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Case report

Corrosion of Polished Cobalt-Chrome Stems Presenting as Cobalt Encephalopathy

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ABSTRACT

Adverse reactions to metallic debris from corrosion of polished cobalt-chromium–cemented femoral stems are reported. Cobaltism (systemic cobalt poisoning) has not been reported from this phenomenon. Three patients presented to their surgeon for ongoing care 10-20 years after primary metal-on-plastic hip arthroplasty with the same polished cobalt-chromium–cemented femoral stems (Heritage, Zimmer). Urine cobalt was elevated, and the patients had symptoms consistent with cobaltism. Quantitative-F^{16}DG-PET-CT brain imaging was performed showing generalized and focal brain hypometabolism consistent with cobalt encephalopathy. At revision, all stems were well fixed and grossly corroded. At 1 year after revision, cobalturia and cognitive symptoms were resolved or improved. Mechanically assisted crevice corrosion at the polymethylmethacrylate interface is a complication of polished cobalt-chromium–cemented stems that can result in systemic cobalt exposure and toxic encephalopathy. Our cases had only minor periprosthetic symptoms. Patients implanted with polished cobalt-chromium–cemented stems warrant monitoring with urine cobalt. Patients with cobaltemia warrant an evaluation for toxic encephalopathy.

Introduction

Corrosion of stainless-steel–cemented stems rarely resulted in fracture of Charnley's flat-backed design [1]. Cement-stem interface corrosion appears to be universally experienced in modern stainless-steel polished cemented stems (Exeter, Stryker, Kalamazoo) with rare clinical periprosthetic consequence [2-6]. Titanium alloy–cemented stems were largely abandoned because corrosion at the stem-cement interface was associated with painful femoral hypertrophy [7,8]. Polished cobalt-chromium–cemented femoral stems (PCoCrCFS) CPT, Zimmer, Warsaw, IN and Ultima TPS, DePuy Synthes, Warsaw, IN have recently been reported to be prone to clinically significant adverse reactions to metallic debris (ARMDs) from stem-cement interface corrosion if a metal-on-metal (MoM) articular couple is used [9,10].

Corrosion or wear of cobalt-chromium implants can result in cobaltism in addition to periprosthetic complications [11]. Arthroprosthetic cobaltism has been largely reported from wear of MoM articulations or from catastrophic wear of revision cobalt-chromium heads by debris from a fractured ceramic implant [11]. Cobaltism most commonly presents with nonspecific constitutional, neurologic, and psychiatric symptoms, with patients who experienced extreme cobalt exposure may experience profound deafness, blindness, peripheral neuropathy, parkinsonism, heart failure, thyropathy, and death [11]. Reversible cobaltism has been recently reported from trunnionosis with nominal elevations of blood cobalt ions [12,13]. Quantitative-F^{16}DG-PET-CT brain imaging (QFDGPETBI) appears to have utility in confirming the diagnosis of arthroplasty cobalt encephalopathy (ACE). A recent report noted a pathognomonic pattern of quantitative brain hypometabolism in 57 neurologically symptomatic arthroprosthetic patients with elevated urine cobalt (≥1 ppb), the majority of whom were implanted with metal-on-plastic (MoP) hips [14]. We report 3 cases of severe PCoCrCFS corrosion in women with ACE implanted with well-fixed Zimmer (Warsaw, IN) Heritage stems. The major

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indication for revision surgery was cobaltism; all patients underwent QFDGPETBI that showed the characteristic pattern of quantitative hypometabolism described for ACE [14].

Case histories

The senior author was the primary and revision surgeon for these cases. Since 2015, all patients presenting to his practice with a hip with a CoCr component are screened for cobaltism with a urine cobalt and a Cobaltism Symptom Inventory. The women consented to have their redacted information used for this case report, and they are part of the senior author's larger study cohort of 68 patients with arthroprosthetic cobalturia who underwent QFDGPETBI [14]. Our clinic first recognized the utility of QFDGPETBI to diagnose ACE in 2015, and we routinely use this diagnostic modality for patients with cobalturia experiencing neurologic or constitutional decompensation [14,15] (Table 1, Figs. 1 and 2).

The primary arthroplasties used Zimmer (Warsaw, IN) Heritage stems with modular cobalt-chromium heads with Trilogy shell with plastic inserts. This construct was the senior author's preferred implant in patients with Dorr B and C bone from 1992 through 2010.

At presentation, radiographs showed well-fixed components with minimal polyethylene wear, and the patients noted new minor exercise-related hip soreness. All subjects had elevated urine cobalt and progressive symptoms consistent with neurocobaltism, most commonly cognitive decline, fatigue, and tremor [11]. Cobalt was also elevated in whole blood and periprosthetic fluid aspirated in clinic or at revision surgery. Focal brain hypometabolism on QFDGPETBI consistent with ACE was found in all cases (Fig. 2) [14]. The primary indication for revision surgery was suspected neurocobaltism [11]. Stem revision required extended trochanteric osteotomy (ETO) in 2 cases, and removal of cement was performed with headlight and osteotomes. Revision titanium alloy stems with ceramic heads were used. The patients had resolution of cobaltemia and cobalturia and noted cognitive improvement 1 year after revision. One patient repeated QFDGPETBI at 1 year after revision which found comparatively improved brain metabolism [14].

Case 1

Right primary arthroplasty was performed in 2006 for osteoarthritis for a 68-year-old fit woman. Asymptomatic elevations of blood and urine cobalt were first noted in 2014. Over the next 4 years, progressive fatigue, cognitive decline, relative depression, and an intention tremor of the right index finger developed. Blood and urine cobalt levels increased, and progressive quantitative brain hypometabolism was found on serial QFDGPETBI consistent with ACE [14]. Simultaneously, progressive activity–related groin pain developed and metal suppression magnetic resonance imaging was consistent with ARMD. Revision was performed in 2018 after 11.8 years in situ, the stem was well–fixed, and gross corrosion residue was noted at the stem-cement interface. Stem and cement mantle were removed, press-fit TiAlV revision stem was placed with a 32-mm Delta ceramic head, and the Zimmer (Warsaw, IN) Longevity XPLE liner was retained because it showed no radiographic or gross evidence of damage. Figure 1(a-c) shows preoperative and postoperative radiographs and intraoperative photograph of the stem. Recovery was uneventful, and 2 years after revision, cognitive function, energy level, mood, and groin pain improved and the hand tremor is resolved. Explant analysis confirmed the gross finding of severe surface pitting corrosion on most of the stem interface with cement (Fig. 3 a–d), and visual examination showed some of the corrosion pits penetrated over 1.5 mm into the stem. The head bore-stem trunnion interface showed minimal corrosion damage (Fig. 4), and thus, the primary source of metal-ion debris came from the stem-cement mantle interface. The color of the corrosion debris was black, white, and/or red depending on the chemical constituents which included cobalt, chromium, iron, phosphorus, molybdenum, chlorine, oxygen, and calcium, as identified with energy-dispersive spectrometry (EDS). with the red-colored residue having significantly higher iron content (Fig. 5).

Case 2

A left primary total hip arthroplasty was performed in 2001 for osteoarthritis for a 60-year-old active fit woman. In 2018, the patient returned to care for symptoms at her contralateral hip. She noted no symptoms at her replaced hip. For a year, she noted short-term memory decline with occasional disorientation while driving and an intention tremor of the left little finger was noted on examination when fingers were extended in supination. Brain hypometabolism was found on QFDGPETBI consistent with ACE [14]. Revision was performed in 2018 after 17.5 years in situ, the stem was well fixed, and gross corrosion was noted at the stem-cement interface. The stem and cement mantle were removed, and a revision TiAlV press-fit stem was placed. Longevity liner was exchanged so that the articulation could be upsized to 32 mm. Figure 1(d-f) shows preoperative and postoperative radiographs and intraoperative photograph of the stem. Recovery was uneventful, and at 2 years, cognitive function was improved and the hand tremor resolved.

Case 3

Right primary arthroplasty was performed in 1997 for osteoarthritis for a 47-year-old active fit woman. In mid-2018, the patient returned for routine follow-up with new onset of cold urticaria, tinnitus, and deafness, and an intention tremor of the right index finger was noted on examination. Blood and urine cobalt were elevated, and over the next 6 months, because of new visual symptoms, progression of deafness, and new forgetfulness and word finding problems, a QFDGPETBI was performed. Brain hypometabolism consistent with ACE was found [14]. Revision was performed in late-2018 after 20.2 years in situ, the stem was well

Table 1
Summary data cases 1–3.

Case	Mass LBS	Head	Taper	Blood cobalt	Urine cobalt	Joint fluid cobalt	Histopathology	Composite QFDGPETBI score before revision [14]	Composite QFDGPETBI score 1 y after revision [14]	Blood cobalt at the last follow-up
1	155	32 + 7	12/14	4.1 ppb	11 ppb	390 ppb	No significant acute or chronic inflammation	−56	Not done	0.4 ppb
2	145	28 + 3.5	12/14	20 ppb	55 ppb	3,200 ppb	No significant acute or chronic inflammation	−72	Not done	1.9 ppb
3	135	26 medium	6 degree	1.3 ppb	14 ppb	290 ppb	No significant acute or chronic inflammation	−172	-145	0.3 ppb

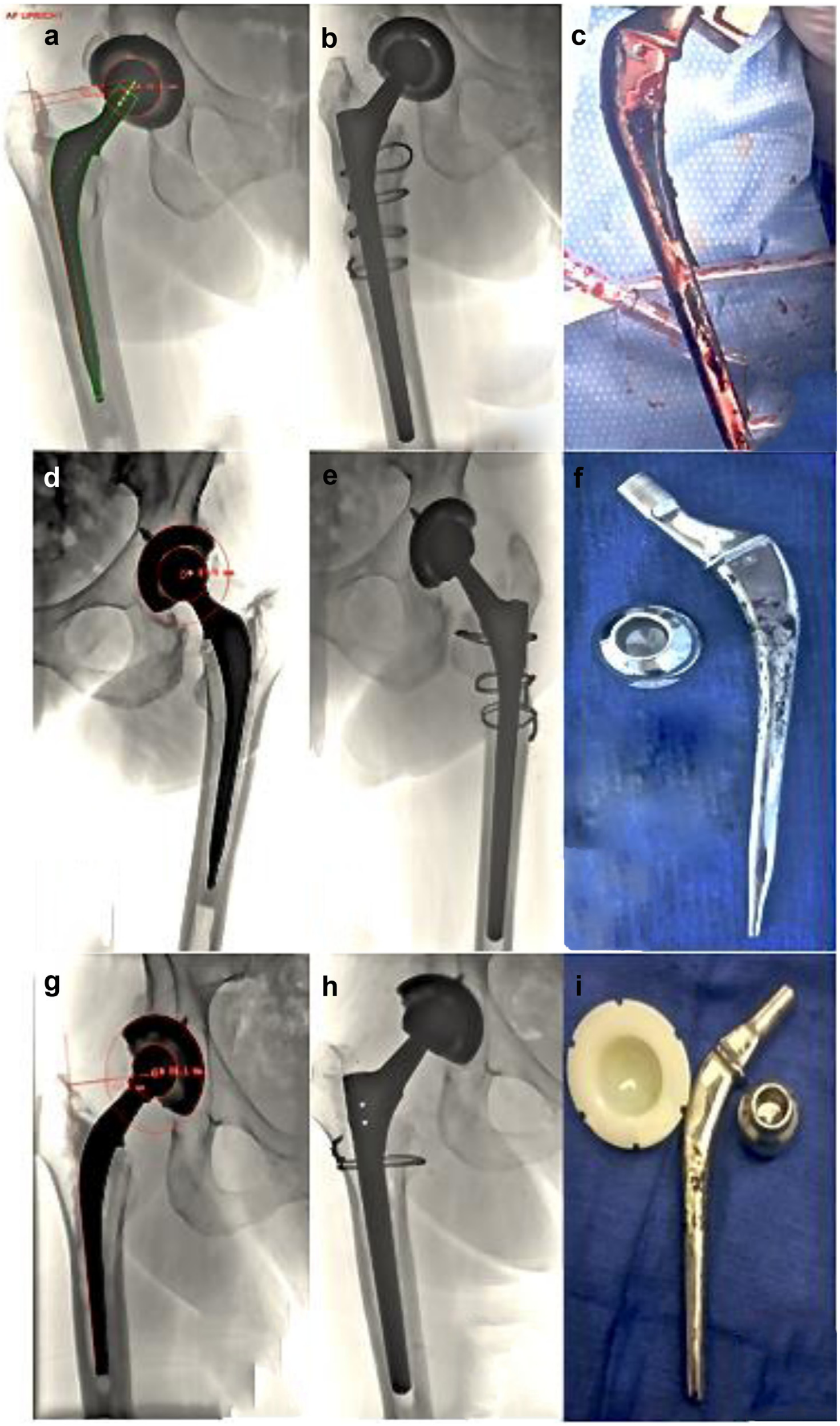

Figure 1. Figure 1. (a) Pre-revision radiograph case 1 shows no measurable polyethylene wear and well fixed stem, (b) case one radiograph post revision to pressfit titanium stem and ceramic head, (c) case 1 primary Heritage stem intra-operative photograph showing gross corrosion of cobalt-chromium cement interface, (d) Pre-revision radiograph case 2 showing no appreciable polyethylene wear and well fixed stem, (e) Post-revision radiograph case 2 showing revision to titanium pressfit stem and ceramic head, (f) Intraoperative photograph of extracted Heritage stem showing corrosion at cemented surfaces, (g) Prerevision radiograph case 3 showing minimal polyethylene wear and well fixed stem, (h) Postrevision radiograph showing revision of stem to pressfit titanium, head to 32 mm ceramic, and liner to crosslinked polyethylene, (i) Case 3 explanted components, Heritage stem shows corrosion of cemented surfaces.

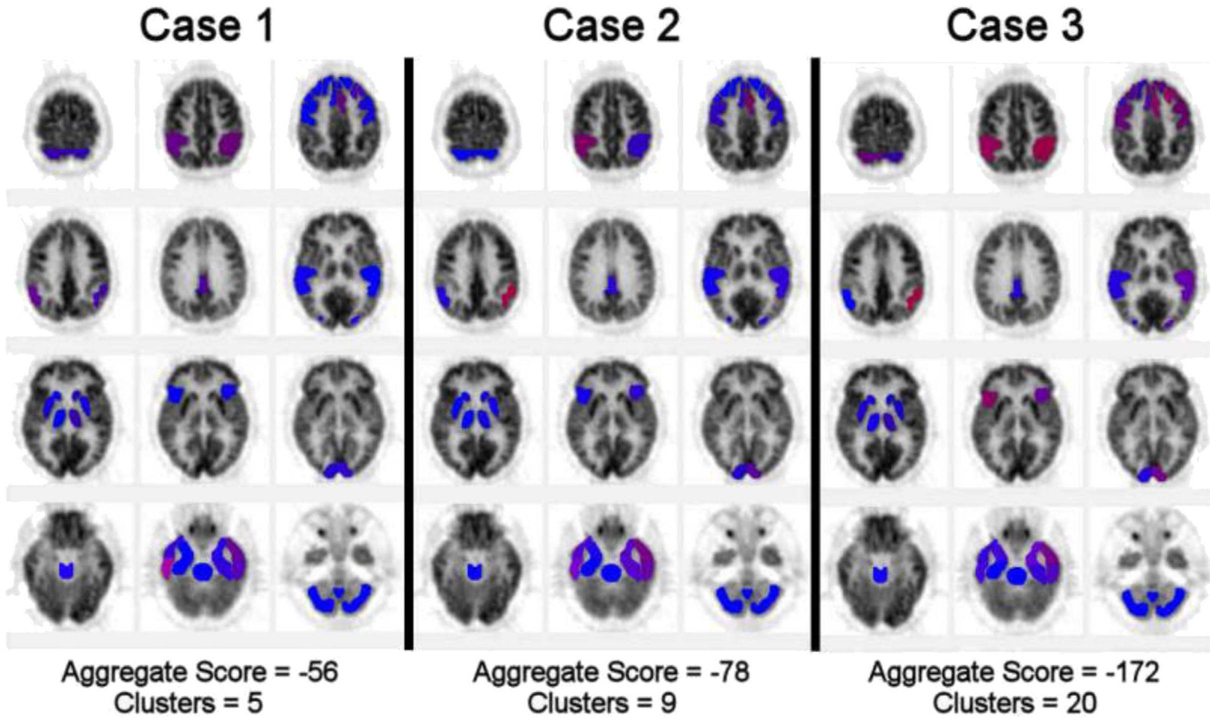

Figure 2. Progression from Blue to Red defining increasing hypometabolism.

Figure 3. (a) An explanted stem with corrosion pits highlighted with arrows, (b) red-colored corrosion residue with high levels of iron, (c) a large corrosion pit with white-colored corrosion residue, and (d) a corrosion pit over 1.5mm deep with black-colored corrosion residue.

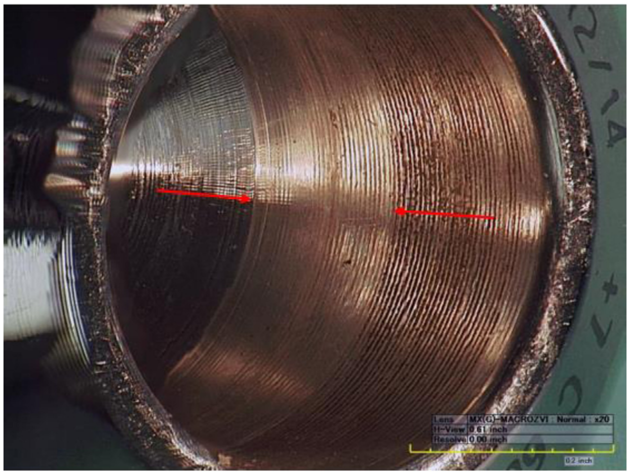

Figure 4. The inside of the trunnion showing minimal corrosion damage. The original circumferential machining marks are still visible.

fixed, and gross corrosion was noted at the stem-cement interface. The stem and cement mantle were removed, and a press-fit TiAlV stem was placed with a 32-mm Delta ceramic head. The ultra high molecular weight polyethylene liner was exchanged for a Longevity liner so that the bearing could be upsized to 32 mm. Figure 1(g-i) shows preoperative and postoperative radiographs and intraoperative photograph of the stem. Recovery was uneventful, and at 1 year, cognitive function and visual and auditory symptoms were improved and the hand tremor was resolved. Repeat QFDGPETBI showed improved brain metabolism.

Discussion

Historically stem-cement interface corrosion occasionally resulted in fracture of stainless-steel stems and thigh pain in patients with well-fixed TiAlV stems [1,7]. Recently, stem-cement interface corrosion of PCoCrCFS has been implicated, provoking ARMD requiring stem revision [9,10]. Our experience indicates that patients implanted with PCoCrCFS with MoP articulations may experience systemic cobalt poisoning without hip symptoms [14].

QFDGPETBI has become an accepted and important diagnostic tool for the assessment of brain disease. While interpretation of these scans has relied on visual interpretation, early stages of disease may not be readily apparent visually [16]. Food and Drug Administration–approved analytic software provides a repeatable and quantitative method for objectively comparing patients' brain scans against an atlas of normal brains matched for age and sex [17,18]. Comparisons use a reference region that classically has been either the pons or cerebellum. Quantitative analysis has increasingly become the standard for initial interpretation and monitoring of brain disease [17,18]. Prior studies report some regions of the brain become hypometabolic before others in patients experiencing the named dementias, injury, or toxins: chemotherapy, organic solvents, and heavy metals [16,19-21]. Subjects with nominally elevated blood cobalt levels from well-functioning MoM hip resurfacings have focal brain substance atrophy in addition to echocardiographic diastolic dysfunction [22,23]. From early work into the neurotoxicity of elevated systemic cobalt levels from failing joint prostheses, QFDGPETBI has shown several regions of the brain to be compromised early due to cobalt [14], these include the temporal, frontal and parietal lobes along with the anterior cingulate gyri. Patients with elevated urine and blood cobalt with those regions of the brain showing hypometabolism should be considered at heightened risk for arthroprosthetic cobalt encephalopathy. Reversibility of neurotoxicity, clinically and by repeat QFDGPETBI, has been documented in patients undergoing revision of their cobalt alloy prostheses to noncobalt alternatives [14]. NeuroQ, the analytic software used in this case report and our previous case series, separates the brain into 240 regions, which are then combined into 47 statistically significant, related areas of the brain, termed clusters [18]. The threshold for abnormality is $\pm$1.65 standard deviations. In addition to the numerical readout of the statistics, the clusters are color coded, with 'blue' being normal and increasing shades toward 'red' being greater degrees of altered metabolism. In these cases, red is the greatest degree of hypometabolism with respect to the pons as the reference region. The summed aggregate of the 240 regions, the number of clusters, and the degree of hypometabolism per cluster quantify the amount of hypometabolism (Fig. 2) [14].

Take-home point from these 3 cases include

- Cobalt-chromium may be a suboptimal material for polished cemented femoral stems comparative to stainless steel because of the potential for systemic cobalt exposure.
- Monitoring of patients with PCoCrCFS for cobalturia and neurologic symptoms should be considered.
- QFDGPETBI appears to be a useful modality to confirm the diagnosis of ACE for patients with neurologically symptomatic cobalturia ($\geq$1 ppb).

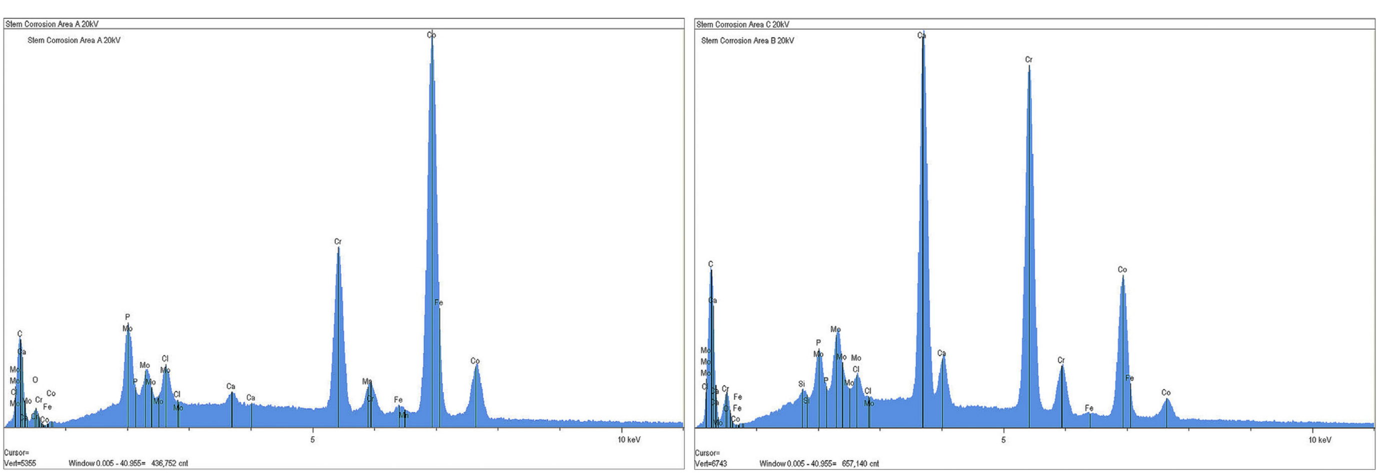

Figure 5. Energy -dispersive spectrometry scans showing the corrosion oxide residue chemistry that includes cobalt, chromium, iron, phosphorus, molybdenum, chlorine, oxygen, and calcium.

- Surgeons revising patients implanted with PCoCrCFS need to be aware that stem revision is required to resolve corrosion-related ARMD or cobaltism regardless of bearing couple or the finding of trunnionosis. In our experience, these stems are well fixed and the corrosion at the stem-cement interface only becomes apparent during stem removal that often requires an extended trochanteric osteotomy.

Summary

Corrosion at the stem-cement interface of PCoCrCFS may be periprosthetically clinically and radiographically silent yet can result in systemic cobalt toxicity. Further use of polished force-closed cobalt-chromium stems should be questioned, and monitoring programs considered for those patients already implanted. Patients with symptoms consistent with cobaltism and a urine cobalt level of $\geq$1 ppb may benefit from quantitative QFDGPETBI to confirm the diagnosis of ACE. In our experience, cobaltism is generally reversible if source control can be reasonably safely achieved by revision surgery to a cobalt-free construct.

Conflict of Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to have appeared to influence the work reported in this paper.

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