Review Article

Decompression Illness

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Abstract

Decompression illness (DCI) describes a syndrome complex caused by inert gas bubbles generated by an inappropriate rate of reduction in ambient environmental pressure or decompression. This "umbrella term" covers both traditional decompression sickness caused by *in-situ* bubble formation from dissolved inert gas and arterial gas embolism (AGE), in which alveolar gas or venous gas emboli (via shunts or by-passing pulmonary vessels) are introduced into arterial circulation. DCI occurs in divers, compressed air workers, aviators, and astronauts, but AGE could also arise from iatrogenic causes unrelated to decompression. A hundred years ago, serious manifestations and deaths were frequent in divers and caisson workers due to DCI, but they decreased greatly when decompression stops were introduced in diving practice. This review article is of interest to the doctors who face the dilemma of treating the rare syndrome of DCI that could present in the clinical spectrum ranging from itching and minor pain to severe neurological symptoms or other systemic pathology. The first aid lies in the administration of 100% oxygen, and definitive treatment is therapeutic recompression. With appropriate and adequate treatment, recovery is complete, but some severe cases may have lifelong residual deficits, even after extended and multiple recompressions.

Keywords: Arterial gas embolism, decompression illness, decompression sickness, therapeutic recompression

Introduction

Over the last several decades, scuba (self-contained underwater breathing apparatus) diving has gained popularity globally. The endeavor to explore newer avenues underwater has resulted in a rapid expansion of recreational, technical, professional, and military diving opportunities. Although diving is relatively safe, significant injury or death can occur with a fatality rate of 3–6 deaths/100,000 divers.[1] Decompression illness (DCI) is one such entity affecting divers with insignificant to grave consequences, particularly if there is absence of regulations for safe diving practices.

Medical professionals, regardless of specialty, need to be aware of the ill effects of altered ambient pressure exposure on the human body. DCI can be of rapid onset, straight forward, or insidiously mild and delayed. Divers with DCI can present late, far away from the dive sites, attributing to its variable presentation, tardy onset, and air travel after diving. Medics need to consider activity in preceding days and be aware of the issues and nuances of diving disorders so that they do not miss the opportunity to diagnose and treat such patients appropriately.

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Pathogenesis

DCI is definitely associated with either vascular or extravascular bubbles, which are produced during or after a reduction in ambient environmental pressure or decompression. It includes two pathophysiological syndromes: the more common Decompression Sickness (DCS) and the rarer Arterial Gas Embolism (AGE). AGE occurs in divers mainly during rapid or breath hold ascent from depth where disproportionately expanding gases stretch the alveoli, and rupture the alveolar capillaries causing pulmonary barotrauma, leading to tracking of alveolar gas into arterial circulation.

The formation and build up in the size of extravascular and intravascular bubbles occur when the sum of the partial pressures of dissolved gas disproportionately exceeds the ambient pressure. In diving and during compressed-air work, this state of supersaturation is made possible due to increase in tissue inert gas partial pressure at high pressure. This is clinically evident during decompression if the rate of ambient

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pressure reduction exceeds the rate of inert gas washout from tissues. Hypobaric exposure too could produce DCI. During sudden increase in altitude for aviators or in space travelers who are exposed to decreased ambient barometric pressure, supersaturation of body tissues is due to preexisting dissolved inert gas at sea level which can cause bubble formation.

Bubbles can have mechanical, embolic, and biochemical effects with a vivid spectrum of clinical features. Manifestations are caused by direct effects from the mechanical distortion of tissues or embolic obstruction causing stroke-like outcome or activation of endothelial mediators causing capillary leak syndrome, extravasation of plasma, platelet activation and deposition, leukocyte–endothelial adhesion, and hemoconcentration.[2] Diving is associated with the generation of gas emboli which arise mostly on the venous side of circulation, and they are usually eliminated as they pass through the pulmonary circulation.[3] Pulmonary vascular obstruction may occur when large amounts of free gas transit through the venous system, which can result in chest pain, dyspnea, cough, and pulmonary edema.[4]

Large bubble load can overwhelm the pulmonary capillary filter and by-pass the pulmonary bed. In addition, the bubbles can "shift to left" in the presence of a patent foramen ovale (PFO) or other right-to-left cardiac shunts which will aid the bubbles' entry into the systemic arterial circulation and can further affect the spinal cord, brain, skin, inner ear, etc., Initial symptoms after recreational diving typically consist of pain or mild neurological manifestations such as numbness or paresthesia. Secondary effects can cause delayed symptoms up to 24 h after surfacing in most cases.

Incidence

Of the 323 diving deaths reported to the Divers Alert Network (DAN) worldwide during the period 2010–2013, 22 were due to AGE.[5] If appropriate decompression procedures are followed, DCS is also uncommon. A DAN sample of 135,000 dives by 9000 recreational divers reveals the rate of occurrence of DCS to be 0.03% dives.[6]

Diagnosis

The erratic presentation of DCI makes it impossible to diagnose by a single, simple evaluation or investigation. Diagnosis is made on thorough clinical evaluation and suspicion; thus, it is pertinent to elicit accurate history and physical examination of individuals with symptoms after diving. AGE should be suspected if a diver has rapid-onset altered consciousness, confusion, and focal cortical signs or seizure during ascent or within a few minutes after surfacing from a compressed gas dive.[7] If the diver spends much time at depth and might have absorbed substantial inert gas before surfacing, serious DCS usually with spinal cord manifestations predominates. Any new symptom arising shortly after decompression should be considered as a possible DCS, especially if the depth-time exposure has exceeded acceptable limits set from diving safety point of view.

Historically, DCS was classified into the following two types based on presenting symptoms and the organs affected: Type I DCS/DCI, the clinical features could include one or more of the following: arthralgia, myalgia, dermal manifestations, fatigue, restlessness, and isolated nausea, and Type II DCS/DCI may present as one or more of the following: shortness of breath, cough, and other cardiopulmonary signs, paralysis, paresis, incoordination, dizziness, headache, nystagmus, reflex abnormality, visual disturbance, paresthesias, dysesthesias, behavioral changes, decreased level of alertness, and other signs and symptoms consistent with injury to the nervous system.[8]

Pain, constitutional symptoms, subjective numbness, paresthesias, or rash initially occur in 85% of cases, and at least one of these mild manifestations is present in all cases. Neurological examination is essential for all divers with suspected DCI, unless the delay in recompression to cater for examination is unacceptable for patient outcome in serious cases, especially cerebral AGE. Post recompression, evaluation would include complete blood count to rule out hemoconcentration and chest radiography for the detection of pneumothorax. Specific neurophysiological tests such as audiometry or electronystagmography for inner-ear DCS and imaging are usually delayed until after recompression. Doppler ultrasonography and echocardiography are valuable for research into venous gas emboli but not for the diagnosis of DCI, which remains entirely clinical.[9] Among the imaging modalities available to study neurologic DCS, magnetic resonance imaging appears to be the most accurate for detecting pathological changes in the brain and spinal cord.[10] Imaging studies are not useful for the assessment of need for recompression treatment and if used should not delay recompression unless there is a strong suspicion of a nondiving-related cause.

Prevention

DCI is better prevented than treated. Meticulous dive planning considering the depth, duration, and type of diving along with the scrutiny of diver for fitness is the first step in the prevention of DCS. AGE is not related to depth-time exposure in diving. The risk of this syndrome can be decreased by avoidance of three factors, namely breath holding, rapid ascent, and diving with lung pathology, which places a diver at an increased risk for pulmonary barotrauma.

Simple predive measures such as endurance exercises in a warm environment, oral hydration, and normobaric oxygen breathing can be applied prior to diving for attenuating decompression stress. The risk of DCS in flying after diving can be decreased by reduction of exposure or by elimination of inert gas before or during decompression with intermittent high-concentration oxygen breathing.[11] In future, drugs that modify bubble generation by improving endothelial function or reducing gas nuclei population could be used as possible candidates for preventing the occurrence of DCI, particularly

in risky operational dives or in emergency situations such as submarine escape.[12]

Observation

Patients with DCS can dramatically improve or have complete resolution in musculoskeletal or neurological symptoms with just oxygen and rehydration. However, all such patients should be evaluated by a diving physician. They should be observed for at least 24 h as relapses occur with worse outcomes. Therefore, referral to a center with recompression facility is strongly advised.

Management

The mainstay of primary first aid for DCI is 100% oxygen even as the patient is being transported to a hyperbaric center for recompression therapy. Breathing 100% oxygen eliminates the inert gas in the inspired mix, enhancing the washout of inert gas from tissues, thus increasing the partial pressure gradient for diffusion of inert gas from bubble into tissue.[13] First-aid oxygen when administered early, i.e., within 4 h, increases the recompression efficacy and reduced number of recompression treatments required.[14] Strategies suggested include maintenance of a horizontal position to prevent the movement of intravascular bubbles into the cerebral circulation and administration of 100% oxygen, intravenous or oral fluids, corticosteroids, anticoagulants, nonsteroidal anti-inflammatory drugs (NSAIDs), lignocaine, and diazepam. It is important to consider that any one of these strategies might modify the outcome of DCS and AGE in opposite directions.[15]

Typically, recompression is done in a multiplace chamber with an attendant. The dictum is to recompress as quickly as possible. Delayed recompression is probably less effective, but the time beyond which recompression is pointless is unknown.[16] The clinical symptoms of spinal cord DCS and their initial course before admission to the hyperbaric center should be considered as the major prognostic factors in recovery.[17]

The most common recompression schedule is the Short Oxygen Recompression Therapy- Royal Navy Treatment Table 61 [Figure 1] or an equivalent procedure. Patients are compressed to 2.8 bar pressure, corresponding to 18 meter sea water (msw) depth while breathing 100% oxygen with intermittent air break. The time at 2.8 bar and 1.9 bar equivalent to 9 msw can be extended with additional cycles of oxygen and air if clinical resolution is not complete.[18]

If treatment pressures have to be >2.8 bar, air or any other suitable mixture such as nitrox is used to reduce the risk of oxygen toxicity. Approximately 6.6% of patients who present after 48 h may not respond to the recompression therapy on oxygen.[19] Such patients will require extended recompression and adjuvant therapy. Rehydration is vital as there is usually a degree of dehydration in these patients.[20] Hypotonic fluids and fluids with glucose should be best avoided in neurological injuries.[21,22]

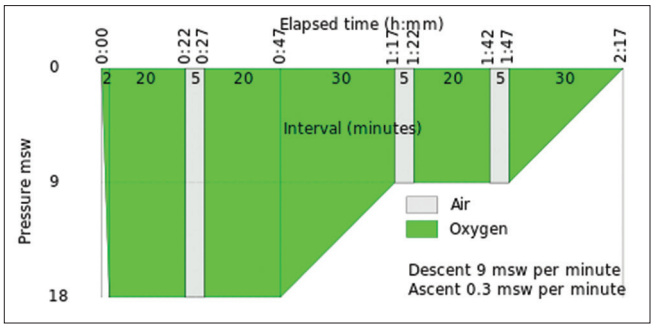

Figure 1: Short oxygen recompression therapy – Royal Navy Treatment Table 61

Although recompression is the primary treatment, especially for severe DCI, other aspects of care for seriously ill patients should not be neglected, i.e., management of airway, coma, hemodynamic instability, temperature control, metabolic derangements, bladder dysfunction, pain, risks of immobility, and long-term disability.[23] The addition of NSAIDs and the use of heliox may reduce the number of recompressions required, but do not improve the odds of recovery.[24] Treatment with NSAID is appropriate if there are no contraindications. Divers should be kept thermally comfortable and warm but not hyperthermic, especially in cases with severe neurological signs and symptoms.[25]

Good diving practice suggests that after diving, divers should be within timely reach of a recompression chamber for treatment, if DCI is suspected, and should be recompressed at the earliest once diagnosed. Following therapeutic recompression, observe the patient for recurrence of symptoms, 2 h for pain-only symptoms and 6 h for serious symptoms. Patients with residual symptoms should fly only with the advice of a diving physician and not fly for 72 h after treatment, at a minimum. After completing treatments, the chamber attendant should also remain in the vicinity of the recompression chamber for 1 h followed by remaining within 1h travel time of a recompression facility for the next 24 h.[26] Divers should be referred for screening of PFO if there is severe or repetitive DCI to assess the risk of continued diving post DCI.[27]

One newer avenue of research in relation to DCS relates to the role of the so-called intravascular "microparticles" which are small fragments shed from the surface of some formed elements of blood and endothelium. These fragments appear to activate or amplify inflammatory processes and coagulation. Microparticles are increased in a variety of disease states including sepsis, myocardial infarction, and vasculitis. Microparticle numbers also appear to be increased by decompression stress. This raises the possibility that some of the pathophysiological events in DCS that are currently attributed to circulating bubbles may in fact be caused or exacerbated by microparticles.[28] Once established, this may provide a better insight for the prevention and management of DCI in future.

Conclusions

DCI affects a specialized group, mostly with mild symptoms, but bears the potential for permanent injury or death in severe cases, especially if unrecognized or inadequately treated. It is a problem that few physicians are trained to recognize or treat. Medical personnel should be aware of the manifestations of DCI in the background of a patient presenting with a history of recent diving or exposure to altered ambient pressures. If DCI is suspected, diving physicians should be consulted. All cases should be treated initially with 100% oxygen until hyperbaric recompression therapy can be provided as per therapeutic recompression tables in the form of breathing air, oxygen, or mixed gas in a recompression chamber. If treated early, there is a significantly higher chance of successful recovery.

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Conflicts of interest

There are no conflicts of interest.

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