Case Report

Spinal Cord Injury in Decompression Sickness: A Case Report

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Abstract

Study Design: Case report.

Objective: To describe an unusual case of deep diving followed by spinal cord injury due to decompression sickness (DCS).

Setting: Princess Royal Spinal Injuries Centre, Sheffield Teaching Hospitals NHS Foundation Trust, England.

Method: Description and observation of management and outcomes, of spinal decompression sickness (DCS).

Results: The patient's symptoms and signs developed after she surfaced after a deep sea diving event. She was managed and treated in a tertiary level care hospital. MRI performed within 24 hours, showed signs of increased signal intensity in the cervical and thoracolumbar regions. She was treated with hyperbaric oxygen which improved her pain symptoms but there was no immediate resolution in motor sensory deficits. Repeat MRI done after a week showed resolution if hyperintensity in the cervical region but not in the thoracolumbar region. Patient progressed to have significant neurological recovery in the next 6 months. She became ambulant with unilateral ankle foot orthotic and a pair of crutches, she continued to have bladder incontinence at 1 year follow-up interval.

Conclusion: Central nervous involvement is not uncommon in decompression sickness in divers. Early diagnosis and proper management can reduce acute symptoms and prevent further complications of permanent neurological disability. Primary prevention by education and adhering to standard diving guidelines is needed to reduce mortality and morbidity in decompression sickness.

Keywords: **Decompression sickness (DCS), arterial gas embolism (AGE), patent foramen ovale (PFO), divers alert network (DAN).**

A 28 years old trained female certified diver with no significant past medical illness, felt unwell after controlled surfacing from depth of 20 metres. No history of injury during the dive. On surfacing she felt confused and had severe pain in her neck and shoulders. The pain was initially attributed to the heavy diving gear she was carrying but suspicion increased as her confusion continued for about 20 minutes. She progressed on to developed sensory loss predominantly noted on her left side with motor weakness on the right upper and lower limbs. She was unable to sit or weight bear, and developed incontinence of bladder. She was rushed to the local tertiary hospital where imaging with T2 weighted and STIR sequence of MRI revealed high signal intensity in the C2-C6 and T9-T10 cord segments (Fig 1).

Diagnosis of spinal decompression sickness was made. She was treated with hyperbaric oxygen following which there was significant improvement in her pain and alertness but the motor weakness and reduced sensations persisted. Subsequently progressive improvement was noted in the proximal muscle groups in the right upper and lower extremities. Repeat MRI done after ten days showed resolution of the hyperintensity in the T9/T10 segment but findings in the C2-C6 segment remained largely unchanged. MRI of the brain showed focal lesion on the right side of the splenium of the corpus callosum. She was transferred to a rehabilitation unit, where she

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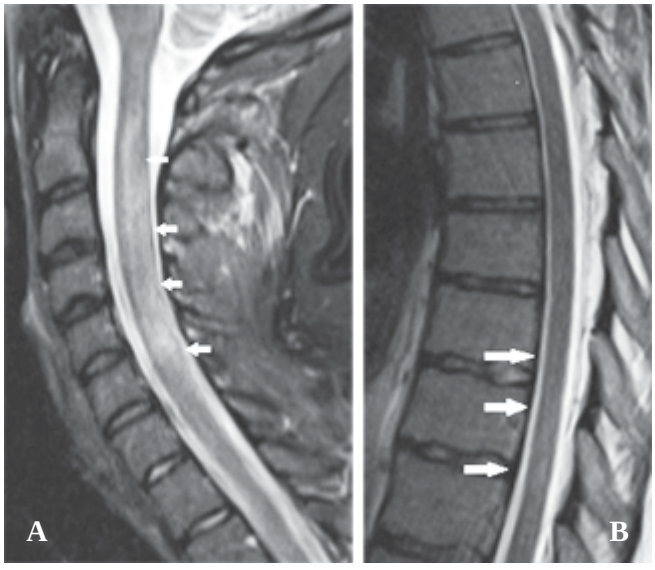

Fig 1- *MRI Showing High Signal Intensity in C_2-C_6 and T_9T_{10} Cord Segments*

continued to neurologically improve over the next few weeks. Following six months from the time of injury she became independent in all her activities of daily living and was able to ambulate with a pair of crutches and right ankle foot orthosis. She remained incontinent and was managing her bladder with clean intermittent catheterisation at discharge but eventually opted to have a suprapubic catheter.

Discussion:

We tend to hear a lot of the word "decompression" following spinal injuries which implies to the fact that the patient has had a surgical decompression of their spinal canal to reduce the impending damage to the spinal cord. Rarely do we hear or see a case of spinal injury and paralysis following decompression sickness (DCS) in our rehabilitation centres.

DCS was first described in 1841 and was commonly called the 'Bends'. DCS is commonly seen in under water and high altitude events and recreational activities, when the individual is subjected to sudden barometric pressure changes. In physics Henry's law states that "At a constant temperature, the amount of a given gas that dissolves in a given type and volume of liquid is directly proportional to the partial pressure of that gas in equilibrium with that liquid" (Figs 2 & 3).

When surrounded by high pressure like in diving, inert gasses like nitrogen dissolve and accumulate in blood and the tissues. Then during rapid accent or decompression, the inert dissolved gases predominantly nitrogen, returns to its gaseous form (out gassing). This out gassing usually happens in the lungs but when out gassing is too rapid, a lag occurs before nitrogen can diffuse back to the non-fluid space. This delay precipitates nitrogen bubbling while still in the fluid state, which if in blood stream may cause embolism, and disrupt cell architecture when within tissues. Dysbarism encompasses the conditions of decompression sickness, arterial gas embolism and barotraumas, whereas decompression sickness and arterial gas embolism (AGE) are commonly classified together as decompression illness[1]. DCS is classified into type I, II and AGE. For decompression sickness, the pressure changes should be of sufficient duration to create a gas load but not necessarily saturate tissues, and the onset of symptoms may take anywhere between 0-36 hours. The neurological deficits can manifest in spinal cord or the brain.

DCS type I (mild, the bends occurring in 70-85%) accompanied by pain, rash and pruritis, are self resolving.

DCS type II (serious) could present as shock and nervous system involvement, of which the spinal cord lesions are the most common. Spinal cord involvement in DCS presents with back pain and signs of paraesthesia, motor weakness and loss of sphincter control. Among these,

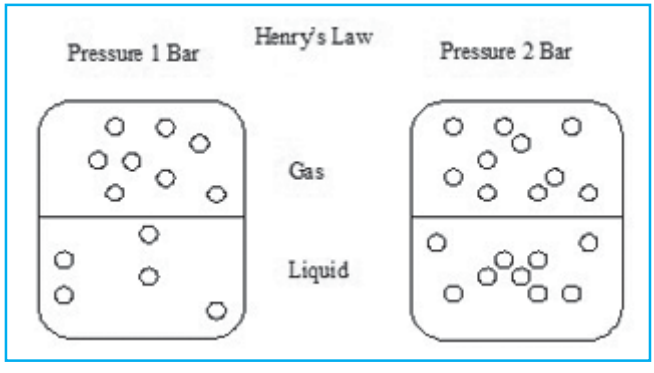

Fig 2- *Diagramatic - Henry's Law (a)*

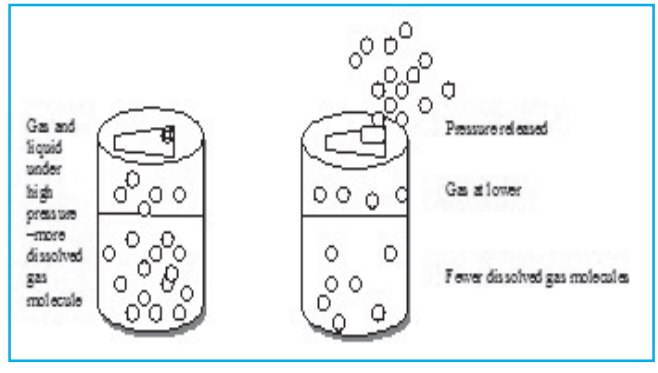

Fig 3- *Diagramatic - Henry's Law (b)*

symptoms of back pain and girdle pain after a diving episode has been shown to be closely associated with a diagnosis of spinal decompression sickness[2,3]. The pulmonary vasculature is able to filter the bubbles and micro-emboli to a certain extent depending on the size of the bubbles. The microbubbles which pass through the pulmonary capillaries and enter the systemic circulation surprisingly do not cause any damage. As this blood circulates in the cerebral vasculature, much of the oxygen is extracted from this blood by the cortical grey matter, and the remaining is drained by capillary free zone, long veins which supply the white matter of the mid brain and the spinal cord. In this area, any compromise by micro-emboli will cause the hypoxic damage and dysfunction of the blood CNS barrier, leading to inflammation, demyelination leading to physiological or physical axonal dysfunction, also termed as perivenous syndrome[4]. This venous ischaemia of the cord causes myelitis in the cord in most patients with a diagnosis of DCS of the spinal cord. Interestingly, studies have shown that the blood bubble interface can cause mechanical stretch of the blood vessels and cause a foreign body effect that causes release of vasoactive amines and activates the complement and the coagulation pathways, aiding in formation of thrombus[5,6].

Another factor which can augment bubbles to come into the systemic circulation directly bypassing the pulmonary filter is patent foramen ovale. The prevalence of probe patent foramen ovale is about 27% in the normal population[7]. The DAN Report on Decompression Illness, Diving Fatalities and Project Dive Exploration, in 2006 had analysed 8,000 divers for around 100,000 dives and has reported an incidence of DCS as 3.6 per 10,000 dives[8]. Analysis of a cohort of divers with a diagnosis of DCS showed 50 -53% of them had a PFO, and in the group of divers who did not have a diagnosis of DCS, the incidence of PFO was 8%[9].

AGE occurs immediately after resurfacing, affecting the brain more than the spinal cord and is associated with complaints of headaches, giddiness, anxiety, altered sensorium and stroke. It can also cause embolism of the coronary vessels and lead to arrhythmia's and myocardial infarction.

Incidence of DCS: According to statistics from the US the incidence of DCS in divers is about 1000 per year[10]. Marx *et al* in 2010 showed the incidence of decompression sickness to be about 2.8 cases per 10,000 dives, with the risk 2.6 times more in males than in females. The DAN report of the "Project Dive Exploration" which reviewed data up to the year 2002 from 50,150 dives showed that the incidence was about 0.05% where as the DAN report based on DCS claims from the year 2000 to 2007 found to be about 217 cases per 100,000 (0.21%) among insured members. There is no substantial data from the Indian subcontinent, but there has been some surveys conducted among the Urak Lawoi fishermen from Thailand who dive using surface-supplied compressed air through rubber tubes. They found the diving related morbidity to be about 550 per 100,000 person-years and a mortality rate of approximately 300 per 100,000 person-years[11].

Management:

Patient must be kept supine, as the head down position increases risk of further damage to the blood brain barrier[12]. Treated with hyperbaric oxygen therapy is recommended, if near a centre with this facility. Otherwise, treating with 100% oxygen washes out and reduces risk of further damage by the dissolved nitrogen. It is more effective if administered within the first four hours and also reduces the number of therapies required[13].

Antiplatelets after ruling out, intracerebral and other hemorrhagic lesions, have been tried, though there is no evidence for this is not intervention[14].

Intubation, cardiopulmonary resuscitation, and chest tube insertions should be performed where ever necessary. Rehydration with intravenous fluids, with constant monitoring of haemodynamic parameters till adequate renal perfusion and urine output is obtained. Neurological symptoms, pulmonary symptoms, and skin changes should be managed with hyperbaric oxygen therapy if seen within 10 to 14 days of development.

The diagnosis of DCS is primarily based on history and clinical findings. Hyperbaric oxygen treatment should not to be delayed if DCS is the most likely diagnosis. Differential diagnosis includes alcohol/substance abuse, deep vein thrombosis, pulmonary embolism, hyperventilation syndromes and vasogenic shock. Full blood counts, electrolytes, blood sugars, arterial blood gases to be measured and appropriately corrected. Chest x-ray is done to rule out lung conditions like pneumothorax, pneumomediastinum, subcutaneous emphysema. If sensorium continues to deteriorate CT of head is required to rule out other causes of brain injury. Imaging with T2 and FLAIR sequences of MRI may show focal lesion in the spinal cord and brain. The sensitivity of picking up a lesion in the brain is more,

than in the spinal cord[15]. One should be aware of the fact that a negative MRI finding does not rule out diagnosis of dysbarism.

Prognosis: Follow-up studies of DCS patients has shown that about 14.3% of the patients continued to have residual neurological deficits and symptoms from type II DCS and 7% from type I DCS. Permanent neurological sequelae have been reported to be around 16% in follow-up cases[16]. Other factors which are associated with poor outcomes are, age over 42 years, depth of the dive being greater that 39 metres, bladder dysfunction, and persisting or worsening of clinical symptoms before recompression therapy. The lag time to recompression therapy did not significantly influence recovery[3].

Prevention: Adhere to standard diving guidelines, limiting the depth and duration of deep water dives, avoiding substance abuse before diving, avoiding repeated dives within a 12-hour period. Slow accent after every dive, no faster than 9 metre per minute. A safety stop, before surfacing at 4.5 metre prior to reaching the surface for 3 to 5 minutes reduces the risk of DCS. This allows for "degassing" before surfacing.

Avoiding air travel within 24 hours of deep dives, avoiding air travel in non-pressurised aircraft, adequate hydration, can reduce risk of DCS.

Further research on improved primary prevention and medical treatment are needed to reduce mortality and morbidity in decompression sickness.

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