DIVING DOCTOR'S DIARY

PULMONARY OEDEMA, DYSPNOEA AND DIVING

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Key Words

Cardiovascular, case reports, drugs, equipment, immersion, medical conditions and problems.

Introduction

Over a decade ago I was contacted by the physician on St Thomas Island in the Caribbean and asked: Why was it that there were so many surface snorkellers developing pulmonary oedema, and who also had hypertension treated with beta-blockers?

I explained to him the significance of the beta-blockers, i.e. the production of asthma-like states with obstructive airways disease, the reduction in effort tolerance and the possible detrimental cardiac effects of bradycardia of immersion. I was not then very interested in snorkellers. We certainly had observed the cardiac death risk in divers who were also on these drugs.[1]

I was reminded of this association by the most recent *Alert Diver*, which described two scuba divers with pulmonary oedema, in two separate articles.[2,3] The first was in a hypertensive female tourist taking beta-blockers (with the implication that if she had been seen by an Australian or New Zealand diving doctor, this would have been avoided).[2] The second was in a middle aged male who went on to cardiac surgery once the cause was clarified.[3]

I was impressed with the coincidence of recently reviewing two similar cases of cardiac induced pulmonary oedema, but with some interesting variations. These were similarly dyspnoeic, but in swimmers, not divers, and both were doctors. Each was identified as pulmonary oedema by the treating institution.

These two physicians present a not uncommon problem, similar to those of St Thomas', but one not well recognised; the production of dyspnoea associated with immersion per se.

Case reports

CASE 1

Late last year I received an e-mail from a Swedish pulmonary physician. She wrote:

"I found your interesting article via the Internet, on snorkelling deaths. I am 61 years old and visited Australia in October for a conference. We went to Cairns and tried snorkelling on the reef. In the water I got pulmonary oedema and had to be transported to hospital by helicopter and stay there for 4 days. I have never had problems like that before although I have some hypertension. I did not aspirate seawater and was not diving. I am not a good swimmer and it has made me a little nervous although I have snorkelled before. That cannot explain what happened. My question: Could it have been caused by a tight wetsuit or the dead space in the snorkel? I do not have large lung volumes, but I am physically active for my age.

Well, I survived and am not going to snorkel any more, but it would be nice to hear your opinion".

I am not enthusiastic about answering questions over the Internet, especially when I know that I am not going to get the full history and am denied the luxury of a physical examination, let alone appropriate investigations. I replied as follows:

"Before I respond to your questions, I need to know:-

1 What medication were you on (especially beta-blockers)?
2 Have you had a stress ECG? If yes, give the full report.
3 Any history of heart or lung disease (e.g. asthma)?"

The reply came almost before I had finished sending my questions!

"Dear Colleague,

Thank you for reading my question (Internet is fantastic).

1 I am on a beta-blocker, metoprolol tartrate (Metoprolol) 100 mg, and an angiotensin 2 receptor antagonist, candesartan cilexetil (Atacand) 16 mg.
2 No stress test was done before the snorkelling but it was before I left the Hospital in Cairns and that was without any problem. It was made because of a raised troponin level.
3 No history of heart or lung disease before. I am physically rather active for my age and my ECG at rest has been normal."

A discharge summary from Cairns Hospital indicated that the diagnosis was a cardiac induced pulmonary oedema, with myocardial ischaemia as the cause. She was commenced on Simvastatin and aspirin. The exercise stress test, while on beta-blockers, produced a maximum blood pressure of 220/100 and a maximum workload of 10.4 mets. There were no ECG changes or chest pains during exercise, but a U-shaped ST depression infero-laterally became more

horizontal during the recovery phase and was considered to be probably significant.

As so often happens is these cases, the patient's description was not completely accurate.

I informed her that I considered further investigations should be carried out regarding her cardiac status and that full lung function tests be performed. The latter were undertaken and there was no sign of any bronchial obstruction, even when on the beta-blockers.

CASE 2

This was more informative, because of the subsequent developments.

A 55 year old male had mild hypertension, was taking a beta-blocker (metoprolol) and aspirin, had no problems associated with scuba diving. But with surfing he regularly developed mild dyspnoea, 5-10 minutes after entering the surf, irrespective of the sea state. It would be relieved as he continued his surfing activities. Over 5 years this observation was verified on hundreds of occasions, but the dyspnoea seemed to be increasing in intensity and was affecting his aquatic fitness.

By the time he had his angiogram, which demonstrated multiple coronary vessel obstruction, he was experiencing dyspnoea not only during surfing but also with other exertion. He did not suffer angina pectoris in the aquatic environment, but this was becoming pronounced with terrestrial activities, being provoked by cold temperature, mild to moderate exertion, emotion, excessive food intake, etc.

Following a successful coronary artery bypass graft, all clinical evidence of cardiac ischaemia disappeared and he had no evidence of ischaemia on either the stress ECG or thallium scan.

When he resumption surfing he experienced no further dyspnoea with immersion and no apparent impairment of physical fitness during this activity.

This case demonstrates the successful influence of coronary flow restoration in the prevention of episodes of dyspnoea and pulmonary oedema during immersion.

Discussion

The pathophysiology underlying these cases was relatively clear because of the absence of any diving activity. With scuba diving there is a greater complexity in the development of dyspnoea and pulmonary oedema, as described in a recent diving medical text.[4]

During the 19th century immersion in a bath was one treatment given for "dropsy", demonstrating the effects of immersion on the redistribution of peripheral fluid.

Linnarsson assessed subjects with cardiac disease during an increasing work load, both in the water and on land, until symptoms or ECG abnormalities developed. The aquatic environment consistently masked the anginal symptoms of myocardial ischaemia.[5]

Diving diseases

Pulmonary oedema has been described in a variety of diving diseases. In some of these, it is a consequence of other diving respiratory pathology, such as in;

- the drowning syndromes, including salt water aspiration,
- pulmonary barotrauma,
- decompression sickness,
- underwater blast,
- some gas contaminants,
- pulmonary oxygen toxicity,
- some marine animal envenomations (e.g. the Irukandji syndrome).

In other instances pulmonary oedema has been described as the primary manifestation from a diving exposure.[6] Sometimes dyspnoea occurs in otherwise healthy individuals during scuba diving. The disorders occur while swimming or diving in shallow water, sometimes in the head-out position. The symptoms include dyspnoea, cough, haemoptysis and expectoration of frothy sputum.

The pathophysiological explanations advanced are often not completely convincing. Nevertheless there are sufficient such cases to warrant concern. The various presumed aetiological types are as follows.

Pulmonary oedema of immersion.

Immersion of the body, with the head above water, will have significant pulmonary effects.[8] Because of the hydrostatic pressure exerted by the water there is a redistribution of the blood (over half a litre) into the large intrathoracic blood vessels and, to a lesser extent, into the pulmonary microcirculation. Immersion also greatly affects the structure and function of the thoracic cavity. The following changes occur;

- reduced vital capacity (approximately 5%),
- reduced static lung compliance,
- increasing closing volumes leading to,
  - increase in functional air trapping,
  - increase in diffusing capacity,
  - increased flow resistance in airways,
  - altered ventilation, increasing in the apical regions

and reduction in functional residual capacity.

Pulmonary oedema and dyspnoea associated with cardiac disease.

This may be related to pulmonary oedema of immersion. Wilmshurst recorded the observation of pulmonary oedema, inducing dyspnoea in divers, associated with hypertension and postulated a number of aetiologies.[9] The stress factors which combine to induce this disorder include; increased cardiac loading due to the effect of water immersion, the respiratory changes noted above and cold induced peripheral vasoconstriction increasing the workload on the heart.

The effect therefore included increased cardiac pre-load, increased breathing resistance and exaggerated cardiac after-load.[10]

Some older divers, especially males, will develop dyspnoea 5 to 10 minutes after immersion. In these cases there is probably also a precipitation of cardiac ischaemia or arrhythmias because of the many physiological effects of immersion.

The following cardiovascular effects of immersion have been observed;

increased central blood volume, with blood passing from the periphery to the lungs due to the hydrostatic gradient,
increased stroke volume with increased cardiac output,
increased peripheral perfusion,
extrasystoles during the early phase of immersion (due to cardiac distension),
the diving reflex, when facial immersion and trigeminal stimulation induce bradycardia, shunting of the blood to the heart/brain axis from the periphery and viscera, and peripheral vasoconstriction,
the cardiac effects of cold exposure
and hyperbaric bradycardia, usually by 10 beats per minute, which is possibly related to increased oxygen partial pressure, increased gas density, increased hydrostatic pressure and a narcotic effect of inert gas.

Each or all of these may play a part in inducing mild pulmonary oedema and the respiratory symptom of dyspnoea. Subsequent investigation will demonstrate the presence of coronary artery insufficiency or arrhythmia and the problem will disappear when this is corrected.

Troponin estimations may supplement the traditional ECG and cardiac enzymes (CK and CK-MB) in determining cardiac involvement.[10]

Asthma provocation.

The scuba situation is likely to induce asthma in those so predisposed. There are multiple provoking factors, including;

exercise, especially if swimming against a significant tidal current,
breathing against a resistance (the demand valve),
breathing cold dry air (decompressed air),
breathing dense air (related to depth),
salt water spray inhalation (leaky demand valve) and
psychological stress and hyperventilation.

Cold urticaria.

Cold urticaria is a specific disease due to histamine release after exposure to cold. It may affect the skin, gastrointestinal tract and the lungs.

In scuba diving there is a reduction in breathing air temperature as the air pressure drops during its passage from the high pressure cylinder to the diver (Charles Law) and by-passes the nasal cavity with its warming and humidification effects.

Deep diving dyspnoea.

Cold gas inhalation at depth makes all divers susceptible to dyspnoea at great depths, due to convective heat loss in the airways and the local response to this.

Other non-diving disorders

Other non-diving disorders may present as dyspnoea while diving, because of the occasional exceptional physical demands of this activity. Examples are lung disease, drug effects from beta-blockers or irritants such as cannabis.

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