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SEVERE DECOMPRESSION SICKNESS IN A DIVER IN THE COURSE OF A TRAINING DIVE

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ABSTRACT

The article describes the case of a neurological form of decompression sickness, which occurred in the course of a short training dive to the medium depth using the air as the breathing mix. The subject was treated according to the 3rd air recompression table. Despite an immediate hyperbaric treatment followed by hospitalisation residual neurological symptoms remained throughout the entire observation period.
Key words: decompression sickness, neurological form, alcohol, case study.

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INTRODUCTION

The growing interest in underwater space along with the rapid development of the diving technique is unfortunately linked with the ever increasing incidence of diving sicknesses and accidents.

The risk of decompression sickness (DCS) is on the basis of the thus far obtained statistical data estimated at approximately 5% of cases in the total number of hyperbaric exposures [2,5,14,15]. In this group ca. 10% of cases is concerned with the neurological syndrome [2,9,10].

The occurring neurological lesions in severe decompression sickness are commonly located in the spinal cord in the Th_6-L_2 section at the border line between white and grey matter of anterior horn [1,4,7], i.e. the most poorly vascularised area of the spinal cord. The above phenomenon, besides the small number and diameter of the blood vessels, is explained by a relatively high lipid content in the nervous tissue of the cord, as well as gas diffusion from neighbouring tissues (1,3,12,16). The scope of damage and the dynamics of lesions in the nervous system in the course of this type of decompression sickness is highly unpredictable due to the fast occurring distribution of generated gas bubbles, usually as a result of an improperly performed diver decompression [7,10,11,16].

The clinical course of spinal cord injury syndrome in the neurological form of the decompression sickness is usually manifested with pain in the lower part of the chest and the lumbosacral area, parestesia, sensory function impairment, paralysis of extremities, usually lower. Moreover, particularly in untreated cases, it is possible to observe pathological tendon and periosteal reflexes, as well as Babiński and Rossolimo symptoms [4,7,13,14,15,16].

Immediate application of proper recompression treatment in a hyperbaric chamber usually leads to remission of the occurred lesions and re-establishment of the disturbed functions of the musculoskeletal system. Nonetheless, in numerous cases despite the application of causal treatment the persistence of small neurological disturbances is noted in the form of the so-called residual symptoms, which commonly recede within the period of 6-8 weeks [5,15,16,20]. Still, it is possible that they will remain permanently as long-term implications of the decompression sickness.

CASE DESCRIPTION

On 17 July 1975 the Neurological Ward of the Naval Hospital admitted a diver, M.G., at the age of 20. According to the interview, on 16 July 1975 at 11.00 a.m. following a medical examination the patient was qualified to perform a training dive at sea in concord with the training programme.

The meteorological conditions in the diving area from the board of a rescue vessel were defined as good: air temperature 22°C, water temperature 19°C, with sea state of 1.5 on Beaufort scale. The diver's task included: descent to the depth of 31 m and performance of work defined as light. The diver remained approximately 9 minutes at the said depth and underwent the decompression procedure according to the Naval tables for the depth of 33 m and the time of 5 minutes. The total stay time under pressure amounted to 23 minutes. Upon the lapse of ca. 10 minutes from dive completion the patient began to feel mild pain in lower part of the chest and the lumbar area, which after another 5 minutes became accompanied by a weakening of the muscle strength of lower extremities (he was unable to stand up).

Due to above symptoms, he reported himself to the physician securing the diving activities, who recognised the decompression sickness and recommended immediate recompression in the chamber located on-board the ship according to recompression table III [9]. During the compression at the pressure of 2.8 ata in the chamber the subject felt a visible relief, whereas at 7.2 ata the pain symptoms and the weakness in the muscle strength of lower extremities completely receded.

In the process of decompression, upon the lapse of approximately 20 hours from symptoms occurrence the diver presented urine retention. For this reason, the physician supervising the treatment and remaining in the chamber with the diver performed bladder catheterization. Following the treatment completion the patient felt quite well. Due to the occurrence of urination disorders he was referred to hospital for the purpose of further observation and possible treatment. As reported by the patient, already in the transport to the hospital the previous symptoms began to reoccur, although in a less intense form.

Extremities – active movements (the patient lifts the limbs by himself to 45°, bent in knees), muscle strength slightly reduced. Spastic muscle strain. Knee reflexes – superficial, ankle jerk reflexes even, live, plantar reflex – reduced. Bilaterally positive Rossolimo symptom. Babiński symptom noted on the left side (positive). Other tests: heel-knee test, the patient was unable to reach the knee. Moreover the patient was unable to urinate. Minor leukocytosis was confirmed with additional tests performed on the patient's blood at the Neurological Ward (10,000 in 1 mm3), OB 17 after 1 hour and approx. 50 erythrocytes in the field of vision.

In the interview the patient also reported that the day prior to the performed dive he had drunk a considerable amount of vodka with his colleagues (approximately 300 g each), which he failed to mention during the medical examination prior to the dive.

On the basis of the interview and the examination, the patient was diagnosed with the symptoms of spinal cord injury as a consequence of the past decompression sickness treated with recompression.

Rest and pharmacological treatment was applied (Hydrocortison, Lignocainum, Cocarboxylase, Sadamin) and bladder catheterization for the period of 3 days due to sustained urination disorder.

As a result, on the 4th hospitalisation day at the Ward the patient began to pass urine and stool. Following 3 weeks of treatment, due to a certain improvement in the health condition the patient was discharged from the Hospital and referred to Military Medical Commission with the request for granting him a 30-days' sick leave.

After the lapse of another 2 months of observations at the unit the patient was once more referred to make an appearance before the Military Medical Commission for the purpose of determination of his further capability to perform military service. On the basis of the conducted examinations it was determined that due to the sustained lesions in the neurological examination confirming permanent injury of the spinal cord in the thoracic section and sensory impairment – as

a consequence of severe decompression sickness – the patient was permanently incapable of performing military service.

Upon dismissal from military service the subject underwent further treatment at a Neurological Clinic, where, as he reported, the lesions receded to a large extent only when approximately 8 months from the accident elapsed. 2 years after the accident the patient reported that the only remaining symptoms concerned disturbances in superficial sensation of the left thigh, with no other detectable neurological changes.

DISCUSSION

When diagnosing the clinical picture of the case, on the basis of the observed neurological changes an organic injury to the spinal cord in the thoracic-lumbar section was confirmed as a result of the decompression sickness developed following a diving activity. The said diagnosis was confirmed with the presence of pathological tendon and perioteal reflexes, positive Babiński symptom on the left side, bilateral positive Rossolimo symptom, as well as sphincter functions disorders. The above symptoms point to pyramidal tract damage [8].

Despite the application of medical recompression immediately after an occurrence of the first symptoms, during a longer time of out-patient treatment the diver manifested persistent residual neurological symptoms, which nearly completely receded approximately 8 months after the incident.

Therefore, it seems that the diagnosis regarding a severe case of decompression sickness should be extremely careful, which is undoubtedly significant for certification.

The cause of sickness occurrence in the discussed case can be sought in the excessive alcohol consumption prior to the day of dive performance. This had a serious effect on the diver's condition and physical fitness, as well as organism desaturation from nitrogen dissolved in the tissues. Many authors point to alcohol as a factor conducive to an occurrence of decompression sickness, thus in the light of the effective regulation its abuse prevents divers from performing diving activities [17,18,19,21].

Moreover it should be emphasised that as certain data show the said sickness commonly occurs in middle-aged divers with a long-term experience in working under water [6]; the discussed case concerned a young subject undergoing training within the diving service. Hence, we can presume that the mentioned fact had a significant influence on the course of treatment [16].

CONCLUSIONS

- Alcohol can constitute a factor conducive to an occurrence of decompression sickness.
- A severe case of decompression sickness requires immediate recompression in a chamber equipped with an oxygen installation.
- During recompression performed on a subject diagnosed with severe decompression sickness the presence of a physician is indispensible.

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