

Open Access

Cobalt Syndrome, a new entity following Hip joint replacement surgery: A first case reported in the Illawarra and Shoalhaven region, NSW, Australia

Abstract

Background: Cobalt poisoning following hip replacement surgery is not much reported in the Australian literature. However, in Australia, there have been notable cases of patients experiencing cobalt toxicity, particularly with the DePuy ASR (articular surface replacement) XL Acetabular Hip System prosthesis containing cobalt and chromium. This prosthesis has been recalled due to these issues [1]. Cobalt toxicity is a potential complication of metal-on-metal (MoM) hip replacement surgeries. Cobalt toxicity has been identified as an emerging clinical problem in patients with metal-on-metal hip prostheses [2]. This issue has been documented in several Australian patients.

Cobalt toxicity can lead to various health problems, including cardiomyopathy, hypothyroidism, lassitude, neuropathy (loss of taste, smell, and hearing), focal and diffuse weakness, and rashes. The Therapeutic Goods Administration (TGA) in Australia has provided guidance and resources for patients with MoM hip implants.

Case presentations: The author presents the first reported case in the Illawarra and Shoalhaven districts of Wollongong, NSW, Australia. The patient is a 71-year-old male who underwent a Stryker ABGII modular hip prosthesis in 2011. Following his surgery, he continued to experience pain and instability, resulting in prosthetic loosening. The prosthesis was recalled in 2012, following which his cobalt levels were checked regularly every two years. The patient had a recurrent left hip dislocation and, in 2021, underwent a revision surgery, following which he gradually improved with hip stability, but his symptoms remained unresolved.

Conclusions: There remains a lack of literature to support the extensiveness of Cobalt-related toxicity despite reported cases in Australia. The author would like to propose a new terminology, "Cobalt Syndrome," instead of using Metallosis and Cobaltism due to the lack of scientific validation of those

Literature Review

Bala Vaidya^{1*}

¹Doctor of Health Science trainee, Campbell University, Raleigh, NC, USA Medical Co-Director, Department of Population and Public Health, Illawarra and Shoalhaven Local Health District, The Wollongong Hospital, Wollongong, NSW

***Correspondence:** Bala Vaidya, Department of Population and Public Health, Raleigh, NC, USA, E-mail: renniez0007@gmail.com

Received: 26 Oct 2024; Accepted: 30 Oct 2024; Published: 06 Nov 2024

Copyright: © 2024 Vaidya B. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

terminologies and to raise awareness within the Australian community on the potential harm secondary to metal on metal hip replacement surgeries done in the last two to three decades.

Keywords

Cobalt poisoning • Cobalt Syndrome • Metallosis • Cobaltism • Metal hip

Introduction

Total hip arthroplasty (THA) is a widely accepted standard procedure to treat hip osteoarthritis. Over the last two to three decades, over one million patients underwent the procedure yearly with hip resurfacing devices with MoM articulation until an official recall was initiated around 2012.

The advantages of MoM articulations include their tolerance of high-impact loading, decreased wear rates, and increased stability provided by the larger head size. Exposure to metal ions after hip arthroplasty surgery is a widely.

reported phenomenon. Multiple studies have shown that increased metal ions can result in local soft tissue reactions, described as adverse reactions to metal debris [3]. The components ultimately had significantly higher revision rates, up to three times those of contemporary hip implants, with a significant decline in their use. Many of these failures were attributed to the breakdown and disintegration of metal debris, resulting in adverse local tissue reactions and potential systemic side effects due to Cobalt metal breakdown [4]. Some reported literature states tissue necrosis, osteolysis, and sterile hip effusion result in significant hip instability and prosthetic loosening.

Mechanism of Metal Disintegration

Initially, it was hypothesized that the result of the Metal-on-Metal articulation was the problem. There were increasing implications for nonmalignant and non-infectious masses in other THA articulations, including metal-on-polyethylene ceramic-on-polyethylene bearings. This and was eventually recognized due to the likely result of metal ion release from the modular junctions. Subsequently, it was recognized that increased cobalt concentrations are often seen after implanting metal-on-metal hip bearings, and this can release ions from the metal (cobalt-chromium) surface either directly (corrosion) or during sliding under load, which may create wear particles (adhesion). Another significant metal particle release source is applying a metal component to revise a fractured ceramic head and/ or a fractured ceramic acetabular liner. In this scenario, massive three-body abrasive wear can be created, as small remaining particles of the fractured ceramic bearing lead to abrasion of the metal surface [5].

Pathophysiology of Cobalt Syndrome

Cobalt disintegration following hip replacement surgery, particularly with metal-on-metal (MoM) implants, can lead to cobalt toxicity. Here's a brief overview of the pathophysiology:

Wear and tear: Over time, the metal components of the hip implant can wear against each other, releasing tiny particles of cobalt and chromium into the surrounding tissues [6].

Inflammatory response: The body's immune system reacts to these metal particles, leading to inflammation. This can

cause pain and swelling around the implant site.

Systemic absorption: Cobalt particles can enter the bloodstream, leading to elevated blood cobalt levels. This systemic absorption can cause various symptoms, including neurological, cardiovascular, and thyroid issues.

Cellular damage: Cobalt-induced reactive oxygen species (ROS) production can lead to cellular damage through apoptosis, autophagy, necrosis, and mutation. This cellular damage can contribute to osteolysis (bone loss) and aseptic implant loosening [7].

Clinical Manifestations

The literature reported clinical toxicity with systemic involvement, including gastrointestinal, neurocognitive, and psychological dysfunction with mood swings and endocrinal abnormalities, in particular, thyroid dysfunction, tinnitus, vertigo, hearing loss, and visual disturbances. Some literature reported cases of increased cancer secondary to chromium leaks. There have been reported cases of cardiotoxicity resulting in cardiac failure. Other neurological manifestation reported includes headaches, convulsions, and peripheral neuropathy. All these systemic manifestations are called (PHACT) Post-hip replacement Cobalt, and chromium toxicity). Given that heavy metal toxicity is widely reported in Industrial hazards and the terminology is coined to reflect the toxicity of the source heavy metal, we do not have widespread accepted medical literature on the definition and classification of this disorder. resulting in underdiagnosis and poor recognition. On that note, the author recommends the term 'Cobalt Syndrome," which encompasses all the systemic side effects of Cobalt toxicity.

Index reported case

The author presents a case where a 71-year-old male with a background history of severe Left hip Osteoarthritis underwent a Left Total Hip Replacement surgery in 2011 with a Stryker ABG II Modular hip prosthesis. Postoperatively, he continued to have left hip pain intermittently, which had progressively worsened over the next 12 months. The operating surgeon received a recall notification in 2012, following which the patient was called to have further investigations by checking Cobalt levels and X-rays of his left hip. The blood tests were carried out regularly, and the patient was observed without further revision surgery. The patient gradually developed left hip instability secondary to muscle weakness around his hip abductors and extensors. He also developed hearing impairment and progression of his left hip pain. Further blood samples were taken, which showed high levels of Cobalt in 2019. The patient had also reported several left hip dislocations, which he was able to relocate without severe pain. Around 2021, the orthopedic surgeon revised his left hip by removing the metal prosthesis and putting a ceramic polyethylene component.

The patient was recommended to undergo strict surveillance and restricted range of movement for six to 12 months. After revision surgery, the patient felt stability around his left hip. However, he is left with chronic left hip pain, left hip abductor muscle wasting, increased left leg lengthening, requiring shoe rise, inability to walk long distances, restriction with functional activities of daily living around his domestic chores, restriction with his avocational interests like rock pool fishing, playing basketball, skiing, etc. The patient had also reported sexual dysfunction, but currently, there is no literature to support sexual dysfunction secondary to cobalt toxicity. The patient continues to work in a limited capacity in an administrative role, but he states that his life has changed entirely after his initial left hip replacement surgery. This is the first reported case of Cobalt toxicity secondary to hip replacement surgery in the districts of Illawarra and Shoalhaven, and more cases may be reported in the following years. There is a general lack of awareness within the medical fraternity and underdiagnosis of this problem, resulting in inappropriate investigations and treatment, poor patient outcomes, and poor healthcare cost utilization.

The author aims to provide further education and information about this condition and raise awareness among primary care physicians and medical professionals for a targeted assessment and recommendation. The other aim is to protect the population from harm by applying the principles of Nonmaleficence and the code of ethics.

Discussion

The terminology used to describe Cobalt-related side effects reported in the literature are "Cobaltism, Metallosis, Cobalt toxicity, etc." In the author's view, this is not getting much traction within the medical fraternity, given the lack of clarity in defining a problem based on signs and symptoms.

On that note, the author wishes to propose terminology based on Cobalt-associated signs and symptoms, such as "Cobalt Syndrome." The author's hypothetical view is that by defining a clinical condition with specific signs and symptoms correlating with etiology and pathophysiology, the medical condition will be recognized more as an entity and receive more attention and awareness.

This will promote the knowledge base, understanding, and appropriate, timely diagnosis and treatment intervention it deserves.

The Index case reported here underwent his first Left replacement surgery in 2011. Following post-operative persistent pain and recall from the manufacturer of the Left hip prosthesis in 2012, he was under surveillance and had regular blood samples to check for Cobalt level and an MRI of his left hip. The patient was assessed by the Orthopedic surgeon in 2018; at that stage, his cobalt level was assessed to be 121 (Normal is below 1.8mcg per liter), and it was compared to the sample done in November 2016, which was 116, and the previous level 12 months ago was 104, and MRI hip done in 2015 showed no erosion, granuloma or pseudotumor, but there were local erosive changes.

The MRI done in 2016 showed some capsular thickening and effusion, ALVAL changes, and metallic debris. It appears that 5 years from the date of surgery, radiological changes start appearing, although the cobalt serum level precedes radiological changes. The patient underwent a revision of his left THR in May 2021. At the time of the surgery, the Serum Cobalt level was assessed to be 149, and an MRI of the left hip revealed worsening metallosis.

The operative notes stated that the perioperative inspection of the hip revealed significant metallosis with an ALVAL lesion. The posterior hip capsule, gluteus minimus, and hip external rotators were utterly absent. The femoral head was posteriorly dislocated, and the head and neck of the femur were dislocated from the stem. There was significant bone loss at the proximal part of the femoral stem. The femoral stem was mobilized with a combination of burr and flexible osteotome maneuvers, and the stem was delivered. The shaft was then reamed accordingly to accommodate a size 13 Corail stem. A bone graft was performed with a morcellised autograft, and the stem was impacted in place with slight anteversion to create an excellent seating position. The neck was fitted with a Delta ceramic head. The patient was recommended to remain protected for 6 weeks, gradually progressing to weightbearing as tolerated. The patient attained a full range of hip motion and regained ambulation without any gait aid. However, the consequences from initial surgery with left hip pain, muscle wasting with hip gluteal abductors, and external rotators remained despite targeted rehabilitation programs. This explains the long-term poor outcomes secondary to focal cobalt and chromium metallic ion destruction of bones and soft tissues.

Conclusion

Cobalt-related local and systemic complications following hip replacement surgery remain underdiagnosed and underreported, at least within the Australian perspective, and there is very limited literature reported in Australia. Various reported cases described symptoms complex affecting local tissue reactions resulting in chronic nociceptive pain, hip instability, secondary muscular degeneration, cardiac side effects, neurological side effects including neuropathy and cognitive changes, thyroid disorders, and visual and auditory side effects. Pathophysiology is secondary to the degradation of the cobalt metal, resulting in the release of metallic ions into the local tissue and systemic absorption, resulting in medium- to long-term side effects that affect various systems.

The diagnostic challenge is due to underreporting and decreased awareness within the medical fraternity. Blood and urine cobalt samples supported by clinical and radiological features remain the cornerstone for early diagnosis and intervention. It is unclear at what stage is considered early to intervene, but a robust retrospective study to look at interventions taken for revision over the last decade or two would shed some light on this.

Finally, the author believes that Cobalt-related clinical signs and symptoms deserve better recognition in medical literature and proposes a syndrome complex for cobalt-related complications, such as "Cobalt Syndrome." The awareness of this condition will increase once it is appropriately defined, categorized, and evaluated based

on clinical, radiological, and pathological investigations. More evidence-based research is required to investigate the full scope of cobalt-related toxicity in the long-term following hip replacement surgeries. However, the author hereby reports this first case in the region of Illawarra and Shoalhaven and hopes to raise awareness of the clinical syndrome for future years.

Acknowledgment

The author would like to acknowledge the traditional landowners of Dharawal and Yuin aboriginal communities past, present, and emergingand the Illawarra and Shoalhaven Health District.

Secondly, the author would like to acknowledge the patient who was willing and provided written informed consent to write about his clinical situation.

Thirdly, the author would like to thank the legal firm, Mr. John Potter Solicitor, principal of Commins Hendriks Law firm in Waga Wagga, NSW, Australia, who referred this patient for a medico-legal assessment.

Funding

Not applicable.

Availability of Data and Materials

Not applicable.

Declarations

Ethics Approval and Consent to Participate

Ethics approval was not applicable.

The patients gave written informed consent to publish these case reports and any accompanying images.

Competing Interests

The authors declare that they have no competing interests.

References

- 1. Mao, Xinzhan, Andrew A. Wong, and Ross W. Crawford. "Cobalt toxicity-an emerging clinical problem in patients with metal-on-metal hip prostheses." Med J Aust 194, no. 12 (2011): 649-651.
- Gessner, Bradford D., Thomas Steck, Erik Woelber, and Stephen S. Tower. "A systematic review of systemic cobaltism after wear or corrosion of chromecobalt hip implants." Journal of patient safety 15, no. 2 (2019): 97-104.
- Crutsen, J. R.W, M. C. Koper, J. Jelsma, M. Heymans, et al. "Prosthetic hip-associated cobalt toxicity: a systematic review of case series and case reports." EFORT Open Reviews 7, no. 3 (2022): 188-199.
- Venkatraman, Vishal, Megan K. Wong, Chidyaonga Shalita et al. "Cobalt-induced toxicity and spasticity secondary to hip arthroplasty: case report and review of the literature." Cureus 12, no. 12 (2020).

- Kim, Chul-Ho, Young Hyun Choi, Mi Yeon Jeong, Jae Suk Chang et al. "Cobalt intoxication heart failure after revision total hip replacement for ceramic head fracture: a case report." Hip & pelvis 28, (2016): 259-263.
- Peters, Rinne M., Pax Willemse, Paul C. Rijk, Mels Hoogendoorn, and Wierd P. Zijlstra. "Fatal Cobalt Toxicity after a Non-Metal-on-Metal Total Hip Arthroplasty." Case reports in orthopedics 1 (2017): 9123684.
- 7. Mastel, M., A. Boisvert, R. Moore, F. Sutherland, and J. Powell. "Metallosis following hip arthroplasty: two case reports." Journal of Medical Case Reports 16(2022):115.

Citation: Bala Vaidya. "Cobalt Syndrome, a new entity following Hip joint replacement surgery: A first case reported in the Illawarra and Shoalhaven region, NSW, Australia." J Healthc Adv Nur (2024): 117. DOI: 10.59462/JHAN.2.3.117