# Metallosis During Partial Component Hip Revision Arthroplasty

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*A partial component revision, where the implant is not entirely removed is a tempting option where only some components are damaged and others are well fixed. This is especially important for young patients where prolonging the survival of the joint replacement is paramount since advantages of partial revision in hip replacement are well-documented. Data in the literature are scarces and disparated on metallosis during partial component hip revision arthroplasty being limited mostly on case series. We therefore consider useful to present a study which aims to discuss this topic based on current knowledge and personal experience. We present a case of metallosis occurring after revision for infection in a 52 years old active male with hip arthroplasty. Fibrotic material from intraoperative debridement was given to histopathological analysis. The results offered a picture of unspecific reaction to metallic debris indicating asymptomatic metallosis. The patient had 4.1 mcg/L of cobalt and 5.9 mcg/L of serum chromium immediately after surgery. These measures were repeated at last follow- up (2 years) and were considerably lower, to 1.5 mcg/L of cobalt and 2.3 mcg/L of serum chromium. The real incidence of metallosis is unknown, although it seems to be more frequent in hip than in knee arthroplasty. It is caused by the infiltration and accumulation of metallic debris into the peri-prosthetic structures, deriving from friction between metallic prosthetic components.*

*Keywords: metallosis, partial component, hip, revision arthroplasty*

Hip arthroplasty is the most common joint replacement performed worldwide. For the last few years, it is associated with survival of 99% at 10 years. However, younger age and high volume increase the need for revision surgery. In our country, initial enthusiasm of primary hip arthroplasties is now followed by increased awareness towards revisions.

A partial component revision, where the implant is not entirely removed is a tempting option where only some components are damaged and others are well fixed. This is especially important for young patients where prolonging the survival of the joint replacement is paramount since advantages of partial revision in hip replacement are well-documented [1]. Isolated acetabular polyethylene exchange is advocated as an advantage of metal-backed cups, since the acetabular bone stock can be spared and operative time is shorter, even though some authors recommend caution in cases with multiple surgeries due to increased dislocation rates [2].

Whenever high friction occurs between metallic surfaces of implant components debris is generated from fretting and accumulates in the surrounding tissues, a condition known as metallosis – this is usually defined as aseptic fibrosis, local necrosis, or loosening of a device secondary to metallic corrosion and release of wear debris. It is an occasional but characteristic clinical finding in patients who have a metal-on-metal design of total hip replacement, or when metal surfaces contact after a failure or erosion of the polyethylene component [3]. It rarely leads to metal poisoning and is most often an incidental finding. However it is an expression of implant malfunction and can increase risks after revision. A study demonstrates that metallosis frequently causes osteolysis and that complete elimination of it is not a prerequisite for the success of revision total hip arthroplasty [4].

Data in the literature is scarces and disparate on metallosis during partial component hip revision arthroplasty being limited mostly on case series. We therefore consider useful to present a study which aims to discuss this topic based on current knowledge and personal experience.

## Experimental part

*Materials and Method*

We present a case of metallosis occurring after revision for infection in a 52 years old active male with hip arthroplasty. The patient had posttraumatic osteoarthritis which led to primary total hip replacement early in life. Initial revision of the index arthroplasty became infected and was replaced with antibiotic impregnated cement spacer (fig. 1). During implant removal the acetabular cup, although slightly malpositioned with excessive anteversion was very well integrated and the decision was made to be left in place, based on invasive bone loss and increased risk of infection propagation to the pelvis [5]. After local infection was cured a new femoral stem (Revitan: Ti6Al7Nb - stem, CoCrMo – morse taper and head and Ti6Al7V – acetabular cup), head and polyethylene liner were implanted. Cam impingement of the femoral neck to the acetabular cup and liner led to early posterior dislocation and direct contact between the metallic femoral head and the posterior wall of the metallic acetabular cup (fig. 2). Abnormal surface abrasion led to metallic debris. Intraoperatively extensive debridement of the impregnated periarticular tissue was performed and the offset was increased by replacing the head (fig. 3). Delayed diagnosis led to incidental metallosis (fig. 4) during revision surgery for head exchange. Careful examination of the cup and stem revealed good stability. Examination of the polyethylene liner also did not reveal signs of wear.

Fibrotic material from intraoperative debridement was given to histopathological analysis. The results offered a picture of unspecific reaction to metallic debris indicating asymptomatic metallosis (fig 5-8) [6].

Similar results were described in the literature such as the works of Natu et al. who presented a study on 123

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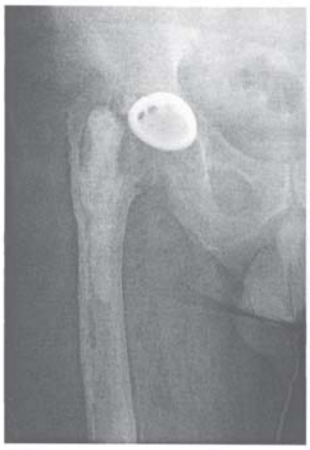

Fig. 1. preoperative X-ray (AP) with the acetabular cup and cement spacer

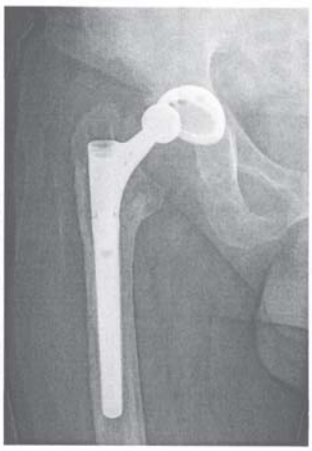

Fig. 2. AP X-ray with the revision stem in place and posterior dislocation

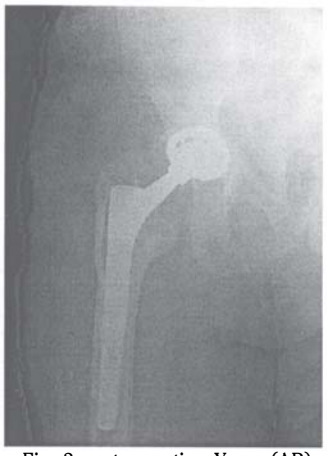

Fig. 3.postoperative X-ray (AP) with the new head and increased offset

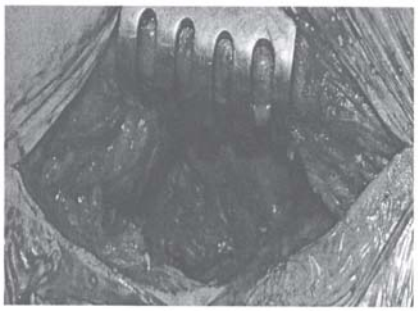

Fig. 4. intraoperative aspect with dark grey staining of fibrotic periarticular tissue suggestive for metalosis

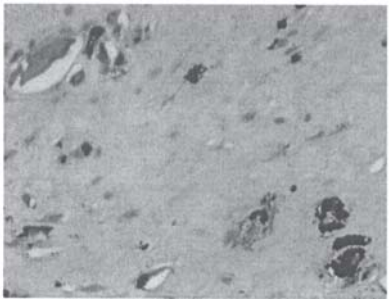

Fig. 5. Connective tissue with metallic deposits and focal microcalcifications (hematoxylin – eozin X400)

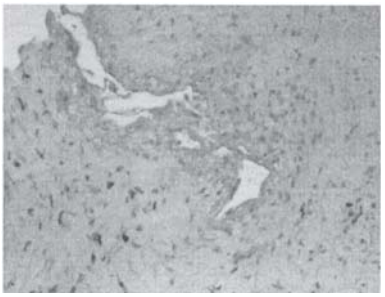

Fig. 6. Connective tissue with metallic deposits and discrete fibrin deposits (hematoxylin – eozin X100)

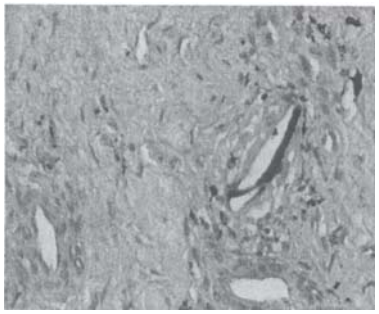

Fig. 7. Connective tissue with rare neoformation vessels, chronic inflammation cells (lymphocytes) and metallic inclusions one of which large, needle shaped (hematoxylin – eozin X200)

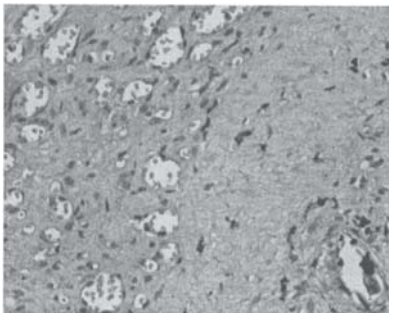

Fig. 8. Granulation tissue with frequent neoformation vessels in the vicinity of metallic deposits and rare chronic inflamation cells (lymphocytes) (hematoxylin – eozin X200)

patient samples of periprosthetic soft tissues biopsied at time of revision from failed metal on metal hip arthroplasties. The inflammatory cell response was categorized into perivascular lymphocytic cuffing, lymphoid aggregate formation with or without germinal centers, metallosis characterised by sheets of macrophages with intracytoplasmic metallic debris and well-defined granulomas [7, 8].

**Results and discussions**

Studies reviewed from the literature concerning ions moving from the prosthetic joint into general circulation due to abrasive wear of metallic surfaces consider Co (cobalt) and Chromium to be responsible for clinical manifestations of toxicity encountered in moderate to severe cases of metallosis and also the best predictors for sickness and implant revision [9, 10]. Therefore, given the composition of the friction surfaces (CoCrMo – for the head and Ti6Al7V – for the cup) we tested for serum levels of these two metals [11].

Although on macroscopic evaluation of the head – cup contact area the acetabular component appeared more abraded, the retreaved head was 1.9 g lighter. The patient had 4.1 mcg/L of cobalt and 5.9 mcg/L of serum chromium immediately after surgery. These measures were repeated at last follow- up (2years) and were considerably lower, to 1.5 mcg/L of cobalt and 2.3 mcg/L of serum chromium. We did not found clinical or radiological signs of metallosis on last follow up visit [12]. The implant has good functional outcome.

A serum cobalt level of > 7 mg/L indicates possible periprosthetic metallosis. A normal serum cobalt level is averaged at 0.19 mg/L and 95%. A value of 1 mg/L indicates

excessive cobalt exposure, and levels of >5 mg/L are considered toxic.

## Conclusions

Campbell et al. found that unlike component wear or serum ion levels, which can be measured with a known degree of accuracy; there are currently no definitive blood tests or histopathology criteria to diagnose metal hypersensitivity. Nevertheless, the authors devised a working postulate to diagnose hypersensitivity: early onset of pain, the absence of other reasons for pain (such as loosening, impingement, infection, or high wear), and the resolution of symptoms after the removal of the cobalt-chromium components. Other clinical reports have noted similar features in patients suspected to have a metal hypersensitivity reaction [13].

Hart et al. found that the 7 ppb cut-off level for the maximum of cobalt or chromium had a specificity of 89% and sensitivity 52% for detecting a pre-operative unexplained failed metal on metal hip replacement. The optimal cut-off level for the maximum of cobalt or chromium was 4.97 ppb and had sensitivity 63% and specificity 86%. Blood metal ions had good discriminate ability to separate failed from well-functioning hip replacements [9].

Morse tapers are frequently used in total hip replacement to achieve precise adjustment of lengths and femoral offset. Corrosion after malpositioning over the Morse taper in hip arthroplasty has been reported with requirement for revision due to mechanical wear [14].

Insert wear, fracture, or dislodgment in modular components may lead to articulation of the prosthetic head with the metallic shell and subsequent metallosis. When metallic debris-induced bone loss is recognized early, surgical intervention may limit its progression [15, 16].

The real incidence of metallosis is unknown, although it seems to be more frequent in hip than in knee arthroplasty. It is caused by the infiltration and accumulation of metallic debris into the peri-prosthetic structures, deriving from friction between metallic prosthetic components. The metallic debris induces a massive release of cytokines from inflammatory cells, making a revision necessary whenever osteolysis and loosening of the prosthesis occur [17, 18].

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