Acute pulmonary oedema in a hypertensive snorkel swimmer

F Michael Davis

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Abstract

(Davis FM. Acute pulmonary oedema in a hypertensive snorkel swimmer. *SPUMS J*. 2005; 35: 209-10.)
A case is reported of a 52-year-old woman with known hypertension on medication, who developed acute pulmonary oedema whilst snorkelling in tropical waters. Subsequent management and investigations are described. No underlying myocardial disease was identified. This is presumed to be a case of immersion pulmonary oedema, contributed to by her medications, which included ß-blockers. Subsequently, a rare dopamine-secreting carotid body tumour, which was likely to have contributed to this event, was diagnosed and successfully resected.

Introduction

The sudden onset of acute left ventricular failure whilst swimming, snorkelling or scuba diving may have a variety of causes. Whilst myocardial ischaemia, particularly in the face of pre-existing cardiovascular pathology in the middle-aged or elderly, would be the first consideration in many cases, acute pulmonary oedema has also been reported in young, healthy individuals.[1–4] An apparent case of immersion pulmonary oedema in a snorkeller occurring in warm tropical waters is reported here.

Case report

An active, mildly obese 52-year-old female anaesthetist was vacationing in Queensland. She was a known hypertensive on nadolol 50 mg tds and diltiazem 280 mg daily, with only modest control. On the third day of a visit to an island on the Great Barrier Reef, she went snorkelling in calm, sunny conditions. She had snorkelled before, but not for some years, and was inexperienced. After about three-quarters of an hour she noticed some difficulty making headway, and she told her companion that she would swim to shore, about 200 m away, with the tide assisting her.

She describes what happened as follows.
"*I found my legs were very weak, which was worrying, but what started to preoccupy me was the presence of a lot of bubbly fluid in my chest. I thought I must have aspirated some sea water, but knew I hadn't. It took a very long time to get out of the water, and I was very breathless and bluer than I have ever seen anyone. Pink frothy stuff was coming out of my mouth. I had no chest pain…but felt weak.*

This is a resort island with a well-stocked first-aid clinic and a nurse. Unfortunately, the newly qualified nurse had no experience in acute medicine, she had never put in an intravenous (IV) line and she started to cry. I reassured her, and we obtained permission from a hospital junior doctor on the mainland to use morphine and frusemide, but the island's management would not allow me to put in my own IV. Half an hour later, by which time I was cross and exhausted, an elderly neurologist appeared who had not put in an IV for 25 years. I wasn't surprised, but welcomed him (through the oxygen mask and pink frothy sputum) with "Sit down and supervise this" and proceeded to insert my own IV. Morphine and frusemide were administered and I began to feel much better."

The snorkeller's condition having improved considerably, and after some discussion, the decision was made to evacuate her by boat to the mainland the following morning. The morning after that she was seen by a cardiologist and immediately admitted to the coronary care unit of a local private hospital. On admission, she appeared fatigued and dyspnoeic at rest. There were crepitations at both lung bases, a soft third heart sound and oxygen saturation on air was 93%. Echocardiography showed an area of infero-lateral left ventricular wall hypokinesis and mild mitral regurgitation. The only abnormality on electrocardiogram was peaked T-waves in the lateral chest leads.

Whilst in hospital, she had two episodes of anginal-type pain and a few brief episodes of palpitations, but cardiac iso-enzymes were not elevated, excluding an acute myocardial infarction, which had been the presumed diagnosis. She made steady progress and was discharged on the sixth day on aspirin, nadolol, diltiazem, frusemide and an ACE inhibitor. At this time, she was able to walk for more than an hour and a half without dyspnoea, and had no orthopnoea or palpitations. Blood pressure was normal and a 3/6 pan-systolic murmur was noted. The attending cardiologist stated in the discharge letter *"my impression is that there are certain pieces missing from this puzzle*."

Echocardiography two weeks later showed a left ventricular ejection fraction of 87% with no evidence of regional dysfunction or mitral regurgitation, the only abnormality being a mildly increased left ventricular mass index, consistent with her hypertension. A Bruce protocol exercise

study was normal, with a heart rate of 89% of predicted maximum being achieved. Coronary angiography showed a dominant left main coronary artery of normal calibre and there were no significant flow-limiting coronary artery lesions.

However, the story does not end here. This woman's labile hypertension persisted, and a right submandibular lump was noted. Further investigations revealed elevated dopamine levels to nine times above normal. Nine months after the immersion incident, she underwent resection of a non-malignant right vagus paraganglionoma, confirmed to be dopamine secreting. Several cranial nerves were sacrificed during surgery and she underwent a prolonged recovery. Six years later she remains well, still in full-time anaesthesia practice.

Discussion

Immersion pulmonary oedema was first reported by Wilmshurst in eleven, middle-aged, hypertensive scuba divers in cold water.[1] The only feature of note in these divers was the development of a high peripheral vascular resistance response to forearm cold water (less than 12^{O}C) immersion. Subsequently, similar episodes in healthy young scuba divers, fin swimmers during long surface swims and in recreational snorkellers have been documented.[2–4] Several factors, such as overhydration, ischaemic heart disease and some drugs, particularly ß-blockers, are believed to be contributory. However, it may arise in an otherwise healthy individual with no apparent risk factors.

The pathophysiology of immersion-induced pulmonary oedema is not fully understood. Immersion and cold exposure cause peripheral vasoconstriction, with an increase in cardiac pre-load and after-load as blood volume is centralised and peripheral resistance increases. There is an increase in mean pulmonary artery pressure and pulmonary capillary wedge pressure. The engorgement of the pulmonary blood vessels may predispose to capillary stress failure. A more detailed discussion of the pathophysiology and the differential diagnosis of immersion pulmonary oedema has been presented by Mitchell.[5]

Immersion pulmonary oedema was not considered at any stage in the differential diagnosis in this patient, though the cardiologist concerned, who had no diving medical training or experience, clearly felt he was missing a key piece of the jigsaw. This appears to be a classic case, with the associated risk factors of hypertensive cardiovascular disease and use of ß-blocker medication in an inexperienced snorkeller, although it is very uncommon for this to occur in warm tropical waters.

Cases of Irukandji envenomation with chest pain, particularly if pulmonary oedema develops, may be misdiagnosed as acute myocardial infarction with developing heart failure.[6] This may be reinforced by a history of swimming (exertion), especially if the history of a mild sting is not elicited, or is forgotten by the victim. Elevated levels of troponins and/or CK-MB are taken as a measure of cardiac damage. In this patient cardiac iso-enzymes were never elevated, and Irukandji syndrome cannot be completely excluded from the differential diagnosis as she was snorkelling within the known geographical distribution of *Carukia barnesi*. However, the general absence of pain during the early post-immersion period makes this very unlikely.

This case of acute immersion pulmonary oedema is unusual in that a contributory pathology was subsequently identified.

Acknowledgments

Permission by the patient to report details of her experience and subsequent clinical course is greatly appreciated.

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Michael Davis, MA, FRCA, FANZCA, MD(Otago), is Associate Professor of Medicine in the Faculty of Medicine and Health Sciences, The University of Auckland, and Medical Director of the Hyperbaric Medicine Unit, Christchurch Hospital, New Zealand.

Address for correspondence:
Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand
Phone: *+64-(0)3-364-0045*
Fax: *+64-(0)3-364-0187*
E-mail: *<michael.davis@auckland.ac.nz>*