

Sudden Bilateral Sensorineural Hearing Loss Following Speedballing

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Abstract

Background: Hearing loss is an infrequently-reported consequence of recreational drug abuse. Although there are sporadic reports of hearing loss from heroin and cocaine ingested separately, there are no reports of hearing loss resulting from the combination of both drugs ingested simultaneously in the form of speedballing.

Purpose: The purpose of this report is to document a case of bilateral sensorineural hearing loss associated with an episode of speedballing.

Research Design: Case Report

Data Collection and Analysis: The subject of this report was a 40-year-old man with a 20-year history of substance abuse. Data collected included a case history, pure tone audiometry, tympanometry and acoustic reflexes, and transient evoked otoacoustic emissions.

Results: The audiologic evaluation indicated a mild to moderate, relatively flat, bilateral sensorineural hearing loss that was worse in the right ear.

Conclusions: A bilateral sensorineural hearing loss involving both cochlear and neural pathology may be a rare complication of cocaine, heroin, or the combination of the two drugs.

Key Words: Autoimmune, bilateral hearing loss, cocaine, cochlear pathology, drug abuse, heroin, otoacoustic emissions, recreational drugs, sensorineural hearing loss, sudden hearing loss

Abbreviations: MRI = magnetic resonance imaging; TEOAEs = transient evoked otoacoustic emissions

Sumario

Antecedentes: La hipoacusia es una consecuencia infrecuentemente reportada del uso de drogas como recreación. Aunque existen reportes esporádicos de hipoacusias por heroína y cocaína ingeridas por separado, no hay reportes de hipoacusias como resultado de la combinación de ambas drogas ingeridas simultáneamente en la forma de “*speedballing*.”

Propósito: El propósito de este reporte es documentar un caso de hipoacusia sensorineural bilateral asociado con un episodio de “*speedballing*.”

Recolección y Análisis de los datos: El sujeto de este reporte era un hombre de 40 años con una historia de 20 años de abuso de sustancias. Los datos colectados incluyeron una historia clínica, audiometría de tonos puros, timpanometría, reflejos acústicos y emisiones otoacústicas evocadas por transitorios.

Resultados: La evaluación audiológica indicó una hipoacusia sensorineural bilateral, leve a moderada, relativamente plana, que era algo peor en el oído derecho.

Conclusiones: Una hipoacusia sensorineural bilateral que involucraba tanto patología coclear como neural puede ser una complicación rara de la cocaína, la heroína o la combinación de ambas drogas.

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Palabras Clave: Autoimmune, hipoacusia bilateral, cocaína, patología coclear, abuso de drogas, heroína, emisiones otoacústicas, drogas de recreación, hipoacusia sensorineural, hipoacusia súbita

Abreviaturas: MRI = imágenes por resonancia magnética; TEOAE = emisiones otoacústicas por transitorios

Many users of recreational drugs are known to use cocaine and heroin sequentially or together. In the case of speedballing, the two drugs are injected simultaneously. The reasons for using both drugs together include a heightened sense of euphoria compared to either drug alone, an attempt to experience the effects of both drugs simultaneously, and a mitigation of the severe physiological effects of withdrawal (Leri et al, 2003). Estimates range from 30 to 80% of abusers of heroin and of cocaine (Leri et al, 2003). People addicted to opioids, including heroin, frequently continue to use cocaine even while under methadone treatment for drug use (Bandettini di Poggio et al, 2006).

Independently, cocaine and heroin have major effects on the body. Cocaine is a potent stimulant of the central nervous system. When used alone, it produces vasoconstriction and activates the sympathetic nervous system to increase heart rate, blood pressure, and body temperature. The acute effects of cocaine overdose include cardiovascular and neurovascular deficits, such as seizures and intracranial hemorrhages (Zimmerman, 2003).

Hearing loss has been related to cocaine use. In both animal and human newborns, prenatal exposure to cocaine has been associated with hearing, vision, and growth deficits that are attributed to vasoconstriction, hemorrhage, malnutrition, or a combination of these conditions (Church et al, 1998). Although hearing deficits may resolve with development, these children may be at risk for language and cognitive delays (Church et al, 1998; Cone-Wesson, 2005). In a case study of an adult, Nicoucar et al (2005) reported that a unilateral hearing loss developed following intranasal ingestion of cocaine by a man who was on methadone substitution treatment and who had also ingested alcohol. The cocaine was associated with the onset of dizziness, tinnitus, and unilateral hearing loss described as "deafness." Although the dizziness eventually subsided, the hearing loss appeared to be permanent. The hearing loss was presumed to be cochlear in origin, caused by an intralabyrinthine hemorrhage that was documented by magnetic resonance imaging (MRI).

Heroin is a central nervous system depressant. Heroin used alone can lead to coma, reduced respiration, pulmonary edema, cardiovascular disorders, and stroke (Buttner et al, 2000). Heroin overdose has been associated with peripheral neuropathy, temporary

paralysis (compressive), chest infections, and seizures (Warner-Smith et al, 2002). Noncompressive acute peripheral neuropathy, presumably caused by toxins from the heroin, has also been identified (Dabby et al, 2006).

Hearing loss from heroin is not often reported. Full-term infants born to heroin-abusing mothers have not been found to be more likely to have severe sensorineural hearing loss than nonexposed infants (Grimmer, Buhner, Aust, & Obladen, 1999). In a study of drug users, Iqbal (2002) interviewed 16 heroin users, including 2 who reported a temporary bilateral hearing loss and 1 who reported improved hearing after heroin use. No objective measures of hearing were obtained. Polpathapee et al (1984) reported a case in which a 25-year-old man overdosed from an injection of heroin; passed out for 2 days; and awoke with muscle aches, fatigue, and reduced hearing. He had been taking nitrazepam for 6–7 years and cocaine for 2–3 years, with previous sensations of fullness in his ears. One month after the overdose, an audiogram revealed a bilateral, moderate to profound, sensorineural hearing loss, which was confirmed by an auditory brainstem response test. Although the authors reported that the hearing loss was of both cochlear and retrocochlear origin, the degree of the hearing loss was too severe to permit a reliable assessment of the site of lesion.

Korteque et al (2005) described a 26-year-old man who sustained a unilateral profound hearing loss in his right ear following an injection of heroin into his right neck. A reevaluation 3 months later indicated that the hearing loss was not improved. No assessment of the site of lesion was possible beyond an MRI that ruled out vascular and space-occupying lesions.

Finally, Mulch and Handrock (1979) reported the case of a 20-year-old who suffered a sudden, bilateral, sensorineural hearing loss after ingesting heroin. In that case, the hearing loss was reversed in 3 days. There are no other published studies of sudden hearing loss caused by the use of heroin.

In summary, there are sporadic reports of hearing loss associated with overdoses of cocaine or heroin, but not of both taken simultaneously. The previously described hearing losses have been unilateral or bilateral, mild or moderate to profound, and permanent or transient, and the underlying pathology generally is not clear. There are no reports in the

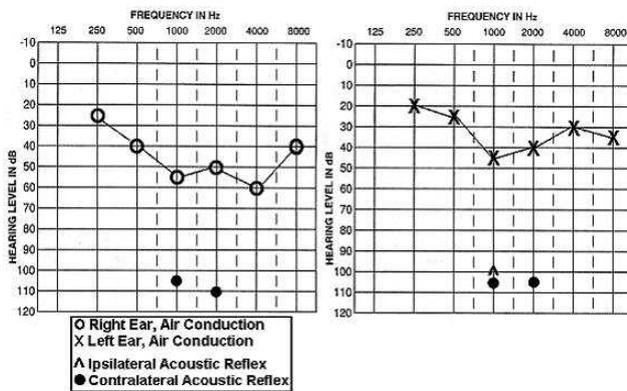


Figure 1. Audiogram for the right ear (left panel) and left ear (right panel) showing air-conduction thresholds and acoustic reflex thresholds. Air-conduction thresholds indicate essentially a bilateral mild to moderate hearing loss, which is worse in the right ear. Bone conduction thresholds and further behavioral testing were not obtainable.

literature of hearing loss caused by speedballing. The purpose of this report is to document a case of bilateral, sensorineural hearing loss associated with an episode of speedballing.

CASE REPORT

A 40-year-old Caucasian man with a 20-year history of polysubstance abuse reported to the emergency room in the midafternoon complaining of a sudden, bilateral hearing loss following a night of speedballing. He had passed out for 12 hours after ingestion of the drugs and had awakened with the hearing loss. He was shaking and sweating profusely when examined. His affect was agitated and distractible, although he was coherent. He reported a bilateral hearing loss and a high-pitched, hissing tinnitus in the left ear, with no significant history of noise exposure and no prior episode of hearing loss. No previous audiogram was available.

Given his physiological state, only limited audiological testing was possible. An otoscopic examination revealed that the ear canals were clear and the tympanic membranes were of normal appearance. Behavioral pure-tone air-conduction tests revealed a bilateral moderate, relatively flat, hearing loss, as shown in Figure 1. The man's responses were somewhat erratic, although they were considered reliable. Because of his distress, bone-conduction thresholds and other behavioral tests were not attempted. Tympanograms were within normal limits in both ears. The acoustic reflexes were absent or elevated in both ears, as shown in the lower part of the audiogram in Figure 1. Transient evoked otoacoustic emissions (TEOAEs) were absent bilaterally from 1,000 to 5,000 Hz.

The audiogram suggested a bilateral mild to moderate, relatively flat hearing loss, which was worse in the right ear. The normal tympanograms ruled out middle ear effusion, traumatic tympanic membrane perforations, and ossicular disarticulation. The presence of acoustic reflexes (though elevated) ruled out ossicular fixation and confirmed the absence of conductive hearing loss in both ears. In the presence of normal tympanograms and a normal otoscopic examination, the absent TEOAEs confirmed that the hearing was no better than a mild loss, and thus the hearing was consistent with the thresholds shown on the audiogram. Because the TEOAEs are cochlear responses, their absence further confirms the presence of significant cochlear pathology in both ears. The reflex pattern, with equal thresholds for ipsilateral and contralateral acoustic reflexes at 1,000 Hz in the right ear, is consistent with an additional retrocochlear site of lesion.

The man failed to show for a follow-up appointment, and he could not be located for rescheduling.

Discussion

Possible explanations for the sudden bilateral sensorineural hearing loss and absent otoacoustic emissions include cochlear anoxia, cochlear toxicity, or autoimmune reaction precipitated by the use of cocaine, heroin, or a combination of the two drugs. Nicoucar et al (2005) suggested that the unilateral hearing loss in their case of cocaine overdose was a cochlear lesion given the MRI evidence of an intralabyrinthine hemorrhage. Korteque et al (2005) surmised that the unilateral hearing loss in their case of a heroin overdose was caused by cochlear toxicity from a carotid injection of the heroin. None of the previously identified cases of bilateral hearing loss following drug use provided adequate evidence of the site of lesion or suggested a cause.

The physiological cause of the lesion in the present case cannot be determined, but the site of the lesion includes both cochlear and neural components. The absence of otoacoustic emissions confirms the cochlear pathology, and elevated acoustic reflexes in the presence of normal tympanograms confirms a neural component. The bilateral nature of the hearing loss is unlikely to have been caused by two independent vascular events. Systemic toxins or autoimmune reaction related to one or both drugs, however, could affect both sides.

Autoimmune-related hearing loss has not been linked thus far to cocaine or heroin abuse. Autoimmune hearing losses, however, are variable and have been described as having sudden or rapid onset, unilateral or bilateral presentation, transient or permanent pathology, and both cochlear and neural sites of lesion (Cadoni et al, 2002; Bovo et al, 2006).

Autoimmune circulatory and brain-related pathologies resulting from the abuse of both cocaine and heroin have been noted (Savona et al, 1985; Jankovic et al, 1991). Hence, an autoimmune reaction cannot be eliminated as a possible mechanism for the bilateral hearing loss in the present case.

In summary, bilateral or unilateral hearing loss may be a rare complication of intoxication from cocaine, heroin, or a combination of the two drugs. In the present case of a sudden, bilateral hearing loss following speedballing, both cochlear and neural pathology were documented. Effects of additional substances that are mixed with the target drugs or ingested separately cannot be discounted. Local police, however, reported that they had no indication of street drugs being cut with aspirin, a drug that in high doses can cause bilateral hearing loss.

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