Severe spinal cord decompression illness after an uneventful North Sea dive

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ABSTRACT

The aim of this case report is to illustrate that, even under moderate conditions, a dive can result in spinal cord decompression illness (DCI). The diver in question completed five dives with the same profile. The first four included substantial physical strain, while the final dive was for observation only, without physical strain.

The spinal cord was the target organ for DCI. We discuss the roles of various diver-related risk factors and of factors related to the dive itself. Older divers have a higher risk for decompression incidents. The nature of the dive profile is a major factor in the uptake and release of inert gas. Physical exertion during pressure-exposure boosts the inert gas load, increases bubbling in tissues and raises the risk of DCI in the decompression phase of the dive. We discuss the causal involvement of such risk factors in this case, given the characteristics of the diver and the circumstances of the dive. Finally, we want to express our concern for physical fitness and smoking habits, especially for divers over the age of 40.

INTRODUCTION

It is widely recognized that the workplace of the professional diver is a potentially hostile environment, and one that can change very rapidly. In general, the professional diving industry uses safe procedures. It is important that the divers are well prepared for the job, that they are physically fit, use appropriate diving procedures for a safe outcome, and that they have the professional knowledge and skills required.

In the so-called "toolbox meeting" prior to the dive, procedures are reviewed and discussed in great detail. A decompression sickness incidence of 0.5% per dive depth profile is reported for the decompression tables recommended by the official Dutch governmental body for diving affairs (the National Diving Center, or NDC) [1].

Decompression disorders have several manifestations caused by the place where bubbles impede circulation or tissue function. The concept of decompression illness (DCI) refers to a multisystem condition that may express itself in a variety of manifestations [2,3].

The first objective of this case report is to illustrate the fact that, even under controlled conditions such as these, an uneventful dive may have serious and unexpected consequences. As a second objective, we investigate possible risk factors. We also attempt to give some recommendations for occupational health and safety for experienced divers over the age of 40.

CASE REPORT

A professional diver (a 45-year-old male) employed by a diving company was carrying out work in the North Sea, about 323 km (200 miles) from the nearest harbor. He had 20 years of experience in the diving industry, with an annual exposure in the order of 250 dives.

On the day of the incident (August 7, 2007), he was scheduled to work the night shift. At 21:00, he and one of his colleagues had a pre-dive "toolbox meeting" with the dive supervisor. The diving procedure for the night was a surface-supplied dive involving a surface decompression procedure in the decompression chamber. The diving depth was estimated to be 31.5 meters and total bottom time was set at 53 minutes. The decom-

pression schedule chosen had a maximum diving depth of 33 msw (meters of sea water) and 60 minutes of bottom time and was used in accordance with a Dutch professional diving table (NDC-Table NSOX88). His dive equipment consisted of a hard-hat diving helmet with underwater communication and a video camera on top. He wore a drysuit and fins. The breathing gas was compressed air supplied from the surface through an umbilical system.

At 21:20, both divers entered the water. This was their first dive of the day. The surface interval for both divers was more than 20 hours. The work in question involved the inspection of concrete mattresses above a pipeline. The dive was not physically demanding (professionals often refer to this type of dive as a "Mickey Mouse" dive). At 22:13, they returned to the surface. The dive profile included two in-water stops during the ascent. The first was a four-minute stop at 12 meters, and the second was a three-minute stop at a depth of 9 meters. This has been verified by the dive supervisor.

Diagnosis and treatment

During the ascent and just before surfacing, one of the divers suddenly felt acute pain in his groin and lost control of his legs. At first he thought that his diving umbilical had twisted around his legs. On investigation, however, this was not the case.

When the diving basket was lowered onto the deck of the ship, the diver was unable to stand and collapsed. He was stripped of his dive gear and, with great difficulty, placed in the hyperbaric chamber. Normally, this procedure takes less than three minutes. In this case, however, it took about seven minutes. The planned surface decompression procedure with oxygen in the chamber was cancelled and replaced by an emergency procedure, in accordance with U.S. Navy Treatment Table 6.

The dive medical attendant, a diver trained in decompression illness management – including diagnostics and treatment under supervision – performed a brief neurological examination in the chamber. The diver was disoriented in regard to his location, the date and time of day. His cranial nerves were intact, but he had a decreased motor response in the muscles of the right arm and paraparesis in both legs. His legs had a bilateral lack of sensitivity to touch, pain and temperature. A more specific examination could not be performed at that time. The patellar tendon reflexes on both knees were depressed, while the feet exhibited a Babinski reflex. Based on these symptoms, it was decided to extend the USN Table 6 with one additional oxygen cycle for 30 minutes at 9 meters. Fifteen minutes before the conclusion of the extension of USN Table 6, the subject developed tingling, pricking and fasciculations in his extremities. This was interpreted as a neurological manifestation of acute oxygen toxicity, and the treatment was aborted.

Before the diver left the chamber he was examined by the dive medical attendant. The neurological examination revealed that the subject had recovered his habitual strength. At that time the diver complained of pain in his bladder and that he could not urinate. A urinary catheter was inserted and 1.2 liters of urine were released. The pain subsided, and the catheter was removed.

The subject left the hyperbaric chamber, supported by the attendant, because he could not walk unsupported. This was probably due to balance problems. Shortly thereafter, he was brought to bed. He experienced malaise, could not sleep and experienced pain throughout his body. Six hours after leaving the hyperbaric chamber, he noted that power and motor control of his legs was deteriorating. He was not able to move his legs voluntarily and had a neurogenic urinary bladder and neurogenic disturbance of defecation.

The dive supervisor consulted the company's dive physician by telephone for further treatment advice. He was advised to treat the subject in the hyperbaric chamber again, in accordance with USN Table 6.

Some 20 minutes after the start of this second treatment, the subject's condition deteriorated still further. He developed numbness in his extremities, and muscle fasciculation. Believing that the diver was once again showing signs of an acute oxygen toxicity, the treatment was aborted. When the diver tried to leave the chamber he could neither stand nor walk, due to loss of muscle strength and pain in his legs. The urinary catheter was again applied because the patient still was unable to urinate. An emergency evacuation by helicopter was considered but the supervisor did not request it, and the ship set sail for harbor. On the advice of the dive physician, the attendant contacted the Royal Netherlands Navy's Diving Medical Center to ask them to take over the treatment of this patient.

On Day 2, a day and a half after the last treatment, the ship arrived in the harbor. Since the subject had undergone two hyperbaric oxygen treatments during the

preceding 24 hours, it was decided not to repeat this procedure a third time. Instead, the diver was transferred to a hospital, where he was medically examined by a neurologist.

The subject was fully alert, and exhibited no disorientation with regard to his identity, location, day and date. The motor function in his arms was impaired, more so on the right side than the left. There was a decline in muscle strength in both legs, as well as a reduced sensitivity to pain, temperature, vibration and proprioception in both legs. Babinski reflexes were still present in both feet.

The patient had no other significant illness or injury other than a history of a herniated lumbar disk some seven years prior. He had continued to use diazepam during periods of lower back pain. His medical history revealed no previous diving-related illness – *e.g.*, DCS-decompression sickness/ CAGE-cerebral arterial gas embolism).

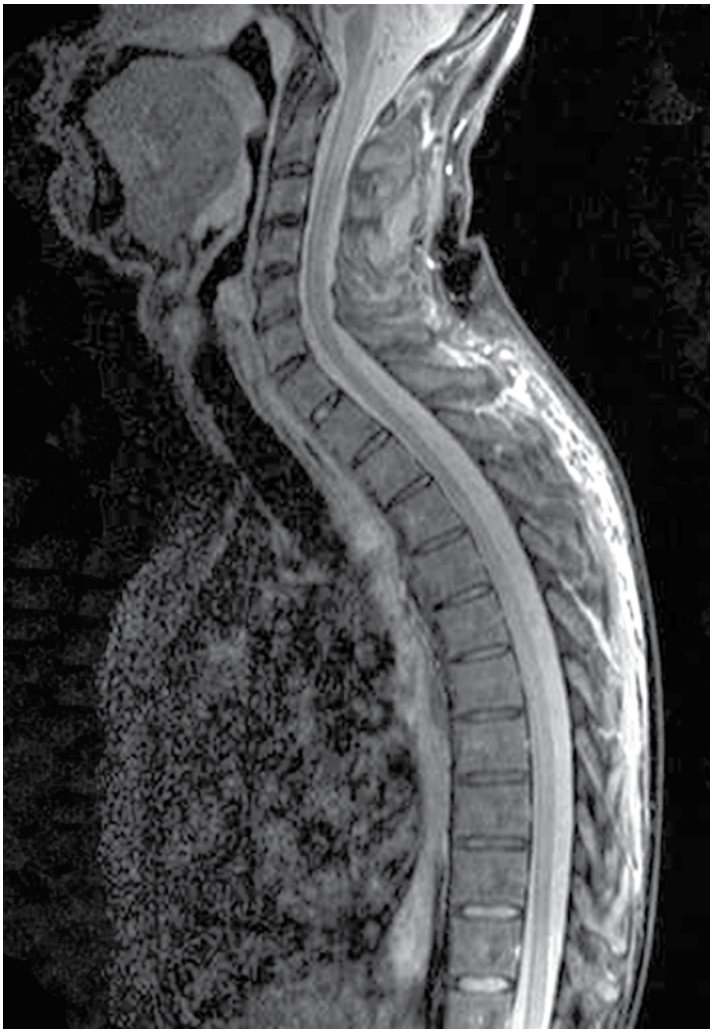
Based on the neurological examination it was concluded that the subject had suffered spinal cord injuries at the cervical and thoracic levels. An MRI scan was administered on Day 3. An area of myelopathy was revealed in the spinal cord, at C2 level. There was no evidence, however, of a herniated lumbar disk. The patient exhibited complete paraplegia, and was treated with a urinary catheter.

The patient was subsequently transferred to a Diving Medical Treatment Center on Day 3 following the incident. The differential diagnosis included spinal vascular incident; spinal cord arterial gas embolism (AGE) due to pulmonary barotraumas and spinal cord decompression sickness. He was treated in accordance with a modified USN Navy Table 9, in which he was given six oxygen blocks (of 20 minutes each) with subsequent air breaks (of five minutes each) at 9 msw). During hyperbaric treatment the patient received an intravenous injection of 100 mg lidocaine followed by 1 mg lidocaine intravenously per minute for the total hyperbaric period.

The subject was treated for seven days, receiving one daily exposure to Table 9 for as long as his clinical situation continued to improve. The final treatment had to be terminated prematurely due to chest pain and dyspnea, which was interpreted as a sign of pulmonary oxygen toxicity.

The final medical examination at the Diving Medical Treatment Center on Day 9 after the event revealed a slight recovery in the motor function of both arms, more so in the left arm than in the right. Motor function in both legs had neither improved nor worsened; the subject was still unable to move his legs. While the diver had partially regained sensitivity to pain and temperature in both legs, proprioception was still absent.

FIGURE 1. MRI cervical spine sagittal recording STIR sequence table position 17.6.



At level C2 the dorsal part of the spinal cord has a higher signal (whiter on the image) compared with the rest of the spinal cord. The signal of the surrounding spinal cord fluid is significantly higher compared to the abnormal part of the spinal cord. This is diagnosed as myelopathy of the spinal cord.

In view of the symptoms of pulmonary oxygen toxicity, and because there had been little improvement in his medical condition, the diver was transferred to a hospital closer to his home for rehabilitation.

Rehabilitation

The neurological ward

The diver was initially treated in the neurological ward. The clinical examination, performed by a neurologist 10 days after the event, revealed that muscle motor function in the patient's left arm had returned to a normal level. However, there still was a distal paralysis of the right arm and paraparesis of the right leg muscle strength (proximal Grade 0, distal Grade 2), and the left leg muscle strength (proximal Grade 2, distal Grade 3).

Fine tactile and kinesthetic sensations below the level of Th9 were abnormal. The sensitivity to pain, temperature and touch was disturbed. These findings imply that both the dorsal column and the anterolateral tract were involved. Patellar and Achilles reflexes in both legs were weak, and Babinski reflexes were present on both sides. The patient exhibited urinary incontinence and abnormal defecation. Medication for bowel stimulation (Macrogol/electrolytes) was prescribed, together with heparin to optimize blood flow and counteract sludging and thrombotic mechanisms. The continuous pain in both legs was treated with amitriptyline.

As part of the treatment 10 days after the incident a high-resolution CT scan of the lungs was performed. Paraseptal and centrilobulair emphysema of both lungs was detected. MRI revealed no abnormalities in the cerebrum and cerebellum. Furthermore, no lesions in the spinal cord could be found.

Rehabilitation ward

Twenty-four days after the event, the diver was transferred to the hospital's rehabilitation ward, where he remained as an inpatient until 38 days after the event. His further treatment was continued on an outpatient basis. Eight weeks after the incident, the diver was performing regular daily rehabilitation exercises. He was able to walk about 10 meters on crutches, but was confined to his wheelchair when carrying out his daily activities. He urinated by means of a condom catheter. The subject suffered sexual dysfunction and required enemas to regulate his bowel movements.

The patient's physical condition showed no further improvement during the two-year period following the incident. At the end of this period, in March 2009, the patient was send to a university hospital for a new MRI examination of his spinal cord. The entire spinal cord was examined using a DTI (digital tensor imaging) technique to examine the continuity of its white matter. This revealed no abnormalities in the structure or shape of nerve fibers in the spinal cord. The clinical picture remained unmistakably paralytic.

The diver was mobilized as much as possible. He undertook regular physical therapy, swimming exercises, and familiarization exercises with crutches and a wheelchair. He is now able to walk some 25 meters with the aid of crutches and has found appropriate work, in the form of a one-year post as an assistant teacher in a secondary school.

DISCUSSION

This case demonstrates that even uneventful dives can result in severe spinal DCI. In our case, the diver presented with symptoms described as spontaneous neurological deficit with muscular weakness and paralysis with pain, reduced level of consciousness and bladder dysfunction. We present some risk factors as possible clues for preservation of health and safety.

Diver-related risk factors

The diver in our case was 45 years old; his height was 188.0 cm, weight 75 kg, which resulted in a BMI of 21.2. This suggests a normal frame.

Advancing age may increase a diver's risk of DCS, as age is statistically correlated with venous gas emboli (VGE) [4,5].

During his medical assessment two years prior, the subject underwent a bicycle ergometer test with direct maximal oxygen uptake measurement. His maximal load was 225 Watts, with a maximal heart rate of 163 beats/minute and a maximal oxygen consumption of 2973 ml/minute, which resulted in a $VO_{2\ max}$ of 42.2 ml/kg/minute. Divers who are physically fit have a lower risk of DCS since physical fitness is correlated with VGE [4,5]. The guideline from the European Diving Technology Committee states that the physical condition $VO_{2\ max}$ should be 40 mlO_2/minute/kg [6]. This is the latest standard of technology and science and is accepted as such in occupational health and safety legislation in the Netherlands.

The subject's lung function test resulted in a FVC (l) = 6.78 (predicted = 5.42), FEV1 (l) = 4.51 (predicted = 4.41), FEV% = 64 (predicted = 80). Divers frequently

have unusually large lung volumes associated with a low ratio of FEV1 to FVC (FEV1%) suggestive for obstructive airway disease. A study by the Israel Naval Institute shows that a low FEV1% in a diving population does not necessarily indicate obstructive airway disease, but may simply reflect the phenomenon of divers' large lungs [7]. The researchers suggest that large lungs represent part of the natural selection of divers, rather than a training-related effect. Prolonged diving experience may result in the development of minor airway disease, which can increase the risk for barotrauma.

The diver was a smoker, using 20 cigarettes a day for 10 years. The available evidence consistently shows that smokers are at a higher risk of decreased FEV1 both in cross-sectional and longitudinal studies with an absolute declination of FEV1. There is also consistent evidence about a dose-response relationship between the amount of smoking and the decline in FEV1 [8]. In addition to its impact on lung tissue, smoking affects the walls of blood vessels. It also initiates airway remodeling, involving reticular basement membrane fragmentation and altered vessel distribution [9]. Venous bubbles may cross the lungs through large extra-alveolar vessels, or via the pulmonary capillaries themselves [10]. Smoking might stimulate bubble formation and the transpulmonary passage of venous emboli [11].

Due to the subject's smoking habits the diving physician had requested a regular inspiratory chest X-ray in 2006. This showed a normal thorax skeleton with no abnormalities in the lungs. No nodular or infiltrative processes could be identified. Furthermore, no bullae or blebs were detected. The chest x-ray showed no signs indicating a predisposition to pulmonary barotraumas.

The diver had suffered a herniated lumbar disk 10 years earlier. Intervertebral disk abnormalities might be predictors of negative clinical outcome in subjects presenting with spinal cord DCS with positive MRI findings [12]. Disk herniation or spinal canal narrowing could affect spinal cord offgassing by altering epidural venous drainage, thus inducing venous stasis and subsequent ischemic myelopathy. The neurological manifestations initially observed in spinal cord DCS are frequently accompanied by acute back pain. It should emphasized that this symptom is generally followed by aggravation of the disease, and that it can be predictive of poor clinical outcome [12].

Dive related risk factors

The work in question involved covering an underwater oil pipeline with concrete mattresses. The final dive involved no physical exertion.

The diver's logbook showed that he had been diving once daily for the preceding four days, with a minimal surface interval of 16 hours between dives. The preceding dives involved the same dive profile, but they were described as very exhausting dives involving considerable physical strain. Exercise during pressure-exposure increases the inert gas load, and raises the risk of DCS [3]. However, the decompression tables do not technically consider a surface interval of 16 hours to be a repetitive dive calling for additional decompression.

Treatment

Decompression illness (DCI) should be treated with hyperbaric oxygen. USN Table 6 is commonly used for this purpose [13,14]. The use of decompression tables with higher pressures in combination with helium, (*e.g.*, Comex CX30), rarely alters the clinical course or produces clinically significant improvements [14]. There was no reason to deviate from standard recompression treatment in the current situation.

The diver was hydrated with standard saline infusion. In addition, local institutional policy prescribed administering a daily intravenous dose of 100 mg lidocaine to help counteract sludging, to induce an anti-inflammatory effect on tissues and reduce neuronal metabolism [15]. However, the extraordinarily adverse signs during the course of DCI as presented in this case are associated with poor outcome, regardless of the choice of treatment undertaken [16].

Risk factors – Summary

The dive itself cannot be regarded as a high risk dive. As discussed above we considered the heavy physical strain during the previous dives, the subject's advanced age (45 years), his smoking habits (20 cigarettes a day for 10 years) and an earlier herniated disk to be potential risk factors.

Recommendations for practice

We have referred to potential causal risk factors in the present accident. A single case does not warrant conclusive procedural changes, but we would like to address some factors that could affect occupational health and safety for divers.

Based on findings in the literature and in this case we feel that a history of herniated lumbar disk and smoking habits should be considered as risk factors during medical assessments, particularly among divers of advanced age.

After studying the existing evaluation forms in this case, we have the impression that communication between diving physician and diving supervisor was less than ideal due to the hectic nature of the situation. Radio or telephone consultation is standard procedure in the event of decompression incidents. We would like to suggest the use of a combination of verbal and written communication lines between the dive medical physician and the medical supervisor, to ensure that messages are fully understood. It can also be used as an evaluation tool, to learn why particular decisions were made and what can be learned for the future.

Research recommendation

We propose that further research be carried out involving a case control study model, with an emphasis on a history of herniated disk. This should provide more information about the factors that might be predictive for a spinal cord DCS, and promote a better understanding of the best treatment results that can be obtained under a given set of circumstances. A multicenter research trial might also be considered that would bring together rare cases at more than one medical center.

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