Decompression Illness in a Vegetarian Diver

A 36-year-old male dive instructor made one dive for 60 min at 18 mt and a second dive for 52 min at a maximum depth of 21 mt, with a surface interval of 2 hours. His dive computer gave a decompression stop for 8 min at a depth of 3 mt, which he accordingly made.

About 45 min after the dive, he felt tingling in his feet and left hand, weakness in his legs, pain in the elbow of the left arm and tiredness. During transfer to the hyperbaric chamber he breathed 100% oxygen and rehydrated himself by drinking 500 ml of water.

On his arrival at the hyperbaric chamber 6 hours later, he was well orientated with normal speech, pupil reaction and cardiopulmonary examination. The neurological examination was normal for cranial nerves, low reflexes in both arms, no reflexes in his legs, normal strength in arms and legs, abnormal sensory aspects for vibration and propriocepsis in his legs. Coordination was normal.

The patient was treated with a USN Table 6 and 4 daily HBO2 sessions (2.4 bar, 90 min). His symptoms gradually improved during the treatment tables, but in between there was a relapse of his sensory symptoms and weakness of his lower legs. On Day 4 of his treatment we heard about his vegetarian nutrition, so we performed additional hematological tests and found abnormal values, suggesting macrocytic anemia with a vitamin B12 concentration of 100 pmol/l (normal range 165-835), folic acid 10.9 nmol (9.2-38), iron saturation percentage 7% (25-50), serum-iron 4 µmol/l (12-30), ferritine 108 µg/l (50-300). The Schilling test performed to exclude malabsorption was negative. 1000 µg cyanocobalamin was administered intramuscular for 5 days, then weekly in the first month and monthly for 3 months. The patient was completely recovered within 4 weeks and prescribed daily multivitamin tablets, including B12. Because of his career as a sport/diving instructor, we also screened him for PFO by TEE, which showed no shunt. His blood values restored after 4 months, and he resumed diving after 6 months.

Discussion

After reviewing the medical literature, we believe this is the first published case of a vegetarian diver presenting a vitamin B12 deficiency in combination with DCS.

Vitamin B12 (cyanocobalamin) is abundant in meat, fish and most animal byproducts. However, strict vegetarians seldom develop a clinical deficiency, as only 2.0-5.0 mcg (microgram) of vitamin B12 is needed a day and an adequate amount is available in legumes. The most common cause of B12 deficiency is malabsorption due to defective intrinsic factor production.

Vitamin B12 deficiency affects the spinal cord, brain, optic nerves and peripheral nerves. The onset of symptoms is gradual, with general weakness and paresthesias (tingling, “pins and needles” feelings etc). As the illness progresses, the gait becomes unsteady and stiffness and weakness of the limbs (mainly the legs), develop. Initially, there may be no objective signs; later on, examination shows a disorder of the posterior and lateral columns of the spinal cord. Loss of the vibration sense is the most consistent sign, noticeable in the legs and often over the trunk; position sense is usually impaired. Loss of strength, changes in tendon reflexes and clonus affect the legs.

In divers, spinal DCS usually starts acutely within a couple of hours after surfacing with numbness, weakness in the legs, progressing with sensory and motor deficits: symptoms suggesting involvement of the spinal cord with a predominance of the dorsal and lateral columns. In histopathological studies, both DCS and vitamin B12 deficiency present spongi changes and foci of myelin and axon destruction in the

white matter of the spinal cord. The most affected regions are the posterior columns in thoracic and cervical levels, but there are also changes in the lateral columns. The pathological findings of the peripheral nervous system are those of axonal degeneration and significant demyelination. In acute DCS, bubbles cause vascular obstruction in the arterial and venous system and liberation of gas bubbles in white matter of the spinal cord with spongiosis, axonal swelling and myelin degeneration.

Monkeys kept on a vitamin B12 deficient diet for a long period develop neuropathological changes indistinguishable from those in humans, in a time comparable to the time required to deplete the vitamine B12 stores of patients with pernicious anemia. In this diver, the Shilling test excluded the pernicious anemia.

The most immediate goal in the treatment of B12 deficiency is to saturate body stores and prevent relapse for as long as possible. The advice is to administer 12 doses of 1 mg of vitamin B12 weekly as initial therapy, then follow a schedule of 1 mg of vit B12 every 3 months. All neurological symptoms and signs improve mostly in the first 3-6 months of therapy and then, slowly, during the ensuing year or even longer.

We concluded that the diver in this case had DCS based on the acute onset of symptoms after a provocative decompression dive; he was at risk because of a vulnerable spinal cord due to a long-lasting vitamin B12 deficiency. We also suspect that some of his symptoms during post-treatment were manifestations of a B12 deficiency enhanced by DCS. However, we do not believe that vegetarians in general are at risk for DCS, but they should be aware of their nutritional status, particularly regarding vitamin B12.

Contacts

Robert A van Hulst, MD, PhD, Diving
Medical Center, Royal Netherlands
Navy, PO Box10.000, 1780 CA Den
Helder, The Netherlands –
Email: ra.v.hulst@mindef.nl

Win van der Kamp, MD, PhD, Department
of Neurology, Medical Center
Leeuwarden, PO Box 888, 8901 BR
Leeuwarden, The Netherlands