The diving doctor's diary

Scuba divers' pulmonary oedema. A case report

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Key words
Immersion, scuba diving, pulmonary oedema, case reports

Abstract

(Edmonds C. Scuba divers' pulmonary oedema. A case report. *Diving and Hyperbaric Medicine*. 2009;39(4):232-3.)
A case of presumed scuba divers' pulmonary oedema (SDPE) is presented. The symptomatology and clinical progress is typical and it illustrates some of the aetiological factors incriminated in this disease, as well as measures taken to avoid the disorder. Such factors include the effects of immersion, negative-pressure inspiration induced by vertical positioning in the water, resistance to breathing by the scuba regulator and advanced age. Dyspnoea may have been exacerbated by the respiratory restriction imposed by the diving equipment. Other aetiological factors not relevant to this case are mentioned, as is the possible differential diagnosis. An accompanying review article discusses SDPE in greater detail.

Introduction

The following is a fairly typical case history of scuba divers' pulmonary oedema (SDPE) that illustrates the difficulty facing a diver who must act on the proposed theoretical mechanisms. Unfortunately, these are mainly speculative and there is little direct experimental evidence that any one specific characteristic of scuba diving is responsible for the pulmonary oedema observed.[1]

Case history

On the first dive of the second diving day, a 72-year-old experienced diver surfaced from a dive to a maximum depth of 15 metres' sea water (msw) for a duration of 50 minutes, with an extra five minutes at a 3–5 metre decompression safety stop. Water temperature was 27^{O}C and there was only a slight current. There was no rapid ascent, no salt-water aspiration or other dive incident. It was an innocuous dive, without any excessive exertion. During the safety stop he noticed increasing dyspnoea, but attributed this to a low-on-air situation, having only 40 bar remaining.

He was at the surface in a head-out vertical position for about 10 minutes, during which time he had inflated his buoyancy compensator (BCD). During that time, he noted increasing difficulty in obtaining sufficient air from his demand valve and snorkel. He also observed the sound of fluid in his lungs (rattling breathing). He attempted to relieve the dyspnoea by pulling on the neck of his tight wetsuit, without effect. Dyspnoea, fatigue, the moist breath sounds, cough and expectoration continued after he boarded the dive vessel, and then in a diminishing manner for the next 2–3 hours, all aggravated by exertion.

When diving on the preceding day, he had complained to the dive organiser that there was resistance to breathing from the main demand valve at 18 msw. He had also tried to exchange the wetsuit for one that fitted him better, but none was available. On commencing the first dive of the second day, it was evident that the hired demand valve had not been repaired or replaced, and this breathing resistance was again noted.

After a 4–5 hour surface interval, he felt normal and so dived again on an almost identical profile, but without any incident or difficulty. For this second dive he dispensed with the tight wetsuit (hired) and rejected the primary demand valve for the one on his octopus rig (also hired). This regulator produced no excessive resistance to breathing. On surfacing, he assumed a horizontal position and breathed through his snorkel.

The diver deduced that his dyspnoea was related to breathing against an appreciable resistance (negative-pressure inspiration), vertical position at the safety stop and during his head-out immersion after surfacing. It was possibly aggravated by his tight wetsuit. He also wondered if the inflated buoyancy compensator had been contributory.

Discussion

In this case, as in many similar reported cases, there was no immediate medical investigation of the incident to verify the diagnosis, but the clinical manifestations were indicative of SDPE. The vertical or head-out immersion position could have contributed to a fluid shift of blood into the lungs and the tight wetsuit and inflated buoyancy compensator could have accentuated the work of breathing by decreasing chest-wall compliance. These factors were all avoided on the second dive by changing regulators, not wearing the wetsuit, not over-inflating the buoyancy compensator and swimming horizontally after surfacing.

Specifically, in this case the dyspnoea seemed *not* to be related to:

- cold exposure (warm water, wetsuit);
- hypertension (normotensive, BP 125/80);
- ischaemic heart disease (maximal stress ECG a month before the incident was normal, and no symptomatology developed afterwards);
- salt-water aspiration (this did not occur);
- asthma (no clinical or spirometric indication);
- excessive depth (symptoms first noted near and on the surface).

There was no other evidence of decompression sickness, and the dives were well within no-decompression limits.

Pulmonary oedema in divers has been reported in the literature since 1981.[2] Though uncommon, it is probably under-reported because it typically resolves rapidly once the diver emerges from the water, as in this diver, and death is only rarely reported. Although it may occur in apparently healthy young divers, it is more frequent in older divers and tends to be recurrent. Clinical presentation includes dyspnoea, fatigue, cough and expectoration, sometimes blood-stained, with hypoxia and auscultatory and radiological signs of pulmonary oedema.

The proposed mechanisms encompass both cardiac and non-cardiac aetiologies. The cardiac causes include physiological changes in both myocardial pre-load and after-load with immersion and the presence of cardiac ischaemia. Non-cardiac causes include changes in pulmonary physiology and equipment-related limitations. Other diving-related conditions such as salt-water aspiration, near drowning, respiratory oxygen toxicity and gas contamination may induce an inflammatory exudate that may present as pulmonary oedema – more protracted than the transudates. Pulmonary decompression sickness and pulmonary barotraumas are diving disorders that may cause diagnostic confusion with other pulmonary oedemas. These various factors in the aetiology of SDPE are discussed in detail in the accompanying review paper.[1]

References

1 Edmonds C. Scuba divers' pulmonary oedema. A review. *Diving and Hyperbaric Medicine*. 2009;39(4):226–31.

2 Wilmshurst P, Nuri M, Crowther A, Betts J, Webb-Peploe MM. Forearm vascular response in subjects who develop recurrent pulmonary oedema when scuba diving: a new syndrome [abstract]. *Br Heart J*. 1981;45:A349.

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The poetry doctor

Radical thoughts

I am an oxygen radical,
A rebel molecule,
With a negative attitude
To disobey the rules.

Electrically I am unpaired,
Marriage ain't for me.
A single electron fully bared
I'll always wander free.

I'm symbolized by a dot
Implying I'm a blob
But don't think I'm running on the spot
For I'm manic on the job.

People think that I am mad
Because I am unstable
But my reactivity
Is my species label.

I can do astounding things,
A "superoxide" God,
Invincible to everything
Except that enzyme SOD!

Because of this I've had bad press
That's caused much angst and raging
Stating that I am the cause
Of cancer and of aging.

These are lies to scare you all
To doubt longevity
So you'll take huge doses of
Vitamins E and C.

I even help defend you
And aid phagocytosis.
My actions are very sane
Not driven by psychosis.

So don't be fooled by all the spin
That I offend with stealth.
I am conservative deep within
And essential for good health.

John Parker
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