LESSON OF THE MONTH

The diagnostic challenge of acute polyarthritis

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Case Report

Eight years ago a 22 year old Indian Lady was seen at the local Casualty Department. She had been knocked unconscious by a motorcycle and suffered a severe head injury. In addition, she sustained fractures to the right clavicle and the right fibula. She was transferred to a neurosurgical unit for an urgent computed tomography (CT) brain scan, which showed a left frontal contusion. She was transferred back to the referring hospital the next day, as no neurosurgical intervention was required. Her progress was slow and after five days she remained semiconscious and irritable. Another CT brain scan was performed, which indicated that the left frontal haematoma was resolving. She remained on the neurosurgical unit and was treated with 4 mg oral dexamethasone initially four times a day and received a total dose of 252 mg over a 30 day period.

Six months after receiving the steroids, the patient complained of pain in both ankles and, five months after this, both knees were painful, especially on weight bearing. She later complained of both hips being painful; the right shoulder and the left elbow were also involved. On examination, mild synovitis of both ankles and bilateral knee effusions were documented. She also demonstrated restricted movement and pain in both hips.

Radiographs at this time suggested early avascular necrosis of the hips and the clinical picture was that of a polyarthritis. Laboratory investigations showed haemoglobin 121 g/l, leucocyte count $7.4 \times 10^9/l$, erythrocyte sedimentation rate 23 mm/1st h, rheumatoid arthritis latex negative, antinuclear antibody negative, immunoglobulins normal. Arthroscopy of the right knee showed minimal erosion of the articular surface and minimal inflammation of the synovial lining. A radioisotope bone scan was non-specific and reported 'activity is increased in the elbows, shoulders, hips, knees and ankles suggesting active involvement', which would suggest specific areas of increased bone turnover.

The patient was reviewed regularly and at one stage was thought to be developing rheumatoid arthritis. She was managed with non-steroidal anti-inflammatory drugs and hydrotherapy.

Three years after the accident, the patient went abroad and presented subsequently two years later with worsening symptoms. Her pain had spread to both shoulders, her left elbow felt worse, and her radiographs showed avascular necrosis in the shoulders, left elbow, and both hips (figure).

Further investigation did not reveal any other predisposing factor such as congenital epiphyseal dysplasia, diabetes mellitus, sickle cell disease, or systemic lupus erythematosus. There was no use of steroids before her head injury.

The clinical signs in her hips have since deteriorated, with positive Trendelenburg's signs, and the patient is currently considering hip arthroplasty.

Discussion

To our knowledge, this clinical problem has not been reported in the past nine years. This young patient developed significant joint problems as a result of steroid treatment for her head injury. As her disease was of a symmetrical nature, she was initially thought to be developing an early inflammatory arthritis. It has been reported that avascular necrosis can occur independently of steroids in patient with an inflammatory arthritis,¹ and it was not until more than 12 months after our patient received her course of steroids that the connection between her head injury, the steroids, and her arthritis was finally made. We were particularly impressed by the extent of joint involvement in this patient, and how closely it mimicked an acute polyarthritis.

The pathogenesis of avascular necrosis attributable to steroids has been considered the result of a venous stasis phenomenon, whereby osteocytes become loaded with fat or intramedullary fat cells enlarge, thus building up pressure within the closed system. An increase in osseous pressure above the arteriolar pressure compromises the arterial blood supply, causing bone to die (K Kawai *et al*, Proceedings of the ORS 29th Annual Meeting, Anaheim, 1983).

Avascular necrosis has been well documented in patients receiving long term steroid treatment, particularly in renal transplant patients,² but in addition, several case reports have highlighted the complication of avascular necrosis occurring following short term steroid use.^{3,4} These cases have indicated that, where dexamethasone was used in circumstances similar to those of our patient and in a similar dosage, avascular necrosis has occurred.

There are many applications for the use of steroids in clinical practice, whether for long term or short term use. However, the value of using dexamethasone in head injuries or in acute strokes is not proven.⁵ Certainly, its use in this young patient was associated with substantial morbidity.

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Crescent sign in the left shoulder (A), and avascular necrosis of the left elbow (B) and both hips (C).

The lesson

- Correct diagnosis and potential treatment⁶ may be delayed when avascular necrosis involves multiple joints and presents as a clinical polyarthritis.
- Marked morbidity is the consequence, and this complication should be considered in patients treated with short courses of steroids.
- Investigation with magnetic resonance imaging may expedite correct diagnosis,⁷ and is now becoming the technique of choice.

1 Mitchell A, Rothman W, Tallett P, Williams K, Pitt P. Survey of the long term incidence of osteo-

necrosis of the hip and adverse medical events in rheumatoid arthritis after high dose intravenous methyl prednisolone. *Ann Rheum Dis* 1988; 47: 930-3.

2 Ibels S L, Alfrey AC, Huffer W E, Weil R. Avascular necrosis of bone following renal transplantation; experience in 194 transplant recipients and review of the literature. *Medicine, Baltimore* 1978; 57: 25-40.

3 McCluskey J, Gutteridge D H. Avascular necrosis of bone after high dose of dexamethasone during neurosurgery. *BMJ* 1982; 284: 333-4.

4 Taylor L J. Multifocal avascular necrosis after short term high dose steroid therapy: a report of 3 cases. *J Bone Joint Surg Br* 1984; 66: 431-3.

5 Milley G, Wilcox R G, Mitchell J R A. Dexamethasone in acute stroke. *BMJ* 1978; 2: 994-6.

6 Camp J F, Colwell C W. Core decompression of the femoral head for osteonecrosis. *J Bone Joint Surg Am* 1986; 68: 1313.

7 Kalunian K C, Hahn B H, Basset L. Magnetic resonance imaging identifies early femoral head ischaemic necrosis in patients receiving systemic glucocorticoid therapy. *J Rheumatol* 1989; 16: 959-63.