Auditory Hallucinations in Patients With Acquired Hearing Loss: A Case Series

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ABSTRACT

Introduction: Auditory hallucinations are a common but underrecognized symptom in those with hearing impairment. The pathophysiology is poorly understood but hypothesized to be related to deafferentation phenomenon.

Case Presentations: We describe the cases of 2 patients with bilateral acquired hearing loss and no previous history of psychosis who reported distressing auditory hallucinations. Case 1 involves a woman in her 60s who presented with both auditory and visual hallucinations and associated delusions that did not respond to olanzapine but responded well to sertraline and a low dose of quetiapine. Case 2 involves a man in his late 70s with progressively worsening auditory hallucinations with associated delusions who responded well to treatment with risperidone.

Discussion: Despite centuries of documentation about hallucinations in sensory impairment, its pathophysiology is not completely understood, nor is there consensus regarding treatment.

Conclusions: Health care providers should be aware of the prevalence of auditory hallucinations in those with hearing impairment, particularly when symptoms are misattributed to primary psychotic disorders or neurodegenerative conditions. A biopsychosocial approach, including psychoeducation, behavioral and environmental modifications, and family involvement can yield significant improvement and even resolution of symptoms.

INTRODUCTION

Auditory hallucinations are an unexpectedly common occurrence in patients with hearing impairment, affecting over 16% of patients.[1] This phenomenon has been observed in individuals with both acquired and congenital hearing loss,[2,3] and it occurs more frequently in individuals with more severe impairment.[1] Despite its prevalence, it remains an understudied and underrecognized clinical entity. The pathophysiology is not well understood, but a leading theory is that these auditory hallucinations represent a deafferentation phenomenon, similar to Charles-Bonnet syndrome in visually impaired individuals.[4] Deafferentation auditory hallucinations can lead to severe distress, depression, sleep impairment, and secondary delusions.[5]

These hallucinations are not thought to be mediated primarily with dopamine; however, multiple case reports have shown benefit from antipsychotics.[2,3] Other proposed treatments include antidepressants, anticonvulsants, and acetylcholinesterases.[5] Many patients have benefitted from nonpharmacological interventions, including psychoeducation, optimizing sensory input (such as hearing aids), and addressing comorbid psychiatric conditions.[4]

In this case series, we describe 2 patients with bilateral acquired hearing loss and no previous history of psychosis who reported distressing auditory hallucinations. The hallucinations were associated with sleep disturbance and secondary delusions that were treated with pharmacotherapy and psychoeducation.

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CASE PRESENTATIONS

Case 1

Patient A is a woman in her 60s with a history of obesity, chronic knee pain, insomnia, and severe to profound acquired sensorineural hearing loss. She presented initially to the emergency department (ED) after several days of frightening auditory hallucinations, visual hallucinations, paranoia, and lack of sleep. She had no prior significant psychiatric history, and extensive chart review revealed only that the only past psychotropics were brief trials of bupropion

(for smoking cessation), topiramate (for weight loss), and zolpidem (for sleep)—all prescribed by a primary care clinician.

Of note, 7 years prior to her initial presentation, the patients experienced bilateral hearing loss, which progressed from mild to severe-profound over several years. Audiologic evaluation, brain magnetic resonance imaging (MRI), and rheumatologic workup failed to identify a definitive cause for her hearing loss. She reported hearing "nothing" with 0% word recognition bilaterally and described concurrent tinnitus—at times with the perception of music, children singing, or a preacher.

During initial psychiatric evaluation in the ED, the patient reported not sleeping for 3 days and described threatening auditory hallucinations, including voices stating that she would be killed and cut to pieces, as well as commands from Satan. She also reported visual hallucinations of rabbits and children. A urine drug screen was positive for opiates, which had been prescribed for osteoarthritis knee pain. She also recently had been prescribed a short course of methylprednisolone for knee pain. The working diagnosis was substance-induced psychosis. She was treated with a single dose of olanzapine, reported improved sleep and resolution of visual hallucinations, and was discharged the following day with a short course of olanzapine.

Five days later, the patient returned to the ED with continued symptoms of poor sleep and auditory hallucinations. She was subsequently evaluated by integrated behavioral health in the outpatient setting. At that time, she demonstrated insight that the auditory hallucinations were not real but described a chip in her ear emitting screeching feedback, which she believed allowed others to read her mind. Olanzapine was increased. One month later, she returned to the ED, and olanzapine was titrated further. She then established care at a psychiatry resident clinic.

At her initial clinic visit, olanzapine dosing was adjusted to 20 mg nightly to target hallucinations and ongoing poor sleep. Hallucinations were more severe at night, particularly prior to sleep and when she was alone in a quiet, dark room. At subsequent visits, she disclosed a history of trauma and other symptoms consistent with posttraumatic stress disorder, as well as depressive symptoms. Sertraline was initiated and was gradually titrated to a therapeutic dose over several months. Despite this, she continued to experience poor sleep and auditory hallucinations. Olanzapine was tapered and discontinued. Trazodone was trialed but was ineffective for sleep. Quetiapine was initiated to address sleep, mood, and hallucinations, resulting in improvemed sleep and decreased distress from auditory hallucinations. Quetiapine was titrated further.

With a combination of sertraline 150 mg daily and quetiapine 100 mg nightly, the patient reported improved sleep and continued but nondistressing hallucinations, including music and nonviolent, noncommand voices. Her mood improved, and she achieved remission from major depressive disorder. She reported she was "very thankful" and "felt like [herself] again."

The patient's subsequent treatment course was complicated by inconsistent follow-up with outpatient psychiatry and suboptimal medication adherence. Two-and-a-half years after initiating the quetiapine and sertraline regimen, quetiapine was transitioned to risperidone with good clinical response and a planned transition to long-acting injectable risperidone.

Case 2

Patient B is a man in his late 70s with history of bilateral sensorineural hearing loss, mild major neurocognitive disorder, panic attacks, hypertension, hyperlipidemia, obstructive sleep apnea, and a seizure disorder secondary to a right frontal abscess, status post craniotomy. He reported progressively worsening auditory hallucinations over several years and denied any substance use. On interview, his hearing impairment was so severe that it impeded conversation even with assistive devices; thus, he relied on his wife to provide history.

His hallucinations consisted of arguing voices and "psychedelic" music, which became increasingly distressing. To manage symptoms, he played loud classical music to drown them out, which in turn became distressing to his wife. The music was unfamiliar to him and occurred daily. Intensity increased when he was alone and in quiet areas with low ambient noise. He also reported paranoid delusions, often attributing the voices and music to neighbors or other nearby individuals and sometimes confronting those he believed were responsible for the noise. He also attempted to silence the music by placing duct taping over air vents.

Previous audiometric testing revealed severe, bilateral sensorineural hearing loss, and he was not a candidate for cochlear implantation. Further workup included computed tomography of the head, which showed no acute intracranial abnormality. MRI of the brain showed diffuse parenchymal loss, though no clear disproportionate loss or microvascular deficits were noted. Neuropsychological testing results were consistent with frontal lobe dementia, with deficits in executive functioning, attention, and learning and memory retrieval (with confabulatory tendencies). Orientation, visual spatial skills, and language abilities were intact.

Low-dose quetiapine was initiated, but after several months there was no change to auditory hallucinations. The patient subsequently began hearing "explosions" that were not seizure-related but reminded him of pre-seizure auras. These episodes frequently woke him from sleep. The hallucinations and explosions became so severe and distressing that he voiced suicidal ideation and was admitted briefly for further evaluation and risk assessment. While hospitalized, quetiapine was discontinued due to lack of efficacy, and risperidone was initiated to treat hallucinations. Electroencephalography showed no seizure activity, and the patient was discharged.

After several weeks, the patient reported complete resolution of the "explosions." Musical auditory hallucinations and mile paranoia continued, though his wife noted improvement in his mood with risperidone. With continued titration of risperidone over several months to 1 mg twice daily, he reported complete resolution of hallucinations, which remained absent 2 years after initial presentation.

DISCUSSION

We present the cases of 2 patients with bilateral acquired sensorineural hearing loss and no prior history of psychosis who developed auditory hallucinations. Consistent with previously reported cases, both patients experienced upsetting hallucinations that began after several years of progressive hearing loss. The hallucinations included both musical passages and voices. In both cases, symptoms intensified during periods of reduced ambient noise and were described as highly distressing.

Several risk factors for musical hallucinations have been identified, including advanced age, female sex, hearing loss, primary brain disease, certain medications (particularly antihypertensives), and social isolation.[6] Both of our patients shared many of these features.

A notable discrepancy between the 2 cases was the patients' response to psychotropic treatment. Patient A did not respond to olanzapine but improved with sertraline and quetiapine, whereas Patient B worsened with quetiapine and resolved with risperidone. This variability is consistent with existing case reports, which describe highly variable responses to psychotropic medications, including antidepressants, anticonvulsants, acetylcholinesterase inhibitors, benzodiazepines, and antipsychotics.[2-4,7] Other proposed interventions include nonpsychotropic treatments, such as optimizing sensory input (eg. hearing aids), psychoeducation, psychotherapy, and treatment of the underlying cause of hearing impairment.[5]

Documented examples of this phenomenon date back to the 19th century. Colman referenced an 1883 case of a patient with unilateral auditory hallucinations of bells, attributed to a wax-covered grain of wheat in the external auditory meatus, with resolution following removal—though one could argue this represents temporary rather than acquired hearing loss.[8] More recently, Hammeke et al reported 2 geriatric patients with bilateral hearing loss and musical hallucinations who experienced only minor improvement after treatment with an anticonvulsant, an antipsychotic, and vitamin supplementation.[9] David and Fernandez described an 88-year-old woman with progressive hearing loss who reported hypnogogic hallucinations of various instruments, including a full orchestra, and responded rapidly to quetiapine.[7] Tuck et al reported at 75-year-old male with posttraumatic stress disorder and bilateral hearing loss who presented with suicidal ideation due to increasingly disturbing hallucinations, including gospel music and an unfamiliar male voice.[3] He was treated with sertraline and aripiprazole; and while his hallucinations did not completely resolve, his mood improved and hallucinations decreased in frequency and volume.

While the cause for these hallucinations remains unclear, they are generally understood to represent a deafferentation phenomenon, in which reduced sensory input lead to disinhibition of perception-bearing neural circuits, followed by "release" of perceptual traces.[9] Griffiths theorized that auditory input into the primary auditory cortex is processed into segmented sounds, which are then encoded and recognized. In the absence of sufficient input, spontaneous pattern recognition of these segmented sounds may result in the perception of musical hallucinations.[10]

While case reports spanning more than 125 years have documented this phenomenon—and the heterogeneity of both causes and treatments—optimal treatment of auditory hallucinations remains unclear. Based on literature, it is reasonable to recommend that first-line treatments include psychoeducation, optimization of sensory input, treatment of reversible underlying medical conditions, and consideration of psychotropic trials, including antipsychotics or selective serotonin reuptake inhibitors, depending on co-occurring mood symptoms.

CONCLUSIONS

These cases highlight the complex and often underrecognized and undertreated phenomenon of auditory hallucinations in individuals with acquired hearing loss. Clinicians should maintain a high index of suspicion for auditory hallucinations in this patient population, particularly when symptoms are misattributed to primary psychotic disorders or neurodegenerative conditions. A biopsychosocial approach—including psychoeducation, environmental modifications, family involvement, and judicious use of psychotropics—can yield significant improvement and, in some cases, complete resolution of symptoms. Early recognition and tailored intervention are essential to reduce patient distress and prevent unnecessary morbidity.

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