

Diver With Decompression Injury, Elevation of Serum Transaminase Levels, and Rhabdomyolysis

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A 43-year-old female recreational scuba diver presented to the emergency department 1 hour after a rapid, uncontrolled ascent. Her presentation included progressing confusion, slow and slurred speech, and complaints of headache and hypesthesia over her forearms and anterior thighs bilaterally. Differential diagnosis included arterial gas embolism and decompression sickness. She underwent recompression therapy with US Navy Table 6 within 120 minutes of her ascent. After recompression therapy, the patient had signs and symptoms consistent with severe rhabdomyolysis, including creatine kinase levels of 36,000 U/L and myoglobinuria.

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INTRODUCTION

Pulmonary barotrauma and arterial air emboli may develop in a scuba diver experiencing a rapid, uncontrolled ascent from a depth as insignificant as 1 m.¹ A scuba diver at depths greater than 7 m may accumulate a tissue partial pressure of nitrogen large enough that, on ascent to the surface, nitrogen exits tissues rapidly, forming bubbles in the blood and other organs,² which is known as decompression sickness (DCS). Although both of these bubble disorders can, in theory, cause injury to skeletal muscles (rhabdomyolysis), this case demonstrates the potential for massive muscle necrosis after decompression injury.

CASE REPORT

A 43-year-old female scuba diver presented to the emergency department 90 minutes after an emergency ascent from 63 ft.

The patient had initially dived to 100 ft (water temperature, 9°C [48.2°F]) for approximately 16 minutes, followed by a 2-hour-and-13-minute surface interval on a

dive boat. She then dove to 63 ft (water temperature, 14°C [57.2°F]). She apparently ran out of air after 27 minutes at depth and had a panic attack when her diving partner's custom regulator did not administer enough air. She took 3 breaths and then rapidly swam to the surface. On arrival at the surface, she was conscious and expelling seawater. Although weak, she climbed into the boat on her own. She complained of dyspnea and was immediately placed on 100% oxygen. On transport to our hospital, she became confused, with slow and slurred speech.

On arrival, her vital signs were stable, with a blood pressure of 122/81 mm Hg, pulse of 97 beats/min, respiratory rate of 22 breaths/min, and oral temperature of 37.4°C (99°F). She had a normal physical examination, except for neurologic findings. She had diffuse headache and hypesthesia over the anterior thighs and dorsal forearms bilaterally. She spoke with slow, slurred speech and had a blunt affect. She was alert and oriented to person, place, and time but could give no details of the accident or how she got to the hospital and only vague details of her past medical history. Her past medical history included treatment for depression, anxiety, and back problems. Her only allergy was to prochlorperazine, and her medications included trazodone for anxiety.

Results of her laboratory studies are presented in the Table. She had an immediate chest radiograph (negative for pneumothorax or pulmonary disease) and a head computed tomographic scan without contrast (negative) as the hyperbaric chamber was being prepared. Her toxicology screen was negative.

Her presentation was consistent with a cerebral arterial gas embolism caused by her rapid ascent (quite possibly with breath holding), and neurologic DCS (DCS type II) was also a possibility.

A US Navy Table 6 decompression protocol was instituted within 2 hours of her injury. She tolerated this therapy quite well, and after 20 minutes at depth, she had improved mentation, resolving headache, and no complaints of numbness on her arms or legs.

The next morning, the patient's mental status again worsened. She was confused, had slow speech, and complained of weakness and numbness of her arms and legs. We elected to retreat her with another US Navy Table 6. This time, she had no noticeable improvement of her symptoms at pressure. On completion of her second treatment, she complained of severe dyspnea and "loss of control" of her limbs. This resolved over approximately 20 minutes without medical intervention. Physical evaluation and an ECG showed no evidence of ischemia. At the time of occurrence, this episode was believed to be a panic attack because the patient suffered from anxiety disorders. In retrospect, it may have been an oxygen embolus caused by ascent from pressure and prior pulmonary barotrauma.

Because the patient did not show any improvement during the second hyperbaric oxygen therapy, supportive care was continued, including physical therapy, occupational therapy, and intravenous hydration. Nonsteroidal therapy was not used because the patient's skeletal creatine kinase (CK) and hepatic enzymes levels were markedly elevated and rising (Table).

Three days after the decompression injury occurred, the patient had a CK level of 36,340 U/L. The patient's urine was "cola" colored, with a myoglobin level of 20,000 µg/mL. Therapy was initiated to protect the kidneys (inducing a metabolic alkalosis with bicarbonate to alkalinize the urine and vigorous hydration). Additionally, because her hepatic transaminase levels were significantly elevated, an ultrasound of her right upper quadrant was performed, which was normal. The patient's renal function remained good, with a plasma creatinine level of 0.7 mg/dL.

A cardiac ultrasound was performed, which did not demonstrate a patent foramen ovale with either injected agitated saline or Valsalva maneuvers.

Over the course of her hospitalization, the patient had small improvements in her neurologic status while receiving intensive physical and occupational rehabilitation. Her muscle and liver enzyme levels peaked and began decreasing. The patient was transferred to a rehabilitation

Table.

Trends in significant laboratory studies over the course of hospitalization.

Hospital Day	Creatinine (mg/dL)	CK (U/L)	LDH (U/L)	AST (U/L)	ALT (U/L)	Urine Myoglobin (µg/mL)	Alkaline Phosphatase (U/L)
ED	0.8	298	220	36			55
1	0.7		746	543	131		60
2	0.7			908	240		
3	0.6	36,340	1,000			20,000	
4		20,073	777	684	359		47
5	0.5	7,401					
6		2,799		170	292		
7	0.7	1,040					
8	0.7	459	387	112	257		53

AST, Aspartate transaminase; **ALT**, alanine transaminase; **ED**, emergency department arrival.

hospital for 4 weeks and then discharged to home. She still complains of difficulty in concentrating and new onset of asthma but otherwise has returned to her normal state of health.

DISCUSSION

Rhabdomyolysis results from skeletal muscle injury and subsequent release of intramuscular cellular contents. It may be associated with myoglobinuria, although this is dependent on the amount of myoglobin released into the plasma, as well as renal function. Rhabdomyolysis is multifactorial in origin. Causes include alcohol abuse, muscle compression, seizures, drug abuse, trauma, hypothermia, sepsis, and severe exertion.³ Our patient did not have any of these risk factors, but she did have an uncontrolled rapid ascent to the surface and a possible systemic gas embolism in the hyperbaric chamber. We believe these events led to her rhabdomyolysis.

A scuba diver rapidly ascending from any depth is at risk for bubble disorders. These range from mild DCS (DCS I) with only a few symptoms largely limited to skin and musculoskeletal systems to more severe forms of DCS (type II) with cardiopulmonary and neurologic symptoms. DCS I and DCS II are caused by the presence of nitrogen bubbles that have developed as ambient pressure is reduced and partial pressure of nitrogen remains above a critical threshold.

The other bubble disorder that an ascending scuba diver is at risk for is a gas embolism. This may occur as ambient pressure rapidly declines and gas in closed spaces (eg, nonventilating lungs during a breath hold or through other air-trapping mechanisms) rapidly expands per Boyle's law. A gas embolism may also occur as a result of DCS because nitrogen bubbles can cross a patent foramen ovale and develop into a paradoxical arterial embolism. Additionally, a paradoxical embolism may arise when large amounts of gas entrained in the venous circulation overload the lungs and pass into the systemic arterial circulation.⁴ Generally, gas that has entered the arterial systemic circulation causes damage by occluding nutrient arteries of the head (cerebral arterial gas embolism) or the coronary vasculature. Bubbles may, however, go to any artery, including those perfusing skeletal muscles.

Elevations in CK, lactic dehydrogenase (LDH), and hepatic transaminase levels may be caused by 1 or more of the above bubble disorders. Our patient did not have a patent foramen ovale, and therefore, this mechanism for generating paradoxical air emboli is unlikely.⁵ However, an episode of dyspnea in the patient was reported shortly

after the dive. Dyspnea in combination with coughing is one of the major cardiopulmonary symptoms of severe DCS (type II), called "chokes," and is caused by large amounts of nitrogen bubbles entering the pulmonary vessels.⁶ In such cases, a spillover of gas bubbles into the systemic circulation is likely.

Smith and Neuman⁷ performed a retrospective study on divers with diagnosed arterial gas embolism and found that all had elevated CK levels. The median peak was 1,218 U/L for the group, with the highest level reported in their group being 45,170 U/L. They do not mention whether myoglobinuria was present with this patient. Further work by Smith and Neuman⁸ and Neuman⁹ describe elevations of LDH and transaminase levels, which are also associated with arterial gas embolism. They suggest that these enzymes may be elevated as a result of hepatic injury caused by the emboli.

Other researchers have demonstrated elevations in these enzymes associated with DCS. Powell et al¹⁰ described elevations in CK and LDH levels in pigs after deliberately induced DCS. Freeman and Philp¹¹ demonstrated that CK and LDH levels were elevated in moderate and severe DCS, whereas aspartate transaminase and alanine transaminase levels were only elevated in cases of severe DCS. Additionally, Williams et al¹² described a case of CK elevation caused by decompression injury, with a peak level of 5,418 U/L.

CK levels have been shown to be elevated with rigorous exercise. Martin and Nichols¹³ measured CK levels weekly in divers undergoing a strenuous dive course. They found an elevation in CK levels in all the divers (maximum peak, 910 U/L). They found that the divers performing the course in cooler water had a greater elevation in CK levels than their warm-water counterparts.

Additionally, there are some reports on rhabdomyolysis associated with near drowning.^{14,15} In these articles, the authors postulate that the mechanism of rhabdomyolysis is caused by hypothermic stress. They believe that cold-induced vasoconstriction and vigorous exercise (or shivering) is part of the mechanism. Our diver was wearing a full dry suit in 14°C (57°F) water (63 feet), and therefore, we believe that the near-drowning mechanisms do not apply to her. We believe multiple arterial gas emboli to the skeletal muscles and liver, severe DCS, or both are the most likely explanations for this patient's presentation.

This case emphasizes another aspect of the many organ systems that can be affected by a decompression injury. Along with the brain, spinal cord, heart, and lungs, the emergency physician evaluating victims of decompression injury should be aware of the musculoskeletal and

potential renal insults that may arise. We suggest, in cases of severe diving accidents, that serum enzymes and CK levels should be closely monitored.

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