A Puzzling Case of Arm Paresthesias.

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A puzzling case of arm paresthesias

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CASE

A 16-year-old previously healthy girl presented to the ED for evaluation of left arm soreness and paresthesias, which began 3 hours earlier. She recently completed a scuba certification course and earlier that day participated in her first two 30- to 45-minute cold-water quarry dives with an instructor. On her first dive, she descended to 25 ft (7.6 m) and paused at 15 ft (4.6 m) on a platform before ascending. After a 45-minute break, she dove to an initial depth of 25 ft, continued to a maximum depth of more than 40 ft (12 m), then ascended to the surface without stopping at the platform. Afterward, she was extremely tired and slept for 2.5 hours. Upon awakening she noticed an aching feeling in her left arm and a tingling sensation from her bicep to her fingers. In the ED, she described the pain as constant and uncomfortable, like her arm was falling asleep. She did not take pain relievers and could not recall injuring her arm. She had no allergies, denied the use of any daily medications, and was a nonsmoker.

On examination, the patient was well appearing. Her vital signs were: BP, 138/85 mm Hg; heart rate, 105; Spo_2, 100% on room air; and tympanic temperature, 99.1° F (37.3° C). The physical examination was significant for mild tenderness of the left mid-forearm and dorsal wrist in addition to a dull sensation when light touch was applied to the affected extremity from shoulder to fingers. The patient's strength was 5/5 in all extremities and her upper and lower extremity reflexes were intact (2+) bilaterally. She denied pain with movement of her left arm and her pulses were equal bilaterally. Her lungs were clear to auscultation bilaterally and her heart rate and rhythm were regular without any murmurs, rubs, or gallops.

Her arm appeared normal to inspection and the remainder of the physical examination was unremarkable. A complete blood cell count, complete metabolic panel, and urinalysis were unremarkable and a urine pregnancy test was negative. An ECG showed normal sinus rhythm with a ventricular rate of 90 beats/minute.

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WHAT IS YOUR DIAGNOSIS?

- type II decompression sickness
- type I decompression sickness
- barotrauma
- upper extremity deep venous thrombosis (DVT)

DISCUSSION

Given the patient's recent scuba dive and presentation of upper extremity joint discomfort, myalgias, and paresthesias, the most likely diagnosis was type I decompression sickness. Although type II decompression sickness also was a consideration, the patient did not have its common symptoms of neurologic deficits, chest pain, or difficulty breathing. Patients with barotrauma from aquatic activities

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often complain of otalgia, vertigo, epistaxis, and dyspnea, which this patient did not report. Although DVT is not directly related to scuba diving, it also was a consideration. However, the patient had no predisposing risks for DVT, was not hypoxic or tachycardic, and had no signs of edema or vascular congestion on examination.

Type I decompression sickness, colloquially known as "the bends," generally is not life-threatening and involves joints, skin, and lymphatics.[1] Type II includes other organ systems, particularly the brain, spinal cord, and lungs, and carries a higher risk of morbidity and mortality. A recent study of military divers concluded that in type I decompression sickness, 42% of patients reported symptoms within 1 hour after diving, 60% within 3 hours, 83% within 8 hours, and 98% within 24 hours.[2,3] Most commonly reported were pain (68%), paresthesias (63%), and constitutional symptoms such as malaise, fatigue, and headache (40%).[1]

Diving-related complications range from barotrauma to decompression sickness; type II decompression sickness includes arterial air embolism.[4] Decompression sickness results when the body is unable to eliminate nitrogen-predominant bubbles in the tissues or circulatory system after a scuba dive. The pressure of dissolved gases in the body exceeds the ambient pressure surrounding the diver, causing intra- and extravascular gas bubbles to collect, typically in joints, nervous and vascular tissues, and the pulmonary circulation.[5] This buildup results in endothelial injury and disruption of the body's microcirculation; capillary leakage and inflammation cause vascular obstruction and neurologic compromise.[1,6]

According to the Divers Alert Network, decompression sickness occurs in two to four per 10,000 recreational dives.[7] Nine divers died in 2007 from decompression sickness.[8]

The risk for decompression sickness is increased with cold water dives, flying after diving, and dehydration. Multiple dives within a single day also increase the risk, as dissolved nitrogen remains in the blood and tissues for up to 12 hours.[1] Divers can reduce their risk for decompression sickness by avoiding rapid ascents, abiding by platform stops, and ensuring that they do not have any chronic lung pathologies or acute infections before diving.[2]

Diagnosis of type I decompression sickness is based on patient history and physical examination. Chest radiographs may be obtained to rule out pneumothorax or arterial gas embolism, and CT or MRI may be obtained if cerebral hemorrhage or infarct is suspected, but these tests should not delay hyperbaric oxygen therapy.[2]

Recompression therapy with hyperbaric oxygen decreases the size of the bubbles that have accumulated in the tissues. The patient breathes 100% oxygen during the treatment, creating a natural oxygen-rich and nitrogen-poor gradient between the patient's blood and the bubbles. This process lets the bubbles decompress and diffuse into the capillary system for elimination.[4,9]

All patients with suspected decompression sickness should receive high-flow oxygen and IV fluid resuscitation. Oxygen accelerates the washout of the inert gases that have accumulated by increasing the gradient between the blood and tissues.[5] Several studies have shown that patients with decompression sickness who received 100% oxygen required fewer hyperbaric oxygen treatments than those who did not.[2] Fluids are also beneficial because tissue dehydration can intensify the symptoms of decompression sickness.[5]

Hyperbaric oxygen therapy is administered according to the US Navy treatment algorithm; length of treatment is determined by the patient's symptoms and dive history. Typically, patients remain in the chamber for 4.5 to 8 hours. Patients with complete resolution of symptoms after one treatment do not require additional interventions but are advised to refrain from diving for 4 weeks.

CONCLUSION

As initial therapy, the patient received 1 L of 0.9% sodium chloride solution and 100% oxygen via nonrebreather mask until she could receive definitive care. She was transferred to a facility with hyperbaric oxygen capability, where she received recompression therapy based on the US Navy treatment algorithm. Following one 271-minute hyperbaric oxygen therapy session, she reported complete resolution of all symptoms and did not require additional follow-up care.

Although decompression sickness is not commonly diagnosed in inland EDs, this diagnosis can occur in noncoastal settings, so all clinicians who work in emergency or urgent care should be aware of this diagnosis and its treatment options. **JAAPA**

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