ORIGINAL PAPERS

DYSBARIC OSTEONECROSIS DIVERS BONE ROT: A CASE REPORT

Carl Edmonds, Richard Harvey and Ray Randle

Abstract

The following case report calls into question the investigatory capabilities available for demonstrating or refuting dysbaric osteonecrosis (the "bone rot" of divers). It also implies a possible hazard associated with decompression meter and multi-level repetitive diving.

Key Words

Case report, dysbaric osteonecrosis, investigations, recreational diving.

Case history

A 36-year old female recreational scuba diver, otherwise very healthy and without a history of any predisposing factors for bone necrosis,[1] developed a pain in her left groin in November 1991. Initially this caused some problems in diagnosis, and it was variously diagnosed as a femoral hernia or an "irritable hip".

Diving history

The patient is a recreational scuba diver who achieved her open water BSAC Certificate in 1988. She had performed only typical recreational dives well within the requirements laid down by the Oceanic decompression meter that she has used on all her dives. Most of her dives were less than 18 m and she rarely dived more than once a day. Nevertheless, she had logged 136 dives and more than 70 hours underwater. She had never undertaken decompression diving, nor had decompression sickness. The only excessive diving exposure comprised 20 dives in all.

Her only "eventful" dive was in May 1991 when she dived on a single dive to a maximum of 31 m for a total 21 minutes. She ascended from 31 to 19 m faster than usual, due to the strong current. She then performed 5 minutes decompression at 10 m and 5 m respectively. At no stage of this multi-level dive did the decompression meter suggest a need for decompression. The only other deep dive was a multi-level one to 37 m maximum, a week later, without incident.

Two months later she performed 3 dives/day for 6 consecutive days, diving from a live-aboard boat on the Great Barrier Reef in July 1991. The sequence of 18 dives (Table 1) was permitted by the dive computer as no-decompression dives (but not when assessed by the US Navy decompression tables).

TABLE 1

DIVES UNDERTAKEN ON SIX CONSECUTIVE DAYS

	Maximum depth in metres	Duration in minutes	Surface interval in hours and minutes
Day 1	18	35	3.05
	12	43	2.34
	10	37	overnight
Day 2	12	47	2.15
	17	43	4.25
	12	42	overnight
Day 3	17	44	3.05
	18	30	2.20
	12	43	overnight
Day 4	21	20	6.25
	21	7	3.05
	9	45	overnight
Day 5	19	37	2.56
	15	41	2.54
	24	25	overnight
Day 6	18	38	3.40
	22	24	4.07
	9	37	overnight

Clinical progress

The symptoms progressed over 18 months, so that she became unable to carry out her normal occupational duties, other lower limb activities or weight bearing. Towards the end of this period she was progressively immobilised and more incapacitated by pain. Eventually she had a left total hip replacement in May 1993.

She was subjected to a number of investigations and operative procedures, to exclude possible causes of the symptomatology. The relevant positive investigations and procedures were:

A plain hip X-ray showed no abnormality (5th November, 1991).

The femoral canal area was explored (18th November, 1991). No abnormality was detected to indicate bowel herniation.

A laparoscopy was performed (22nd November, 1991), also without any abnormality being detected.

A technetium bone scan revealed a "hot spot" on the lower half of the left femur (27th November, 1991). An X-ray verified a 3 x 1.5 x 1.5 cm focus of benign appearing calcification in the medullary cavity at the junction of the distal and middle thirds, corresponding with the focal area of increased activity of the technetium bone scan. It was suggestive of either a medullary bone infarct or a benign enchondroma. As this lesion was the only pathology detectable, it was thought that the symptoms may have been due to that, and it was removed surgically (2nd December, 1991). This had no effect on the symptomatology.

Diving physicians and others retrospectively diagnosed a typical dysbaric osteonecrosis B2 type lesion on the X-rays. Such lesions are typically asymptomatic.

Because of the continuing symptomatology, an MRI scan was performed. This revealed no abnormality in the hip region but there was a cystic swelling of the left ovary. An ultrasound confirmed the presence of a left ovarian tumour. Oophorectomy was performed (26th February. 1992). This had no influence on the symptoms. An epithelial inclusion cyst was verified pathologically.

A local anaesthetic injection with Depo-Medrol (methylprednisolone) into the left hip produced relief of symptoms for some hours.

A CT scan of the hip region (26th May, 1992) revealed no abnormality.

An arthroscopy (14th September, 1992) of the left hip revealed normal articular surfaces, as far as could be ascertained, and no obvious reason for the symptoms.

Repeat technetium bone scans, repeat hip x-rays and tomograms, CT scans and MRI showed no abnormalities of the left hip.

A repeat injection of Depo-Medrol, with Marcain (bupivacaine), to the left hip successfully removed the pain for a few hours. Nevertheless, the patient was seen and examined by five independent orthopaedic groups over 18 months. There was conformity of clinical opinion, in that all agreed that the problem was with the left hip.

Investigations

In summary, the patient has had the following investigations, with the results in parentheses:

1 X-ray of the left hip:
November 1991 (calcified lesion with lower part of femur, possible B2 lesion)
February 1992 (NAD)
September 1992 (NAD)
January 1993 (NAD)

2 Tomograms left hip
January 1993 (NAD)

3 CT scan left hip and surrounding area:
May 1992 (NAD)

4 Technetium bone scan:
November 1991(hot spot in lower aspect of left femur, possible B2 lesion).
February 1992 (NAD)
May 1992 (NAD)
September 1992 (NAD)

5 MRI Scan
February 1992 (incidental left ovarian tumour observed)
May 1992 (NAD)
September 1992 (NAD)

6 Ultrasound
February 1992 (left ovarian tumour)

7 Arthroscopy
September 1992 (NAD)

Because of the difficulty in diagnosis, independent assessments were obtained of all the X-rays, technetium bone scans, CT scans and MRIs. No positive investigatory findings to support a diagnosis of hip disease were observed by any imaging specialist.

Pathological abnormalities

The only pathological abnormalities demonstrated in the above investigations were:

1 the bone lesion in the distal half of the left femur, similar to dysbaric osteonecrosis. The investigations supporting this included the X-ray and technetium scan.

2 left ovarian tumour demonstrated by MRI and ultrasound.

Both these lesions were removed early in the 18 month period but had no influence on symptomatology. Initially the bone pathology was thought to be consistent with an ossifying enchondroma.

It was decided, at the patient's instigation and insistence, to carry out a total hip replacement, because of the severe incapacity. At surgery, when the hip was removed, there was a chondral softening and separation with adjacent areas of obvious collapse of the hyaline cartilage (Fig 1).

Gross pathology of the hip revealed a necrotic top of the femur. The cartilage appeared normal, apart from minor transverse splits and mild superficial changes. Histologically, the bone of the femoral head showed areas of normal bone and bone marrow, but with focal areas of bone and bone

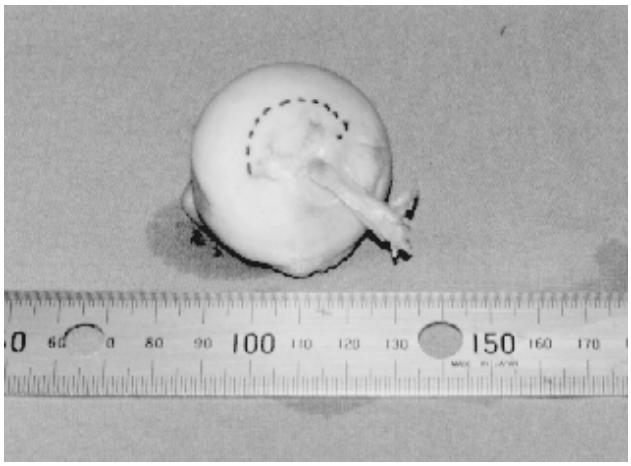

Figure 1. The area affected is under the dotted line.

marrow necrosis, and surrounding areas of bone repair. One of the necrotic areas was in the subchondral position, so that there was collapse of the overlying cartilage, which was fragmented. Occasional medium sized vessels contained thrombus, and this could have been part of the explanation for the multiple focal necrosis. There was no total or massive necrosis of the femoral head.

In summary, the appearances were those of small separate infarcts in the femoral head, immediately under the articular surface.

Discussion

At a radiological/pathology "Bone" meeting at Royal Prince Alfred Hospital in Sydney, the unusual characteristics of this case were reviewed and the unanimous opinion was that the radiological and imaging examinations did not demonstrate abnormal bone morphology of the left hip. In particular in the MRI examinations there is no evidence to suggest avascular necrosis within the femoral neck or head, nor joint effusion.

The only orthopaedic abnormality in any of the investigations was the lesion within the medullary cavity of the distal left femur on the plain x-rays, with scan findings consistent with dysbaric osteonecrosis.

The radiology assessments were performed by 5 different radiology groups and there was consistency with all the reports. The reports were available from the radiology and nuclear medical departments of 3 independent teaching hospitals.

The pathology of the hip does not indicate aetiology, however there was no doubt of the multiple and small aseptic necrotic areas under the articular surface. Pathology reports were obtained from Royal Prince Alfred Hospital, Sydney and the Royal Free Hospital, London.

This case illustrates four important problems:

1 The dependence that we have placed on the investigatory techniques (plain X-rays, tomograms, technetium bone scans, CT scans and MRI) to demonstrate significant lesions of dysbaric osteonecrosis, may not always be adequate to exclude such a lesion. This observation has been made elsewhere,[2] however no other reported case has had so many "negative" investigations performed.

2 The safety of the repetitive dives permitted by some of the decompression meters is in doubt. This has also been described elsewhere,[3] but in relation to decompression sickness more than dysbaric osteonecrosis. Many decompression meters allow recreation divers to undertake longer repetitive exposures to shallower (often multi-level) depths, more approximating to caisson workers' exposures than the square wave profiles of the conventional decompression dive tables. Whether this will make the recreational diver more prone to the occupational diseases of caisson workers (such as dysbaric osteonecrosis) is now in question.

3 There seems to be a paucity of pathological data to demonstrate the range of dysbaric osteonecrosis in humans, except for the more typical cases in which there is gross and extensive necrosis present. The pathology of non-traumatic idiopathic osteonecrosis has been described, and the fact that clinical symptoms can predate the articular surface involvement and the investigatory findings in this disorder is well recognised,[4] as is the multiple small vessel pathology which probably causes it.[5] This contrasts with the gross osteonecrosis presented in the common diving medical texts.[1,6]

4 This case demonstrates the possible superiority of clinical assessment over current imaging techniques, even though they are of great value when they are positive and to demonstrate clinically silent areas.[1,2, 4-6]

Acknowledgments

Acknowledgments for assistance in this case must be made to: Dr Greg Briggs, Radiology Unit, North Shore Medical Centre, Sydney; Dr John Paul Jones, Orthopaedic Surgeon, California, USA; Dr Stan McCarthy, Pathology Department, Royal Prince Alfred Hospital (RPAH); Dr Leo Pinczewski, North Sydney Orthopaedic Centre; Professor P A Revell, Pathology Department, Royal Free Hospital, London; Dr Ronald Shnier, Radiologist, RPAH Medical Centre, Sydney; Dr John P H Stephen, Orthopaedic Department, Prince of Wales Hospital, Sydney.

References

1 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine, 3rd Edition*. Oxford: Butterworth/Heinemann Publications, 1991
2 Steiberg ME, Thickman D, Chen HH et al. Early diagnosis of AVN by MRI. In *Bone Circulation and Bone Necrosis*. Arlet J and Maziers B. Eds. New York: Springer-Verlag, 1990; 281-285
3 Lang MA and Hamilton RW. *Proceedings of the American Academy of Underwater Sciences Dive Computer Workshop*. California: Sea Grant Publications, 1989
4 Lange TA. The staging of aseptic necrosis. A Review. Chapter 4 in *American Academy of Orthopaedic Surgeons Instructional Course*. Park Ridge, Illinios: AAOS, 1988-1993; 33-40
5 Jones JP Jr. *American Academy of Orthopaedic Surgeons Instructional Course*. Park Ridge, Illinios: AAOS, 1988-1993
6 Bennett PB and Elliott DH. *The Physiology and Medicine of Diving. 4th Ed.* Oxford: Saunders, 1993

Dr Carl Edmonds, FRANZCP, Dip DHM, who was the one of the founders and the first President of SPUMS, is Director of the Diving Medical Centre, 66 Pacific Highway, St Leonards, New South Wales 2065, Australia. Phone +61-(02)-9437-6681. Fax (02) 9906-3559.

Dr Richard Harvey, MBBS, is a General Practitioner. His address is Laurel Avenue, Lismore, New South Wales 2480, Australia. Phone +61-(066)-21-8606.

Dr Ray Randle, FRACS, is an Orthopaedic Surgeon. His address is 75 Hunter Street, Lismore, New South Wales 2480, Australia. Phone +61-(066)-21-2200.

EFFECTS OF HYPERBARIC PRESSURE ON THE GROWTH PLATES OF RATS

Peter Walker, Edward Bates, Wui Chung, William Walsh and Andrew Leicester

Abstract

Children with open growth plates are exposed to raised atmospheric pressures when scuba diving and during treatment for medical conditions such as osteomyelitis and gas gangrene in a hyperbaric chamber. This study was to determine if raised pressures have any detrimental effect on growth plate potential.

Immature rats were exposed for different periods of time to raised atmospheric pressures in a hyperbaric chamber. The animals were then sacrificed and their tibias examined macroscopically, radiologically and histologically. No differences in growth were detected between those exposed and the control groups. It is our conclusion that there are no detrimental effects to the growth plate of rats as a result of the pressures used in this study.

Key Words

Dysbaric osteonecrosis, hyperbaric research.

Introduction

Longitudinal bone growth is confined predominantly to the growth plates located at each end of the long bones. Cartilage is added at the top of the plate and is replaced by bone at the bottom. The cartilaginous portion of the growth plate is divided, by its morphology and function, into reserve, proliferative and hypertrophic zones.

The relationship between oxygen tension and bone and cartilage formation is a complex one. It is possible that oxygen tension may be an important physiological control mechanism governing growth at the epiphysial plate.

Brighton[1] studied the effects on growth of the epiphysial plate in vitro under different oxygen tensions using the costochondral junctions of rats. He showed that the highest growth rate occurred in 21% oxygen and the lowest growth rate in 90% oxygen. This and other experiments indicate that there is an optimal oxygen concentration for growth to occur and that high oxygen concentrations are detrimental to growth.[2,3] The explanations for this oxygen toxicity are numerous, but are not fully understood.

Oxygen is carried in the blood in two ways, bound to haemoglobin and in solution. By increasing oxygen partial pressures, either by scuba diving or in a hyperbaric chamber, the amount of dissolved oxygen increases in a linear fashion.[4]

Effective treatment of disorders using increased pressure was introduced in the 19th century. It is used in the treatment of gas gangrene, decompression sickness, gas embolism, carbon monoxide poisoning, cyanide poisoning, acute peripheral arterial insufficiency, crush injury, refractory osteomyelitis and to improve the viability of skin grafts. The treatment of some disorders may be prolonged, involving several weeks of daily hyperbaric exposures.

Destructive bone lesions have long been recognised as a latent problem associated with exposure to compressed gas atmospheres in divers. Extensive surveys have shown the incidence of dysbaric osteonecrosis to range from 4% in Royal Navy Divers (almost all of whom had been