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Cobalt-Induced Ototoxicity Treated With Corticosteroids, Chelation, and Hyperbaric Oxygen Therapy

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Abstract

Systemic cobalt toxicity, including sensorineural hearing loss, may occur in patients with cobalt-chromium (CoCr) hip prostheses. This report describes a 45-year-old woman who underwent unilateral total hip revision surgery due to mechanical failure of her original implant. Intraoperative findings included fractures of her original ceramic prosthesis that were debrided. A new CoCr dual mobility liner and femoral head were implanted. Within a few months postoperatively, she developed sensorineural hearing loss and was diagnosed with markedly elevated plasma cobalt and chromium concentrations. She underwent a second revision procedure, but her hearing loss persisted. After treatment with prednisone, succimer, N-acetylcysteine (NAC), and hyperbaric oxygen therapy, her hearing acuity improved. For patients with arthroprosthetic cobalt toxicity, prosthesis explantation and revision are primary treatments that often result in symptom improvement, but patients who experience persistent ototoxicity may benefit from additional therapies to improve symptoms and reduce the systemic cobalt burden.

Categories: Otolaryngology, Emergency Medicine, Orthopedics
Keywords: arthroplasty, cobalt toxicity, hyperbaric oxygenation, poisoning, sensorineural hearing loss

Introduction

Cobalt-chromium (CoCr)-containing hip implants are commonly associated with systemic cobalt exposure that often results from chronic corrosion of the prosthesis [1]. Infrequently, systemic cobalt exposure may also be precipitated by third-body wear of CoCr implants that occurs when debris in the joint space becomes trapped within the articular surface, leading to abrasion of cobalt-containing prosthetic components [2]. Patients with cobalt exposure related to CoCr implants are frequently asymptomatic, but systemic toxicity can also occur [3]. Cardiotoxicity, peripheral neuropathy, including ototoxicity, and thyroid dysfunction are common manifestations of cobalt toxicity [4]. Cobalt-related ototoxicity, including sensorineural hearing loss and tinnitus, may remain permanent for some individuals, even after revision surgery is performed [5]. We present a rare case of a patient with severe cobalt toxicity related to third-body wear of a CoCr hip implant who experienced improvements in hearing acuity after hip revision surgery and treatment with prednisone, chelation therapy, and hyperbaric oxygen therapy. Written informed consent for case report publication was obtained from the patient.

Case Presentation

A 45-year-old woman with a history of hypertension, mitral valve prolapse, and congenital hip dysplasia underwent unilateral total hip revision arthroplasty due to mechanical failure of her original joint implant. Intraoperative findings included fractures of the original ceramic implant, which were debrided. A new CoCr dual mobility acetabular liner and femoral head were implanted. The patient experienced progressive hip pain postoperatively, and subsequent hip aspirations yielded black-colored fluid. Four months after the revision arthroplasty, she experienced two episodes of vertigo, followed by sudden-onset bilateral hearing loss one month later (Table *1*).

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2003	Initial total hip arthroplasty surgery (ceramic components)
October 2024	First left total hip revision surgery (CoCr dual mobility acetabular liner and femoral head)
February 2025	Developed vertigo
March 2025	Developed bilateral hearing loss; audiogram revealed bilateral mild sloping to moderately severe sensorineural hearing loss
April 2025	Initial plasma cobalt (991 mcg/L) and chromium (89.8 mcg/L) concentrations obtained
May 2025	Second left total hip revision surgery (polyethylene liner and ceramic femoral head)
May 2025	Initiation of chelation therapy with succimer (10 mg/kg three times daily for 5 days, followed by 10 mg/kg twice daily for 14 days); initiation of hyperbaric oxygen therapy
July 2025	Repeat plasma (44.2 mcg/L) and whole-blood (288 ng/mL) cobalt concentrations obtained; N-acetylcysteine administered; subjective improvement in hearing acuity noted
August 2025	Completion of final hyperbaric treatment; repeat audiogram revealed mild to moderate bilateral sensorineural hearing loss
April 2026	Repeat plasma (7.8 mcg/L) and whole-blood (10.6 ng/mL) cobalt concentrations obtained
August 2026	No further reported decrements in hearing acuity

TABLE 1: Chronological timeline of events

CoCr, cobalt-chromium

An audiogram revealed bilateral mild sloping to moderately severe sensorineural hearing loss (Figure *1*).

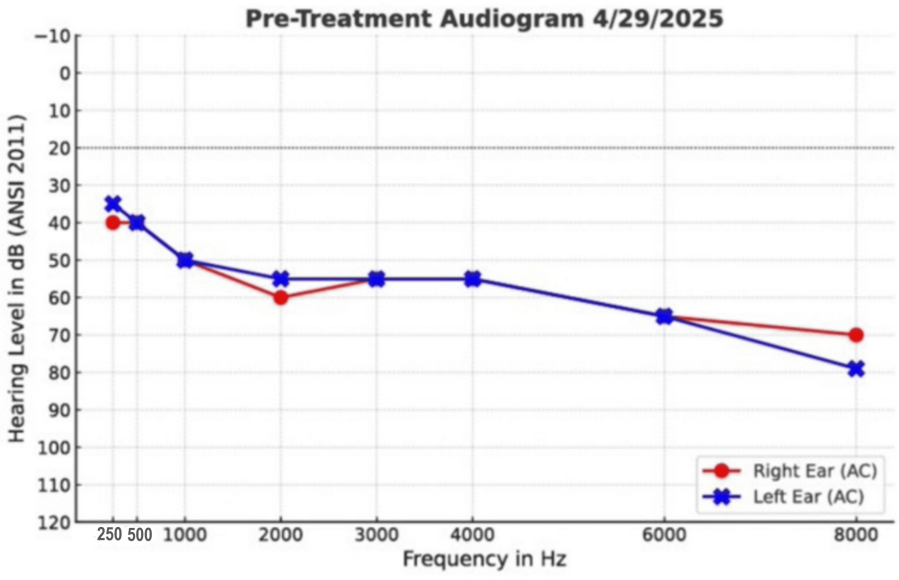


FIGURE 1: Pre-treatment audiogram

Pre-treatment audiogram demonstrating bilateral mild to moderately severe sensorineural hearing loss.

ANSI, American National Standards Institute

The patient obtained hearing aids due to the severity of her hearing impairment. Due to the persistence of

her symptoms, she underwent a toxicology laboratory assessment six months postoperatively that revealed significantly elevated plasma levels of cobalt (991 mcg/L, range: <0.9 mcg/L) and chromium (89.8 mcg/L, range: <4.8 mcg/L). A diagnosis of metallosis and cobalt toxicity was established, and urgent explantation and revision of the CoCr hip implant was performed with placement of a polyethylene liner and ceramic femoral head. Intraoperatively, significant metallosis was noted; this may have been related to third-body wear of the metallic implant surfaces from microscopic ceramic particles that remained after her initial revision surgery. The patient was referred for medical toxicology and otolaryngology evaluations due to cobalt and chromium toxicity with associated sensorineural hearing loss.

At the time of her initial medical toxicology and otolaryngology evaluations, the patient reported having persistent bilateral hearing loss that had not improved in the month since the explantation surgery, even though her plasma cobalt level had decreased precipitously postoperatively to 130.6 mcg/L (with a whole-blood cobalt concentration of 537.5 mcg/L). She worked as a schoolteacher, did not smoke cigarettes, and occasionally consumed ethanol. She denied use of dietary supplements and had no identifiable recreational or occupational exposures to cobalt or chromium. Her physical examination, including neurological examination, was unremarkable aside from the hearing loss. A brain MRI revealed potential mineralization in the bilateral basal ganglia, possibly related to cobalt toxicity. Evaluations for other systemic manifestations of cobalt toxicity were unrevealing. The patient's left ventricular ejection fraction was 50-55%, or within the normal range, on transthoracic echocardiography, with mild right ventricular dilation and hypertrophy noted. Thyroid function, as measured by thyroid-stimulating hormone (TSH), was within normal limits (4.2 mcIU/mL, range: 0.45-4.5 mcIU/mL). A 19-day course of chelation therapy with oral succimer was prescribed to enhance elimination of her systemic cobalt and chromium burden. For treatment of her sensorineural hearing loss, she was prescribed oral prednisone and began hyperbaric oxygen therapy in a monoplace hyperbaric chamber that involved daily pressurization to 2.5 absolute atmospheres for 90 minutes. She tolerated these treatments without complications. Near the completion of her prednisone taper and initial course of succimer, and after completion of 15 hyperbaric treatments, the patient experienced subjective improvements in her hearing acuity. A repeat audiogram, performed the following week, demonstrated 10-15 dB improvements in hearing acuity at lower frequencies, although her hearing loss remained moderately severe at the highest frequencies (Figure *2*).

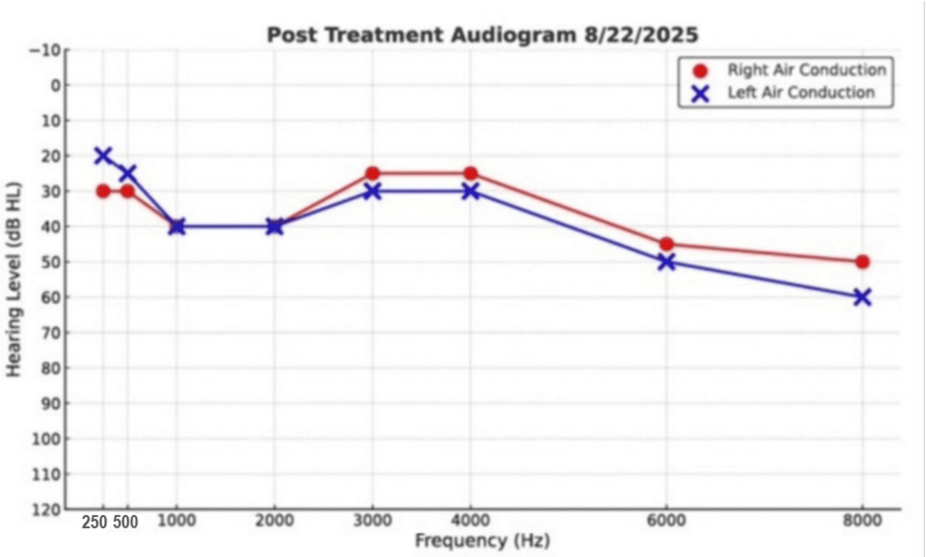


FIGURE 2: Post-treatment audiogram

Post-treatment audiogram demonstrating improvements in bilateral hearing acuity.

Her whole-blood cobalt and plasma cobalt concentrations continued to decline two months after explantation surgery (288 and 44.2 mcg/L, respectively), and her whole-blood and plasma chromium concentrations also decreased during that time. A 21-hour course of N-acetylcysteine (NAC) was administered to enhance cobalt and chromium excretion. She was able to discontinue use of her hearing aids and did not experience further decrements in her hearing acuity. A repeat audiogram performed three weeks after the conclusion of 34 hyperbaric treatments revealed mild to moderate sensorineural hearing loss bilaterally. Her hearing improvements remained sustained over the next year, with no further subjective hearing impairments reported. One year after the second explantation and revision surgery, her whole-blood and plasma cobalt concentrations remained detectable (10.6 and 7.8 mcg/L, respectively).

Discussion

Orthopedic prosthetics are often crafted from metal alloys that contain cobalt, chromium, or molybdenum [6]. The physical properties of these alloys, including resistance to wear, corrosion, and heat, make them biocompatible and suitable for use as long-term implants [7]. Although orthopedic prostheses are generally considered to be biologically inert, the release of metal ions from these implants can occur over time due to ongoing friction, corrosion, or loosening of individual components [4]. Systemic exposure to these metal ions, including chromium and cobalt, may result in significant human toxicity, including signs and symptoms of arthroprosthetic cobaltism. Systemic chromium exposure may also occur in patients with orthopedic implants, but this is typically in the form of trivalent chromium, which is less toxic than the hexavalent state of that metal [4].

Patients affected by arthroprosthetic cobaltism may experience cardiac compromise, endocrine dysfunction, and peripheral neuropathy, including ototoxicity. Ototoxicity is a common finding in patients with systemic cobaltism related to metal-based hip implants, affecting up to 50% of this patient population [8]. The clinical manifestations of cobalt-associated ototoxicity are varied and may include sensorineural hearing loss, tinnitus, and imbalance [9,10]. While some systemic effects of cobalt toxicity are reversible, ototoxicity associated with cobalt exposure is often permanent in nature [4,5].

The pathophysiology of cobalt-induced ototoxicity has been investigated in both human and animal studies. Cobalt is directly cytotoxic to neural cells and affects multiple organ systems, mimicking mitochondrial cytopathy [11]. In a study involving rabbits, chronic cobalt and chromium exposure resulted in a time- and dose-dependent reduction of spiral ganglion cells, degeneration of the organ of Corti, and loss of sensory hair cells [12]. Similar findings were noted in an in vivo investigation of cobalt exposure in rat postnatal cochlear organotypic cultures; in that study, cobalt exposure caused damage to the hair cells and spiral ganglion neurons [13]. Human studies involving the ototoxicity of cobalt have evaluated both the auditory and vestibular sequelae of cobalt intoxication. Leyssens et al. (2020) determined that cobalt exposure is associated with hearing decrements at higher frequencies in humans, similar to previous reports of drug-induced ototoxicity involving platinum-based chemotherapy and aminoglycosides, although the results of vestibular outcome testing were inconclusive [9]. In the case reported herein, the patient's hearing decrements were greatest at the higher frequencies, consistent with the previously reported data.

In patients with systemic cobaltism related to metal-based joint implants, explantation of the affected prosthesis removes the source of heavy metal exposure and is typically followed by a precipitous decline in systemic cobalt burden as well as symptom improvement [4]. Despite the significant decrease in the patient's blood cobalt concentration that soon occurred after explantation in the patient described in this case, a similar rapid clinical improvement (i.e., hearing recovery) did not occur, and thus additional treatments were considered. NAC may reverse the cytotoxicity induced by cobalt and has been utilized as a treatment for patients with cobalt intoxication [14,15]. However, the systemic bioavailability of oral NAC is low, ranging from 6-10%, and for that reason it was not utilized as a primary treatment for this patient [16]. Oral and intratympanic corticosteroid administration and hyperbaric oxygen therapy are standard therapeutic interventions for patients with idiopathic sudden sensorineural hearing loss and were utilized in the management of this patient due to her lack of symptom improvement after explantation. Hyperbaric oxygen therapy, which involves the systemic breathing of 100% oxygen by patients enclosed in a pressurized treatment vessel, may promote hearing recovery through modulation of antioxidant enzyme activity, cochlear tissue hyperoxygenation, and reduction of inflammation in patients with idiopathic sudden sensorineural hearing loss [17]. In patients with cobalt toxicity, the cochlear hyperoxygenation induced by hyperbaric oxygenation may also mitigate the cytotoxic effects of cobalt on the inner ear. Although it has not been previously reported as a treatment for cobalt-induced hearing loss, the hearing recovery experienced by the patient in this report after receiving hyperbaric oxygenation suggests that the elevated oxygen gradients induced by this treatment may result in favorable outcomes for patients with cobalt-induced ototoxicity.

Hyperbaric oxygen therapy is an accepted adjunctive treatment for idiopathic sudden sensorineural hearing loss, and the patient described in this case experienced improvements in her hearing acuity after receiving hyperbaric oxygen therapy, oral prednisone, and chelation therapy with succimer. While hyperbaric oxygen therapy was utilized primarily for the management of this patient's sensorineural hearing loss, it is possible that this therapeutic intervention may also have a role in the treatment of cobalt toxicity, even in the absence of hearing loss or vestibular symptoms, although this association remains experimental at this time and requires further study. Cobalt induces mitochondrial oxidative stress similar to hypoxia, leading to cellular toxicity and apoptosis [18]. The systemic hyperoxygenation induced by hyperbaric oxygen therapy may counteract the hypoxia-like effects of cobalt exposure and limit the systemic toxicity of this element. Hyperbaric oxygenation is currently not a recognized standard treatment for cobalt intoxication, and further investigations are needed to define the potential clinical uses of hyperbaric oxygen therapy in patients with manifestations of cobalt intoxication.

In the case described above, the improvements in the patient's hearing acuity were noted after she had received multiple treatments, including corticosteroids, chelation, and hyperbaric oxygenation. It is unclear which, if any, of these interventions were the most effective in restoring her hearing, and it is also possible that the patient's hearing acuity would have resolved independently in the absence of specific treatments. Since the ototoxic effects of cobalt are known to be directly related to the dose and length of exposure, the

physicians treating this patient chose an aggressive treatment approach with the hopes that multimodal therapeutic interventions would more effectively reduce her systemic cobalt burden and duration of exposure.

Conclusions

Cobalt intoxication induces hypoxia-like cytotoxicity that results in cochlear damage and loss of sensory hair cells. Ototoxicity associated with cobalt poisoning may cause vestibular symptoms in addition to sensorineural hearing loss predominantly affecting higher frequencies. Cobalt-induced ototoxicity is time- and dose-dependent, suggesting that effective reduction in the systemic cobalt burden may result in a more favorable clinical outcome. The outcome presented in this case indicates that hearing recovery is possible in patients with cobalt-induced ototoxicity, even when systemic cobalt concentrations are markedly elevated. As evidenced by this case, patients with cobalt-induced ototoxicity may require a multidisciplinary treatment approach, including surgical explantation of cobalt-containing devices, chelation therapy, corticosteroids, and hyperbaric oxygen therapy, although further investigations are needed to define the exact roles and clinical utility of each of these interventions.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kelly Johnson-Arbor, Hung Jeffrey Kim

Acquisition, analysis, or interpretation of data: Kelly Johnson-Arbor, Daniel Swanson, Hung Jeffrey Kim

Drafting of the manuscript: Kelly Johnson-Arbor, Daniel Swanson, Hung Jeffrey Kim

Critical review of the manuscript for important intellectual content: Kelly Johnson-Arbor, Daniel Swanson, Hung Jeffrey Kim

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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