

A tale of three divers: recompression therapy for divers with severe Type II decompression sickness with neurological deficits

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ABSTRACT

Decompression sickness (DCS) is manifested in a myriad of symptoms, and can affect any part of the body. It is attributed to the formation of inert gas bubbles in the blood and tissues. Following a diving incident, the pathogenesis of DCS is a result of mechanical obstruction caused by the inert gas bubbles and the body's immunological response to the bubbles. Neurological DCS may present with unusual sensory/motor symptoms that may lead to paralysis. This report describes three divers who suffered severe neurological Type II DCS and underwent recompression therapy at the Naval Hyperbaric Centre in 2007.

Keywords: decompression sickness, diving complication, hyperbaric oxygenation, neurological deficits, recompression therapy

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INTRODUCTION

Decompression sickness (DCS) is a condition that manifests in a myriad of symptoms that may affect any part of the human body. It is attributed to the formation of inert gas bubbles in the blood and tissues. DCS is a potentially life-threatening disease, often requiring recompression hyperbaric therapy. DCS is further categorised as Type I (limb and/or joint pains or skin rash) and Type II (cardiopulmonary and/or neurological system involvements). The incidence of Type II DCS is about 62% of all dive-related illnesses.⁽¹⁾ Type II neurological DCS may present with sensory/motor symptoms or paraparesis. Another important condition to recognise in the treatment of decompression sickness is cerebral arterial gas embolism (CAGE). CAGE causes severe neurological deficits/coma due to the obstruction of cerebral blood flow from air emboli which lodge distally in smaller arteries and arterioles of the brain. This results in cerebral ischaemia, hypoxia and cerebral oedema. The Naval Hyperbaric Centre (NHC) has been

providing recompression therapy (RCT) as part of the provision of underwater and hyperbaric medicine to both military and civilian divers for the past 30 years. We report three recent cases of severe Type II neurological DCS with good recovery post-RCT at the NHC.

CASE REPORTS

Case 1

Our first case was a 45-year-old Chinese man, an experienced commercial diver with no past history of DCS. He completed a single uncomplicated 37 m dive (20 minutes underwater) and adhered to the decompression schedule as stated in United States Navy dive tables. During the dive ascent, he experienced some left ankle/left wrist pain which he initially attributed to physical exertion. His symptoms did not resolve on surfacing, and he subsequently developed musculoskeletal symptoms of bilateral upper limb pain, left wrist swelling/pain and left ankle pain. He also had suffered from numbness in his left wrist. He was first seen at a local hospital emergency department and was referred to the NHC for further assessment. An examination revealed that his left wrist was erythematous and swollen. He also had paraesthesia on his left C6/C7/C8 dermatomes and a limited range of motion in his left wrist. Generalised tenderness over his bilateral shoulders, elbows, left wrist and left ankle was noted. A diagnosis of Type II DCS (musculoskeletal and neurological) was made on the basis of the presence of peripheral paraesthesia coupled with limb/joint pains. He underwent three courses of uncomplicated RCT (Three × Royal Navy Treatment Table 62 [TT62]) and had complete symptom resolution following his third RCT.

Case 2

Our second case was a 32-year-old man, a commercial diver who presented to a local hospital after a boat salvage dive off Changi Naval Base. He descended to 40 m for 30 minutes and did not adhere to the stipulated diving decompression schedule on ascent. He presented initially with bilateral upper/lower limb pain and numbness. At the

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emergency department, he was unable to ambulate and was noted to have tetraparesis of power 3/5, affecting all four limbs, associated with diminished sensation over the C5–T1 and T10–S4 dermatomes. Anal tone was lax and the patient was unable to void urine. He was given pain relief at the hospital before he was transferred to NHC. A diagnosis of Type II DCS (spinal and musculoskeletal) was made and the patient underwent immediate RCT (TT62).

A review post-recompression showed an immediate improvement in the power of all four limbs from 3/5 to 4/5. He subsequently underwent a total of eight uncomplicated RCTs (seven TT62, one TT61) with concurrent intravenous steroids over a period of eight days, with gradual recovery of symptoms. He regained full motor power in his limbs (except in the left lower limb) and was ambulating after four sessions of TT62. However, he continued to have mild residual weakness of his left lower limb and diminished sensation over left L5–S4 dermatomes despite further RCT. Further investigation at the hospital revealed a significantly prolapsed L4/L5 intervertebral disc on magnetic resonance imaging.

Case 3

Our third case was a 31-year-old Swedish man, a recreational diver with a past history of gastroesophageal reflux disease. He had just embarked on technical diving, (technical dives may be defined as being either dives to depths deeper than 40 m or dives in an overhead environment with no direct access to the surface or natural light. Such environments may include fresh and saltwater caves and the interior of shipwrecks. In many cases, technical dives also include planned decompression carried out over a number of stages during a controlled ascent to the surface at the end of the dive) and completed six days of dives (total of 30 dives). On his seventh day, he completed two Trimix (Trimix is a breathing gas, consisting of oxygen, helium and nitrogen, and is often used in deep commercial diving and during the deep phase of dives carried out using technical diving techniques) dives (67 m) of 25 minutes' duration. He adhered to all the decompression stops and surfaced with no complications. He complained of rashes, dizziness and shortness of breath, which started 20 minutes after surfacing. He also vomited several times. In-water recompression at 72 feet (22 m) was attempted, with marginal relief of symptoms, before he was evacuated straight to the NHC.

Examination at the NHC revealed tetraparesis with reduced motor power of all four limbs (power 4/5) with sensation unaffected. Deep tendon reflexes were normal and plantars were downgoing bilaterally.

The patient complained of diplopia on left gaze with no other significant cranial nerve deficits. He did not exhibit any cerebellar signs or nystagmus. His vomitus tested positive for blood (suspected Mallory-Weiss tear from repeated vomiting). He was diagnosed with Type II DCS (neurological) and underwent two sessions of RCT (TT62) without complications. He regained full motor power as well as resolution of left diplopia post-treatment. He was subsequently referred to a local hospital for the management of his suspected Mallory-Weiss tear.

DISCUSSION

The presentation of Type II neurological DCS is varied and unpredictable. The patterns of weakness in a study of divers with Type II DCS were as follows: paraparesis 27%; paraplegia 26%; lower extremity monoparesis 14%; lower extremity monoplegia 6%; quadriparesis 4%; hemiparesis 4%; hemiplegia 3%; and quadriplegia 2%.⁽²⁾ In the three severe Type II cases reported above, it is noted that they each presented with a unique set of neurological deficits. Clinically, sensory and motor neurological DCS usually present independently, and this is attributed to sensory and motor deficit dissociation in the spinal cord.⁽³⁾ Hence, it is important to recognise the signs and symptoms of decompression sickness and to administer prompt treatment. The differential diagnosis of decompression sickness includes cerebrovascular accident and CAGE.

Emergency treatment of severe DCS on site includes providing basic life support, horizontal positioning of the victim, the administration of 100% normobaric oxygen, followed by an early evacuation to the nearest hyperbaric facility for definitive recompression treatment in order to prevent serious neurological sequelae.^(4,5) The subsequent management of DCS should be guided by repeated clinical neurological examination and assessment of symptoms.⁽⁶⁾ Ball reviewed 49 cases of spinal DCS from a United States naval station and classified them according to severity and time to recompression with oxygen.⁽⁴⁾ A delay in treatment was found in studies to worsen the outcome for severely-injured divers. DCS occurs due to the liberation of gas bubbles following an oversaturation of tissues with inert gas. These bubbles can cause cerebral blood flow obstruction leading to brain ischaemia. Several other mechanisms have been postulated to explain the pathophysiology behind Type II neurological DCS. They include arterial bubble embolism in neural vasculature, epidural venous obstruction leading to infarction and the formation of autochthonous (formed *in situ*) bubbles.

RCT involves the inhalation of 100% oxygen at pressures greater than atmospheric pressure. Inhalation

of pressurised oxygen will bring the arterial partial pressure of oxygen to 1,500 mmHg at a pressure equivalent to two absolute atmospheres. The delivery of high levels of oxygen is important to counteract the ischaemic and hypoxic effects of vascular obstruction. This process will also create a pressure gradient which forces the inert gases back into solution (Fick's Law). According to Boyle's law, the volume of a gas is inversely proportional to the amount of pressure exerted on the gas. RCT reduces the size/surface area of the bubbles to decrease the inflammatory effect at the bubble-blood interface. Using standard treatment tables and adherence to decompression stops, the inert gases are gradually displaced out of the tissues, and eventually transported to the lungs to be removed. Hyperbaric oxygen treatment also helps to relieve hypoxia, improves microcirculation, decreases cerebral oedema by vasoconstriction and prevents secondary brain damage in Type II neurological DCS. Currently, the value of adjunctive medication, such as intravenous steroids, remains controversial.⁽⁷⁾

Currently, besides DCS, hyperbaric oxygen treatment is also approved by the Undersea and Hyperbaric Medical Society (UHMS) as a recommended therapy for conditions such as arterial gas embolism, acute carbon monoxide poisoning, acute necrotising infections, clostridial myonecrosis (gas gangrene), crush injury/compartment syndrome and other traumatic ischaemias, exceptional anaemia resulting from blood loss, refractory osteomyelitis, radiation tissue damage (osteoradionecrosis), compromised skin grafts/flaps and thermal burns.⁽⁸⁾ In conclusion, we have described three cases of severe DCS that responded well to early

RCT. It is important to note that taking a complete diving history and its correlation to the symptoms remains key to establishing the clinical diagnosis of DCS. The favourable response to subsequent RCT further confirms the diagnosis and we need to emphasise that early RCT improves the overall prognosis of DCS.

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