POLYETHELENE WEAR AND OSTEOLYSIS

Dr.Vajara Wilairatana
Department of Orthopaedics
Chulalongkorn University Hospital, Bangkok Thailand

The most frequent long - term complication of joint replacement is loosening. Acrylic bone cement has been blamed for aseptic loosening of totol joint arthroplasty components and the term "Cement disease" has been coined for this phenomenon. The osteolysis associated with polyethelene debries has become a major concerned. Polyethelene particles are often associated with macrophages in failed joint replacement. These osteolytic granulomatous process are widely considered to be the result of foreign body reaction to particulate debries. We study two case of extensive osteolysis behind a well fixed acetabulum component that had been inserted with cement. Revision with Muller acetabular reinforcement ring and bone graft was done. Histologicallly the membrane were found to contain numerous multinucleated giant cells and foreign-body granulomatous inflammation.

INTRODUCTION

Osteolysis or bone loss is a well recognized complication of total hip replacement. Acrylic bone cement is commonly blamed for aseptic loosening of total joint arthroplasty component and the term "Cement disease" has been coined for this phenomenon. Miller[11] et al demonstrated the benefits of low viscosity cement to improved component fixation. Ling, et al[9] demonstrated that a dry bone bed facilitated bone cement interdigitation and improved interfacial integrity. Harris[5] showed the other means of improving cement fixation included vacaum mixing and centrifugation that removed air bubbles and prevented void in cement. Engh, et al[4] chose to eliminate cement entirely throught the use of biologic ingrowth surface but these have not solved the problem of aseptic loosening and have created others such as stress shielding and difficulty of removal in revision.

MECHANISM OF WEAR DEBRIS-INDUCED OSTEOLYSIS

Macrophages and giant cells has been described at the interfaces of nonloosened implant.[3] These may be remnant populations from the tissue repair process. In loosened implants also found to have

macrophages with wear debris. Interfacial tissue produce potent osteolytic, one of which is interleukin1[7]. Interleukin 1 is synthesized by serveral cell types, namely fibroblasts, endothelial cells and macoephages. Interlukin 1 may have bone resorptive activity, bone resorption is belived to be largely mediated by tumor necrosis factor alpha (TNF-α). TNF-α is also known as osteoclast activating factor (OAF), a cytokine secreted largely by macrophages.

Jiranek, et al.[8] studied 10 hip membranes surrounding loose cemented acetabular component and they can demonstrate production of two potent cytokines, Interleukin 1-α and platelet derived growth factor. These cytokines appear to be involved in the loosening process.

The macrophages can induce bone loss by the relase of modulating factors such as OAF, as well as directly, by the relase of oxide radicales and hydrogen peroxide[2]

Particulate debris, bacteria and cell death are activators of macrophages and can trigger further macrophage cell recruitment, phagocytosis and relase of osteolytic factor. Horowita, et al[6] demonstrated the particle size between 1 to 12 μm that phagocytized by macrophages produce active membrane and bone resorption. The concept of activated macrophage osteolysis is fundamental to the following concept implant fixation failure.

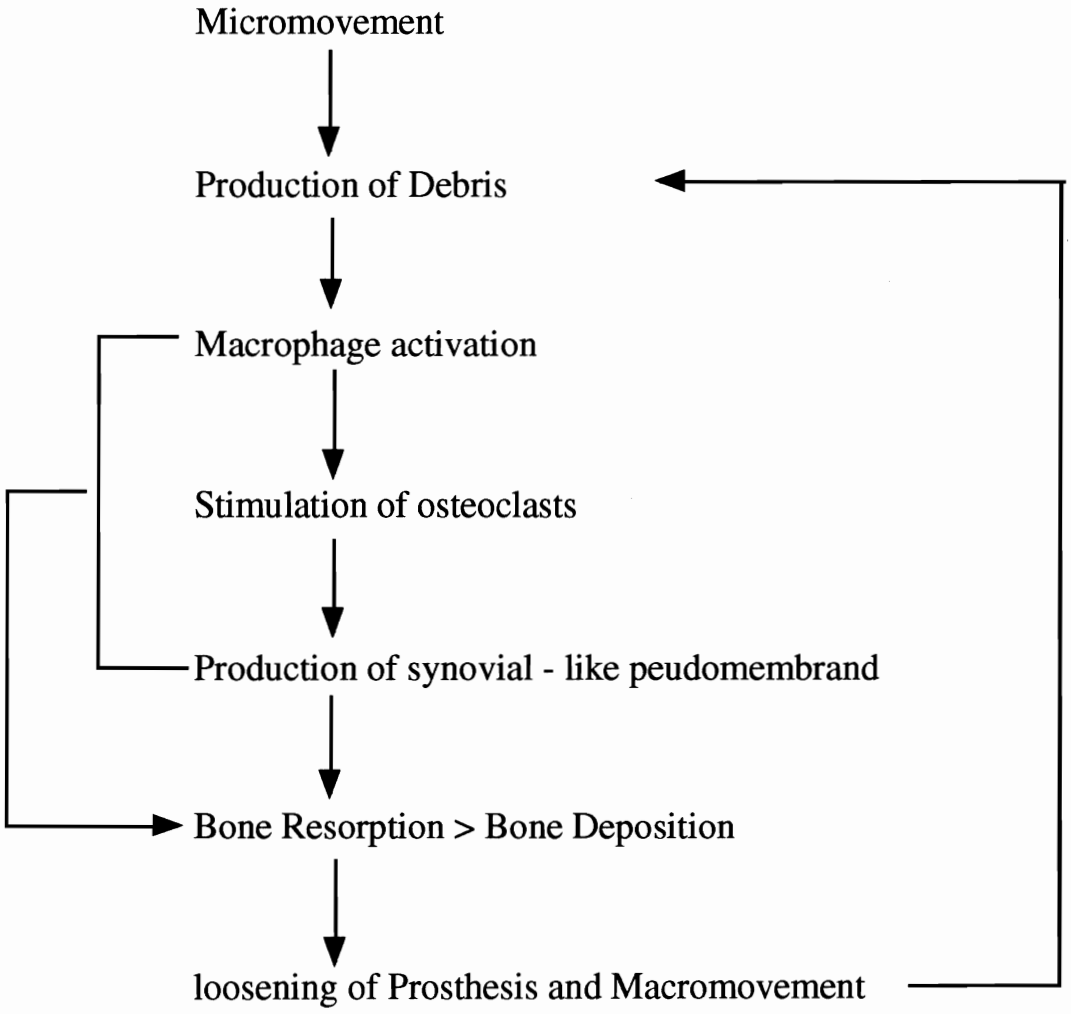
Amstertz, et al[1] concluded that macrophages are the primary agents in the bone loss seen in failing implants. The evidence for this is circumstantial 1) Macrophages are present in the tissue around loosened implants usually with an an abundance of intra cellular polymeric and metallic wear debris 2) Macrophages are known to secrete products that can cause bone lysis. (Table 1) and they can be and

Table 1. Cells that can produce osteolylic enzyme-or cytokins

Cells	Enzyme/cytokines
Macrophages fibroblasts Vascular endothelial cells	Collagenase Gelatinase Prostaglandin E2 Interleukin-1, Interlukin 1- β TNF-α Platelet derived growth factor

they can be these agent after phagocytosis 3) Tiny scallopsed edges are observed that correspond in size to the adjacent macrophange and that are seen in the absence of osteoclast.

The pathogensis of loosening process is seem to be multifactorial. Maquire, et al.[10] described pathogenesis of loosening and foreign body giant cell reaction (figure 1). Once the cellular and enzymatic destruction phase begin and the lymphatic/reticuloendothelial system is no longer able acceptabley to clear the particulate matter, regardless of its make up, the bone destruction proceeds at an ever-in creasing rate.



Finger 1. Pathogenesis of loosening and the foreign body, giant cell reaction

In 1996. We study histopathology from aseptic loosuing total hip repalacement. The techique has been apply from Mirra, et al[12]. We found polyethelne debris, metal debris and foreign body granuloma reaction from loosening acetabular component. Two hips

arthroplasty failures due to mark osteolysis at acetabular has abundant polyethelene debris.

SUMMARY

Aseptic implant loosening is a mechanical and biological problems in that it is intiated and aggravated by joint articulation and interfacial micromotion. The solution to the problems, however is not to deal with this mechanical aspect alone, since the wear characteristic of the materials will determine the generation of debris particles.

REFERENCE

1. Harlan C. Amstutz, M.D., Pat Campbell, B.Sc., Nir Kosso VSKY, M.D., and Ian C. Clarke, PH.D. : Mechanism and Clinical significance of Wear Debris-induced osteolysis. Clin. Orthop 1992 ; 276 : 7-18.
2. Coe, M.R., Fechner, R.E., Jeffey, J.J., Balian, g., and Whitehil, R., Characterization of tissue from the bone-polymethylmethacrylate interface in a rat experimental model. J bone Joint Surg. 71A : 1989 ; 863-75
3. Charnley, J. : The reaction of bone to self-curing acrylic cement. J. Bone Joint Surg 52B : 1970 ; 340-53.
4. Engh, C.A. : Hip arthroplasty with a Moor prosthesis with porous coating : A five-year study. Clin. Orthop 1983 ; 176 : 52-65.
5. Harris, W.H., and Davies, J.P. : Modern use of cement for total hip replacement. Orthop. Clin North Am 1988 ; 19 : 581-90
6. Horowitz SM, Doty SB, Lane JM,et al : Studies of the mechanism by which the mechanical failure of polymethymethacrylate leads to bone resorption. J Bone Joint Surg 1993 ; 75A : 802-813.
7. Goldring, S., Schiller, A., Roelke, M., Rourke, C.M., O'Neill, D.A., and Harris, W.H.: 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 1983 ; 65A : 575-84.
8. Jiranek WA, Machado M, Jasty M, et al : Production of cytokines around loosened cemented acetabular components J. Bone Joint Surg 1993 ; 75A : 863-879.
9. Ling, R. S.M. : Observation or the fixation of implants to bony skelaton. Clin. Orthop. 1986 ; 210 : 805-13.
10. James K. Maguire, JR., M.D.,Michail F. Coscia, M.D.,and Michael H.Lynch, M.D., Foreign Body Reaction to Polymeric Debris Following Total Hip arthroplasty. Clin. Orthop. 1987 ; 216 : 213-223.

11. Miller, J., Krause, W.R., Burke, D.L., Krug, W., and Kelebay, L.:Pressur penetration of low viscosity acrylic cement for improved fixaton of arthroplasty components. J. Bone Joint Surg, 64B : 619,1982.
12. Joseph M.Mirra, M.D., Harlan C.Amstutz, M.D., Maximo Matos,M.D. and Richarcl Gold, M.D. : The pathology of the Joint Tissues and its Clinical Relevance in Prosthesis failure. Clin. Orthop 1976;117:221-40.