as currently there is no requirement for abalone divers to undergo commercial diving medical examinations.

There were 2 fatalities, one from entrapment (1999) and the other from shark attack (1998).

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THE WORLD AS IT IS

ASEPTIC BONE NECROSIS AS A DIAGNOSTIC PUZZLE

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Key Words

Legal and insurance, medical conditions and problems, osteonecrosis.

Introduction

Aseptic Bone Necrosis (ABN) is also known as Avascular Necrosis (AVN) and, when it occurs in divers and compressed air (caisson and tunnel) workers, as Dysbaric Osteonecrosis (DON).

Apart from hyperbaric exposure, there are many non-dysbaric causes. A number of non-dysbaric causative factors have been listed in text-books, some of these are, in alphabetical order:

Alcaptonuria
Alcoholism
Arteriosclerosis
Cirrhosis of the liver
Diabetes mellitus
Gaucher's disease
Gout
Haemoglobinopathies
Hepatitis,
Hyperlipidaemia
Idiopathic
Inflammatory bowel disease
Organ transplant recipients
Pancreatitis
Rheumatoid arthritis
Radiation
Systemic Lupus Erythematous

Radiological and pathological features of both dysbaric and non-dysbaric osteonecrosis are indistinguishable and both are characterised by intramedullary venous stasis, ischaemia and bone necrosis.

Occurrence in divers

The prevalence of DON has been reported to be as high as 79% in Greek professional divers,[1] 85.7% in Turkish sponge divers,[2] 65% in the Hawaiian diving fishermen,[3] while other diving fishermen in Japan also recorded in excess of 50%.[4] Australian abalone divers had a prevalence of 32%.[5]

Of course, these reports were about divers who often ignored normal decompression procedures, overstayed their bottom time and had inadequate decompression.

For divers who adhere to decompression tables, however, the incidence is much lower. Royal Navy Clearance divers had an incidence of about 5-7%.[6] Commercial divers had a 4.2% incidence.[7] Although it is very rare in recreational divers, nonetheless, DON has been described.[8,9]

Non-dysbaric ABN

Excluding hyperbaric exposure, trauma is a common cause of ABN. It has been stated that up to 30% of patients with certain medical conditions such as Systemic Lupus Erythematous, Sickle Cell Anaemia, etc. may develop avascular necrosis.[10] In non-traumatic osteonecrosis, about two thirds of the cases are related to hypercortisonism and/or increased alcohol intake.[10]

ABN is the underlying diagnosis in 5% to 18% of the more than 500,000 total hip arthroplasties performed for advanced stages of osteoarthritis in the US and Western Europe.

A diagnostic problem

A man with osteoarthritis of his left hip was diagnosed as having AVN. A number of aetiological factors had to be entertained. Whatever the cause of his condition, it presented a dilemma to his Workers Compensation as he could no longer continue to work in his job.

The history

A 37 year old married man (OA), a non-drinker with 3 children, had been employed for 14 years as a "pumper" in a colliery. His duties involved de-watering areas of the pit, with some heavy work, such as moving pumps, laying hoses and pulling pipes which reputedly could weigh up to 150 kg. Occasionally, he was required to drive machinery.

His initial problem, in May 1995, was low back pain radiating to the buttock areas for which he attended a chiropractor. He obtained relief from this treatment and continued to work without much difficulty.

In June 1996, OA reported a slow and progressive onset of pain over his left thigh and groin. By November 1996, he complained of increasing pain and discomfort in his left hip region such that he had difficulty lying on his left side at night. This time the chiropractor was unable to provide any relief. He claimed that he had been limping for some 3 months before this visit to the chiropractor.

Eventually, OA attended his GP and was prescribed anti-inflammatory medication, which provided significant relief. Nonetheless the pain persisted and he also noted morning stiffness of his left hip. The discomfort was such that he even tried acupuncture therapy for relief. On 16 December 1996, OA had an X-ray of his hips. This showed advanced osteoarthritis of both hips, the left hip being worse than the right.

He had no significant past history. His only injury had been to his lower back, while lifting a water container some years earlier, which kept him off work for 2 weeks. He had no problems in his hips as a child or as a teenager, nor had he suffered any traumatic injury.

After the diagnosis of osteoarthritis was made he was referred to an orthopaedic surgeon, whose examination confirmed moderate osteoarthritis of both hips, the left being worse than the right. Since osteoarthritis is quite unusual in men in their 30s, the orthopaedic surgeon was of the opinion that his heavy work over the years had contributed significantly to the onset of the symptoms.

As OA was unable to perform the task for which he was employed and with this orthopaedic opinion, his problem became a Workers' Compensation case. Whether his work has caused or contributed significantly to the onset of his arthritic symptoms became a major issue.

The company's Occupational Physician was of the opinion that OA's work was most unlikely to be a contributing factor. The Occupational Physician, if it was thought that the osteoarthritis was work related, was interested in investigating all the other workers doing the same work to see what proportion of them had developed arthritis.

The employer informed the Occupational Physician that OA has been known to use a petrol driven compressor in a boat for a surface supply (hookah), for "prolonged periods" (up to 3-4 hours at a time), to catch crayfish. It was also stated that one of OA's friends was being prosecuted by the Fisheries Department for illegal fishing (this was all hearsay and not documented). The implication was that OA must have been a party to this illegal fishing and must have done substantial diving.

On the basis of this information, the Occupational Physician decided that OA's osteoarthritis was most likely to be due to diving.

Eventually, the employer decided to seek another opinion and asked RMW to review OA about the likelihood of him having developed his condition from diving.

The history OA gave me was that outlined above. He claimed that he had suffered from neck and lower backache in May 1995, for which he saw a chiropractor as well as a medical practitioner.

When RMW reviewed him, OA stated that his condition had remained static. He mentioned that cortisone injection given by his local medical practitioner did help to ease the pain and that he could sleep reasonably well at night.

OA did not deny that he had dived but his story differed from the employer's story. He had been doing breath-hold diving since he was 12 years old. About 1992, some 6 years before I saw him, he was taught to "hookah" dive by friends. He never formally learned to dive nor had a diving medical. He was not a regular compressed air diver, diving only during Christmas holidays with his friends to get a few crayfish, abalone and fish. He might make 3 or 4 dive trips during the holiday season. The dive profile he claimed would be to a depth of 6 m (20 ft) with a bottom time of no more than 30 minutes. Sometimes, he would dive 2 or 3 times in a day, with a surface interval of 1 to 2 hours. He had not done any commercial diving nor engaged in multi-day diving. He denied having any incidents of DCS or prolonged diving. When saw OA he had not dived for two years.

On 9 September 1997 OA had another radiological examination and a MRI of his hip. The radiological report stated that "this patient has developed Avascular Necrosis of the femoral head on the left side. When comparing this area with the previous film in 1996 there is no doubt that there was a bony abnormality at this stage. On the previous study no evidence of old Perthe's or slipped femoral epiphysis or deformity of the articular surface but there has been progression and characteristic development of avascular necrosis, with structural failure, and focal areas of ischaemia and bony regeneration and repair...". The radiologist was confident of the diagnosis of avascular necrosis and advised that a MRI would not be necessary.

Discussion

OA's history and findings leave the cause of his ABN up in the air. Could his ABN be the result of other conditions apart from trauma, or hyperbaric exposure? There are at least four questions that need an answer.

1 What is the likely cause of ABN in this patient?
2 Was it a work related consequence of dragging heavy weights around for a protracted period (years) in a man in his 30s?
3 Was it due to his diving activity?
4 Was it due to one of the many medical conditions that could give rise to ABN?

He did not have a history of trauma. Although admitted to being a "diver", his hyperbaric exposures would not be considered as likely to be the causative factor. He has never suffered decompression sickness at any stage. He is a non-drinker and not on any medications (the cortisone injections he received for pain relief after the diagnosis would hardly be responsible).

To exclude the non-traumatic conditions, a series of laboratory investigations were done, and the results are shown in Table 1. The asterisks * indicate abnormal results.

Reviewing OA's history and various investigations the abnormal results were that his lipids, cholesterol, triglycerides and apolipoprotein were elevated.

FIGURE 1

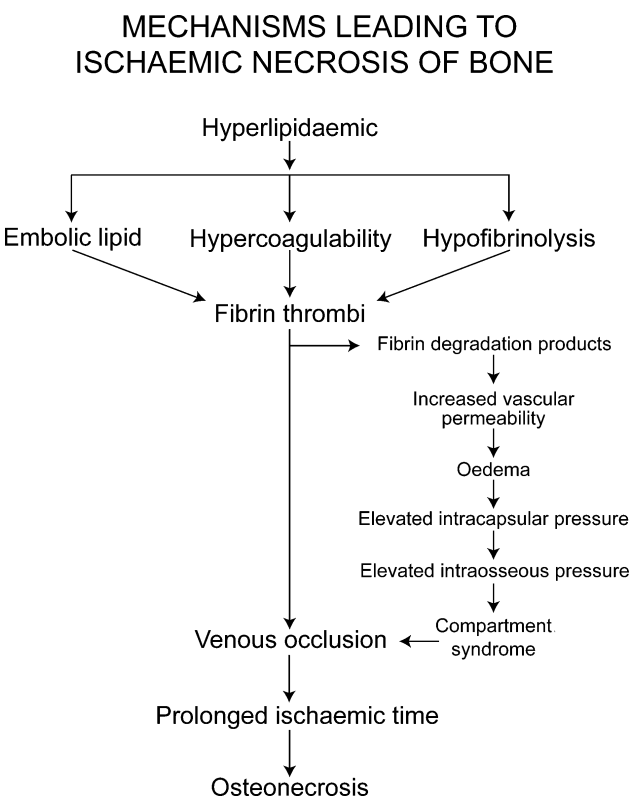

TABLE 1
(* is an abnormal result)

Haemoglobin studies

	Result	Normal range
Hb A2	2.6%	(1.5-3.5%)
Hb F	0.6%	(0.1-1.0%)
Hb H inclusions	negative	
Unstable HB	negative	
Qualitative Hb electrophoresis - electrophorectic mobility as Hb A/A		

Biochemistry studies

Cholesterol	6.2*	(<5.5 mmol/l)
Triglycerides	4.6*	(<2.0 mmol/l)
HDL cholesterol	1.20	(>1.0 mmol/l)
LDL cholesterol (cannot be calculated when triglycerides are 4.5 mmol or greater)		
Alpha fetoprotein	4	(<11 ku/l)
Apolipoprotein B	1.17*	(<1.00 g/l)
Lipoprotein a	0.1	(<0.3 g/l)
Blood glucose	5.3	(3.9-6.2 mmol/l)
Bilirubin	5	(3-20 umol/l)
Alkaline phosphatase	108	(35-135 u/l)
Alanine aminotransferase	29	(0-40 u/l)
Gamma glutamyl transferase	60	(10-65 u/l)
Albumin	45	(35-50 g/l)
Uric acid	0.30	(0.20-0.42 mmol/l)

Urine

24 hour creatinine	21.6*	(7.0-18.0 mmol/d)
24 hour urate	5.3	(<6.1 mmol/d)
24 hour urine volume	1.47 l	

It is known that Type II and IV Hyperlipidaemia predispose to AVN.[11] Jones has proposed that AVN could result from vascular stasis, hypercoagulability, endothelial damage (by free fatty acids) and intravascular coagulation.[12] Hyperlipidaemia has been linked to hypercoagulability and hyperfibrinolysis, which are further associated with a tendency to venous thrombosis.[13] It has also been proposed that osteoarthritis and AVN could result from obstruction of susceptible joint microvasculature by embolic lipid and thrombi.[14]

Fig 1 shows the proposed mechanism leading to ischaemic necrosis of bone (modified from Cheras).[14]

The pathways show how lipid abnormalities could promote fibrin thrombi, leading to occlusion of susceptible joint microvasculature. Increased vascular permeability subsequent to thrombolysis leads to elevated intracapsular pressure (ICP) and intraosseous pressure (IOP), establishing

a compartment syndrome and further compounding venous occlusion.

In OA's case the first X-ray (December 1996) was reported as showing only osteoarthritis. Nine months later he was diagnosed as AVN. The 1997 report, quoted above, can be read as suggesting that there was a bony abnormality in the 1996 film, but the words "at this stage" do not clearly refer to 1996. In any case there was generalised osteoarthritis of both hips on the 1996 X-rays.

Was the osteoarthritis due to years of lugging heavy weight around? Was it due to diving? These factors were considered unlikely to be responsible for his AVN.

Or was it, assuming the above scenario, that OA's AVN was due to hyperlipidaemia? This was considered likely. Could all these factors be synergistic in producing his condition? From the evidence it is hard to tell.

If the reader was referred such a case, what advice should be offered to the compensation tribunal? Also how would one expect the court to settle such a Workers Compensation claim?

To find out what happened in this case turn the page upside down.

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Months later

The worker was not happy about the medical report, that it could be his constitutional problems and went to his union.

The discussion that ensued convinced the worker and the Union representative that the settlement offered by the employing company was reasonable, especially as the company lawyer pushed the issue of his diving using hookah. The only person who suggested that it was work related was the orthopaedic surgeon.

Finally, he conceded that his ABN was not work related, took the money and left his employment.

We have no further information about this man.