Acta Biomaterialia 9 (2013) 8046–8058

Contents lists available at SciVerse ScienceDirect

Acta Biomaterialia

journal homepage: www.elsevier.com/locate/actabiomat

Review

Osteolysis around total knee arthroplasty: A review of pathogenetic mechanisms

J. Gallo [a,*], S.B. Goodman [b], Y.T. Konttinen [c,d,e], M.A. Wimmer [f], M. Holinka [g]

[a] Department of Orthopaedics, Faculty of Medicine and Dentistry, University Hospital, Palacky University Olomouc, I.P. Pavlova Str. 6, CZ-775 20 Olomouc, Czech Republic
[b] Department of Orthopaedic Surgery, Stanford University School of Medicine, Stanford, CA, USA
[c] Institute of Clinical Medicine, Department of Medicine, FIN-00029 HUS, Finland
[d] ORTON Orthopaedic Hospital of the Invalid Foundation, Helsinki, Finland
[e] COXA Hospital for Joint Replacement, Tampere, Finland
[f] Department of Orthopedic Surgery, Rush University Medical Center, Chicago, IL, USA
[g] Department of Orthopaedics, Karvina Mining Hospital, Czech Republic

ARTICLE INFO

Article history:
Received 7 February 2013
Received in revised form 12 April 2013
Accepted 2 May 2013
Available online 10 May 2013

Keywords:
Total knee arthroplasty/replacement
Osteolysis/aseptic loosening
Wear particles
Joint fluid
Knee biomechanics

ABSTRACT

Aseptic loosening and other wear-related complications are some of the most frequent late reasons for revision of total knee arthroplasty (TKA). Periprosthetic osteolysis (PPOL) pre-dates aseptic loosening in many cases, indicating the clinical significance of this pathogenic mechanism. A variety of implant-, surgery- and host-related factors have been delineated to explain the development of PPOL. These factors influence the development of PPOL because of changes in mechanical stresses within the vicinity of the prosthetic device, excessive wear of the polyethylene liner, and joint fluid pressure and flow acting on the peri-implant bone. The process of aseptic loosening is initially governed by factors such as implant/limb alignment, device fixation quality and muscle coordination/strength. Later, large numbers of wear particles detached from TKA trigger and perpetuate particle disease, as highlighted by progressive growth of inflammatory/granulomatous tissue around the joint cavity. An increased accumulation of osteoclasts at the bone–implant interface, impairment of osteoblast function, mechanical stresses and increased production of joint fluid contribute to bone resorption and subsequent loosening of the implant. In addition, hypersensitivity and adverse reactions to metal debris may contribute to aseptic TKA failure, but should be determined more precisely. Patient activity level appears to be the most important factor when the long-term development of PPOL is considered. Surgical technique, implant design and material factors are the most important preventative factors, because they influence both the generation of wear debris and excessive mechanical stresses. New generations of bearing surfaces and designs for TKA should carefully address these important issues in extensive preclinical studies. Currently, there is little evidence that PPOL can be prevented by pharmacological intervention.

1. Introduction

Total knee arthroplasty (TKA) relieves knee pain and improves function, and has a significant impact on the health-related quality of life. Currently, it is estimated that more than 1.5 million TKAs are performed worldwide each year (Health at a glance 2011; OECD indicators). However, some TKAs fail during the period of service and require revision surgery. Revision surgery brings less satisfactory outcomes and increased risk of complications [1]. In addition, it is more expensive than primary operations. Assuming an increasing number of primary TKAs in the coming decades, it is clear that the number of revision surgeries will also be increasing [2,3]. This could have a significant economic impact on the health care system. Therefore, understanding current failure mechanisms of primary TKA, and especially the potential for prevention, is critical in order to reduce an expected TKA revision burden.

Although infection, instability and patellofemoral problems dominate as reasons for reoperation in the first five years after index surgery, one of the most frequent late reasons for failure is aseptic loosening accompanied by periprosthetic osteolysis (PPOL), (Fig. 1) [4]. Chronologically, PPOL pre-dates aseptic loosening in the majority of cases, creating conditions facilitating implant loosening via weakening of the bone–implant interface. There is a relative paucity of reported studies related to the pathogenesis of osteolysis around TKA in comparison with total hip arthroplasty [5,6]. As in total hip arthroplasty, the pathogenesis of aseptic loosening and osteolysis in TKA is multifactorial, with contributions from surgeon-, patient- and implant-related factors. However, there are also important differences between total hip arthroplasty

* Corresponding author. Tel.: +420 588443548; fax: +420 58844548.
E-mail address: jiri.gallo@volny.cz (J. Gallo).

1742-7061/$ - see front matter © 2013 Acta Materialia Inc. Published by Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.actbio.2013.05.005

and TKA that preclude a direct translation of results from hip to knee arthroplasty. These differences are related to joint anatomy and kinematics, biomechanical function, implant design, biomaterials and resulting tribology.

The aim of this review is to summarize the present knowledge on the etiology and pathogenesis of PPOL of TKA.

2. Early bone changes after TKA surgery

The critical structure supporting TKA components is the periprosthetic bone bed. The fate of the TKA depends on the long-term ability of bone to withstand mechanical stresses and strains without deterioration in its micro-architecture and density, which would jeopardize the integration and support of the implant. This depends at least partially on the magnitude of mechanical stresses, the bone tissue vitality and the remodeling around the bone–implant interface.

The bone mineral density around a TKA decreases within several months after implantation. The loss in bone density reaches up to almost 23% within 1 year postoperatively. The reported values depend on the preoperative and post-surgical mechanical alignment, location of measurement and the method of assessment [7,8]. Postoperative loss of bone density is the result of the surgical procedure, peri-operative inflammation and bone remodeling associated with postoperative alterations in mechanical load. However, there is no evidence that an extensive periosteal soft tissue release around the medial part of the proximal tibia is associated with an increased risk of bone damage. Regardless of the particular cause of early bone mineral density loss, periprosthetic bone density generally normalizes in the majority of patients at the end of 3 years [9].

3. Mechanical theories underlying PPOL around TKA

Development of PPOL can be linked to the long-term influence of mechanical forces on both the joint replacement device and the bone bed. Under ideal conditions, the interface between the implant and bone bed should withstand repeated mechanical stresses associated with the activities of daily living. In this context, the major factors that would limit the mechanical stability of TKA and its longevity are the accuracy of the surgical reconstruction, the fixation of the implant to the bone bed, and factors influencing bone vitality and remodeling.

There is considerable evidence that relates abnormal mechanical stresses/strains to prosthesis/knee malalignment [10–12]. Abnormal forces associated with a limb/prosthetic malalignment can degrade the bone cement layer anchoring implants to the bone [13]. The integrity of the bone–cement interface is especially critical to implant survival [14–16]. In this context, deterioration of bone structure around the areas of cement fixation observed in some TKA postmortem specimens might be understood as a reaction to long-term overload of bone-implant interface [17]. How-

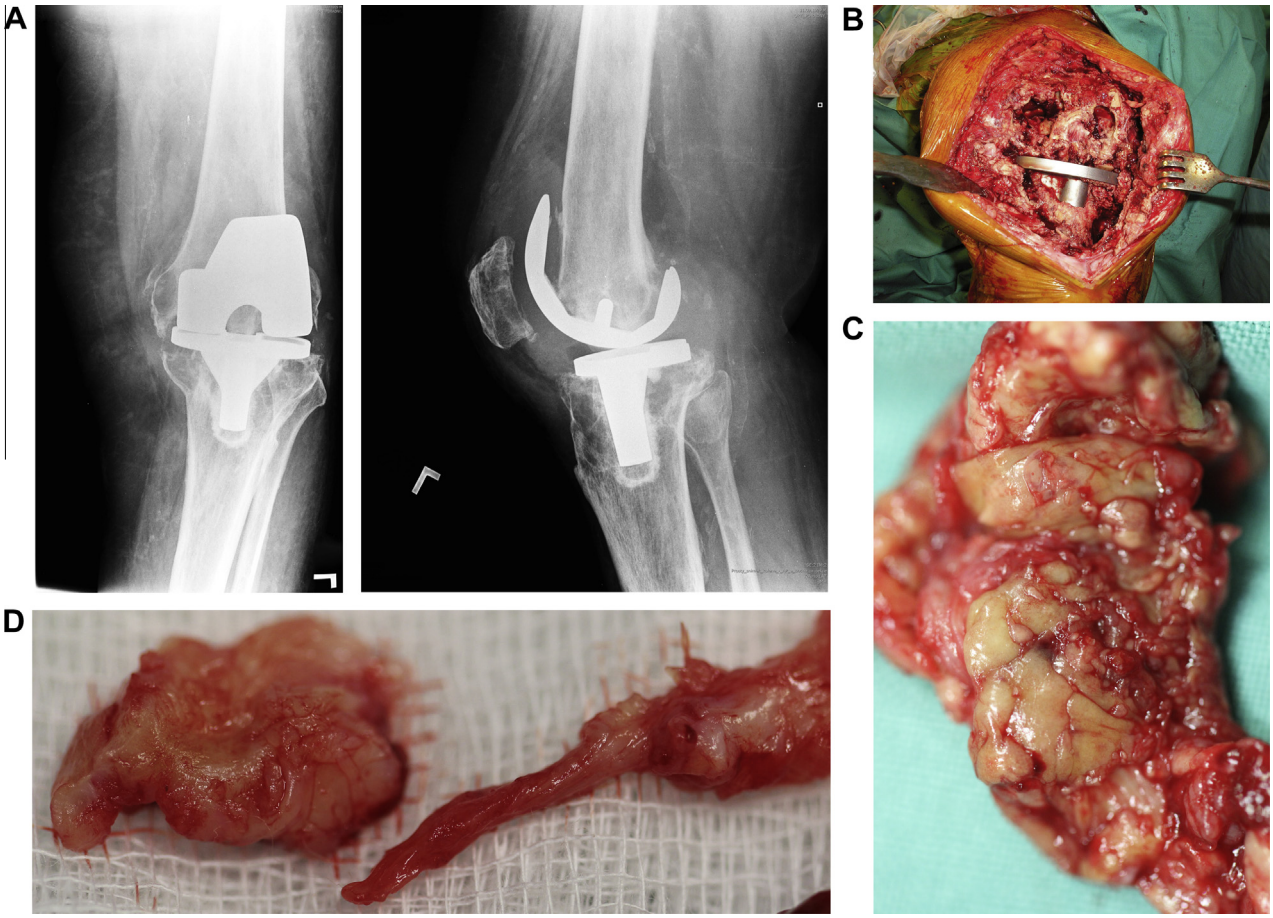

Fig. 1. (a) Anteroposterior and lateral X-ray of extensive osteolysis around the femoral and tibial component of TKA 11 years postoperatively; (b) intraoperative view on the size of bone defects after removal of the failed femoral component and debridement of the osteolytic cavity; (c) from the surgeon's viewpoint, the interface tissue membrane surrounding a failed TKA appears to be similar to the same tissues from (d) aseptically failed total hip arthroplasty.