

Rapidly destructive hip disease

Jeremy A.L. Lawrance, M.D.¹, Nicholas A. Athanasou, M.D.²

¹ Department of Radiology, Nuffield Orthopaedic Centre, Headington, Oxford, UK

² Department of Pathology, Nuffield Orthopaedic Centre, Headington, Oxford, UK

Case report

A 70-year-old woman presented with a 1-year history of increasing knee pain and reduced mobility. Her past medical history was unremarkable. At presentation she had been taking naproxen 250 mg three times daily for 1 month and physical examination revealed limited mobility of both hips, but no other significant abnormality.

A pelvic radiograph showed severe bilateral osteoarthritis (OA) of the hips (Fig. 1). A repeat pelvic radiograph 7 months later showed complete destruction of both femoral heads and fractures of the left inferior pubic ramus and left acetabulum (Fig. 2). A radiograph of the hand showed mild OA of the interphalangeal and metacarpophalangeal joints without chondrocalcinosis. Surgery was deferred for 4 months during which time she developed spontaneous dislocation of the right shoulder joint with partial destruction of the humeral head (Fig. 3). This shoulder had appeared normal on the chest radiograph taken 4 months earlier. Pelvic radiography showed further progression of the destructive arthropathy (Fig. 4). Magnetic resonance imaging of the cervical spine showed a normal cervical and upper dorsal spinal cord with mild spondylotic changes at C5–6 and C6–7.

Correspondence to: J.A.L. Lawrance, Department of Radiology, John Radcliffe Hospital Headington, Oxford OX3 9DU, UK

A left total hip replacement was performed at this stage. The differential diagnosis prior to surgery included osteonecrosis, crystal-associated arthropathy, neuroarthropathy, inflammatory arthritis and analgesic arthropathy.

Pathological examination of the femoral head and neck showed complete loss of articular cartilage, the articular surface being covered by fibrous tissue, fibrocartilage and eburnated bone. There was no evidence of established avascular necrosis and the synovium was not inflamed. Undecalcified sections of bone from the femoral neck showed no evidence of osteoporosis, osteomalacia or any other metabolic bone disease (Fig. 5). Joint tissues and synovial fluid were examined for crystals, including hydroxyapatite, and none were found. Bacterial culture was negative. The features were in keeping with severe degenerative arthritis.

In considering the differential diagnosis, neuroarthropathy was excluded on the basis of the absence of neurological or vascular abnormalities, negative *Treponema pallidum* haemagglutination test, rapid plasma reagin test and normal vitamin B12, folate, glucose and thyroxin levels. Inflammatory arthropathy was unlikely due to normal rheumatoid factor, antinuclear antibody, normal inflammatory markers and absence of any clinical, radiological or pathological evidence of inflammatory arthritis. The patient had been taking

naproxen 250 mg three times daily for 4 months, this being replaced by diclofenac sodium retard 100 mg nocte for 3 months. Additional prescriptions had been co-amilofide one mane for 2 months and finally morphine sulphate (MST) 10 mg twice daily for uncontrolled pain for 1 month, immediately prior to the second pelvic radiograph.

Discussion

Rapidly destructive hip disease (RDHD) occurs in elderly patients, usually female, who develop rapid destruction of the hip or hips, often occurring within months of the onset of symptoms. Radiographs demonstrate femoral head and acetabular destruction with a notable paucity of osteophytes, in contrast to classic OA. A proportion (22% in the series of Bock et al. [1]) develop similar changes in other articulations, notably the shoulder joints [1, 2].

Spontaneous collapse and sudden disintegration of the whole femoral head is described in OA secondary to the formation of large geodes which subsequently collapse [3]. Geodes were not evident on the presentation radiograph.

Osteonecrosis is an important consideration, but there were no predisposing factors and radiological and histological findings do not support this as the primary pathology. In osteonecrosis there are bony lucencies and sclerosis with varying degrees of

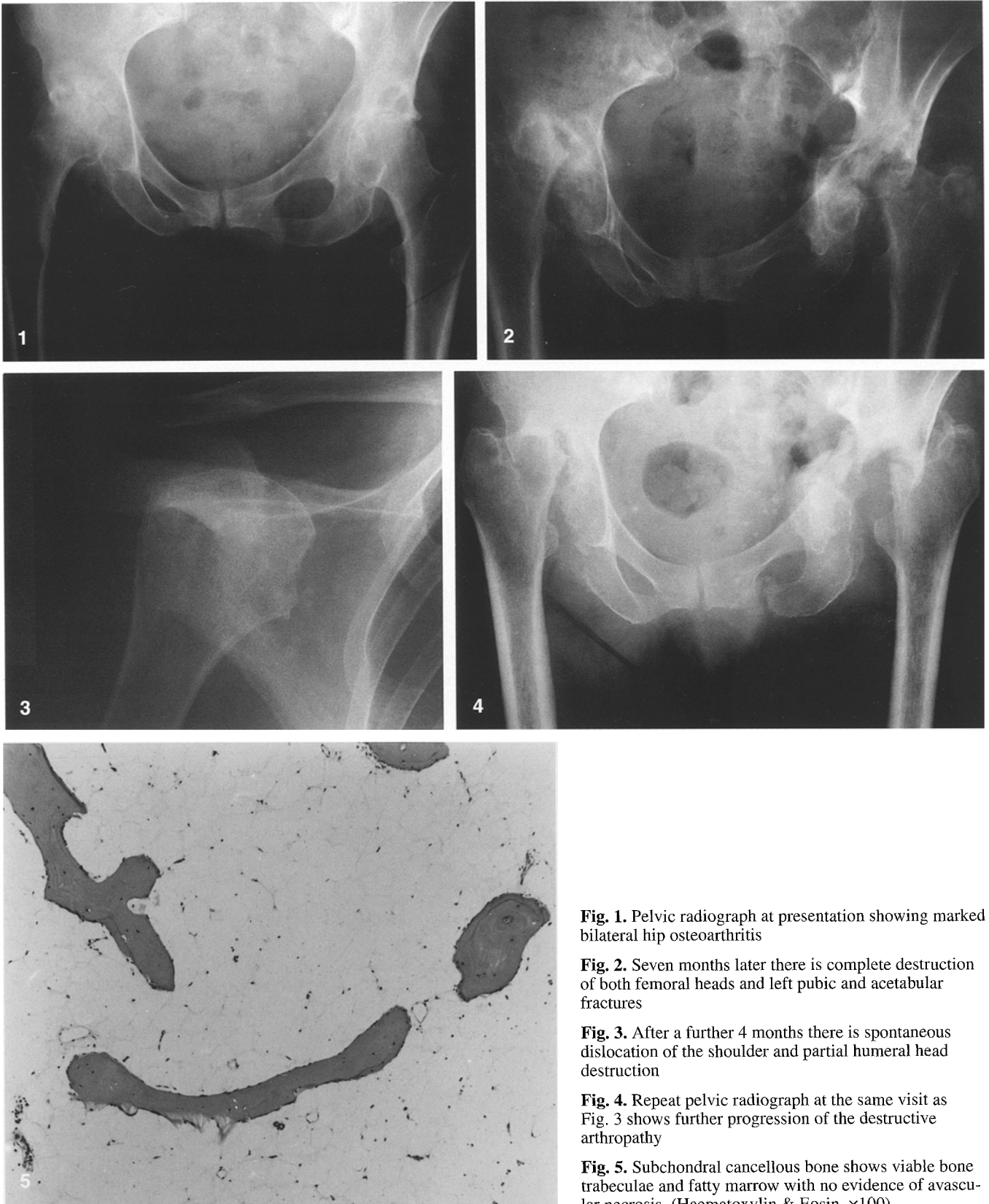


Fig. 1. Pelvic radiograph at presentation showing marked bilateral hip osteoarthritis

Fig. 2. Seven months later there is complete destruction of both femoral heads and left pubic and acetabular fractures

Fig. 3. After a further 4 months there is spontaneous dislocation of the shoulder and partial humeral head destruction

Fig. 4. Repeat pelvic radiograph at the same visit as Fig. 3 shows further progression of the destructive arthropathy

Fig. 5. Subchondral cancellous bone shows viable bone trabeculae and fatty marrow with no evidence of avascular necrosis. (Haematoxylin & Eosin, $\times 100$)

subchondral bony collapse, the joint space being relatively preserved [4].

Calcium pyrophosphate dihydrate deposition disease is characterized by joint space narrowing, subchondral sclerosis and prominent subchondral radiolucencies, which may progress to rapid destruction of the femoral head and acetabulum. This is usually associated with hyaline cartilage calcification and fibrocartilaginous calcification, which may be noted in the acetabular labra [4, 5].

Calcium hydroxyapatite crystal deposition leads to apatite-associated destructive arthritis, the concept of the "Milwaukee shoulder syndrome", where microspheres containing hydroxyapatite crystals produce shoulder joint disorganisation after rotator-cuff rupture and release of crystals into the joint, the syndrome having been subsequently recognised in other joints, including the hip [6–8]. Crystals were not found in the present case.

Neuroarthropathy is associated with bony eburnation and deformation of the articular surfaces, exuberant osteophyte formation and free intra-articular bony fragments, but we found no clinical evidence of neuropathy and the radiological pattern is atypical for this condition. Analgesic arthropathy may be considered a subgroup of the latter. The patient's drug history is modest apart from the addition of MST 10 mg twice daily one month before repeat radiography. Al-

though non-steroidal anti-inflammatory drugs have been implicated in accelerated OA [9], we have not come across cases of neuroarthropathy secondary to MST treatment. This view has been challenged by Doherty et al. [10], who believe that "analgesic hip" is primarily a manifestation of apatite-associated destructive arthritis [10].

Erosive OA is a consideration, but this is usually a disease of the small joints, affecting predominantly the interphalangeal and carpometacarpal joints, accompanied by acute inflammatory episodes [2, 11].

Infectious arthritis is the most important differential diagnosis in a monoarthropathy.

In conclusion, this is an example of the relatively recently characterised RDHD. It is particularly unusual as we are aware of only one previous report of RDHD associated with destructive shoulder arthropathy and pelvic insufficiency fractures [1]. Extensive work-up and deferred surgery could have been avoided by awareness of this condition.

References

1. Bock GW, Garcia A, Weisman MH, Major PA, Lyttle D, Haghighi P, Greenway GD, Resnick D. Rapidly destructive hip disease: clinical and imaging abnormalities. *Radiology* 1993; 186: 461–466.

2. Rosenberg ZS, Shankman S, Steiner GC, Kastenbaum DK, Norman A, Lazansky MG. Rapidly destructive osteoarthritis: clinical, radiographic, and pathologic features. *Radiology* 1992; 182: 213–216.
3. Ilardi CF, Sokoloff L. Secondary osteonecrosis in osteoarthritis of the femoral head. *Hum Pathol* 1984; 15: 79–83.
4. Resnick D, Niwayama G, Coutts RD. Subchondral cysts (geodes) in arthritic disorders: pathologic and radiographic appearance of the hip joint. *Am J Roentgenol* 1977; 128: 799–806.
5. Martel W, Champion CK, Thompson GR, Carter TL. A roentgenologically distinctive arthropathy in some patients with the pseudogout syndrome. *Am J Roentgenol* 1970; 109: 587–604.
6. Campion GV, McCrae F, Alwan W, Watt I, Bradfield J, Dieppe PA. Idiopathic destructive arthritis of the shoulder. *Semin Arthritis Rheum* 1988; 17: 232–245.
7. Dieppe PA, Doherty M, MacFarlane DG, Hutton CW, Bradfield JW, Watt I. Apatite associated destructive arthritis. *Br J Rheumatol* 1984; 23: 84.
8. McCarthy DJ, Halverson PB, Carrera GF, Brewer BJ, Kozin F. "Milwaukee shoulder" – association of microspheroids containing hydroxyapatite crystals, active collagenase, and neutral protease with rotator cuff defects. *Arthritis Rheum* 1981; 24: 464–491.
9. Ronningen H, Langeland N. Indomethacin treatment in osteoarthritis of the hip joint. *Acta Orthop Scand* 1979; 50: 169–174.
10. Doherty M, Holt M, MacMillan P, Watt I, Dieppe P. A reappraisal of "analgesic hip". *Ann Rheum Dis* 1986; 45: 272.
11. Keats TE, Johnstone WH, O'Brien WM. Large joint destruction in erosive osteoarthritis. *Skeletal Radiol* 1981; 6: 267–269.