

Sleep Paralysis and Decompression
SicknessOrtega-Albás JJ^{1*}, Gomis Devesa AJ¹, Martínez A^{1*}, Salvador-Marín M²¹SleepUnit, University General Hospital, Castellón, Spain²HyperbaricTherapeutic Unit, University General Hospital, Castellón, Spain

Article Information

Received date: Mar 04, 2016

Accepted date: Apr 30, 2016

Published date: May 03, 2016

*Corresponding author

Juan José Ortega Albás, SleepUnit,
University General Hospital, Castellón,
Spain, Email: jjoral@ono.com

Distributed under Creative Commons
CC-BY 4.0

Abstract

We are presenting the case of a 38-year-old patient with no significant medical history. He used to work as a professional diver but currently only partakes in recreational diving activities. Thirteen years ago, the patient reported self-limited para-paresis (24 hours) after diving, which improved after administering nor-mobaric oxygen and corticosteroids. This process was diagnosed as probable neurological Decompression Sickness (DCS).

Case Report

The patient went to the hospital after controlled diving reporting severe pain of sudden onset in the upper thoracic region, dysesthesia in the trunk of the body and right crural para-paresis. The medical examination showed bilateral and symmetric hypoesthesia with a clinical sensory level between the fifth and sixth thoracic vertebrae, absence of abdominal-cutaneous reflexes and present and symmetrical osteotendinous reflexes. The diving manoeuvre lasted for fifty minutes at a depth of up to twenty-five metres, stopping for three minutes at a depth of five metres.

The analytical study, electrocardiogram, chest X-ray and brain and spinal Magnetic Resonance Imaging (MRI) with and without injected contrast were all normal.

The supra-aortic trunk echo-Doppler showed a spontaneous pattern, as well as a “shower-curtain” pattern following application of the Valsalva manoeuvre.

A contrast transoesophageal echocardiography was performed after intravenously injecting physiological saline solution, revealing bubbles and a right-to-left shunt in the first heartbeats after the injection. Although small, the shunt was sufficiently large enough to reveal the presence of a Patent Foramen Ovale (PFO).

Several sessions were completed in a hyperbaric chamber (USN Table 6), resulting in the patient’s significant clinical improvement and discharge from hospital, completely asymptomatic, four days after admission.

The sum of the atmospheric and hydrostatic pressures during diving contribute test to the increased dissolution and transport of gases (particularly nitrogen) to the tissues. During the ascent to the surface when the environmental pressure decreases, excess gas is removed from the tissues by means of exhalation. However, inert gas supersaturation can manifest if the variation in environmental pressure occurs faster than the elimination of nitrogen from the tissues, which is the main cause of DCS.

At the same time and on successive days, the patient started manifesting nocturnal awakenings, where no motion other than eye movement could be perceived. Respiration was normal. Most of these acute episodes were long lasting (up to 45 minutes) and hypnopompic. There recovery from this “sleep-paralysis status” was gradual, progressive and from proximal to distal, starting with movement of the neck and finally the lower limbs. No visual, tactile or experiential hallucinations were referred, nor did the patient experience excessive day time sleep illness or sleep attacks. HLA DQB1*0602 typing was negative.

No specific treatment was needed to achieve a spontaneous and progressive clinical improvement, experiencing increasingly shorter and less frequent episodes.

Muscle atonia, a characteristic of REM sleep, is triggered by the glycinergic inhibition of the motor neurons of the spinal anterior horn. When the “REM-generating neurons” are active, their axonal projections excite the glycinergic spinal neurons located inside the paramedian, magnocellular and gigantocellularthalamic reticular nucleus, via a glutaminergic transmission pathway. At the same time, these spinal neurons send their axonal sprouting directly to the motor neurons located in the brain-stem and the spinal-cord, precisely where glycine is released. As a result, inhibitory postsynaptic potentials are generated in REM sleep that ultimately open the channels and induce

changes in the cellular membrane with hyperpolarisation of the motor-neurons and ultimately somatic muscle atonia [1]. Recently, an additional cholinergic mechanism has been shown to be involved in the control of muscle atonia in REM sleep [2].

Isolated sleep paralysis is characterized by a short period of time (several seconds to minutes) where the movements of the voluntary muscles are inhibited [3] but the eyes and respiratory muscles remain active. It is usually recurrent, does not have any hypnagogic, hypnopompic or hypnometric preferences and may occur after awakening during the REM sleep phase [4]. Its pathophysiological mechanism continues to be debated [5]. In this case report, hypnopompic predominance, long duration and recovery from proximal to distal are all remarkable events not often found in isolated and recurrent sleep paralysis.

Patients developing DCS related to PFO tend to have earlier symptoms once they get to the surface after immersion. Spinal DCS represents about 50% 60% of all cases [6] and it may involve both venous and arterial circulation (anterior spinal artery).

In this case report, the paraparesis as well as the clinical thoracic sensory level suggest spinal damage at the upper thoracic level, while the sleep paralysis may be related to the medulla oblongata.

To the best of our knowledge, this is the first published case of sleep paralysis as a clinical expression of Spinal Decompression Sickness (DCS).

References

1. Chase MH. Motor control during sleep and wakefulness: clarifying controversies and resolving paradoxes. *Sleep Med Rev.* 2013; 17: 299-312.
2. Torontali ZA, Grace KP, Horner RL, Peever JH. Cholinergic involvement in control of REM sleep paralysis. *J Physiol.* 2014; 592: 1425-1426.
3. Sharpless BA, Barber JP. Lifetime prevalence rates of sleep paralysis: a systematic review. *Sleep Med Rev.* 2011; 15: 311-315.
4. Girard TA, Cheyne JA. Timing of spontaneous sleep-paralysis episodes. *J Sleep Res.* 2006; 15: 222-229.
5. Brooks PL, Peever JH. Identification of the transmitter and receptor mechanisms responsible for REM sleep paralysis. *J Neurosci.* 2012; 32: 9785-9795.
6. Rosińska J, Ąukasik M, Kozubski W. Neurological complications of underwater diving. *Neurol Neurochir Pol.* 2015; 49: 45-51.