

CASE REPORT

Understanding a Strange Phenomenon: Lilliputian Hallucinations

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Abstract

Lilliputian hallucinations are infrequent but reported in a variety of states ranging from toxic metabolic states, medical, neurological and psychiatric conditions. We report three cases with different psychiatric diagnoses all presenting with vivid Lilliputian hallucinations. An attempt to apply the current biological mechanisms that explain complex visual hallucinations to Lilliputian hallucinations is made. The phenomenon of size constancy and a functional break between the visual cortex and association cortices explains the phenomena of micropsia, a sensory distortion. We propose whether this principle could also determine the form of hallucinations and produce Lilliputian hallucination (German J Psychiatry 2007; 10: 21-24).

Keywords: Lilliputian Hallucination, schizophrenia, micropsia

Received: 27.7.2006

Revised version: 17.11.2006

Published: 23.1.2007

Introduction

Lilliputian hallucinations are formed complex visual hallucinations of people, objects or animals greatly reduced in size (Asaad and Shapiro 1986). The hallucinations are vivid and evoke varied responses including fear, anxiety or even pleasure (Leroy 1909; Yawgner 1925; Savitsky and Tarachow 1941). The earliest descriptions of this strange phenomenon are attributed to Macarius, who in 400 AD described such hallucinations as 'little strangers' (Cohen et al.1994). The widespread use of the term Lilliputian Hallucination dates back to its description in Gulliver's Travels. Leroy (1909) introduced this term into the scientific literature.

Lilliputian hallucinations have been described in a variety of conditions i.e. delirium, dementia, toxoplasmosis, basilar migraine, mesencephalic lesions, schizophrenia and alcohol withdrawal states (Harper and Knothe 1973; Lishman 1987; Cohen et al.1994; Hendrickson and Adityanjee 1996; Takaoaka and Takata 1999; Podoll and Robinson 2001). What

underlies the formation of these special types of complex visual hallucinations in such diverse conditions? We attempt to address this issue by first reporting three unusual cases seen in our psychiatric practice, followed by a review attempting to integrate literature from different specialties.

CASE REPORTS

Case 1

Mr. R, a forty-year married male, with 10 years of formal education was brought to the emergency with complaints of irritability, wandering and running away from home, abusive and violent behavior of six months duration. History also revealed hallucinatory behavior and gross decline in personal care.

During serial mental status examinations, he complained of hearing multiple male voices discussing him. He also re-

ported that his thoughts were being broadcast in television and radio but did not elaborate further.

In addition, the patient reported seeing small sized people "looking like dolls" all over his food. He was very frightened that these small sized people would choke him. He therefore stopped taking food and on being forced, would vomit it out. After a few days, he saw these 'doll like' people chasing him and trying to mutilate him. He was very fearful and on few occasions tried to run away from the ward. His mood was flat. His orientation and cognitive functions were intact.

Patient's past psychiatric history was unremarkable. Patient's mother had a history of post-partum psychosis, from which she had recovered following treatment. His elder brother had history suggestive of alcohol abuse.

General and systemic examination at the time of admission did not reveal any significant abnormality. Baseline investigations including blood counts and serum biochemistry were normal. Electroencephalography showed occasional sharp waves in frontal areas. Magnetic Resonance Imaging of brain did not reveal any pathological findings.

A diagnosis of paranoid schizophrenia was made and the patient received tablet risperidone up to 8mg over the next four weeks. As his response was poor, he was shifted to olanzapine up to 20mg without significant change. He then received a course of seven electroconvulsive therapies with some behavioral improvement, but psychopathology persisted. Inj. zuclopenthixol decanoate 200mg once in two weeks was added with tablet olanzapine and patient showed considerable improvement.

Case 2

Mr. B, a twenty-five year, middle class educated married male was brought to hospital with excessive consumption of alcohol and abnormal behavior. History revealed that he was a regular drinker since four years and had increased the amount of alcohol for the last two years. His drinking pattern fulfilled International Classification of Disorders-10 criteria for alcohol dependence syndrome. His usual quantity of daily drink was around one to one and half liters of alcohol (approximately 50 units).

Two weeks prior to admission, the patient had decreased the amount to half to one fourth of regular intake. Following this, his sleep had markedly reduced and he started seeing little people all over the house. They were about a foot high, with funny colorful dresses, weird faces, big eyes and mouths. Some of them were also wearing spectacles. They would follow him all around the house and he could hear their footsteps. Patient would also see them drinking his blood (did not elaborate further) and complained of physical weakness as a result. Initially, patient attributed his experiences to some evil spirits present in the house and changed the house. But the experience continued. Informants did not report any past or current history of withdrawal seizures or any physical problems.

Mental status examination revealed that patient was partially oriented to time and place. He complained of visual halluci-

nations of Lilliputian nature and secondary delusions of persecution. He denied any other psychopathology and had impaired insight. General physical examination showed tremors, which were more marked in the extremities. Physical investigation showed raised liver enzymes. Computerized Axial Tomography of brain was normal. His hallucinations, along with other withdrawal symptoms subsided completely within three to four days of abstinence along with tablet lorazepam 8mg/day on first day and tapering down 2mg every alternate day.

Case 3

Mrs. C, a case seen by author 2 in the UK, was an eighty-year-old widow with an eighteen-month history of diminution of vision and visual hallucinations. She reported seeing very short men, women and little girls wearing blue and /gray leaves, whom she referred to as "*blue-bottles*". They would peep at her from cupboards and doors, or hide behind the curtains or lie on the carpets. She would see them eating yellow, black fish like creatures and they would enjoy watching her undress. Sometimes she would see little black cats roaming inside the house. Apart from these, she also complained of multiple tactile and olfactory hallucinations. She was initially afraid of the *blue-bottles* and could not sleep at night.

Historical and cross sectional mental status examination did not reveal any features suggestive of cognitive decline, mood symptoms or Schneiderian first rank symptoms. Physical examination revealed bilateral decrease in visual acuity i.e. left eye 0.5ft, right eye one foot and osteoarthritis of left knee. Computerized Axial Tomography scan of head revealed age related changes.

On mental status examination patient reported visual hallucinations and auditory hallucinations detailed earlier. She also looked fearful and had ideas of persecution secondary to the perceptual abnormalities. She had intact cognitive functions with partial insight to her problem. She was diagnosed as suffering from Charles Bonnet Syndrome. Patient was initially treated with tablet flupenthixol 1mg/day, thioridazine 50mg/day and diazepam 5mg/day, which were later withdrawn in next one week. She was detected to be suffering from glaucoma with retinal artery occlusion and prescribed timoptol eye drops by the ophthalmologist. Gradually her visual hallucinations subsided dramatically both in frequency and in severity. She was discharged after one month of hospital stay.

Discussion

All the three patients reported here with diverse psychiatric syndromes (schizophrenia, complicated alcohol withdrawal and Charles Bonnet Syndrome) experienced visual hallucinations of Lilliputian nature along with other psychopathology.

In the first case, the patient suffered from schizophrenia. Visual hallucinations have been fairly frequently reported in

schizophrenia, as much as in 72 % of patients interviewed, along with hallucinations in other modalities (Bracha et al. 1989; Mueser et al. 1990). This contradicts conventional thinking that visual hallucinations are indicative of underlying structural damage on the one hand, and on the other, supports that schizophrenia is a brain disease. Although Lilliputian hallucinations were first described in schizophrenia by Lewis (1961) and have been subsequently reported (Hendrickson and Adityanjee 1996), hallucinations of this nature are rare in schizophrenia.

In case of alcohol withdrawal syndrome, illusions and hallucinations have been reported in the literature. Rats, snakes and other small animals of Lilliputian nature can appear in vivid and colorful forms. They may have a lot of movement i.e. dancing or playing are quite common (Lishman 1987). In our index second case, patient had similar hallucinations of seeing little funny people following him and demanding his blood.

In the third case, an elderly lady with partially impaired vision without any cognitive dysfunction presented with Lilliputian hallucinations along with other perceptual abnormalities. This clinical presentation is suggestive of Charles-Bonnet Syndrome, which is characterized by vivid, complex and recurrent visual hallucinations associated with eye pathology in a cognitively intact person (Gold and Rabins 1989). The characteristic findings of the index case were that Lilliputian hallucinations subsided with the treatment of glaucoma.

The above three cases have three different diagnosis but interestingly all presented with visual hallucinations of Lilliputian nature, suggesting a possible dysfunction or involvement of either the same areas or pathways in the central nervous system to produce similar phenomenology.

Complex Visual Hallucinations - Underpinnings

Manfred and Andermann (1998) have published an extensive review on the pathogenesis of complex visual hallucinations occurring in different conditions i.e. hypnagogic and hypnopompic states, peduncular hallucinosis, narcolepsy-cataplexy syndrome, Parkinson's disease, Lewy-Body Dementia, Charles Bonnet Syndrome, schizophrenia, delirium tremens and epilepsy. In Charles Bonnet Syndrome, the hallucinations occur in psychologically healthy individuals who have recently become blind or have impaired vision. These are called release hallucinations because it is thought that they are 'released' or instigated by the 'removal of normal visual afferent input into the association cortex'. It is found that direct stimulation of visual cortex area in epilepsy and loss of inhibition on this area in vascular stroke involving occipital area produce similar visual hallucinations. Most of these conditions are associated with disturbance in the sleep-wake cycle indicating involvement of thalamus and fibers radiating to reticular activating system.

Another insight in understanding these condition has come from the use of hallucinogenic substance i.e. LSD and mescaline, potent serotonin agonists (5HT₂) which on intoxica-

tion produce similar complex hallucinations. Apart from serotonin, GABA is also implicated in some of these conditions (Manfred and Andermann 1998).

Extending the theory to Lilliputian Hallucinations

Lilliputian hallucinations are also a type of complex visual hallucinations and can be interpreted on the above theories. At this point of time there is no separately defined neurological basis for this strange phenomenon.

Traditional phenomenology differentiates hallucinations (without afferent sensory signals) from illusory percepts (false percepts with afferent sensory signals) but it is unlikely that these phenomena have different neural substrates (Ffytche and Howard 1999). On the same lines, we also suggest that an analogy could be drawn between Lilliputian hallucinations and the so called 'Lilliputian Sight' or micropsia which is a sensory distortion rather than a false perception.

Micropsia or the 'Alice in Wonderland Syndrome' where objects are perceived substantially smaller than in reality is a condition of altered perception where the mechanics of the eye are not affected, only the brain's interpretation of information passed from the eyes (Takaoka and Takata 1999).

The gestalt psychologists have demonstrated that retinal afterimages change their size depending on where the image is projected (Emmert's Law) (Emmert 1881). Micropsia, an illusion is hypothesized as a dysfunction of the mechanism that underlies the "size constancy" phenomenon. Size constancy denotes the tendency of animals and humans to see familiar objects as having standard shape, size, color, or location regardless of changes in the angle of perspective, distance, or lighting. The impression tends to conform to the object as it is or is assumed to be, rather than to the actual stimulus. Perceptual constancy is responsible for the ability to identify objects under various conditions, which seem to be "taken into account" during a process of mental reconstitution of the known image. Even though the retinal image of a receding automobile shrinks in size, the normal, experienced person perceives the size of the object to remain constant. Indeed, one of the most impressive features of perceiving is the tendency of objects to appear stable in the face of their continually changing stimulus features. Though a dinner plate itself does not change, its image on the retina undergoes considerable changes in shape and size as the perceiver and plate move. What is noteworthy is stability in perception despite gross instability in stimulation. Such matches between the objects as it is perceived and the object as it is understood to actually exist (regardless of transformations in the energy of stimulation) are called perceptual constancies (Tovee et al. 1996).

Precise neurobiological details on the locus of the lesion responsible for micropsia are lacking in most of the reported cases. An overview of the literature reveals that the deficit is much more common after damage of the visual association cortex (Frassinetti et al. 1999). More recently, Cohen et al. (1994) described two cases of hemimicropsia resulting

from a lesion affecting the lower part of areas 18 and 19 and the underlying white matter. In the patients described by Ceriani et al. (1998), who complained of seeing objects smaller than they were in the whole visual field, the lesion apparently involved the right temporoparietal cortical junction and the occipital white matter.

It is proposed that anything that produces a functional imbalance between intact and damaged visual areas is likely to produce micropsia (Bender and Teuber 1949). If this explains the dysfunction of size constancy for objects in the external objective space, the same theory can explain decisions of size the hallucinating brain makes, due to functional disconnections between primary and association visual cortices. So hallucinations of Lilliputian nature might be described as micropsia for internally generated images or percepts. After all, hallucinations themselves are a behavioral manifestation of brain function, just as is interpreting reality the way we do. The different conditions that produce Lilliputian hallucinations, all have evidence suggestive of underlying neurobiological disturbances, the details of which are still poorly understood.

We have attempted to postulate a common mechanism for strikingly similar phenomena in three different conditions. More studies in the form of functional neuroimaging are necessary to understand and establish the pathophysiology of this strange phenomenon.

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