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COMPRESSION FRACTURE OF THE FEMORAL HEAD IN ASSOCIATION WITH CORTISONE THERAPY

By

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Osteoporosis in Cushing's disease was shown by Albright to be due to excessive production of adrenocortical steroids, such as hydrocortisone. Therapeutic stimulation of the adrenal cortex with pituitary adrenocorticotrophic hormone ACTH, or administration of adrenocortical substances, such as cortisone or hydrocortisone-like substances, may also produce osteoporosis.

The anti-inflammatory and anti-allergic effect of cortisone and hydrocortisone has resulted in a wide therapeutic use of such preparations. They are believed to act directly upon the tissue cells. Though their mode of action is not properly understood, they are believed to interfere with protein synthesis, with deficient regeneration of the albumin matrix of the bone tissue. The skeleton is therefore not able to absorb sufficient calcium to satisfy the requirements of regeneration of bone tissue. Prolonged excess of adrenocortical substances leads to osteoporosis with the risk of spontaneous fractures. The risk of such fractures is, of course, greater in pre-existing osteoporosis, resulting from disuse or ageing, and in postmenopausal osteoporosis or rheumatoid arthritis.

Spontaneous fractures owing to osteoporosis in rheumatoid arthritis are well known (*Baer* 1941). Since 1950 a number of spontaneous fractures have been reported in patients undergoing cortisone therapy for rheumatoid arthritis. (*Boland & Headly* 1950, *Steinbrocher et al.* 1951, *Boland* 1952, *Demartini et al.* 1952, *de Sèze et al.* 1953, and others).

Spontaneous fractures have also been described in association with cortisone therapy for other diseases (*Soffer & Bader* 1952, *Teicher & Nelson* 1952, case 39221 from Massachusett General Hospital 1953, *Irwin et al.* 1954, and *Eisenstedt & Cohen* 1955). The diseases in these cases were: pemphigus vulgaris in 2, bronchial asthma in 2, and lupus erythematodes 1.

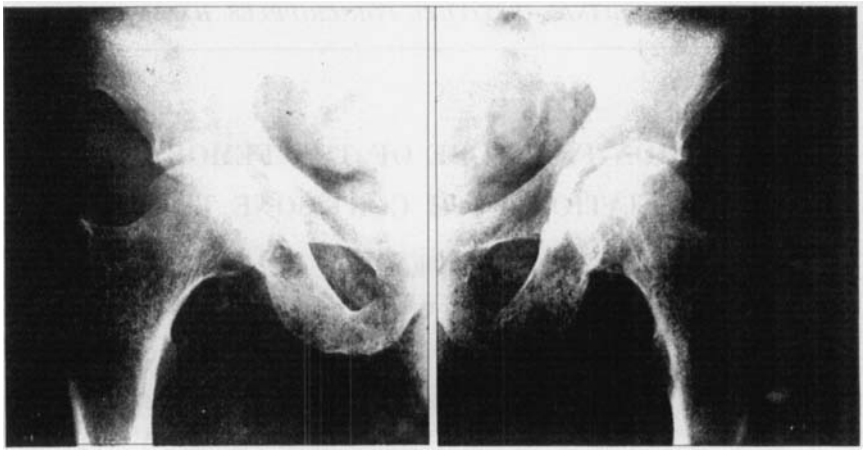

Fig. 1.

In view of the relatively few publications of spontaneous fracture in association with cortisone treatment, it was considered legitimate to report on a further 2 cases. In addition, the form of osteoporotic fracture in these two cases deserves special attention.

Case 1. The patient, a previously healthy farmer, had been receiving cortisone for about one year because of psoriasis. During this period the dosage had ranged between 50 and 200 mg. a day.

In June 1955 his grandchildren visited him at the hospital where he was staying. He went for a walk with them in the park. After strolling about with them for a

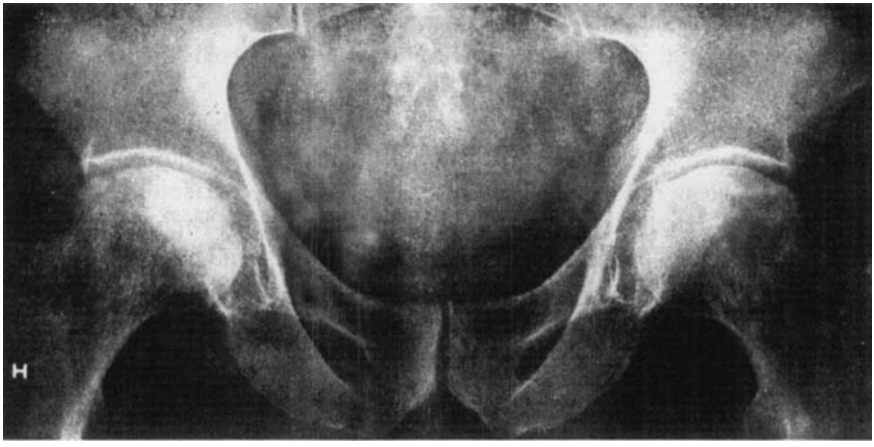

Fig. 2.

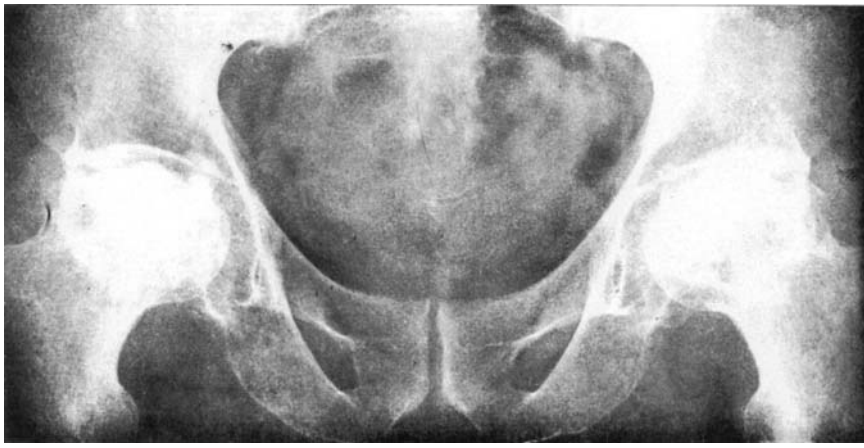

Fig. 3.

couple of hours he had such severe pain in both groins that he had to take a taxi back to the hospital.

Roentgen examination of the hips gave the picture in Fig. 1. Cortisone treatment was stopped, but the examination was not considered to indicate any other particular treatment or instruction and the patient was sent home. Follow-up at the outpatient department in September 1955 (Fig. 2) showed compression of both femoral heads. The patient was informed that the hip pain might have been due to previous treatment with cortisone. The pain gradually became more intense and in May 1956 and in April 1957 he was examined at his local hospital. The latter examination showed further compression of the femoral head on either side (Fig. 3),

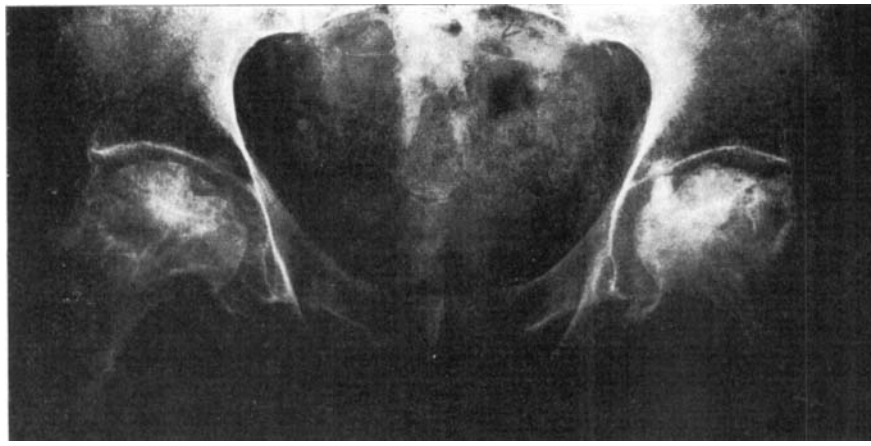

Fig. 4.

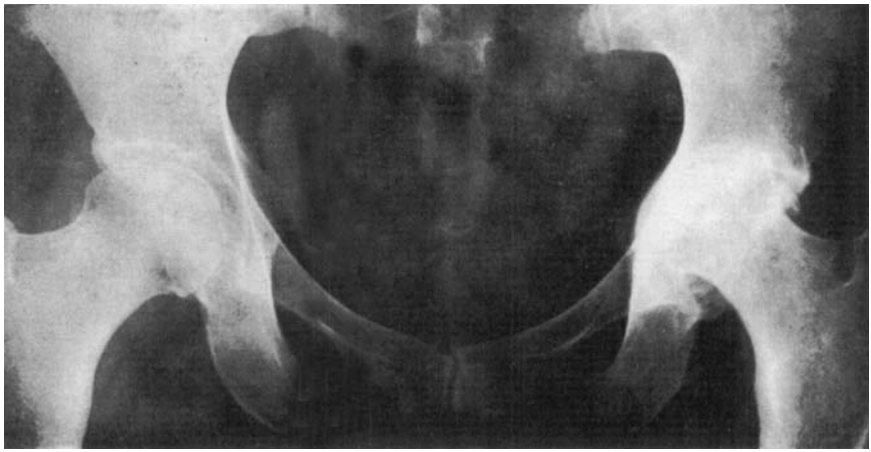

Fig. 5.

and in May 1957 the patient was referred to the Orthopaedic clinic, Hälsingborg, for investigation.

Walking was obviously difficult: he walked with a stoop and used two sticks. The range of mobility of the both hips was markedly decreased, but there was no malposition or contractures. He refused operation and was therefore instructed to rest in bed at home. He was re-examined in August 1957 (Fig. 4) when he reported that the hip pain was no longer so intense.

Case 2. The patient was a housewife, aged 53, who for many years had chronic polyarthritis. During the last 8 years she had been treated on various occasions

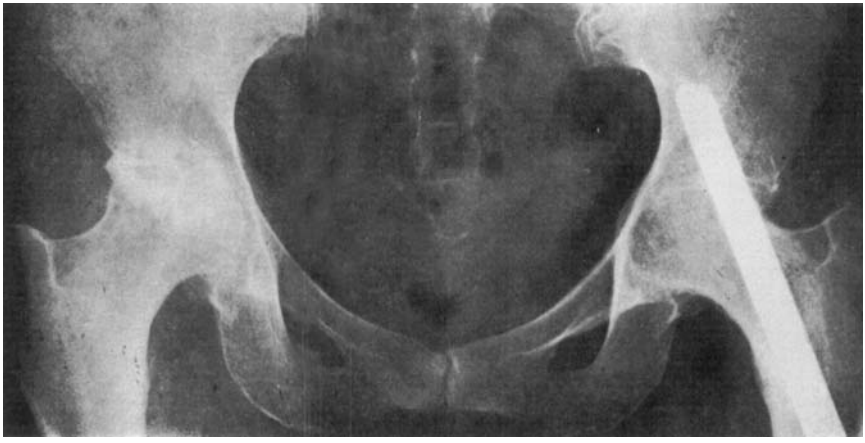

Fig. 6.

with cortisone. Since May 1958 she had had severe pain in the left hip. Examination in June 1958 revealed flexion and adduction contractures of the left hip joint, and roentgenography showed severe arthrosis (Fig. 5).

Arthrodesis of the left hip was done. The postoperative course was complicated by polyserositis. In the treatment of this complication cortisone (Kenacorte) was instituted one month after the operation. After gradual withdrawal of cortisone the patient was sent home in September 1958: she was then ambulant. At follow-up in October the state of the joints was found to be satisfactory.

In January 1961 the patient re-appeared complaining of pain in the right, unoperated hip. During the last two years she had been almost continually treated with cortisone (Kenacorte, 4 mg. three times daily). Roentgen examination revealed compression of the femoral head (Fig. 6).

DISCUSSION

It appears that the frequency of spontaneous fractures in patients with rheumatoid arthritis has increased since the introduction of cortisone and ACTH therapy. Evaluation of the significance of cortisone in the causation of such fractures in individual cases of rheumatoid arthritis is, however, difficult because calcium loss and osteoporosis are not uncommon accompaniments of this disease, and spontaneous fractures are not rare.

Stronger evidence for cortisone favouring or causing osteoporosis is therefore provided by the development of osteoporosis in association with cortisone treatment for diseases not otherwise known to be accompanied by osteoporosis, such as the present case of psoriasis.

Of orthopaedic and roentgenologic interest is the nature of the fracture, i.e. compression of the crown of the femoral head which in advanced stages resembles necrosis of the head after medial fracture of the neck.

Before and during the development of the fractures both patients had been ambulant and had to their knowledge not sustained any trauma capable of causing fracture of the femoral neck with subsequent necrosis of the femoral head. The greatest strain to which the bone had been exposed was the vertical load when walking.

Hirsch & Brodetti (1957) focused attention on the tensile strength of the proximal end of the femur and in a mechanical-experimental study they were able to show that application of a certain type of pressure to the femoral neck invariably caused the same type of fracture of the neck and that a force of 400 to 1100 kg. was sufficient to produce such fractures. Vertical loading produced a vertical fracture in the middle of the femoral neck. Fractures of this type can be excluded in the pre-

sent cases. Even if detection of a fracture, if any, in the roentgenogram might have been made impossible by the rarefaction of the skeleton, one would have expected demonstrable changes during the course of healing, if such fractures can heal in ambulant patients. But no such changes could be demonstrated. In addition case 1 was followed from the onset of the symptoms. Roentgen examination at that time (Fig. 1) and 4 months later (Fig. 2) showed that fracture had occurred with compression of the head during the interval between the two examinations. This shows that spontaneous fracture of the femoral head may occur in severe osteoporosis.

Greig (1931) and *Watson Jones & Roberts* (1934) stressed that osteoporosis can only develop in bones with a good blood supply to the bone substance. *Trueta & Harrison* (1953) and *Claffey* (1960) have shown that the calotte of the femoral head is supplied by a strikingly rich arterial network.

It may therefore be assumed that the demands placed on metabolism and on osteoblastic activity in this part of the head are greater than in the rest of the head and neck. Consequently impaired osteoblastic function, as in osteoporosis, will pave the way for a more rapid development of the osteoporosis with a shift of locus minoris resistentiae to weight bearing from the femoral neck to the calotte of the femoral head.

SUMMARY

Two cases are described in which unilateral and bilateral spontaneous fractures of the calotte of the femoral head occurred in association with cortisone treatment.

It is suggested that the rich vascular supply to the calotte of the femoral head is responsible for the relatively rapid advance of the osteoporosis in this region with a shift of the locus minoris resistentiae to weight bearing from the femoral neck to the calotte of the femoral head.

RESUME

L'auteur rend compte du cas de deux malades chez lesquels, en liaison avec un traitement à la cortisone, il s'est produit une fracture spontanée, bilatérale et unilatérale, respectivement, de la calotte de la tête fémorale.

L'auteur considère que c'est l'abondante alimentation sanguine de la calotte fémorale qui est la cause du développement plus rapide d'une

porosité osseuse dans cette région pouvant donner lieu à un décalage de la résistance de la tête fémorale à la charge du col fémoral.

ZUSAMMENFASSUNG

Der Verfasser berichtet über zwei Patienten, die sich im Zusammenhang mit einer Cortisonbehandlung eine doppelseitige, beziehungsweise einseitige Spontanfraktur der kranialen Femurkopfkalotte zuzogen.

Die reichliche Blutversorgung innerhalb der Femurkopfkalotte sieht der Verfasser als die Ursache einer sich rascher entwickelnden Osteoporose in dieser Region an. Diese schafft die Voraussetzung für eine Verschiebung des punctum minoris resistentiæ während der Belastung vom Schenkelhals zur Kopfkalotte.

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