A Free Diver with Hemoptysis and Chest Pain

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Case Reports from the Alpert Medical School of Brown University Residency in Emergency Medicine

DR. CHANA RICH: Today’s patient is a 24-year-old man who presented to the Emergency Department (ED) with a cough and hemoptysis. The patient was freediving (breath-hold diving) in order to spearfish and had submerged to 50 feet while using an 11-pound belt. The patient ascended without expiration and developed chest pain. Upon surfacing, he coughed up approximately 5 tablespoons of bright red blood. After a brief rest on the boat, he dived to a depth of 30 feet in order to catch a large striped. Upon surfacing the second time, the patient had pleuritic chest pain and mild shortness of breath.

Due to his symptoms, the patient presented to the ED. He did not complain of headache, visual changes, ear pain, nausea, joint or muscle pain, and he had no additional episodes of hemoptysis. Vital signs revealed BP 127/68 mm Hg, heart rate 53, respiratory rate 16, and the patient had an oxygen saturation of 98% on room air. On exam, he was comfortable and non-toxic appearing and was in no distress. His lungs were clear to auscultation bilaterally; his cardiac exam revealed a normal s1s2 and he had a regular rate and rhythm with no murmurs. There was no crepitus. The remainder of his physical exam was unremarkable.

DR. WILLIAM BINDER: Free-dive spearfishing at 50 feet must cause significant pressure changes as our bodies are subject to almost 3 atmospheres of pressure. What disorders occur at increased atmospheric pressure?

DR. RICH: Spearfishing and other underwater activities may be done while freediving, snorkeling, or scuba diving. Our patient and his friends were freediving, or breath-hold diving. Unlike scuba diving, breath-hold divers do not use supplemental air underwater. Divers face a unique set of underwater hazards in addition to the general aquatic problems such as drowning, hypothermia, water-borne infectious diseases, and interactions with hazardous marine life. When diving deep, free divers are exposed to increased pressure, causing a spectrum of injuries to the body.

Pressure contributes either directly or indirectly to the majority of serious diving-related medical problems. As a diver descends underwater, absolute pressure increases much faster than in air. The pressure change with increasing depth is linear, although the greatest relative change in pressure occurs nearest the surface, where it doubles in the first 33 feet of sea water from 1 to 2 atmospheres of pressure. The body behaves as a liquid and follows Pascal’s law; pressure applied to any part of a fluid is transmitted equally throughout the fluid. When a diver submerges, the force of the tremendous weight of the water above is exerted over the entire body. The body is relatively unaware of this change in pressure.

While this is true of the body, the spaces within the body containing gas [air], including the lungs, sinuses, intestines, and middle ear, follow a different law. The gases in these spaces obey Boyle’s law; the pressure of a given quantity of gas at constant temperature varies inversely with its volume. Therefore, as one dives deeper, the volume of air in the middle ear, sinuses, lungs, and gastrointestinal tract is reduced, but upon ascent, the volume expands. Inability to maintain gas pressure in these body spaces equal to the surrounding water pressure leads to barotrauma.

Barotrauma can potentially involve any area with entrapment of gas in a closed space. In addition to sinuses, lungs and the GI tract, barotrauma can occur in the external auditory canal, in the teeth, the portion of the face under a face mask, and skin trapped under a wrinkle in a dry suit. The tissue damage resulting from such pressure imbalance is commonly referred to as a “squeeze.”

Illustration 1. Pascal’s Law: pressure applied to any part of a fluid is transmitted equally throughout the fluid.
Source: https://upload.wikimedia.org/wikipedia/commons/thumb/2/27/Pascals-law.svg/2000px-Pascals-law.svg.png
DR. ELIZABETH SUTTON: How does this account for the patient’s hemoptysis?

DR. RICH: There are two possibilities accounting for the patient’s chest pain and hemoptysis. Pressure-related injury to the lung can occur on descent, or as a diver ascends to the surface. If one were able to completely exhale, the absolute minimum lung volume remaining is called the residual volume (RV). Lung squeeze occurs when the diver descends to a depth at which the total lung volume is reduced to less than the residual volume. The increased pressure on the pulmonary vascular bed can damage the integrity of the pulmonary capillaries. At this point, transpulmonic pressure exceeds intraalveolar pressure, leading to transudation of fluid or blood from ruptured pulmonary capillaries. [1] Patients can exhibit signs of pulmonary edema and hypoxemia. This is the reason that many free divers cough up blood after a deep dive, although there are a number of case reports of lung squeeze with repetitive shallow dives with brief surface intervals. [2]

Despite this presumed mechanism of barotrauma of descent, free divers are able to dive to depths beyond those that should cause mechanical damage to the lungs. Other physiologic mechanisms must play a role, although the exact pathophysiology of this condition remains unclear. When diving deep, the chest cavity itself gets smaller and there is central pooling of blood in the chest from the surrounding tissues. The central pooling of blood in the chest equalizes the pressure gradient when the RV is reached and thereby decreases the effective RV. These mechanisms allow the lungs to be compressed down to about 5% of total lung capacity in highly trained breath-hold champions. [3] An individual’s anatomy, physiologic reserves, underlying pathology and the conditions of the day all play a role in the development of pulmonary barotrauma. [3]

Alternatively, and perhaps more likely, the patient suffered alveolar injury upon ascending. As a diver ascends, the pressure within the alveoli of the lung increases as the pressure around the diver decreases. If intrapulmonary gas is trapped behind a closed glottis, as the diver ascends and the surrounding pressure decreases, the volume of the intrapulmonary gas increases in accordance with Boyle’s law. Increased pressure within the lung causes an increase in trans-alveolar pressure leading to overexpansion injury, alveolar rupture, and intraparenchymal hemorrhage. [4] A situation of rapid ascent to the surface, such as if a diver runs out of air, panics, or drops his weights, is often the cause of pulmonary barotrauma of ascent. Divers who hold a breath as they ascend and those with obstructive airway diseases, such as asthma or chronic obstructive pulmonary disease, are at increased risk. This was likely the case with our patient – he did not exhale and relieve the building pressure as he ascended, causing his pulmonary barotrauma.

DR. KRISTINA M CATEER: What were your next diagnostic steps for this patient?

DR. RICH: Laboratory studies were normal and an ECG revealed sinus tachycardia without signs of ischemia or right heart strain (no ST depression or T-wave inversions in V1-V4). The chest X-ray [Figure 1] reveals bilateral, patchy, airspace disease.

DR. JAMES RAYNOR: Did the patient require positive airway pressure or other airway management?
**DR. RICH:** The patient was placed on supplemental oxygen and was admitted to the ICU for observation and supportive care. A CT scan [Figure 2] was performed in order to evaluate for any underlying pulmonary parenchymal disorders and a repeat chest X-ray was obtained the following morning which demonstrated no appreciable change.

**Figure 1.** Chest X-ray reveals bilateral, patchy, airspace disease.

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**Figure 2.** Single image from chest CT scan showing bilateral patchy airspace disease.

**DR. OTIS WARREN:** It appears that this patient did quite well. However, did he risk other consequences from his pulmonary barotrauma?

**DR. RICH:** Yes, this patient was quite fortunate. The intrapulmonary pressure can become so elevated that air is forced across the pulmonary capillary membrane. Pulmonary interstitial air can dissect along bronchi to the mediastinum causing pneumomediastinum. Air from the mediastinum can track down the esophagus and great vessels causing retroperitoneal emphysema or pneumoperitoneum. [5] Mediastinal air can track superiorly to the neck, resulting in subcutaneous emphysema. Rarely, air may reach the visceral pleura, causing a pneumothorax.

One of the more concerning consequences of pulmonary barotrauma is due to an air embolism. If air enters the pulmonary vasculature, it can travel to the heart and embolize to other parts of the body. While a frequent cause of air embolism is iatrogenic secondary to interventional procedures, many other causes, including blunt and penetrating trauma, childbirth, and diving can lead to an air embolism. Clinical manifestations and severity of illness can range from asymptomatic to sudden onset circulatory collapse and are dependent upon the amount of air as well as the location of the air bubble. [6]

In our patient, due to his chest pain and hemoptysis suggesting alveolar injury, air embolism was a primary concern. Venous air embolism is less likely to cause severe symptoms. However an arterial gas embolism (AGE) can be devastating. Approximately 4% of divers who suffer an arterial gas embolism die immediately from total occlusion of the central vascular bed. [7,8] Furthermore, AGE patients who survive and present to the hospital frequently suffer neurologic deficits. Air entering the pulmonary vasculature can result in a cerebral air embolism (CAE) which can cause seizures, stroke, and death. [9]

**DR. VICTORIA LEYTIN:** What treatments are available for patients suffering from an air embolism?

**DR. RICH:** All cases of AGE must be considered for hyperbaric oxygen treatment as rapidly as possible. Treatment is required even if manifestations resolve prior to reaching an ED in order to prevent progression of subtle neurologic deficits that are not immediately detected. [10] Our patient showed no signs of right heart strain and had no neurologic deficits. He remained hemodynamically stable and had no respiratory distress. He was discharged on hospital day 2. The patient had no shortness of breath and was at his baseline after 2 weeks, and a follow up X-ray at that time revealed complete resolution of the bilateral infiltrates.
References


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