

What guidelines/algorithms (both operative and nonoperative) are there for the treatment of osteolysis?

Osteolysis is the conclusion of a complex particle-induced biologic process resulting in bone loss and in some cases implant loosening.¹⁻⁵ Early diagnosis of osteolysis requires interval radiographic evaluation of patients with joint replacements. The success of early treatment underscores the need for consistent longitudinal surveillance.

The incidence of osteolysis is likely increasing as patients live longer and remain more active. Twenty-five years ago, a total hip replacement in a 65-year-old patient was expected to last for a lifetime. With increased longevity, however, hip replacements remain in service for many years. Significant activity demands can result in marked wear particle production.

Treatment of osteolysis in asymptomatic patients⁶ is far different than treatment of patients with symptoms of a loose implant or pain from impending pathologic fracture. The goals of treatment of asymptomatic patients are preservation of bone stock, reduction of the risk of catastrophic periprosthetic fracture, and restoration of the articulation with the best material combinations currently available. Any treatment designed to address osteolysis must accomplish two key functions—debridement of the lesion, and modification of the articulation to decrease particle generation.⁷ These treatment components are frequently referred to as the osseous lesion and the wear generator.

Although there is no clear consensus on the necessary frequency of radiographs following total hip replacement, most authors agree that images should be obtained 1 year after surgery and then at intervals of 1 to 2 years. When periprosthetic osteolysis is diagnosed, the radiographic evaluation should be more frequent in order to quantify the rate of progression and provide a clinical opportunity to question the patient for clues that might indicate a loose implant or impending pathologic fracture. The patient can also be informed about the process and its treatment. Patients are followed at 3- to 6-month intervals for several visits until a predictable pattern can be determined. If stable clinical and radiographic patterns are identified, then less frequent follow-up is acceptable. Each encounter should be used to improve patient understanding of the disease and its treatment. The addition of oblique pelvic views increases the sensitivity for the detection of osteolysis in total hip patients.

Acetabular Osteolysis

Several groups have proposed classifications of acetabular osteolysis. Paprosky and associates⁸ developed a classification system for cemented implants that is based on the integrity of the acetabular rim and predicts the type of bone grafting that will be required to attain a stable implant. Type I defects involve minimal deformity. These are small, contained defects and are amenable to cancellous bone grafting. A cementless acetabular component, usually larger in diameter than the shell used in the primary arthroplasty, can be used to achieve stable fixation. Type II defects represent distortion of the normal acetabular hemisphere. In these defects, the anterior and posterior columns are intact although the medial wall and superior dome may be deficient. The reconstruction options include a high hip center, cancellous bone graft, femoral head allograft, and a variety of specialized components designed to replace deficient bone. Type III defects have severe bone loss requiring the use of structural allograft; examples include severe acetabular protrusio, column deficiencies, and pelvic discontinuity with associated global deficiency.

The American Academy of Orthopaedic Surgeons Committee on the Hip (COH) classification has two basic categories, segmental and cavitary. Segmental defects result from complete bone loss in one area of the acetabulum and are subclassified into peripheral and central defects. Cavitary bone deficiencies represent contained bone loss with the acetabular rim remaining intact. This system has not been as useful as Paprosky's because it has not been combined with a specific treatment algorithm.

For uncemented acetabular components, a new classification system with treatment algorithm has been created (Fig. 1).⁷ In the presence of acetabular osteolysis, type I implants are stable and have intact locking mechanisms. This combination can be treated with liner exchange, downsizing the femoral head if desirable, and cancellous bone grafting. Because lesions have been demonstrated to heal without grafting after liner exchange,⁷ it is not always necessary to place cancellous graft into the lesions. Type II components are also stable; however, the function of the cup is compromised. Examples of Type II include a broken locking mechanism, backside wear of the shell so that the polyethylene liner would be unsupported or abraded, or component malposition. Treatment options include placement of a new polyethylene liner (possibly even a custom type) and acetabular shell retention with cementation of a new polyethylene liner. Acetabular shell removal may be necessary. Preoperative planning is critical if an existing component is to be left in place. Type III implants are loose and may have migrated. Acetabular screws should be removed at the time of revision to allow for stability testing and to permit improved access for lesion debridement. If grafting is to be performed, sufficient access may be achieved through the screw holes but may require a pelvic window. Debridement through the shell may be accomplished by curettage. Cancellous bone grafting or placement of bone graft substitutes is done through the same access. It is not yet known if grafting is required for some of these smaller lesions to heal.

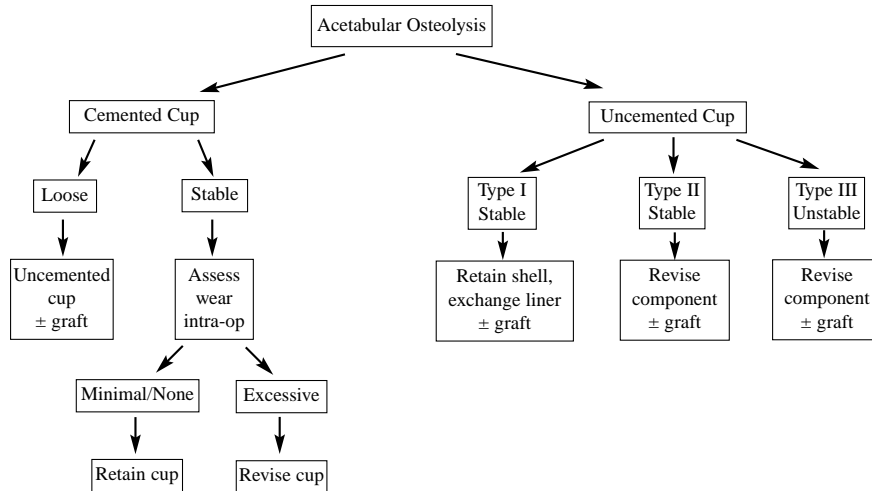


Figure 1 Treatment of acetabular osteolysis. (Adapted with permission from Rubash HE, Sinha RK, Maloney WJ, Paprosky WG: *Osteolysis: Surgical treatment. Instr Course Lect* 1998;47:321-329.)

Removal of the continuous source of particles is necessary. For cemented cups, the treatment depends on whether the implant is loose or stable (Fig. 1).

Femoral Osteolysis

The classification of femoral osteolysis proposed by the COFH has been useful. Femoral defects are classified as segmental, cavitory, or combined lytic. Segmental defects are characterized by erosion of the cortical bone, and are subclassified into complete or partial deficiencies. Cavitory defects represent contained lesions with destruction of endosteal bone. Combined defects, which include some element of both segmental and cavitory bone loss, are the most common type of femoral bone loss from osteolysis.

Treatment of asymptomatic femoral osteolysis varies with the extent and the progression. The extent of lytic changes should be carefully delineated; extensive radiographic evaluation may be required. Comparison of early postoperative radiographs with the most recent images demonstrating lysis is critical. This important comparison has become increasingly difficult, however, because of newer digital film storage and destruction of older radiographs. The economic demands of the health care environment must not lead to destruction of these important early evaluations.

The surgeon must determine if the femoral component is loose, if there is an impending pathologic fracture, and if there is eccentric polyethylene wear, all of which are relative indications for surgical intervention. In the presence of osteolysis, two special situations suggest more expedient surgical treatment—a loose or debonded femoral component with a rough surface texture, and a worn acetabular component with thin polyethylene.

Femoral osteolysis treatment goals are to maximize fixation and overall bone quality and minimize iatrogenic bone loss from revision surgery.

Treatment algorithms can be developed for femoral revision after considering the stability of the implant and its fixation, the need for cancellous and structural grafting, and the surgical approaches for removal (Fig. 2).

Pharmacologic Treatments

As our understanding of the biologic sequences that lead to osteolysis improves, nonsurgical treatment of osteolysis may become possible. Investigators have studied the use of nonsteroidal anti-inflammatory drugs and even gene transfer techniques to inhibit the inflammatory component of osteolysis.⁹⁻¹¹ In addition, newer medical therapies have been developed that may interrupt osteolytic progression. Shanbhag and associates¹²⁻¹⁴ demonstrated in a canine model that bisphosphonates such as alendronate can be successfully used to prevent osteolysis. Although the peri-implant bone resorption was prevented in their model, the inflammatory response was not affected. This finding was not unexpected because bisphosphonates exert their effect primarily on the osteoclasts with no known anti-inflammatory effects. The efficacy of this drug in treating periprosthetic osteolysis is currently being evaluated in a multicenter placebo-controlled clinical trial. Such pharmaceutical therapy may be clinically useful in the early stages of osteolysis before the lesion has compromised implant stability, or in cases where surgery is either too complex or risky.

Relevance

Periprosthetic osteolysis is a progressive disease that requires careful radiographic evaluation. Progression of osteolysis to the level of substantial bone loss, impending pathologic fracture, or to the degree that future reconstruction would be compromised are indications for revision surgery. Limited revision surgery that involves exchange of the polyethylene liner, debridement of osteolytic lesions, retention of a stable femoral component, and

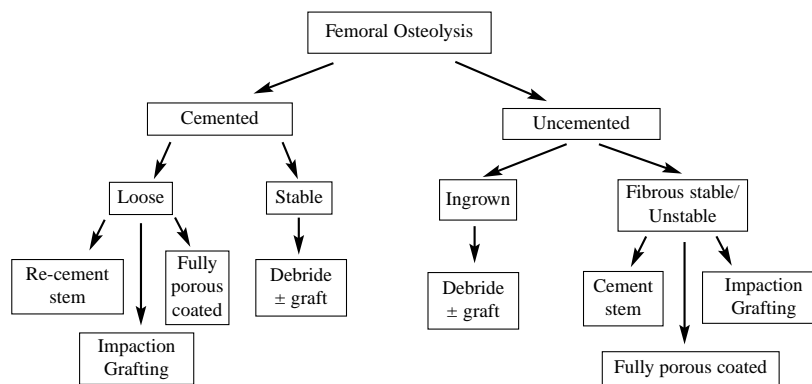


Figure 2 Treatment of femoral osteolysis. (Adapted with permission from Rubash HE, Sinha RK, Maloney WJ, Paprosky W.: *Osteolysis: Surgical treatment. Instr Course Lect* 1998;47:321-329.)

placement of a new femoral head have been successful in appropriate cases. The indications for bone grafting of acetabular and femoral lesions are not well defined. Finally, nonsurgical treatment of osteolysis could become a clinical reality.

Future Directions for Research

Will the newer materials (highly elevated cross-linked polyethylenes, ceramic-ceramic, and metal bearings) prevent osteolysis? If lysis does occur, will the clinical manifestations be similar? What modifications can be made to implant surfaces to prevent periprosthetic osteolysis? Is there an optimal articulation—lowest wear, least osteolysis, best range of motion and stability—and how should it be defined? Should advanced imaging techniques such as radiostereometric analysis or in-office digitized radiography be routinely used to detect early component wear? Are there low risk, inexpensive medical therapies for osteolysis?

References

1. Goldring SR, Schiller AL, Roelke M, Rourke CM, O'Neil DA, Harris WH: The synovial-like membrane at the bone-cement interface in loose total hip replacements and its proposed role in bone lysis. *J Bone Joint Surg Am* 1983;65:575-584.
2. Goodman SB, Chin RC, Chiou SS, Schurman DJ, Woolson ST, Masada MP: A clinical-pathologic-biochemical study of the membrane surrounding loosened and non-loosened total hip arthroplasties. *Clin Orthop* 1989;244:182-187.
3. Harris WH: The problem is osteolysis. *Clin Orthop* 1995;311:46-53.
4. Jiranek WA, Machado M, Jasty M, et al: Production of cytokines around loosened cemented acetabular components: Analysis with immunohistochemical techniques and in situ hybridization. *J Bone Joint Surg Am* 1993;75:863-879.
5. Schmalzried TP, Jasty M, Harris WH: Periprosthetic bone loss in total hip arthroplasty: Polyethylene wear debris and the concept of the effective joint space. *J Bone Joint Surg Am* 1992;74:849-863.
6. Maloney WJ, Herzwurm P, Paprosky W, Rubash HE, Engh CA: Treatment of pelvic osteolysis associated with a stable acetabular component inserted without cement as part of a total hip replacement. *J Bone Joint Surg Am* 1997;79:1628-1634.
7. Rubash HE, Sinha RK, Maloney WJ, Paprosky WG: Osteolysis: Surgical treatment. *Instr Course Lect* 1998;47:321-329.
8. Paprosky WG, Perona PG, Lawrence JM: Acetabular defect classification and surgical reconstruction in revision arthroplasty: A 6-year follow-up evaluation. *J Arthroplasty* 1994;9:33-44.
9. Goodman SB, Chin RC, Chiou SS, Lee JS: Suppression of prostaglandin E2 synthesis in the membrane surrounding particulate polymethylmethacrylate in the rabbit tibia. *Clin Orthop* 1991;271:300-304.
10. Spector M, Shortkroff S, Hsu HP, Lane N, Sledge CB, Thornhill TS: Tissue changes around loose prostheses: A canine model to investigate the effects of an antiinflammatory agent. *Clin Orthop* 1990;261:140-152.
11. Wooley PH, Sud S, Robbins PD, Whalen JD, Evans CH: Contrasting effects of gene therapy to inhibit interleukin-1 β or tumor necrosis factor alpha in the murine inflammatory response to wear particles. *Trans Orthop Res Soc* 1998;23:122.

Implant Wear in Total Joint Replacement

12. Shanbhag AS, Hasselman CT, Rubash HE: The John Charnley Award: Inhibition of wear debris mediated osteolysis in a canine total hip arthroplasty model. *Clin Orthop* 1997;344:33-43.
13. Shanbhag AS, Jacobs JJ, Black J, Galante JO, Glant TT: Macrophage/particle interactions: Effect of size, composition and surface area. *J Biomed Mater Res* 1994;28:81-90.
14. Shanbhag AS, Jacobs JJ, Glant TT, Gilbert JL, Black JM, Galante JO: Composition and morphology of wear debris in failed uncemented total hip replacement. *J Bone Joint Surg Br* 1994;76:60-67.