J Arthroplasty. 2016 Oct;31(10):2332-6. doi: 10.1016/j.arth.2016.03.009. Epub 2016 Mar 18.

Trunnionosis: Does Head Size Affect Fretting and Corrosion in Total Hip Arthroplasty?

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Author information

Abstract

BACKGROUND:

Wear and tribocorrosion at the modular head-neck taper interface may be a cause of failure in metal-on-polyethylene total hip arthroplasty (THA). The present investigation endeavored to elucidate the effect of femoral head diameter on fretting and corrosion in retrieved head-neck tapers.

METHODS:

A retrieval analysis of THA prostheses in vivo for a minimum of 1 year was performed. Twenty-three femoral heads of 32-mm diameter were matched with 28-mm heads based on time in vivo and head length (-3 mm to +8 mm). All included implants featured a single taper design from a single manufacturer. Fretting and corrosion damage scoring was performed for each implant under stereomicroscopic visualization.

RESULTS:

Head diameter was observed to affect fretting ($P = .01$), with 32-mm femoral heads exhibiting greater total fretting scores than 28-mm heads. Fretting damage was greatest ($P = .01$) in the central concentric zone of the femoral head bore tapers, regardless of head diameter, length, or stem offset. No significant effect on total corrosion scores was observed for any head or stem variable. Retrieved implant total corrosion scores were positively correlated ($\rho = 0.51$, $P < .001$) with implantation time.

CONCLUSION:

Increased femoral head diameter in THA may produce greater fretting damage owing to and increased head-neck moment arm. There is no associated increase in corrosion with 28-mm and 32-mm heads of this taper design. The longer a THA prosthesis is implanted, the greater the risk of damage due to corrosion.

Bone Joint J. 2015 Jul;97-B(7):911-6. doi: 10.1302/0301-620X.97B7.35149.

Taperosis: Does head length affect fretting and corrosion in total hip arthroplasty?

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Abstract

Tribocorrosion at the head-neck taper interface - so-called 'taperosis' - may be a source of metal ions and particulate debris in metal-on-polyethylene total hip arthroplasty (THA). We examined the effect of femoral head length on fretting and corrosion in retrieved head-neck tapers in vivo for a minimum of two years (mean 8.7 years; 2.6 to 15.9). A total of 56 femoral heads ranging from 28 mm to 3 mm to 28 mm + 8 mm, and 17 femoral stems featuring a single taper design were included in the study. Fretting and corrosion were scored in three horizontally oriented concentric zones of each taper by stereomicroscopy. Head length was observed to affect fretting ($p = 0.03$), with 28 mm + 8 mm femoral heads showing greater total fretting scores than all other head lengths. The central zone of the femoral head bore taper was subject to increased fretting damage ($p = 0.01$), regardless of head length or stem offset. High-offset femoral stems were associated with greater total fretting of the bore taper ($p = 0.04$). Increased fretting damage is seen with longer head lengths and high-offset femoral stems, and occurs within a central concentric zone of the femoral head bore taper. Further investigation is required to determine the effect of increased head size, and variations in head-neck taper design.

Hip Int. 2015 Jan-Feb;25(1):7-14. doi: 10.5301/hipint.5000180. Epub 2014 Oct 27.

Clinically significant corrosion at the head-neck taper interface in total hip arthroplasty: a systematic review and case series.

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Abstract

Corrosion of the head-neck junction of the femoral component in total hip arthroplasty has been associated with symptomatic adverse local tissue reactions, trunion fracture and elevated serum metal ions. An analysis of risk factors and treatment strategies for corrosion at this interface is lacking in the literature. We therefore performed a systematic review of AAOS proceedings, MEDLINE and EMBASE databases, and included our own case series. A total of 24 articles representing 776 cases of head-neck corrosion met inclusion criteria. The combination of large femoral head sizes and small taper dimensions comprised the majority of published corrosion cases. Revision to ceramic head and ceramic/polyethylene liner was the most commonly utilised treatment. Coating precipitation, mixed alloy coupling and head-neck modulus mismatch collectively appear to contribute to the corrosive process.

Clin Toxicol (Phila). 2014 Sep-Oct;52(8):837-47. doi: 10.3109/15563650.2014.944977. Epub 2014 Aug 16.

Systemic toxicity related to metal hip prostheses.

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Abstract

INTRODUCTION:

One in eight of all total hip replacements requires revision within 10 years, 60% because of wear-related complications. The bearing surfaces may be made of cobalt/chromium, stainless steel, ceramic, or polyethylene. Friction between bearing surfaces and corrosion of non-moving parts can result in increased local and systemic metal concentrations.

OBJECTIVES:

To identify and systematically review published reports of systemic toxicity attributed to metal released from hip implants and to propose criteria for the assessment of these patients.

METHODS:

Medline (from 1950) and Embase (from 1980) were searched to 28 February 2014 using the search terms (text/abstract) chrom* or cobalt* and [toxic* or intox* or poison* or adverse effect or complication] and [prosthes* or 'joint replacement' or hip or arthroplast*] and PubMed (all available years) was searched using the search term (("Chromium/adverse effects"[Mesh] OR "Chromium/poisoning"[Mesh] OR "Chromium/toxicity"[Mesh]) OR ("Cobalt/adverse effects"[Mesh] OR "Cobalt/poisoning"[Mesh] OR "Cobalt/toxicity"[Mesh])) AND ("Arthroplasty, Replacement, Hip"[Mesh] OR "Hip Prosthesis"[Mesh]). These searches identified 281 unique references, of which 23 contained original case data. Three further reports were identified from the bibliographies of these papers. As some cases were reported repeatedly the 26 papers described only 18 individual cases. Systemic toxicity. Ten of these eighteen patients had undergone revision from a ceramic-containing bearing to one containing a metal component. The other eight had metal-on-metal prostheses. Systemic toxicity was first manifest months and often several years after placement of the metal-containing joint. The reported systemic features fell into three main categories: neuro-ocular toxicity (14 patients), cardiotoxicity (11 patients) and thyroid toxicity (9 patients). Neurotoxicity was manifest as peripheral neuropathy (8 cases), sensorineural hearing loss (7) and cognitive decline (5); ocular toxicity presented as visual impairment (6). All these neurological features, except cognitive decline, have been associated with cobalt poisoning previously. Type of prosthesis and blood metal concentrations. Where blood or serum metal concentrations were reported (n = 17 for cobalt and n = 14 for chromium), the median cobalt concentration was 398 (range, 13.6-6521) μg/L and the median chromium concentration was 48 μg/L (in whole blood) (range, 4.1-221 μg/L including serum and blood values). Those patients reported to have systemic features who had received a metal-on-metal prosthesis (n = 8) had a median peak blood cobalt concentration of 34.5 (range, 13.6-398.6) μg/L; those with a metal-containing revision of a failed ceramic prosthesis (n = 10) had a median blood cobalt concentration of 506 (range, 353-6521) μg/L. Management. The most common treatment was removal of the metal-containing prosthesis, undertaken in all but 2 patients. This

was usually associated with a fall in circulating cobalt concentration and improvement in some or all features. Clinical and toxicological assessment of systemic features. We propose the following criteria for assessing the likelihood that clinical features are related to cobalt toxicity: clinical effects consistent with the known neurological, cardiac, or thyroidal effects of cobalt, and for which any other explanation is less likely; increased blood cobalt concentrations (substantially higher than those in patients with well-functioning prostheses) several months after hip replacement; a fall in the blood cobalt concentration, usually accompanied by signs of improvement in features. When judged by these criteria, the systemic features in 10 of the reported cases are likely to be related to cobalt exposure from a metal-containing hip prosthesis.

CONCLUSIONS:

Rarely, patients exposed to high circulating concentrations of cobalt from failed hip replacements develop neurological damage, hypothyroidism and/or cardiomyopathy, which may not resolve completely even after removal of the prosthesis. The greatest risk of systemic cobalt toxicity seems to result from accelerated wear of a cobalt-containing revision of a failed ceramic prosthesis, rather than from primary failure of a metal-on-metal prosthesis.

Clin Toxicol (Phila). 2014 Feb;52(2):98-112. doi: 10.3109/15563650.2013.857024. Epub 2013 Nov 20.

Interpreting cobalt blood concentrations in hip implant patients.

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Abstract

INTRODUCTION. There has been some recent concern regarding possible systemic health effects resulting from elevated blood cobalt concentrations in patients with cobalt containing hip implants. To date there are no blood cobalt criteria to help guide physicians when evaluating an individual hip implant patient's risk of developing systemic health effects because historically there was little or no concern about systemic cobalt toxicity in implant patients. OBJECTIVE. Our purpose is to describe recently completed research regarding the relationship between blood cobalt concentrations and clinical health effects. We discuss the possibility of systemic health effects in patients with metal containing implants and propose various blood cobalt concentrations that are not associated with an increased risk of developing certain adverse effects. METHODOLOGY. The primary literature search was conducted using PubMed and Web of Science using the following search terms: cobalt AND (toxicity OR health effects OR cardiotoxicity OR hematological OR endocrine OR immunological OR reproductive OR testicular effects OR neurological OR case report OR cohort OR Roncovite). The searches identified 6786

papers of which 122 were considered relevant. The Agency for Toxic Substances and Disease Registry toxicological profile for cobalt and the U.S. Environmental Protection Agency Office of Research and Development's National Center for Environmental Assessment's documentation on the provisional peer-reviewed toxicity value for cobalt were also utilized to identify secondary literature sources. RESULTS. Our review of the toxicology and medical literature indicates that highly elevated blood cobalt concentrations can result in certain endocrine, hematological, cardiovascular, and neurological effects in animals and/or humans. These studies, in addition to historical clinical findings involving the therapeutic use of cobalt, indicate that significant systemic effects of cobalt will not occur below blood cobalt concentrations of 300 μg/L in most persons. Some individuals with specific risk factors for increased susceptibility (e.g., severe and sustained hypoalbuminemia) may exhibit systemic effects at lower cobalt blood concentrations. This review also describes several cobalt dosing studies performed with human volunteers that consumed cobalt for 15, 30, or 90 days. Overall, the results of these dosing studies indicate that sustained blood cobalt concentrations averaging 10-70 μg/L for up to 90 days cause no significant clinical effects (maximum concentrations approached 120 μg/L). Some proposed blood criteria for assessing implant wear and local tissue damage have been suggested by several medical groups. For example, the UK Medicines and Healthcare Products Regulatory Agency has proposed a blood cobalt guidance value of 7 μg/L, and the Mayo Clinic has suggested serum cobalt concentrations greater than 10 μg/L, but both of these values are primarily intended to address implant wear and to alert physicians to the possibility of an increased incidence of local effects. There is a clear lack of consensus regarding how to identify a specific numerical blood concentration of concern and whether whole blood or serum is a better matrix to assess total cobalt concentration. CONCLUSIONS. Based on currently available data, only under very unusual circumstances should a clinician expect that biologically important systemic adverse effects might occur in implant patients with blood cobalt concentrations less than 300 μg/L. Patients with metal-containing hip implants who exhibit signs or symptoms potentially related to polycythemia, hypothyroidism, neurological, or cardiac dysfunction should be clinically evaluated for these conditions. Polycythemia appears to be the most sensitive endpoint.

Clin Toxicol (Phila). 2016 Nov;54(9):874-877. Epub 2016 Aug 5.

Fatal cobalt toxicity after total hip arthroplasty revision for fractured ceramic components.

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Abstract

CONTEXT:

Post-arthroplasty metallosis, which refers to metallic corrosion and deposition of metallic debris in the periprosthetic soft tissues of the body, is an uncommon complication. Systemic cobalt toxicity post-arthroplasty is extremely rare. The few known fatal cases of cobalt toxicity appear to be a result of replacing shattered ceramic heads with metal-on-metal or metal-on-polyethylene implants. Friction between residual shards of ceramic and cobalt-chromium implants allows release of cobalt into the synovial fluid and bloodstream, resulting in elevated whole blood cobalt levels and potential toxicity.

CASE DETAILS:

This is a single patient chart review of a 60-year-old woman with prior ceramic-on-ceramic right total hip arthroplasty complicated by fractured ceramic components and metallosis of the joint. She underwent synovectomy and revision to a metal-on-polyethylene articulation. Ten months post-revision, she presented to the emergency department (ED) with right hip pain, dyspnea, worsening hearing loss, metallic dysgeusia, and weight loss. Chest CTA revealed bilateral pulmonary emboli (PE), and echocardiogram revealed new cardiomyopathy with global left ventricular hypokinesis with an ejection fraction (EF) of 35-40% inconsistent with heart strain from PE. Whole blood cobalt level obtained two days into her admission was 424.3 mcg/L and 24-h urine cobalt level was 4830.5 mcg/L. Although the patient initially clinically improved with regard to her PE and was discharged to home on hospital day 5, she returned 10 days later with a right hip dislocation and underwent closed reduction of the hip. The patient subsequently decompensated, developing cardiogenic shock, and respiratory failure. She went into pulseless electrical activity (PEA) and expired. Autopsy revealed an extensive metallic effusion surrounding the right hip prosthesis that tested positive for cobalt (41,000 mcg/L). There was also cobalt in the heart muscle tissue (2.5 mcg/g). A whole blood cobalt level obtained two days before she expired was 641.6 mcg/L.

DISCUSSION:

This is a case of fatal cobalt-induced cardiomyopathy in a patient whose ceramic components of a total hip arthroplasty fractured causing metallosis with worsening cobalt toxicity. We recommend that when a fractured device is revised with a prosthesis with cobalt-chromium components, whole blood and urine cobalt measurements should be obtained and periodically monitored to evaluate for rising concentrations. Providers should be aware of clinical signs and symptoms of cobalt toxicity in patients who have prostheses with cobalt-chromium components. If suspected, toxicology and orthopedics should be involved for possible chelation and removal of the prosthesis.