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Case Report

Titanium and Cobalt Neurotoxicity due to Catastrophic Failure of a Metal/Polyethylene Total Hip Arthroplasty (THA)-Case Report and Literature Review

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Abstract

Cobalt metallosis from failed total hip arthroplasty (THA) has been implicated in brain toxicity with, sometimes, severe neurological symptoms. We present a case of a destroyed metal on polyethylene THA, in a male patient that resulted in extreme metallosis from Titanium and Cobalt within 5 years and presented with severe neurological impairment. A literature review revealed that little is known regarding titanium toxicity from orthopaedic implants. We discuss the case and the literature aiming to raise awareness for this rare and potentially serious complication.

Keywords: Failed THA; Metallosis; Neurotoxicity; Titanium; Cobalt; Metal on Polyethylene

Introduction

Cobalt metallosis from wear of articulating surfaces is a well described complication of total hip arthroplasty (THA) that can have severe systemic effects, including brain toxicity [1,2]. However, titanium metallosis, has received very little attention in association to THA and there is a scarcity in existing literature about its toxicity, alone or in combination with cobalt. We present a case of sudden and severe neurological impairment in a patient with significant titanium and cobalt metallosis due to a destroyed polyethylene liner that resulted in catastrophic wear of the titanium shell and the cobalt-chrome head of a THA within 5 years.

Case Presentation

A mesomorphic (1.95m tall, 115 Kg), 80 year-old man, who had a primary, cementless, metal on polyethylene, hip arthroplasty of his right hip in February 2013 due to osteoarthritis, was admitted in a private hospital with sudden onset neurological symptoms in February 2018. Initial neurological evaluation revealed that for the past 6 months he had exhibited progressive motor regression and neurocognitive decline with no history of fever, head trauma or new medication. At the time of admission he was delirious, confused, agitated and uncooperative. He had visual hallucinations, impaired communication and depression. On physical examination he was quadra-kinetic, although with decreased muscle power, and hyper-reflective, with normal plantar reflexes. MRI of the brain revealed extensive multi-infarct leukoencephalopathy. He was started on trazodone 100 mg/24h and risperidone 2,5 mg/24h and showed mild improvement.

Alongside the neurological symptoms, the patient complained of right hip pain, an audible clunk and inability to walk. On examination, the right hip felt unstable, all movements were painful and a metallic clunk was obvious. Radiographs (Figure 1A) revealed a quite uncommon problem. The femoral head had penetrated the acetabular shell superolaterally and was in contact with the acetabular bone. A shadow medially suggested that the polyethylene insert had been displaced and was not contributing to the articulation. Further investigation, by questioning the patient's family, revealed a chronic problem that had started just 2.5 years post THA. At that time the aforementioned symptoms appeared and had been increasingly debilitating since then. In August 2016, the patient had visited his surgeon with radiographs (Figure 1B), because his hip symptoms started affecting his every-day activities. Although an eccentric position of the femoral head is clearly seen on those X-rays, the operating surgeon dismissed him.

Figure 1A: Radiograph showing cup penetration. The arrow points to the shadow of a polyethylene fragment.

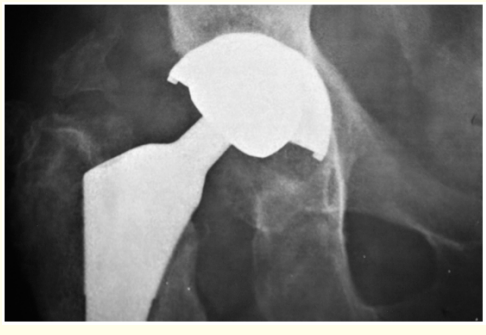

Figure 1B: Previous radiograph with eccentric head position, indicative of significant polyethylene wear.

Cobalt and Titanium levels were extremely high in blood, urine and cerebrospinal fluid samples (Table 1).

	Blood	Urine	CSF
Cobalt **µg**/L	16.2	34.0	1.8
Titanium **µg**/L	179	610	19

Table 1

With the suspicion of heavy metal brain toxicity and a completely worn-out THR, revision surgery was indicated and was performed under epidural anesthesia, through a lateral (omega) approach. Immediately after the deep fascia incision abundant fluid discharge of black discolouration and black-stained soft tissues were evident. Following capsulotomy, more metal-stained fluid had to be drained to allow joint visualization. Inferomedially, a fractured part of the polyethylene liner, showing significant wear, was found dislodged. With the hip dislocated, affected membranes had to be removed from the joint to allow access to the cup. The 32 mm femoral head exhibited significant abrasion and was removed revealing a smaller fragment of the polyethylene liner, also extremely worn out, inside a titanium cup that had been completely penetrated superomedially and was loose (Figure 2A and 2B). Cup removal revealed a thick, black membrane covering the entire acetabulum, and this was thoroughly removed to show some bone loss at the acetabular floor, but quite good acetabular rim. Careful reaming to clean bone was performed and a new, cementless, tantalum cup was inserted that achieved good fixation. The femoral stem was found stable, with only a 1 - 2 cm metal-stained membrane around its neck that was removed leaving healthy bone. Copious pulsatile lavage was used to remove as much of the remaining metallic and, obviously, polyethylene debris as possible. A thick polyethylene liner with an elevated rim superolaterally and a 28 mm head were inserted, providing a stable reduction. Layers were closed over suction drainage, which continued discharging black fluid for 4 days postoperatively, before it could be removed. Post two weeks, a 3cm long separation of the surgical incision, draining more stained fluid, was noted. Infection was excluded and a new surgical debridement with more lavage was performed. Following this the wound healed uneventfully and physical therapy was initiated aiming at getting the patient mobile. Progression was very slow due to his neurological status that started a steady but slow improvement following surgery. The first obvious improvement included the patient's mood and his cognitive and communication abilities. Motor improvement was greatly delayed by his decreased mental status, prolonged immobilization, muscle weakness and his build.

Figure 2: A: Intraoperative picture. Severe staining of tissues and polyethylene fragment can be seen. B: The extracted implants revealing the extent of the damage.

Eighteen months postoperatively the patient had a satisfactory mental capacity, but did not as yet gain independent mobility. He presently uses a walker, with some assistance, for short indoor walks and can sit in a chair for a limited time. Unfortunately, full recovery cannot be expected.

Discussion and Conclusion

Metallosis following THR has been recognised as a common problem with metal-on-metal articulations and increased awareness has been raised for this complication [2]. Revision to a metal-polyethylene articulation following fracture of a ceramic component has also been recognised as a significant cause of metallosis and is not currently recommended [3]. However, metallosis from a primary metal-polyethylene THR is quite rare and the sources of metal particles can be mode 2 wear (articulating surface moving against a non-articulating surface), mode 3 wear (third-body interposition between articulating surfaces), or mode 4 wear (2 secondary surfaces rubbing together) [4]. Liner wear, fracture or dislodgment can lead to a cobalt-chrome head abrading a soft titanium shell, creating large amounts of cobalt and titanium particles. Although wear-through of the shell is very rare, it has been seen in cases with delayed diagnosis [5]. Hip pain, instability, audible crepitus or clunk and an eccentric femoral head on X-ray help with a definite diagnosis [5]. Unfortunately, our patient was dismissed by the operating surgeon when he presented with those symptoms, although the x-ray showed the eccentricity of the head. This resulted in significant treatment delay, with liner fracture and complete wear-through of the titanium shell. The produced polyethylene and metal debris caused cup loosening and, more importantly, the circulating metal particles had a detrimental systemic effect on the patient and especially his brain function.

Cobalt systemic and particularly brain toxicity has been recognised clinically and termed Arthroprosthetic cobaltism and Arthroplasty Cobalt Encephalopathy (ACE) respectively [1,6]. Neurological symptoms vary and can include short-term memory loss, cognitive changes, dementia like and psychiatric symptoms, mood disorders, fatigue, vision and hearing loss, impaired motor and sensory function and more [7].

PET scans in patients with this condition suggest that there is a status of global brain hypometabolism, with some areas affected more than others and correlate this effect with cobalt blood levels [8]. Moreover, an MRI study revealed subtle structural brain changes associated with cobalt exposure even from well-functioning MoM prostheses [9]. Normal blood cobalt values are < 1 μg/L, 0.4 g/L according to Lauwerys and 0.8 g/L according to the INRS [10]. Previous research showed blood cobalt values < 1 μg/l to be the usual for the general population, > 4.5 μg/l to indicate abnormal wear in MOM THA, > 10 μg/l to predict high risk of early failure and > 20 μg/l to be associated with joint metallosis [10]. Moreover, a systematic review of patients with prosthetic hip-associated cobalt toxicity (PHACT) showed much higher Co blood and CSF concentrations than the ones recorded in our patient [11]. We encountered extreme local metallosis and acetabular loosening during revision surgery, as well as severe neurological impairment in our patient despite a Co blood level of 16.2 μg/l and a CSF level of 1.81 μg/l. However, it was evident that most of the metal debris originated from the acetabular shell that was made from Titanium and Ti levels were very high both in blood and in CSF. Although many orthopaedic implants are made from titanium alloys, very little is found in the literature regarding titanium metallosis and its systemic or brain toxicity. An *in vitro* study on rat and human glial cells showed that Ti nanoparticles induced a strong oxidative stress and caused morphological changes and damage to mitochondria in these cells, indicating toxicity [12].

The combination of a 32 mm head and a thin liner resulted in accelerated wear and liner fracture and should be avoided. Polyethylene particles can be blamed for cup loosening. However, it is not possible to state if cobalt alone, titanium alone or their combination led to such a severe neurologic damage to a previously asymptomatic patient. More research is necessary to examine toxicity of metals used for orthopaedic implants (Co, Ti, Mo, Al) and their combined toxic effects on human tissues and organs. Increased

awareness is required on the surgeons' behalf, for early identification of abnormal wear of hip implants, in order to prevent destructive local and systemic complications.

Bibliography

1. Gessner BD., *et al.* "A Systematic Review of Systemic Cobaltism after wear or corrosion of Chrome-Cobalt Hip Implants". *Journal of Patient Safety* 15.2 (2019): 97-104.

2. Delaunaya C., *et al.* "Metal-on-metal bearings total hip arthroplasty: The cobalt and chromium ions release concern". *Orthopaedics and Traumatology: Surgery and Research* 96 (2010): 894-904.

3. Harris A., *et al.* "Cobalt toxicity after revision to a metal-on-polyethylene total hip arthroplasty for fracture of ceramic acetabular component". *Arthroplasty Today* 1 (2015): 89-91.

4. Silva M., *et al.* "Bearing surfaces". In: Callaghan JJ, Rosenberg AG, Rubash HE (editions). The Adult Hip". 2nd ed. Philadelphia, PA: Lippincott Williams and Wilkins (2007): 251.

5. Cipriano CA., *et al.* "Metallosis after metal-on-polyethylene total hip arthroplasty". *American Journal of Orthopedics* 37.2 (2008): E18-E25.

6. Tower SS. "Arthroprosthetic cobaltism: neurological and cardiac manifestations in two patients with metal-on-metal arthroplasty: a case report". *Journal of Bone and Joint Surgery* 92.17 (2010): 2847-2851.

7. Cheung AC., *et al.* "Systemic cobalt toxicity from total hip arthroplasties. Review of a rare condition part 1 - history, mechanism, measurements, and pathophysiology". *The Bone and Joint Journal* 98-B (2016): 6-13.

8. Bridges R and Tower S. "PET/CT Brain Imaging of Arthroplasty Cobalt Encephalopathy (ACE) due to failure of Chromium-cobalt hip protheses - A works in progress". *Journal of Nuclear Medicine* 58.1 (2017): 484-484.

9. Clark MJ., *et al.* "Brain Structure and Function in Patients after Metal-on-Metal Hip Resurfacing". *American Journal of Neuroradiology* 35.9 (2014): 1753-1758.

10. Sidaginamale RP., *et al.* "Blood metal ion testing is an effective screening tool to identify poorly performing metal-on-metal bearing surfaces". *Bone and Joint Research* 2.5 (2013): 84-95.

11. Devlin JJ., *et al.* "Clinical Features, Testing, and Management of Patients with Suspected Prosthetic Hip-Associated Cobalt Toxicity: a Systematic Review of Cases". *Journal of Medical Toxicology* 9 (2013): 405-415.

12. Huerta-García E., *et al.* "Titanium dioxide nanoparticles induce strong oxidative stress and mitochondrial damage in glial cells". *Free Radical Biology and Medicine* 73 (2014): 84-94.