on approximately 40 of his exposures. On the day he developed symptoms he dove in a wet suit to 15 ft maximum depth to clean the hull of a boat. After 15 min at relatively constant depth he noted the sudden onset of dyspnea. He ascended to the surface and climbed onto the dock. Dyspnea persisted out of the water, associated with a "wheezing" sound within his chest. Shortly after surfacing he began to expectorate watery, blood-tinged secretions. He estimated that he expectorated up to one-half cup of secretions over 30 min.

He went to an emergency department for evaluation. Dyspnea and cough resolved en route. Physical examination and chest radiograph were unremarkable. Arterial hemoglobin saturation measured by pulse oximetry was initially 93% with the patient breathing room air. Arterial blood gas analysis demonstrated pHa 7.42, Pa_{CO_2} 40 mmHg, and Pa_{O_2} 70 mmHg (alveolar-to-arterial O_2 difference 30 mmHg). Over the course of emergency-department observation, arterial saturation by oximetry normalized to 98% and the patient was discharged.

He was taking no medications at the time of the incident. Subsequent evaluation included normal pulmonary function studies and surface echocardiogram demonstrating normal left ventricular systolic and diastolic function. He experienced no recurrence of symptoms with an estimated 100 subsequent dives, 95% in a dry suit and 5% in a wet suit.

DISCUSSION

The syndrome of pulmonary edema among scuba divers was first described by Wilmhurst and colleagues in 1989 (2). They reported 11 individuals who developed pulmonary edema while diving in cold, British waters. All developed dyspnea at depth without excessive exertion, variably associated with a number of other symptoms (Table 1). Two of the individuals had also experienced similar symptoms while surface swimming. All patients had normal cardiopulmonary evaluations. However, each demonstrated high resting forearm vascular resistance and exaggerated vascular reactivity to cold challenge. As the water temperature was less than 12°C when the syndrome occurred, and none had developed symptoms when diving in warmer water, the syndrome was believed to be caused by excessive vascular reactivity to cold, resulting in overwhelming cardiac afterload and left ventricular decompensation.

The next description of the syndrome was published by Pons et al. in 1995 (3). They described three divers who developed dyspnea and cough near the end of uneventful scuba profiles in cold, Swiss lakes (water temperature less than 6°C). All demonstrated rales on physical examination, pulmonary edema on chest radiograph, and resolution of signs and symptoms within 12–96 h. Interestingly, the individuals exhibited forearm vascular reactivity at rest and during cold challenge that was not different from values in

Table 1: Symptoms Associated With Pulmonary Edema Developing During Scuba Diving

	Current Series	Wilmhurst (2)	Pons (3)
Dyspnea	6/6	11/11	2/3
Cough	5/6	11/11	3/3
Orthopnea	2/6	11/11	0/3
Frothy sputum	3/6	7/11	2/3
Hemoptysis	2/6	6/11	2/3
Chest discomfort	2/6	0/11	0/3
Syncope	0/6	2/11	0/3

control volunteers. In addition, Doppler echocardiography was normal at rest and during cold challenge. The authors questioned the importance of cold exposure as a prerequisite for the syndrome. They proposed that a combination of mechanisms was responsible for a transient elevation of pulmonary capillary pressure, with resultant hydrostatic pulmonary edema.

Our patient group exhibited a clinical syndrome very similar to those previously reported. As can be seen in Table 1, symptoms associated with the syndrome are quite uniform among all three series. In our patient population, most episodes occurred during diving in Puget Sound. Water temperatures there are considered "cold" for diving, averaging 12°–18°C at depths less than 50 ft and 4°–8°C at greater depths. In those waters, dry suits are often worn for thermal protection. Despite this, cases of pulmonary edema have been seen among divers wearing either dry suits or wet suits.

It is probable that a variety of factors contribute to the development of pulmonary edema while scuba diving. As suggested by Pons and co-workers (3), the condition is likely to be one of hydrostatic pulmonary edema rather than the result of increased capillary permeability. This is supported by the fact that signs and symptoms of affected individuals often resolve rapidly and spontaneously upon removal from the inciting exposure. Factors potentially contributing to an elevation of pulmonary capillary pressure during diving include increases in cardiac preload and afterload. Water immersion has long been known to increase preload. Head-out immersion in water at 35°C has been shown to increase mean pulmonary artery pressure by more than 12 mmHg (4). Additionally, cold thermal stress increases both preload and afterload (5).

When initially reported by Wilmhurst et al. (2), the syndrome was described as "cold-induced pulmonary edema." It is likely that cold thermal stress does play a significant causative role in the syndrome, because cases reported to date have typically occurred while diving in very cold water. The diving exposures reported in the current series also support the concept of cold thermal stress as an inciting factor for the syndrome, although one individual did experience well-documented recurrent pulmonary edema while diving in the Gulf of Mexico (diver 2). Water temperatures at that location are considered warm for diving, averaging 27°C to depths as great as 250 ft year round. It must be recognized, however, that "warm" is a relative term. While water may feel warm, it can still be colder than body core temperature. While the central pooling of blood associated with immersion is accentuated by cold, it is not prevented even when water temperature is only 1°–2°C cooler than normal core temperature (6).

It should be noted that similar cases of pulmonary edema have also been reported in swimmers. Among the 11 divers with the syndrome described by Wilmhurst et al. (2), two had additionally developed the syndrome while surface swimming in cold water. Two individuals in the report by Pons et al. (3) had developed pulmonary edema while swimming in 18°–21°C water. Finally, pulmonary edema has also been described among 8 of 30 individuals involved in a strenuous military fitness swimming exercise in 23°C water (7). Support for the hypothesis that the mechanism of the syndrome is hydrostatic pulmonary edema is provided by the fact that the individuals in that report consumed an average of 5 liters of water before the exercise to avoid becoming dehydrated. Because of these reports among swimmers, it would appear that immersion is necessary for the syndrome, but not necessarily to a critical minimum depth.

Finally, there is probably an undefined individual physiologic predisposition to the syndrome. Several of the individuals reported to date have experienced recurrent episodes, whereas many more divers perform identical profiles under similar conditions for many years without developing symptoms of pulmonary edema. It is possible that advanced age is a risk factor for the syndrome. The average age of the 20 affected divers described by Wilmhurst et al. (2), Pons et al. (3), and the current report is 42.7± 2.2 yr (mean ± SEM). Demographic data on divers with decompression sickness or arterial gas embolism are collected annually and reported by Divers Alert Network (DAN). In DAN's 1993 report (8), the average age of 508 injured divers was 35.5 ± 0.4 yr, significantly less than the group developing pulmonary edema. Furthermore, only 29% of divers reported with decompression-related injuries were 40 yr of age or older.

At this time, recommendations for treatment of the syndrome and advice for future diving by affected individuals must be based on the limited experience available. According to the history obtained from these individuals, mild cases of diving-related pulmonary edema often resolve spontaneously. Nonetheless, individuals developing pulmonary symptoms in association with diving should seek immediate medical evaluation. Alternate causes for the symptoms must be excluded and appropriate treatment initiated for more severe cases. To date, reported cases of the syndrome have responded well to standard medical therapy for pulmonary edema including oxygen, diuresis, and positive pressure ventilation for respiratory support when necessary.

Individuals diagnosed with diving-related pulmonary edema should be cautioned against future diving. The syndrome may be recurrent among affected individuals and

is unpredictable with regard to water temperature and diving profile. The hypoxemia associated with the syndrome may be severe (e.g., diver 5). Furthermore, some individuals have experienced loss of consciousness in association with the syndrome (Table 1). Until the mechanism of the syndrome is better understood and a method identified to define risk factors for future recurrence, we believe that affected individuals should be advised to forego scuba diving.

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