SCINTIGRAPHIC DIAGNOSIS OF OSTEONECROSIS

Robert Ware

This will be a discussion about bone scanning and suggestions for screening. A bone scan involves the external imaging of radioactive tracers which are localised in the skeleton. The main tracer we use now is technetium diphosphonate which is readily available. The bone uptake depends on bone blood flow but primarily on bone metabolism. The tracer is taken up in areas of new bone formation and reflects regional osteoblastic activity. After intravenous injection the tracer is distributed to the plasma, to the non-bone extracellular fluid and also to the bone extracellular fluid and a small component of the tracer is deposited in stable lamellar bone. But the largest portion is deposited in active new bone, if the person has significant quantities of that. The remaining tracer, about 30%, is excreted in the urine. The examination involves a fairly low radiation exposure and one of the advantages is that, as one introduces all the radiation at the start of the procedure, taking additional views involves no additional radiation burden. If one is talking about screening young people yearly one has to think about radiation exposure because it may become significant. Because of low radiation dose scintigraphy is very well suited to whole body scanning and screening procedures.

The central feature in the pathogenesis of osteonecrosis is ischaemic and cell death involving the marrow, osteocytes and fat. This can be caused by vascular disruption as in trauma and it can be caused by other things, such as fat cells, which presumably cause vascular occlusion. Growing gas bubbles is the postulated mechanism of interference with the blood supply in the dysbaric disorders. Sickle cells, vasculitis, corticosteroids and some other mechanisms we do not fully understand also cause osteonecrosis.

Not all osteonecrosis is visible on an X-ray. I have an X-ray of a resected femoral head where there were two large necrotic areas but one cannot see them and that is on an X-ray of the specimen not of a patient. So obviously on an X-ray of a patient it is much more difficult to pick up any changes.

Any time one injures bone there is a reparative process starts fairly rapidly. The reparative process involves bone resorption and simultaneous new bone formation. At this stage the scan shows increased activity. We cannot pick cold areas in the femoral head, which represents the necrotic bone, because of the layer of new bone formation surrounding it. Figure 4 in Dr Shevland's paper (page) is the CT scan of a patient who had completely normal plain X-rays. Following a bone scan he had the CT which demonstrated his subcortical fracture.

If the area of bone necrosis and bone resorption is large enough, and particularly if the patient continues to weight bear, mechanical deficiencies appear as manifested by subchondral fractures. This leads very rapidly to articular surface collapse and osteoarthritis which is obviously a disaster in a young person.

That is the theory, how well does it stand up in practice? A study, which is fairly representative of a large number of clinical studies looking at X-rays versus bone scans, was done in 36 patients with lupus who were commencing steroids. The authors looked at bone scans, X-rays and bone marrow pressures, which they took as their gold standard. 27 joints developed avascular necrosis in this group. In the symptomatic group scanning was much more sensitive than X-ray as it was in the asymptomatic group. An interesting experimental model, which may have particular reference to dysbaric disorders, looked at rabbits who had microsphere emboli into their external iliac arteries. X-rays and scans were compared over a period of 12 weeks and these were compared to the histology. Very early on the X-ray is insensitive compared to the scan and the situation does not change very much by 12 weeks. So one is well down the pathogenic path by the time one starts picking up changes on X-ray. All the abnormal areas on the scan were shown at necropsy to be osteonecrosis. However there were 14 areas with osteonecrosis which were not detected on the scans. These were microscopic foci only and therefore probably of minor importance.

Because a large alteration in bone density is needed to demonstrate changes, plain x-rays are insensitive. The features of mechanical deficiency are specific. But one wants to be making a diagnosis before the patient is in a situation where joint replacement may be needed.

Bone scan by contrast is very sensitive. But, because almost anything one does to bone immediately sets up a reparative reaction, the specificity is poor. Recently developed tomographic capabilities (Single Positron Emission Computed Tomography or SPECT) have the advantage that one can section in three planes transaxial, coronal and sagittal. One is able to remove the influence of overlying and underlying activity in a particular plane of reference. Because of this one is able to find the areas of avascular necrosis, or cold areas which do not take up the tracer, when they are smaller in the earlier stage of the disease. So one has increased sensitivity using this technology and as one can find areas of avascularity in the midst of new bone formation, so one increases ones specificity. SPECT is able to be applied straight after the plain bone scan and therefore one is not increasing the radiation dose and one can be directed by the abnormalities on the plain scan.

SPECT studies do not have the resolution of magnetic resonance imaging (MRI) but one gets functional information. In one case where the femoral neck and head had areas of increased activity SPECT showed a large cold

area which was an area of avascular necrosis. From the ordinary scan one could not say what the etiology of the condition was but from the SPECT one could be very certain that it was avascular necrosis.

Coming to aseptic necrosis in commercial divers I must say I could find very little in the literature about the application of bone scans to commercial divers. A paper, in the Lancet, from the Decompression Sickness Central Registry looked at a large number of divers, almost 5,000 over a period of 5 years from 1976 to 1980, with many divers having multiple films. The overall prevalence of osteonecrosis from an X-ray diagnosis was 4.8%. The majority of these were head, neck and shaft lesions which are felt not to be of major significance in terms of producing disability. In fact they are often asymptomatic. Juxta-articular lesions were present in 1.2% and these are the ones that are potentially going to give the divers problems in the long run. Hip avascular necrosis, which is probably the most important site, has a very low prevalence in this group. Shoulders were slightly more commonly affected and these can also produce disabling features if there is articular collapse.

Should one screen divers routinely? I am going to be making suggestions based perhaps on inadequate information. The points against screening are firstly that the prevalence really is very low. Juxta-articular lesions are comparatively rare. It is, as far as I understand, totally unknown whether the presence of head, neck and shaft lesions predicts the future occurrence of juxta-articular lesions. Secondly, it is obviously going to be a fairly costly exercise to screen people. A standard bone scan costs around $300. That would put it in the same ball park as the CT and skeletal survey. The main argument for screening divers is the terrible morbidity of a young person having to have a joint replacement. People who have had decompression sickness are more likely to develop osteonecrosis. The incidence of dysbaric osteonecrosis was 10.7% with definite evidence of decompression sickness and this included the large majority of people with joint damage. Without decompression sickness the prevalence was only 1.7%. It was also apparent that multiple episodes of decompression sickness puts one at greater risk of developing osteonecrosis. They also found the depth of diving was an important predictive factor in developing osteonecrosis. For those diving less than 30 metres there was no osteonecrosis, going up to 15.8% in those diving to more than 200 metres.

I think a reasonable screening plan, if one accepts that it is a worthwhile thing to do and was effective in divers, is to take baseline X-rays of the humeri, the femora and the tibias. I think any screening plan has to include bone scan because of its sensitivity in the early phase of the disease. So a base line bone scan is also indicated. One should repeat the scan if the person develops skeletal pain after an episode of decompression sickness. Perhaps repeat the scan yearly if they are diving to more than 50 metres. As the specificity of bone scanning is not high the correlation is warranted. I think the primary method for following people, if one is going to do it, should be the bone scan. I should emphasise that I am not saying this from any published studies on the subject that I know of, it is just my general feeling about the sensitivity of bone scanning in diagnosing skeletal disorders.

The above has been adapted, by the SPUMS Journal Editorial Staff, from the transcript of a lecture presented at a meeting on Hyperbaric and Diving Medicine, sponsored by SPUMS and the Royal Hobart Hospital, 4th - 6th November, 1988, at the Royal Hobart Hospital, Tasmania.

Dr Robert Ware is a radiologist at the Royal Hobart Hospital, Hobart, Tasmania 7000, Australia.

MEDICOLEGAL ASPECTS OF AVASCULAR NECROSIS IN DIVERS

Audrey Mills

Introduction

I propose to talk briefly about the diving industry in Tasmania particularly abalone divers. I will use this as a background to the question of what legal remedies are available to divers who suffer avascular necrosis. I will then examine the legal problems involved in diagnosing the condition as that affects medical practitioners.

I would like to acknowledge the assistance of Mr D. Wolfe of The Department of Sea Fisheries for the information on the Abalone Industry in Tasmania.

Professional Divers in Tasmania

Abalone divers would represent the largest group of divers in Tasmania. The abalone industry in Tasmania is one of the biggest abalone industries in the world and is responsible for 22% of the world market. At present, there are 125 abalone licenced divers.

Commercial divers are employed by the Marine Board, CSIRO and Police Department and a few private companies working in salvage and construction areas. The total working in these areas would be approximately 15.

The abalone industry has very few regulations concerning work practices. Whilst diving tables are available and are recommended, it is doubtful that they are strictly