Lower motor neuron weakness after diving-related decompression

Robert D. Henderson, FRACP; and Michael P. Pender, MD

Myelopathy is a recognized complication of diving and usually presents with upper motor neuron weakness and sensory loss of the lower limbs due to thoracic spinal cord damage. Here we present a case of lower motor neuron upper limb weakness due to infarction of the anterior horn cells of the spinal cord following diving. To our knowledge, this is the first report of an isolated lower motor neuron syndrome following diving-related decompression.

A 46-year-old woman was referred for electromyography (EMG) with a clinical diagnosis of ALS. Two months earlier, she had noted weakness in her right hand without sensory symptoms. The patient was a recreational scuba diver and recalled diving to 90 ft, 1 week prior to the onset of weakness. She ascended quickly but not in violation of her diving table and 20 hours later took an international flight. There was no significant past medical history.

On examination, there was wasting of the small muscles of the right hand and a prominent inability to extend the right middle finger. Scattered fasciculations were seen in the small muscles of the right hand. There was mild weakness of right elbow extension, forearm pronation, wrist extension and thumb abduction, and moderate weakness of finger extension and abduction. Left finger abduction was mildly weak, but power was otherwise normal in the left upper limb. The biceps, supinator, and triceps jerks were normal bilaterally. The upper limb sensation, cranial nerves, and lower limb examination were normal.

Nerve conduction studies showed low amplitude right ulnar and median compound muscle action potentials (CMAPs) with normal conduction velocities, F waves, and corresponding sensory studies. No conduction block was present. The right radial CMAP recording over the extensor digitorum was normal. The EMG showed fibrillations and fasciculations in the right first dorsal interosseous and opponens pollicis muscles with reduced recruitment of complex large motor units. There was increased size of motor units in the right deltoid, triceps, pronator teres, and lower cervical paraspinal muscles. Large motor units were also present in the left first dorsal interosseous and opponens pollicis muscles.

MRI of the cervical spine at the time of the clinical examination showed increased signal in the spinal cord from C3 to C7 vertebral bodies on sagittal T2 imaging. On T2 axial cuts, the increased signal was predominantly in the right anterior horn, with hypointensity on T1 imaging (figure). T2 hyperintensity was also present to a lesser extent in the left anterior horn. No enhancement occurred following the injection of gadolinium. The MRI was consistent with focal infarction of the cervical spinal cord. On follow-up, 4 months after the onset of weakness, her neurologic condition was unchanged.

Our patient’s hand weakness is explained by infarction involving the anterior horn cells in the spinal cord due to diving-related decompression. Spinal cord damage following diving is recognized, but our case is unusual because of the cervical cord involvement with lower motor neuron weakness of the upper limb, in contrast to the more typical thoracic spinal cord involvement with upper motor neuron weakness of the lower limbs and sensory loss. In our case, this presentation had led to the initial diagnosis by a neurologist of ALS.

Spinal cord infarction is usually reported with type II decompression sickness but can occur in the absence of clinical signs of decompression sickness. In a study of goats with different diving exposures, the presence of spinal cord lesions did not correlate with the typical features of decompression sickness. Spinal cord involvement may occur following dives conducted in accordance with US Navy decompression tables. Rapid ascent has been associated with spinal cord involvement.

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The most likely mechanism responsible for decompression-related myelopathy is spinal cord ischemia due to congestion of the epidural vertebral venous system by nitrogen gas bubbles. The epidural vertebral venous system (Batson plexus) can be obstructed by gas bubbles that collect, coalesce, and grow, when other major veins do not become obstructed, because the system functions as a valveless relatively stagnant venous lake in which the direction of flow changes frequently, in contrast to the unidirectional conduit function of other veins.⁶ In addition to mechanically obstructing venous outflow, gas bubbles in the venous bed may accelerate coagulation, leading to more complete venous obstruction.⁶

From the Department of Neurology, Royal Brisbane and Women’s Hospital, School of Medicine, University of Queensland, Australia.

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References