Dysbaric osteonecrosis in technical divers: The new 'at-risk' group?

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Abstract
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Introduction: Dysbaric osteonecrosis (DON) in people working under increased atmospheric pressure is well documented. It is generally less common in military and commercial divers than in caisson workers, except in some high-risk groups, such as in many indigenous diving industries where workers have little or no understanding of decompression principles. With the increasing popularity within the recreational diving community of deep air and mixed-gas decompression diving ('technical diving'), it is likely that diving physicians may see an increase in the prevalence of DON in this group in the future.
Methods: The case report is presented of a technical diving instructor, with a 30-year history of deep diving, who developed bilateral humeral head DON and required a right shoulder hemi-arthroplasty. A focused literature search was also undertaken to identify published cases of DON in recreational divers.
Results: The frequency, duration and depth of exposure to pressure, inadequate decompression, the occurrence of DCS and increasing age have been common features associated with DON in both divers and caisson workers. Many of these features were present in this technical diver.
Conclusions: Whilst DON is uncommon in recreational air scuba divers, all the above risk factors are present to a greater degree in technical diving. It is suggested that medical review for DON is merited from time to time in this potentially high-risk group of recreational divers.

Introduction

Aseptic bone necrosis (AVN) is the final common pathway of various conditions leading to bone death,[1] most commonly long-term, high-dose steroid use and alcoholism. Other contributing conditions include pancreatitis, lupus, sickle cell disease, Gaucher's disease, radiotherapy, trauma, pregnancy and thrombotic conditions.[1,2] A recent review paper discusses the diagnosis and staging of AVN of the femoral head.[3] AVN in caisson workers and divers, termed dysbaric osteonecrosis (DON), is well documented. In the 1970s, the Medical Research Council (MRC) Decompression Sickness Panel reported an incidence of 19% in caisson workers and reported a link to decompression sickness (DCS).[4,5] It is reported to be less common in military[6] and commercial divers[7,8] except in some high-risk groups[9] and those with little or no understanding of decompression principles.[10] Readers are referred to a useful review of DON in professional divers.[11] Lesions may be either juxta-articular (Type A, Table 1) or in the shaft of long bones (Type B), the former being far more likely to produce symptoms.

DON is considered rare in recreational divers, though individual cases have been reported.[12–15] With the increasing popularity within the recreational diving community of deep mixed-gas decompression diving ('technical diving'), it is likely that diving physicians may see an increase in the prevalence of DON in this group in the future because of the nature of their diving activities. For this reason, we present an illustrative case of a technical diving instructor with DON and briefly review the relevant literature.

Literature review

PubMed, the Rubicon Foundation Research Repository (http://archive.rubicon-foundation.org/xmlui/), the complete collection of the *South Pacific Underwater Medicine Society Journal* and *Diving and Hyperbaric Medicine* journal and major textbooks on diving medicine were searched using the terms avascular necrosis, dysbaric osteonecrosis, and caisson disease and in combination with the terms diving, decompression sickness, scuba and technical diving. Further articles of potential interest from reference lists were also reviewed. The intention was not to perform a comprehensive

Table 1

Classification of dysbaric osteonecrosis lesions (after the United Kingdon Medical Research Council). * = classification of the present case

Lesion	Subtype	Comments
A type lesions Juxta-articular	A1 Dense area with intact articular cortex	Prevalence of A lesions: Tunnellers and saturation divers Femur > Humerus Other divers Humerus > Femur
	A2 Spherical opacities	
	A3 Linear opacities	
	A4 Structural failures - Translucent subcortical bands - Collapse of articular cortex - Sequestration of cortex*	
	A5 Secondary degenerative osteoarthritis	
B type lesions Shaft	B1 Dense areas	n/a
	B2 Irregular calcified areas	
	B3 Translucent and cystic areas	

literature search, but to focus on published evidence relevant to recreational and technical diving.

Case report

A man in his 40s had been actively involved in recreational diving since 1991. He qualified as a dive instructor in 1994, teaching a mix of recreational and technical scuba diving around the world, doing 200 or more hours a year in the water. During this period, technical mixed gas diving took up about a quarter of his diving hours, but no detailed records of his diving were kept. Between 2010 and 2018, he recorded on his annual medical questionnaires over 1,800 dives of which 80% were to depths greater than 30 metres' sea water (msw) and over 1,500 were on mixed gas/trimix using closed-circuit rebreathers.

In 2002, he did a wreck dive to 115 msw using trimix, followed by a gas switch to air at 30 msw during the ascent. At the 9 msw decompression stop, he developed severe vertigo which persisted post dive. Audiometry was normal. A diagnosis of inner-ear decompression sickness (DCS) was made and he underwent a RNZN heliox Table 1A (similar to a Comex 30 treatment table) with a good response. Following two further short hyperbaric oxygen treatments he was symptom-free. Two years later in 2004, he dived on the same wreck to a depth of 120 msw. Again, a gas switch to air was made, this time at 40 msw. On reaching the 15 msw stop, he developed severe vertigo, vomiting and tinnitus. Because symptoms had improved at 6 msw after switching to 100% oxygen, he did not present for assessment until the following day when examination revealed a fine right nystagmus, very poor sharpened Romberg test and a 70 dB hearing loss at 8 KHz in the right ear. The diagnostic difficulty of differentiating between inner-ear DCS and barotrauma necessitated a cautious approach to recompression on a Royal Navy Treatment Table 62; however, his symptoms responded well. Again, after two further short hyperbaric oxygen treatments, he was symptom-free.

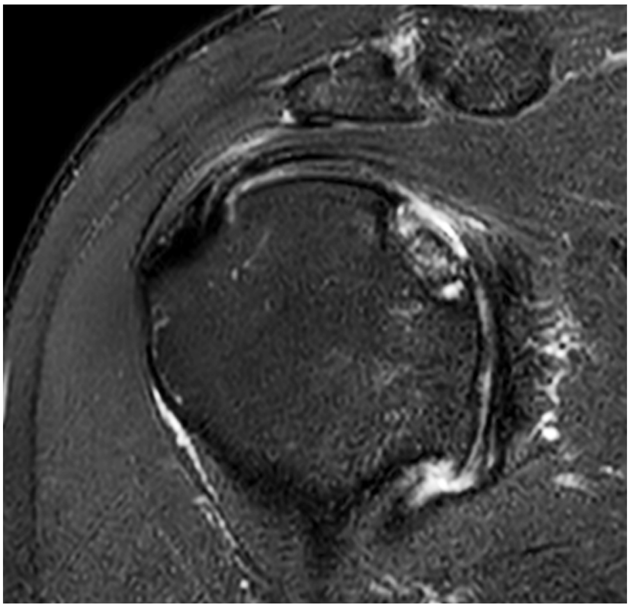
In 2011, aged 42 years, he developed pain in the left shoulder. Given his diving history, he underwent a long-bone X-ray survey which showed "*a localised lucency in the left humeral head with a surrounding sclerotic rim, the right shoulder was normal in appearance and there were faint, slightly serpiginous-appearing sclerotic lesions in the proximal left and distal right femoral shafts.*" In 2013, he was seen by an orthopaedic surgeon (BC) for assessment of the left shoulder lesion, which was managed conservatively at that time. No other risk factors for DON other than diving were identified.

In late 2018, now 49-years old, he presented following minor trauma to his right shoulder, with significant pain and limitation of movement associated with crepitus. Plain X-ray (Figure 1) and magnetic resonance imaging (MRI) (Figure 2) of the right shoulder showed a juxta-articular DON lesion of the humeral head measuring 8 mm by 24 mm with fragmentation of the articular surface and disruption of the articular cartilage. Given his symptoms were impeding his ability to work, he proceeded to surgery, at which time the articular surface of the right humerus had a 25 mm by 20 mm unstable osteochondral fragment in the central head. A right shoulder pyrocarbon hemiarthroplasty of the humeral head was inserted.

Recovery was uneventful with steady return of strength and range of motion such that he was cleared to return to work as a diving instructor four months post-operatively. For the hemiarthroplasty, there is little increased risk of aseptic loosening above what is already expected for patients under the age of 55. These patients have approximately double the failure rate of over 55-year olds.[16] The prognosis for the opposite shoulder is for gradual deterioration in symptoms over time due to the presence of DON in the humeral head with subchondral fracturing and collapse of the joint surface. This will eventually lead to arthritis or displacement of the fragmentation of the osteonecrotic segment, resulting in arthroplasty when the symptoms warrant.

Figure 1
Plain X-ray of the right shoulder of a recreational and technical diving instructor showing an avascular lesion of the humeral head

Figure 2
Coronal t2-weighted magnetic resonance image of the right shoulder of a recreational and technical diving instructor showing an avascular lesion of the humeral head



Discussion

AVN secondary to DCS was first noted in the late nineteenth century, culminating in work such as that of the MRC Decompression Sickness Panel in the 1960s and 1970s,[4,5,7] and later studies in China[17] and elsewhere. A high incidence of DON has been confirmed in high-risk diving groups such as long-term Japanese commercial divers,[9] and Hawaii's diving fishermen.[10] The United Kingdom DCS Central Registry and Radiological Panel determined that the risk of DON in caisson workers and commercial divers was directly related to the degree of pressure and the number of exposures,[5,7] whilst in Japanese commercial divers, diving exposure over 17–25 years was associated with DON, particularly in those diving to 35 msw or deeper and where there was a history of DCS.[9] In the majority of reports, juxta-articular lesions appear to be more common in the humeral head than in the femoral head of divers,[7,8] though this was not so in one study of 450 'hard-hat' Japanese divers in which the distribution was similar for both sites.[9] The prevalence of DON in divers in these various studies shows widely differing rates.

Thus, the frequency, duration and (deeper) depth of pressure exposure, inadequate decompression, the occurrence of DCS (and possibly delayed recompression) and increasing age have been common features associated with DON in both divers and caisson workers.[4–9,17] Whether age is, in fact, an independent risk factor or secondary to the length of the pressurisation/diving career is debatable. Although inert gas embolism is thought to be a mechanism linked to DON,[18] there is no clear evidence of a cause-and-effect relationship,[19,20] this more likely being a gradual process over many dives. Other mechanisms, such as a hypercoagulable state and fat emboli, also may be involved.[21] No certain aetiology for DON has been established.

DON is uncommon in typical recreational diving. The first report of dysbaric osteonecrosis lesions in sports divers was in an Australian study of 110 navy, professional and sport divers.[22] There were three lesions (one juxta-articular) in the 19 sport divers in the study. In the single cases reported,[12–15] in one,[15] subsequent enquiry from the author (Laden G, personal communication, 2019) revealed that this diver was a technical diver with several hundred deep and mixed-gas dives; in another,[13] the diver had performed 190 dives over six years, over 100 having been to depths greater than 30 msw. By contrast, in a third,[12] only no-decompression air diving had been undertaken, mostly to less than 18 msw. None had had symptoms suggesting DCS. Nevertheless, single events of DCS, especially if there was a delay to recompression treatment, appear to be associated with an increased risk of DON even in recreational divers.[23,24]

In a long-bone survey of 56 Turkish dive instructors each of whom had performed in excess of 500 dives, DON lesions were detected in 14, with only one juxta-articular lesion in the humeral head.[25] On univariate analysis of a wide range of factors, only increasing age was associated with DON and this, in itself, was correlated with diving experience and the total numbers of dives performed.

For occupational divers, long bone surveys are "*optional and as medically indicated*" in most jurisdictions.[26] In the Australian/New Zealand Standard,[27] juxta-articular DON is not considered to be a contraindication to continued diving. Under these regulations, this dive instructor with known DON was cleared to dive following the condition and risks associated with continued diving having been fully discussed and understood, and a document signed by the diver to that effect.

Several features of the present case – many years of deep and decompression diving resulting in a higher likelihood of DCS and the possible role of gas switches in inducing a gas phase in sensitive tissues such as the inner ear[28] or bone – illustrate why technical divers are more likely to be at risk of DON than the average recreational diver. Therefore, we believe that recreational technical divers need greater regular medical screening than open-circuit air scuba divers. Early radiological referral in the presence of joint symptoms which might suggest a juxta-articular DON lesion is warranted, as in this case.

Reporting an amateur scuba diver who developed DON, the authors state "*avascular bone necrosis … may lead to joint dysfunction and lifelong disability*".[15] Modern orthopaedic surgery for shoulder and hip joint pathology now allows a range of treatment options, including hemi- and total arthroplasty for patients with juxta-articular DON.[29] Joint preserving surgery for osteonecrosis of the shoulder includes core decompression, bone grafting or autologous bone marrow grafting. Core decompression can be effective in early stages of the disease when there is no collapse of the humeral head but is less effective once the humeral head shows signs of collapse.[30] Bone grafting, either simple autologous grafting, strut bone graft or vascularised bone graft are complex procedures with increased morbidity and variable results.[31,32] It remains unclear as to the most effective joint sparing treatment of osteonecrosis. The natural history of conservatively treated advanced osteonecrosis of the humeral head is poor. Once the condition results in structural damage (equivalent of stage A4, Table 1), almost half will require shoulder arthroplasty.[33]

Hyperbaric oxygen treatment (HBOT) has been reported in several studies to show long-term benefit for early (equivalent to stages A1–A3; Table 1) femoral head AVN. In one double-blind, randomised study of 20 patients, the HBOT group showed a significant reduction in pain at the end of 30 treatments ($P < 0.001$) compared with the sham air group who were then offered HBOT, which they all accepted.[34] At seven years' follow up of 17 of the 20, "*all patients remained substantially pain-free … with none requiring hip arthroplasty. Substantial radiographic healing … was observed in seven of nine hips* [on MRI]."[34]

The medicolegal aspects of DON in working divers in the Tasmanian fish farming and abalone industries were discussed at a conference in Hobart in 1988 with reference to a new Workers' Compensation Act that came into force that year in Australia.[35] This Act recognised DON in Schedule 4 as "*compressed air illness including avascular necrosis caused by any work involving exposure to increased or reduced atmospheric pressure from working underground or underwater or from working at high altitudes*". Similarly, this diving instructor's DON was approved for treatment under the Accident Compensation Corporation (ACC) in New Zealand. Whether the ACC would recognise DON in a recreational diver who was not an 'employed' diver remains a moot point.

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