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CASE REPORT

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# Decompression illness type II with stroke: challenging situation in acute neurorehabilitation

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## ABSTRACT

*A professional 55-year-old female experienced diver, who surfaced after the second dive, had a lucid interval before dropping Glasgow Coma Scale (GCS) to 3/15. She was admitted to intensive care unit and commenced on hyperbaric oxygen therapy. Her initial computed tomography of the head was normal but her magnetic resonance imaging of the brain at 48 hours showed extensive bilateral cortical watershed territory infarcts. She developed acute respiratory distress syndrome which resolved within a few days. Her GCS gradually improved from 3/15 to 6/15, was repatriated to United Kingdom after about 2 weeks of the insult and admitted to a tertiary care hospital where she had myoclonic seizures and was started on anti-epileptics. Then she was transferred to the Rehabilitation Medicine Ward of Leicester General Hospital, with GCS 14/15 with poor sitting balance, for her management and rehabilitation. She had weakness of right upper and lower limbs, dysarthria, neuropathic bilateral shoulder pains, pressure ulcer of left heel, bladder and bowel incontinence and cognitive issues. She improved to have significant neurological recovery within next 3 months, became ambulant independently and bladder and bowel continent. Her Barthel index (from 4 to 17), Montreal Cognitive Assessment Test, Adembrook Cognitive Examination and Berg Balance scale (from 33/56 to 44/56) improved significantly. Early diagnosis, treatment and rehabilitation can have a significant impact on the recovery of decompression illness.*

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**Key words: decompression sickness, scuba diving, the bends, hyperbaric oxygen therapy, hyperbaric chamber**

## INTRODUCTION

Decompression illness (DCI) is a clinical manifestation of Henry's Law which states that while ascending up after a dive, reduction in pressure can cause release of dissolved nitrogen into blood and tissues leading to intravascular and extravascular bubble formation [1]. The clinical manifestations can range from mild pains to complete vascular obstruction, respiratory and circulatory disturbances. Management involves securing and maintaining airway, breathing and circulation as well as hyperbaric oxygen therapy which should be instituted as early as possible followed by intensive inpatient rehabilitation.

This report describes a 55-year-old experienced diving instructor who has had more than 1000 successful dives in the past. She met with this fate in spite of the fact that she followed all prescribed procedures required during ascent. This report intends to understand the rare clinical presentation of decompression illness with stroke and to appreciate the importance of early inpatient rehabilitation programme.

## CASE REPORT

A 55-year-old female professional diving instructor, who was a trained and certified diver, left handed, ex-smoker (left 12 years ago), occasional drinker, with no past medical history, delivered two dives while scuba diving in Cyprus. She had been diving for the past 24 years and had a history of more than 1000 successful dives in the past. First dive was 31 m in depth having duration of about 1 h using Nitrox 32% (oxygen enriched air having oxygen percentage as 32% and nitrogen percentage as 68%). She undertook decompression stop for 2 min duration at 9 m of depth and

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for 5 min duration at 6 m depth. This was followed by the second dive which was 30 m in depth, 43 min of duration using Nitrox 34% (having oxygen percentage as 34% and nitrogen percentage as 66%). During this dive the decompression stop was at 6 m depth, for 8 min. She followed all prescribed procedures for a safe ascent while ascending up, and during both dives, she undertook the decompression stops according to the standard guidelines. The surface interval in between the dives was 1 h and 59 min, during which she kept herself well hydrated. She surfaced after the second dive. She was well hydrated before, in between and after both the dives. She had no significant past medical history. There were no technical problems and emergency ascent was excluded. She had a lucid interval of around half an hour before dropping Glasgow Coma Scale (GCS) to 3/15, that is she had no eye opening, no verbal and no motor response. She was rushed to the local tertiary hospital in Cyprus, was intubated and commenced on hyperbaric oxygen therapy (100% oxygen in high pressure chamber). Her initial computed tomography (CT) of the head was normal, while CT head after 48 h showed large left frontal infarct with diffuse cerebral oedema with pressure effects on ventricular system. Her magnetic resonance imaging of the brain showed extensive bilateral cortical watershed territory infarcts, more extensive on left than right, particularly involving pre-motor and primary motor cortex. There was involvement of dorsolateral prefrontal cortex bilaterally. Period of hypotension and presence of recent cerebral oedema could have resulted in further ischaemic injury/watershed infarcts which were not visible on CT head. Extensive bilateral cortical watershed territory infarction was consistent with a moderate hypoperfusion injury and the low GCS. Electroencephalography showed moderate encephalopathy. She developed acute respiratory distress syndrome during the first week of her stay in Cyprus, which resolved within a few days. She was tracheostomised after a week. Her GCS gradually improved from 3/15 to 6/15 wherein she started opening eyes to pain and showed extensor motor response. Her verbal response was incomprehensible sounds or speech. After around 2 weeks of the insult, she was repatriated to United Kingdom, by air ambulance and admitted to neurology department in a tertiary care hospital, initially in intensive therapy unit and after a couple of days was transferred to neurology ward. She underwent around 7 sessions of hyperbaric oxygen therapy (HBOT) according to the established standard protocols. HBOT was initiated with at a depth of around 18 metre sea water (msw). Total duration of the session being around 4 h. Subsequent sessions were of around 100 min duration each at a depth of 14 msw. During the course of her stay in hospital, she also had myoclonic seizures and was started on anti-epileptic drugs in the form of Phenytoin and Levetiracetam. Phenytoin was subsequently tapered off while she was continued on maintenance dose of Levetiracetam 1 g twice a day. She even had episodes of hypotension, requiring fluid resuscitation. She was suffering from hypoxic ischaemic encephalopathy secondary to cerebral decompression sickness causing extensive bilateral watershed territory ischaemic infarcts (left > right) which were likely air embolic. Clinical presentation of the patient at the time of admission to rehabilitation medicine ward comprised of weakness of right upper and lower limbs, poor sitting and trunk balance, dysarthria, moderate to severe expressive and receptive dysphasia with semantic difficulties, neuropathic bilateral shoulder pains plus restriction of range of motion of bilateral shoulders, subluxated right shoulder, mild to moderate dysphagia. Initially she required moderate assistance with bed mobility, transfers, feeding, grooming and upper body dressing, and maximal assistance with lower body dressing and ambulation. In the rehabilitation medicine ward, she underwent a comprehensive rehabilitation programme comprising of active and passive range of motion exercises, stretching and strengthening exercises, retraining of sitting balance, activities of daily living and gait training. On completion of a 4-week course of inpatient rehabilitation, the patient improved to modified independence with all activities of daily living and transfer and required no assistance for ambulation. Her motor strength normalised in the lower extremities and her dysmetria improved. She became continent of both bowel and bladder, and urodynamic study demonstrated normal bladder function. For management of her neuropathic pain in bilateral shoulders, she was prescribed Gabapentin 300 mg up to 3 times a day (gradual escalation of dose). She was even given ultrasound guided steroid injection around the bicipital tendon for her bicipital tendonitis. Apart from regular rehabilitation techniques, she underwent hydrotherapy, dynamic balance training, functional electrical stimulation for right shoulder subluxation, dynamic splinting for improving hand functions, transfer training comprising of hoist transfer, followed by rotunda transfer followed by step transfer, activities of daily living training comprising of large handle cutlery. Outcome measures included were 10 m walk test, Berg Balance Scale, JFK Coma Recovery Scale, 9 hole peg test, Barthel Score. She continued to gradually improve over the next few weeks, became independent in all her activities of daily living and was able to ambulate independently. She became continent in bladder and bowel.

## DISCUSSION

Decompression illness was first reported in 1841, also called Caisson disease. It is common in under water, high altitude events and recreational activities. In spite of a drastic increase in number of recreational dives as well as the

growing popularity of the same, the incidence rate of DCI remains only 0.02% to 0.03% per dive [2]. According to Vann et al. [2], the risk of DCI increases with age and body mass index. Two main mechanisms are thought to be responsible for pathomechanism of DCI. The first one being, acute increase in pressure in the lungs, leading to stretching and rupturing of alveolar capillaries (pulmonary barotrauma), which causes alveolar gas to enter arterial circulation (air gas embolism) [2]. On the other hand, an excess amount of inert gas which is released from the saturated tissues can form bubbles in venous blood. This may cause pulmonary and arterial embolisms due to right to left shunting. Main causes for DCI have been documented to be very rapid ascent and lack of decompression stops [3]. Decompression stops can prevent decompression illness as these delay ascent to the surface and allow inert gases to be eliminated in dissolved form rather than as bubbles [4, 5].

Decompression illness can be classified into two types on the basis of severity and type of symptoms. Type I, the minor type is the 'pain only' DCI, which requires lesser magnitude of recompression therapy. It occurs in 70–85% and is self-resolving. Type II is the DCI which is characterised by neurological symptoms and can even present as shock. Spinal cord lesions are the most common presenting as back pain, paraesthesias, motor weakness, loss of sphincter control. DCI II has worse prognosis and requires greater magnitude of recompression therapy [6]. Air gas embolism on the other hand, occurs immediately after resurfacing and occurs more commonly in brain than spinal cord. This can also cause embolism of coronary vessels leading to arrhythmia and infarction. DCI has several risk factors.

The diving risk factors include, the depth and the duration of the dive, the breathing gas used, the ascent rate and if the dive was conducted at altitude higher than sea level or flying after diving. Among individual risk factors are the quantum of exercise during the dive, older age, higher body fat content, and presence of a patent foramen ovale (PFO) [2]. To be precise, age more than 42 years, depth of dive more than 39 m, having bladder dysfunction, having clinical symptoms before recompression therapy are the factors associated with worse outcome [7].

Patent foramen ovale is associated with severe neurological decompression sickness, inner ear decompression sickness and cutis marmorata. A workshop at the South Pacific Underwater Medicine Society (SPUMS) Annual Scientific Meeting 2014 with representatives of the United Kingdom Sports Diving Medical Committee (UKSDMC) present and subsequent discussions including the entire UKSDMC resulted in a consensus statement according to which right-to-left shunt across a persistent or PFO is a risk factor for some types of DCI. It was agreed that divers with a history of cerebral, spinal, inner-ear or cutaneous DCI, migraine with aura, a family history of PFO or atrial septal defect and those with other forms of congenital heart disease should undergo routine screening for PFO. Screening should be undertaken by bubble contrast transthoracic echocardiography with provocative manoeuvres, including Valsalva release and sniffing. In case of presence of shunt, experienced diving physician should take a decision based on the clinical context and size of the shunt. Transcatheter device closure of PFO may be considered in order to return to normal diving [8].

In the present case report, the patient had hypoxic ischaemic encephalopathy secondary to cerebral decompression sickness type II, causing extensive bilateral watershed territory ischaemic infarcts (left > right) which were likely air embolic. Out of the risk factors, she had older age, higher body fat content and bladder symptoms. As stated by Jüttner et al. [9], treatment of DCI, should comprise of initial normal pressure 100% oxygen inhalation. The two purposes solved by pure oxygen are to improve tissue oxygenation and increase the partial pressure gradient leading to passage of nitrogen out of the bubbles formed during decompression [1].

Furthermore, whether ventilation occurs via invasive or noninvasive means, re-expansion barotrauma may be a contributory factor in DCI, thus peak pressures should be minimized to any extent possible. Additionally, adequate fluid resuscitation should be ensured by emergency department providers to ensure no compounding effects of dehydration or shock physiology [10].

However the mainstay of treatment of DCI remains rapid hyperbaric oxygenation in pressure chamber. This helps to reduce the size of existing bubbles, increase inert gas clearance from tissues and blood [9]. This should be initiated as soon as possible. Recompression therapy should be initiated without waiting for the conclusion of investigations [2]. The fastest available method of transportation is often by helicopter air ambulance. However, the manifestations of DCI may be exacerbated by decreases in atmospheric pressure [11]. In spite of the limited studies available on establishing safe altitudes for patients with DCI, current recommendations include ensuring that the cabin altitude does not exceed 500 feet (152 m) above the departure location [12].

Mechanism of action of hyperbaric oxygen comprises of recompressing bubbles and forcing gas back into solution for a more controlled ascent. Inert nitrogen is replaced by rapidly-metabolised oxygen, and bubbles move either to the lungs where they are excreted, or to smaller vessels where obstruction is less important, and surface tension forces eventually collapse the bubbles. Hyperbaric oxygen also counteracts platelet and leukocyte activation and endothelial interactions [13].

## CONCLUSIONS

Decompression illness is rare with the rate of occurrence estimated to be around 0.03% in recreational divers [2]. Adherence to standard diving guidelines is essential. Regardless of the fact that all diving guidelines being followed, and even if the diver is an experienced one, the medical team should be ready for any emergencies. Early recognition of DCI and its prompt management incorporating early treatment with hyperbaric oxygen and comprehensive rehabilitation is the key to recovery of DCI. First aid treatment comprises of 100% oxygen and definitive treatment is recompression therapy that is HBOT according to established standard protocols. Recompression therapy should be initiated without waiting for the conclusion of investigations.

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