

# A case report of cobalt cardiomyopathy leading to electric storm and cardiogenic shock: the importance of the orthopaedic background in patients with heart failure of unknown aetiology

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## Background

The first series of cobalt cardiomyopathy was described in the 60s in relation to the abuse of a cobalt containing beer. Since then, millions of metal hip arthroplasties have been performed and a small number of cobalt cardiomyopathies related to metal prosthesis have been reported.

## Case summary

We report a case of a 48-year-old man who developed a severe non-dilated restrictive cardiomyopathy in the setting of a systemic metallosis following several hip arthroplasties. The diagnosis was suspected by exclusion of other more common causes for restrictive cardiomyopathies and confirmed by the levels of cobalt and chromium in the serum and the endomyocardial biopsy performance that showed metal deposits in myocardial tissue. Despite the removal of the metal prosthesis and a significant decrease in serum metal levels, he suffered cardiogenic shock (CS) and electric storm that required emergency mechanical circulatory support as a bridge to heart transplant.

## Discussion

Cobalt cardiomyopathy is a rare condition that has been observed in patients who develop cobalt toxicity after metal hip arthroplasty. The condition may improve after diagnosis and removal of the prosthesis or get worse and progress to end-stage heart failure or CS. The concern about the metal toxicity associated with metal hip prosthesis has increased in the last few years. Orthopaedic surgeons and cardiologists should be aware of this severe complication that is probably under diagnosed.

## Keywords

Cobalt cardiomyopathy • Cardiogenic shock • Mechanical circulatory support • Heart transplant

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## Learning points

- Cobalt toxicity may appear after metal hip arthroplasty. It can potentially cause hearing loss, skin problems, polycythaemia, thyroid disorders, impairment of the peripheral nerves, and heart failure.
- Cobalt cardiomyopathy is a restrictive cardiomyopathy that can be fatal. Reaching a diagnosis can be challenging so a high index of clinical suspicion is required.
- When cobalt cardiomyopathy is diagnosed, removal of the metal hip prosthesis is mandatory. After the surgery, even when the levels of cobalt in the blood decrease, the cardiac condition can either improve or get worse quickly, leading to acute heart failure or cardiogenic shock. Mechanical circulatory support as a bridge to heart transplant is feasible in this scenario.

## Primary specialities involved in addition to cardiology

Orthopedic Surgery  
Cardiac Surgery  
Otorhinolaryngology  
Radiodiagnosis  
Pathology

## Introduction

Cobalt toxicity is an uncommon but potentially severe complication of metal-on-metal (MoM) hip arthroplasty.<sup>1</sup> Hip prosthesis can be made of a different combination of materials such as ceramic-on-ceramic (CoC), MoM, ceramic-on-polyethylene or metal-on-polyethylene (MoP). Cobalt and chromium metal ions could potentially be released into the circulation, especially in the setting of an MoM arthroplasty<sup>2</sup> or an MoP implanted after the fracture of a previous ceramic arthroplasty.<sup>3</sup> Elevated levels of cobalt in the blood could lead to a specific kind of cardiomyopathy and affect other organs and systems like peripheral nerves or hearing.<sup>1</sup> This case shows the severity of this illness and the possible evolution to electrical storm and cardiogenic shock (CS) even after the removal of the metal source.

## Timeline

Prior history	Bilateral total hip arthroplasty in 2008 and 2010 Replacement of right prosthesis after breakage in 2014 Rosacea and sensorineural hearing loss in January 2018
March 2018	Restrictive cardiomyopathy diagnosed at referral hospital after 1 year—history of heart failure and weight loss Neurohormonal blockade therapies started
April 2019	First decompensation which required admission Trans-thoracic echocardiogram showed severe biventricular dysfunction

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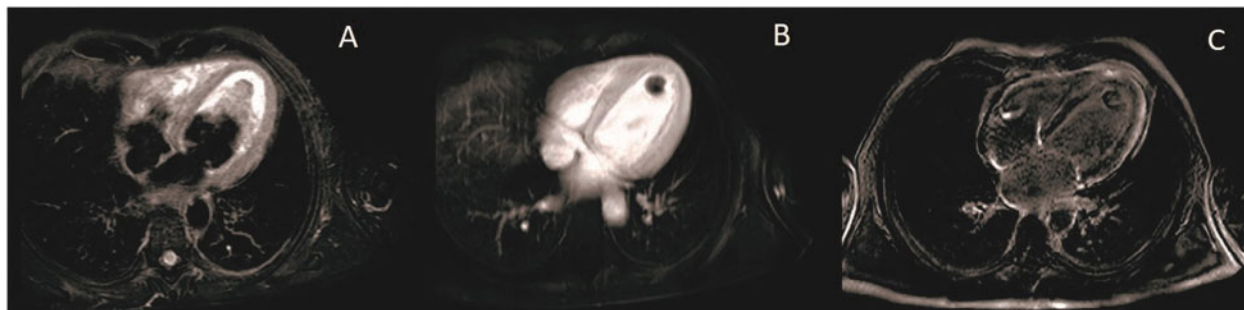
May 2019	Implantable cardioverter-defibrillator New admission due to heart failure Transferred for heart transplant candidacy evaluation Complete diagnosis work-up performed excluding common causes for restrictive/hypertrophic cardiomyopathy High cobalt levels in blood and myocardial samples
27 September 2019	Prosthesis replacement (ceramic-on-ceramic) at his referral centre finding extensive metallosis
6 October 2019	Hospital admission due to an electrical storm Transferred to cardiac intensive care unit, evolving to an INTERMACS 1 situation Venoarterial ECMO and Intra-aortic balloon pump
7 October 2019	Temporary biventricular assist device (BIVAD) implantation due to insufficient left ventricle unloading on ECMO
8 October 2019	Low flow events in the setting of severe transitory myocardial oedema Cardiac tamponade requiring reoperation
27 October 2019	Stable after 19 days on BIVAD Sensitization ruled out Decline in metal levels confirmed Listed for heart transplantation
30 October 2019	Heart transplantation performed Favourable postoperative response
8 November 2019	Discharged to the ward Normal graft function Hearing improvement
22 November 2019	Discharged home

## Case presentation

In May 2019, a 48-year-old male patient was referred to the advanced heart failure unit in our hospital.

He had no cardiovascular risk factors and had been very athletic in the past. He had been diagnosed with rosacea years before and suffered sudden severe sensorineural hearing loss in 2018.

A total hip arthroplasty with CoC prosthesis on the right side had been performed in 2008 and on the left side in 2010, both in the setting of avascular necrosis. In 2014, he felt a clicking noise in the right



**Figure 1** (A–C) Cardiac magnetic resonance imaging showing normal wall thickness, an apical left ventricle thrombus, and diffuse late gadolinium enhancement.

hip prosthesis while he was getting out of a van. A modular-neck prosthesis fracture on the right side was diagnosed and new total hip arthroplasty was required. The acetabular line and the ceramic head were removed and an MoP prosthesis was implanted.

He had no relevant family medical history.

In May 2018, he was attended to in his referral hospital because of heart failure symptoms. The echocardiogram was compatible with an early stage restrictive vs. hypertrophic cardiomyopathy with a mildly dilated left ventricle (LV), slightly thickened walls, and moderate LV dysfunction. Additionally, the magnetic resonance imaging (MRI) showed mild myocardial oedema, a small apical thrombus and subepicardial delayed enhancement in both atria and ventricles that was not present in the basal and medium septum ([Figure 1](#)). A 99m TC-DPD scintigraphy was performed showing no ATTR amyloidosis compatible data. A coronary angiogram ruled out significant disease. A next-generation sequencing panel for genes associated with hypertrophic, dilated, and restrictive cardiomyopathies did not reveal any likely pathogenic variants. At that point, he was in NYHA class II under treatment with diuretics, warfarin, angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, and ivabradine.

One year later, he was admitted because of heart failure. An echocardiogram showed a decline in systolic LV function. No thrombus in the apex was visualized. An implantable cardioverter-defibrillator (ICD) as a primary prevention was implanted and he was referred to our heart transplant unit.

When first evaluated in our hospital (May 2019), he was in NYHA class III. In the physical examination, a striking third sound and elevated jugular venous pressure were noticed. Severe hearing loss was present.

The electrocardiogram (ECG) showed sinus rhythm 80 b.p.m., right atrial enlargement data, low QRS voltage in the frontal plane, and a pathologic q wave in the inferior leads. The blood tests revealed polycythaemia (haemoglobin 18.8 g/dL; normal range 13–18 g/dL) without any other significant findings.

The echocardiogram revealed severe systolic dysfunction of both ventricles with an ejection fraction of 15% and a TAPSE value of 12 mm. Diastolic function assessment showed a restrictive filling pattern. There was mild hypertrophy of the interventricular septum

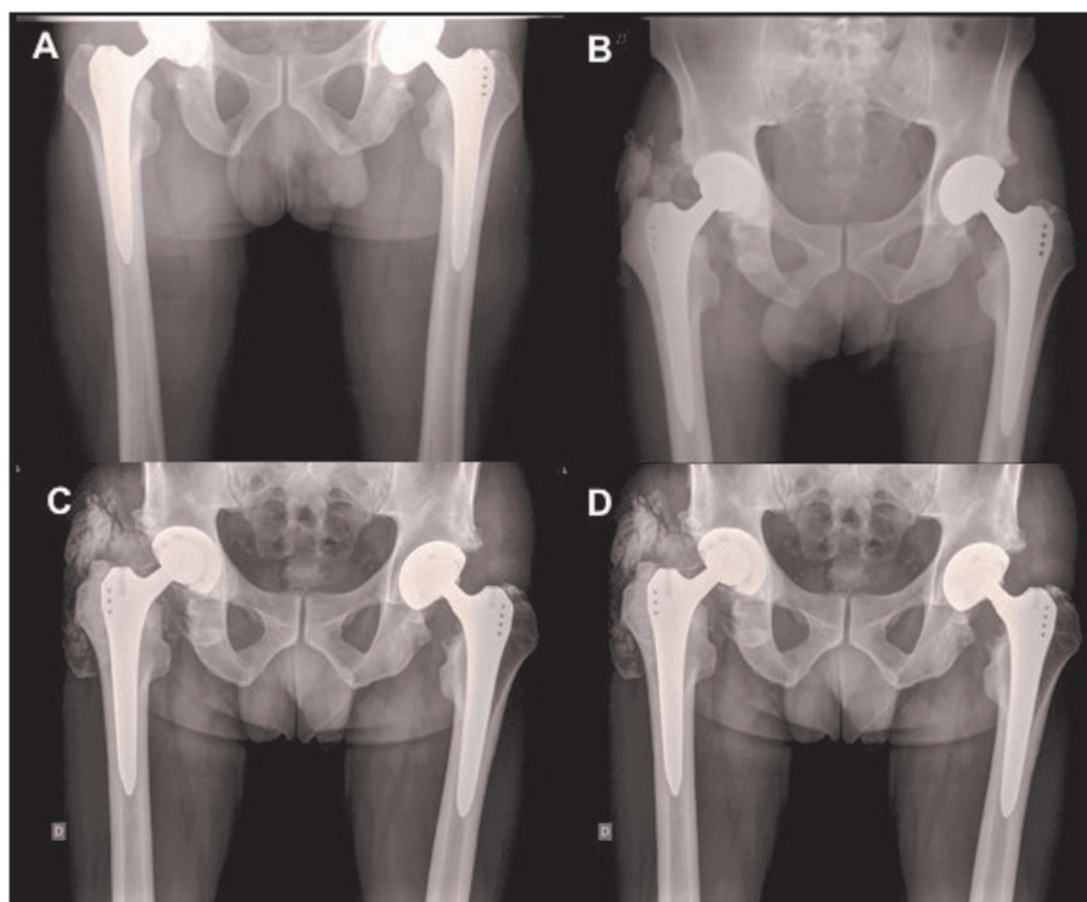
measuring 13 mm. Regarding valve evaluation, the only remarkable finding was a moderate mitral regurgitation.

Several differential diagnoses for the possible causes of cardiomyopathy were considered. Mitochondrial syndromes were not compatible since the lactic acid was normal (0.9 mmol/L; normal range 0.5–2 mmol/L) and the multigene panel test was negative, haemochromatosis was ruled out due to a normal ferritin level (171 ng/mL; normal range 22–322 ng/mL), Sarcoidosis was not very probable since there was no pulmonary affection and a normal ACE level (12.4 U/L; normal range 20–95 U/L) was detected. There was no GLA gene mutation either so Fabry disease was discarded. Lastly, a serum protein electrophoresis test dismissed multiple myeloma.

A right heart catheter showed pulmonary wedge pressure 15 mmHg (normal range 6–12 mmHg), right atrial pressure 5 mmHg (normal range 2–6 mmHg), pulmonary arterial pressure 25/15/18 mmHg (normal range 15–30/8–15/9–18 mmHg), cardiac index 1.7 L/min/m<sup>2</sup> (normal range 2.5–4.0 L/min/m<sup>2</sup>), and pulmonary vascular resistance 1 WU (normal value < 3.125 WU). Preliminary endomyocardial biopsy analysis did not show amyloid deposits but mild perivascular fibrosis was reported.

Considering the orthopaedic history, the abrupt onset and rapid progression of the cardiomyopathy coupled with deafness and the lack of other common causes, serum levels of chromium and cobalt were determined. Both were found to be severely elevated. Chromium levels were 166 µg/L and cobalt levels were 595 µg/L (normal value for a hip prosthesis carrier <7 µg/L and <5 µg/L, respectively). The myocardial sample was tested for metals too, finding high levels of chromium and cobalt (1.96 and 2.15 µg/g, respectively, in the absence of known reference values).

Post-intervention hip X-ray studies ([Figure 2](#)) were reviewed showing progressive development of an increased radiological opacity on the right hip. These findings had been misinterpreted as heterotopic calcifications. A computed tomography was then performed showing intense metallosis at that level ([Figure 3](#)). It was then decided to remove the MoP prosthesis. In September 2019, the surgery was performed uneventfully although extensive metallosis and 'dark grey tissue' in pericapsular and gluteal regions were found. Another total



**Figure 2** Radiological evolution after exchange of broken ceramic on ceramic right hip prosthesis for a metal-on-polyethylene prosthesis surgery. It shows progressive development of an increased radiological opacity surrounding right hip. (A) February 2014, (B) September 2017, (C) February 2019, and (D) June 2019.

hip arthroplasty with a CoC approach was performed (Figure 4). Nevertheless, 2 weeks after the surgery, the patient suffered an electrical storm with several ICD shocks in the setting of multiple episodes of different morphologies ventricular tachycardia.

He was transferred to our critical care unit deteriorating quickly to an INTERMACS 1 situation, leading to an emergent peripheral VA ECMO implantation. Electrical stability was achieved but he showed persistent signs of insufficient LV unloading so an intra-aortic balloon pump was added. However, LV unloading was still inadequate despite high doses of intravenous inotropes and counterpulsation, so a short-term biventricular assist device (BIVAD; Levitronix Centrimag<sup>®</sup>) was implanted, with LV apex to aorta and right atrium to pulmonary artery cannulation.

The first postoperative hours were marked by the impossibility of maintaining appropriate flows with repeated suction events in the left ventricle assist device (LVAD). A severe myocardial oedema and the absence of any native cardiac output were noticed by transoesophageal echocardiography (Figure 5). After fluid administration, endovenous corticosteroids, inhaled pulmonary vasodilators and optimization of the right ventricle assist device speed, LV filling improved, and stable optimal flows were achieved.

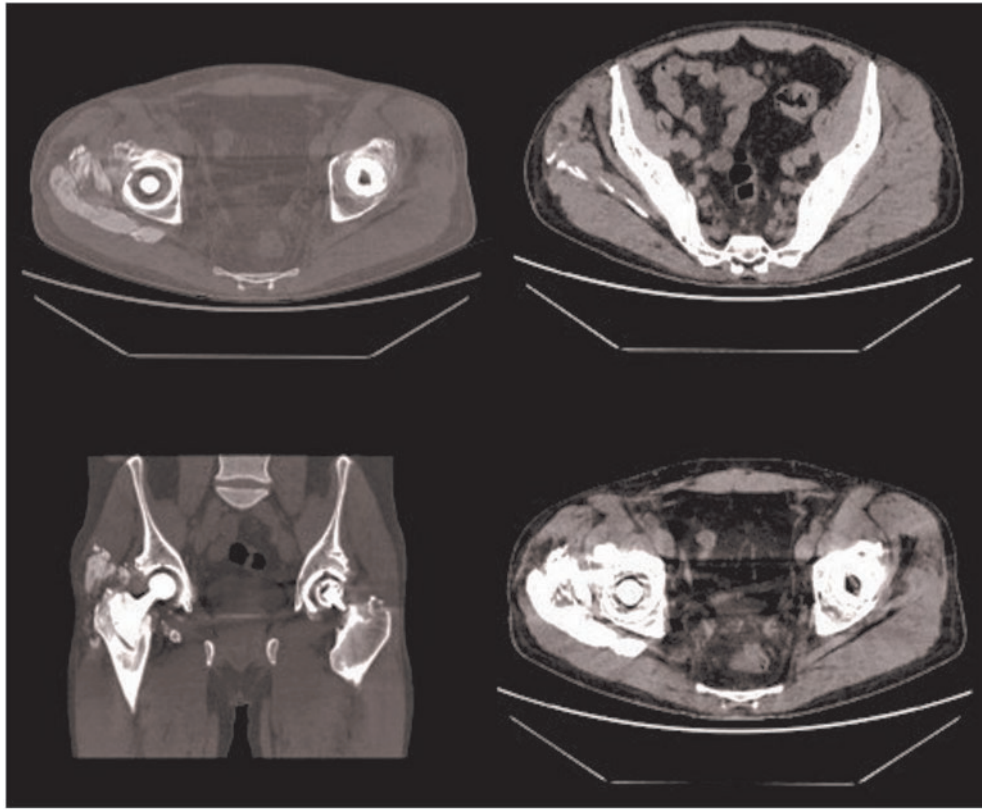
Nevertheless, 3 hours later, BIVAD flow suddenly dropped because of a cardiac tamponade, requiring surgical review for haematoma evacuation and haemostasis. The subsequent evolution was overall favourable.

On the 19th day of support, an important decrease in the chromium and cobalt levels was achieved, measuring 71 and 85  $\mu\text{g/L}$ , respectively (Figure 6). At that point, the patient was feeling very well being capable of doing physical rehabilitation. Humoural sensitization was ruled out and he was listed for heart transplant in an Urgency 0 status. After 23 days of support, the transplant was performed with a bicaval technique. The operation was complicated by severe bleeding secondary to adhesions, but he was weaned from the cardiopulmonary bypass with low doses of inotropes, achieving a normal LV function and moderate right ventricle dysfunction.

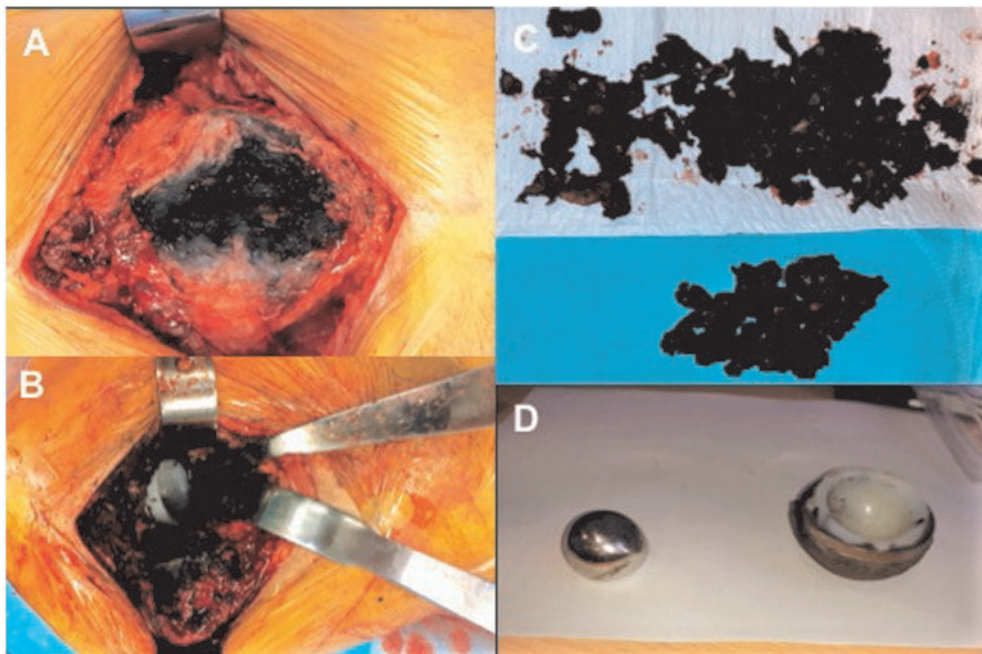
After 8 days, he was discharged to the ward, remaining stable afterwards. There was an improvement in hearing, confirmed by an audiometry. He was discharged 24 days post-operation, with normal biventricular function.

Finally, 5 months after the heart transplant, the patient is doing well, in an NYHA functional class of I, with normal graft function.

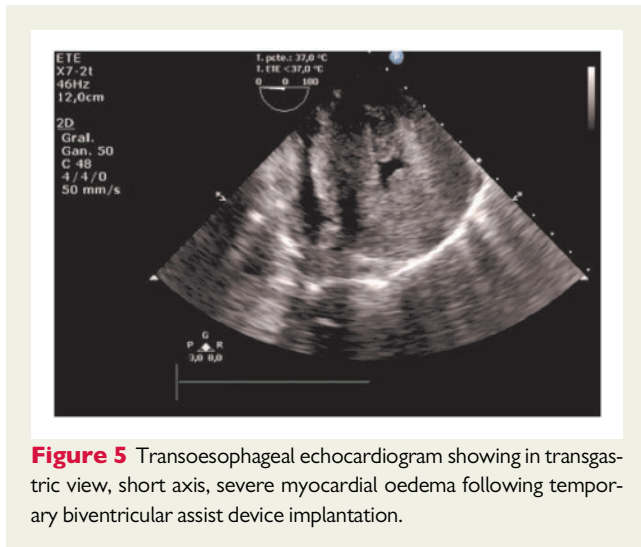




**Figure 3** Computed tomography scan showing radiodense debris surrounding the right hip joint.



**Figure 4** Surgical findings during right hip prosthesis exchange. (A–C) Intense metallosis of the soft tissue adjacent to the prostheses. (D) Corrosion in the polyethylene liner and the surface of the metallic head with loss of sphericity in polyethylene embedded microparticles of ceramic.



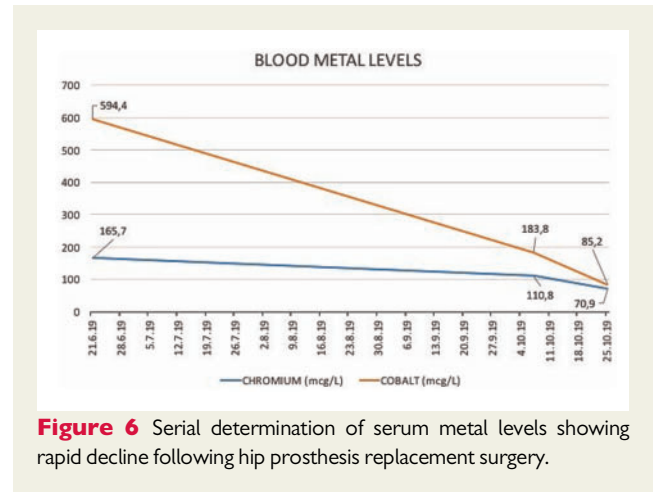
**Figure 5** Transoesophageal echocardiogram showing in transgastric view, short axis, severe myocardial oedema following temporary biventricular assist device implantation.

## Discussion

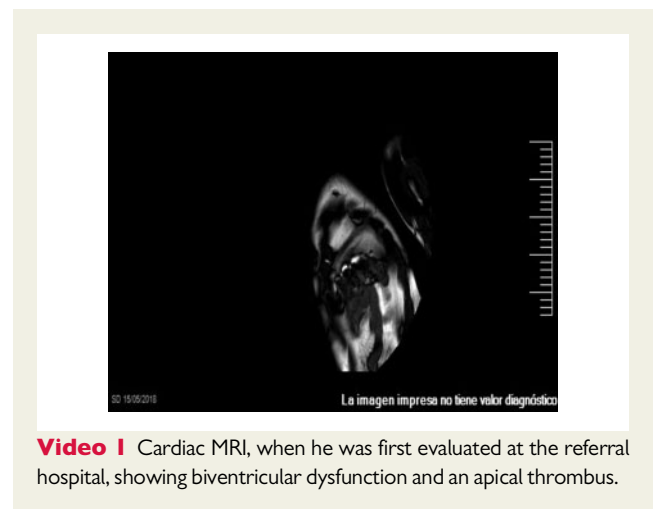
Cobalt cardiomyopathy is an uncommon cause of heart failure and CS. It was first described in patients who were consuming a specific kind of beer that contained an elevated amount of cobalt.<sup>4</sup> The heart involvement of cobalt toxicity usually mimics a hypertrophic or restrictive cardiomyopathy with not very enlarged LV diameters, at least in the first stages, increased left ventricular mass, diastolic dysfunction that progresses to both diastolic and systolic dysfunction and, sometimes, severe pericardial effusion at the diagnosis or during the evolution.<sup>1,5-7</sup> The clinical presentation might be associated with neurological symptoms, polycythaemia, skin problems, sensorineural hearing loss, and hypothyroidism although none of these problems are pathognomonic. The level of suspicion must be high in patients without other previous heart conditions or cardiovascular risk factors who develop a restrictive cardiomyopathy in the presence of an orthopaedic clinical history that supports the diagnosis.

Differential diagnosis should be made with hypertrophic cardiomyopathy and other restrictive cardiomyopathies such as cardiac amyloidosis, sarcoidosis, haemochromatosis, Fabry disease, and mitochondrial disorders. Unfortunately, there are no pathognomonic signs in the image studies although some authors have reported similar findings to the ones usually seen in Amyloidosis in the MRI studies. Mosier *et al.*<sup>8</sup> described diffuse myocardial hyperenhancement of the anterior, lateral, and apical walls with sparing of the base and mid-septum which are concordant with cardiac Amyloidosis but with other differential issues like the blood-pool kinetics during contrast administration and the diffuse oedema noted on T2-weighted MRI. This group also highlights the importance of the MRI to rule out other aetiologies like post-infarct, infiltrative cardiomyopathy, etc. and suggests that in the absence of other causes, the hyper-enhancement and oedema could be strong supportive evidence of cobalt infiltration.

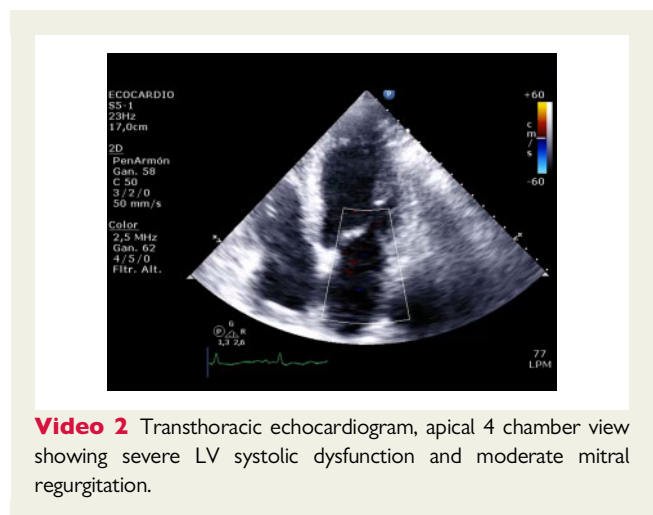
In order to confirm the cobalt cardiomyopathy, some groups suggest the use of endomyocardial biopsy. Myocardial hypertrophy and interstitial fibrosis are common. Other findings such as increased vacuolation and lipofuscin, myofibre disarray and abnormal



**Figure 6** Serial determination of serum metal levels showing rapid decline following hip prosthesis replacement surgery.

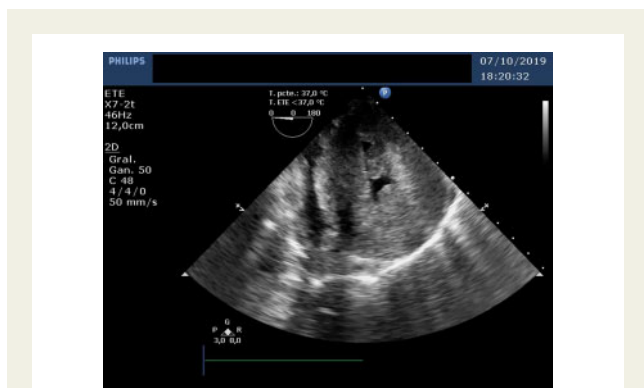


**Video 1** Cardiac MRI, when he was first evaluated at the referral hospital, showing biventricular dysfunction and an apical thrombus.



**Video 2** Transthoracic echocardiogram, apical 4 chamber view showing severe LV systolic dysfunction and moderate mitral regurgitation.

mitochondrial forms with electro dense deposits, have been described as cobalt toxicity-specific features both in biopsies and autopsies.<sup>2,6,8-10</sup>



**Video 3** Transesophageal echocardiogram in transgastric short axis view showing massive LV oedema.

Due to its difficult diagnosis, clinicians should maintain a high rate of clinical suspicion of this entity. Orthopaedic records must be reviewed in patients who present recent onset heart failure and cardiologists must be aware of the risk of metal toxicity when an MoM arthroplasty has been performed.<sup>11</sup> Metal-on-metal arthroplasties began in the 70s and they were supposed to improve stability, reduce osteolysis, and conserve bone.<sup>12,13</sup> In recent years, an increased risk of implant failure and high revision rates secondary to adverse local tissue reactions have been documented, so the use of this technique has decreased significantly.<sup>2</sup>

Most of the patients who carry an MoM prosthesis remain free of toxicity or only develop local reactions. However, a small percentage of them suffer from intense metallosis with variable degrees of different organ involvement. According to some authors, this is particularly frequent when a malposition of the prosthesis is detected.<sup>2</sup>

Interestingly, there has been an increase in concern over metal arthroplasty after ceramic prosthesis fracture in recent years. The patients who get a metal arthroplasty after fracturing their former ceramic prosthesis, seem to have higher blood cobalt levels and are more likely to develop toxicity when compared with MoM patients.<sup>1,3</sup> A possible explanation could be that the broken ceramic prosthesis may produce retained ceramic components that provoke abrasions and destruction of metal head components. This would also explain the higher blood cobalt levels.<sup>1</sup> In this sense, Rambani *et al.*<sup>14</sup> recommend not to perform MoP after a ceramic prosthesis fracture.

Not all the patients who have cobalt toxicity develop a cobalt cardiomyopathy. In fact, to our knowledge, there are only 23 cases of cobalt cardiomyopathy described in the literature.<sup>1</sup> The mean time from the hip surgery to the onset of heart failure is around 2 years and a half.<sup>1</sup> Other toxicity signs are not always present but are more likely to happen when the cobalt levels are higher.<sup>1</sup> Cobalt and chromium blood levels above 7 mg/L, in two determinations separated by 3 months have been established as a threshold to intensive surveillance and to rule out toxicity<sup>11</sup> with a sensitivity and specificity of 52% and 89%, respectively. Other groups recommend a threshold of 5 µg/L with a sensitivity and specificity of 63% and 86%.<sup>13</sup> Some studies pointed out cobalt levels above 100, 250, and 500 µg/L for cardiac, peripheral neuropathy, and thyroid toxicity whereas sensorineural hearing loss would be related with much lower levels.<sup>15</sup>

Replacement of the MoP and debridement of affected tissues must be considered in all patients who develop cardiac toxicity. A CoC prosthesis should be used.<sup>1</sup>

Even when the metal prosthesis is removed, the clinical evolution of the patients who develop cardiac involvement is unpredictable, with some patients achieving normalization of the ejection fraction<sup>16</sup> and others requiring the implant of urgent mechanical circulatory support (MCS) due to CS or an LVAD due to heart failure.<sup>2,8,10,12</sup>

The role of chelation therapy is not clear. Different drugs have been used in this field including N-acetyl-cysteine,<sup>15,17</sup> 2,3-Dimercaptopropane-1-sulfonate<sup>7</sup> and ethylene diamine tetraacetic acid.<sup>5</sup> All these drugs were capable of diminishing the cobalt levels, although the effect on the recovery of the target organs remains controversial. In fact, they are considered as an adjunct therapy and they could also be considered in patients who are not suitable for surgery.

To our knowledge, this is the first case described in literature of a patient with cobalt cardiomyopathy who has suffered an electrical storm needing MCS after the removal of the metal prosthesis. It is not clear whether the cobalt toxicity on the myocardium could be an arrhythmogenic substrate. The myocardial oedema that was seen immediately after the implant of the BIVAD was astonishing and completely unusual. We speculate that this oedema could be secondary to the cobalt infiltration of the myocardium and could be compatible with the MRI findings reported by some groups.<sup>8</sup> This risk should be considered when the cannulation strategy for MCS is discussed in these patients. An atrial cannulation for LVAD inflow could provide a more stable support, at least in selected patients.

In our case, after some initial instability, it was finally possible to maintain an adequate circulatory support and bridge the patient to a heart transplant that was performed once the cobalt and chromium levels decreased. The clinical course after the transplant was positive as previously described by other groups.<sup>18</sup>

In conclusion, cobalt cardiomyopathy is a rare complication of metal hip arthroplasty. It appears to be a restrictive cardiomyopathy that can present as heart failure and CS. It can be accompanied with other signs of systemic toxicity such as deafness, paraesthesia, and polycythaemia. Mechanical circulatory support and heart transplantation are both feasible in this setting. Cardiologists should be aware of this condition and suspect it when treating a patient with an MoM prosthesis or metal arthroplasty after a ceramic prosthesis fracture.

## Lead author biography



I am a cardiologist MD specialized in acute cardiac care and mechanical circulatory support. I work in the Acute Cardiac Care Unit in Hospital Universitario Marqués de Valdecilla in the north of Spain. One of our aims as a team made up of cardiologists, is to emphasize the necessity to lead the acute cardiac care separating cardiac units from the conventional intensive care units. In this sense we have

developed several programmes to train the young cardiologist of the country in this field.

## Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

**Consent:** The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** None declared.

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## References

1. Umar M, Jahangir N, Faisal Khan M, Saeed Z, Sultan F, Sultan A. Cobalt cardiomyopathy in hip arthroplasty. *Arthroplast Today* 2019;**5**:371–375.
2. Martin JR, Spencer-Gardner L, Camp CL, Stulak JM, Sierra RJ. Cardiac cobaltism: a rare complication after bilateral metal-on-metal total hip arthroplasty. *Arthroplast Today* 2015;**1**:99–102.
3. Fox KA, Phillips TM, Yanta JH, Abesamis MG. Fatal cobalt toxicity after total hip arthroplasty revision for fractured ceramic components. *Clin Toxicol* 2016;**54**:874–877.
4. Morin Y, Daniel P. Quebec beer-drinkers' cardiomyopathy: etiological considerations. *Can Med Assoc J* 1967;**97**:926–928.
5. Choi H-I, Hong JA, Kim M-S, Lee SE, Jung S-H, Yoon PW et al. Severe cardiomyopathy due to arthroprosthetic cobaltism: report of two cases with different outcomes. *Cardiovasc Toxicol* 2019;**19**:82–89.
6. Zywił MG, Brandt J-M, Overgaard CB, Cheung AC, Turgeon TR, Syed KA. Fatal cardiomyopathy after revision total hip replacement for fracture of a ceramic liner. *Bone Joint J* 2013;**95-B**:31–37.
7. Pelclova D, Sklensky M, Janicek P, Lach K. Severe cobalt intoxication following hip replacement revision: clinical features and outcome. *Clin Toxicol* 2012;**50**:262–265.
8. Mosier BA, Maynard L, Sotereanos NG, Sewecke JJ. Progressive cardiomyopathy in a patient with elevated cobalt ion levels and bilateral metal-on-metal hip arthroplasties. *Am J Orthop* 2016;**45**:132–135.
9. Moniz S, Hodgkinson S, Yates P. Cardiac transplant due to metal toxicity associated with hip arthroplasty. *Arthroplast Today* 2017;**3**:151–153.
10. Allen LA, Ambardekar AV, Devaraj KM, Maleszewski JJ, Wolfel EE. Clinical problem-solving. Missing elements of the history. *N Engl J Med* 2014;**370**:559–566.
11. Medicines and Healthcare products Regulatory Agency. <https://www.gov.uk/drug-device-alerts/all-metal-on-metal-mom-hip-replacements-updated-advice-for-follow-up-of-patients>. (9 May 2020).
12. Charette RS, Neuwirth AL, Nelson CL. Arthroprosthetic cobaltism associated with cardiomyopathy. *Arthroplast Today* 2017;**3**:225–228.
13. Kwon YM, Lombardi AV, Jacobs JJ, Fehring TK, Lewis G, Cabanela ME. Risk stratification algorithm for management of patients with metal-on-metal hip arthroplasty: consensus statement of the American Association of Hip and Knee Surgeons, the American Academy of Orthopaedic Surgeons, and the Hip Society. *J Bone Joint Surg Am* 2014;**96**:99–102.
14. Rambani R, Kepecs DM, Mäkinen TJ, Safir OA, Gross AE, Kuzyk PR. Revision total hip arthroplasty for fractured ceramic bearings: a review of best practices for revision cases. *J Arthroplast* 2017;**32**:1959–1964.
15. Giampreti A, Lonati D, Raghianti B, Ronchi A, Petrolini VM, Vecchio S et al. N-acetyl-cysteine as effective and safe chelating agent in metal-on-metal hip-implanted patients: two cases. *Case Rep Orthop* 2016;**2016**:1–7.
16. Tilney R, Burg MR, Sammut MA. Cobalt cardiomyopathy secondary to hip arthroplasty: an increasingly prevalent problem. *Case Rep Cardiol* 2017;**2017**:1–4.
17. Luczak MW, Zhitkovich A. Role of direct reactivity with metals in chemoprotection by N-acetylcysteine against chromium (VI), cadmium (II), and cobalt (II). *Free Radic Biol Med* 2013;**65**:262–269.
18. Sanz MI, Rico AM, Moreno A, Bartolomé S, Campo J. Heart transplant secondary to cobalt toxicity alter hip arthroplasty revision. *Hip Int* 2019;**29**:NP1–NP5.