ASEPTIC NECROSIS OF BONE IN A COMPRESSED AIR WORKER

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This paper reports a detailed examination of three lesions of aseptic necrosis of bone occurring in a man who had worked in compressed air eleven years previously. Although two of the lesions were juxta-articular, in neither had the bone collapsed. Histology of only one similar lesion is reported in the literature (McCallum et al. 1966). Necropsy material is considered, together with radiographs and bone scans of the same lesions taken four years earlier. Similar changes have been shown to occur in commercial divers.

On the evidence of this case it becomes clear that radiography is limited in its use and that bone scans as an indicator of repair may be open to misinterpretation. There may be a place for operation before the onset of symptoms in these cases of aseptic bone necrosis.

In the last few years it has become evident that aseptic necrosis of bone can occur in compressed air workers and divers even if they adhere strictly to so-called "safe" decompression tables (McCallum et al. 1966). Because this condition, known also as caisson disease of bone, affects otherwise fit young men it is important to prevent disabling complications if possible.

The Medical Research Council's Decompression Sickness Research Team in Newcastle upon Tyne has established a central registry for the collection of radiological and medical information about compressed air workers and divers in order to elucidate the natural history of the condition. The team is also investigating methods for the early detection of bone necrosis, as well as studying the mechanisms by which joint disrupiton and secondary osteoarthritis develop in some of the patients. One object of this research is to devise schemes of management for the different stages of caisson disease of bone, in order that a more adequate repair of the damaged tissue can occur than seems possible at present.

Part of the difficulty in the study of this condition hitherto has been the almost total lack of human necropsy material. Our interest in compressed air workers and divers is now known nationally and we are being given the opportunity to study relevant post-mortem material when one of the men affected dies.

The man described in this paper has been known to us for several years, and the Central Registry in Newcastle holds a detailed account of his exposure to compressed air during his tunnelling experience, together with a radiographic survey of his bones and radioisotope scans of detected lesions.

CASE HISTORY

The man, who was born in 1934, worked in compressed air as a labourer during the construction of the Tyne Road Tunnel between August 1964 and February 1965. During this time he underwent fifty-three exposures to pressure, all but four of which were full eight-hour shifts, and the maximum working pressure was 30 pounds per square inch gauge. The British 1958 Decompression Tables were used (Ministry of Labour and National Service 1958). He was recompressed on one occasion for symptoms of decompression sickness (simple bends; type 1).

In March 1972 a routine follow-up skeletal survey was carried out on men who had been employed in this contract. This man was reported to have radiographic changes typical of aseptic necrosis in the head of the left humerus, the head of the left femur and the distal shafts of both femurs (Figs. 1 to 3). The lesions were reported to be of type A3, A3, B2 and B2 respectively, using the classification scheme developed by the Medical Research Council's Decompression Sickness Panel (Davidson and Griffiths 1970). In addition, tomographs were taken of the left hip to assess the extent of the lesion and a Sr⁸⁷m bone scan was also carried out. No asymmetry between the hips was found on the scan but there was increased uptake in the left shoulder.

In February 1976 the man was found dead in bed. The cause of death at necropsy was reported as asphyxia from inhalation of vomit. Gastric contents were present in the major airways and throughout the majority of the smaller bronchi. The blood alcohol concentration was

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estimated as 176 milligrams per cent. The other pathological findings were: slight left ventricular hypertrophy, severe coronary artery disease and focal fatty infiltration of the liver. The medical history in the case included references to the treatment of hypertension, depression and alcoholism.

At necropsy the left humerus and left femur were removed. Standard radiographs of each bone were taken in the antero-posterior and lateral positions and this was followed by tomography and microfocal radiography of all bone lesions seen on the standard radiographs. The bones were then sectioned and prepared for histological examination.

**FINDINGS**

A comparison of the radiographs and tomographs taken in 1976 with those taken in 1972 (Figs. 1 to 3) show that almost no change had occurred in the appearance of the lesions in this period.

The only abnormalities of the sectioned bones to be seen by the naked eye were in those areas already identified radiographically as being affected.

On histological examination the articular cartilage of the hip and shoulder joints appeared normal. However, the area involved in each lesion, as determined by the presence of trabeculae with empty lacunae, was greater than that shown radiographically (Fig. 4). A dense line, corresponding to the margin as seen in the radiograph, transversed each lesion. This line divided the area of dead trabeculae into two regions. In the region (labelled (a) on Fig. 4) between the dense line and the articular surface the dead trabeculae had not been covered by new bone and had retained their original architecture (Fig. 5). They were surrounded by marrow fat cells which had lost their nuclei, although by the use of special stains it was shown that fat was still present. In the
region outside the perimeter of the dense line, the dead trabeculae were covered by new bone. The dense line itself (labelled (b) on Fig. 4) was made up of dead trabeculae between which there was a thick meshwork of collagen and reticulin. In places this showed dystrophic calcification, both as granules and as confluent masses, as well as a mixture of rather disorganised new woven and new lamellar bone. In all three lesions, but most noticeably in those in the heads of the femur and humerus, much of the new bone was present in the form of arches between adjacent trabeculae (Fig. 6). Patent blood vessels were seen in the viable inter-trabecular marrow around each lesion.

At certain points around the shaft lesion large mononuclear cells of uncertain type were seen (Fig. 7). Much of the femoral cortex at the level of the shaft lesion was dead, as judged by the absence of osteocytes.

DISCUSSION

Although it has been suggested that the aseptic necrosis of bone seen in compressed air workers could be due to excessive consumption of alcohol, Zinn (1971) found no evidence to support the theory that alcohol is a factor in the pathogenesis of aseptic bone necrosis. In the workman reported in this paper the lesions were typical of those that occur in caisson disease and, we believe, resulted from his exposure to compressed air eleven years earlier.

A comparative study of the histology and radiographs at necropsy confirmed that the radiographic outline of each lesion was a result of the calcification and new bone formation which had occurred around the central area of necrosis. As there had been no radiographic change in the outline of the lesions between 1972 and 1976 it was concluded that, for this time at least, there had been no reduction in the size of each lesion.

Positive bone scans in aseptic necrosis of bone have been reported to be due to a repair reaction (Bauer 1971). The finding of a positive scan in the left shoulder of this
scans in bone necrosis may indicate some aspects of repair, this does not necessarily mean a concomitant reduction in the size of the lesion as determined radiographically. This observation is further supported by recent work carried out by this Department in which Technetium $^{99m}$-labelled diphosphonate has been used in scanning the bones of men with established lesions of caisson disease. It has been found that lesions which have shown an unchanging radiographic appearance for more than ten years may still be associated with positive radioisotope scans.

The histological changes that occur with each lesion of aseptic necrosis of bone are usually interpreted as evidence that bone necrosis has been followed by a certain amount of revascularisation and repair, but that the process has halted before completion (Welfling 1971; Jaffe 1972; Catto 1976). The cause for this apparent failure to repair completely remains unknown. The repair process may outstrip its blood supply, or there may be a critical size to a lesion that can be repaired, and this critical size has been exceeded. For the present, however, the significance of the histological changes and the mechanisms of their production remain speculative.

We believe that not enough attention has been paid to mechanical factors in determining the changing appearance of these lesions. All lesions of aseptic necrosis of bone in compressed air workers are initially symptomless and many may remain so. This means that repair of the necrotic bone, which presumably begins soon after the causal event, proceeds in the presence of full load-bearing. As one of the principal functions of bone is that of support, it might be expected that reparative changes occur to replace the mechanical strength lost from ischaemia of some of the trabeculae. For this reason the histological finding that much of the new bone is present in the form of arches at the margins of the lesions deserves comment (Fig. 8). While the development of such a pattern may reflect the mechanism by which new bone forms in association with the blood vessels at the viable margins of the lesions, it is interesting, and perhaps significant, that this arrangement also provides the most effective structural support at these sites for continued load-bearing. Further evidence that might support the hypothesis that the principal object of repair is to re-establish mechanical support is the constant finding that new bone formation occurs apparently in the almost complete absence of osteoclastic activity and dead bone removal (Catto 1976).

As dead bone has different mechanical properties from living bone (Stevens 1963), continued load-bearing will result in alteration of the stresses and strains being set up in the trabeculae at and around the junction between living and dead bone. Mechanical forces are known to influence bone formation (Wolff 1892; Bassett 1962, 1968; and Young 1975), and it is possible that the resultant alteration in stresses and strains may be responsible for some of the changes seen. Whether the changes that occur around the lesions result from vascular, mechanical or other factors, there is no doubt that once established further revascularisation of the necrotic mass is unlikely. Therefore, the deposition of new bone in the form of arches may be a causal factor in the arrest of the repair process.

Another interesting histological finding in this case was the presence of many large mononuclear cells, of uncertain identity, at points around the femoral shaft lesions. Eight cases of sarcomata have now been reported in association with old bone infarcts, and in three of these cases the men were known to have been caisson workers (Dorfman, Norman and Wolff 1966; Mirra, Bullough, Marcove, Jacobs and Huvos 1974). The shortest interval between the infarct and a recognised tumour was seventeen years. In two cases the tumour was reported.  

Fig. 8
Diagram of marginal region of a lesion.
as a malignant "fibrous histiocytoma" and in one it was a fibrosarcoma. Although these figures are of no statistical significance, the long interval between initial diagnosis and malignant change suggests that long continuing surveillance of shaft lesions would be wise.

Until now it is only the juxta-articular lesions in caisson disease of bone which have given rise to a need for surgical intervention. Collapse of the necrotic trabeculae leads to disruption of the joint contour, the onset of symptoms, and later the development of disabling arthritis. The hip and shoulder are the joints affected. The incidence of collapse is not known, nor is the reason why collapse may occur within months in some cases and not for years in others. In the absence of symptoms the policy of management has been one of non-interference, but this is not totally satisfactory because operation delayed until after bone has collapsed does not necessarily result in the complete relief of symptoms or the total restoration of function. Since collapse occurs through the necrotic trabeculae it might be expected that revascularisation, which will result in the reinforcement of dead trabeculae by appositional new bone, might prevent collapse from occurring. Boetwchar, Bonfiglio and Smith (1970) reported a 79 per cent success rate in restoring viability to the non-traumatic femoral head by making drill holes across the margin of the lesion and inserting bone grafts around which revascularisation could take place. These authors stressed that the overall result was better when the procedure was carried out before collapse occurred.

With a normal joint contour and normal articular cartilage as in this case, such surgical prophylaxis would have much to commend it. The effective block to more extensive revascularisation and repair, demonstrated by our histological studies, would lend support to this concept of earlier surgical intervention. However, until collected information on compressed air workers and divers can define more precisely the likelihood of non-symptomatic lesions becoming painful and disabling the present policy of wait and see is likely to continue.

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REFERENCES


