CASE REPORT

Chromium, cobalt and titanium metallosis involving a Nottingham shoulder replacement

W. S. Khan, M. Agarwal, A. A. Malik, A. G. Cox, J. Denton, E. M. Holt

From the University of Manchester, Manchester, England

Metallosis after shoulder replacement has not previously been described in the literature. We report a patient who developed extensive metallosis after implantation of an uncemented Nottingham shoulder replacement. He underwent a revision procedure.

Examination of the retrieved prosthesis showed that the titanium porous coating was separating from the humeral stem and becoming embedded in the ultra-high-molecular-weight polyethylene glenoid component, resulting in abrasive wear of the humeral component. There was metallosis despite exchange of the modular humeral head. Both components had to be exchanged to resolve the problem.

Metallosis is the accumulation of metal wear debris in the peri-prosthetic tissues which results in a cellular reaction characterised by a macrophagic response with formation of giant cells and fibrosis. Few cases have been reported in the literature and mostly after hip replacement. We describe a patient who developed extensive metallosis after implantation of an uncemented shoulder replacement. Extensive metallosis after shoulder replacement has not previously been described.

Case report

A 55-year-old right-handed man underwent uncemented Nottingham shoulder replacement (Biomet, Bridgend, United Kingdom) for destructive rheumatoid arthritis. The humeral stem and head were made from cast cobalt-chromium-molybdenum alloy. The proximal part of the humeral stem had an argon plasma-sprayed titanium coating to encourage osteointegration. The glenoid component was made of ultra-high-molecular-weight polyethylene (UHMWPE) and was attached by a snap-fit mechanism to a glenoid tray secured to the bone by two screws. The glenoid tray and screws were made from titanium alloy.

He complained of persistent pain and underwent exploration 28 months after implantation. Radiographs taken one week before the exploration (Fig. 1) showed no radiolucent lines, osteolysis or vertical or horizontal migration of the components. They did, however, show that one of the titanium screws used to attach the glenoid tray was broken. The bubble sign, the outlining of the joint cavity by metal-

Fig. 1a

a) Anteroposterior and b) lateral view radiographs of the right shoulder one week before the initial exploration procedure with the uncemented Nottingham shoulder replacement in situ.

Fig. 1b


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lic debris, was not noted on the radiographs. At operation blackening was found involving intra-articular and extra-
articular tissues and the subchondral bone. There was no evidence of osteolysis involving the humerus or glenoid,
loosening of the components or tear of the rotator cuff. The findings did not suggest infection and microbiological sam-
pies taken at the time of surgery confirmed this. Other than obtaining material for biopsy, no further surgery was
undertaken.

Histology showed a profound macrophage response to the debris with some giant-cell formation (Fig. 2). The macrophages and giant cells contained numerous black intracellular particles mostly between 1 µm and 5 µm in size. Spectrometry using a single-line Microprobe Laser Ablation-Inductively Coupled Plasma-Mass Spectrometer system indicated that the intracellular particles contained the elements chromium, cobalt and molybdenum. These three elements were in the same relative proportions as those in the prosthetic humeral head. Occasional large extracellular particles 200 µm in size were identified as titani-
um with an identical elemental signature as the osteointe-
gration layer on the humeral component. No UHMWPE particles were identified. Urine analysis 30 months after the initial procedure showed elevated levels of the essential trace elements cobalt and chromium and of the non-essential trace elements titanium and aluminium (Table I).

In view of the continuing pain and the histological diag-
nosis of metallosis he underwent further debridement and exchange of the modular 22 mm humeral head to one of 20 mm, 30 months after the initial operation. Once again there was no evidence of osteolysis or loosening intra-
operatively. Macroscopic examination of the retrieved humeral head showed numerous radial scratches on the head consistent with abrasive wear (Fig. 3), raising concerns regarding ongoing metallosis and the potential of cobalt and chromium toxicity. The pain continued and it was decided to revise the implant to a cemented Bio-
modular total shoulder replacement (Biomet UK) 38 months after the original operation. Although macroscopic analysis of the titanium porous coating of the removed stem indicated some signs of osteo-integration, it also showed that the coating had started to delaminate and disintegrate peripherally (Fig. 4). Plasma-mass spectrometry analysis of the particles embedded in the glenoid component had the same elemental composition as those in the coating layer of the humeral component and those in the larger peri-prosthetic particles. Three years after the revision, he remains free from pain.

**Discussion**

We are not aware of previous reports of metallosis follow-
ing shoulder replacement. In our case it seems likely that

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**Table I.** Urine analysis was performed 30 months after the initial procedure to identify the concentration (µl) of metals present in alloys used in the components. Raised levels are highlighted in bold.

<table>
<thead>
<tr>
<th>Trace element</th>
<th>Early morning</th>
<th>Mid morning</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobalt</td>
<td>10.0</td>
<td>66.0</td>
<td>&lt; 2.0</td>
</tr>
<tr>
<td>Chromium</td>
<td>&lt; 20.0</td>
<td>20.0</td>
<td>&lt; 20.0</td>
</tr>
<tr>
<td>Molybdenum</td>
<td>26.0</td>
<td>100.0</td>
<td>&lt; 28.0</td>
</tr>
<tr>
<td>Titanium</td>
<td>190.0</td>
<td>210.0</td>
<td>&lt; 160.0</td>
</tr>
<tr>
<td>Aluminium</td>
<td>&lt; 10.0</td>
<td>46.0</td>
<td>&lt; 10.0</td>
</tr>
<tr>
<td>Vanadium</td>
<td>1.0</td>
<td>3.6</td>
<td>&lt; 2.0</td>
</tr>
</tbody>
</table>

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![Fig. 2a](image1)  
![Fig. 2b](image2)
the cobalt and chromium particles were from the articulating surface of the humeral head, and detached titanium particles became embedded in the polyethylene surface producing abrasive wear. This would explain why the metallosis continued despite an exchange of the humeral head.

After hip replacement, metallosis has been reported as being a cause of osteolysis, but the latter was not seen in our case. It has been suggested that osteolysis may be due to the synergistic interaction between metal and polyethylene particles causing a profound activation of histiocytes. In our case, however, histological analysis revealed only metal particles composed of chromium, cobalt, and titanium. Although metallosis can mask the radiological features of osteolysis, there was no radiological or intra-operative evidence to support osteolysis or loosening in our patient.

Titanium particles were identified in the surface of the polyethylene. Since titanium was not used in the bearing surface, it seems probable that the titanium porous coating had detached from the humeral stem. Alternatively, the titanium debris may have originated from the broken screw, but since the screw was covered by the glenoid tray, this seems less likely. Cobalt-chromium alloy is almost twice as hard as titanium, but the embedded particles seem to have been sufficiently hard to produce attrition of the cobalt-chromium head as demonstrated by the radial scratches on the humeral head. Previous reports of titanium-induced metallosis have been reported but only in cases in which the softer metal had been used in the bearing surface.

The possible long-term effects of metal wear debris in orthopaedic patients has recently been reviewed. Cobalt toxicity is known to cause thyroid dysfunction and cardiomyopathy, and is carcinogenic. Chromium and vanadium toxicity can cause cardiac and renal dysfunction and psychosis. Titanium toxicity can induce pulmonary disease and platelet dysfunction while aluminium produces bone-marrow suppression, renal failure and neurological dysfunction. These toxicities result from extremely high circulating concentrations of the elements which are unlikely to occur from degradation of an implant. To date, there is no study which has shown a cause-and-effect relationship between implant devices and toxic levels of metal release. Likewise, no epidemiological evidence is available to suggest that the risk of carcinogenesis is anything more than theoretical.

Our report supports previously documented evidence that radiographs may appear to be normal and cannot always reliably demonstrate the presence of metallosis.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References


