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REVIEW ARTICLE

Why do some People see Ghosts?

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ABSTRACT

Visual illusions and hallucinations differ in whether the perceived objects exist in reality. visual misperceptions have a common underlying neural mechanism. The hallucinators in parkinsonism were significantly older and cognitively impaired.Previous structural brain magnetic resonance imaging studies suggest volume loss in the mesial temporal lobe and limbic regions in subjects with Parkinson's disease with visual hallucinations, relative to those without visual hallucinations.Grey matter atrophy in the hallucinators occurred predominantly in brain regions responsible for processing visuoperceptual information. Structural changes in the brain occurred independent of cognitive function and age.Identification of distinct structural differences in magnetic resonance imaging associated with hallucinations in Parkinson's disease may permit earlier detection of at risk patients and develop therapies targeting hallucinations and visuoperceptive functions.

Keywords:Parkinson’s disease, dementia, hallucinations, magnetic resonance imaging,cortical atrophy.

INTRODUCTION

Influence of cultural folklore on the patterns of hallucinations:

Visual hallucinations were experienced by a patient with progressive posterior cortical atrophy (PCA) which was very similar to Japanese folktales and personal ghost stories.Neurological examination showed Bálint syndrome accompanied by dressing apraxia, constructional apraxia, agraphia, and dyscalculia. Magnetic resonance imaging (MRI) revealed bilateral parieto-occipital, occipital and mild left temporal cortical atrophy and xenon computed tomography (CT) perfusion showed decreased cerebral blood flow in the bilateral parietal and frontal regions[1].Context affected the content of false perceptions through the activation of stored beliefs and cultural values, that differ between individuals.Thus giving a mechanism for the effect of context on idiosyncratic content of hallucinations in schizophrenia[2].Traumatic grief, included a prominent component of separation distress characterised by yearning and searching with frequent bitter and sweet recollections of the deceased, was associated with long term dysfunction. Traumatic grief treatment protocol was aneffective intervention[3].The Cambodian panic response to sleep paralysis (SP)was greatly reinforced by elaborate cultural ideaswith SP generating concerns about physical status, good luck or bad luck status, sorcery assault and ghost assaultand by trauma associations[4].Dissociation and depressive symptoms and misinterpretations of seeing a ghost etc., were more common among those who had experienced sleep paralysis than among those who denied having experienced it[5].

A bimodal distribution was found for 20 and 22 carbon unsaturated fatty acids in RBC membranes of schizophrenic patients.The same fatty acids in normal RBC membranes showed anunimodal distribution[6].Understanding of the genetic mechanisms underlying the evolution of the recent human brain regions and paleoneurologyis the key to get solutions for the focal, asymmetrical or systemic character of neurodegeneration, the pathologic heterogeneity and overlap of syndromic presentations associated with gait, hand, cognition,language, mood and behaviour disorders[7].A number of different neuropathologic

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disorders like optic ataxia, simultanagnosia, ocular apraxia, alexia, acalculia and visual hallucinations, were associated with posterior cortical atrophy[8].

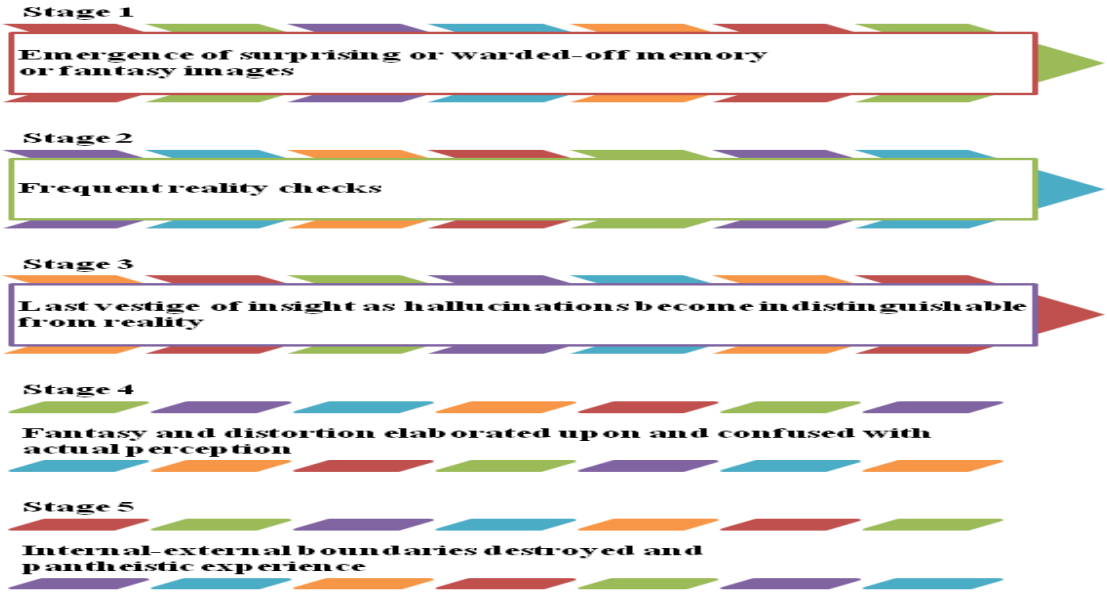

Figure 2: Stages of hallucination

Cortical atrophy associated with neurodegenerative disorders:

Hallucinations in patients with PCA were associated with parkinsonism, rapid eye movement sleep behavior disorder, and myoclonic jerks. The voxelbasedmorphometry results showed that hallucinations in PCA involves a circuit of thalamocortical connections along with atrophy of the posterior association cortices[9].Clinical manifestations of PCA caused by Alzheimer's Disease (AD) included visual agnosia, cortical blindness, optic apraxia, delusions, hallucinations, agitation, amnestic deficit,depression, Wernicke's aphasia, acalculia, and disorientation. Atrophy was demonstrableby magnetic resonance imaging, positron emission tomography, and electroencephalography.Etiology of atrophy was revealed by tissue examination[10].The neural networks involving visuoperceptual pathways contributed to the pathophysiology of visual hallucinations whencognitive performance was similar in hallucinators and nonhallucinators. This explains the predominant visual nature of hallucinations in Parkinson's disease. Identification of distinct structural MRI differences associated with hallucinations in Parkinson's disease aided in early detection of atrisk patients and to develop targeted therapies against hallucinations and visuoperceptive functions[11].The diagnosis of sporadicCreutzfeldtJakob disease sCJDhas be considered in cases of rapidonset dementia even when the clinical criteria are not present. The detection of the 14.3.3 protein and multifold increase in total Tau with normal or slightly increased phosphorylatedTau in the CSF further aid the diagnosis[12].Correlation analysis between cortical thickness and Neuropsychiatric Inventory hallucination item scores showed that the structural alteration in the dorsal visual regions of superior parietal gyrus and precuneus correlated with the occurrence and severity of VH. The study suggests that structural changes in key regions of the dorsal visualnetwork play a crucial role in the physiopathology of VH in patients having dementia with Lewybodies[13].The relationship among subtypes of Parkinson's Disease (PD) subgroups, and the relationship between cognitive,motor and psychiatric factors in PD was discussed in a study done by Moustafa AA andPoletti M 2013[14].Dementia with Lewy bodies (DLB)usually affected elderly individuals in Chinese population. Clinical features were dementia, fluctuating cognition, recurrent visual hallucinations and spontaneous features of parkinsonism[15].

The frequency of the C9ORF72 hexanucleotide repeat expansions in a population of schizophrenia patientsdid not influence the phenotype significantly[16].The role of the occupational therapy in using the Describe, Investigate, Create and Evaluate(DICE) approach for Neuropsychiatric symptoms (NPS) management was described in a study done by Fraker J et al 2014 as a nonpharmacologicalapproach of management of NPS in clinical settings[17].Patients with Charles Bonnet syndrome developed dementia with Lewy bodies, suggesting

that delirium, dementia with Lewy bodies, and Charles Bonnet syndrome must be considered as potential inducers of visual hallucinations in the elderly for differential diagnoses[18].Hallucinations in LBD and euphoria and disinhibition in frontal variant frontotemporal dementia (FTD)were related to the structural brain alterations responsible for cognitive decline in dementia patients. Damage to the frontal cortical areas involved in executive functions, resulted in apathy[19].Assessments done on community dwelling demented individuals, by usingbehavioral pathology in Alzheimer's Disease Rating Scale, Neuropsychiatric Inventory, Etiological Assessment of Psychotic Symptoms in Dementia, Activities of Daily Living, and MiniMental State Examination, revealed that family members and staff members perception varied in different parts of the total picture. The combination of both points of view is essential in order to establish an accurate, comprehensive assessment of dementiasymptoms and to enhance the understanding of the reality of the different parties[20].Considerable reduction in frequency and content of auditory verbal hallucinations was observed in a patient treated with a highfrequency temporoparietal (T3P3) transcranial magnetic stimulationprotocol[21].

Effect of drug therapy:

Significant decreases were found in the following Neuropsychiatric Inventory (NPI) total score and five NPI subscales symptoms of delusions, hallucinations, agitation, irritability, and aberrant motor behaviour by usingmemantine therapy.But no significant differences were seen between the memantine therapy group and the control group[22].Psychosis was associated with volume loss in specific regions of the lateral parietal and frontal lobes including anterior cingulate gyrus[23].High prevalence of psychotic phenomena was found in patients who had C9ORF72 mutations as well as FTD or amyotrophic lateral sclerosis[24].Treatment seeking was predicted by how individual felt about hallucinations, but patient's decision was not logically consistent. This suggests that treatment must be provided based on patients' recollections and opinionsabout hallucinations[25]. Mirtazapine improved refractory psychotic symptoms, and visual hallucinations, without worsening of motor symptoms in Parkinson's disease with psychosis[26].It was found that misattribution of an externally generated source which was either held as a memory or as a traumatic re experiencing the same as an internally generated one, underlies the generation of hallucinations in some non psychotic children[27].Early in the course of psychotic illness, external source monitoring bias was not contributing to the cognitive processes underlying hallucinations thus disproving the theory of linking childhood trauma and external source misattribution[28]. Excessive neuronal noise was found in specific thalamic nuclei in the generation of hallucinations in schizophrenia. Nicotinic receptor abnormalities, dopaminergic hyperactivity and glutamatereceptor hypofunctionwere reconciled within a model of psychotic symptom generation that placed emphasis on dysfunction of the reticular thalamic nucleus[29].Clinical continuity was found between early and later onset schizophrenia, suggesting that very early onset schizophrenia is more severe form of disorder and secondary to greater familial vulnerability[30].Multiple sclerosis (MS)was associated with deficient rapid eye movement (REM) sleep inhibitory neural mechanisms resulting in sleep paralysis secondary to the intrusion of REM sleep atonia and dream imagery into the waking state. Pineal melatonin and monoaminergic neurons were implicated in the induction and maintenance of REM sleep[31].Psychotic symptoms were commonin overloaded individuals (stressed) and were treated with risperidone[32].Caregivers providing care to patients with Alzheimer disease experienced multidimensional problems, but could not find any professional support, suggesting that theyneeded institutional assistance[33].The link between psychotic and psychotraumatic symptoms were complex and multidirectional. Personality structure of the subject must be considered to articulate the psychotherapy and the needed pharmacological treatment[34].

Mental state in post traumatic stress disorder:

Anxiety and depression were common after stays in intensive care units (ICU), and is often mixed with PTSD, which includes fear, feelings of horror, helplessness, avoidance, neurovegetative symptoms and intrusive thoughts[35].Maintaining ICU diaries has shown to have potential to fulfil the existential needs of patients who struggle to make sense of their experiences and construct their own illness narratives[36].For the first time, PTSD was caused by the misattribution of mental states accompanying a seizure[37].

Hallucinations in children:

Magnetic resonance imaging of the brain demonstrated lesions in keeping with healed

parasitic disease of neurocysticercosis in a patient from third world nation. She also had hallucinations in the context of the relationship between a traumatic childhood and psychosis. The hallucinations resolved as stepmother became more available and reduced use of antipsychotic medication[38].Typical symptoms of excessive daytime sleepiness, cataplexy, sleep paralysis, hypnagogic/hypnopompic hallucinations were reported in children with narcolepsy. Additional features of obesity and nocturnal bulimia were present along with poor school performance and emotional disorder[39].Significant associations were found withfamily dysfunction, family breakup in a population of non psychotic children who had auditory hallucinations. Anxiety and depression were also present. Some children reported the presence of imaginary companions[40].

The study done on late onset PTSD patients with traumatic experiences from World War II, revealed that the psychotic PTSD patients were significantly older, later traumatized, dementedfrequently, and widowed. The contents of psychotic features were related to early life traumatic experiences[41].Psycho educational program on schizophrenia increased the knowledge and attitude of caregivers suggesting that the interventions for successful treatment and rehabilitation of schizophrenic patients must include the caregivers also for good results[42].Pan dysmaturationwas reported from the first months of life in children who later develop into childhood onset schizophrenia, suggesting a more severe and early disruption of brain development than in adolescent and adultonset disorders[43].History of neurological diseases, thrombolytic treatment, reduced left ventricle ejection fraction <40%, handicap of visual ability, amiodarone or atropine in pharmacological treatment and nicotinism were found to be important factors to predict psychiatric abnormalities in patients with acute myocardial infarction [44].Acute manifestations of hallucinatory phenomenon in young children, with a focus on tactile, visual and phobic hallucinations was discussed in study by Pao M *et al* 2004. Symptoms were anxiety based, shortlived and occurred at night[45].Inclusion of the family and close friends in the treatment strategy was benevolent as a complement to drug therapy and psychotherapy given to the adolescent[46].Negative symptoms of ongoing hallucinations and delusions in a family member were the most disturbing to normal siblings. Social isolation and lack of motivation were the symptoms of the illness (schizophrenia) but not due to laziness[47].

In a study done on latah in a malay extended family, performers were found engaged in conscious, ritualized social gain through the apparent exploitation of a neurophysiological potential.Latahwas a social construction of western trained universalist scientists as a concept of malingering and fraud[48].Children with conduct and emotional disorders having auditory hallucinations were found to be older than other children seen with similar diagnoses[49].

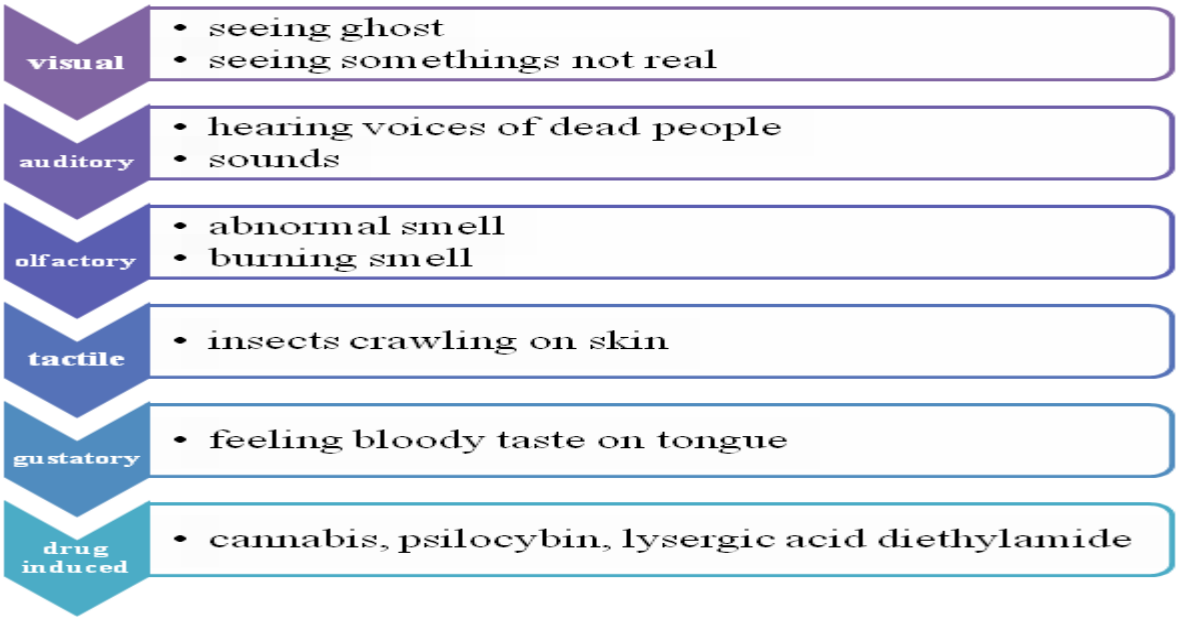

Figure 1: Types of hallucinations

An in depth understanding of hallucinations in monozygous quadruplets concordant for schizophrenia was given in a study done by Rosenthal D and Quinn OW 1977. Concordance was due to the effect of genetic factors[50].Nonpsychotic patient with hallucinations was a socially immature teenage girl who experienced depression and anxiety due to stress within the family[51].Dementia with prominent visual cortical symptoms, not associated with anterior visual pathology, presenting with dyslexia, visual agnosia, Balint's syndrome, and spatial disorientation was reported in a presenile female patient[52].The PD VH patients showed significant cortical atrophy compared to the PDpatientswith out VH in the bilateral dorsolateral prefrontal cortex, left ventral section of the cingulate cortex, left rostral region of the prefrontal cortex,bilateral primary visual cortex, and secondary visual cortex including the left inferior occipital gyrus, right supramarginalgyrus,right lingual cortex, and left fusiform gyrus. Subcortical atrophy was observed in the white matter of the right parahippocampalgyrus, the bilateral posterior part of the cingulate gyrus, the left lingual gyrus, and the right middle occipital gyrus[53].Language deficits with interindividual variability were found in cortical neurodegenerative diseases[54].

Variability in scene perception was exhibited by individuals with PCA indicating the importance of exposure duration in the perception of complex scenes[55].The visual PCA rating scale was quantitatively reflectedgrey mater atrophy in parietal regions with validity and reliability, making it a valuable tool for the radiological assessment of dementia[56].Visual complaints related to visuospatial and visuoperceptualdisorders,were the manifestations of focal degeneration in the posterior cortical areas called PCA,in a focal variant of Alzheimer's disease[57].

CONCLUSION

False perceptions of a religious type were reported more in participants measuring high on religiosity than those low on religiosity. Context affects the content of false perceptions through the activation of stored beliefs and values, which vary between individuals, offering a mechanism for the effect of context on idiosyncratic content of visual hallucinations in brain disorders. The effect of context and individual differences on falseperception content suggests the need for future work regarding the underlying nature of hallucinations and their treatment. Disturbances in the neural networks involving visuoperceptual pathways and improper release and imbalance in total quantity of neurotransmitters like Dopamine, and Serotonincontribute to the pathophysiology of visual hallucinations and disbeliefs. Medical drugs can control the symptoms, but cannot cure totally. A combination of drug and behavioural therapies are needed.

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