

CASE REPORTS

Acute shortness of breath: an unusual cause

H Cosgrove, H Guly

Abstract

A case of cold induced pulmonary oedema in a scuba diver is described. This is rare, but with the increasing popularity of the sport it is important for accident and emergency staff to be aware of the condition. Treatment is symptomatic and the outlook is good.

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Key terms: cold induced pulmonary oedema; scuba diving

Cold induced pulmonary oedema in scuba divers is a rare condition. No definite incidence has been reported but Wilmhurst¹ was able to locate 11 people in Britain with the condition. It is recognised by those involved in the medical management of divers, but does not appear to be well known among the general medical population. We present a case report of this condition.

Case report

The patient was a 58 year old woman. She was an experienced warm water diver, but this was her first dive in cold water. Her breathing tanks had all been filled at the same shop as those of her companions. Within the previous six months she had passed a routine medical assessment of her fitness to dive. She was a life-long non-smoker on no medication. Her only documented health risk was "slightly" raised cholesterol discovered at an insurance medical. She had no treatment for this.

She made her first ever cold water dive on a bright sunny May morning. The sea temperature was 11°C. She was wearing a wetsuit with hood, gloves, and shoes. Less than 10 minutes after entering the water, before she had started to dive, she started to feel short of breath and weak. She had difficulty in attaining the 10 metre depth of the dive and had to be helped down by her companions. There was no suggestion of malfunction of her equipment. She continued to feel unwell and reports that the shortness of breath and exhaustion became more extreme. She returned to the surface almost immediately and had to be helped from the water. She promptly coughed up copious amounts of "pink froth". She was brought to the accident and emergency department by car, arriving approximately one hour after leaving the water.

On arrival she was grey, cold, clammy, and pale. She had a pulse of 82 beats/min, regular but small volume, blood pressure of 68/40 mm Hg, respiratory rate of 28, and an oxygen saturation of 70% on air. She was able to talk in short phrases and complained of feeling very tired. She was more comfortable sitting upright. Examination of her chest revealed symmetrical air entry and widespread coarse crackles throughout all zones. Chest *x* ray revealed pulmonary oedema. She was given high flow oxygen by face mask and 40 mg frusemide intravenously. She started to look and feel better quickly. Ten minutes after this treatment her oxygen saturation was 98% and her blood pressure was 100/60 mm Hg. Electrocardiogram (ECG) and routine blood testing were normal. She was admitted for observation and oxygen treatment. She maintained a good urine output throughout her admission and required no further medication; serial electrocardiograms and cardiac enzymes were normal.

She was discharged home the next day, well, with plans for follow up in her local hospital.

Discussion

The differential diagnosis of the diver who is dyspnoeic after a dive includes cardiac disease, contaminated gases, pneumothorax, near drowning, and cold induced pulmonary oedema.¹

Acute pulmonary oedema occurs when the pulmonary capillary permeability is increased, or when the hydrostatic pressure in the pulmonary circulation exceeds the plasma oncotic pressure. This second situation may arise if the preload and the afterload rise acutely. Immersion causes a sudden and temporary rise in cardiac output. The effects of gravity are negated and external pressures enhance the venous return of fluid in the limbs to the heart. This increase in the central blood volume increases the preload on the heart.^{1,2}

Immersion in cold water also produces an increase in sympathetic activity which among other effects produces a peripheral vasoconstriction. This increases both the central blood volume and preload on the heart, and the afterload. This response is exaggerated in divers with a history of acute pulmonary oedema on immersion in cold water. Wilmhurst *et al*¹ have shown that the forearm vascular resistance in these individuals in

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creases more in response to a standardised cold stimulus than control divers with no problems diving in cold water.

There are no long term complications of this condition. A group of 11 divers who had had documented pulmonary oedema on immersion in cold water was followed for a mean of eight years. All had above average exercise tolerance. Seven went on to develop mild hypertension and one developed atrial fibrillation. There were no deaths, and no one developed symptomatic heart disease.

CONCLUSION

The development of pulmonary oedema on immersion in cold water has obvious repercussions for the recreational diver. With the

increased popularity in diving it is important that staff in accident and emergency departments are aware of this condition. Treatment is easy and is symptomatic only. There do not appear to be any long term serious complications, except that a watch should be kept for the development of hypertension in the ensuing years and the patient should be encouraged to develop other leisure interests.

- 1 Wilmhurst PT, Nuri M, Crowther A, Webb-Peploe MM. Cold-induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. *Lancet* 1989;i:62-5.
- 2 Edmonds C, Lowry C, Pennefather J. *Diving and subaquatic medicine*, 3rd ed. London: Butterworth Heinemann, 1994.
- 3 Bennett PB, Elliott DH. *The physiology of medicine and diving*, 4th ed. Philadelphia: W B Saunders, 1993.

Silent myocardial infarction during hypoglycaemic coma

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Abstract

A case of silent myocardial infarction associated with hypoglycaemic coma is described. A hypoglycaemic episode represents a risk factor for a patient with underlying ischaemic heart disease. A routine ECG may be indicated in such circumstances.

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Key terms: hypoglycaemia; diabetes mellitus; myocardial infarction

Case report

A 75 year old woman with ischaemic heart disease and non-insulin-dependent diabetes mellitus controlled by metformin presented to the emergency department following a period of unconsciousness at home. The patient had been found by her daughter and was described as being cold, clammy, and unrousable. An ambulance was called, and on arrival at the scene the crew made a provisional diagnosis of hypoglycaemia and put jam and sugar into the unconscious patient's mouth. Within minutes she had regained consciousness and was talking to her rescuers.

On arrival at the emergency department the patient was fully conscious and alert, with no history of chest pain either before or after her period of unconsciousness. Clinical observations were normal and a Glucostix test gave a reading of 16.4 mmol/litre. A routine ECG was requested by the nursing staff and this revealed evidence of an acute inferior myocardial infarction, with 5 mm of ST elevation in leads II, III, and aVF and T wave inversion in III and aVF. The patient was immediately transferred

to the resuscitation area and the medical staff informed.

Clinical examination revealed a raised jugular venous pressure, mild ankle oedema, and severe diabetic retinopathy, but was otherwise unremarkable. Formal laboratory blood glucose estimation confirmed the reading of 16.4 mmol/litre.

A diagnosis of silent myocardial infarction was made and the patient was admitted to an acute medical ward. Serial ECGs and serum aspartate transaminase and creatine kinase estimations over the next 72 hours confirmed the diagnosis. She had an uneventful hospital stay and was discharged home seven days later.

Discussion

It is recognised that hypoglycaemia increases damage to the ischaemic myocardium.¹ However, few cases of myocardial infarction associated with hypoglycaemia have previously been reported.^{2,3} Others have described ischaemic ECG changes during severe hypoglycaemia which resolved completely after treatment with intravenous glucose.⁴ The development of hypoglycaemia was not preceded by chest pain in any of these cases. It has been shown that autonomic neuropathy in diabetes mellitus leads to disturbed cardiac perception and thus may play a role in silent myocardial infarction.⁵

Myocardial infarction normally tends to increase the blood sugar because of a decrease in insulin sensitivity⁶; therefore, had it been the primary event in our patient, symptomatic hypoglycaemia would have been less likely. Although we have no biochemical proof that

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