Rapidly Destructive Hip Disease: Clinical and Imaging Abnormalities

The clinical and radiographic records of 23 patients (15 women, eight men) with rapidly destructive hip disease (RDHD) were retrospectively reviewed. Criteria for RDHD included a history of hip pain of 1–6 months duration and the radiographic appearance of a rapidly progressive atrophic form of bone destruction involving both the femoral head and the acetabulum. Radiographs of the remainder of the appendicular skeleton were assessed in 14 patients. The mean patient age was 72 years. The average time from clinical presentation to the appearance of severe hip destruction was 14 months. Five patients demonstrated similar atrophic bone destruction around other articulations. No patients had clinical or laboratory evidence of sepsis or neurologic disease. Although previous reports have suggested that RDHD is degenerative in nature, similar involvement of other articulations suggests that it may represent a focal finding of a more generalized process.

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A peculiar, rapidly destructive form of osteoarthritis involving the hip has recently been reported in the radiology literature (1). Previously published reports have suggested that this rapidly destructive hip disease (RDHD) is degenerative in nature (1–3), although the rapid clinical course and atrophic bone destruction seen radiographically in patients with RDHD differ from those seen with a degenerative process. The purpose of this study was to further characterize the clinical and imaging features of RDHD and to call attention to the abnormalities of the other appendicular joints that may occur in this disease.

MATERIALS AND METHODS

A retrospective review of patients seen at the Veterans Administration Medical Center, San Diego, University of California San Diego Medical Center, and Health Sciences Centre of the University of Manitoba, as well as cases referred for consultation between 1985 and 1991, revealed 23 patients who met our criteria for RDHD. The criteria for RDHD were a clinical history of hip pain of 1–6 months duration, a radiographic appearance of rapidly progressive atrophic bone destruction involving the femoral head and the acetabulum, the absence of clinical or laboratory evidence of sepsis, and no evidence of neurologic disease. A retrospective analysis of clinical and radiographic records was performed, and all available imaging studies were reviewed. Radiographs of the hips, hands, wrists, knees, and shoulders were studied. The presence or absence of chondrocalcinosis or inflammatory arthropathy was noted. Septic arthritis was excluded on the basis of results of joint aspiration and/or clinical and laboratory examinations. Neurologic disease as well as a history of steroid medication was assessed. Ischemic necrosis was excluded on the basis of the radiographic appearance of atrophic destruction and failure to identify the usual stages of osteonecrosis on serial radiographs.

Radiographs of the hip were evaluated for joint space width, subchondral sclerosis, osteophyosis, cyst formation, and the degree of bone destruction by using a modification of Danielsson’s method as described by Ronningen and Langeland (4). When available, serial radiographs of the hips were similarly evaluated, and the time between the last normal radiograph and that showing the most severe destruction was recorded.

Serial radiographs were evaluated, and the morphologic appearance and the pattern of migration of the femoral head were recorded. Results of synovial fluid analysis for crystals as well as culture were also reviewed. Finally, the surgical findings at joint replacement or biopsy were recorded. In cases in which histologic examination had been performed, these data were reviewed.

RESULTS

Of the 23 patients who were identified with RDHD, 15 were women and eight were men. Four of the 23 patients had bilateral disease. The right hip was involved in 17 cases, and the left hip was involved in 10. The mean age of the patients at onset of disease was 72 years (range, 47–90 years).

All patients presented with hip pain. The mean time for the radiographic appearance of joint destruction after a negative radiograph was 14 months (range, 2 months to 5 years). In one patient, the time between a negative radiograph and appearance of joint destruction was 5 years. No interval radiographic studies were available in this patient; however, the clinical history was considerably shorter, suggesting a more rapid course. If this patient had been excluded, the mean time for the appearance of joint destruction would have been 4 months. Medication histories were available in 18 patients (Table). Fifteen patients had been receiving some form of NSAID. Two

Abbreviations: AP = anteroposterior, NSAID = nonsteroidal antiinflammatory drug, RDHD = rapidly destructive hip disease.
patients had received corticosteroids, one intraarticularly in the affected hip 2 months before the final radiograph was obtained (patient 18) and one orally at a dose of 15 mg hydrocortisone a day, beginning several months before the last radiograph was obtained (patient 15).

Serial radiographs were available in 23 patients, and progressive loss of joint space was evident in 20. In 12 patients in whom more than two serial radiographs were available, progressive loss of joint space was the first manifestation of disease. Evaluation of the hip radiographs demonstrated a striking hatchetlike deformity in 19 of 27 hips (70%) (Fig 1). Five hips (18%) demonstrated a peculiar intrusion type of deformity of the ilium (Fig 2). Osteophytes were not apparent in 22 of the 27 hips. Bone sclerosis was mild and limited to either the acetabulum or the femoral head, but not both. No cases demonstrated severe bone sclerosis. Subchondral cyst formation was uncommon. In 12 of the 27 hips, no cyst formation was seen initially, and none developed in the study period. Multiple cysts were seen in only one hip. The degree of bone destruction was marked in 17 hips, resulting in a grossly deformed joint. In 12 hips, bone destruction occurred when the most recent radiographic examination of the hip had revealed a normal joint. Two hips demonstrated fragmentation of either the acetabulum or the femoral head.

Radiographs of other joints were available in 14 patients (Table). Radiographs of the entire appendicular skeleton were available in five patients. Six patients demonstrated findings typical of osteoarthritis in the hands, wrists, and knees. Four patients demonstrated characteristic abnormalities of osteoarthritis in the contralateral hip. One patient was serologically positive for rheumatoid factor, with typical rheumatoid involvement of her hands and ankles. One patient demonstrated chondrocalcinosis of the contralateral hip, and one had chondrocalcinosis of the knee. One patient with ankle pain had pyrophosphate crystals aspirated from her ankle. Severe destructive changes were noted in other joints in five patients. Glenohumeral joint involvement was seen in three patients (Fig 3), with both shoulders affected in two. Subluxation and atrophic destructive changes were demonstrated in the first metacarpophalangeal joint in one patient and in both tibiotaral joints in another.

Additional imaging studies were available in six patients. Arthrograms of the hip had been obtained in three patients; these demonstrated a tight capsule with a limited articular capacity, suggestive of adhesive capsulitis. No growth of organisms or crystals was found at analysis of the synovial fluid in these three patients. Bone scans were obtained in two patients and demonstrated increased uptake of the radiopharmaceutical agent around the affected hip. Computed tomographic (CT) scans of the hip were available in three patients. In one patient, a CT scan of the pelvis was obtained before the onset of marked hip disease. This scan was obtained to further evaluate multiple fractures involving the sacrum, pubic
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biochemical disease seen developed.

Figure b.

sio lateral trauma.

rami, and symphysis that had been seen on the routine radiograph of the pelvis (Fig 4). These fractures occurred in the absence of substantial trauma. Although a metabolic bone disease was considered, no cause could be identified despite extensive biochemical and clinical evaluation. In the remaining two patients, CT examination of the pelvis revealed superolateral and anterior displacement of the femoral head. Fragmentation of the acetabulum with marked protrusio deformity and thinning of the acetabular wall was seen (Fig 5). Magnetic resonance (MR) examinations of the hips were performed in two patients. In the coronal planes, T1-weighted images (repetition time msec/echo time msec = 600/20) showed absence of cartilage with bone resorption in the femoral and acetabular regions (Fig 6a). There was superolateral displacement of the femoral heads in both cases. On T2-weighted images (2,500/80), abundant synovial fluid was evident with normal-appearing bone marrow (Fig 6b).

Results of synovial fluid analysis were available in eight of the 23 patients. Results of bacteriologic studies were negative in all eight. Synovial fluid analysis was performed in three of the five patients with polyarticular disease. Calcium pyrophosphate crystals were found in one of these patients. Three of these five patients had undergone serologic tests for rheumatoid factor; results were negative in all three. The aspirates were not evaluated for the presence of hydroxyapatite crystals.

Fourteen patients eventually required total joint replacements. The macroscopic findings at surgery were similar for all 14 patients. Severe cartilage and subchondral bone resorption was seen in both the femoral head and the acetabulum. Abundant synovial fluid with extensive fibrosis in the joint capsule and synovium was evident. Histologic analysis demonstrated findings compatible with acute and chronic inflammation of the bone and synovium. Severe cartilage and bone attrition was seen.

Retrospective microscopy was performed in three cases and showed marked fragmentation of the hyaline cartilage and areas of acellular necrotic-looking cartilage surrounded by fibrosis and granulation tissue. Chronic inflammatory cells were seen infrequently (Figs 7, 8). Small fragments of bone were noted within regions of fibrosis (Fig 9). Less striking histologic findings included disappearance of the subchondral bone plate, foci of sclerotic lamellar bone, areas of reactive bone formation, focal marrow fat necrosis, and areas of fibrosis in the marrow spaces. No crystals or organisms were identified.

Figure 2. Patient 23. (a) AP radiograph of the left hip of an 81-year-old woman obtained at onset of symptoms reveals mild joint space narrowing axially. (b) AP radiograph obtained 3 months after a shows that an "intrusion" deformity of the ilium has developed.

Figure 3. Patient 10. (a) AP view of left shoulder of a 73-year-old woman at initial radiographic evaluation demonstrates elevation of the humeral head and acromial erosion. (b) AP view of left shoulder obtained 23 months after a reveals marked destruction of the humeral head, glenoid, and acromion.

DISCUSSION

RDHD has been the subject of several reviews (1–11). These reviews have highlighted the rapidity of joint destruction (average, 1 year), the age of the patient (average, 72 years, which is older than that of most patients with osteoarthritis who are presenting for total hip replacement), and the initial complaint of pain with relative preservation of range of motion. Our study demonstrated similar findings: rapid articular destruction averaging 14 months in duration, elderly patients with an average age of 72 years, and the universal complaint of hip pain. In our study, five patients demonstrated similar destruction of other articulations, suggesting that RDHD may be a focal manifestation of a systemic arthropathy in some patients.

Several disorders can lead to rapid and severe destruction of large joints. These processes include crystal-induced arthropathy (12,13), ischemic necrosis (14), iatrogenic drug-induced
arthropathy (14), septic arthritis (15), and neuroarthropathy (16).

A distinctive, destructive arthropathy related to chondrocalcinosis was described in 1971 independently by Lagier et al (17) in France and Martel and colleagues (18) in the United States. Menkes et al (19) reported 15 patients with destructive arthropathy in association with chondrocalcinosis, five of whom had hip involvement. Clinically, these patients had pseudo-gout attacks, recurrent joint effusions, and severe disability. Radiographically, joint space narrowing, erosions and destruction of bone, prominent osteophytes, osteosclerosis, subchondral cysts, and subluxation were evident. More recently, Menkes et al (7) found that among 86 patients with RDHD, 28 (32.5%) had evidence of chondrocalcinosis, which was almost twice the frequency of chondrocalcinosis in a matched group of control subjects with typical osteoarthritis of the hip. The radiographic appearance of the hips of our patients was unlike that described in previous reports of pyrophosphate arthropathy. Osteophytes, sclerosis, and subchondral cyst formation were not found in our patients, and chondrocalcinosis was seen in only two.

Apatite-associated destructive arthropathy is another disorder that could account for our observations. This arthropathy was initially reported in 1976 by Dieppe et al (20). In two subsequent reports, Dieppe and others described rapid joint destruction in a group of 12 patients (10,11). Three patients had hip involvement, and shoulder abnormalities were evident in 10. The radiographic findings in these previous reports were similar to those seen in our cases and included marked joint destruction, loss of interosseous space, attrition of subchondral bone, mild subarticular bone sclerosis, paucity of osteophytes, and absent cyst or geode formation. Results of synovial fluid analysis demonstrated large numbers of red alizarin-positive bodies in all cases described by those investigators (10,11). Results of synovial biopsy revealed fibrosis and the presence of collagen with fragments of calcified material. One patient had a 22-year history of rheumatoid arthritis before onset of rapidly progressive destructive changes in the knees and shoulders. Similarly, one patient in our series had rheumatoid arthritis.

Because aspirates were not evaluated for the presence of hydroxyapatite crystals, we cannot verify their importance in the development of RDHD. The multicenter, retrospective nature of this review precluded a uni-

![Figure 4. Patient 8.](image1)

(a) AP view of pelvis of 66-year-old woman obtained at onset of symptoms shows fractures of the parasymphyscal region (long arrow) and left sacral ala (short arrow). (b) AP view obtained 16 months after a shows progressive fracturing of the pubic rami (arrows) and development of an intrusion deformity of the left ilium.

![Figure 5. Patient 9.](image2)

Transaxial CT scan of the right hip of a 59-year-old woman shows marked protrusio deformity with thinning and fragmentation of the acetabular wall.

![Figure 6. Patient 8.](image3)

(a) T1-weighted coronal MR image (600/20) of the pelvis of a 66-year-old woman obtained 16 months after onset of symptoms shows bone resorption in femoral and acetabular regions with cartilage loss. There is superolateral displacement of the femoral head. (b) T2-weighted coronal MR image (2,300/80) of the pelvis obtained 16 months after onset of symptoms shows large joint effusion with normal-appearing surrounding bone marrow.
crystal-associated disease be applied to apatite arthropathy because the hydroxyapatite crystals may be a “necessary but not sufficient” prerequisite for symptomatic disease.

The radiographic appearance of shoulder involvement in the patients reported by Doherty et al (11) is similar to that seen in the cases of “Milwaukee shoulder” reported by McCarty et al (24). Glenohumeral joint space narrowing, humeral head elevation, and large effusions were described. Electron microscopy of the synovial fluid in these patients revealed calcium hydroxyapatite crystals. Comparable radiographic findings were seen in three patients in our series, with bilateral involvement in two cases.

The use of NSAIDs has been implicated as a cause of hip destruction. The importance of analgesic-induced joint disease, first reported, to our knowledge, in 1967 by Coke (25), has been subsequently debated in the literature. Laboratory evidence has demonstrated in vitro altered cartilage metabolism in humans and reduced fracture healing and inhibited bone remodeling in rabbit and rat models with NSAID administration (26–28). Furthermore, clinical evidence has suggested that indomethacin may have a deleterious effect on osteoarthritic hips (4). Another recent report has described acetabular bone destruction related to NSAID administration (29). Conversely, Watson (30) could not demonstrate an injurious effect of NSAIDs on cartilage repair in a laboratory model of joint degeneration.

Finally, in a study of 19 patients with typical radiographic findings of “analgesic hip,” Doherty et al (11) found that only 10 patients had received NSAIDs. Five patients had received analgesics other than NSAIDs, and four had not received any medication of any kind. In our study, 15 patients had received some form of NSAID. Although a contributory effect of NSAID cannot be excluded as a cause of RDHD, it is evident that this arthropathy can occur in the absence of NSAID use. In addition, the persistence of pain in these patients, despite the use of NSAID, makes it unlikely that a neuropathiclike effect of NSAID contributed to the arthropathy (11).

An idiopathic form of RDHD has been described by several French investigators (2,3,5–9). This disorder resembles apatite deposition disease. In a recent review of 27 cases of RDHD, Lequesne and Ray (8) reported finding apatite crystals in all cases in which fluid could be obtained.

Among the different types of osteoarthritis of the hip described by Cameron and Macnab (31), the capital collapse pattern is the least common. Although the morphologic characteristics of the femoral head and the pattern of joint space narrowing in this type of osteoarthritis appear similar to those seen in our cases, the rapidity of osseous destruction and the absence of osteophytes and histologic evidence of osteonecrosis at pathologic examination suggest that RDHD represents an entity distinct from the capital collapse type of osteoarthritis.

Subchondral bone necrosis and cell necrosis recently have been emphasized as major factors in the development of RDHD by Mitrovic and Riera (32). Although alizarin-positive material was found in 50% of the synovial fluid aspirates at joint replacement, these authors believed that this was a consequence of the joint destruction rather than a primary factor. Mitrovic and Riera concluded that further studies are required to determine the cause of the observed subchondral ischemia in their histopathologic study of RDHD.

The presence of osteoarthritis in the hands, wrists, and knees of six of our patients raises the possibility of a subset of patients with both RDHD and nodal osteoarthritis. Among the 19 patients with apatite-associated destructive arthritis reported by Doherty et al (11), seven had nodal osteoarthritis. Gerster et al (33) reported a greater frequency of destructive arthropathy in patients with generalized osteoarthritis. Age-matched control subjects would likely show a similar frequency of osteoarthritis, however, and the association of these findings remains uncertain.

Erosive osteoarthritis has been reported to involve large joints. The case presented by Keats et al (34) demonstrated many radiographic similarities to our cases. Although the involvement of the shoulder and hip in their patient was suggestive of apatite-associated disease, the presence or absence of intraarticular crystals was not noted.

RDHD may mimic septic arthritis or acute neuroarthropathy (15,16). The absence of fever and leucocytosis and the inability to recover microorganisms from joint fluid exclude sepsis. Patients with neuroarthropathy usually have mild or absent pain, abnormal findings at neurologic examination, and radiographic evidence of
bone fragmentation and detritus. None of our patients had evidence of a neurologic disease or deficit, and all patients presented with moderate to severe pain.

Solomon et al (35) have suggested that the appearance of hip osteoarthritis is determined by three interacting factors: mechanical stress, cartilage degeneration, and bone response. The presence of insufficient fractures in one of our patients is suggestive of an abnormal repair response.

It may be argued that the identification of RDHD is of little clinical importance. Most affected patients will require total joint replacements as treatment for their debilitating pain and deformity. Two differential diagnostic considerations in these patients are septic arthritis and neuroarthropathy. If the clinician is made aware of RDHD, the need for extensive investigation to exclude sepsis may be obviated. Furthermore, placement of a total joint prosthesis in a patient with neuropathy often leads to failure. In our series, as well as some of those previously reported (1.3), the results of total joint replacement in patients with RDHD have been good; therefore, total joint replacement may be offered to the patient with RDHD with less concern for subsequent failure. Finally, in the presence of rapid destructive changes in the hip, a search should be made for involvement of other joints, particularly the shoulders.

Because the cause of this disorder remains unclear, further study and follow-up of these 23 patients, including detailed synovial fluid analysis, are required to elucidate the importance of hydroxyapatite in RDHD. The use of age-matched control subjects to evaluate the importance of the polyarticular disease that was observed in our patients may provide further evidence to support the concept of RDHD as a focal finding of a more generalized process.

References