

Immersion pulmonary oedema

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Key words

Pulmonary oedema, pulmonary edema, diving, scuba, immersion, cold, beta blockade

Abstract

Pulmonary oedema of immersion is a rare complication of scuba diving. A case and brief discussion of the differential diagnosis and pathophysiology are presented. Onset of symptoms typically occurs during the dive, and is characterized by dyspnoea, cough and frothy sputum. Spontaneous recovery frequently occurs once the diver leaves the water. If investigated, the findings are of hypoxaemia and chest X-ray changes typical of pulmonary oedema. The differential diagnosis includes decompression illness, pulmonary barotrauma, near drowning, salt water aspiration syndrome and non-diving causes of pulmonary oedema. The condition may be precipitated by the increase in pulmonary capillary hydrostatic pressure that occurs on immersion, coupled with the imposition of a pressure gradient between mouth pressures and hydrostatic pressure at the chest in the upright diver, and the imposition of more negative intra-alveolar pressures during breathing of a denser gas or from a poorly tuned regulator. Divers with heart disease, hypertension, or taking negative inotropic agents may be at greater risk.

Introduction

Diving physicians are sometimes presented with patients complaining of isolated respiratory symptoms and signs arising during or immediately after diving. A diagnosis that is frequently overlooked in this setting is immersion pulmonary oedema. It is important to consider this problem because recompression is not indicated in its treatment. Recognition may prevent a costly and unnecessary evacuation to a hyperbaric facility. Immersion pulmonary oedema is a rare condition but case reports and series have steadily appeared over the past 10 years. The pathophysiology is poorly understood. Dr Joanne Grindlay and I recently reviewed the problem, and this presentation is a synopsis of the important points made in that paper.¹

Case report¹

A 31-year-old female medical practitioner who had no relevant past history and was on no medications was undertaking the second pool session of a PADI open water diving course. The first pool session had been conducted two nights previously and she had experienced no problems. The pool session in question involved a number of skills including taking the regulator in and out of her mouth underwater, and she experienced no problems with any of the skills being practised. The session lasted 90 minutes and was conducted in a chlorinated pool, four metres in depth, water temperature 20°C. She was wearing reasonable thermal protection, a two-piece 5 mm wetsuit with a hood but no gloves.

Immediately after the end of the session, which involved quite a few ascents and descents throughout the evening, she noticed that she was short of breath. She developed a wheezy cough productive of some watery sputum which

was slightly pink and she became cyanotic. This observation was made by medical colleagues who were doing the diving course with her. She self-presented at an emergency department with progressive shortness of breath. On admission, respiratory rate was 20 breaths per minute with widespread crepitations on auscultation. Oxygen saturation was 88% on room air. Blood gases were recorded while she was on oxygen with an inspiratory fraction of 50%. P_aO_2 was 63 mmHg, oxygen saturation 92%, and P_aCO_2 and acid-base state normal. Chest X-ray showed an interstitial pulmonary oedema pattern.

She was treated with 30 minutes continuous positive airway pressure by mask and given 20 mg frusemide intravenously. She made a very rapid symptomatic recovery and was discharged. For about two weeks afterwards she felt slightly dyspnoeic on exertion but at rest she was asymptomatic.

A number of investigations were carried out. Her dive equipment was examined and was found to be in working order. An electrocardiogram and echocardiogram were normal with no evidence of a patent foramen ovale or valvular heart disease. A stress test, histamine and hypertonic saline challenge tests, and finally a high resolution inspiratory and expiratory thoracic helical CT scan, were all normal. She returned to diving, completing her course and at last follow up had done about 50 dives in a wide range of conditions without incident.

Discussion

The main feature of this case was acute pulmonary oedema with no other symptoms and no latent period after diving. The initial diagnosis made at the time of presentation in the emergency department was decompression illness (DCI), notwithstanding such a non-provocative dive profile.

DCI due to venous bubble formation from dissolved nitrogen may cause dyspnoea and possibly pulmonary oedema, but to the author's knowledge such manifestations are unreported after such unprovocative diving. Moreover, pulmonary manifestations of DCI are usually accompanied or followed by other, more specific, neurological and musculoskeletal symptoms.² Therefore, the diagnosis of DCI in this case seems implausible.

Pulmonary barotrauma may certainly occur in a swimming pool situation, but is not known to present as pulmonary oedema. More typically, pulmonary barotrauma presents either with neurological or cardiac symptoms and signs from cerebral arterial gas embolism, or with evidence of pneumothorax and/or mediastinal and subcutaneous emphysema. None of these were present.

Near drowning produces pulmonary oedema of short latency. However, clinically apparent near drowning requires a history of aspiration, which is usually brought on by panic, a malfunctioning regulator, or something else. There were no such events in this case and it seems unlikely that a sufficient occult aspiration of water could have occurred without this informed diver noticing.

Salt water aspiration syndrome is a condition attributed to aspiration of small amounts of water through a diving regulator.³ The classical symptoms and signs of cough, fever, constitutional malaise and sometimes patchy consolidation on chest X-ray (but not of a pulmonary oedema pattern) were not present in this woman. Moreover, this syndrome is reported as having a long latent period, and does not typically onset during the dive as is often the case in immersion pulmonary oedema.

Exercise-induced pulmonary oedema is an obscure disorder of uncertain relevance to humans, but it may have contributed to several cases reported recently in fin swimmers exercising hard for long periods.⁴ Pulmonary capillary fragility is increased by catecholamine release during exercise in other species, eg. horses, but it is not commonly seen in humans. This patient was not exercising vigorously.

Non-diving causes of pulmonary oedema or of symptoms suggestive of pulmonary oedema include asthma, myocardial infarction, trauma, allergy, anaphylaxis and exposures to toxic gas. None of these conditions appeared relevant to this case.

In the absence of other adequate explanations, it was presumed that this woman had suffered immersion-induced pulmonary oedema. This condition is probably a form of acute onset left ventricular failure, but its pathophysiology is not definitively described. There are probably several important components. Firstly, it is known that immersion itself, and the peripheral vasoconstriction associated with either immersion or exposure to cold, both cause a

simultaneous increase in cardiac pre-load and after-load as blood volume is centralised and peripheral resistance increases. Not surprisingly, there is an increase in mean pulmonary artery pressure, and therefore pulmonary capillary hydrostatic pressure.

Secondly, immersion of a diver in an upright position results in the basal lung tissue being exposed to a hydrostatic pressure approximately 15–20 cm greater than that of the airway pressure at the mouth (which equilibrates with alveolar pressure during breathing). This differential is volume-compensated by engorgement of the pulmonary blood vessels, which may predispose to capillary stress failure.

Thirdly, it seems likely that the negative intra-alveolar pressures generated during inspiration due to airways resistance are greater during diving because turbulent flow is more likely when breathing a denser gas. Finally, and in relation to the latter point, if the diver is breathing from a poorly tuned regulator that requires increased negative pressure to activate gas flow, this will also enhance negative intra-alveolar pressure.

It is plausible that this combination of increased pulmonary capillary hydrostatic pressure, pulmonary vessel distension, and exaggerated negative cyclical intra-alveolar pressure promotes the transudation of fluid through the capillary walls into the alveoli. This sequence of events would be more likely in a 'predisposed' person whose myocardial response to increased load is impaired. Interestingly, most cases seen by the author recovered spontaneously over a period of hours once the diver was removed from the water. The case described here was one of the few that received active treatment.

A number of contributory or risk factors have been proposed for this condition. The earliest descriptions suggested that this was a cold immersion induced phenomenon.⁵ Cold water immersion produces a greater degree of vasoconstriction and a consequent increase in pre-load and after-load that has been confirmed experimentally. However, other reported cases and our own unpublished experience indicate that extreme cold is clearly not a prerequisite for pulmonary oedema of immersion.⁶

Hypertension and coronary artery disease have been reported in some cases, but in others there have been no identifiable predisposing factors whatsoever, as in the present case. All of the 11 cases reported by Wilmshurst et al (1982) had an abnormal peripheral resistance with a high resting forearm vascular resistance and an exaggerated vascular response to cold exposure.⁷ However, none of the three cases reported by Pons exhibited these features.⁸

Another potential contributory factor is beta blockade or medication with negative inotropic agents. Of six cases seen by the author, three were on beta blockers.¹ Hampson and

Dunford reported six patients, one of whom was on beta blockers and one on a calcium-channel blocker.⁶ Medication with these agents might simply be an epi-phenomenon, reflecting the presence of hypertension or other cardiovascular disease. Nevertheless, it makes sense that a negative inotrope might predispose a swimmer or diver to cardiac decompensation in the presence of a sudden increase in pre-load and after-load.

Over-hydration has been reported as a potential risk factor.⁴ Eight subjects in a strenuous military swim developed pulmonary oedema after drinking five litres of water prior to the swim to avoid dehydration. The heavy exercise in this group was proposed as another risk factor (see above).

What advice should be given to someone about diving after an episode like this? The simplest advice would be to never dive again, as one could not guarantee this would not happen again even if no predisposing factors could be identified. This may become a risk/benefit decision for the diver to make after being counselled about the issues. Certainly, if investigation identified an obvious risk factor advice not to dive is clearly indicated. Appropriate counselling would include advice to wear good thermal protection, including a dry suit, boots, gloves and hood in cooler waters, avoidance of over-hydration and overexertion, and use of a well tuned regulator. Other advice would include diving from a platform where oxygen is immediately available at the surface, and not at some remote location.

Summary

The syndrome of sudden onset pulmonary oedema may occur in divers of all ages and levels of fitness but may be more likely in older divers, those with cardiac disease or hypertension, and those using negative inotropic agents or who are over-hydrated. With respect to the dive, it can occur in all temperatures of water but the risk is probably higher in colder water and with vigorous exertion. Onset of pulmonary oedema may occur any time during or soon after the dive. It does not appear to be a phenomenon associated with decompression, since almost all cases develop symptoms during the period at depth. It is characterised by dyspnoea and cough productive of white or pink frothy sputum. Rapid spontaneous recovery occurs after leaving the water in many cases. It appears that divers may return safely to diving after such an event but this should only occur after careful assessment for predisposing factors and appropriate counselling. This is a rare phenomenon whose pathophysiology is not clearly established.

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Audience participation

Davis, New Zealand: The incidence of secondary drowning has been raised. The figures quoted by Dr North were 5% from John Pearn's work in Australia on children,¹ and 25% from Modell's work in Florida.² It may be anywhere between these. There are only three useful definitions in the drowning literature as far as I am concerned; drowning which is death by immersion, near drowning which is survival from an immersion incident and secondary drowning which is a syndrome of pulmonary oedema as described here by Dr Mitchell. This latter definition has now gone out of favour, but secondary drowning may occur after a variable latent period of up to 24 hours following a near-drowning episode. It does not appear to be dependent on the severity of the initiating event. In other words it can be a life-threatening complication following a minor aspiration incident. I suspect that is what the case described suffered from. Pulmonary oedema following an immersion incident usually responds extremely rapidly and effectively to continuous positive airway pressure or to mechanical ventilation combined with positive end expiratory pressure.

On another subject, comment was made on buddy pairs dying. In the New Zealand series, there were three multiple drowning incidents, two buddy pairs and three out of a group of seven divers – the so called French Pass Incident.³ SPUMS will hear more about this accident once the legal processes are complete, which could take several years. In this tragic diving accident, seven scuba divers were sucked to a depth of over 90 meters by strong currents. Amazingly, four of them survived, but three did not.

Mitchell, Brisbane: Apropos your comment about my misdiagnosis, I just wonder if you have ever seen a case of secondary drowning with florid symptoms that occurred in a victim who had absolutely no awareness of aspirating anything. (**Davis:** No) Secondly, I am not aware of any case of secondary drowning that occurred with no latency at all. Of course the aspiration could have occurred early in the evening and there would have been a latent period but there was no awareness of any aspiration. Therefore, I do not agree with you.

Bennett, Sydney: You mentioned the question of returning to diving and the fact that at least some of the people you know returned to diving with no more events. Have you actually treated someone multiple times for emergent pulmonary oedema?

Mitchell: No I have not. None of the cases that I am aware of that went back to diving have suffered it again. However, two of them who had a single event, recovered and went diving again before they saw me, and developed the same problem again within days of the first episode. Clearly, they had not fully recovered and something was still predisposing them to the second event. No one who has had it, seen me, heard my advice, had some time off and then gone diving again, has had another event to my knowledge.

Bennett: My other question is, would you comment on the reason for being given 50% oxygen on arrival at the emergency department. When were you involved and what would your advice have been?

Mitchell: I was not involved acutely. I was contacted after one of the consultants suggested she might need recompression therapy, which I did not believe she needed. She had an adequate arterial O₂ on 50%, but I do not know why they chose that. If I had been managing her in the first aid setting, I would have put her on 100% oxygen but whether that would have been necessary I am not sure. Have we any emergency physicians here who might be able to cast any light on it? (No audience response.)

Brogan, Perth: It does not matter what you do with your gear, you can still run into trouble. I had mine serviced about three weeks before I came. I did about 10 dives before I left Australia. On the third dive here I had a first stage problem, total loss of air at 40 feet, and had to take off for the surface. So you can always be unlucky!

Bennett: A number of speakers have made the comment that they cannot understand how with modern dive training methods there could possibly be an out-of-air situation. Let me tell you a true story from about a week ago. This involved an experienced dive buddy pair, both of them in excess of 600 dives, and buddied up for most of that time. One of the divers had a progressive leak in the high pressure hose which became more and more obvious until one of the crew heard it, and drew her attention to it. The decision was made by the diver to just remove that hose and block the plug so now she had no contents gauge. About three dives later, the other diver, the one who actually had a contents gauge, noticed early in the dive it was a little difficult to breathe. Looking at his contents gauge for the first time, it read zero, so he swam over to his buddy who did not have a contents gauge and pointed to his contents gauge and indicated it was a bit difficult for him to obtain air. The buddy's response was to tap the gauge and indicate it must be broken.

They then took off further down the reef, and at about 20 metres it was really very difficult for him to breathe. So he indicated to his buddy that he needed to breathe on her octopus. They successfully managed this manoeuvre without any problems, except, just as he was getting himself settled, she saw something of great interest lower down the reef and took off like a rocket, leaving him with only the rubber mouthpiece in his mouth. Now, he said, he was slightly concerned because he was not sure just how far away she was going. He managed to catch up with her and put what was left of the octopus in his mouth, in the process of which he dropped the mouthpiece he was holding. She then saw this and took off after it. By now she was at about 35 metres. He recovered from that, followed her down, and breathed on the octopus without the aid of a mouthpiece.

They then made their way up to about 20 meters and completed a pleasant 40 minute dive, both of them breathing from a tank with no contents gauge, having already realized that at least one of their tanks had not been filled prior to the dive. The most amazing thing about this story is their reaction when we all got back on the boat, which was to wonder why anybody thought this was a difficult problem because they felt they had dealt with it very well; it was a perfectly safe dive as far as they were concerned!

Thomas, Sydney: Dr Mitchell, I presume the cases you describe had normal electrolytes and renal function? Has anyone looked at renal function acutely in these patients and whether they produce normal amounts of urine, or whether they perhaps are concentrating? I would assume that if they have normal electrolytes then that does tend to make the diagnosis of either neardrowning or salt water aspiration unlikely.

Mitchell: Electrolytes were done on the case I presented and they were normal, but nothing more formal in terms of renal function tests was done. I would have to go back to

the literature to tell you whether or not they were done on some of the other reported cases. In most of the cases that I have they were not done. Many of them I saw in the context of their seeking an opinion on what happened and what they should do about it in future, so I was not involved in the acute management. There was one whom we admitted, who had a full blood count and renal function tests, which were normal, but I could not tell you about the other cases. As to whether or not all salt water aspirations result in abnormal renal function tests I am not sure about that. I was of the opinion that they did not necessarily.

Haller, Melbourne: With all these people running out of air, I was just wondering whether anyone has done any trials or whether anyone would know the physiology of how long someone would last using the air out of their mask? If you run out of air, could you breathe in and out of your nose to get to the surface using the air in your mask as a form of emergency air source?

Bennett: There is not enough volume in it to get past your dead space is one answer, I think. But it would help you feel more comfortable on the ascent essentially. Not a bad idea.

Jones, New Zealand: There was a technique widely touted 25 years ago of breathing from your buoyancy compensator (BC). But this was killed by the description of Key West scuba divers disease, which was *Legionella basmaneei*. It was recommended that you flush out your BC between dives with hypochlorite to sterilise it, just in case you needed to breathe from it again.

Bradley, Central Coast, NSW: Just to comment on safety. Often on these conferences we place reliance on our dive masters who in some cases may have a lot less experience than us, which comes back to having a good dive plan regardless of what dive masters are saying to us. A case in point occurred in Layang Layang two years ago, where the directions of the dive master were taken, when in fact it was quite an unsafe practice that was being proposed. In this case, the current picked up during the dive to a considerable rate. Against the strong current, the dive master was trying to home everyone back into a particular spot that we had dived several times before, whereas the safest practice was to go with the current and do our deco in mid-water, away from the reef. Half the team actually did that, while the other half struggled valiantly to stay on the reef. We all ended up doing mid-water deco.

Knight, Melbourne: One thing to remember is that as you rise in the water, the pressure differential between your tank and the second stage rises and you can get some air out of the tank. Divers should keep their regulators in their mouth if they run out of air and try to breath in and out on the ascent. If they did not have enough air to inflate their compensator, dropping the weight belt would make them buoyant. Encouraging this technique markedly reduced the

incidence of injury and death from rapid ascents in one of the Canadian National Parks some decades ago.

Bennett: How many of you have run stone motherless cold out of air whilst diving? (Several members of the audience raise their hands.) So there are a few people amongst this group. None of you are diving tomorrow!

Carney, New Zealand: Dr Mitchell, are the people with pulmonary oedema that you have treated all similar in type or are they men and women of all ages?

Mitchell: That is one of the surprising things. It seems to be able to affect anybody but typically they're older, perhaps less fit, possibly with some sort of cardiac problem like hypertension or another predisposition. It is probably safest to just see it as something that can happen to anybody.

Robyn Walker, Sydney: We have had one clearance diver candidate on a four-hour surface swim to Manly and back, done in winter fully dressed in appropriate wetsuit, gloves and hood, pulled from the water halfway back in frank pulmonary oedema. Similar cases have been reported in swimmers as you described.⁴ He was given oxygen and frusemide and made an uneventful recovery overnight. Intensive cardio-respiratory work up was normal and he was permitted to continue diving. On a repeat swim, he went into pulmonary oedema for the second time. He was superbly fit in all other respects and we found no other abnormality with him, but the Navy could not risk him every time he did a long surface swim going into pulmonary oedema.

Bennett: He presumably did the same level of exercise but in different settings and he was okay then?

Walker: Yes. Navy divers have a high physical activity load, and in part of his acceptance test he would have done a two-week course where we estimate they need about 10,000 calories a day just to get them through the level of physical activity that they do on that course. So he was superbly fit in other respects and, in fact, had no problem diving in water of the same temperature as on the long surface swim.

Davis: This is an interesting phenomenon. Because of my long-distance sea swimming activities when I was younger, I took a special interest in the physiological work on English Channel swimmers who spend many hours swimming in cold water.⁵ I do not recall from that literature this phenomenon being reported at all, and this puzzles me. Dr Mitchell, do you remember whether in the Israeli swimmers paper, they were using fins, mask and snorkel or was it just ordinary swimming? In other words, were they like Dr Walker's case, or not? I wonder whether this is an issue of using swimming aids and protective suits that creates the difference.

Mitchell: I think they were using swimming aids, and were diver trainees like Dr Walker's diver.

Davis: It would be worth reviewing the old long-distance swimming physiology work to see whether it is documented anywhere. It is the use of swimming aids in one form or another that perhaps creates a different respiratory and cardiovascular workload which someone swimming without aids in cold water for long periods does not experience. Another interesting aspect is that many of these swimmers were not youngsters; Gerry Forsberg broke the two-way English Channel record in his late forties, for instance.

von Neullen, Holland: Dr Mitchell, could a contaminated air source be a cause of pulmonary oedema, and have you ever seen such cases?

Mitchell: You are absolutely right. If you inhale irritants or noxious substances you can definitely suffer pulmonary oedema. The air in this patient's cylinder was tested and no contaminants were found. So it was not the problem in her case. I personally have not seen any diver suffer pulmonary oedema because of contaminants in the breathing mix but I believe such cases have been reported in the literature.

Thomas: Do you know if they routinely test for oxides of nitrogen in the air? (**Mitchell:** I do not think so.) These are described as a cause of pulmonary oedema. Her air was probably tested for carbon monoxide and hydrocarbons as it was a diving situation. Presumably though, you would expect a large number of people to be affected if the system was contaminated.

Deborah Yates, Sydney: It is highly unlikely that they would have been able to test for oxides of nitrogen because it is difficult to get hold of the equipment. We have recently seen some cases in blast furnace workers, but we had considerable difficulty measuring levels. Quite variable levels were seen (paper in preparation).

Thomas: If you measure the levels of nitric oxide in hospital compressed air they vary with the urban pollution. We have recorded up to about 300 parts per billion in our hospital air. This is probably not important, but you do not know what else is being compressed out of the atmosphere to give to the patients. Oxides of nitrogen have been described as being present in ventilator circuits in intensive care units.

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