CASE REPORT

A case of decompression illness during saturation diving

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INTRODUCTION

Decompression illness (DCI) is the manifestation of clinical symptoms due to formation of free phase gas (bubbles) as sequelae of suddenly reduced ambient pressure on the dissolved inert gases in the various body tissues. It is seen in the settings of diving, aviation, and space exploration. In diving, DCI is most commonly encountered in those dives which are excessively long or deep or in whom the mandatory 'stops', which are halts during ascent to surface to harmlessly remove the dissolved inert gas in the various body tissues, have been omitted. In saturation diving, meticulous steps in the form of a prolonged monitored decompression in the controlled environment of a decompression chamber are taken to obviate DCI.[1] This is further reinforced by providing a high fraction of inspired oxygen (FiO_2) to decrease the inert gas uptake as well as to enhance the removal of the dissolved inert gas harmlessly. Hence, occurrence of DCI during saturation diving, reflects either poor dive execution or an idiosyncratic phenomenon.[2,3] We present a case of musculoskeletal decompression illness (Type I) after saturation diving, which is reflective of the idiosyncratic nature of the illness and has implications for the employability restrictions for the individual. This case also highlights the limitations of completely preventing it even by meticulous execution of the dive.

CASE REPORT

A 30-year-old serving sailor, by trade a clearance and saturation diver, presented with pain in the left knee three hours post surfacing from a saturation dive. The dive was to a depth of 60 m in the chamber while breathing a mixture of helium–oxygen, with partial pressure of oxygen being maintained at 1.4 atmosphere absolute [ATA] during compression and 1.6 ATA during decompression. The pain was insidious onset, dull aching type on the medial aspect of the left knee with mild exacerbation on full extension, and mild relief in the semi flexed position. There was no radiation or referred pain, nor involvement of any other joint. There was no history of definitive trauma or physical exertion. There was no history of fever, tingling or numbness of left lower limb, restriction of movement of left knee joint, incontinence, vertigo, visual symptoms, otolaryngological (ENT) symptoms, skin rash, or any respiratory symptoms. During the course of the dive, the individual was comfortable and asymptomatic. There was no instance of sudden loss of pressure during the course of the dive or any other deviation from the standard decompression schedule. He had a history of similar symptoms of the right shoulder six months prior to the current incident post surfacing from a 'no-stop' 30 m 'wet' dive while breathing compressed air. The symptoms resolved on breathing 100% oxygen. This incident was not monitored by a physician and is likely to be an incidence of musculoskeletal decompression illness.

Clinically, the patient had pulse of 80/min, blood pressure of 118/70 mmHg and a body temperature of 98°F. He weighed 62 Kg with body mass index of 22.78. On examination of the affected joint, there was no swelling, deformity or redness. It was non-tender and a pressure cuff inflated over the knee resulted in mild relief of the pain. The range of motion of the joint was full and free and there were no signs of distal neurovascular deficit. The examination of the central nervous system revealed normal higher mental functions, no motor or sensory deficits, normal deep tendon jerk reflexes, normal cranial nerve function, and no signs of meningeal irritation or cerebellar ataxia. Tympanic membranes were bilaterally intact and mobile. The examination of the respiratory system, cardiovascular systems, and the abdomen did not reveal any abnormality. Based on the presentation, history, and examination a diagnosis of Type I decompression illness without the evidence of barotrauma was entertained. Concurrent haematological and biochemical studies were within normal limits. He was managed with the standard recompression treatment of pressurising to 2.8 ATA on 100% oxygen and on evidence of relief of symptoms, he was decompressed to surface over a period of four hours and 45 minutes. The individual reported complete relief of symptoms on conclusion of the recompression therapy. Subsequently, he was again investigated and his haematological and biochemical parameters were found to be within normal limits. Chest radiography, activated prothrombin test time, electrocardiography (ECG) and spirometry were found to be normal. Echocardiography did not reveal any evidence of patent foramen ovale (PFO). There was no recurrence of symptoms after being observed closely for 48 hours. He was recommended to avoid flying for seven days and diving for four weeks.

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DISCUSSION

Decompression illness results from the physical and biochemical effects of free phase inert gas or bubbles which have precipitated due to an abrupt drop in ambient pressure. The quantum of these bubbles depends on the duration and magnitude of pressure to which the body was exposed as well as to the composition of breathing mixture that was inspired. Once precipitated, the bubbles have multiple physical and biochemical effects in the form of compression and stretching of the surrounding structures and activation of the complement cascade.[4] These manifest clinically as pain, numbness, and tingling, sensory and motor neurological deficits, and bleeding, the site depending on the precise location of the precipitation of the offending free phase gas. The incidence of DCI open water dives from minutes to several hours in duration varies according to the diving population: typically 0.015% for scientific divers, 0.01–0.019% for recreational divers, 0.030% for US Navy divers, and 0.095% for commercial divers.[3]

Decompression illness is classified into two types: Types I and II based on the severity and variety of symptoms. Type I is the 'pain only' DCI, this is considered to be a minor type which requires considerably lesser magnitude of recompression therapy. Type II is the DCI where symptoms in addition to pain are present, particularly neurological symptoms. These require greater magnitude of recompression therapy and have worse prognosis in terms of residual neurological deficits.[5] Our patient did not present with any neurological symptom and hence was classified as Type I DCI.

Decompression illness is to be entertained as a differential diagnosis in patients presenting with *any* symptom during or after a dive. The diagnosis of DCI can be difficult due to the myriad symptoms with which it manifests and the involvement of nearly all systems. The diagnosis is mainly based on the history of an incriminating dive profile and the clinical examination. Relief of symptoms on recompression clinches the diagnosis. There is little role for investigations in diagnosis although they are supportive in cases where associated barotrauma is suspected or for early determination of involvement of coagulation abnormalities. First aid treatment is 100% oxygen and definitive treatment is recompression to increased pressure, breathing 100% oxygen. Adjunctive treatment, including fluid administration and prophylaxis against venous thromboembolism in paralysed patients, is also recommended. Treatment is, in most cases, effective although residual deficits can remain in serious cases, even after several recompressions. The recompression therapy has to be initiated without waiting for the conclusion of investigations.[3] In our patient, although the dive profile was not incriminating, the characteristic symptomatology and the relief of symptoms on recompression established the diagnosis of DCI.

Decompression illness has several risk factors involving the dive profile as well as the individual characteristics. 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 PFO.[4]

In the dive, the patient undertook, there were no recorded violations of the time or depth limit or of the standard decompression schedule. In effect, it was an 'undeserved' DCI. Also, the individual had a similar incident which was possibly another 'undeserved' DCI. These point towards a likelihood of an idiosyncratic predisposition to DCI. In patients, suspected to have such a predisposition, PFO has to be ruled out.[6,7] If present, this effectively rules out the individual from combat diving. However, the opinion regarding further employability in cases of recurrent 'undeserved' DCI having no demonstrable PFO, is divided. In some militaries, two incidence of Type II DCI results in the individual being denied further diving employment.[8] This has been criticised as this results in loss of trained manpower of combat divers without adequate scientific basis and may also lead to dangerous non-reporting due to implied threat of consequent unemployment. On the other hand, continued employment of such individual's risks future combat diving operations, as development of DCI during such an operation will have disastrous consequences. In this case, a deliberate decision was taken to retain the individual for the diving duties in view of no demonstrable evidence of PFO; the minor severity of the DCI which occurred; and the lack of diagnostic evidence in the first instance of shoulder pain to classify it as DCI. Nevertheless, the individual is placed under careful watch for his forthcoming diving duties.

CONFLICTS OF INTEREST

None identified.

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