Case reports

Pulmonary oedema in breath-hold diving: an unusual presentation and computed tomography findings

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Abstract

(Gempp E, Sbardella F, Cardinale M, Louge P. Pulmonary oedema in breath-hold diving: an unusual presentation and computed tomography findings. *Diving and Hyperbaric Medicine*. 2013 September;43:162-163.)
Haemoptysis and pulmonary oedema following deep breath-hold diving have been described in recent years. We describe the case of a 33-year-old healthy military diver who presented symptoms suggestive of pulmonary oedema after two breath-hold dives, the first lasting 0.5–1 min and the second 1–2 min, to 6 metres' depth in the sea. The diagnosis was promptly confirmed with chest computed tomography showing bilateral interstitial infiltrates in the upper regions of the lungs. To our knowledge, this is the first report to document pulmonary oedema in this setting of shallow breath-hold diving with atypical radiological presentation. A definite mechanism for this specific distribution of lung injury remains unclear.

Key words
Breath-hold diving, freediving, pulmonary oedema, radiological imaging, case reports

Introduction

In recent years, haemoptysis and pulmonary oedema have increasingly been observed in competitive breath-hold divers and underwater fishermen after deep dives.[1–3] These disorders have also been described in endurance swimmers and scuba divers but there is no case reported in very shallow diving depths except after experimental breath-hold dives preceded by full expiration.[4,5] This report, presented with the patient's consent, is the first to document computed tomography (CT) findings consistent with interstitial-alveolar damage due to capillary stress failure in this setting and is presented with the patient's consent.

Case report

A 33-year-old man was admitted complaining of cough and pink, frothy sputum immediately after two breath-hold dives to a depth of 6 metres' sea water, the first lasting 0.5–1 min and the second 1–2 min, with a surface interval of less than 1 minute. He noted wheezing but had no chest pain nor dyspnoea after exiting the water. The patient was a healthy military diver candidate on the first day of a naval ship diving course. He denied aspirating sea water and performing manoeuvres such as glossopharyngeal insufflation and voluntary diaphragmatic contractions. He was not taking any medications and was a non-smoker with no past medical or family history of cardiovascular disease or immersion pulmonary oedema. The water temperature was 10^{O}C and he was wearing a 5-mm neoprene wetsuit with 3 kg of weight. He was immersed at rest for about 10 minutes waiting his turn to dive. No significant strenuous fin swimming or physical exertion was performed during the hours preceeding the diving session.

On admission 90 minutes after initial symptoms, the diver was comfortable. Physical examination revealed mild, diffuse, bilateral 'crackles' on chest auscultation. His pulse was 72 bpm and blood pressure 130/70 mmHg. Arterial blood gases, laboratory investigations (including clotting tests, D-dimer and cardiac biomarkers) and electrocardiogram an (ECG) were normal. Chest CT scan showed bilateral interstitial and alveolar infiltrates in the upper lobes while the lower zones were remarkably normal (Figure 1). Follow-up investigations at one month, including ECG, exercise testing, spirometry with lung volumes and chest X-ray, were unremarkable.

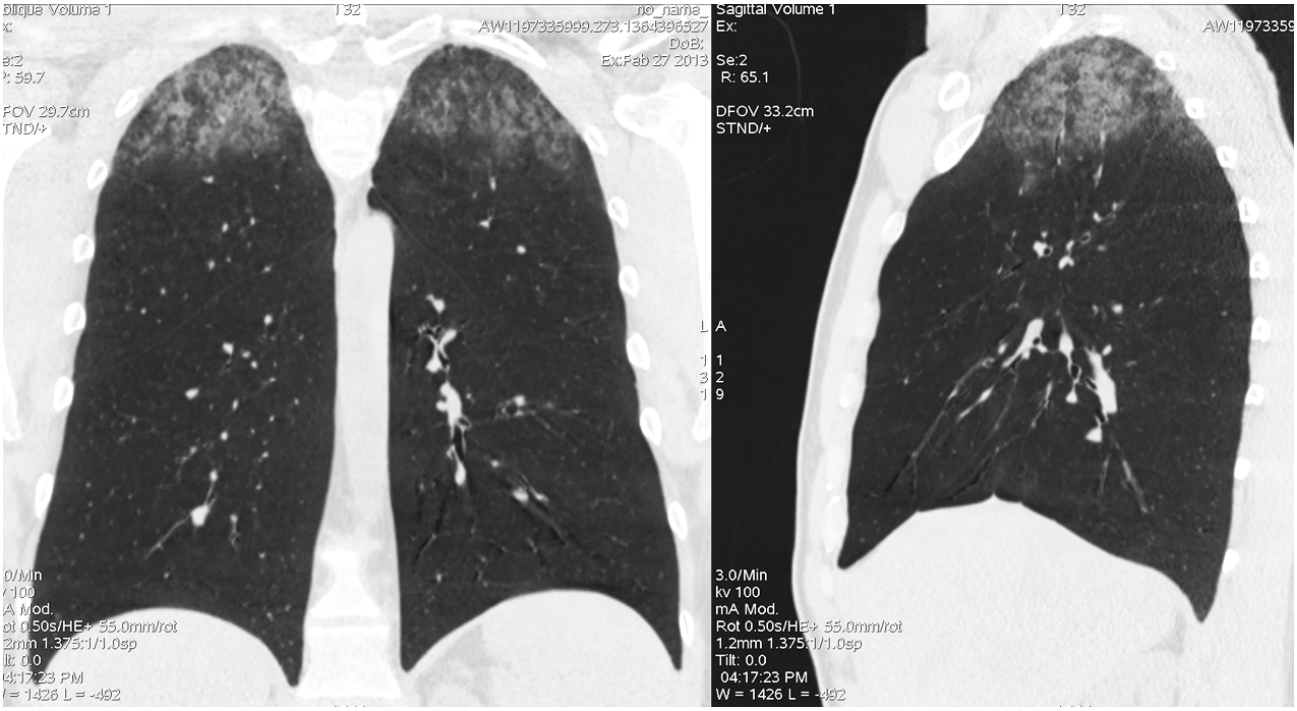
Discussion

Several factors contributing to pulmonary oedema in breath-hold diving have been identified, including cold water immersion leading to central pooling of blood and haemodynamic alterations, reduction of lung gas volume during descent ('lung squeeze'), exertion, hypoxia and diaphragmatic contractions.[3,6,7] Taken together, these mechanisms result in an increased trans-capillary pressure gradient that would disrupt the blood-gas barrier and cause alveolar haemorrhage. Some authors argue that aspirin or other non-steroidal anti-inflammatory drugs may also promote this condition.[8,9] It has also been postulated that neurohumoral stimulation resulting from emotional stress may be involved, as might be the case here.[10]

Although the radiographic features associated with pulmonary oedema in breath-hold divers have been well described in the literature, there are no data examining the distribution of these lesions within the lungs.[7,8] The striking preferential bilateral localisation of ground-glass opacities to the upper-zones in our case is rather

Figure 1

Chest CT scans with coronal and sagittal reconstruction (minimal intensity projection technique) that demonstrate areas of ground-glass opacities involving the apices of both lungs and sparing the rest of the parenchyma. Note the lack of Kerley lines or visibly enlarged vessels



unexpected. A proposed mechanism for the development of this pathological condition is the changes in alveolar pressure regimen between the lower and upper regions of the lungs that tends to be more negative in the apices when the diver has a vertical head-down posture during the descent. As a result, the transmural vascular pressure gradient and capillary stress should be maximal in those regions, thus leading to this specific pattern of interstitial oedema and subsequent alveolar injury.

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