

# Hip painful prosthesis: surgical view

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## Summary

**Painful hip prosthesis is the most feared immediate and remote complication of a primary implant and usually represents the failure of one or more therapeutic moments. In cases of aseptic implant failure, the causes invoked may be represented by an incorrect indication, the quality of materials, local and general condition of the patient and especially from a bad joint biomechanics. In cases of septic loosening, however, the cause of failure to be found in the location of pathogens within the implant. In planning a revision is necessary to respect many important steps. They are represented by the exact identification of the causes of failure, the correct preoperative planning, by respecting the skin incisions, the proper choice of the prosthesis, planning the surgical technique, and finally by an appropriate rehabilitation program.**

**In the evaluation of hip failure the first diagnostic step is to recognize exactly those aseptic and septic forms anyway to exclude the diagnosis of infection.**

*KEY WORDS: THA; loosening; infection; bone-loss.*

## Introduction

The long-term success after total hip arthroplasty depends on maintaining a good fixation of prosthetic components. The periprosthetic osteolysis is the most common complication after primary arthroplasty, and the consequent easing pain are the main causes of prosthetic revision. The loss of acetabular component fixation occurs in approximately 2-8% of hip replacement, femoral component loosening occurs in percentages between 6% and 18% of cases (1, 2). Bone loss is predominantly due to wear-mediated inflammation. However, bone resorption may be multifactorial, and micromotion and altered mechanical loading may also play a role. Periprosthetic osteolysis (Figure 1) is progressive and may be com-

plicated by joint failure or periprosthetic fracture with the subsequent need for surgical revision (3, 4). Therefore, diagnostic imaging may be helpful in accurately evaluating the extent and distribution of osteolysis, in anticipation of further surgical management (5, 6).

Hip revision surgery is rapidly increasing because of the increasing number of arthroplasties implanted in recent years.

## Discussion

A painful hip must be considered infected until the contrary. It is therefore important to assess the type and sequence of pain, general and local conditions of the patient, evaluate laboratory tests, the X-ray and if in doubt do other imaging examinations. It is most important to distinguish aseptic periprosthetic osteolysis from loosening caused by infection. Infection complicates approximately 2% to 3% of hip arthroplasties (7). The diagnosis of infection depends on a combination of clinical features (erythema, warmth,



Figure 1 - Periprosthetic osteolysis of the proximal femur due to poly wear.

joint swelling, effusions), laboratory tests (elevated white blood cell count, erythrocyte sedimentation rate, C-reactive protein), and diagnostic imaging. While both aseptic periprosthetic osteolysis and infection may demonstrate similar imaging characteristics, infection is more likely to be associated with endosteal scalloping, acute multisite periosteal reaction, and periprosthetic bone resorption (8, 9).

The pre-operative planning is critical to the success of a prosthetic revision surgery.

The objectives to be met are:

- identify the causes of failure
- assess the patient's general condition
- assess acetabular and femoral bone-loss
- evaluate the biomechanical changes

The assessment of these points yields the following results:

- choose the most appropriate therapeutic strategies
- anticipate and be ready to treat any intraoperative complications
- make a stable and pain-free system
- preserve or increase bone stock
- restore joint biomechanics

The mobilization of the hip is defined as the loss of stability of a prosthesis or a failure or stabilization of a prosthesis that requires a secondary integration to complete the initial primary stability.

The mobilization is a gradual process that recognizes biological and mechanical causes.

The mechanics have had more to design and prosthetic materials, bone quality, the technique of implanting (positioning, cemented or cementless), the quality of the bone-prosthesis or bone-cement-prosthesis interface.

The biological one consists of a gradual wear of materials (polyethylene, metal, etc.) that involves the release of debris in joint bring inflammation reaction that result in the prosthesis mobilization (10-12).

Another cause of implant loosening may be the periprosthetic fracture that can occur following an efficient trauma, or more frequently in the presence of osteolytic areas as a result of inefficient trauma.

In fact, the mechanical and biological causes are not always separable, the prosthesis change the distribution of mechanical forces on the bone that involves biological reactions of physiological adaptation and on the other hand, the production of debris is related to mechanical factors that accelerate material's wear (13). Currently, the problem of the longevity of the system is essentially linked to osteolysis; osteolysis is the initial findings reported in indurated and not mobilized. Osteolysis is the biological response to wear debris originating materials by mechanical or chemical action. The greatest source of debris is the acetabular articular surface between the liner and head prosthesis (14, 15). Wear particles from the bearing surfaces play a major role in initiating periprosthetic osteolysis, which is also potentiated by mechanical factors such as increased synovial fluid pressure. The precise mechanisms by which wear particles induce periprosthetic osteolysis have not been fully elucidated and remain an active subject of research.

Particle characteristics such as composition, size, shape, and number (especially for particles in the most biologically active, sub-micrometer-size range) are recognized to significantly affect the overall cell and tissue response. The polyethylene particles that are smaller than 1 micron cause greater tissue reaction. Those produced by the friction of metal collar at the junction of the prosthetic head, and screw cup of modular components are larger than the particles of polyethylene. Their action may be indirect, acting as a third body in poly-wear or live as free metal ions can trigger the cellular response. Debris stimulate an inflammatory respon-

se to foreign body: those less than 1 micron are engulfed by macrophages while the larger (15-20 microns) are incorporated by multinucleated giant cells may be extracellular or inducing a chronic inflammatory response. The production of corrosion products, especially from metal-on-metal implants, also is a clinically significant issue, and individual variability in innate and adaptive immune responses is important but not yet completely defined (16, 17) (Figure 2).

However, wear particles are resistant to enzymatic degradation, digestion, or inflammatory factors. This results in the prolonged presence of particles, continued activation of an inflammatory response, and the release of various cytokines that affect osteoclast differentiation and activity (TNF, RANKL, IL-6, IL-1, and IL-11). The prolonged duration of inflammatory activity promotes progressive osteolysis (17).

In the past the molecular pathogenesis of osteolysis was attributed to the release of inflammatory mediators (cytokines, interleukins, etc.), now identified a direct action of macrophages that can initiate the bone resorption by osteoclasts. Thus was osteolysis and the formation of a periprosthetic fibrous membrane that spreads within the "effective joint space": the distribution of the debris can be passively because of pressure from joint or through the lymphatic vessels. The extension of the osteolytic areas leads to an increase of micromovements of the bone-prosthesis interface resulting in increased production of debris, and then amplification



Figure 2 - Breakage of the stem probably due to fatigue.

of the phenomenon that inevitably leads to the mobilization of the implant. For this reason, the periprosthetic bone loss is the main problem in aseptic loosening, it is the expression of the phenomena of aggression by the reactive granulation tissue debris penetrating to the bone-cement or bone-prosthesis interface. The correct evaluation of the extension, repeatable and extent of loss of substance is a key parameter for the choice of the technique of revision. There are in literature different classifications of bone loss in acetabular and femoral bone (18-23). Currently the most widely used is that of Paprosky, which allows the classification of bone defects with plain radiographs of the pelvis and hip than evaluating the migration and / or medialization of the acetabular component, osteolysis ischial and radiographic drop.

- Type I: Minimal loss of metaphyseal cancellous bone. Intact diaphysis. Consider cemented vs cementless fixation.
- Type II: Extensive loss of metaphyseal cancellous bone. Intact diaphysis. Loss of cancellous bone makes cemented fixation more suspect, consider uncemented fixation (24-26) (Figure 3).
- Type III-A: The metaphysis is not supportive. There remains greater than 4 cm of bone in the diaphysis to allow for a scratch fit. Consider uncemented fixation with a fully porous-coated stem.
- Type III-B: The metaphysis is not supportive. There remains less than 4 cm of bone in the diaphysis to allow for a scratch fit. Due to short segment of cylindrical bone to support a fully-porous coated stem, the failure rate is high with such a device. Consider a modular tapered stem.
- Type IV: Wide open canal without any appreciable isthmus to support an uncemented stem. Consider impaction grafting if the proximal tube is intact +/- an intact calcar. Other alternatives would include a modular tumor megaprosthesis.

Similar to his femoral defect classification, this system attempts to stratify the degree of host bone loss in order to estimate the ability to achieve stable cementless fixation for any given bone loss pattern.

Four landmarks require evaluation:

- Femoral head center as measured from Hilgenreiner's line (horizontal line connecting the inferior aspects of the teardrops or the superior margins of the obturator foramina). Note any superior displacement greater than 3 cm and if the displacement tends to go medial or lateral.
- Ischial osteolysis- as measured from Hilgenreiner's line inferiorly to the edge of the osteolytic lesion in the ischium. Greater than 1.5 cm of bone loss represents 20-25% loss of the acetabular bone stock.
- Tear Drop- Loss of the radiographic tear drop indicates damage to the medial wall as well as the inferior portions of the columns, ~ 10-15% host bone loss.
- Kohler's line- breakthrough medial to this line (the ilioischial line) represents medial wall destruction and likely damage to the midportion of the columns.
  - Migration relative to this line can be graded as follows:
    - Grade 1- the socket remains lateral to the line
    - Grade 2- the socket has migrated to, not through, the line
    - Grade 3- the socket has migrated medially into the pelvis

Type 1- there is an intact rim with little or no migration superior or medial. The teardrop and ischium are intact.

- Treat with an uncemented hemisphere (with screws) and possibly cancellous bone grafting to small defects.

Type 2A- minimal increase in bone destruction, but any superior migration is less than 3 cm (i.e. superomedial bone loss with an intact rim).

- Treat with an uncemented hemisphere (with screws) and cancellous bone grafting to defects.

Type 2B- greater distortion of the superior rim (small superolateral



Figure 3 - Revision with Wagner stem using Wagner osteotomy.

segmental rim defect of less than 1/3 rim circumference) but less than 3 cm superior migration. The dome remains supportive and lysis in the teardrop/ischium is minimal. Medial migration to the medial wall, but not violated.

- Treat with an uncemented hemisphere (with screws) and cancellous bone grafting to any contained defects. There may be a role in some cases for a small structural bone graft superolateral to the segmental defect. However, as a rule this graft would be to restore bone stock because it would be a Type 3 if the bone graft was required for implant stability.

Type 2C- similar to 2B with migration medial to Kohler's line and moderate to severe teardrop osteolysis. Minimal ischial osteolysis. The dome remains supportive. This is the case with an intact rim but no medial wall.

- Treat with an uncemented hemisphere (with screws) and cancellous bone grafting to defects. Consider the use of a "medial wafer" structural bone graft.

Type 3A- significant superior dome destruction with greater than 3 cm superolateral migration. Kohler's line is intact, but there is moderate ischial and teardrop lysis (Figure 4). There is usually adequate host bone for ingrowth, but the cup requires some form of augmentation to achieve implant stability.

- Treat with an uncemented cup with screws and:
  - A structural bone graft- femoral head
  - A modular prosthetic graft- e.g. wedges
  - Place the cup into the high hip center

Type 3B- significant superior dome destruction with greater than



Figure 4 - Luxation of the stem due to a massive periacetabular bone loss subsequent to osteolysis.

3 cm superomedial migration. Kohler's line is broken. There is severe ischial and teardrop lysis and likely a pelvic discontinuity. There is less than 40% host bone available for ingrowth and the rim defect is greater than  $\frac{1}{2}$  the rim circumference.

- Treatment follows two main principles:
  - Establish initial implant stability
  - Attempt to achieve biological fixation where possible
  - Structural allograft and uncemented cup with screws +/- plate the column
  - Cup-cage construct
  - Structural allograft + cage + cemented cup
  - Cage + cemented cup

Bone defects therefore represent a difficult problem to solve in revision hip surgery. In fact it is often not possible to have sufficient quantities of autologous bone and it is therefore necessary to use a bank or allograft.

The conditions required for successful grafting are representative:

- Bone graft sterile and biologically active
- Host bone without infection and well vascularized
- Make a mechanical stability between the host bone graft

The grafts currently available are of various qualities and dimensions:

- Morcelized cancellous bone
- Chips of cancellous bone
- Blocks of cancellous bone
- Cortico-cancellous blocks
- Single or massive bone graft

The use of bone grafts is intended to restore an adequate bone stock and / or to make a mechanical support for the revision pro-

sthesis. The choice of graft is made according to the classification of bone defects presented above.

Acetabular bone grafts showed massive structured long-term high percentage of failures. In cases of segmental defects using grafts str-utturati support stabilized with screws or cemented cups for 8-10 years showed 54% of failures (28). So nowadays it is preferable to use the grafts in the form of morcellizzato and then place the acetabular component in neocotile or metal rings reinforced cups or jumbo-cup.

A femoral treatment of bone defects can be performed with cancellous grafts or grafts morcellizzati structured. The use of massive segmental transplantation is indicated in cases where there are proximal circumferential bone defects with greater length to 5 cm.

## References

1. Desal MA, Bancroft LW. Periprosthetic osteolysis. *Orthopedics* 2008; 31:518.
2. Abu-Amer Y, Darwech I, Clohisy JC. Aseptic loosening of total joint replacement: mechanism underlying osteolysis and potential therapies. *Arthritis Res Ther.* 2007; 9 Suppl 1:S6.
3. Harris WH. The problem is osteolysis. *Clin Orthop Relat Res.* 1995; (311):46-53.
4. Harris WH. Osteolysis and particle disease in hip replacement. A review. *Acta Orthop Scand.* 1994; 65(1):113-123.
5. Berquist TH. Imaging of joint replacement procedures. *Radiol Clin North Am.* 2006; 44(3):419-437 Howie DW, Neale SD, Stamenkov R, McGee MA, Taylor DJ, Findlay DM. Progression of acetabular periprosthetic osteolytic lesions measured with computed tomography. *J Bone Joint Surg Am.* 2007; 89(8):1818-1825.
6. Romanò CL, Romanò D, Logoluso N, Meani E. Septic versus aseptic hip revision: how different? *J Orthop Traumatol.* 2010 Sep;11(3):167-74.
7. Fujishiro T, Moojen DJ, Kobayashi N, Dhert WJ, Bauer TW. Perivascular and Diffuse Lymphocytic Inflammation are not Specific for Failed Metal-on-metal Hip Implants. *Clin Orthop Relat Res.* 2010 Oct 29.
8. Catelas I, Jacobs JJ. Biologic activity of wear particles. *Instr Course Lect.* 2010;59:3-16.
9. Aspenberg P, Herbertsson P. Periprosthetic bone resorption. Particles versus movement. *J Bone Joint Surg Br.* 1996; 78(4):641-646.
10. Engh CA, O'Connor D, Jasty M, McGovern TF, Bobyn JD, Harris WH. Quantification of implant micromotion, strain shielding, and bone resorption with porous-coated anatomic medullary locking femoral prostheses. *Clin Orthop.* 1992:13-29.
11. Burke DW, O'Connor DO, Zalenski EB, Jasty M, Harris WH. Micromotion of cemented and uncemented femoral components. *J Bone Joint Surg Br.* 1991;73:33-37.
12. Engh CA Jr, Young AM, Engh CA Sr, Hopper RH Jr. Clinical consequences of stress shielding after porous-coated total hip arthroplasty. *Clin Orthop Relat Res.* 2003;417:157-163.
13. Phillips TW, Nguyen LT, Munro SD. Loosening of cementless femoral stems: a biomechanical analysis of immediate fixation with loading vertical, femur horizontal. *J Biomech.* 1991;24:37-48.
14. Panjabi MM, Trumble T, Hult JE, Southwick WO. Effect of femoral stem length on stress raisers associated with revision hip arthroplasty. *J Orthop Res.* 1985;3:447-455.
15. Gallo J, Zdarilová A, Rajnochová A, Ulrichová J, Radová L, Smižanský M. Synovial fluid from aseptically failed total hip or knee arthroplasty is not toxic to osteoblasts. *Acta Chir Orthop Traumatol Cech.* 2010 Oct;77(5):416-24.
16. Carr AM, DeSteiger R. Osteolysis in patients with a metal-on-metal hip arthroplasty. *ANZ J Surg.* 2008; 78(3):144-147.
17. D'Antonio J, McCarthy JC, Bargar WL, Borden LS, Cappel WN, Collis DK, Steinberg ME, Wedge JH. Classification of femoral abnormalities in total hip arthroplasty. *Clin Orthop Relat Res.* 1993 Nov;(296):133-9.
18. Paprosky WG, O'Rourke M, Sporer SM. The treatment of acetabular bone defects with an associated pelvic discontinuity. *Clin Orthop Relat Res.* 2005 Dec;441:216-20.

19. 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(1):33-44.
20. Della Valle CJ, Paprosky WG. The femur in revision total hip arthroplasty evaluation and classification. *Clin Orthop Relat Res*. 2004; (420):55-62.
21. Gruen TA, McNeice GM, Amstutz HC. Modes of failure of cemented stem-type femoral components: a radiographic analysis of loosening. *Clin Orthop RR*. 1979;141:17-27.
22. Gibbons CER, Davies AJ, Amis AA, Olearnik H, Parker BC, Scott JE. Periprosthetic bone mineral density changes with femoral components of differing design philosophy. *In Orthop*. 2001;25:89-92.
23. Talmo CT, Shanbhag AS, Rubash HE. Nonsurgical management of osteolysis: challenges and opportunities. *Clin Orthop Relat Res*. 2006; (453):254-264.
24. Engh CAJ, Ellis TJ, Koralewicz LM, McAuley JP, Engh CAS. Extensively porous-coated femoral revision for severe femoral bone loss: minimum 10-year follow-up. *J Arthroplasty*. 2002;17:955-960.
25. Dorr LD, Absatz M, Gruen TA, Saberi MT, Doerzbacher JF. Anatomic Porous Replacement hip arthroplasty: first 100 consecutive cases. *Semin Arthroplasty*. 1990;1:77-86.
26. Paprosky WG, Greidanus NV, Antoniou J. Minimum 10-year-results of extensively porous-coated stems in revision hip arthroplasty. *Clin Orthop*. 1999:230-242.
27. Lee PT, Clayton RA, Safir OA, Backstein DJ, Gross AE. Structural Allograft as an Option for Treating Infected Hip Arthroplasty with Massive Bone Loss. *Clin Orthop Relat Res*. 2010 Nov 16.
28. Kwong LM, Jasty M, Harris WH. High failure rate of bulk femoral head allografts in total hip acetabular reconstructions at 10 years. *J Arthroplasty*. 1993 Aug;8(4):341-6.