Complicated decompression illness over-the-phone diagnosis, hyperbaric oxygen therapy delay at several days, favorable outcome (diving telemedicine clinical case study)

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Telemedical support for diving operations, case study. Late diagnosis and HBO2 therapy delay at several days in DCI complicated with Inner Ear disorder. An unusual decompression sickness manifestation, previously the Inner Ear ICD has been believed to accompany severe forms of the disease, or follow deep dives. This time the Inner Ear ICD followed not deep dives and was not accompanied with general severe symptoms, it followed repetitive multiple dives to 20-30 m of water. The victim called the doctor only a few days after the onset of the disease, for a long time he refused an appropriate treatment. Treatment in the pressure chamber — Hyperbaric Oxygen Therapy (HBO2 Therapy) — started on the 4th day of the disease. However, a very good result was obtained with favorable outcome. The case illustrates the modern features of medical care for occupational divers and for workers engaged in other activities under high pressure in Russia, it demonstrates implementation of HBO2 treatment for caisson disease (decompression sickness) in the specialized hospital monoplace HBO2 chamber with maximum pressure not exceeding 20 m of water (3 ATA). This is an example of successful “telephone treatment” — sometimes this option may be the only one possible in vast Russian territories.

Keywords: telemedicine, occupational diseases in divers, diving medicine, decompression illness (DCI), hyperbaric oxygen therapy (HBO2 Therapy).

Introduction

Existing perceptions of the prevalence and severity of various forms of diving-associated disease and injury are consistent with 30-50 years old industrial realities and equipment type. Due to changes in underwater technology, there has been a shift in the patterns of symptoms, incidence, and severity of syndromes, and these changes will increase in the upcoming years. It is reasonable to introduce diving specifics to a wide scope of general practitioners and to help them understand diving physiology, hazards, and health problems in people engaged in underwater work. Recreational diving growth will contribute to this knowledge relevance.
Clinical case study

September 14, 20… a qualified occupational male diver Mr. X., a freelance contractor N employee (the city of K., Russia), sought advice at the 24-hour medical consultation service. Mr. X. declared to have all the necessary diving training, and to be familiar with diving physiology basics and diving safety issues.

Mr. X. had made daily dives to 12–15 m underwater for several weeks, breathing air in a hard-hat rig switched to an open-circuit mode. Deco-computer had been used to plan and to control all dives, and it showed all dives to be no-decompression dives, requiring no deco stops (decompression-free dive profiles). On the last day — September 10 — Mr. X. made three consecutive dives to 30 m, and by the evening the same day he felt weakness, unpleasant sensations in the head, and slight dizziness.

Fearing specific decompression disorders, Mr. X. had tried the so-called test trial-dive to a depth of 5 m and noted a short-term relief of all symptoms. His subsequent strong alcohol intake (about 100 g of vodka) did not improve the situation. He remained feeling stable dull pressure, itching deep in the head, radiating into the ear. No skin changes were present, neither pain or any discomfort in the joints or limbs, except for an intermittent itching sensation inside the left heel.

Mr. X. assumed himself to be hit with caisson disease (decompression sickness) and he turned to the emergency room of a local hospital. He was offered hyperbaric oxygen (HBO2) treatment in a monoplace chamber. Since the maximum treatment pressure attained in a monoplace oxygen chamber corresponds to only 20 m of water (3 atmospheres absolute, ATA), Mr. X. refused the treatment offered. He had sought for treatment under a pressure of 100 m (11 ATA), as prescribed by Russian diving safety regulations. As a result, Mr. X. remained without medical care for several days: he considered the treatment available as inadequate and therefore refused to undergo it. Other modes of pressure therapy, which Mr. X. wanted to get, were not available in the city of K. By the end of the fourth day of the unsuccessful search Mr. X. phoned our consultation service.

According to the patient he had perfect general health status and had never been ill with specific diving-associated diseases before. In all four days that followed the incident, Mr. X.’s complaints and symptoms were virtually unchanged, limited to slight dizziness, unpleasant sensations in the head and in the deep parts of the left ear that resembled pressure and scratching, as well as itching in the deep parts of the left heel. His voice over the phone was ordinary and sonorous; there was no shortness of breath. Mr. X. noted normal pulse rate and blood pressure, stool and urination unchanged, and no skin symptoms of pruritus or mottling, no paresthesia, and no skin sensation disorders.

The duty Doctor-Consultant assumed Mr. X. had Inner Ear Decompression Disorder — the so-called decompression Ménière-like syndrome of mild severity (ICD-10 Codes: T70.3† H82*). Diagnostic considerations included specific diving-associated injuries, and acute and chronic diseases not linked to diving. The diagnostic conclusion was based on the following. 1) Direct temporary link of unpleasant sensations with under water activity. 2) Disease clinical and pathophysiologic patterns well matched with the incident delineation. 3) Positive trial recompression test.

Mr. X. has been given the following recommendations.

1. Visit neurology clinic and obtain ENT consultation in order to objectify the disease pattern and to monitor subsequent changes. These specialists should verify focal
brain and spinal cord lesions, and inner ear deficiency; they are likely to prescribe MRI or CT, depending on examination results.

2. Contact the local hospital HBO2 department equipped with monoplace HBO2 chamber with maximum pressure not exceeding 20 m of water (3 ATA). A course of HBO2 therapy is essential, the patient needs a series of daily treatment sessions with a total number of 10 at least, each session of 45-60 minutes duration, and two or three sessions are eligible on the first treatment day. The first 3–5 sessions should attain the highest possible oxygen partial pressure (up to 3 bar, which corresponds to breathing pure oxygen at the depth of 20 m of water), parameters for subsequent follow-up treatment sessions — at the discretion of the doctor — barotherapist.

3. Time to return to diving work will depend on the results of treatment and the degree of residual symptoms. A pause of two weeks to one month will be most suitable. It will be reasonable to get advice of diving physician before resuming under water activities.

4. While continuing a career in diving, it will be rational to undergo special medical test for patent foramen ovale.

We reviewed long-term results in a month and a half. As the patient himself was not available for an interview, we had to ask his close friends. From their words Mr. X. had not had contacted specialist doctors — neurologist, ENT, — had rejected the opportunity to undergo MRI or CT, and immediately after the telephone consultation Mr. X. had gone straight to the HBO2 hospital department, and had received 10 daily sessions of HBO2 therapy in the specialized hospital monoplace HBO2 chamber with maximum pressure not exceeding 20 m of water (3 ATA). The results of treatment were satisfactory: complaints and all symptoms had disappeared; Mr. X. had recovered in full, and he was fit for his occupation. By the time of our investigation (the interviews with his friends), Mr. X. continued to work as a diver and went on a business trip to the remote northern region of Russia.

Discussion

The essence of health problems in Mr. X. was decompression gas production. While diver descends under water, gas molecules of the breathing mixture saturate the human body. Special physiological mechanisms control the oxygen and the carbon dioxide concentrations; oxygen and carbon dioxide variations with pressure changes have little effect on decompression gas formation. The case is different with so called inert gases, e.g. nitrogen. The deeper and the longer descent accumulates more nitrogen in the diver’s body, and on the way back to surface excess nitrogen needs to leave the body. The desaturation process has a finite speed. The diver has to rise from bottom to surface slowly, through a series of specially calculated deco stops. Otherwise at a high rate of ascent the rapid release of excess nitrogen would lead to the bubbling of blood and tissues, as nitrogen molecules would not have time to leave the body by diffusion through natural barriers (lungs, skin, mucous membranes) and they would crowd in gas bubbles.

In Mr. X. decompression bubbling has happened in the endolymph cavities and in the inner ear channels. This kind of disorder for many years was thought to accompany severe forms of decompression sickness, and to complicate diving descents to great depths. In recent years, many diving-associated Ménière-like syndrome cases have been linked not to
great depths and not to severe forms of DCS [1]. Nowadays these incidents tend to happen against the background of air breathing dives to relatively small depths (up to 30 m), and these dives are done in a series day by day and sometimes they are multiple dives in one day. At the same time, the parameters of each dive usually remain within safe limits with no obvious deviations from decompression rules and modes.

The reason of the case discussed is related to intricacy of the inner ear desaturation. The inner ear elements are “slow tissues” and do not absorb and get rid of excess nitrogen easily and quickly. In a single short dive slow tissues accumulate very few nitrogen, and this involves low risk of decompression bubbling when the diver comes to a surface... However, when dives are repeated day by day, and even more so with multiple dives in one day, low rate of nitrogen elimination contributes to retention of residual nitrogen in the inner ear. And although endolymph accumulates very few nitrogen with each dive, by the beginning of the next consistent dive, excess gas does not have time to exit [2]. As a result, the nitrogen piles up in the inner ear, and sooner or later amassed nitrogen may be enough to cause decompression bubbling. Moreover, the disorder is localized, i. e. limited exclusively to slow tissues of inner ear, because in other parts of the body nitrogen saturation and desaturation go faster, and residual nitrogen does not accumulate so significantly.

The case illustrates the modern features of medical care for occupational divers and for workers engaged in other activities under high pressure in Russia, it demonstrates implementation of HBO2 treatment for caisson disease (decompression sickness) in the specialized hospital monoplace HBO2 chamber with maximum pressure not exceeding 20 m of water (3 ATA) [3].

The patent foramen ovale and its role in diving associated injuries has been a hot topic in diving medicine in recent years, PFO is thought to be imperative protagonist in DCS mechanics [4]. Since atrial communication is considered an important player in the game of bubbling disorders, it is reasonable to recommend all DCS victims who desire to continue diving career to undergo a special examination to identify the right-left intracardial shunt and to evaluate the feasibility of its correction. This was the recommendation issued to diver X.

Conclusions

1. Diving related injuries and diseases often tend to display polymorphous symptoms and complaints. The diagnosis should be based on diving activities preceding the development of a health disorder, as well as on association of leading syndromes and symptoms with pathophysiology of decompression or barotrauma.

2. The most important treatment modality for specific diving diseases and injuries is recompression in barochamber (barotherapy). Unfortunately, nowadays recompression regimens involving the maximum chamber pressure corresponding to 50–100 m of water (6–11 ATA), which have been considered extremely efficient in Russia, are very often impossible to implement due to several reasons. Under the circumstances, the only treatment method available is hyperbaric oxygen therapy (HBO2 therapy) in a monoplace chamber with maximum pressure not exceeding 20 m of water (3 ATA), available in many hospitals providing emergency medical care. Routine medical practice demonstrates good results when affected divers get treatment with HBO2 therapy in a monoplace chamber.
References


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