The Journal of International Advanced Otology

J Int Adv Otol 2019; 15(3): 459–62 • DOI: 10.5152/iao.2019.6891

Case Report

Cochlear Implantation after Bromate Intoxication–Induced Bilateral Deafness: A Case Report

Choi Sung-Won, Cho Youngmo

Department of Otorhinolaryngology, Pusan National University Hospital, Busan City, Korea, Republic Of (CSW)
Department of Emergency Medicine, Pusan National University Hospital, Busan City, Korea, Republic Of (CY)

ORCID IDs of the authors: C.S.W. 0000-0002-7463-7720; C.Y. 0000-0002-1840-3305.

Cite this article as: Sung-Won C, Youngmo C. Cochlear Implantation after Bromate Intoxication-Induced Bilateral Deafness: A Case Report. J Int Adv Otol 2019; 15(3): 459-62.

Hearing loss is a common consequence of the strong acidosis induced by bromate poisoning. Partial hearing recovery has been achieved through medical or rehabilitative therapy but reports of surgical otology treatment for this condition are rare. We report the case of a 48-year-old female patient who underwent cochlear implantation after bromate intoxication had induced bilateral deafness. In cases with life-threatening renal damage, the diagnosis of hearing loss is sometimes delayed, but in our case, hearing impairment was unavoidable despite early detection of symptoms and early disruption of the use of diuretics that could cause hearing damage. Hearing loss 12 hours after bromate ingestion was successfully reversed through cochlear implantation (CI) six months after completing acute phase treatment, including dialysis for acute kidney injury. The benefit of CI for deafness by bromate intoxication is highlighted by this case.

KEYWORDS: Cochlear implantation, hearing loss, bromates

INTRODUCTION

Sodium bromate (hereafter, "bromate") is a strong oxidizing agent. It is commonly used as a neutralizer for permanent hair wave agent. Bromate intoxication predominantly results from deliberate ingestion by patients who are suicidal [1]. Several intoxication cases have been reported in Western countries due to accidental ingestion by children [2]. Recently, bromate has been replaced by the less toxic agents such as sodium perborate, sodium hexametaphosphate, and hydrogen peroxide. Although still used by professional hairdressers, bromate is not available for home use [3].

Bromate poisoning results in symptoms of the gastrointestinal (GI) tract, and central and peripheral nervous system, acute renal injury (AKI), and hearing loss [4-7]. Only two of these clinical manifestations are known to be irreversible: AKI and hearing loss [2-4, 6]. In particular, hearing impairment due to bromate occurs in most cases, progresses very fast, and leads to severe to profound bilateral sensorineural hearing loss.

Cochlear implantation (CI) has been effective in treating hearing impairment due to bromate ingestion [2, 8] and the use of ototoxic therapeutic agents [9, 10]. We report treatment of bromate-induced deafness with CI, and use postoperative audiometry to actively assess this method as a general option to treat the irreversible hearing loss caused by bromate. The long-term outcome that we aimed for was to improve the quality of life for this patient, and inform future decisions for other patients with this condition based on our experience with this case.

CASE PRESENTATION

A 48-year-old female patient presented at the emergency room at 10:00 pm complaining of vomiting and diarrhea that started 1 h after drinking approximately 180 cc of permanent neutralizer solution. Physical examination on her arrival showed no abdominal abnormality except for mild tenderness and increased bowel sound. In the initial head, ears, eyes, nose, and throat examination, hearing was preserved without any abnormality and communication was normal. Vital signs were as follows: blood pressure, 110/70 mmHg; pulse, 109 bpm; respiration, 18/min; body temperature, 36.1°C; and saturation, 98% at room air. Laboratory tests showed the following: white blood

Corresponding Address: Cho Youngmo E-mail: specialkey7@naver.com
Submitted: 18.02.2019 • **Revision Received:** 19.04.2019 • **Accepted:** 07.05.2019
Available online at www.advancedotology.org

cell count, 4520/μL; hemoglobin, 14.0 g/dL; platelet count, 198,000/μL; serum sodium, 144.0 mEq/L; potassium, 4.52 mEq/L; chloride, 106.5 mEq/L; blood urea nitrogen, 14.2 mg/dL; and serum creatinine, 0.82 mg/dL. Liver transaminase levels were normal. Urinalysis showed specific gravity, 1.010; pH, 6.5; urine protein, 2+ score; and trace scores for occult blood and glucose. Arterial blood gas analysis showed pH, 7.43; partial pressure of carbon dioxide ($PaCO_2$), 27.1 mm Hg; partial pressure of oxygen (PaO_2), 157.8 mm Hg; bicarbonate (HCO_3), 18.2 mM; and oxygen (O_2) saturation, 99.8% on 3 L/min of oxygen via nasal cannula.

The patient remained in the emergency room overnight; the next morning, she underwent endoscopy to identify the effects of strong acid ingestion. She was diagnosed with erythematous gastritis, reflux esophagitis, and corrosive injury grade I. We determined that she was able to take food by mouth.

We started continuous renal replacement therapy (CRRT) 26 h after admission because serum creatinine monitoring increased rapidly from 0.82 mg/dL measured 2 h after admission, to 2.93 after 19.5 h, and 3.50 at 25 h after admission, and vital signs became unstable. Considering that AKI is a life-threatening condition for the patient, she was admitted to the Department of Nephrology for dialysis. A kidney biopsy taken on the 19th day after admission showed acute tubular damage. After two hemodialysis sessions during 19 days of inpatient treatment, renal function returned to normal.

After 12 h in hospital, the patient complained of sudden bilateral hearing loss. An otoscopic examination was normal. The results of pure tone audiometry suggested profound sensorineural hearing loss (Figure 1); tympanometry showed type A tympanograms on both sides. An auditory brain stem response test showed no response at the maximum limits. High-resolution temporal bone computed tomography and magnetic resonance imaging showed normal inner ear structure on both sides (Figure 2). She was diagnosed with bromate-induced sudden deafness, and systemic steroid therapy was administered for a week with dose reduction for another week.

However, her hearing did not recover in the six months following ingestion. We decided to perform CI on her right ear. A Med-El Concerto Flex 28 (Med-El GmbH, Innsbruck, Austria) was used, and full insertion of all electrodes was achieved with no difficulty. Intraoperative neural response telemetry tests were positive. No post-implantation infection or other complications occurred.

Following mapping, programming, and aural rehabilitation, the patient's auditory performance was excellent, with categories of auditory performance of 7. She was very satisfied with her hearing skills for both speech and environmental sounds. Eight months after implantation, an aided pure tone audiogram showed a threshold level of 32 dB (Figure 3). The patient scored 100% on the open set sentence perception test, and 100% on the open set word perception tests with monosyllabic and bisyllabic words. The authors appreciate the cooperation of the patient, who provided her consent for this study.

DISCUSSION

Bromate is a strong oxidizing agent. It is colorless, tasteless, odorless, and water-soluble. It is used in a 2%-10% aqueous solution as a permanent neutralizing agent, with a lethal dose of 160-500 mg/kg [11, 12]. Clinical features include GI symptoms such as vomiting, diarrhea, and abdominal pain, which occur within hours after bromate intoxication [7, 12, 13]. GI symptoms are caused by hydrobromic acid produced by the reaction of bromate with hydrogen chloride in the stomach to stimulate the gastric mucosa [14]. These manifestations are most common after ingestion and appear before other clinical symptoms and in severe cases may cause GI bleeding due to corrosive effect of bromate [15, 16].

The rapid, irreversible, bilateral sensorineural hearing impairment due to bromate intoxication is known to occur within 4-16 hours of ingestion [12]. The mechanism of ototoxicity, although not yet established, can be attributed to a decrease in enzyme activity that causes damages to the stria vascularis and degenerative changes in the outer hair cells. A breakdown of the endolymph-perilymph barrier

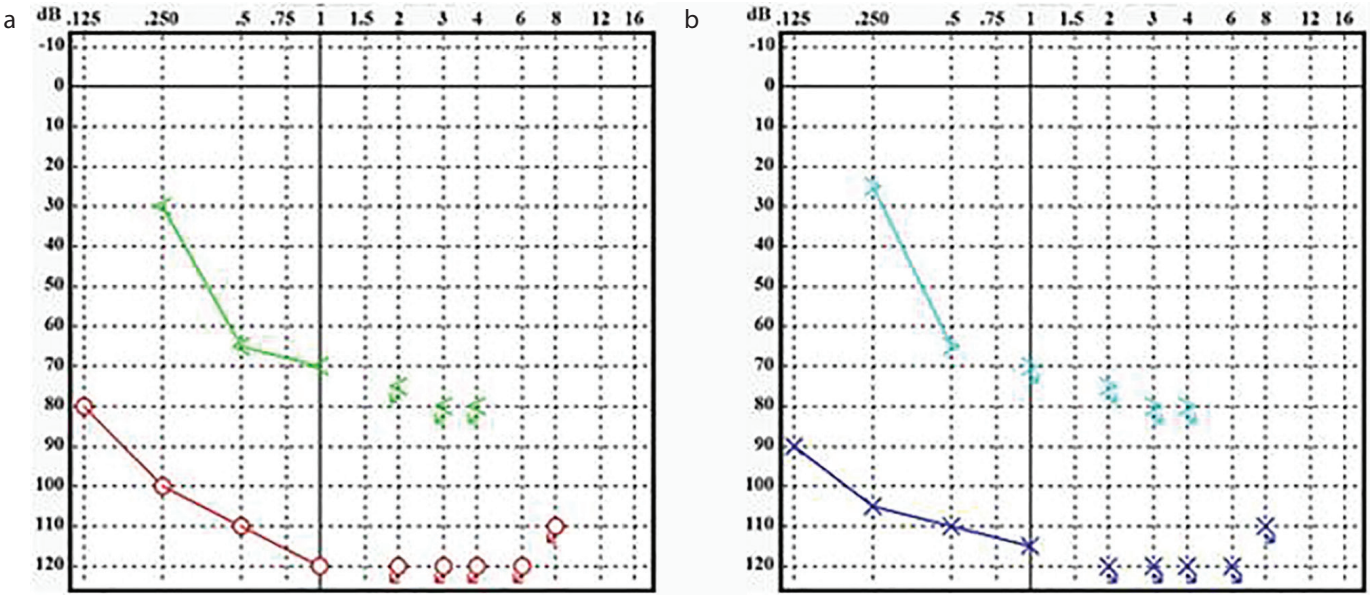

Figure 1. a, b. Preoperative pure tone audiometry showed bilateral profound sensorineural hearing loss. (a) Right. (b) Left.

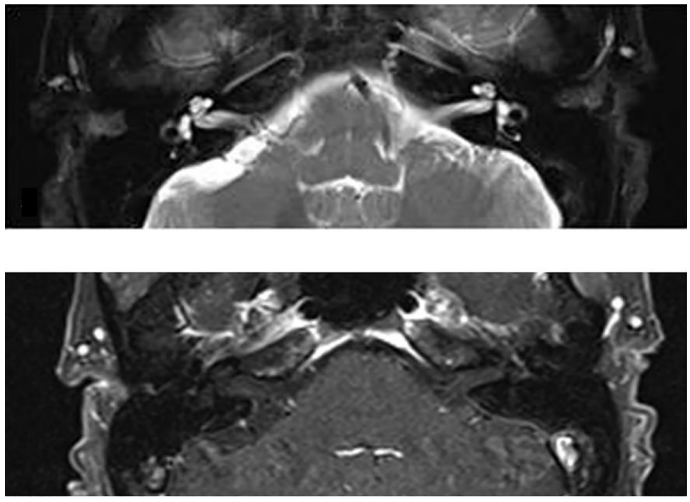

Figure 2. Preoperative MRI T2-weighted image of both ears demonstrating normal high signal intensity of cochlear and vestibulocochlear nerve.

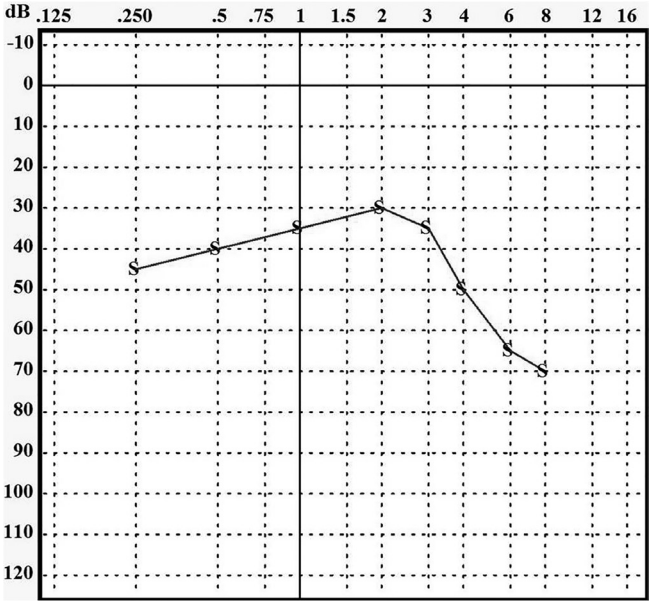

Figure 3. Postoperative implant aided pure tone audiogram, six months after implantation.

appears to be involved, resulting in a decrease in the endocochlear potential [17, 18]. Also, the mechanism of AKI has not yet been fully elucidated. The hypothesis is that direct tubular damage caused by activated oxygen radicals and reduced renal perfusion due to intravascular volume depletion and modulation of vasomotor tone [7, 12].

Early dialysis is recommended to restore compromised renal function in these cases, and AKI, that two decades ago was considered irreversible, is now being successfully treated [1-3, 19]. In our case, renal failure was well treated through CRRT with hemodialysis (HD), and renal function was maintained well without additional dialysis until the time of this report. The reason for the recovery of renal function through dialysis is due to the presence of bromate in the form of sodium bromate ($NaBrO_3$, molecular weight 150.91 g), and potassium bromate ($KBrO_3$, molecular weight 167.01 g). The bromate absorbed into the blood after ingestion has low molecular weight and is soluble, so it can be removed easily by hemodialysis, rather than peritoneal dialysis. In CRRT, continuous veno-venous hemofiltration and continuous veno-venous hemodiafiltration (CVVHDF) based on the dialysis mechanism, including the diffusive transport of molecules method, will be effective. Indeed, some studies have suggested that early application of CRRT can reduce bromate-induced hearing loss [1, 3]. We therefore support use of CRRT for such cases, if it is available [20].

CONCLUSION

For patients in whom irreversible hearing loss has already occurred due to bromate ototoxicity, replacement of the hearing organ to restore hearing ability should be considered as a definitive treatment. Although two reports of CI to remediate bromate-induced hearing loss exist, [2, 8] this treatment is very rare. In this case, we successfully restored the patient's hearing through CI, with good results and complete patient satisfaction. We therefore recommend CI as a treatment to improve the quality of life of patients with deafness due to bromate intoxication.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – C.S.W.; Design - C.Y.; Supervision - C.Y.; Resource - C.S.W.; Materials - C.Y.; Data Collection and/or Processing - C.S.W.; Analysis and/or Interpretation - C.S.W.; Literature Search - C.Y.; Writing - C.Y.; Critical Reviews - C.S.W.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: This work supported by clinical research grant from Pusan National University Hospital in 2019.

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