

Selected Topics: Toxicology

METHADONE, ANOTHER CAUSE OF OPIOID-ASSOCIATED HEARING LOSS: A CASE REPORT

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Abstract—Background: Methadone has been used for many years in the clinical setting and has many well-described side effects. In recent years, the use of methadone and other opioids have been increasing throughout the United States (US), and presentations to US Emergency Departments (EDs) due to opioid use and abuse are increasing as well. **Objectives:** As methadone and opioid use increases, ED physicians should be aware of infrequently seen side effects and toxicities associated with the use of these drugs. **Case Report:** We report the case of a previously healthy 20-year-old man who presented with acute onset of bilateral hearing loss secondary to an unintentional methadone overdose. At follow-up, the patient's hearing had returned to normal, with the only intervention being abstinence from methadone. **Conclusion:** Although bilateral hearing loss is a rare toxic finding of opioid ingestion, given the prevalence of opioid use, this etiology should be considered in any patient presenting with this chief complaint. © 2011 Elsevier Inc.

Keywords—hearing loss; methadone; overdose; opioid; ingestion

INTRODUCTION

Methadone has been used for many years in the clinical setting. The United States (US) Food and Drug Administration-approved uses include detoxification treatment of opioid addiction, maintenance treatment of

opioid addiction, and for the treatment of moderate to severe pain. Methadone's use in the United States has dramatically increased in recent years, and this increase in use has outpaced oxycodone, hydrocodone, and hydromorphone (1). Like other opioid medications, methadone has the potential for abuse. The common side effects of methadone are well described and include sedation, constipation, respiratory depression, lightheadedness, dizziness, nausea, and vomiting. Other side effects include dysrhythmias, itching, sweating, rhabdomyolysis, and orthostatic hypotension (2). With the increase in methadone's use and abuse, infrequent and rare side effects or toxicities may become more prevalent. We report a rare case of transient deafness after an intentional misuse and inadvertent overdose of methadone in a previously healthy young man.

CASE REPORT

A 20-year-old man with no significant past medical history was brought to the Emergency Department (ED) by his roommate, who stated that the patient was difficult to wake that morning and looked "pale." Via writing, the patient also described a new onset of deafness. The patient admitted to drinking "liquid morphine" and alcohol the prior evening. The patient denied any previous hearing loss or dyspnea. He also denied recent illness, co-ingestion, trauma, headache, travel, or sick contacts.

On arrival in the ED, the patient had a temperature of 35.8 °C (96.5°F), blood pressure 126/70 mm Hg, heart rate 112 beats/min, respiratory rate 24 breaths/min, and oxygen saturation of 86% on room air. A Masimo Rad-57 (Masimo Corporation, Irvine, CA) co-oximeter (a device similar to the pulse oximeter) was used to non-invasively detect the patient's carboxyhemoglobin and indicated a level of 35%. His glucose was 120 mg/dL. The patient was awake and answering questions slowly. On physical examination, he appeared markedly cyanotic. His physical examination was significant for miosis, tachycardia, tachypnea, and mild respiratory distress; he had rales throughout all lung fields. His neurologic examination was non-focal with the exception of a dense bilateral hearing loss.

With 100% oxygen via a non-rebreather face mask, his oxygen saturation rose to 97%. Chest radiographs demonstrated pulmonary edema, with mild bibasilar infiltrates. The patient received 0.4 mg of naloxone intravenously in an effort to improve his mental status and the quality of his respirations. There was no improvement at this dose, therefore the dose was not escalated and other interventions were initiated. Bi-level positive airway pressure (BiPAP) was begun for presumed acute lung injury. The patient was improving on BiPAP but subsequently vomited. Therefore, he was orotracheally intubated for airway protection.

Laboratory analysis was significant for a leukocytosis of 25,000/dL, serum sodium of 149 mEq/L, and a serum creatinine of 1.6 mg/dL. His creatinine kinase was elevated at 2452 IU/L, with an alanine aminotransferase and aspartate aminotransferase of 227 and 243 IU/L, respectively. Acetaminophen, salicylate, ethanol, and methemoglobin levels were non-detectable. His carboxyhemoglobin was 4%. Urine drug screening immunoassay was performed on a Beckman Coulter UniCel Dx C 800 using SYNCHRON reagents (Beckman Coulter Inc., Brea, CA). It revealed the presence of methadone using a cutoff value of 300 ng/mL and cannabinoids using a cutoff value of 50 ng/dL, but was negative for the presence of other opioids, amphetamines, barbiturates, phencyclidine, or cocaine. His venous blood gas at the time of arrival demonstrated a respiratory acidosis, with a pH of 7.13, and a pCO₂ of 96 mm Hg. An electrocardiogram revealed sinus tachycardia, with right axis deviation but a normal QRS and QT interval. Computed tomography of the brain was negative for acute processes. A sample of the "liquid morphine" was obtained and identified as methadone using gas chromatography/mass spectroscopic analysis.

The patient was admitted to the Medical Intensive Care Unit, where he was treated for aspiration pneumonitis and rhabdomyolysis. His creatine phosphokinase trended downwards and his creatinine normalized after

24 h. He was extubated on hospital day 2 and discharged home on hospital day 4 with complete resolution of his hearing loss. A follow-up phone call confirmed complete resolution of the patient's hearing loss.

DISCUSSION

Acute bilateral hearing loss is an uncommon presentation to the ED. Hearing loss can be classified as conductive or sensorineural. In conductive hearing loss, damage to the mechanical components of the inner ear lead to inadequate transmission of sound from the external ear to the middle ear. Conductive hearing loss can be caused by cerumen impaction, otitis media, foreign bodies and otosclerosis. Sensorineural hearing loss occurs when there is dysfunction at the level of the cochlea or the neural pathway to the auditory cortex. Sensorineural hearing loss results from inherited disorders, noise exposure, ototoxic drugs, and presbycusis (age-related hearing loss) (3). Ototoxicity is not considered a common side effect of opioid medications but has been described with exposure to multiple xenobiotics; aminoglycoside and other antimicrobial agents, diuretics, chemotherapeutic agents, anti-inflammatory agents, and antimalarial medications are some of the more common ototoxic xenobiotics (4) (Table 1).

Ototoxicity is a known rare adverse effect of opioid medications. However, significant case literature supports the phenomenon of opioid-associated hearing loss (OAHL). Mulch et al., in 1979, published the first case report of hearing loss after hydrocodone abuse (5). Since that time, multiple case reports have reported reversible and irreversible bilateral hearing loss after abuse of hydrocodone/acetaminophen and heroin (Table 2) (5–11).

OAHL has been described after acute or chronic opioid use. Two patients who abused hydrocodone daily over a period of years experienced irreversible hearing loss, which rapidly progressed over a period of weeks. One of these patients reported a feeling of fullness in his ears and had intermittent tinnitus (6). In cases of heroin abuse, patients frequently report a period of abstinence from the drug followed by a relapse before presentation. There is usually a period of unconsciousness after use of the drug and, upon awakening, patients note hearing loss (5,7–9). Patients may report other vestibulocochlear symptoms such as tinnitus, aural fullness, and vertigo (7,8,10). Heroin-associated hearing loss resolved over days to weeks in three cases (5,7,9). However, in a case report where heroin was injected directly into the carotid artery, the patient had continued hearing loss 3 months after the exposure (10). Permanent hearing loss was reported in a heroin addict with prior transient heroin-associated ototoxicity (8).

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