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IMAGING (D MINTZ, SECTION EDITOR)

Rapidly Progressive Osteoarthritis: a Review of the Clinical and Radiologic Presentation

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Abstract
Purpose of Review The purpose of this paper is to review the distinct clinical and radiographic features that may lead to prompt diagnosis of rapidly progressive osteoarthritis (RPOA) and thus obviate unnecessary and costly diagnostic workup.
Recent Findings RPOA is uncommon but is more frequently seen in practice because of the aging population. RPOA is a destructive arthropathy that occurs most commonly in elderly women but can also be seen in patients that have sustained trauma. The dramatic radiologic manifestations of RPOA can lead to diagnostic confusion with other arthropathies, infection, and osteonecrosis. RPOA was originally described in the hip but may also involve the shoulder. The etiology of RPOA is not well understood, but subchondral fracture probably plays a role in the development of dramatic destruction of the joint that is seen in affected patients. Early diagnosis may reduce the complexity of surgical management.
Summary RPOA is an uncommon condition that occurs most frequently in elderly woman or in patients who have sustained trauma. Prompt recognition of the clinical and radiologic features of this arthropathy can reduce unnecessary diagnostic workup and complexity of surgical intervention.

Keywords Rapidly Progressive Osteoarthritis · Subchondral Fracture · Chondrolysis · Post-traumatic Arthritis

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Introduction

Rapidly progressive osteoarthritis (RPOA) was once considered to be a rare entity but is more frequently encountered in clinical practice as the average age of the population increases. This clinical entity was originally described in the hip [1], but the shoulder may also be involved [2]. The clinical presentation and destructive changes on radiography can be very dramatic leading to diagnostic confusion. While rheumatoid arthritis, septic arthritis, osteonecrosis, and crystalline arthropathy may lead to rapidly progressive arthropathy, RPOA has a specific clinical and radiologic presentation that suggests the diagnosis. It is therefore important that both radiologists and clinicians are aware of this entity to avoid unnecessary diagnostic workup and to ensure prompt appropriate treatment.

Clinical Presentation

Hip

Rapid progression of hip disease has been defined as joint space loss at a rate greater than 2 mm per year or if more than 50% of joint space is lost in 1 year [3]. Patients with rapidly progressive osteoarthritis of the hip are divided into two distinct populations: those with or without history of recent trauma.

Hip Atraumatic (Fig. 1a, b)

The presentation in patients without history of trauma has received the most attention in the literature. These patients are typically elderly females (61–90% female) with average age of 72 years (range 47–90) [4–8]. Unilateral presentation is typical with bilateral involvement seen in up to 17% of patients [6]. Pain, often severe, is the most common symptom, and it is experienced for

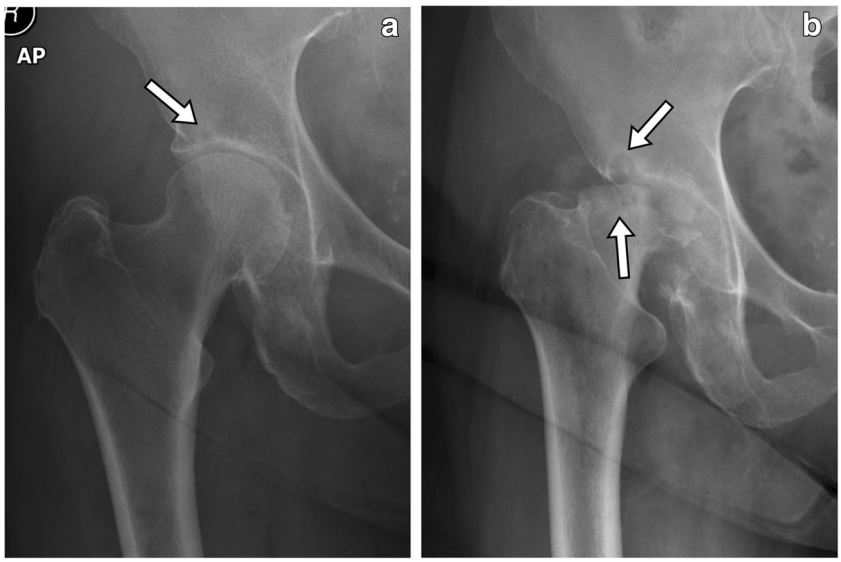

Fig. 1 68 year old woman with RPOA. **a** Minimal subchondral cyst formation (arrow), medial femoral calcar osteophyte and preserved joint space on seen on initial radiograph. **b** AP radiograph of the hip obtained 11 months later shows typical flattening of the femoral head which is migrated superior and laterally. Subchondral lucencies are appreciated in the acetabulum and femoral head (arrows)

14 months on average at presentation (range 2 months–5 years) [4–7].

Hip Traumatic (Fig. 2a, b)

Patients following acetabular fracture are at risk for developing osteolysis of the femoral head in a pattern that is similar to atraumatic RPOA. Rapidly progressive destruction of the hip joint was seen in 6.1% of patients that sustained an acetabular fracture in one series [9]. Unlike atraumatic RPOA, the patients in this series were more likely to be male and were a decade younger in age (mean 61.6 years).

Shoulder (Fig. 3)

Patients with rapidly destructive arthritis of the shoulder are also more commonly elderly women with a mean age of 72 [10•]. Insidious onset of atraumatic pain and restricted range of motion are the presenting symptoms. Interestingly, severe destructive shoulder involvement has been reported in 13% of patients with coexistent or history of RPOA of the hip in one case series [6].

Regardless of joint involved or traumatic or atraumatic presentation, patients with RPOA usually have normal white blood cell count and C-reactive protein levels. Erythrocyte sedimentation rate may be mildly elevated. These normal values can help reduce the concern for septic arthritis as a cause for the rapid destruction of the joint that is seen in patients with RPOA. The patient history is usually negative for steroid use or alcoholism which helps exclude concern for osteonecrosis. A careful history and physical examination eliminates clinical concern for neuropathic disease.

Pathogenesis

The cause of RPOA is still not well understood despite the fact that it was first described in the literature by Coste in 1959 [8,

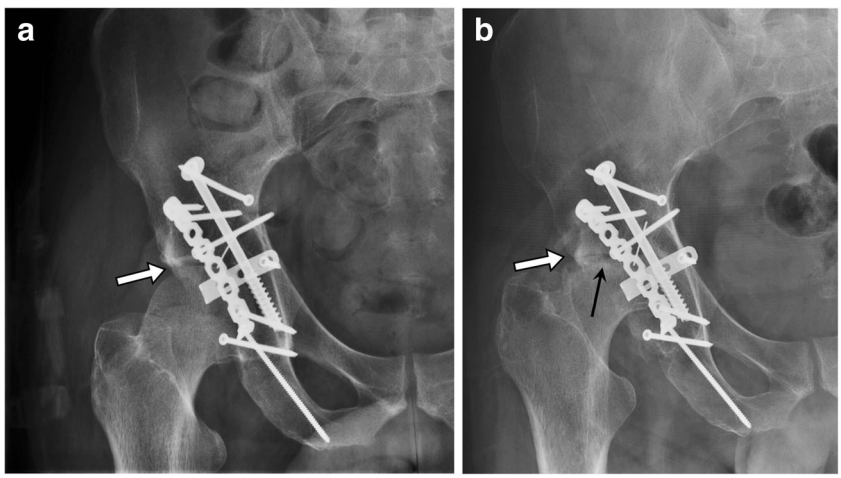

Fig. 2 41 year old man with progressively worsening right hip pain after fixation of an acetabular fracture sustained in a motor vehicle accident. **a** AP radiograph of right hip obtained two months post-operatively demonstrate changes of acetabular ORIF and maintained hip joint space (*arrow*). **b** Nine month post-operative radiograph shows complete joint space collapse (thick white arrow) and marked flattening with remodeling of the superior femoral head (thin black arrow)

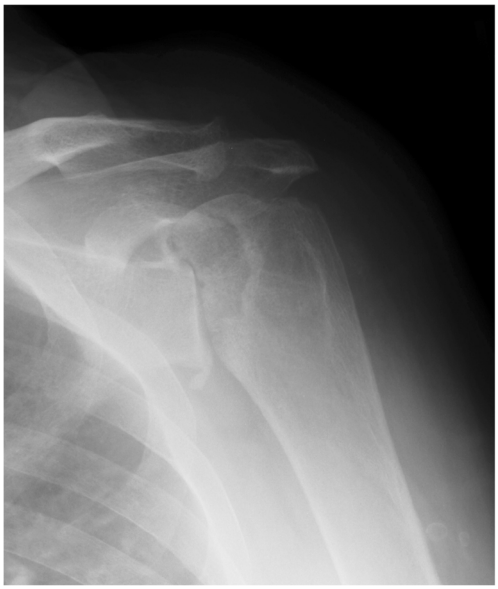

Fig. 3 59 year old woman with atraumatic shoulder pain and rapidly destructive arthritis. Posterior oblique radiograph shows joint space narrowing flattening of the medial humeral head and remodeling of the superior glenoid

11]. Several theories have been proposed in the past. Infectious etiologies have been excluded by culture and/or intraoperative biopsy in several case series. Primary osteonecrosis has been discounted as the cause of rapid destruction of the femoral or humeral head based on histology of resected specimens in multiple studies [4–6, 10•, 12].

Crystal-induced arthritis is widely considered to be a possible source for shoulder disease. Calcium pyrophosphate crystal does not appear to be the basis for RPOA despite that it is a well-known cause of arthritis. On the other hand, hydroxyapatite crystals have been detected in the synovium of shoulders affected by rapidly progressive arthritis (Milwaukee shoulder) as first described by McCarty et al. in 1981 [13] and is widely considered to be a trigger for inflammation leading to joint destruction [14•]. However, the role of basic calcium phosphate crystal as the primary basis of joint destruction is still debated [15, 16]. Interestingly, hydroxyapatite crystal is not widely implicated as the cause of RPOA of the hip despite the overlap in clinical, histological, and radiographic presentation with shoulder disease. This may be, in part, because detection of hydroxyapatite crystals requires special stains that are not routinely performed in or available for pathologic assessment of resected hip specimens. In one pathologic series evaluating RPOA of the hip, 30% of joints did in fact demonstrate significant amounts of alizarin S-positive osteochondral debris in the pathologic specimen [17] indicating the presence of hydroxyapatite crystal.

While neuropathy can be excluded as the cause of RPOA based on clinical grounds, analgesics have been implicated as a contributor to this disorder. Indocin has been reported to cause acceleration of joint space narrowing in knee osteoarthritis [18], but non-steroidal anti-inflammatory drugs (NSAIDs) have not been reported as a risk factor in the RPOA case series literature, although this was not specifically studied in these retrospective reviews. Nerve growth factor antagonists have been evaluated in drug trials for effectiveness in treatment of pain in patients with osteoarthritis. The FDA abruptly terminated these randomized trials in 2010 because of the increased incidence of RPOA in the cohorts receiving these agents [19]. The role of intraarticular steroid and anesthetic injection as a potential cause of the development of RPOA has not been systematically evaluated. Any relationship between intraarticular steroid injection and the onset is likely temporal only. The MRI literature suggests that patients that will go on to develop the destructive changes of RPOA can be predicted in the early stages of the disorder based on the presence of bone marrow edema in the hip [12]. These findings suggest that the die may already be cast for such a patient and that an intraarticular steroid injection administered in this situation may have no impact on the progression to RPOA. One study has shown that intraarticular steroid injection in a patient with known RPOA does not reduce the need for eventual total hip replacement [20].

Increasing evidence suggests that subchondral fracture may play a role in rapidly destructive arthritis in both the hip [12, 21–23] and shoulder [10•, 24–26]. Destruction of the hip joint in a radiographic pattern of RPOA has been described in patients with prior acetabular fracture [9]. Articular collapse of humeral heads in rapidly destructive arthritis [10•, 24–26] occurs in zones of known subchondral osteoporosis [27]. Additionally, the majority of patients with rapidly destructive arthritis of the shoulder have full thickness rotator cuff tears on MRI, and abnormal biomechanics may contribute to the development of subchondral fracture in these patients. Interestingly, body mass index and osteoporosis have no impact on the development of RPOA of the hip. While there is clear evidence of subchondral fracture in patients with rapidly destructive arthritis, it is not understood whether subchondral fracture is the primary initiator of chondrolysis and bone destruction or a downstream consequence of another process. It is also not understood why some patients with subchondral fracture develop RPOA and others do not.

Histology

Evaluation of the synovium in RPOA shows varying degrees of synovitis with variable degrees of mild acute and chronic features that are characterized as reactive rather than inflammatory [4, 6, 8, 17, 28]. Calcium pyrophosphate crystal is either absent or scant. Calcium hydroxyapatite crystal may be present in some specimens [17] but is absent in other case series that have tried to assess its presence [21]. Extensive

capsular fibrosis may be appreciated [6]. Subchondral fracture may be seen histologically [21]. Variable degrees of dead bone, from scant [4, 6, 12] to more extensive [5, 17], have been described in the resected specimen. The overall histologic picture in patients with RPOA is that of osteoarthritis.

Radiologic Manifestations

The radiologic presentation of osteoarthritis is dependent on joint destruction and bone repair. The four interacting variables that produce what we recognize as osteoarthritis have been described as (1) mechanical overload, (2) inflammatory disease, (3) good bone response, and (4) poor bone response [29]. Osteoarthritis has classically been considered a purely mechanical arthropathy, but the impact of inflammation on disease progression and patient symptoms has only recently been more widely appreciated and investigated [30, 31•, 32]. Within this framework, RPOA has two radiographic stages: (1) rapid chondrolysis followed by (2) rapid and marked subchondral bone resorption and destruction [8].

Hip

Early Chondrolysis Stage

The first conventional radiographic manifestations of RPOA of the hip reflect chondrolysis. Joint space narrowing, usually superior lateral, is appreciated but is accompanied by minimal bone repair [4–6, 8] (ESM 1). Osteophytes, if present, are small, and subchondral sclerosis is scant. Variable subchondral cyst formation can be seen.

MRI in the early stages of RPOA of the hip may be clinically useful because it may predict the dramatic destructive changes that will be seen in the coming weeks and months. MRI obtained at this stage will show marked loss of articular cartilage that is accompanied by effusion and synovitis [12, 22, 23] (Fig. 4). Extracapsular edema may also be appreciated [12] which may cause diagnostic confusion with septic arthritis as this finding is commonly attributed to infection rather than degenerative disorders (ESM 2). The most dramatic MRI presentation, however, is in the bone marrow. An edema-like pattern is appreciable in the femoral head in all patients and is accompanied by similar findings in the acetabulum in most patients. Focal subchondral fracture may be seen in early stages of RPOA [12, 21–23] (ESM 3, 4, and 5). Subchondral fractures present as a curvilinear band of low signal on all sequences that may be seen in the weight-bearing subchondral bone in up to 33% of patients [12]. This band of low signal parallels the overlying subchondral bone and is usually less than 1 cm deep to it. These two characteristics help distinguish

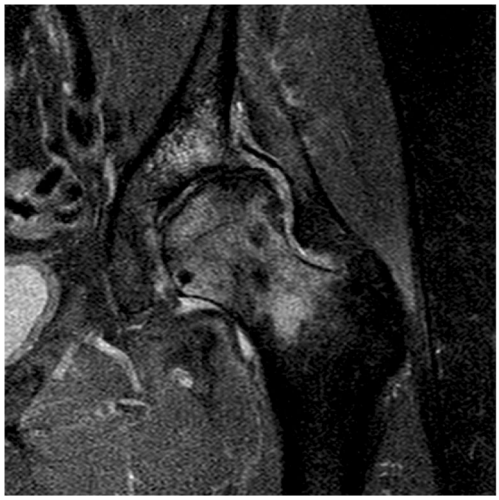

Fig. 4 60 year old woman with early MRI findings of RPOA. Coronal fat-suppressed T2 weighted image of the left hip shows superior lateral joint space narrowing synovitis and bone marrow edema that is more prominent in the femoral neck than acetabulum

a subchondral fracture from avascular necrosis in which the linear band of low signal is concave to the articular surface and serpentine [33]. Subchondral cysts may be appreciated on MRI in either the femoral head and/or acetabulum and are usually seen at the site of bone-on-bone articulation. Contrast-enhanced images parallel the findings seen on fat-saturated fast spin echo T2-weighted images and rarely increase the diagnostic value of non-contrast images.

Late-Subchondral Destruction Stage

Flattening of the femoral head is seen in all patients with RPOA in this stage of the disorder. Femoral head remodeling may be so extensive that it appears sheared off thus mimicking findings that can be seen in neuropathic arthropathy [4–6, 8] (ESM 6). Unlike neuropathic arthropathy, fragmentation and intraarticular osseous debris are not dominant features of RPOA. Superior lateral joint space narrowing is typical with subchondral sclerosis appreciated at the site of bone-on-bone contact. Joint space widening however can be seen at this stage on non-weight-bearing radiographs [4] (ESM 7). Varying degrees of remodeling of the acetabulum are appreciated that usually parallel the femoral head destruction. In 18% of patients in one series [6], the destroyed femoral head subsided into the acetabulum leaving the lateral acetabular rim relatively intact.

Unlike in the early stages of RPOA, MRI offers little diagnostic information in the late destruction phase. The findings of joint remodeling parallel those of conventional radiography. Synovitis and bone marrow edema are still apparent.

Shoulder

The radiographic findings of rapidly destructive arthropathy (RDA) of the shoulder have many similarities to that of its presentation in the hip [10•]. A distinctive separation into a chondrolysis and bone destruction phase has not been described in the shoulder. One feature that is shared between shoulder and hip is flattening of the articular surface. Flattening of the humeral articular surface is either medial or superior medial and is accompanied by subchondral sclerosis and minimal osteophyte formation (ESM 8). Destruction and remodeling can be dramatic with up to 100% destruction of the humeral head [34]. Additionally, intraarticular ossific debris is more commonly appreciated in the shoulder than in the hip [34]. Dramatic destruction of the humeral head and the presence of ossific debris may lead to the erroneous diagnosis of neuropathic joint (ESM 9). Variable remodeling of the glenoid may be appreciated. A distended subacromial bursa containing calcified debris may be appreciated in patients with concomitant full thickness rotator cuff tear (ESM 10, 11).

MRI findings in RDA of the shoulder share many features with RPOA of the hip (ESM 12, 13). Effusion, that is often large, and synovitis are ubiquitous. Bone marrow edema is also appreciated but is more striking in the humeral head than the glenoid in many cases [10•]. Subchondral insufficiency fracture may be identified in the humeral head on T1 and T2-weighted images [24, 26]. Collapse and remodeling of the humeral head and glenoid parallel findings seen on conventional radiography. Full thickness rotator cuff tears are common in patients with RDA but are not seen in all patients.

Treatment

The primary treatment for RPOA of the hip [35] and shoulder [10•] is joint replacement. Blood loss during total hip arthroplasty is higher than in routine osteoarthritis [36]. Survivorship of hip joint reconstruction at 5 years is greater than 95% in the setting of RPOA [37–40]. It is ideal for hip replacement surgery to be performed before the development of acetabular defects [7], which are common in the later stages of RPOA of the hip. Close monitoring of patients at risk for RPOA has been advocated [7] to reduce operative time and complexity of reconstructive efforts.

Conclusion

Rapidly destructive arthritis of the hip and shoulder is relatively rare in comparison to other forms of osteoarthritis. It is most commonly seen in women over 70 years of age, and its rapid progression and dramatic radiologic manifestations may lead to diagnostic confusion with other entities such as septic arthritis, neuropathic arthropathy, crystalline arthropathy, and osteonecrosis. The characteristic radiologic presentation of rapid joint space narrowing with little or no osteophyte formation in an elderly women or patient with recent acetabular fracture will aid in recognition of this disorder. Careful history, detailed physical examination, and routine laboratory evaluation will exclude most of the other diagnostic considerations. Joint aspiration will likely be performed before surgery to exclude the remote possibility of infection.

The exact etiology of RPOA remains unclear, but subchondral fracture and/or hydroxyapatite crystal disease are two possibilities that may explain the development of this unusual presentation of degenerative disease. Regardless of etiology, patients that demonstrate rapid joint space narrowing should be closely monitored for possible bone destruction. Early MRI findings of bone marrow edema and subchondral fracture in this setting may help identify patients that are at risk for rapid and progressive articular collapse. The only known treatment for RPOA at this time is surgical management. Joint replacement should ideally be performed before the development of acetabular destruction to reduce operative time and complexity.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subject performed by any of the authors.

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