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Papers

Pulmonary oedema and haemoptysis induced by strenuous swimming

D Weiler-Ravell, *consultant pulmonologist*,[a] **A Shupak**, *consultant in otolaryngology and director*,[a] **I Goldenberg**, *physician*,[a] **P Halpern**, *consultant in emergency medicine*,[a] **O Shoshani**, *physician*,[a] **G Hirschhorn**, *physician*,[a] **A Margulis**, *physician* [a]

[a] Israel Naval Medical Institute, IDF Medical Corps, PO Box 8040, Haifa 31080, Israel

Correspondence to: Dr Shupak.

Pulmonary haemorrhage induced by exercise is well known in racehorses.[1] Recent studies attribute the phenomenon to rupture of pulmonary capillaries because of a large increase in pulmonary blood flow and pressure.[2] Pulmonary oedema has previously been described in scuba divers and swimmers who have been immersed in very cold water, albeit without excessive exertion.[3] We report on a group of highly trained swimmers in whom severe dyspnoea and haemoptysis developed during the first 45 minutes of strenuous swimming in temperate Mediterranean waters.

Patients, methods, and results

Thirty young men on a military fitness training programme were engaged in a swimming time trial over 2.4 km in the open sea. The sea was calm and the measured water temperature 23°. They swam in the supine position wearing only a bathing suit and using fins. Because of the high heat load expected, the swimmers had been instructed to drink large quantities of water before the swim, to avoid becoming dehydrated. They each drank about five litres of water during the two hours preceding the exercise.

Summary of patients' characteristics and laboratory results

	Case No							
	1	2	3	4	5	6	7	8
Age (years)	18	18	19	18	18	19	18	18
In clinic at training facility:								
Cough	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Haemoptysis:								
Complained of	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observed	No	No	No	Yes	Yes	No	No	No
Oxygen saturation (%)	95	90	98	84	81	88	87	96
In emergency department:								
Partial pressure (mm Hg)								
Oxygen	84	82	85	66	62	77	82	85
Carbon dioxide	38	36	39	39	37	40	37	38
pH	7.42	7.38	7.45	7.38	7.41	7.39	7.41	7.37
Infiltrates in chest x ray film	Yes	No	No	No	No	No	Yes	No
Recurrent episodes	No	No	No	No	Yes	No	Yes	No

Pronounced shortness of breath developed within 45 minutes in eight of the subjects, forcing five of them to terminate the swim prematurely. Since dyspnoea persisted, they were examined in the clinic at the training facility 30-60 minutes after the drill, and oxygen saturation was measured using a pulse oximeter. Those in more severe distress were given 20 mg intravenous frusemide and all were transferred, receiving oxygen by mask, to a local hospital. The patients' findings and the results of the laboratory investigation carried out in the emergency department are shown in the table. None had abnormal cardiovascular signs or hypertension. Their symptoms and signs resolved spontaneously during an overnight stay in hospital. All eight returned to similar activities and completed the training programme, apart from two (cases 5 and 7) who had recurrent episodes of pulmonary oedema or haemoptysis, or both, when they swam.

Comment

We surmise that three precipitating factors combined to produce a transient increase in the pulmonary capillary pressure: maximal exertion, immersion, and overhydration. Exercise causes an increase in cardiac output to meet the tissues' increased demand for oxygen. However, the increase in cardiac output will by itself rarely if ever raise pulmonary capillary pressure to the point of rupture in humans; pulmonary oedema and haemoptysis have never been reported even in top Olympic athletes with above normal cardiac performance. Immersion causes central blood pooling, thus increasing cardiac preload.[4] Water overload, the result of well intentioned but overzealous concern about dehydration, probably further increased pulmonary vascular pressure.

Haemoptysis and pulmonary oedema recurred in two of the patients without an increased fluid intake; recurrences gradually became less severe, gradually proceeding to complete spontaneous resolution. They may have been the result of residual damage to the alveolar-capillary unit, which finally healed.

Pulmonary oedema associated with immersion has previously been described in 11 divers and swimmers exposed to cold water.[3] Although the subjects did not engage in heavy exercise and their water balance was probably normal, increased vascular reactivity was shown by high resting peripheral vascular resistance and a reduced response in a cold pressor test. Vasoconstriction, induced by cold that led to an increase in cardiac preload and afterload, combined with augmentation of cardiac preload associated with immersion was thought to explain the development of pulmonary oedema.[3] We suggest that the simultaneous occurrence of contributing factors is required for the development of pulmonary haemoptysis and oedema associated with immersion: cold induced vasoconstriction, as previously reported,[3] or the combination of exercise and fluid overload as in our cases.

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