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Recompression Therapy Under Emergency Conditions: A Case of Type II Decompression Sickness Acil Durumlarda Rekompresyon Tedavisi: Tip II Dekompresyon Hastalığı

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ABSTRACT

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Decompression sickness presents with a great variety of signs and symptoms that might affect any part of the human body including joints, skin, cardio respiratory and nervous systems. In this manuscript, we reported a case of severe neurological Type II Decompression sickness who received recompression therapy. A 53-year-old man presented at the emergency service of our hospital with history of diving in the Black Sea. He had made an uncontrolled ascent at the final dive. The patient arrived at the emergency service with loss of consciousness. A physical examination revealed paresthesia of the left arm and hand after he had regained consciousness. Anal sphincter tonus was reduced. The patient had urinary and fecal incontinence as well as a characteristic rash (cutis marmorata) on the body. After the initial treatment the patient was transported to the Hyperbaric Unit where he recovered completely after administration of hyperbaric treatment. Emergency treatment of this potentially life-threatening disease includes providing basic life support, horizontal positioning of the patient, administration of 100% normobaric oxygen, followed by an early transfer to the nearest hyperbaric facility for definitive recompression treatment to prevent serious neurological sequelae.

Keywords: Scuba diving, decompression sickness, emergency recompression, hyperbaric oxygenation, air embolism Received: 06.04.2011 Accepted: 24.06.2011

ÖZET

Dekompresyon hastalığı eklemler, cilt, kardiyorespiratuvar sistem ve sinir sistemi gibi vücut kısımlarını etkileyebilen çok farklı semptom ve bulgularla ortaya çıkar. Bu yazıda rekompresyon tedavisi uygulanan ciddi nörolojik Tip II Dekompresyon hastalığı olan bir olgu sunulmuştur. Elli üç yaşında bir erkek hasta Karadenizde dalış yapması sonrasında bayılma nedeniyle acil servise getirildi. Olgu son dalışında kontrolsüz yükselmişti. Hasta acil servise getirildiğinde bilinci kapalıydı. Bilinci acıldıktan sonra fizik muayenesinde sol kol ve elinde parestezi mevcuttu. Anal sfinkter tonusu azalmıştı. Hastanın idrar ve gayta inkontinansıyla beraber vücudunda karakteristik raş (kutis marmoratus) vardı. Hasta başlangıç tedavisinden sonra hiperbarik tedavi merkezine sevk edildi. Yaşamı tehdit edici bu hastalığın acil tedavisi temel yaşam desteği uygulamaları, hastanın horizontal pozisyonda tutulması, %100 normobarik oksijen uygulaması ve ciddi nörolojik sekelleri önleyecek rekompresyon tedavisinin yapılacağı hiperbarik tedavi merkezine sevkini içerir.

Anahtar Kelimeler: Tüplü dalış, dekompresyon hastalığı, acil rekompresyon, hiperbarik oksijenasyon, hava embolisi Geliş Tarihi: 06.04.2011 Kabul Tarihi: 24.06.2011

Introduction

Decompression sickness (DCS) might occur in various contexts including SCUBA diving and flight in a non-pressurized aircraft (1). DCS is caused by injury secondary to nitrogen bubble formation in the circulatory system and tissues. The clinical presentation of DCS depends on the degree and localization of these nitrogen bubbles. In addition, the depth and duration of diving are also of critical importance. Symptoms of DCS are classified as type I and type II indicating simple and severe disease, respectively. Cerebral arterial gas embolism (CAGE) should also be considered in the differential diagnosis of DCS (2).

The incidence of Type II DCS is about 62% in all diving-related illnesses (3). In this manuscript, we report a case of Type II neurological DCS with good recovery following hyperbaric recompression therapy.

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Case Report

A 53-year-old man presented at the emergency service of our hospital with a history of diving in the Black Sea in July 2010 to collect fresh mussel. The patient had dived seven times for 60 minutes at 25 meters in one day. He had made an uncontrolled ascent at the final dive and missed his decompression safety stop. The patient arrived at the emergency service with loss of consciousness.

Glasgow coma score (GCS) improved from 10 to 15 shortly after the patient regained consciousness. The patient remained confused and disorientated with bilateral upper and lower limb pain plus numbness. Physical examination revealed paresthesia of the left arm and hand. Anal sphincter tonus was reduced. The patient had urinary and fecal incontinence as well as a characteristic rash (cutis marmorata) on the body. Vital signs were normal and no other findings were determined on physical examination. Computed tomography examination of the head, plain chest X-ray, arterial blood gas, complete blood count and biochemical parameters were normal. A urinary catheter was inserted and two intravenous lines were inserted for physiological saline infusion. A reservoir oxygen mask was used to administer oxygen at 10 Lt/min from a high-flow oxygen circuit. The patient's history revealed that he was a smoker, consumed alcoholic beverages occasionally but avoided this habit before diving. Body mass index was 25 kg/m² The patient reported previous uncomplicated dives since 1985. After the initial treatment, the patient was transported by overland ambulance to Istanbul for hyperbaric unit. He recovered completely after administration of hyperbaric treatment. No diving for one year was suggested to him but he has been diving as from the sixth month and had not experienced any trouble due to diving.

Discussion

DCS might manifest with a wide variety of signs and symptoms and is typically classified as type I or type II. Type I (non-systemic or musculoskeletal) DCS is characterized by the absence of neurological and other systemic symptoms, and usually presents with musculoskeletal symptoms including pain that is often dull or throbbing and poorly localized around a joint-the shoulder and elbow being the most common sites. Rash and pruritis are common cutaneous manifestations; whereas cutis marmorata or skin marbling is also observed, although less commonly (3).

Type II (neurologic or systemic) DCS affects the neurological, vestibular, or pulmonary systems. Neurological involvement is most common among professional divers and might be caused by either spinal cord or cerebral involvement. Symptoms of neurological involvement include hemiparesis, hemisensory disturbance, dysarthria, vertigo, nausea, dizziness, headache, visual changes, hearing loss, disturbances in consciousness and speech, an inability to concentrate, and even sudden death after ascent. Similar to DCS Type I, the onset is rapid, with half of the patients developing symptoms within 1 hour and 90% of the patients developing symptoms within 6 hours of ascent (4). Obesity, age, tobacco use, alcohol consumption, excessive physical exhaustion during the dive, pre-dive physical condition, dehydration, plane flight after diving and cold environment are considered among factors that create a predisposition for DCS (2). The differential diagnosis of DCS includes stroke, CAGE, spinal cord injury, middle/inner ear barotrauma, and metabolic/ electrolyte derangements. Our case had an altered mental status, bilateral upper and lower limb pain plus numbness, paresthesia of the left arm and hand, reduction in anal sphincter tone, and urinary and fecal incontinence. Therefore we conclude that the case was type 2 decompression sickness.

There are no known drugs to cure or reduce the symptoms of DCS. Emergency treatment of all severe DCS cases on site includes providing basic life support, horizontal positioning of the patient, administration of 100% normobaric oxygen (using a reservoired mask), and intravenous fluid resuscitation regardless of the hydration status (3). Management of DCS involves hyperbaric oxygen treatment, which is best initiated as soon as possible because recovery is improved with early treatment in order to prevent serious neurological sequelae. Our hospital had no HBO treatment facility. For this reason the patient was referred to another hospital. Overland transportation should be preferred; however the cabin pressure should be kept near 1 atm of pressure for air transport of a DCS patient. This generally requires specialized fixed-wing aircraft, or flying at low altitudes (~300 m) in a helicopter or normal fixed-wing aircraft (1).

Conclusion

We reported this case to emphasize the importance of having a good knowledge of the clinical presentation and treatment of DCS which is a relatively uncommon condition. Emergency physicians should note how to contact the nearest available hyperbaric chamber and which vehicle to use for transport since these chambers are of critical importance in the success of DCS treatment.

Conflict of interest

No conflict of interest was declared by the authors.

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