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A unique case of a 70-hour decompression sickness latency

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We report a unique and well-documented case of a type II decompression sickness (DCS) with a latency interval of 70 hours. It may raise divers' awareness and help medical practitioners to keep suspect divers under close observation longer than before and identify and treat DCS accordingly.

KEY WORDS: *diving; hyperbaric oxygenation; neurological differential diagnosis; prophylactic decompression; therapeutic recompression*

The time between the end of diving, that is, returning to surface atmospheric pressure and the appearance of decompression sickness (DCS) (1) is called symptom latency. The symptoms usually appear within an hour of surfacing (ranging between 42 to 85 % of cases), after which time the chances for the development of DSC drop exponentially. In rare cases of an incident or gross negligence of standard decompression procedures, DCS may develop even before surfacing, that is, at decompression stops during ascent (2-6).

CASE PRESENTATION

Diagnosis and treatment

Our patient was an experienced (CMAS three-star) diver aged 43, who was admitted to the emergency ward of the Hyperbaric Medicine Polyclinic OXY in Pula, Croatia on 10 September 1996 for recompression treatment, as he presented with the symptoms of type II DCS. He had no previous history of DCS and he had always been in good health until that incident.

According to history, he was in a party of ten searching for a missing diver in the waters of the port of Pula, and, allegedly, followed the same routine as the other nine of the party, none of whom developed DCS in the aftermath. He dived in a group of three to seven for seven days in a row, one dive a day, using complete diving gear, including a standard scuba [2x10 L / 200 bar (20 MPa)] and an Aladin® dive computer (Aladin Pro; UWATEC; Hallwil; Switzerland). The descents went between 35 and 39 m, and the scheduled dive time was 20 min, but the group rarely exceeded 15 min. The procedures were much the same as described for the following two dives.

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On 6 September 1996 at 12:30 h, our patient made a single dive with two other divers in good diving conditions and descended to 36 m below the surface. There he spent 18 min and went through the decompression procedure observing the readings on his dive computer. That dive went without any DCS symptoms. On 7 September 1996, he again made a single dive with two other divers at 16:30 h in good conditions, descended to 39 m, spent there 16 min, went through decompression observing his dive computer, stopped at 6 m and decompressed for one minute, and then again stopped at 3 m to decompress for another two minutes. In the evening, he went to a wedding and stayed up all night. He did not dance or drink alcohol. He also did not fly during the no-fly period.

Over the following three days, he did not dive or have any DCS symptoms, not even an unusual fatigue. He felt a little drowsy, which he attributed to the sleepless night at the wedding.

On 10 September (treatment day 1) at 15:00 h, his left leg went numb and his upper and lower leg muscles felt tight, so he could not step up a stair. At 16:00 h, his left forearm also went numb from the fist to the elbow. He could not control its movement properly and could barely hold a glass. This prompted him to request recompression therapy at our polyclinic.

On admission, the patient presented with left side mild to moderate pyramidal insufficiency and loss of sensation at spinal level T1. His condition was diagnosed as type II cerebral and spinal DCS, and at 17:00 h, two hours after the onset of the first symptoms, recompression treatment was started, following the US Navy Table 5 schedule (2). The patient was placed in the hyperbaric unit, set at the initial pressure of 2.8 bar (0.28 MPa). Even before oxygenation started, the patient reported that his left arm and leg numbness had gone and that he could move his limbs normally. This remained throughout the first recompression treatment.

Immediately after the treatment, the patient was referred for blood biochemistry and accompanying tests and for neurological examination. He still reported no symptoms, but the neurologist established discrete signs of disorder, such as veering to the left while walking, weakened abdominal skin and plantar reflexes, loss of tactile sensation (hypoesthesia) in the palm-size area above the left knee, mild tension along the ulnar nerve, numbness, and weaker motor function of the fourth and fifth digit on the left arm. Thermal and vibration sensations were normal.

Physical examination revealed no other pathology. Ophthalmology showed no hypertonic changes.

After the required one-day pause, recompression treatment was resumed with hyperbaric oxygen at 2.8 bar (0.28 MPa), following the US Navy Table 5 schedule (2), and was extended to two 60-minute sessions a day.

On 13 September (treatment day 3), the CT brain scan revealed a suspect ischaemic lesion in the anterior horn of the right lateral ventricle.

On 20 September (treatment day 10) during hyperbaric oxygen treatment, the patient complained of burning on the ulnar side of the left forearm toward the 4th and 5th digit but also reported that his muscles were no longer tight and that he could now move his fingers freely. Since the admission until 20 September, the patient had also gradually stopped veering to the left. Only the loss of surface sensation above the left knee remained. After this oxygen treatment, signs of lesion at the spinal level disappeared and only a mild pyramidal deficit remained.

All recompression and hyperbaric oxygen treatments were accompanied by corticosteroid therapy with a single 8 mg tablet a day. At the time, the use of corticosteroids was a usual protocol for some types of swelling and inflammation.

All blood biochemistry findings were normal throughout the treatment, including the risk parameters for the cerebrovascular insult (CVI).

On 24 September (treatment day 14), transcranial cerebral artery and extracranial carotid and vertebral artery Doppler showed normal findings. Bubble study was not done, as our patient was a weathered diver with no history of diving disorders that would point to patent foramen ovale.

The treatment continued with single hyperbaric oxygen 60-minute sessions at 2.2 bar (0.22 MPa) a day until the sensation above the knee was completely returned and the neurological exam showed normal findings on 10 October (treatment day 33), when the patient was discharged as fully recovered.

The follow up visit on 21 October with the somatosensory evoked potential test (SSEP) showed normal findings.

The patient continued to dive occasionally following similar diving schedules as before without any incident. We followed him up for 20 years through direct contact and occasional examinations and not once did the patient have abnormal neurological findings.

Differential diagnosis

Besides DCS, the differential diagnosis included patent foramen ovale and pulmonary alveolar barotrauma caused by intrathoracic overpressure during exertion, but these were excluded immediately because of their very short latency.

Cerebrovascular insult (CVI) or stroke was also excluded based on the disease history and negative clinical and laboratory findings for the CVI risk factors (7). Furthermore, the multifocal (cerebral, spinal, and possibly peripheral) localisation of the neurological lesions did not support the CVI diagnosis. Additional tests, such as electrocardiogram (ECG) and colour Doppler imaging of intra- and extra-cranial arteries turned out normal. Only the brain CT showed a suspect ischaemic lesion as described above, which can hardly be related with the neurological insufficiency we observed.

Another possible diagnosis was multiple sclerosis (MS), but the two decades of follow up showing normal neurological findings also exclude this diagnosis. MS diagnosis is based on the spreading of neurological insufficiencies over time, documented with clinical, paraclinical, and laboratory evidence. Before MS is diagnosed, other causes need to be excluded (8-10). In our case, we excluded the following types of MS: relapsing-remitting, primary and secondary progressive, and progressive-relapsing.

We also considered the unlikely possibility that clinical isolated syndrome (CIS) could manifest itself, not as much as one of the MS diagnosing criteria, but rather to follow up disease progression (11). Unfortunately, we could not do the MR scan with our patient because of a number of dental implants, whereas spinal fluid analysis was not justified as time went by.

We excluded Lyme disease, as it was not supported by the patient's history and clinical findings. In addition, this part of Croatia is virtually free of the tick species *Ixodes ricinus*, which transmits *Borrelia burgdorferi*, and the patient denied having recently visited the areas where such risk is high (continental Croatia).

DISCUSSION

Longer latency is very uncommon, but cases of much longer latencies in DCS patients have also been reported, the longest being 48 h (2, 12), followed by 36 h reported by Rivera (13), 29 h reported by Hadanny et al. (14), and 24 h reported by Wilmhurst and Bryson (15). Haas et al. (16) reported a latency span between immediately on resurfacing to one week (with a median of 1.5 h) in 520 DCS patients, but this retrospective descriptive analysis should be taken with reserve, as the authors themselves warned of incomplete information to safely establish latencies.

We hope to have provided enough evidence of type II DCS. However, this would be nothing new if the patient had not had the latency period of over 70 h after the last dive, which, to the best of our knowledge has never been reported and reliably documented before.

Latency as long as this requires that divers and, more importantly, medical practitioners should review the adopted procedures and look for DCS signs and symptoms beyond the usual latency periods and even introduce hyperbaric treatment as prophylaxis as soon as suspicion arises.

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Conflicts of interest

None to declare.

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Asimptomski površinski interval kod dekompresijske bolesti trajao 70 sati: prikaz slučaja

Pridržavanje profilaktičkih procedura omogućuje da ronjenje bude bez ronilačkih bolesti. Odstupanje od profilaktičkih procedura vodi dekompresijskoj bolesti (DCS), to prije i izraženije što su odstupanja veća. Vrijeme od izrona do pojave prvih simptoma DCS-a naziva se simptomska latencija ili površinski asimptomski interval. Do sada najduža publicirana latencija iznosila je 48 sati. Prikazuje se jedinstveni i dobro dokumentirani slučaj ronioca oboljeloga od DCS-a tipa II nakon latentnog intervala od 70 sati, koji prema našemu najboljemu saznanju dosad nije dokumentiran u literaturi. Upravo to – da se ronioca suspektnoga na DCS drži duže na opservaciji (sada do 70 sati) i pri porastu sumnje profilaktično odmah primijeni inicijalni rekompresijski tretman – teoretski je i praktično značajno ne samo za ronioce nego i za njihove liječnike i ronilačke instruktore.

KLJUČNE RIJEČI: *hiperbarična oksigenacija; neurologijska diferencijalna dijagnoza; profilaktička dekompresija; ronjenje; terapijska rekompresija*