Pulmonary edema in scuba divers: recurrence and fatal outcome.

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Cochard G, Arvieux J, Lacour J-M, Madouas G, Mongredien H, Arvieux CC. Pulmonary edema in scuba divers: recurrence and fatal outcome. Undersea Hyperb Med 2005; 32(1):39-44. Pulmonary edema occurring in divers using a self-contained underwater breathing apparatus (scuba) is an uncommon, probably under-reported, but potentially life-threatening and recurrent condition. We report six episodes of pulmonary edema in five scuba divers seen during a period of 15 months. The four men and one woman ranged in age from 37 to 56 years and two were treated for hypertension. Symptoms were mostly dyspnea onset at depth, cough, hemoptysis and hypoxemia, which in the recurrent case led to cardiac arrest and death. All cases occurred in rather cold water. Findings on thoracic computed tomography (CT) scanning ranged from pleural effusion to ground-glass opacities restricted to a few areas of the lung. The complex underlying mechanisms that would contribute to a raised transalveolar pressure or to a disruption of the blood-gas barrier are discussed. It is important for emergency care providers to be aware of this syndrome for prompt recognition and optimal treatment.

INTRODUCTION

The occurrence of acute pulmonary edema during recreational scuba diving has been infrequently reported in apparently healthy individuals (1-6). Because this sport enjoys great popularity, the diving population no longer includes only the young and fit. The incidence of scuba diving-related pulmonary edema (SDPE) should thus increase, especially with the stresses imposed on the respiratory and cardiovascular systems by the combined effects of immersion, cold, and compressed air breathing. The pathophysiological mechanisms as well as the risk factors that would contribute to a raised transalveolar pressure gradient leading to capillary breaks in SDPE have been described (7-8). We report our experience with six episodes of SDPE in middle-aged divers with special emphasis on (i) the spectrum on clinical presentation ranging from minor cases to a fatal outcome; (ii) the possibility of recurrence, though presently unpredictable; and (iii) the findings on thoracic CT, which have not been well-documented in this setting.

CASE REPORTS

The Hyperbaric Unit of our hospital serves as a referral center for diving accidents occurring in Brittany (population about 3 million) especially when gas embolism is suspected, although a number of emergency departments less remote from the numerous diving spots are also involved.

Despite this policy, we observed five patients who had experienced six episodes of pulmonary edema while scuba diving from May 2002 to August 2003. Data regarding medical history, incident dives, symptoms, diagnostic evaluations, treatments and outcomes are summarized in Table 1.

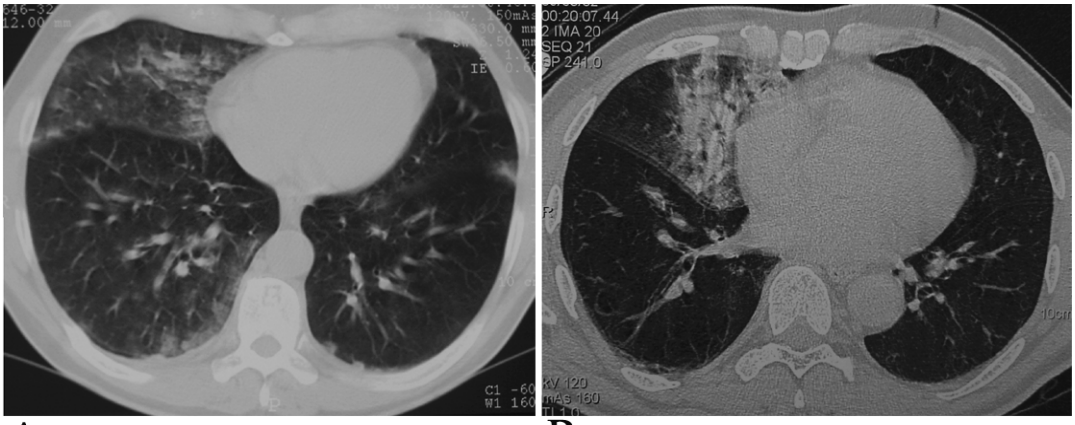
Table 1 – Case summaries

Variables	Case 1	Case 2	Case 3a	Case 3b	Case 4	Case 5
Age (yr) / Sex	53 / F	55 / M	55 / M	56 / M	37 / M	46 / M
Water temperature	15°C	17°C	14°C	10°C	16°C	14°C
Depth / Duration	38 msw/14 min	20 msw/24 min	24 msw/7 min	17 msw/17 min	32 msw/10 min	43 msw/9 min
Symptoms at depth	respiratory distress	respiratory distress	respiratory distress	respiratory distress	loss of consciousness	none
Symptoms on surfacing	dyspnea, cough frothy sputum cyanosis	dyspnea, cough pink frothy sputum, chest tightness	dyspnea, cough hemoptysis	dyspnea, cough bloody sputum cardiopulmonary arrest	unconscious dyspnea, cough bloody sputum	dyspnea, cough chest tightness mild hemoptysis
Medical history	hypertension	arthritis	hypertension dyslipemia arteriopathy	hypertension dyslipemia arteriopathy	none	dyslipemia prior episode ETT (-)
Medications	Quinapril	Rofecoxib Pantoprazole	Candesartan Hydrochloro-thiazide, Acetyl-salicylic acid, Pravastatin	Candesartan Hydrochloro-thiazide, Acetyl-salicylic acid, Pravastatin	none	Simvastatin
First examination	BP 80/60, P 120 bilateral rales	BP 140/90, P 105 bilateral rales	BP 150/90, P 112 unremarkable	cardiopulmonary arrest, bloody frothy tracheal aspiration	conscious, headache BP 130/70, P 100 bilateral rales	BP 130/70 P 90 bilateral rales
Pulse oximetry	SaO_2 85% 9L O_2				SaO_2 79% room air	
Arterial blood gases $pH/PaO_2/ PaCO_2$	7.42 / 92 / 33 9L O_2	7.42 / 80 / 41 CEPAP 70%O_2	7.48 / 62 / 32 9L O_2	major abnormalities	nd	7.42 / 76 / 33 room air
Chest radiograph	diffuse bilateral edema	patchy edema (right lung)	normal	diffuse bilateral edema	perihilar infiltrates	normal
Chest CT scan	nd	right lung : GGO in the 3 lobes + pleural effusion left lung:lingular GGO	GGO in right middle lobe	both lungs : disseminated alveolar opacifications	both lungs : GGO in apices + basal segments	pleural effusion
Diagnostic studies	ECG : ischemia echo : akinesia troponine (+) CPK (+) coronarography (-)	ECG (-) echo (-)	ECG (-) PFT (-) 15 days later	echo (-) after resuscitation	ECG (-) echo (-) PFT (-) 2 days later	ECG (-) echo (-) PFT (-)
Treatment	O_2 by mask Furosemide Nitroglycerin recompression	O_2 , CEPAP Furosemide Nitroglycerin	O_2 by mask	Adrenalin resuscitation intubation	O_2 by mask Furosemide Corticosteroids recompression	O_2 by mask
Outcome	recovery within 48 h	recovery within 36 h	recovery within 8 h	death at 72 h	recovery within 18 h	recovery within 4 h

BP : blood pressure ; CPK : creatinine phosphokinase ; echo : echocardiography ; ETT : exercise tolerance test ; GGO: ground-glass opacities ; nd : not documented ; P : pulse (beats/min) ; PFT : pulmonary function test

In brief, none of the five patients were current smokers or were obese, but two had a treated hypertension, and two had dyslipemia treated with statins. All divers were middle-aged, experienced, but of average fitness. No diving equipment failure (i.e. regulator, tank, air quality) or aspiration of water was encountered. The maximum dive depth ranged from 17 to 43 meters of sea water (msw) at water temperatures of 10 to 17°C. Acute shortness of breath in the absence of strenuous exertion occurred while at depth for all patients, except for patient number 5 who presented with mild tachypnea and bloody sputum shortly after surfacing. This diver had suffered a similar self-limited episode 3 years earlier. Hemoptysis with marked hypoxemia was noted in patients 2-5 after controlled ascent not requiring decompression stops. The female patient, who had a history of essential hypertension, reached the surface after an emergency ascent coughing up frothy white fluid and presented with hypotension. All patients except for patient 1 had a thoracic CT scan showing ground-glass opacities, either diffuse or focal, and/or pleural effusion (Figure 1).These CT examinations were conducted either using the conventional technique (patient 2), or in the high-resolution mode for the remaining patients.

Echocardiograms were performed in all patients, and in patient 1 showed acute ventricular dysfunction. Myocardial akinesia in the apical and inferior territories (ejection fraction of 31%) lasted 24 hours, and was accompanied by a rise in troponin but coronary arteriography was normal. Recompression therapy was performed for possible pulmonary decompression sickness (nitrogen bubble embolization or "chokes") in patient 1 and for initial loss of consciousness (likely related to hypercapnia) in patient 4. The patient for whom two episodes were documented 8 months apart (3a and 3b) died of cerebral edema 72 hours after a cardiac arrest sustained while swimming on the surface to shore. Complete recovery occurred within 4 to 48 hours in the other patients.



A B

Figure 1. Chest CT scan performed immediately after the accident in case 2 (A) and in case 3a (B), showing patchy ground-glass opacifications predominantly within the right middle lobe.

DISCUSSION

A small number of reports (1-8) have described the development of acute pulmonary edema in scuba divers, of whom some have experienced recurrence and similar episodes when surface swimming (Table 2).

Table 2 – Summary of literature on pulmonary edema developing during scuba diving

Author (ref)	Subjects number /male	Age mean (range)	Lowest water temperature	Dive depth (msw) /Duration	Symptoms H/LOC *	Recurrence (D,S) **
Wilmshurst (1)	11 / 8	45.6 (38-60)	< 12°C	nd	6 / 2	2 (S)
Pons (2)	3 / 2	30.7 (26-39)	< 6°C	24-42 /nd	2 / 0	1 (D,S)
Roeggla (3)	1 / 0	54	14°C	5 /10 min	1 / 0	0
Cosgrove (4)	1 / 0	58	11°C	10 /5 min	1 / 0	0
Hampson (5)	6 / 2	43.3 (24-60)	4-27°C	4 -30 /5-20 min	2 / 1	4 (D)
Gnadinger (6)	1 / 1	52	25°C	3 /2 min	1 / 0	0
Slade (7)	8 / 3	52.4 (45-61)	10-26°C	4 -34 /nd	6 / 0	3 (D)
Halpern (8)	1 / 1	50	mild	12 /25 min	0 / 0	0
Our series	5 / 4	49.4 (37-56)	10-17°C	17-43 /7-24 min	3 / 1	2 (D)

*H : hemoptysis; LOC : loss of consciousness
**D : diving; S : swimming

Despite an absence of obvious precipitating causes, we stress that middle-aged divers are most often at risk. In the original description by Wilmshurst et al (1), "cold-induced pulmonary edema" was attributed essentially to hemodynamic modifications (i.e. increased preload and afterload) secondary to immersion and exposure to cold water. The underlying pathophysiological mechanisms in SDPE are actually complex and multifactorial (7-8). Our female patient, who was being treated for hypertension, showed acute hypotensive myocardial dysfunction. In such a case, peripheral vasoconstriction and central blood shift probably play a prominent role by increasing ventricular workload when oxygen delivery is impaired and increased work of breathing is necessary. In all our cases, except patient 1, pulmonary edema was associated with hemoptysis. West et al. have demonstrated that pulmonary edema and hemoptysis are related to stress failure of pulmonary capillaries (9). Increased capillary transmural pressure stretches the capillary wall and alveolar epithelium leading to high-permeability edema with a peculiar "patchy" distribution, indicating focal lung involvement as in cases 2, 3a and 4 (7,9). Furthermore, two divers (2 and 3) had taken medications, aspirin and rofecoxib, which may have predisposed to bleeding. Another factor that could injure the pulmonary blood-gas barrier and contribute to SDPE is excessive negative intrathoracic pressure during inspiration, in relation to elevated gas density at depth and the added resistance of the breathing apparatus (7,9). Negative pressure effects have also been involved in the alveolar hemorrhage observed in breath-hold divers performing voluntary diaphragmatic contractions, and in swimmers with intense exertion (10,11). In this respect, combined exposure to inspiratory resistive loading and head-out water immersion transiently reduced the transfer factor of the lung for carbon monoxide, probably reflecting mild interstitial pulmonary edema (12).

The main differential diagnosis of SDPE is pulmonary barotrauma (with hemoptysis being a hallmark symptom) and decompression sickness ("chokes"), which can

occur either alone or together with SDPE (13). The possibility of "chokes" with coronary air embolism was considered only in case 1 in view of the diving profile, and justified the recompression therapy. None of our patients showed evidence of pulmonary barotrauma on CT scan. In all respects, CT scan proved more sensitive than conventional chest radiography in SDPE to demonstrate discrete pathological changes such as isolated pleural effusion (case 5) or ground-glass opacifications limited to a few areas of the lung (cases 2, 3a and 4). Mechanisms potentially contributing to the focal distribution of pulmonary edema shown in Figure 1 include an increment in closing volume, ventilation-perfusion inequality, uneven vasoconstriction, regional air trapping or lung overinflation. Such a CT scan pattern differs from the more disseminated one observed in breath-hold divers with intra-alveolar hemorrhage (10,14).

Our case series emphasizes that SDPE is a potentially life-threatening syndrome. A loss of consciousness at depth, as in case 4, may have led to drowning. Dyspnea and hypoxemia proved to be severe enough to induce cardiac arrest before the diver was rescued from the water (case 3b). A previous fatal case of SDEP has been reported by the Divers Alert Network based on the autopsy of an experienced female diver with no cardiac abnormalities (7). In sharp contrast, the condition is reputed to quickly resolve spontaneously or with symptomatic medical care, in agreement with the observation in experimental models of rapid healing of the injured blood-gas barrier after normalization of the pulmonary venous pressure (7,9). It is thus likely that a significant number of minor cases of SDPE are undiagnosed, as suggested by diving history in case 5.

Prominent features of SDPE in both previous (1-8) and this series are the diver's age and frequent recurrence with scuba diving and/or surface swimming. This raises the possibility that as yet undefined factors predispose a subgroup of divers to the development of pulmonary edema. Of note, our SDPE patients were significantly older than a group of 98 subjects referred to us for other diving-related injuries (mean age ± SEM of 50.3 ± 7.5 years *versus* 34.0 ± 9.2 years; $p < 10^{-3}$). Age may well represent a risk factor for SDPE through the development of hypertension and physiological changes in respiratory function (5,7). Indeed, progressive changes beginning in the third decade of life have been described in the compliance of the chest wall and lung parenchyma, which may cause air trapping and increased work of breathing (15). However the main problem lies in the decision to resume diving by individuals who have experienced a prior episode, since no testing is available to predict the risk of SDEP. In this report, pulmonary function tests and CT scanning were normal a few days after the accident. Despite a lack of consensus (4,5,7), it seems wise to advise affected divers under age 40 with a normal cardio-respiratory evaluation to avoid cold water or strenuous diving, and older divers with cardiovascular risk factors or pulmonary complaints to forego scuba diving.

In summary, SDPE is an uncommon, probably under-diagnosed but potentially severe and recurrent condition. Although SPDE is a clinical diagnosis of exclusion, chest CT scanning is a valuable diagnostic tool and may increase our insight into a complex pathogenesis. The goal of the present report is to reinforce the need for the clinician to be aware of this syndrome in order to provide optimal medical management.

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