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The mind with a radio of its own: a case report and review of the literature on the treatment of musical hallucinations

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ABSTRACT

Musical hallucinations (MH) have been labeled Oliver Sacks syndrome, and in the majority of cases, they occur in the context of a hearing loss. In these instances, they have been described as auditory Charles Bonnet syndrome because they are thought to represent a cortical release phenomenon. Patients with MH tend to have intact reality testing, and as such, the condition may also be described as musical hallucinosis. The temporal course of MH is variable, but given that they may improve or remit with time, education on their benign nature is often sufficient. MH also may improve when hearing loss is reversed. The use of ambient noise potentially ameliorates mild to moderate MH; however, where this is insufficient, somatic treatments may be considered. Case reports have documented successful use of low-dose antiepileptics, atypical antipsychotics and donepezil. We present a case of a 52-year-old man who received only partial relief from serial treatment with several psychotropic agents. He developed major depression with suicidal ideation in the context of persistent, intrusive MH that were refractory to several medication trials, and whereas a course of electroconvulsive therapy led to remission of depressive and suicidal symptoms, it provided only transient relief of his MH. In this article, we also provide a review of the literature on the neurobiology and treatment of MH.

1. Introduction

Musical hallucinations (MH) are auditory phenomena characterized by a subjective experience of hearing music or aspects of music in the absence of ambient auditory stimuli [1,2]. Their phonology is generally of well-known music from memory, particularly pop or religious songs [1–3]. Hallucinations with a musical quality have been labeled Oliver Sacks syndrome after the British neurologist and author of *Musicophilia* as well as auditory Charles Bonnet syndrome, particularly when they occur as cortical release phenomena. Although the literature on MH is limited, MH have been associated with hearing abnormalities, adverse effects of pharmacological treatment, advanced age, female gender and psychiatric illness. It is unclear to what extent advanced age and gender play independent roles in the development of MH or whether these may be explained by confounders including systemic pathology or other psychosocial factors.

Previous reviews have supported the hypothesis that this phenomenon is frequently linked to otological impairments, with reported hearing loss in up to 67% of individuals with MH [4], although they may occur in neurocognitive disorders such as Alzheimer's disease or in psychiatric illness [5]. The most common associated finding in cases of MH is impaired hearing, especially peripheral hearing loss. Similar to Charles Bonnet syndrome (visual hallucinations in those with impaired sight) and the phantom limb phenomenon (sensory experience in an amputated limb), MH are generally nonpsychotic in nature and found in those with a clear sensorium and insight into the fact that their sensory experience is not based on an environmental percept. States of persisting MH could also be termed musical hallucinosis when reality testing is intact. Despite the clear association with hearing loss, a few reports have documented MH in young patients with a history of sensory deprivation without otological pathology [6,7]. As a rule, the presence of musicality in hallucinations should raise the clinician suspicion for hearing impairment.

Among mental health diagnoses, obsessive compulsive disorder (OCD) may have the highest prevalence of MH, but investigations to date have failed to identify a common neurological process that provides a substrate to link these two disorders [8]. Hermesch and colleagues (2004) developed a questionnaire for assessing MH, the Geha Short Interview for Musical Hallucinations, and with this, they reported a 41.4% lifetime prevalence of MH among patients with OCD [8]. It has been argued, though, that these musical experiences reported by patients with OCD may be phenomenologically distinct from either release phenomena or psychotic hallucinations; moreover, OCD may present with senseless, intrusive musical obsessions that are experienced as originating from within one's own head rather than as ambient sensory percepts [9]. In addition, certain medications have been associated with an increased risk of MH, with opioids,

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antiepileptics and tricyclic antidepressants being described most commonly [10–16].

In this article, we present a detailed clinical case of a patient with MH who was treated serially with several interventions that have been reported in the literature; however, they provided only partial relief of symptoms in our patient. This report is unique in that previous documented cases generally describe only one or a couple of interventions and seldom capture the clinical difficulty of managing this potentially difficult-to-treat and distressing condition. Following the case report, we review documented approaches to the management of MH.

1.1. Case

Mr. P is a 52-year-old right-handed male with a 25-plus-year history of tinnitus who presented to the outpatient mental health services at our tertiary medical center with a chief complaint of MH that had begun suddenly several months prior, having awoken him from sound sleep, and persisted since. Initially, he had presented to his primary care physician who referred him to neurology and otolaryngology for chronic and distressing MH accompanied by diffuse headache. A thorough neurological exam was unremarkable. Head computed tomography and magnetic resonance imaging revealed dependent right mastoid mucosal thickening that was interpreted as consistent with mild, chronic mastoiditis; several scattered hyperintensities in the subcortical white matter of unclear clinical significance were also seen. Otolaryngology notes documented having treated him with "several courses of broad spectrum antibiotics and prednisone with minimal to no response" of MH. Both neurology and otolaryngology were in agreement that his symptoms were out of proportion to radiologic findings. Audiometry revealed a speech recognition threshold of 55 dB in each ear with discrimination of 92% at 90 dB when masked, which was diagnosed as sensorineural hearing loss.

On presentation to psychiatry, his symptoms were diagnosed as nonpsychotic MH and adjustment disorder with anxiety. He had been part of a rock band in his youth and recalled being exposed to deafeningly loud music for long periods of time. He endorsed a family history of faint musical tinnitus in a sibling and mother and a remote personal history of cocaine and LSD use, current use of cannabis every other day, daily tobacco cigarettes and current consumption of upwards of a six-pack of beer on weekends socially. He reported previous medication trials of lorazepam, paroxetine and gabapentin of unknown doses with limited to no benefit. As his mood symptoms increased and met criteria for major depression, he was given several medication trials of more than a month each at the following doses: citalopram 40 mg daily, venlafaxine 75 mg TID, mirtazapine 15 mg at bedtime and risperidone 2 mg at bedtime, with only transient improvements in mood or MH. Notably, buspirone 30 mg BID did offer mild but clinically significant improvement. After several months of scant clinical benefit, the patient developed suicidal ideation, and electroconvulsive therapy (ECT) was pursued. Although the immediate indication for ECT was to treat depression with suicidal ideation, it was hoped that it may provide ancillary relief of MH as well.

He was admitted to a psychiatric unit in the community and underwent six right unilateral sessions of ECT over 2 weeks with an additional session 2 weeks later. Over the course of his month-long hospitalization, he was started on quetiapine 150 mg and donepezil 10 mg at bedtime as well as trifluoperazine 5 mg and clonidine 0.2 mg both BID. Although he experienced a durable improvement of mood and resolution of suicidal ideation, MH proved only transiently improved after each ECT session for the following few days.

Upon discharge, given the limited improvement of MH overall, the patient self-discontinued donepezil and trifluoperazine, but he continued quetiapine, clonidine and buspirone, which he found moderately effective. One final medication trial included slow titration of carbamazepine with a maximum dose of 600 mg in the morning and 800 mg at bedtime, which yielded a blood level ranging from 9 to 11 μg/ml. With each dose increase, he reported several days of improvement followed by return of prior MH intensity. At last contact, his hallucinations persisted in an attenuated form.

1.2. Phenomenology of MH in our patient

Over the course of treatment, he reported extremely loud, vivid and complex hallucinatory phenomena with intact reality testing. Initially, the experiences were characterized by the sound of a blaring alarm that awoke him from sleep, and from that time onward, he experienced all-but-constant auditory hallucinations. He described the sounds as variously operatic, mish-mashes of popular songs, melodic, whooshing or hissing sounds, classical music and many others. Although he intermittently heard vocals with the songs, he reported never hearing any percussion or bass. He found the MH distressing as a result of their intensity, pitch and incessant quality; at times, he experienced sheer cacophony. He discovered that being in the presence of loud ambient noises such as a passing train or white noise provided temporary relief and that, in the presence of moderately loud rhythmic noises and music, his MH would synchronize with these environmental tempos.

1.3. Neurobiology of MH

The pathophysiology of MH is poorly understood. Preexisting hearing loss represents a frequent but unnecessary condition in the development of MH. As with Charles Bonnet syndrome, MH often represent a release phenomenon that occurs in the context of diminished sensory input to the auditory cortex. Such differentiation is thought to create a state of excessive neuronal excitability [17]. Brain imaging studies such as positron emission tomography with the use of fluoro-2-deoxy-D-glucose, functional magnetic resonance imaging, single photon emission computed tomography and electroencephalography with patients presenting with MH have revealed cortical dysfunction of the temporal lobe corresponding to Wernicke's area [2,18–21]. In addition, several studies have shown hyperactivity in the prefrontal cortex, with additional involvement of the cingulate, striatum and basal ganglia [22]. These areas are involved in processing and production of speech as well as emotion processing, attention and memory. It appears that changes in activity gradients, focal brain lesions or inflammation within these brain areas is able to induce MH. Additionally, sensory deprivation or evidence of lesions in or decreased perfusion of connecting pathways between primary acoustic areas and associated ascending and descending fibers has been demonstrated to cause similar presentation of MH [23]. For instance, one case study documents the reduction of auditory hallucinations for a limited time after the injection of the sodium channel blocker lidocaine by decreasing activity in cortical areas involved with speech, attention and memory [24]. In general, evidence implicates a cholinergic deficit in hallucinatory syndromes [1,2,25–27]. This hypothesis is further supported by the observation that anticholinergic medication use is associated with worsening of symptoms [26]. Patients who are more prone to present with MH, such as the elderly or those with underlying central nervous system disorders, also may have heightened central nervous system response to inflammatory mediators in the absence of acetylcholine.

1.4. Treatment of MH

In the absence of large-scale, randomized clinical trials, recommendations on the treatment of MH necessarily rely heavily on clinical judgment and the limited case reports published in the literature. The first step in the treatment of MH often involves reassurance on the benign nature of the phenomenon. When the sensory experience is not unpleasant or intrusive, patients may opt for