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The implication of the osteolysis threshold and interfacial gaps on periprosthetic osteolysis in cementless total hip replacement

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ABSTRACT

Osteolysis around joint replacements may develop due to migration of wear particles from the joint space into gaps between the interface bone and the implant where they can accumulate in high concentrations to cause tissue damage. Osteolysis may appear in various postoperative times and morphological shapes which can be generalized into linear and focal. However, there are no clear explanations on the causes of such variations. Patients' degree of sensitivity to polyethylene particles (osteolysis thresholds), the local particle concentration and the access route provided by the interface gaps have been described as determining factors. To study their effects, a 2D computational fluid dynamics model of the hip joint capsule in communication with an interfacial gap and the surrounding bone was employed. Particles were presented using a discrete phase model (DPM). High capsular fluid pressure was considered as the driving force for particle migration. Simulations were run for different osteolysis thresholds ranging from 5×10^8 to 1×10^{12} particle number per gram of tissue and fibrous tissue generation in osteolytic lesion due to particles was simulated for the equivalent of ten postoperative years. In patients less sensitive to polyethylene particles (higher threshold), osteolysis may be linear and occur along an interfacial gap in less than 5% of the interfacial tissue. Focal osteolysis is more likely to develop in patients with higher sensitivity to polyethylene particles at distal regions to an interfacial gaps where up to 80% of the interfacial tissue may be replaced by fibrous tissue. In these patients, signs of osteolysis may also develop earlier (third postoperative year) than those with less sensitivity who may show very minor signs even after ten years. This study shows the importance of patient sensitivity to wear particles, the role of interfacial gaps in relation to morphology and the onset of osteolysis. Consequently, it may explain the clinically observed variation in osteolysis development.

1. Introduction

The relation between the presence of polyethylene wear particles and osteolysis around joint replacements is widely recognized (Schmalzried et al., 1992; Maloney et al., 1990; Willert et al., 1990). Once wear particles are released from bearing surfaces into the joint space, they may be carried to the accessible periprosthetic spaces and tissues by the joint fluid (Schmalzried and Callaghan, 1999). However, the exact mechanism of osteolysis development is not completely understood. While some patients exhibit linear osteolysis, others develop focal ones and some remain immune to osteolysis despite excessive wear generation in their prosthetic joint (Jasty et al., 1997). In addition, the onset of osteolysis can vary between one to seven years (Tanzer et al., 1992). Although many factors are likely to contribute to these variations in osteolysis development, patient's sensitivity to polyethylene, local particle concentration which relates to particle migration to the interfacial tissue by means of interface gaps could be determining factors.

It has been shown that, if particle concentration reaches a certain threshold in periprosthetic tissue, osteolysis may develop. Revell et al. (1997) and Kobayashi et al. (1997) showed that sites containing more than the critical value of approximately 1×10^{10} particles per gram of tissue had developed focal osteolysis. However, Elfick et al. (2003) reported a lower threshold of 1×10^9 particles per gram of tissue for osteolysis generation. Koseki et al. (2005) showed that the particle concentration in focal lesions (2.10×10^9 particles per gram of tissue) was significantly greater than the linear ones (2.91×10^8 particles per gram of tissue). These findings show that once the threshold is reached, the bone tissue is replaced with fibrous tissue with lower permeability which may encourage more particles to flow in that region. In addition, Ise

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et al. (2007) observed a correlation between the ratio of osteolysis in radiographic images and patient sensitivity to polyethylene particles. These findings suggest that the biological threshold for osteolysis generation may be patient specific and that osteolysis generation in the long term could be dependent on this threshold.

A number of studies have investigated particle migration to the periprosthetic tissue to some extent. Bobyn et al. (1981) observed that polyethylene debris (1×10^8 particles twice a week) injected in the joint migrated easily along the smooth surfaces of cylindrical implants implanted into the distal femur and proximal tibia of dogs. They observed that, during this time, particles penetrated the intratrabecular spaces up to approximately 2 mm. The findings of von Knoch et al. (2000) showed that the path of least resistance for distal migration is provided by the interfacial gaps and not through the porosity of the bone and fibrous tissues.

The above studies considered only the end-stage of osteolysis and do not describe how particle-induced osteolysis develops and expands during the lifespan of an implant. The current study aims to investigate how osteolysis expansion and development may be affected by an osteolysis threshold and an existing interfacial gap which may facilitate particle transportation. A computational fluid dynamics (CFD) model which simulates particle migration and entrapment in periprosthetic gaps and bone tissue as well as fibrous tissue generation was employed.

2. Method

The 2D geometry of the model was based on a realistic 3D model of a femur reconstructed from a computerized tomography (CT) scan implanted with a cementless femoral stem (Summit stem, DePuy, Warsaw USA) and the surrounding joint capsule. The 2D geometry was generated by cutting through the symmetric axis of the implant on the coronal plane (c.f. Fig. 1a). The boolean operation for implantation and 2D geometry generation were carried out in Solidworks (Dassault Systemes, Velizy, France). The model geometry was meshed in GAMBIT (Ansys Inc.) using structured (gap region) and unstructured (capsule and bone regions) quadrilateral cell volumes and then imported into the commercial CFD software FLUENT (version 12.00, Ansys Inc.) for analysis.

The meshed geometry of the model is shown in Fig. 2. The geometry was meshed with approximately 10 k, 21 k, 42 k, and 140 k cells for mesh convergence studies. For each of these meshes, different combinations of the core and boundary layer meshes (which included varying the height of the first layer and the total number of layers) were generated. Mesh convergence studies demonstrated that the 42 k mesh with an appropriately sized boundary layer and core meshes (c.f. Fig. 2) showed less than 5% variation in fluid velocity in regions of high flow gradient, relative to the finest mesh. Therefore, this mesh was deemed appropriate for this study.

The capsule and gap regions were modelled as free fluid continua, the bone was represented as a porous medium and the implant wall was described as a rigid wall. Simulations were steady state and did not incorporate the fluid pumping mechanism caused by micromotion between the implant and bone. High capsular fluid pressure was considered to be the driving force for particle transportation (Alidousti et al., 2014; Schmalzried et al., 1997; Robertsson et al., 1997). Fluid pressure inside the capsule was generated by a pressure inlet boundary defined on the soft tissue wall of the capsule. High capsular pressure was considered to be 60 kPa (Hendrix et al., 1983). The pressure over the porous bone region was assumed to be zero with respect to the capsule (i.e. 0 kPa). This was achieved by defining a pressure outlet on the endosteal and periosteal surfaces of the bone (c.f. Fig. 1b). This boundary condition may represent the fluid drainage through many embedded venous sinuses in proximal femur which leave the bone through a great number of openings on the external surfaces of the femur in medial, lateral and superior regions (Churchill et al., 1992). In addition, one source can extend to supply other regions when there is impairment to another. This is indeed the case for an implanted femur in which the femoral cavity is filled with the implant stem and the blood supply is provided from the external periosteal surface. In fact, it has been shown that two thirds of the total blood supply and drainage in the bone surrounding the implant is carried out by periosteal, epiphyseal and metaphyseal arteries which penetrate the bone through external periosteum layer (Bridgeman and Brookes, 1996). Rhinelander (1972) has also shown that the greater portion of blood in diaphysis is drained from the periosteal surface rather than endosteal. In the cortical bone, it has been shown that the blood supply entering the Haversian system may also exit the system through the periosteal blood supply (Cooper et al., 1966; Montgomery et al., 1988). These observations are further supported by the fact that intravascular pressure drops from about 8 kPa to around 2 kPa from endosteum to periosteum surfaces (Brookes, 1971) which indicates the fluid drainage from the

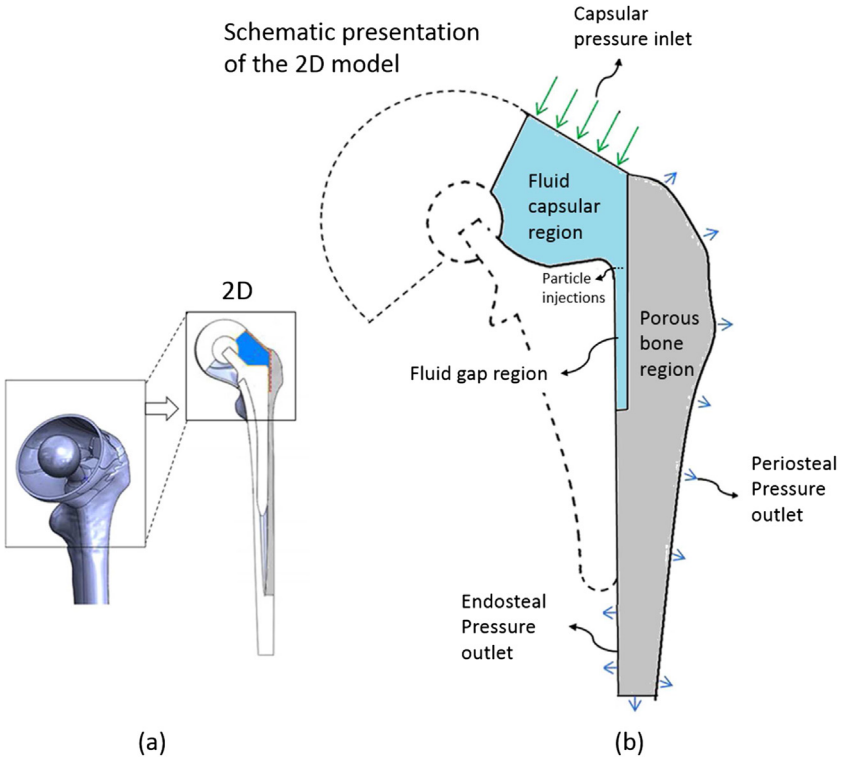

Fig. 1. (a) Realistic 3D model of an implanted femur and the surrounding joint capsule (left) and the 2D cross-sectional geometry generated based on coronal cut (right). (b) Schematic of the system consisting of the fluid capsule and gap regions in communication with the surrounding porous bone region. The size of the gap is exaggerated for demonstration purposes. Capsular pressure inlet (green) and periosteal and endosteal pressure outlets (blue) are shown. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

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