Case report

Bead debonding from a modern cementless total hip femoral component with concomitant taper corrosion

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A B S T R A C T

We report a case of bead shedding from a cylindrical extensively porous-coated cementless femoral component with concomitant taper corrosion at the modular head-neck junction of a metal-on-polyethylene total hip prosthesis. The patient presented with chronic thigh pain 4 years after primary total hip arthroplasty, and radiographs revealed significant osteolysis and metallic debris around the femoral stem. Intraoperatorically, the patient had a grossly loose femoral component with debonding of sintered beads from the femoral stem, as well as evidence of taper corrosion. We identify a failure of a modern beaded femoral component in conjunction with taper corrosion.

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Introduction

Cementless fixation of components can reliably be achieved using implant surfaces coated with sintered cobalt-chromium beads [1]. The beads provide a 3-dimensional porous surface into which bony ingrowth can occur [2]. This surface coating is subject to a rare but potentially catastrophic failure mode known as bead shedding, in which the sintered beads are debonded from the implant [3,4]. This failure mechanism has been reported frequently in acetabular components in total hip arthroplasty, but is rarely described for femoral components, particularly with respect to contemporary implants [5,6].

Metal ion release in total joint arthroplasty can occur secondary to taper corrosion at modular interfaces, metal-on-metal bearing surfaces, or corrosion related to debonded beads from cementless ingrowth surfaces [3,7-9]. Metal ion production has been shown to lead to a variety of adverse local tissue reactions [8,10,11]. The range of responses to metallosis is variable, but includes soft tissue destruction, osteolysis, aseptic loosening, and implant failure [12].

In this report, we describe a case of bead shedding from a modern cylindrical extensively porous-coated femoral stem that was associated with head-neck taper corrosion in a modular hip prosthesis. The patient was informed and provided consent that information regarding the case would be submitted for publication.

Case history

A 59-year-old male with a history of hypertension and prior right total hip arthroplasty (2008) underwent an uncomplicated left total hip arthroplasty for osteoarthritis via a posterior approach in 2012 at an outside institution (Figs. 1 and 2). The components used included Zimmer Trilogy uncemented acetabular cup, Zimmer Beaded FullCoat uncemented femoral stem, cobalt-chrome head, and highly cross-linked polyethylene liner (Zimmer Inc., Warsaw, IN). These were the same components that were used in the contralateral hip 4 years before.

Several months after the operation, the patient developed activity and position-related thigh pain that limited ambulation. This pain did not subside and the patient was eventually seen in clinic 4 years after the index procedure in 2016. Clinical evaluation revealed a normal gait pattern with a decreased cadence. Hip range of motion yielded flexion past 90°, full extension, internal rotation to 20°, and external rotation to 30°. Body mass index was 46 kg/m².

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Figure 1. Preoperative (a) anteroposterior (AP) pelvis and (b) AP hip radiographs demonstrate severe degenerative changes at the left hip.

Figure 2. (a) AP and (b) frog lateral hip radiographs taken 3 months postoperatively demonstrate a left cementless primary total hip arthroplasty. There is irregularity at the surface of the femoral component in zones 2 and 3 suggestive of possible bead dislocation.

Figure 3. (a) AP pelvis, (b) AP hip, and (c) frog lateral hip radiographs taken at the time of patient presentation, 4 years after total hip arthroplasty, showing multiple areas of osteolysis around the proximal and midfemoral stem and metallic debris at the most distal areas of osteolysis.
Review of prior radiographs from 3 months after surgery (Fig. 2) demonstrated irregularity at the surface of the femoral component in zones 2 and 3 suggestive of possible bead dissociation. Radiographs taken at the time of presentation in 2016 demonstrated multiple areas of osteolysis around the proximal and midfemoral stem and metallic debris collecting at the most distal areas of osteolysis. The acetabular component appeared well-fixed with decreased anteversion (Fig. 3). Inflammatory markers included a C-reactive protein level of 3.29 mg/L (normal 0-10 mg/L) and erythrocyte sedimentation rate of 9 mm/h (normal 0-20 mm/h). Serum metal ion levels included a cobalt level of 6.6 parts per billion (ppb; normal <1.0 ppb) and chromium level of 2.0 ppb (normal <0.3 ppb). A hip aspiration resulted in a white blood cell count of 163 with 10% polymorphonuclear leukocytes and no growth on culture. Magnetic resonance imaging with metal artifact reduction sequencing demonstrated a small hip effusion and trace fluid surrounding the femoral component in zones 1 and 7, but no definitive evidence of capsular thickness >4 mm and synovitis; there was no fluid collection noted surrounding the contralateral hip.

Intraoperatively, a pseudocapsule was identified and debrided, and pathology examination of this tissue revealed fibrous tissue with chronic, but not acute, inflammation. The abductors were intact. Tissue and fluid samples sent for culture demonstrated no growth. There was visible corrosion at the head-neck taper junction with debris on both the head and femoral components (Fig. 5). The femoral component was grossly loose and easily removed. The mid to distal portions of the stem had significant gross debonding of beads, especially along the medial face (Fig. 6). Debridement of the femoral canal resulted in removal of multiple loose clumps of beads (Fig. 7). The acetabular component was well fixed and there was no significant polyethylene wear. However, the cup was in slight retroversion and was removed. The patient was revised to a revision tapered femoral stem, a ceramic femoral head, and a revision cementless hemispherical acetabular component with a cross-linked polyethylene liner. At 3-month follow up, the patient’s pain had resolved and the implant was functioning appropriately. Radiographs showed well-aligned and stable implants (Fig. 8).

The case was reported to the implant manufacturer. No retrieval analysis was performed.

Discussion

Bead shedding from arthroplasty implants has been reported frequently in acetabular components after total hip arthroplasty [5,6,13-15] and femoral and tibial components after total knee arthroplasty [4,15,16]. In contrast, there is very limited recent information for debonding in femoral components after total hip arthroplasty, with a literature search yielding no reports of femoral component bead debonding in the last 20 years [3,17,18]. Here, we present a case of bead debonding from a modern cementless femoral component affecting a substantial portion of the ingrowth surface.

Several etiologies of bead shedding have been proposed. These include bead loss during component impaction, bead shearing related to micromotion between a loose implant and bone, or bead debonding because of corrosion between the implant surface and the bead [5]. Risk factors for bead shedding include bead-implant fusion characteristics and component loosening [4,15,16,18]. In a study of 1600 bead-coated implants from an implant retrieval database, Swarts et al found that over 50% of acetabular and total knee components with bead loss were clinically loose [15].

Our patient was found to have taper corrosion at the modular head-neck junction. Modular hip implants provide intraoperative flexibility to optimize hip biomechanics and joint stability.
However, despite a proven track-record for modern modular implants, there is increasing concern for failure mechanisms associated with modular junctions [19]. Adverse local tissue reactions, similar to those seen with metal-on-metal total hip arthroplasty, have been evident due to corrosion at the head-neck taper in modular femoral components [7-9]. Risk factors have been described for development of head-neck taper corrosion including mismatch of taper geometry, trunnion size, femoral head size, neck angle, use of dissimilar metals, obesity, long neck sizes, and inappropriate impaction technique [11,19-22], several of which (obesity, long neck) were present for this case patient. The local response to corrosion products is highly variable with regard to both the patients’ symptoms and the extent of tissue destruction [12]. In addition to soft tissue damage, adverse local tissue reactions can cause osteolysis and aseptic loosening [7,10,12,23-25].

It is our hypothesis that osteolysis occurred secondary to corrosion products in this case patient. One possibility is that initial aseptic loosening occurred secondary to head-neck taper corrosion. In this scenario, the proximal osteolysis would then have concentrated stresses on the distal aspect of the stem, causing it to receive the full burden of weight-bearing. This increased force may have exceeded the fatigue tolerance at the bead-implant interface resulting in debonding of the distal beads and gross implant loosening. This theory is further supported by the patient’s obesity, a risk factor for trunnionosis, as well as the particular use of the initial femoral stem, which has a higher than expected rate of corrosion [26]. An alternate explanation is that nonintegration of

![Figure 6](image1.png)

**Figure 6.** (a) Explanted femoral component with visible bead loss at the distal portion of the stem. (b) Close-up of the distal medial phase of the explanted femoral stem with bead loss.

![Figure 7](image2.png)

**Figure 7.** Photograph of removed contents of the femoral canal after component explant showing debonded metallic beads.

![Figure 8](image3.png)

**Figure 8.** (a) AP pelvis, (b) AP hip, and (c) frog lateral hip postoperative radiographs demonstrate a revision tapered femoral stem, a ceramic femoral head, and a revision cementless hemispherical acetabular component.
the femoral component after the index procedure led to ongoing micromotion between implant and bone causing bead shedding with subsequent corrosion of the debonded beads. In this scenario, the head-neck taper corrosion would be an incidental finding. An additional possibility is that bead debonding during initial implant impaction, as suggested by the femoral component surface irregularity noted on the initial postoperative radiographs (Fig. 2), was the precipitating factor that led to the formation of corrosion products. In this scenario as well, the head-neck taper corrosion would be an unrelated finding. Lastly, it is possible that errors in manufacturing or metallurgy could explain the failure of both the trunnion and the bead-implant interface. Definitive conclusions regarding a causal link between the taper corrosion and bead debonding is beyond the scope of this report.

Summary

To the best of our knowledge, this is the first case study to show femoral component bead shedding in a modern fully porous-coated implant after primary total hip arthroplasty. Although partially-coated femoral components are the most commonly used in modern practice, this report nonetheless emphasizes the need for an improved understanding of both corrosion phenomenon as well as bead debonding in modular hip arthroplasty systems.

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