Air embolism with bilateral pneumothorax after a five-meter dive

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Friehs I, Friehs GM, Friehs GB. Air embolism with bilateral pneumothorax after a five-meter dive. Undersea & Hyperbaric Med 1993; 20(2):155–157.—After an emergency ascent from very shallow depth, a diver suffered a triad of symptoms after bilateral barotrauma of the lungs: air embolism with subsequent paraparesis, pneumomediastinum, and bilateral pneumothorax. This is the first case of its kind in 20 yr of experience in the hyperbaric center at Graz. The patient was successfully treated by recompression with HBO.

barotrauma, pneumothorax, pneumomediastinum, air embolism, diving accident

The two main causes for diving accidents while using scuba devices are DCS (caisson disease) and barotrauma of the lung with consequent air embolism (1). Inexperienced divers sometimes fail to comply with recommended ascent rates, especially in emergency situations. With breath holding or air trapping, the volume of air in the lungs inhaled at depth expands on ascent in direct relation to the decrease of the surrounding pressure (Boyle-Mariotte’s law). When the lung’s elasticity of approximately 100 mbar is exceeded, the pulmonary parenchyma is damaged, sometimes resulting in pneumomediastinum and pneumothorax. Air bubbles entering the pulmonary circulation are transported into the arterial blood stream and cause neurologic deficits of differing degrees. Symptoms may occur as soon as the diver reaches the surface (1).

CASE REPORT

A 24-yr-old male, admitted to our hospital after a diving accident, reported that at a depth of only 5 m he had planned and undertaken an emergency ascent. At a depth of 1 m he held his breath rather than exhale. On reaching the water surface the patient complained of sudden onset of dizziness, paraparesis of the lower extremities, and shortness of breath. On auscultation, a left side pneumothorax was noted. An x-ray taken in the local hospital confirmed the suspected diagnosis and revealed
an extensive mediastinal emphysema. Chest drainage was instituted immediately, and the patient was transported to our hospital by helicopter, flying no higher than 850 m above sea level while breathing 100% oxygen. A subsequent x-ray in the emergency room showed that the left lung was fully expanded, but an incomplete pneumothorax was diagnosed on the right.

Recompression was started at once in the large hyperbaric chamber; the left side pneumothorax was kept drained under hyperbaric conditions while the right side, incomplete pneumothorax was monitored closely by the attending physician, especially during decompression, and further treated by self-absorption. However, surgical instruments were prepared in case a chest tube on the right side was needed. The patient was kept at 3 bar breathing 100% oxygen for 62 min, according to the recompression tables by Goodman and Workman (2). After the HBO, an x-ray showed a totally expanded left lung, the right side pneumothorax at the same minimum size, and an almost vanished mediastinal emphysema. The patient no longer suffered from paraparesis in the lower extremities or dizziness.

The chest tube was removed on Day 8 after admission, and the patient was discharged on Day 11 without residual neurologic symptoms or any other discomfort. On routine examinations 3 wk and 3 mo. later, two ventilation–perfusion scintigrams were taken which depicted no pathologic findings; therefore, the ventilation defects known as trapped gas phenomenon (3) could not be demonstrated.

**DISCUSSION**

As opposed to bends (caisson disease), lung barotrauma associated with diving is not dependent on diving depth or time (1). Barotrauma can occur even at a depth of 1–2 m. The main reason for this type of accident is panic emergency ascent by young, inexperienced divers. Divers should ascend to the water surface by continuously exhaling to compensate for gas expansion in the lungs. An intrapulmonary airway pressure that exceeds the pressure produced by the lung’s elasticity results in extraalveolar air leakage. The perivascular sheaths then mediate the air to the mediastinum, to the subcutaneous tissues, and to the intrapleural space (4). This explains the occurrence of mediastinal and subcutaneous emphysema (5). The rupture of lung parenchyma may also allow gas to enter small blood vessels, and anterograde migration of bubbles in pulmonary venules may then lead to arterial emboli formation (6). Possible platelet aggregation and other coagulation disorders induced by bubble surface and plasma interaction, followed by vessel occlusion and reduction of perfusion (7), lead to hypoxic damage of the organs supplied by these vessels. Resulting neurologic deficits range from hypesthesia to decerebrate symptoms, to death in worst cases.

Pneumothorax due to barotrauma of the lung after a diving accident is rare. Saywell (8) reports a case of a young diver with recurrent pneumothorax after diving to 9 m. Increased intrapulmonary pressure caused by artificial respiration can also result in barotrauma and pneumothorax; such cases are frequently reported in the literature (5, 9, 10).

The appropriate treatment for air embolism of the CNS with neurologic deficits combined with mediastinal and subcutaneous emphysema after a diving accident is recompression with HBO. Radiologically diagnosed pneumothorax, even when
bilateral, does not contraindicate HBO therapy, as our case clearly shows, as long as a large hyperbaric facility and medical attendance during recompression are provided. This agrees with Daugherty's (11) opinion. There is only one limiting factor for prognosis: the longer the time between the accident and the HBO, the less the chance for complete recovery without residual symptoms.

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REFERENCES
