

# STEROID-INDUCED AVASCULAR NECROSIS OF THE HEAD OF THE HUMERUS

## NATURAL HISTORY AND MANAGEMENT

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Ninety-five patients with steroid-induced avascular necrosis of bone have been personally treated by the author. Of these, eighteen had a lesion of the head of the humerus, on one or both sides. The conditions for which the steroids were given included post-transplantation, lupus erythematosus, glomerulonephritis and asthma. The characteristic lesion began as a subchondral osteolytic area which frequently progressed to collapse. The articular cartilage divided from the subchondral bone, either becoming detached as a free cap or at a later stage reattaching. In some cases the lesion was minimal and the symptoms were slight. Conservative treatment has consisted of pendulum exercises and avoidance of abduction, particularly against resistance. In fourteen patients this led to satisfactory function with only intermittent symptoms. Four patients required replacement of five humeral heads with Neer's prostheses. After one to seven years the results of all five were classified as excellent in terms of absence of symptoms and a free range of movement.

Cortisone has proved to be an extremely useful drug capable of profoundly affecting the course of many diseases. However, in terms of complications and side effects the price which the patient must pay has at times been high, and the medical profession has been faced with the problem of avascular necrosis of bone. So far there has been no report in the literature of the natural history and management of steroid-induced avascular necrosis of the head of the humerus. This paper outlines the author's experience of eighteen cases.

### MATERIAL

A total of ninety-five patients suffering from steroid-induced avascular necrosis of bone have been treated. The bones affected have been the femoral head (ninety-one), the head of the humerus (eighteen), the distal femur or proximal tibia (eighteen), the talus (six) and the capitulum (three).

The eighteen patients with avascular necrosis of the head of the humerus had the following conditions: renal homotransplantation (eight), lupus erythematosus (five), asthma (two), glomerulonephritis (one), hypopituitarism (one), and Guillain-Barré syndrome (one).

In two patients both humeral heads were affected without radiographic evidence of lesions in other bones. The other sixteen patients also had at least both femoral heads affected; two had involvement of both femoral heads and both distal femoral articular surfaces. One patient showed avascular necrosis of both humeral heads, both femoral heads, both distal femoral articular surfaces, both proximal tibial surfaces, one talus and one capitulum.

### CLINICAL MANIFESTATIONS

The exact time of onset of symptoms after the commencement of steroid therapy was difficult to determine. All eighteen patients complained of pain developing gradually in the affected shoulder but not before six months of steroid therapy. The longest interval was eighteen months. In two patients symptoms appeared in the shoulder only when they had to use crutches because of involvement of both femoral heads, and then rapidly progressed to become quite disabling. Characteristically the pain was less in those patients for whom steroids had been continuously administered, and worse if the steroid had been discontinued because of improvement in the systemic disease.

Pain on movement was the usual complaint, and in general rest pain and night pain were not prominent. Late in the course of the disease an audible click on movement was often heard in the shoulder; the click was always painful. The active range of movement gradually decreased, mainly because of pain. On the other hand passive movement was nearly always free until late in the course of the disease. In three of the patients who required operation, passive lateral rotation was eventually reduced to 5 degrees, with a decrease in gleno-humeral abduction to 70 degrees.

### RADIOLOGICAL AND PATHOLOGICAL FEATURES

The radiographic pattern of progression seen in the femoral head was also seen in the humeral head (Merle d'Aubigné, Postel, Mazabraud, Massias and Gueguen 1965; Cruess, Blennerhassett, MacDonald, MacLean and



FIG. 1

A radiograph showing a thin subchondral radiolucent area just visible at the place where the outlines of humeral head and glenoid meet, taken eight months after the commencement of steroid therapy for lupus erythematosus.



FIG. 2

A radiograph showing separation of a layer of subchondral bone from the underlying area of collapsed bone eight months after renal homotransplantation.

Dossetor 1968; Fisher and Bickel 1971). Subchondral resorption of bone first appeared in a well localised area of the head (Fig. 1). This was accompanied by collapse of the underlying bony architecture, giving a characteristic appearance in which the calcified subchondral bone with some attached bony debris was left articulating with the glenoid over what appeared to be an empty space (Fig. 2). Pathologically, this resembled the organisational diagrams of Solomon (1973), showing articular cartilage to the deep surface of which necrotic bone was attached (Fig. 3) and then an empty space. The superficial bone beneath this again was necrotic, as was the marrow. There was then encountered an area of active resorption of necrotic bone with new bone formation and fibrous marrow (Fig. 4), immediately below which was normal bone and marrow. Thus the subchondral lesion was well localised and did not represent total head necrosis. No local or general osteoporosis was visible on standard radiographs. Later the articular cartilage separated from the underlying bone, and it is probable that the click that was often heard represented indentation of this cartilage (Fig. 5). If the symptoms allowed a conservative approach, the separated cartilage would in time become reattached to underlying bone (Fig. 6). With the passage of time degenerative arthritis with sclerosis and spur formation would occur if incongruity of the joint surfaces was severe.

Five humeral heads were available for pathological study from two to four and a half years after treatment; four were stained for intravascular fat according to the method of Jones and Sakovich (1966). In three, persistent intravascular fat in an area of necrosis was visible (Fig. 7). In the fourth, from a patient whose symptoms were basically due to degenerative arthritis, revascularisation had occurred in the entire head and no fat was found.

The site of the area of deformed articular cartilage was characteristic in all cases. The glenoid cavity indented

the humeral head over the area of contact with the glenoid at 80 to 90 degrees of gleno-humeral abduction. Bio-mechanical studies have shown that the greatest stress is exerted across the joint in this position, and it is probable that this determines the site of the anatomical lesion (Inman, Saunders and Abbott 1944).

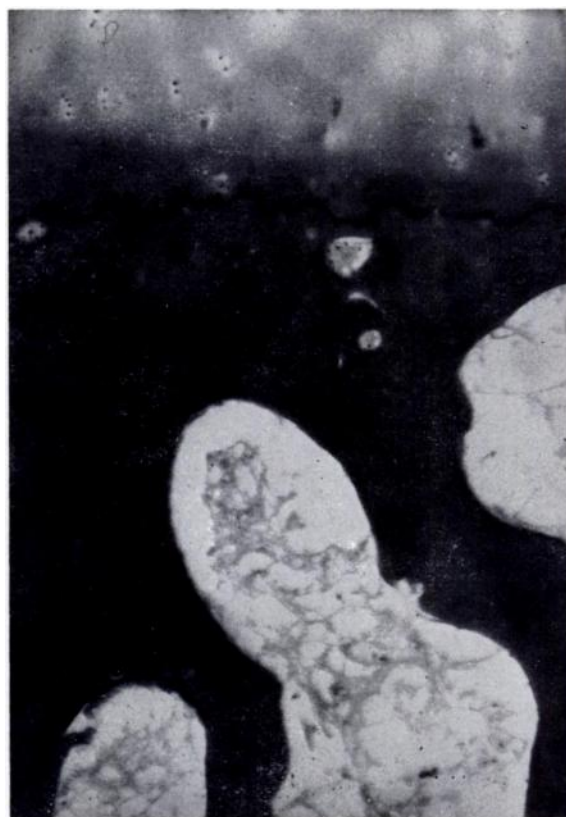


FIG. 3

A photomicrograph of the articular cartilage and subchondral bone of an avascular humeral head removed two years after the onset of symptoms following steroid therapy for asthma. (Haematoxylin and eosin,  $\times 120$ .)

## CLINICAL MANAGEMENT

It is difficult to predict early in the course of the lesion the degree of deformity that will occur in the humeral

discouraged from active abduction or heavy work with their arms. The rationale was, of course, to diminish the stresses across the joint in the hope of minimising collapse and avoiding serious degenerative changes when

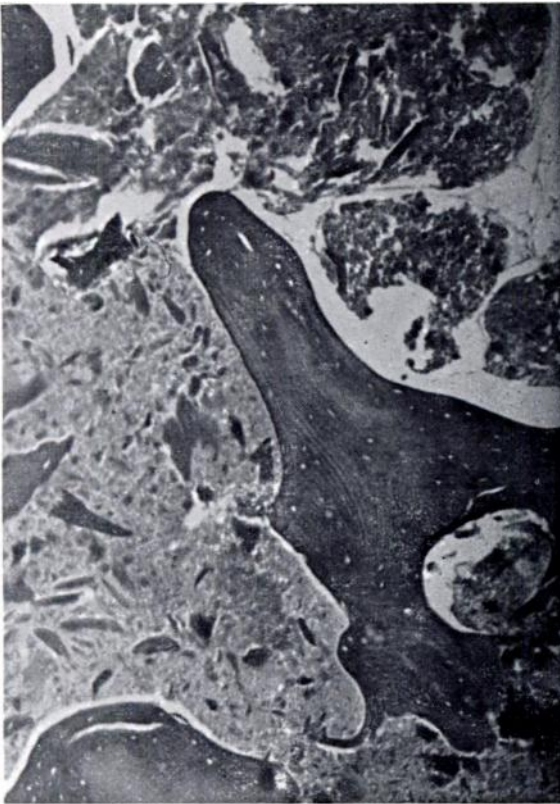


FIG. 4

A photomicrograph showing the zone of invasion of granulation tissue and early resorption of necrotic bone. The necrotic area is above and the normal bone is below. (Haematoxylin and eosin,  $\times 120$ .)



FIG. 5

A photograph of a specimen obtained at operation showing apparently normal articular cartilage separated from the underlying humeral head. The indentation on the left corresponds to the glenoid margin. This patient was being treated for lupus erythematosus and symptoms had been present for eighteen months.

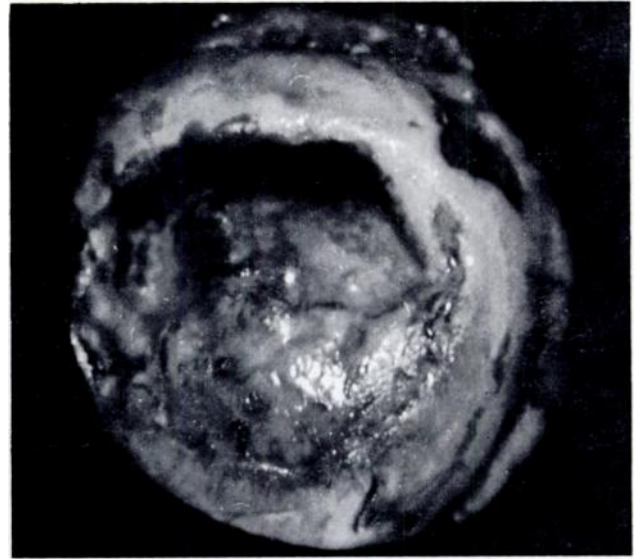


FIG. 6

A photograph of a specimen obtained at a later stage than that shown in Figure 5. The round indented area corresponded to the glenoid margin. The articular cartilage appeared to have become re-attached to the underlying bone.



FIG. 7

A photomicrograph taken from the humeral head of the patient mentioned in Figure 5. It demonstrates a globule of intravascular fat in necrotic bone immediately beneath live cartilage. (Oil red O and toluidine blue,  $\times 300$ .)

head, and equally difficult to assess how much disability will arise. For these reasons the early management in these cases has been conservative. All patients were instructed how to try to maintain the range of movement of the shoulder joint with pendulum exercises, but were

revascularisation and reattachment occurred. Because these patients suffered from serious systemic illness, some difficulties were encountered with the programme. In particular, when painful hip lesions occurred simultaneously the patients had to use crutches, and in two

instances there was an immediate exacerbation of symptoms in the shoulders. A third patient with a minimal shoulder lesion was able to walk on crutches without serious pain. In thirteen patients, however, the regime was successful in maintaining an adequate range of painless movement for ordinary use.

The follow-up of these patients revealed that they fell roughly into three groups. The first group contained eight patients in whom the anatomical deformity was mild and allowed a return to full activities, including

use of the arm overhead. The range of motion was either full or nearly so. In some instances tennis and badminton were possible (Fig. 8).

The second group of six patients had more severe deformity of the humeral head with limitation of active movement and inability to use the arm above the shoulder. However, their daily living did not require extensive use of the shoulder, all continued on a maintenance dosage of steroid which appeared to decrease the amount of pain, and no ongoing therapy was required (Figs. 9 and 10).

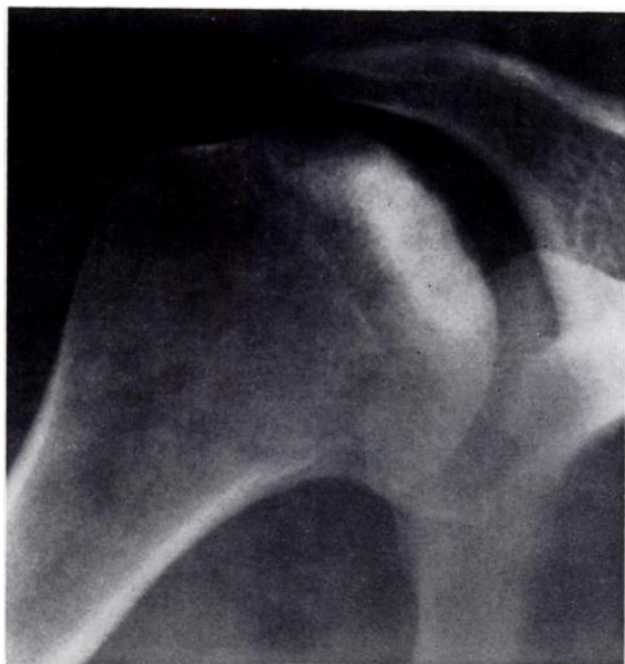


FIG. 8

A radiograph of a healed lesion in a patient previously treated for lupus erythematosus, taken two years after the onset of symptoms, when the patient was fully active.



FIG. 9

The radiological appearance of the shoulder of a patient suffering from asthma. Steroids were being continued and the symptoms were not disabling.



FIG. 10

Radiographs of a shoulder five and a half years after renal homotransplantation. The patient was able to carry on full activities with the shoulder despite some pain.

TABLE I  
A RECORD OF GLENO-HUMERAL MOVEMENT IN FOUR PATIENTS TREATED BY REPLACEMENT

| Age at operation (years) | Sex | Diagnosis           | Before operation* |         |           |                  |                   | After operation |          |           |                  |                   | Follow-up           | Pain    |          |
|--------------------------|-----|---------------------|-------------------|---------|-----------|------------------|-------------------|-----------------|----------|-----------|------------------|-------------------|---------------------|---------|----------|
|                          |     |                     | Abduction         | Flexion | Extension | Lateral rotation | Medial   rotation | Abduction       | Flexion  | Extension | Lateral rotation | Medial   rotation |                     | Pre-op. | Post-op. |
| 47                       | F   | Lupus erythematosis | A —<br>P 60       | —<br>70 | —<br>40   | —<br>5           | —<br>T12          | 95<br>100       | 80<br>80 | 70<br>70  | 20<br>20         | T10<br>T8         | 6 years             | 4+      | 0        |
| 27                       | F   | Asthma              | A —<br>P 50       | —<br>40 | —<br>40   | —<br>0           | —<br>T12          | 100<br>110      | 90<br>90 | 70<br>70  | 20<br>20         | T10<br>T9         | 6 years             | 4+      | 1+       |
| 18                       | M   | Post-transplant     | A† —<br>P 70      | —<br>70 | —<br>60   | —<br>10          | —<br>T12          | 95<br>95        | 70<br>80 | 60<br>60  | 25<br>25         | T8<br>T7          | 1 year§<br>6 weeks  | 3+      | 0        |
|                          |     |                     | A‡ —<br>P 70      | —<br>60 | —<br>60   | —<br>15          | —<br>L1           | 100<br>120      | 90<br>90 | 70<br>70  | 40<br>40         | T9<br>T9          | 1 year§<br>8 months | 3+      | 0        |
| 17                       | F   | Lupus erythematosis | A —<br>P 80       | —<br>60 | —<br>50   | —<br>0           | —<br>T12          | 115<br>115      | 60<br>70 | 60<br>60  | 40<br>40         | T9<br>T7          | 2 years             | 4+      | 0        |

KEY—Pain: 0 = none; 1+ = occasional pain on strenuous activity; 2+ = pain uniformly on strenuous activity; 3+ = pain on minimal activity; and 4+ = pain at rest.

A = active, P = passive movement. † Right ‡ Left.

\* Pre-operative movement recorded is passive only due to variable active movement depending on pain.

§ Patient deceased due to septicaemia from gram-negative organisms.

|| Medial rotation measured by noting the spinous process to which the hand can be placed.

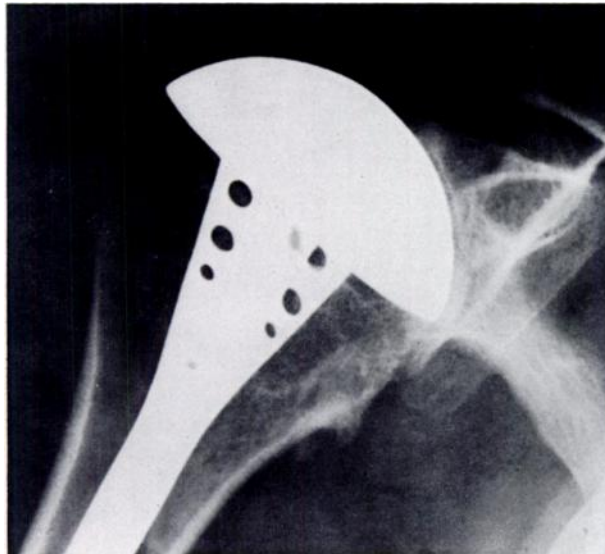


FIG. 11

A radiograph of the right shoulder of a patient suffering from asthma six years after insertion of a Neer's prosthesis. The ranges of active and passive movement were nearly full, with occasional pain on strenuous activity (see Table I).

The third group of four patients required five operative procedures because of pain so persistent and severe that it interfered with the normal enjoyment of life. All had restriction of active movement as well as some restriction of passive movement (Table I). All four had insertion of a replacement of the Neer type between two and four and a half years from the beginning of steroid therapy.

The operations were carried out as described by Neer (1955, 1974) with particular attention to maintaining the normal amount of retroversion of the humeral head (Fig. 11). No operative complications were encountered and the end-results have been most gratifying. The final range of motion approached normal in all cases (Table I) and all four patients had complete relief of pain. One patient was able to return without difficulty to intermittent crutch walking because of hip disability. On the basis of this experience, it is felt that prosthetic replacement of the humeral head can be recommended for steroid-induced avascular necrosis. The excellence of the results is probably due to preservation of the glenoid articular cartilage, which appeared to be essentially normal in all instances.

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