Case Report

Carbamazepine in Treatment of Visual Hallucinations: A Case of Chronic Hallucinatory Psychosis

Sayantanava Mitra, Sourav Khanra, Supriya Kumar Mondal[1], Anjana Rao Kavoor, Basudeb Das

ABSTRACT

Visual hallucinations are commonly present in various neurological and psychiatric conditions such as schizophrenia and other hallucinatory psychosis. Current conceptualization of hallucinations assume pattern completion model of thalamus to be responsible for the origin of this type of the perceptual abnormality and proposes that central inhibition of such circuits may treat hallucinations. We present a case of chronic hallucinatory psychosis with significantly distressing visual hallucinations, resistant to antipsychotics, which successfully responded to carbamazepine. This case illustrates the novel use of an antiepileptic in the treatment of resistant visual hallucinations. Targeted therapy of this kind can be considered in the future, although more evidence is required in this field.

Key words: *Antiepileptic, carbamazepine, resistant hallucinations, visual hallucinations*

INTRODUCTION

Hallucinations have been described since antiquity, and are well-recognized as components of mental illness. Chronic hallucinatory psychosis is one entity which falls under other nonorganic psychotic disorder in International Classification of Disease-10th revision.[1] While auditory hallucinations are more commonly encountered in nonorganic psychotic disorders, visual hallucinations can be seen in a range of neurological, psychiatric and ophthalmological conditions. Antipsychotic medications are the principle mode of treatment[2,3] but they often fail to exert any response in persistent visual hallucinations.

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It has been reported that antiepileptics are useful to treat visual hallucinations in Charles Bonnet syndrome,[4,5] but their efficacy in functional cases is less studied and less reported. We present a case of successfully treated, antipsychotic resistant visual hallucinations in a patient with chronic hallucinatory psychosis by carbamazepine.

CASE REPORT

Mr. MH, a 45-year-old male, residing in urban Jharkhand, India from a poor socioeconomic background, with nil-significant past or family history and no known psychoactive substance use, was admitted to Central Institute of Psychiatry with 4 years' continuous history of seeing images of two-dimensional human figures or faces with associated fearfulness and suspiciousness. The patient had developed low mood and occasional death wishes for last 6 months secondary to these symptoms. Initial evaluation revealed a depressed affect, hopelessness, death wishes, delusion of persecution and reference, and complex visual hallucinations. General and systemic physical examinations were unremarkable. Routine investigations including neuroimaging (computed tomography brain, magnetic resonance

Department of Psychiatry, Central Institute of Psychiatry, Ranchi, Jharkhand, [1]Department of Psychiatry, Murshidabad Medical College, Murshidabad, West Bengal, India

Address for correspondence: Dr. Sayantanava Mitra
Department of Psychiatry, Central Institute of Psychiatry, Ranchi, Jharkhand, India. E-mail: sayantanava@gmail.com

imaging brain and electroencephalogram) were also within normal range. He was diagnosed as a case of chronic nonorganic hallucinatory psychosis.

He had been treated earlier with an adequate dose of atypical (risperidone 6 mg for 6 months and olanzapine 15 mg for 3 months) and typical antipsychotics (fluphenazine decanoate 25 mg long-acting depot every 2 weeks for 9 months) with no improvement in the visual hallucinations. After admission, he was started on haloperidol (initial dose of 10 mg/day, hiked up to 15 mg/day) and fluoxetine (initial dose of 20 mg/day, hiked up to 40 mg/day). After 6 weeks of treatment, patient showed improvement in persecutory delusions and depressive symptoms, but the visual hallucinations persisted unabated. Considering case reports of persistent visual hallucinations that responded to antiepileptic medications in organic Hallucinosis,[4,5] antiepileptic medication was planned to be added to the present regime. Patient was started on 100 mg of Carbamazepine per day that was increased to 800 mg/day over a period of 1 week. After 2 weeks of treatment, patient started showing improvement in visual hallucinations. The frequency and duration of the hallucination improved, and within 4 weeks, visual hallucinations resolved completely.

DISCUSSION

In clinical settings, complex visual hallucinations have varied etiologies, and the treatment varies accordingly. Recent literature suggests that visual hallucinations result from "release phenomenon" due to the modulation of the thalamo-cortical network. Pattern completion in the neural network model of thalamus has been hypothesized in visual hallucinations of various etiologies such as Charles Bonnet syndrome, psychedelic drug consumption, somatic hallucinations in phantom limbs, cognitive hallucinations in schizophrenia, and other psychotic disorders.[6] In this model, hallucinations are postulated to be produced when the thalamus fills deprived areas in order to complete patterns, and hence effective treatment of some of these disorders involve peripheral stimulation along with central inhibition and hence that the neural circuits generating the disorders are depressed according to the proposed model of synaptic plasticity.[6] The ability of carbamazepine to decrease gamma-aminobutyric acid turnover and increase glutamate turnover[7] probably inhibit the hyper-excited neuronal circuits responsible for visual hallucinations, resulting in the required "central inhibition."

Contrary to our present report, few reports on the development of visual and auditory hallucinations following treatment with carbamazepine and other antiepileptics have been published in the past. More recently, complex visual hallucinations have been described with carbamazepine[8] and phenytoin sodium;[9] auditory hallucinations[10] have been reported with carbamazepine at therapeutic serum levels. In these cases, after extensive investigation, the authors concluded that the hallucinations resulted from a direct effect of the antiepileptic drugs and ruled out all other possibilities including migraine and seizures.

Though in our patient visual hallucinations resolved with carbamazepine, rather than having worsened, this is not in contradiction with the past reports. The hallucinations reported in the past are likely to have occurred due to a faulty "central inhibition," where the inhibitory neurons were affected instead of hyper-excited neurons, in vulnerable individuals (seizure or migraine diathesis[8] or genetic predisposition[9]) with rapid changes in serum drug concentrations resulting in "release hallucinations." This proposal of "vulnerability factors" giving rise to paradoxical responses is further supported by the rare occurrence of such hallucinations[10] with carbamazepine use in general clinical practice.

Thus, our case illustrates a novel use of carbamazepine in the treatment of visual hallucinations in the nonorganic psychosis. Often the most challenging cases faced in clinical psychiatry are those with treatment-resistant symptoms, which can prove distressing to patients. Our approach in this case was to combine contemporary ideas in neurophysiology and therapeutic evidence in related disorders and apply these to clinical practice in a targeted way. We recognize this to be a single case of novel therapeutic use of carbamazepine; however, we feel that further research in this field is indicated.

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