Myocardial infarction during scuba diving: A case report and review

Andrew S. Kanter, MD, MPH,[a]
B. Fendley Stewart, MD,[a] John A. Costello, MD,[a] and
Neil B. Hampson, MD[b] *Seattle, Wash.*

Scuba diving is one of the safest water sports. The incidence of diving injuries is only 0.07% per participant per year, compared to 0.17% for swimming and 0.24% for water skiing.[1] There are, however, hazards particular to the sport. We describe a case of scuba-related coronary air embolism and myocardial infarction in a young woman and review the pathophysiologic mechanism and treatment of arterial gas embolism.

A 43-year-old female sport scuba diver had a medical history of mild reactive airway disease and no risk factors for coronary disease. She was night diving in Puget Sound in cold water. Her first dive, to a depth of 15 feet with a total bottom time of approximately 30 minutes, was uneventful. She was on the surface for 6 hours between dives. Thirty minutes into her second dive (maximum depth of 35 feet), her regulator began to leak sea water. She began a slow ascent toward the surface while hyperventilating. Because of increasing salt water aspiration, she rapidly ascended the final 10 to 15 feet to the surface. Her diving computer did not indicate that she had ascended too rapidly, and she remained within the U.S. Navy no-decompression dive tables for the entire exposure (including both dives and surface interval). When she reached the surface of the water, she complained of dyspnea and chest tightness that impaired her ability to take a deep breath. She was transported to a nearby hospital, where a chest radiograph demonstrated bilateral lower lobe infiltrates consistent with aspiration. An electrocardiogram demonstrated minor elevation of the ST segments in the anterior precordial leads most consistent with early repolarization. She was transferred to our facility, where she no longer had chest pain or shortness of breath. An electrocardiogram showed 1.0 mm ST-segment elevation isolated to lead V_2, and she was given aspirin while myocardial infarction was ruled out. Ten hours after arrival at our facility, cardiac enzyme test results showed total creatine kinase (CK) of 183 U/L and MB fraction of 9% (normal 0 to 100, MB < 8%). Additional enzyme measurements drawn at 16 hours and 23 hours after the event were 159 (8% MB) and 136 (8% MB), respectively. A repeat electrocardiogram revealed 1.0 mm ST elevation and diminutive Q waves in leads V_2 and V_3 and T-wave inversions across the precordium and in leads I and aVL (Fig. 1). An urgent echocardiogram demonstrated severe hypokinesis of the anterior wall and interventricular septum. She was immediately given anticoagulant therapy (heparin) and transported to a nearby facility equipped with a hyperbaric chamber. Recompression was performed per U.S. Navy protocol (U.S. Navy Treatment Table 6)[2, 3] 36 hours after her initial transfer to our facility. A repeat echocardiogram performed soon after hyperbaric treatment demonstrated no improvement in wall motion. Because of concern about persistent coronary air and/or thrombus, coronary angiography was performed. The angiogram was completely normal, and a right anterior oblique left ventriculogram confirmed akinesis of the anterior wall. The temporal relation of diving activity to the patient's cardiac event makes a diving-related complication likely. Her clinical course is consistent with an arterial gas embolism to the left anterior descending coronary artery.

During scuba diving, ambient pressure increases 1 atm absolute (ATA) for each 33 feet of sea water below the surface. When compressed air is breathed at depth, the partial pressures of oxygen, carbon dioxide, and nitrogen increase within both alveoli and blood. Oxygen and carbon dioxide are respectively consumed and produced, as would be expected by the level of exercise undertaken. However, nitrogen is inert. During diving, high partial pressures of arterial nitrogen result in tissue uptake of the gas. Nitrogen moves in the opposite direction during ascent from depth, moving from tissue to venous blood to alveoli. Decompression illness occurs if the diver undergoes decompression too rapidly. When dissolved nitrogen comes out of tissue, it may form bubbles that result in pain or neurologic symptoms, depending on location.[3]

The pathogenesis of barotrauma and arterial gas embolism is different from decompression illness and is related to the physics of pressure and gases. When diving, gas in the lung expands as the ambient pressure decreases during ascent. Normally, these gases are exhaled harmlessly, and pressure equilibration occurs. If air is trapped in the lung, as behind a closed glottis, the pressure cannot equalize and the lung can sustain barotrauma similar to that found in patients receiving positive-pressure ventilation. Pressures as low as 100 to 500 mm Hg (0.13 to 0.20 ATA or only 4 to 7 feet of sea water) can exceed the alveolar elastic limits and cause alveolar rupture.[3, 4] Intraalveolar gas cannot decompress when the upper airway is closed or when functional obstruction of the smaller airways exists. Alveolar overpressure accidents may be associated with gas escape into the chest cavity (causing a pneumothorax

From the [a]Department of Medicine, University of Washington, and the [b]Section of Pulmonary and Critical Care Medicine, Virginia Mason Medical Center.

Reprint requests: B. Fendley Stewart, MD, Division of Cardiology, Harborview Medical Center, Box 359748, 325 Ninth Ave., Seattle, WA 98107.

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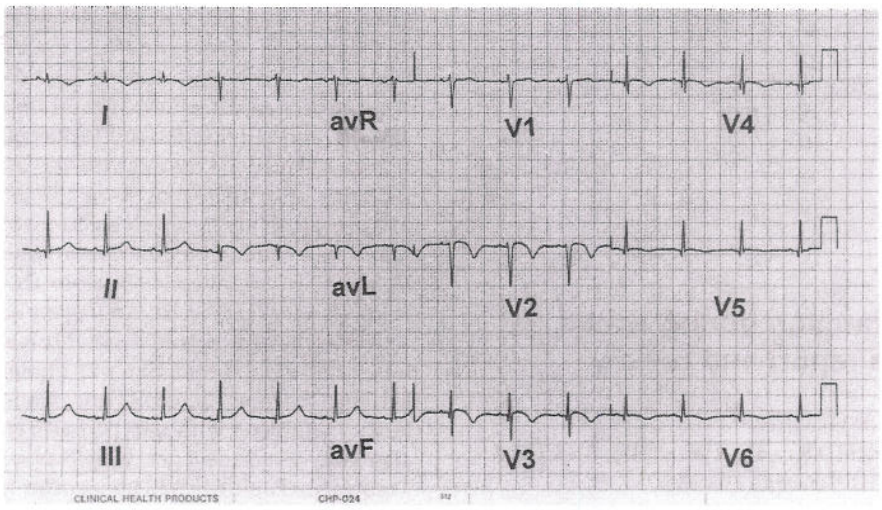

Fig. 1. Electrocardiogram demonstrates 1.0 mm ST elevation in leads V^2 and V^3 and T-wave inversion in lateral limb and chest leads. Small Q waves in chest leads likely represent normal variant. No old tracing was available for comparison.

or mediastinal emphysema) or into the pulmonary venous system with subsequent arterial gas embolism. Diving-related central nervous system gas embolism has been reported.[5] In addition, numerous studies have now documented that arterial gas embolism to the coronary circulation does occur,[6] and evidence for diving-related arterial gas embolism to the coronary circulation is available. Between 1928 and 1957 eight cases of fatal arterial gas embolism occurred in U.S. Navy recruits during training at the Submarine Escape Training Tank in New London, Conn. All victims had left-sided cardiac chamber and coronary artery air.[7] Finally, elevations of the MB isoenzyme of serum CK and electrocardiographic changes suggesting myocardial injury have been described in victims of diving accidents,[8] but specific evidence of myocardial injury in this setting is lacking.

When arterial gas embolism is suspected, initial treatment should include the immediate administration of oxygen and placement of the patient in a supine position. The use of hyperbaric oxygen is the cornerstone of therapy for arterial air embolism.[3, 9] In accordance with U.S. Navy treatment tables, patients undergo compression in a hyperbaric chamber to 2.8 or 6.0 ATA, depending on the acuity of injury.[9] At pressure, 100% oxygen is administered over a period of several hours. As predicted by Boyle's law, the increased pressure of recompression causes an immediate proportional decrease in gas bubble volume. Arterial partial pressure of oxygen is dramatically increased in the hyperbaric environment, and bubble size is also decreased by counterdiffusion.[3] The smaller arterial bubbles either pass distally or are metabolized by the tissues when nitrogen is replaced by oxygen. Although early identification of systemic air embolism is important to successful treatment, bubbles may remain in tissues for several days, and recompression for cerebral embolism has been successful days after the initial accident.[10] In addition, gas bubbles can serve as a nidus for thrombus formation, and although recompression may be successful at removing the offending gas bubble(s), the presence of thrombus could prevent normal blood flow from resuming.[9]

In the present case, the patient ascended rapidly through the water column from a depth of 10 to 15 feet. She could have had functional air trapping from aspiration of sea water and/or bronchoconstriction, or she may have inadvertently closed her glottis during the ascent. Expansion of intraalveolar gas during ascent resulted in local pulmonary overpressure and alveolar rupture. A small amount of air passed into the pulmonary venous component of the bronchovascular bundle, returned to the left side of the heart, and embolized to the left coronary circulation. A myocardial infarction in the territory of the left anterior descending coronary artery was confirmed by the electrocardiographic changes, CK isoenzyme elevation, and large anterior wall motion abnormality. In light of the small CK elevations, the large wall motion abnormality most likely represented myocardial stunning (a follow-up echocardiogram is not available, but the patient currently has no physical limitations and practices race walking as a hobby). A paradoxic embolus caused by severe decompression illness appears unlikely because she did not exceed the U.S. Navy no-decompression tables, and she did not exhibit other signs and symptoms of decompression illness. We cannot exclude coronary vasospasm as the cause of her myocardial infarction, but the coronary angiogram ruled out other potential causes such as congenital coronary anomaly or hemodynamically significant coronary atherosclerosis. This case confirms that myocardial infarction can occur as a complication of arterial gas embolism during scuba diving. The benefits of prompt hyperbaric therapy will be realized only if physicians maintain a high index of suspicion for myocardial injury during the evaluation of diving accident patients.

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