Arthroplasty Today 2 (2016) 15–18

Contents lists available at ScienceDirect

Arthroplasty Today

journal homepage: http://www.arthroplastytoday.org/

Case report

Rapidly destructive osteoarthritis can mimic infection

Gavin Hart, MD [a], Thomas Fehring, MD [b, *]

[a] Department of Orthopaedic Surgery, Carolinas Medical Center, Charlotte, NC, USA
[b] OrthoCarolina, Hip and Knee Center, Charlotte, NC, USA

ARTICLE INFO

Article history:
Received 15 April 2015
Received in revised form
19 November 2015
Accepted 20 November 2015
Available online 9 January 2016

Keywords:
Total hip arthroplasty
Rapidly destructive osteoarthritis
Avascular necrosis
Septic arthritis

ABSTRACT

The intraoperative appearance of rapidly destructive osteoarthritis and septic arthritis can be similar. Three patients at our institution demonstrated preoperative or intraoperative findings potentially consistent with infection during primary total hip arthroplasty; however, none of these patients were found to have an actual infection. One of these patients underwent an unnecessary 2-stage total hip arthroplasty secondary to the intraoperative appearance of their joint fluid. We advocate performing an infection workup preoperatively when patients present with rapid degenerative changes of their hip joint to diminish the uncertainty of proceeding with arthroplasty.

Introduction

Rapidly destructive osteoarthritis (RDO) was first described in the European literature in 1957 [1]. Subsequently, it has been given several different names including rapid destructive coxarthrosis, rapidly destructive arthrosis of the hip, Postel's osteoarthrosis, and destructive osteoarthritis [2-5]. It is a rare syndrome of unknown etiology and appears to be distinct from osteonecrosis as it tends to involve both the acetabulum and the femoral head [6]. The rapid bone loss seen in patients with RDO can mimic septic arthritis, inflammatory arthritis, or neuropathic osteoarthropathy; however, the histologic changes seen in hips of patients with RDO are consistent with osteoarthritis [7]. Clinically, the intra-articular fluid and debris associated with femoral and acetabular bone loss can resemble septic arthritis. When this purulent fluid is encountered at the time of hip arthroplasty, a clinical dilemma arises as active infection would be a contraindication for primary total hip arthroplasty (THA). Without additional information, the appearance of such fluid could lead the surgeon to perform an unnecessary 2-stage procedure.

In this case series, we report a group of patients who had rapid joint destruction and an intraoperative appearance of joint sepsis during primary hip arthroplasty. A strategy to preoperatively evaluate these patients to differentiate between RDO and septic arthritis is necessary to avoid a 2-stage procedure.

Case histories

Case 1

A 65-year-old Caucasian woman who was seen for routine follow-up of her left THA (Fig. 1a). Three months later, she presented with a 6-week history of insidious onset of pain that worsened with prolonged ambulation. She had been taking nonsteroidal anti-inflammatory information with only partial relief of her pain. The patient did not have any pertinent medical history. On physical examination, she had pain with range of motion and diminished internal and external rotation in comparison to the contralateral side. She had full strength and sensation in her bilateral lower extremities. Radiographs of the pelvis at that time demonstrated degenerative joint disease of the right hip with significant bony destruction of the femoral head and acetabulum. Plans were made to proceed with arthroplasty (Fig. 1b). Preoperative blood work demonstrated a white blood cell count of 10.1 $\times$ $10^3/\mu L$ (normal: 3.8-10.8 $\times$ $10^3/\mu L$). During the THA, a capsulotomy was performed and the joint was noted to be filled with cloudy,

One or more of the authors of this paper have disclosed potential or pertinent conflicts of interest, which may include receipt of payment, either direct or indirect, institutional support, or association with an entity in the biomedical field which may be perceived to have potential conflict of interest with this work. For full disclosure statements refer to http://dx.doi.org/10.1016/j.artd.2015.11.003

* Corresponding author. 2001 Vail Avenue, Suite 200A, Charlotte, NC 28207, USA. Tel.: +1 704 323 2564.
E-mail address: Thomas.Fehring@orthocarolina.com

http://dx.doi.org/10.1016/j.artd.2015.11.003

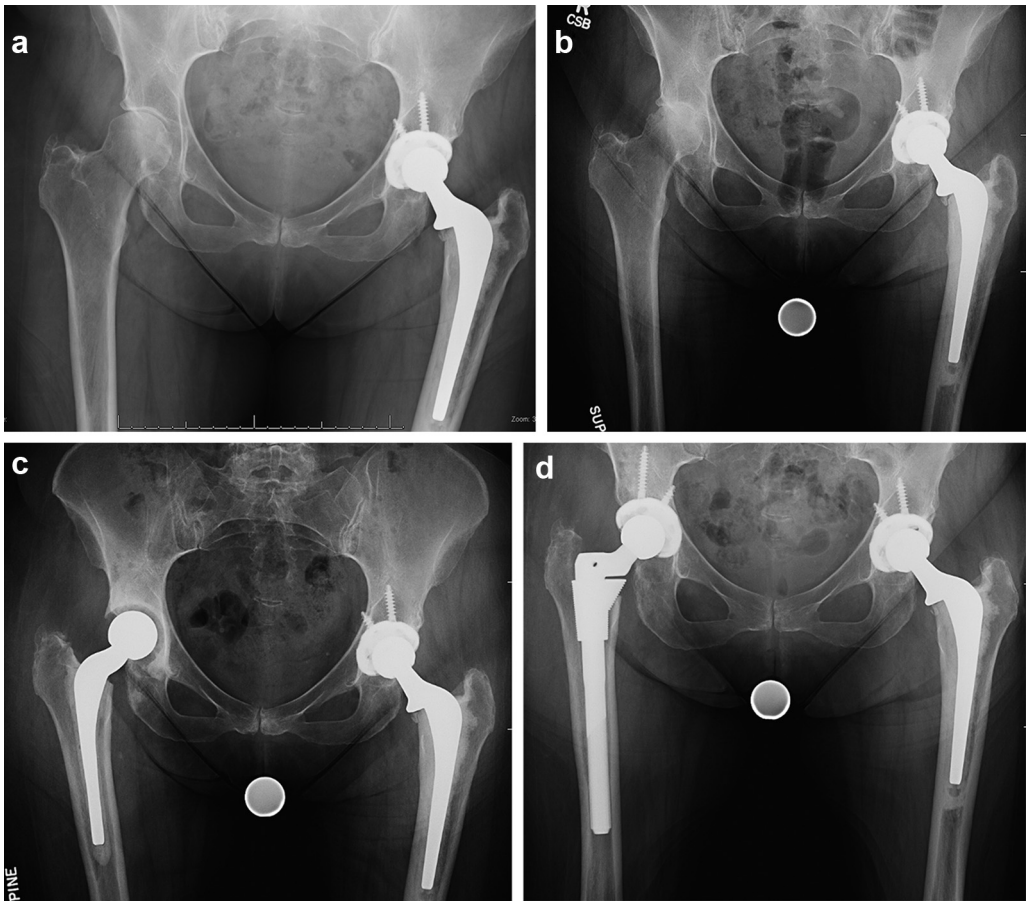

Figure 1. Patient 1 at follow-up for left hip (a), 3 months later with right hip pain (b), initial postoperative radiograph (c), and post-operatively 9 months after THA (d).

purulent fluid. The decision was made intraoperatively to obtain cultures and place an antibiotic spacer (Fig. 1c). The patient underwent insertion of a peripherally inserted central catheter and received 6 weeks of vancomycin. All surgical cultures from this procedure failed to grow any organisms. At conclusion of these antibiotics, repeat laboratory studies were obtained: white blood cell count of 7.7 $\times$ $10^3/\mu L$ (normal: 3.8-10.8 $\times$ $10^3/\mu L$), C-reactive protein (CRP) of <4 mg/L (0-5 mg/L), and erythrocyte sedimentation rate (ESR) of 10 mm/h (0-20 mm/h). An aspirate obtained at 3 months postoperatively returned with 60 nucleated cells and no growth on culture. After this aspiration, the patient underwent reimplantation of her THA components without complication. At 2-year follow-up, the patient is pain free and ambulating without a limp. Her incision was well healed and she exhibited no clinical signs of infection. Her radiographs demonstrated well-ingrown components without any radiographic lucencies (Fig. 1d).

Case 2

A 64-year-old African American male presented with a 4-month history of right hip pain and a 2-month history of left hip pain. This pain worsened to the point that the patient required a cane for ambulation. At his initial visit, the patient reported recent low-grade fevers without any other systemic symptoms. Pertinent medical history included hypertension, deep vein thrombosis, depression, and gout. On physical examination, the patient demonstrated significant pain with hip range of motion and pain with a resisted straight leg raise test. The patient did not have pain with knee range of motion or with a passive straight leg raise test. His initial radiographs showed bone loss of both the femoral head and acetabulum on the right side and the appearance of avascular necrosis on the left (Fig. 2a). As a result of this imaging, the patient was sent for serology. His laboratory studies returned with an elevated ESR of 85 mm/h (0-20 mm/h) and CRP of 2.0 mg/L (0-0.8 mg/L), and the patient was sent for bilateral hip aspirations. The aspirate for the right hip demonstrated cloudy fluid with a cell count of 240 nucleated cells and a negative culture, with the aspirate of the left hip negative as well. The patient underwent right THA 6 months after initial presentation. Intraoperatively, the hip was found to be filled with fibrinous debris and multiple femoral head fragments. At 3 months postoperatively, his right hip pain had dramatically improved, whereas his left hip pain had increased and further degenerative changes were present on radiographs. Plans were made to proceed with left THA. At 2 years follow-up, the patient had pain-free hips. Follow-up radiographs demonstrated that the patient's components were well fixed without evidence of lucency (Fig. 2c).

Case 3

A 69-year-old Caucasian woman presented with worsening bilateral hip pain of 6-week duration. Although she did have pain bilaterally, she reported that her right hip was more painful than her left. The patient had been diagnosed with avascular necrosis several months before initial presentation to our office but had not had any treatment. Her medical history was significant for asthma,

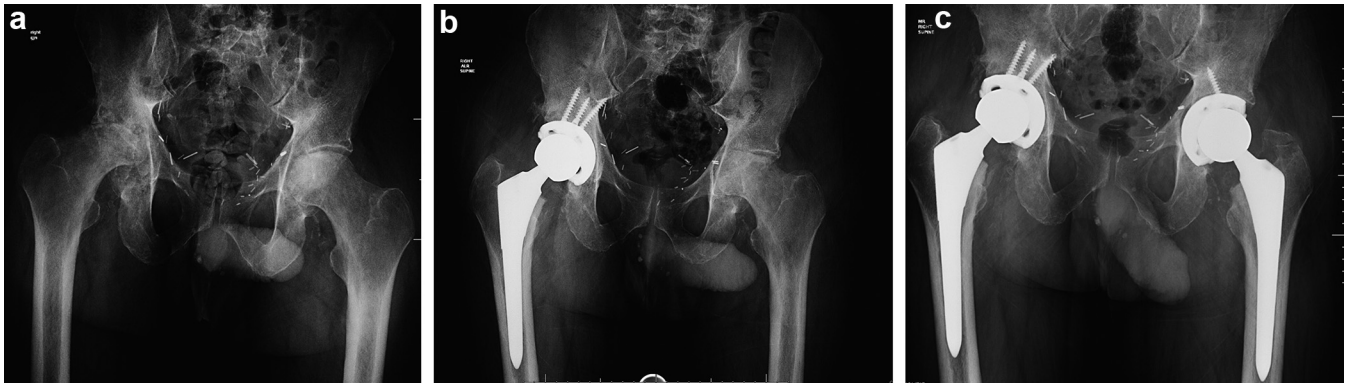

Figure 2. Patient 2 at presentation (a), follow-up visit 3 months later (b), and postoperatively 2 years after initial presentation (c).

hypertension, hyperlipidemia, and hypothyroidism. On physical examination, she had severe pain with hip range of motion and associated hip flexion weakness secondary to pain. Radiographs at that time demonstrated right femoral head bone loss with associated acetabular bone loss and significant arthritis in the left hip (Fig. 3a). A decision was made for a right THA, and the patient was sent for serology and an aspiration. Her laboratory studies returned with an ESR of 13 mm/h (0-30 mm/hr) and a CRP of 2.37 mg/L (0-4.9 mg/L). Her joint aspiration was negative for growth on aerobic and anaerobic cultures. She subsequently underwent right hip arthroplasty. Intraoperatively, cloudy joint fluid with dissolution of the femoral head and part of the posterior wall was noted. The patient returned to clinic at 6 weeks with a well-healed wound without signs of infection and a pain-free hip range of motion. Radiographs at this time demonstrated good position of the right-sided implants; however, rapid dissolution of the left hip was noted (Fig. 3b). As the patient had continued left hip pain, she was subsequently taken to the operating room for a contralateral THA 2 months later. At 18-month clinical follow-up, the patient was pain free in both hips.

Discussion

A disease of unknown etiology, RDO has a painful and debilitating course that often ends in THA. Although other bone loss conditions of the femoral head such as osteonecrosis have been associated with corticosteroid use [8], hemoglobinopathies [9], and alcohol consumption [10], no such relationships have been demonstrated for RDO. When encountering radiographs demonstrating rapid bone loss of the femoral head, the physician must be able to make an accurate diagnosis to provide the patient with the best possible treatment. A thorough preoperative workup can help guide clinical decision-making if unexpected intraoperative findings are encountered.

A review of the literature regarding RDO reveals a better understanding of its clinical course than its pathophysiology. Previous case series have described a predominantly laterally based disease of the femoral head that tends to present in elderly women [3,7,11]. As the name implies, the pain and disability associated with RDO tend to develop relatively quickly, often within 6 months [6]. The femoral and acetabular bone loss seen in the condition is classically atrophic in nature, with an almost complete absence of osteophytes [12].

Summary

As previously described in the literature, patients with RDO tend to lack any obvious risk factors for osteonecrosis and have no clinical or laboratory evidence of an infectious, neurologic, metabolic, endocrinologic, or inflammatory conditions [6,7]. A proposed theory regarding its development suggests that it results from the interaction of 3 factors: mechanical stress, cartilage degeneration, and bone response. If cartilage degeneration is relatively slow and bone response is good, then reparative sclerosis and osteophyte formation can occur. Conversely, atrophic or destructive osteoarthritis can result if cartilage degeneration is rapid and bone response is poor [4,11,13]. Other authors have implicated nonsteroidal anti-inflammatory medications as an etiology of RDO;

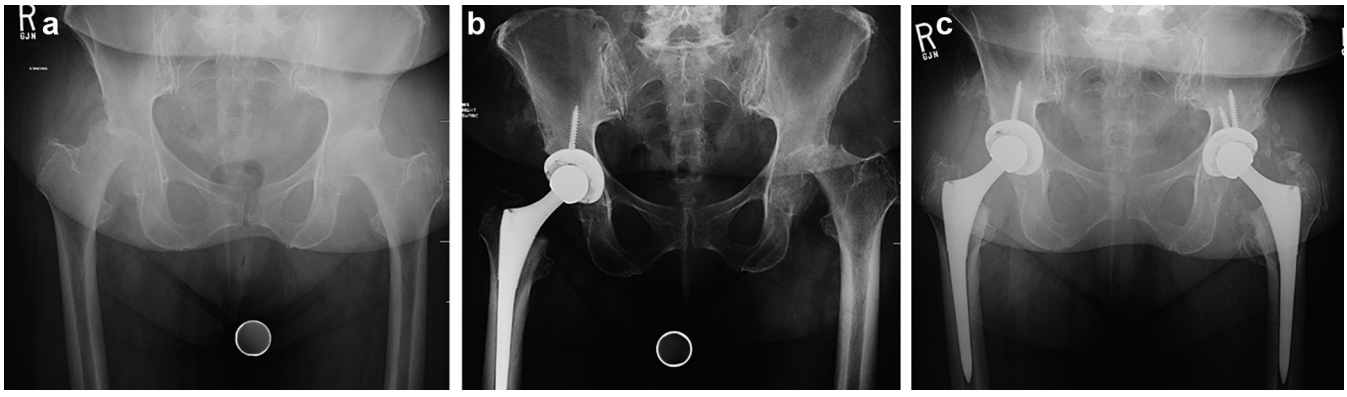

Figure 3. Patient 3 at presentation (a), at 1 month postoperatively (b), and 18-month follow-up (c).

however, nonsteroidal anti-inflammatory medication use is not universal in RDO patients, and a causal relationship has been difficult to establish [14]. Intra-articular hydroxyapatite or pyrophosphate crystal deposition has also previously been cited as a cause of RDO; however, crystal deposition is a nonspecific and inconsistent finding and has been demonstrated in 30%-50% of patients with conventional osteoarthritis [15-17]. What has been observed histologically is a marked increase in the osteoclast count in hips with RDO compared with hips with conventional osteoarthritis [18]. In addition, elevated intra-articular levels of interleukin 6, interleukin 1β, and matrix metalloproteinases have been documented [19,20]. These biological factors may play a role in joint destruction in RDO; however, their exact contribution is incompletely understood.

We propose a diagnostic algorithm for managing patients presenting with rapidly destructive bone loss around the hip. When rapidly progressive, atrophic bone loss is noted on radiographs, we suggest obtaining a preoperative ESR and CRP. If these studies are elevated, then a hip aspirate should be performed. If these studies are not consistent with infection, these patients are felt to have RDO and may undergo THA regardless of the appearance of the intra-articular fluid. If they are consistent with infection, a 2-stage procedure is performed. If an aspirate was not performed and one is still concerned about the appearance of the joint fluid during THA, an intra-operative cell count, frozen section, or leukocyte esterase dip stick can help guide management. We feel that this strategy can help resolve the uncertainty of proceeding with arthroplasty when encountering rapidly progressive joint destruction preoperatively or purulent-appearing fluid at the time of surgery, thus helping to avoid the morbidity of an unnecessary 2-stage procedure.

References

1. Forestier F. Coxites rhumatismales subaigues et chroniques; 1957 ([Thesis. Paris. Cited in: Rosenberg ZS, Shankman S, Steiner GC, et al. Rapid destructive osteoarthritis: clinical, radiographic, and pathologic features. Radiology 1992;182:213]).
2. Lequesne M. La coxarthrose destructrice rapide. Rhumatologie 1970;22:21.
3. Postel M, Kerbouli M. Total prosthetic replacement in rapidly destructive arthrosis of the hip joint. Clin Orthop Relat Res 1970;72:138.
4. Solomon L, Schnitzer CM. Pathogenetic types of coxarthrosis and implications for treatment. Arch Orthop Trauma Surg 1983;101(4):259.
5. Typpo T. Osteoarthritis of the hip: radiologic findings and etiology. Ann Chir Gynaecol Suppl 1985;201:1.
6. Batra S, Batra M, McMurtie A, Sinha AK. Rapidly destructive osteoarthritis of the hip joint: a case series. J Orthop Surg Res 2008;3:3.
7. Rosenberg ZS, Shankman S, Steiner GC, et al. Rapid destructive osteoarthritis: clinical, radiographic, and pathologic features. Radiology 1992;182(1):213.
8. Felson DT, Anderson JJ. A cross-study evaluation of association between steroid dose and bolus steroids and avascular necrosis of bone. Lancet 1987;1(8538):902.
9. Axelrod AR, Clifford GO, Tanaka KR. Sickle cell anemia (homozygous S) with aseptic necrosis of the femoral head. Blood 1956;11(11):998.
10. Matsuo K, Hirohata T, Sugioka Y, Ikeda M, Fukuda A. Influence of alcohol intake, cigarette smoking, and occupational status on idiopathic osteonecrosis of the femoral head. Clin Orthop Relat Res 1988;(234):115.
11. Danielsson LG. Incidence and prognosis of coxarthrosis. Clin Orthop Relat Res 1993;(287):13.
12. Delia Torre P, Picuti G, Di Filippo P. Rapidly progressive osteoarthritis of the hip. Ital J Orthop Traumatol 1987;13(2):187.
13. Solomon L, Schnitzler CM, Browett JP. Osteoarthritis of the hip: the patient behind the disease. Ann Rheum Dis 1982;41(2):118.
14. Ronningen H, Langeland N. Indomethacin treatment in osteoarthritis of the hip joint. Acta Orthop Scand 1979;50(2):169.
15. Dieppe PA, Doherty M, MacFarlane DG, et al. Apatite associated destructive arthritis. Br J Rheumatol 1984;23(2):84.
16. Dieppe P, Watt I. Crystal deposition in osteoarthritis: an opportunistic event? Clin Rheum Dis 1985 Aug;11(2):367.
17. Martel W, Champion CK, Thompson GR, Carter TL. A roentgenologically destructive arthropathy in some patients with the pseudogout syndrome. Am J Roentgenol Radium Ther Nucl Med 1970;109(3):587.
18. Mitrovic DR, Riera H. Synovial, articular cartilage and bone changes in rapidly destructive arthropathy (osteoarthritis) of the hip. Rheumatol Int 1992;12(1):17.
19. Komiya S, Inoue A, Sasaguri Y, Minamitani K, Morimatsu M. Rapidly destructive arthropathy of the hip. Studies on bone resorptive factors in joint fluid with a theory of pathogenesis. Clin Orthop Relat Res 1992;(284):273.
20. Tamai M, Sagawa K, Kawabata R, Inoue A, Itoh K. Production of IL-6 by T cells from the femoral head of patients with rapidly destructive coxopathy (RDC). Clin Exp Immunol 1996;103(3):506.