De compression sickness in a vegetarian diver

Are vegetarian divers at risk? A case report

ROBERT A VAN HULST 1, WIM VAN DER KAMP 2

1 Diving Medical Center, Royal Netherlands Navy, The Netherlands
2 Department of Neurology, Sint Elisabeth Hospital, Willemstad, Curaçao

CORRESPONDING AUTHOR: Dr. R.A. van Hulst – ra.v.hulst@mindef.nl + ravhulst@planet.nl

ABSTRACT
We present a case of a diver who suffered decompression sickness (DCS), but who also was a strict vegetarian for more than 10 years. He presented with symptoms of tingling of both feet and left hand, weakness in both legs and sensory deficits for vibration and propriocepsis after two deep dives with decompression. The initial clinical features of this case were most consistent with DCS, possibly because of a vulnerable spinal cord due to cobalamin deficiency neuropathy. This case illustrates the similarities between DCS and a clinically defined vitamin B12 deficiency. The pathophysiology of vitamin B12 deficiency and common pathology and symptoms of DCS are reviewed.

INTRODUCTION
Decompression sickness (DCS) can present with a great variety of signs and symptoms, e.g., joint pain, skin, and cardiorespiratory and neurological problems. Based on the history of the dive time, dive depth and interval of the presenting symptoms, the diver will be treated for DCS according to regular hyperbaric oxygen treatment tables.

However, another neurological disease not related to diving may appear after hyperbaric treatment. For example, progressive ulnar palsy, Guillain Barré polyneuritis and multiple sclerosis have been reported (1-3).

We describe a case of a dive instructor who presented with DCS but did not recover completely after several treatments. Based on his additional medical history, we found that he had been a strict vegetarian for more than 10 years, with abnormal blood chemistry associated with macrocytic anemia and vitamin B12 deficiency. After treatment of the B12 deficiency, he recovered completely. We discuss whether vegetarian divers are at risk for DCS or only at risk in the recovery phase of the hyperbaric treatment.

Case report
A 36-year-old Caucasian male dive instructor made one dive for 60 minutes at 18 meters (m) and a second dive for 52 minutes at a maximum depth of 21 m. Surface interval between the two dives was two hours. His dive computer gave a decompression stop for eight minutes at a depth of 3m, which he accordingly made.

Approximately 45 minutes after the dive he noticed tingling in both his feet and his left hand, weakness in both legs, pain in the elbow of the left arm and a general tiredness. He started to breathe 100% oxygen and rehydrated himself by drinking 500 ml of water. During transfer to the hyperbaric chamber he breathed 100% oxygen.

Arrival at the hyperbaric chamber was six hours later. On arrival he was well oriented with normal speech, normal pupil reaction and a normal cardio-pulmonary 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 U.S. Navy Table 6 and four daily HBO2 sessions (2.4 bar, 90 minutes). His symptoms gradually improved during the treatment tables, but in between there was a relapse, particularly of his sensory symptoms and weakness of his lower legs; this persisted after the last HBO2 treatment. His medical history showed no neurological abnormalities, and he had no neurological signs or symptoms in the recent past.

Based on his history of vegetarian nutrition (which came to us on Day 4 of his treatment), we performed additional hematological laboratory tests. The values were abnormal, suggesting macrocytic anemia with:

- a vitamin B12 concentration of 100 pmol/l (normal 165-835);
- folic acid 10.9 nmol (normal 9.2-38);
- iron saturation percentage 7% (normal 25-50);
- serum-iron 4 μmol/l (normal 12-30); and
- ferritine 108 μg/l (normal 50-300).

A Schilling test was performed to exclude malabsorption and was found negative. Clinical neurophysiological examinations for EMG and SEP (n. tibialis anterior) were normal; there was no polyneuropathy. After consultation with internal medicine, we administered 1000 μg cyanocobalamin intramuscularly for five days and repeated these parenteral injections weekly in the first month, and then monthly for three months. Within four weeks the patient was completely recovered from his residual neurologic symptoms, and we advised him to take multivitamin tablets that include B12 on a daily basis. In addition, because of his career as a professional sport-diving instructor, we screened him for patent foramen ovale by transesophageal echocardiography (TEE), which showed no shunt. His blood values restored after four months, and he resumed diving after six months.

**DISCUSSION**

Neurological signs and symptoms presenting acutely after diving are most likely manifestations of DCS and require oxygen and hyperbaric treatment (4). However, different neurological diseases and neurological DCS share similarities in their presentations and thus may be mistaken for one another (1-3, 5). After reviewing the medical literature, we believe this is the first published case of a vegetarian diver with a vitamin B12 deficiency presenting in combination with DCS.

Vitamin B12 – cyanocobalamin – is one of several cobalamines having biological activity in the body. They are abundant in meat, fish, and most animal byproducts. Although vegetables are generally devoid of this vitamin, strict vegetarians almost never develop a clinical deficiency, as only 5 mg of vitamin B12 is needed a day, and an adequate amount is available in legumes. Intrinsic factor, a binding protein secreted by gastric parietal cells, is needed for internal absorption of B12; malabsorption due to defective intrinsic factor production is probably the most common cause of B12 deficiency (6).

Vitamin B12 deficiency affects the spinal cord, brain, optic nerves and peripheral nerves. The onset of manifestations is gradual over months; the spinal cord is usually affected first and often exclusively (7). Patients first notice general weakness and paresthesias consisting of tingling, a “pins and needles” feeling and other vaguely described sensations. As the illness progresses, the gait becomes unsteady and stiffness and weakness of the limbs, especially the legs, develop.

Early in the course of the illness, there may be no objective signs. Later on, examination discloses a disorder of the posterior and lateral columns of the spinal cord. Loss of the vibration sense is by far the most consistent sign, more pronounced in the legs and frequently extending over the trunk. Position sense is usually impaired as well. The motor signs, usually limited to the legs, include loss of strength, changes in tendon reflexes and clonus.

In divers, spinal DCS often starts acutely within a couple of hours after surfacing, with numbness, weakness in the legs and is often progressive with an ascending level of both sensory and motor deficits (5). These symptoms suggest involvement in the spinal cord with a predominance of the dorsal and lateral columns. In histopathological studies,
both diseases (i.e., DCS and vitamin B_{12} deficiency) present spongi changes and foci of myelin and axon destruction in the white matter of the spinal cord. The most severely affected regions are the posterior columns in thoracic and cervical levels, but changes are also seen in the lateral columns. The pathological findings of the peripheral nervous system are those of axonal degeneration and significant demyelination (8,9). The mechanisms in acute DCS are bubbles which cause vascular obstruction in the arterial and venous systems and liberation of gas bubbles in white matter of the spinal cord with spongiosis, axonal swelling and myelin degeneration (10,11).

Monkeys who are sustained on a vitamin B_{12}-deficient diet for a prolonged period develop neuropathological changes indistinguishable from those in humans (7). The time required for the production of neurological changes in monkeys (33-45 months) is comparable to the time required to deplete the vitamin B_{12} stores of patients with pernicious anemia, in whom parenteral vitamin B_{12} therapy has been discontinued. In the diver described here, performing the Shilling test excluded pernicious anemia.

Serum cobalamin should be measured whenever the diagnosis of vitamin B_{12} deficiency is in question. Serum levels of less than 100 pg/ml are usually associated with neurological symptoms and signs of vitamin B_{12} deficiency.

The most important goal in the immediate treatment of B_{12} deficiency is to saturate body stores and prevent subsequent relapse for as long as possible, in case patients discontinue maintenance therapy at some later time (12). Based on empirical observations, the advice is to administer 12 doses of 1 mg of vitamin B_{12} on a weekly basis as the initial therapy; once the complete series has been given, the patient can be on a schedule of 1 mg of vitamin B_{12} every three months (12, 13). In general, all neurological symptoms and signs may improve, mostly during the first three to six months of therapy and then, at a slower rate, during the ensuing year or even longer (7, 12).

Recently, MRI has been employed to diagnose subacute combined degeneration due to cobalamin deficiency. It shows a very typical pattern, with T2 hyperintensive signal alterations (usually confined to the dorsal columns), and allows early differentiation of this disease from other myelopathies (14,15).

In contrast, MRI has been used in divers with acute neurological DCS to show cerebral and spinal lesions. However, the technique was relatively insensitive and often failed to detect lesions in patients with obvious neurological signs and symptoms (5).

In the case presented here, we concluded that the diver initially had DCS based on the acute onset of symptoms after a provocative decompression dive. We speculate that he was at risk because of a vulnerable spinal cord due to a long-lasting vitamin B_{12} deficiency. In addition we also suspect that some of the symptoms in the post-treatment period were manifestations of a B_{12} deficiency enhanced by DCS.

Dive physicians should add vitamin B_{12} deficiency to the list of differential diagnoses of DCS because of the similarity of neurological signs and symptoms. 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 B_{12}. If in doubt, and especially if they do not take supplements, divers need to consider an evaluation by laboratory tests in those who are dive instructors and in those who frequently make decompression dives.
REFERENCES


