

Chapter 44

Scuba Divers' Pulmonary Oedema

Scuba Divers Pulmonary Oedema (SDPE) was previously thought to be an uncommon disorder. It can occur in apparently healthy individuals, but is sometimes based on cardio-vascular pathology. In a survey of scuba divers about 1% described it. An individual predisposition is a likely factor since recurrences are common with diving, snorkeling or swimming.

Symptoms

It presents clinically with difficulty in breathing - with fast shallow respirations and a sensation of tightness, wheezing or crackling sounds in the chest. Symptoms are often aggravated during ascent or if the diver/swimmer remains immersed, but are relieved if the victim is removed from the water before the condition becomes too severe.

It may be associated with fatigue, cough, whitish or sometimes blood-stained expectoration, and possibly a bluish tinge to the lips, tongue and face (cyanosis).

Symptoms usually resolve rapidly (some hours) after the immersion, but deaths have been reported and it may be indistinguishable from drowning, at autopsy.

Clinical Signs

Hypoxia may be demonstrated by the cyanosis. Weakness, confusion or impaired consciousness may occur. Paramedics may detect signs of pulmonary oedema by listening to the chest. Later a mild temperature may develop.

Clinicians may demonstrate reduced spirometry and compliance, hypoxaemia and characteristic radiological (plain x-ray or CT scan) abnormalities. These usually resolve rapidly (hours) in most cases.

General

SDPE is more frequently seen in older divers, probably more common in females and tends to recur, either whilst diving or snorkeling. Most are in the 30-60 year age group and there may be an association with hypertension, ischaemic or other heart diseases or impaired respiratory function.

The actual incidence is unknown, but very likely it is under diagnosed. Both clinically and pathologically, the appearances are similar to salt water aspiration, near-drowning and drowning (Chapters 25 and 26).

Extreme exertion may be observed in some cases, but it is often specifically denied.

Predisposition

An individual predisposition for pulmonary oedema is likely since a diver, snorkeler or swimmer with pulmonary oedema may have other episodes of SDPE, previously or subsequently (in at least 30% of cases). Whether the recurrences relate to the individual diver's medical status, the dive profile, environmental conditions or the dive equipment, is conjectural. We do not know why most cases occur or recur.

Causes

Many causes have been incriminated. The common factor is a damage to the pulmonary capillaries, with leakage of fluid from the pulmonary capillaries into the lung alveoli ("drowning from within"). This may be more likely if more than one "stress" is put on these capillaries.

The stresses may include;

Pre-existing cardiac disease (possibly not known to the diver)

High blood pressure,

Cold exposure, inducing hypertension,

Salt water aspiration. See Chapter 26.

Intrathoracic blood pooling induced when the body is submerged

Negative pressure during inspiration, which could occur from:

- Immersion per se, especially with a head-up/vertical or head-out position
- Inspiratory breathing resistance from diving equipment (regulator, snorkel)
- Reduced gas supply/pressure (low on air)
- Excessive gas density with depth
- Increased ventilation, as occurs with anxiety and hyperventilation
- In rebreathing equipment, when the counter-lung is positioned above the lung
- Tight chest clothing (wet suits)

Drugs, such as beta-blockers

Treatment

Rescue the patient from the water. Administer oxygen and rest. Positive pressure respiration may be needed in severe cases. Although improvement is relatively rapid after leaving the water, cases of unconsciousness have been well recorded, as have deaths. Deaths are likely to be attributed to drowning, like so many other deaths in the underwater environment.

Medical assessment is required to verify the **diagnosis** and exclude any predisposing features. Although SDPE may develop in divers with no medical problem, **often** it is based on other diseases, such as cardiac or respiratory diseases. Thus, once it has happened, it tends to recur. Investigations to exclude such predisposing factors need to be undertaken. Sometimes the cardiac diseases are structural (ischaemia),

sometimes mainly physiological (arrhythmias) and often due to transitory abnormalities (reversible myocardial dysfunction, takotsubo etc.). Thus SDPE, especially in older divers, should be an indication for comprehensive cardiac investigation, not only for possible therapy but also to avoid further SDPE episodes.

It seems reasonable that unless the cause can be identified, verified and corrected, divers with SDPE should be advised of the possible risks of continuing with the activity which provoked it, and should be advised against further diving, snorkeling or energetic swimming.

Differential Diagnosis

Other diseases that can produce pulmonary oedema and cause diagnostic confusion are the salt water aspiration syndrome, drowning, respiratory oxygen toxicity, gas contaminations, cold urticaria, the Irukandji syndrome (jellyfish envenomation) and diving induced asthma. Pulmonary decompression sickness, pulmonary barotrauma and the so-called 'deep diving dyspnoea' are diving disorders that may cause diagnostic confusion with SDPE. Anxiety produced hyperventilation may also cause some diagnostic confusion, but this has none of the other respiratory manifestations.

Immersion Pulmonary Oedema (IPE)

There are three forms of acute pulmonary oedema associated with immersion. It may be induced by swimming/snorkeling, free diving ("lung squeeze" at end of Chapter 11) or scuba diving. They have some features in common, but there are significant differences in their demographics, causation and therapeutic implications.

The swimming induced cases tend to be young and fit, but exposed to excessive exertion. Most of the swimmers affected were otherwise healthy. In special forces combat swimmers, extreme exertion was incriminated. It was observed in both cold and warm waters, sometimes over 20⁰C. Over-hydration may have contributed to some of these

Explanations for IPE include; increased cardiac output due to physical exertion, pulmonary vascular blood pooling due to immersion, increase in pulmonary vascular resistance due to cold exposure, hydrostatic pressure effects and increased perfusion in the dependent lung with side-stroke swimming.

DIVING MEDICAL ADAGE

If a diving accident occurs and you are not sure of the cause, and do not take precautionary action, it will re-occur but with more serious consequences